

**AN INTERDISCIPLINARY APPROACH TO UNDERSTANDING
LOW BACK PAIN IN ELITE ADOLESCENT TENNIS ATHLETES**

by

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Submitted in fulfillment of the requirements of the degree of
DOCTOR OF PHILOSOPHY

2020

ABSTRACT

This thesis explores potential risk factors for low back pain in elite adolescent tennis players. Low back pain (LBP) is one of the most significant causes for loss of playing time in elite junior tennis players and can result in up to 4-5 months away from competition. Current research has explored lumbar spine abnormalities as well as serve and groundstroke biomechanics in tennis in an attempt to better understand the manifestation of LBP in the sport. However, the vast majority of this research has been cross-sectional and limited to male playing populations, meaning that the mechanisms of LBP remain unclear. To address the lack of knowledge on the mechanisms of LBP and to provide improved evidence-based prevention for players, this course of studies investigated multiple proposed causes for LBP; including lumbar spine abnormalities, serving biomechanics and workload in a sample of elite adolescent tennis players over a 12-month period.

During this study, 19/25 players presented with lumbar spine abnormalities. The most common abnormalities were disc degeneration, pars abnormalities (including bone marrow oedema (BMO)) and facet joint degeneration. Interestingly, the presence of BMO did not influence LBP, with only 3/12 players who presented with BMO experiencing LBP. Further the severity of BMO did not appear associated with LBP, which is contrary to the findings in other sports such as cricket. Lastly, the link between pars abnormalities and facet joint orientation still remains unclear due to contradicting results at baseline and follow-up.

The biomechanics of the adolescent serve, specifically peak leg, trunk and racquet kinematics and kinetics, were not related to the presence of pars abnormalities. However, there was evidence to suggest that the timing of these peak mechanics are. Players with pars abnormalities entered peak right knee flexion and peak lumbar right lateral flexion earlier than those without pars abnormalities. Also, the players with pars abnormalities entered the trophy position (racquet high point) and experienced peak lumbar posterior force later than those without pars abnormalities. Therefore, this provides cause for coaches to reconsider the importance of the timing of the tennis serve in the context of pars abnormalities and potentially LBP.

Lastly, when referring to players who experienced LBP in this cohort, all players had a spike in workload with an acute chronic workload ratio (ACWR) ≥ 1.5 . Previous research has flagged this ratio as a risk factor for LBP, yet the high frequency of these spikes in adolescent tennis players complicates their direct link with LBP.

It is difficult to conclude the specific causes for LBP in elite junior tennis players. This thesis reveals that BMO is not linked to LBP as many players with diagnosed BMO remained pain-free. The order in which players perform key serving events (for example racquet high point) do appear related to the presence of pars abnormalities and potentially LBP. Lastly, the relationship between workload and LBP remains unclear and warrants further investigation. Overall, this thesis contributes to the small but growing research into LBP among tennis players and progressively increases our understanding of LBP risk factors.

STUDENT DECLARATION

I, Molly Connolly, declare that the PhD thesis entitled “An interdisciplinary approach to better understanding low back pain in elite adolescent tennis players” is no more than 100,000 words in length including quotes and exclusive of tables, figures, appendices, bibliography, references and footnotes. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work.

Signature:

A solid black rectangular box redacting the student's signature.

Date: 20/03/2020

ACKNOWLEDGEMENTS

This thesis required help from a significant number of people that I'd now like to acknowledge. This dissertation could not have happened without you!

I want to start by thanking my supervisors – Machar Reid, Kane Middleton and Damian Farrow.

Machar, words cannot describe how grateful I am to have had you as my supervisor. You have the most brilliant mind and it has been so inspiring to work with someone who thinks so differently and creatively. Thank you for working tirelessly to provide your feedback and advice whether it be journal articles, thesis chapters or any other endeavor I embarked on (e.g. biomechanics serve reports). I cannot thank you enough for the opportunities you've presented me external to this thesis within the national academy. I am truly grateful for my experiences with Tennis Australia, especially working as part of the Tennis Lab team.

Kane, where would I be without you! Thank you for putting up with my countless phone calls, office invasions, equipment hogging (bordering on theft) and of course, my repetitive questions. I'm truly thankful for the countless hours you've put into my PhD and for your infinite patience for the entire biomechanics component of my thesis. Whether it be sitting in your office for 4 hours to find the one line of code that didn't work (what a hoot that was!), or explaining how to define anatomical coordinate systems for the billionth time to me, your commitment to helping me never wavered. I'm also thankful for your encouragement in exploring my curiosity within applied biomechanics

and never making me feel silly over some very (let's face it) basic, questions! Your help has been truly invaluable to this thesis and my confidence as a sports scientist.

Damian, I was so excited to learn that I'd have you on my team having met you at the AIS during my honours degree. Thank you for being a great help on the VU front by assisting with all the academic components to my PhD; Candidatures, ethics, budgets etc. Your laid back persona has been a great compliment to my (admittedly) stressed and uptight self whenever it came to milestones! I made it through them all (as you said I would) and I'm truly thankful for your support over the last few years at both TA and VU.

Next I would like to thank the radiologists from Victoria House Imaging: **Andrew Rotstein, Frank Malara, Justin Roebert, Rafal Grabinski** and **Tomas O'Shea**. The MRI component of my thesis has been my absolute favourite! I've learned SO much from you and I'm truly grateful for your contributions to this thesis. I'm especially thankful for your hard work in not only analyzing all the MRI scans, but completing the familiarity tests, reliability tests as well as learning a heap of new grading systems for each abnormality. I want to especially thank **Andrew** for your invaluable advice and insights for these studies. You were involved right from the start when I was making the online survey for the scans! You've always been willing to help around your busy schedule and have also been fantastic with all my phone calls and visits to ask you a million questions! You've been incredibly patient with me from the start, and again I'm just so thankful for your input to this thesis.

To my two medical mentors – **Tim Wood** and **Melanie Omizzolo**! The ultimate duo and the horse power to get this thesis going. I wouldn't be writing these acknowledgements if you hadn't have wanted this project as badly as you did! Needless to say, you've both been a HUGE help to me over the last 4 years, I cannot thank you enough. **Mel**, you are the bubbliest person I know – I've always looked forward to our catch-ups! Thank you for always being there for me as a mentor, advisor and friend throughout this journey. Your applied clinical insight has been instrumental for this thesis and I would have been so lost without your help! I'm also thankful for the opportunities you presented me external to this thesis with the professional players. You've encouraged me and believed in what I've been doing from the start, I'm so grateful to have had you in my corner. **Tim**, you have been amazing. I remember when I first met you in the physio room with Dave Whiteside and Mel. I was so nervous to meet the "Chief Medical Officer" of Tennis Australia, though there was clearly no need! You were so so excited that this thesis was going ahead and were mentioning all the things we could look at (facet joint angles, pars, BMO etc) even though I was still figuring out which exact part of the vertebra was the "pars"! I'm sincerely grateful to learn from you these last 4 years and for all your help and contributions to the write-ups.

Next I'd like to thank – **Danielle Gescheit, Graeme Spence, David Whiteside** and **NA Vic Academy!**

Dani, thank you for your constant encouragement and help over the last 4 years. Thank you also for been the queen of inclusion and inviting me to all your exercise groups, Wobbly Leg Wednesday, Tuesday Spin (vomit), boxing and so on. I've genuinely loved being part of the groups. I also want to thank you for lending me the responsibility of

Tournament AMS. It was a small slice of work for TA that made me feel super important! **Graeme**, your R and statistical knowledge was incredibly helpful to me! Thank you for never judging my R-codes (and simply suggesting “alternative” ways to write code!) and for your patience in teaching me the countless statistical processes we tried! I really enjoyed learning from you throughout this experience, thank you for your time in helping me with this project. **Dave**, although around for only a short while, you were the perfect person to help me bring my PhD to life. I was genuinely nervous coming into an entirely new environment and you helped me settle in right away. I appreciate the insights and advice you offered during my first year, I’ve certainly carried them with me throughout the remainder of this PhD. Though if I’m completely honest, thanks for our deep and meaningful conversations pertaining to who our favourite Kardashian was, or our weekly briefings on the latest Geordie Shore episode. I’ve missed you! **NA Vic Academy**, thank you to all the staff at the Victorian National Academy (coaches, S&Cs, physios, performance analysts). I cannot express how much I have enjoyed being a part of this family. I’m going to miss you all so much! Thank you for being so supportive of me over the last few years and giving up/rescheduling your time to allow me to conduct my PhD biomechanics studies. I’ve learned so much from each of you at some point along my journey, I’ll truly miss our hellos every day!

I would also like to thank my fellow Tennis Lab teammates for making this experience an enjoyable one. In particular, I’d like to thank **Olivia Cant** and **Lyndon Krause** for all the fun that we’ve had over the last few years. Words cannot describe how lucky I feel to have had the daily support from each of you. Whether it’s the small things such as muffin runs from Edwins, or critiquing the latest superhero, or the big – reeling in a 150kg bull

ray (!!!) on our annual fishing trip, the experiences I've had with the both of you I'll cherish forever. Thank you for helping with my studies, not because you had to, though because you wanted to. I look forward to more laughs, heated debates and interesting conversations on Lyndon's fishing boat in future.

Furthermore, I'd also like to thank the research assistants that helped me over the last few years, **Laura Johnson, Bryanna Armao and Marika Giamerelos**. My biomechanics studies could not have happened without you!

I'd also like to thank **Tennis Australia** and **Victoria University** for allowing me this opportunity. I've undergone tremendous growth and a researcher and applied sport scientist throughout this experience and would not have been possible without your technical, financial and professional support.

Lastly, I'd like to thank my family and friends. Thanks to **Annabel Smith, Molly Cunningham** and **Sally Godwin** for being incredibly supportive of me throughout this whole process. Be it 6am walks around the Botanic Gardens, beach visits or eating our body weight in Italian in De Graves, I've truly appreciated these times more than you'll ever know. I'm incredibly lucky to have such supportive friends, even if every time we arrange a catch up, I now get the response "depends... are you going to ditch us for Rafa again?"!

To my parents, "**Marns and Bruce**" thank you for always believing in me and being the supportive and proud parents that you are. I wouldn't be where I am today without you! Thank you for the constant encouragement throughout my PhD, I'm truly blessed to have parents as wonderful as you!

Lastly and not least, I'd like to thank my amazing fiancé **Sam Fernandez**. Thank you for your unwavering patience, support and belief in me. Thank you for being by my side through this entire journey, words will never explain just how grateful I am to you. Thank you for being an endless source of laughter and always keeping me smiling. Let's get married!

LIST OF PUBLICATIONS AND PRESENTATIONS

PUBLICATIONS

Connolly, M., Middleton, K., & Reid, M. (2019). Differences in tennis serve kinematics between elite adolescent male and female players. *ISBS Proceedings Archive*, 37(1), 431.

Connolly, M., Rotstein, A. H., Roebert, J., Grabinski, R., Malara, F., O'Shea, T., Wood, T., Omizzolo, M., Kovalchik, S., & Ried, M. (2020). Lumbar spine abnormalities and facet joint angles in asymptomatic elite junior tennis players. (*In review*)

Connolly, M., Middleton, K., Spence, G., Cant, O., & Reid, M. (2020). Lumbar spine abnormalities in elite adolescent tennis players: A link with serve kinematics? (*In review*)

Connolly, M., Middleton, K., & Reid, M. (2020). Is lumbar loading linked to pars abnormalities in elite adolescent tennis players? (*In review*)

OTHER PUBLICATIONS

Whiteside, D., Cant, O., **Connolly, M.,** & Reid, M. (2017). Monitoring hitting load in tennis using inertial sensors and machine learning. *International Journal of Sports Physiology and Performance*, 12(9), 1212-1217.

Busuttill, N. A., Reid, M., **Connolly, M.,** Dascombe, B. J., & Middleton, K. J. (2020). A kinematic analysis of the upper limb during the topspin double-handed backhand stroke in tennis. *Sports Biomechanics*, 1-19.

MEDIA RELATED WORK

Feature news segment on Sky News. This piece promoted Tennis Australia's "Tennis Lab", promoting my affiliated role with Tennis Australia. View the news segment here: https://www.skynews.com.au/details/_6053712209001

Feature article published by Chris Clarey in New York Times. This piece promoted Tennis Australia's 'Racquet Room', promoting my affiliated role with Tennis Australia. View article here: https://www.nytimes.com/2017/02/10/sports/tennis/smart-court-racket-australia.html?_r=0

Feature article published by Will Swanton in The Australian. This piece promoted Tennis Australia's and Victoria Universities 'Racquet Room', promoting my affiliated role with Tennis Australia. View article here: <https://www.theaustralian.com.au/sport/tennis/the-bazooka-nadal-will-use-to-defend-french-crown/news-story/06aa33a8b2c542e3e880729c0b4c3055>

Feature article published by Simon Briggs in The Daily Telegraph. This piece promoted Tennis Australia's and Victoria Universities 'Racquet Room', promoting my affiliated role with Tennis Australia. View article here: <https://www.pressreader.com/similar/282660393166387>

SCIENTIFIC CONFERENCE PRESENTATIONS

Connolly, M., Pease, D., Gordon., B. (2016). “The use of Inertial Measurement Units to assess velocity during freestyle swimming”. Presented at the 2016 Exercise and Sports Science Australia conference in Melbourne, Australia.

Connolly, M., Middleton, K., Reid, M. (2019). Differences in tennis serve kinematics between elite adolescent male and female players.” Presented at the 2019 International Society for Biomechanics in Sport conference in Ohio, USA.

COACHING CONFERENCES

Connolly, M. (2018). “A multidisciplinary approach to understanding low back pain in elite adolescent tennis players”. Presented at the 2018 Tennis Australia High Performance Workshop, Canberra, Australia

Connolly, M. (2019). “A multidisciplinary approach to understanding low back pain in elite adolescent tennis athletes”. Presented at the 2019 Tennis Australia National Coaching Workshop, Melbourne, Australia

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CHAPTER 1
INTRODUCTION & OVERVIEW OF THESIS

1.1. INTRODUCTION

1.1.1. LOW BACK PAIN

Low back pain (LBP) is a multifactorial condition (Balagué, Dudler, & Nordin, 2003) that is primarily associated with repetitive stress as a mechanism of injury in athletes (Hjelm, Werner, & Renstrom, 2010; Swärd, Eriksson, & Peterson, 1990). As such, there are both mechanical and workload considerations in the development of this pathology. The condition is also associated with a variety of underlying morphological abnormalities, which can only be detected through diagnostic imaging.

1.1.2. LOW BACK INJURIES IN TENNIS

LBP is a symptom and not a diagnosis (Mortazavi, Zebardast, & Mirzashahi, 2015), though in high performance sport LBP must not be ignored as it might be related to a lumbar spine injury. For example, unlike most other sports, tennis playing schedules involve year-round competition and can subsequently lead to a high prevalence of overuse injury. They are particularly prevalent amongst younger players in whom low back injuries account for the largest proportion of all injuries (Roetert & Ellenbecker, 2007) and a considerable amount of lost playing time (Campbell, O'Sullivan, Straker, Elliott, & Reid, 2014; Campbell, Straker, O'Sullivan, Elliott, & Reid, 2013). Indeed, there is recent evidence of rising prevalence of low back injury among elite Australian juniors, the cause of which remains unknown (Figure 1.1). Further, Australian male juniors have been shown to experience low back injuries at a higher rate than Australian female juniors (Figure 1.1)

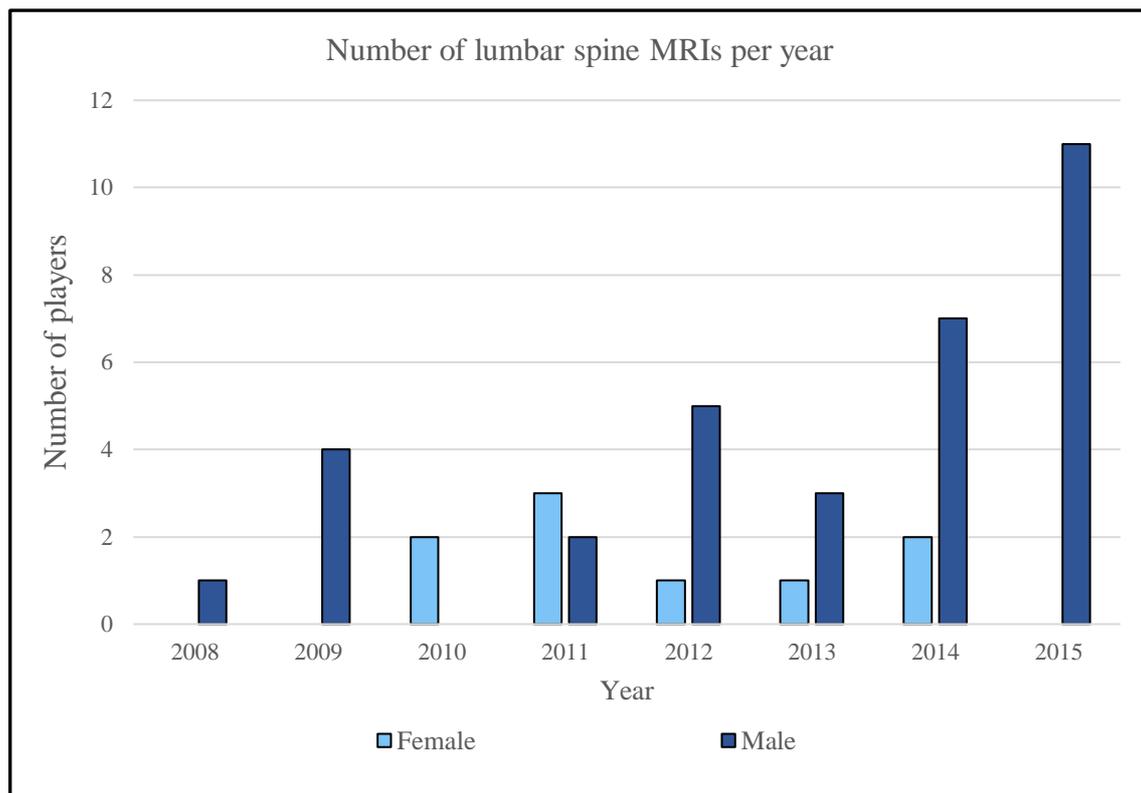


FIGURE 1.1. Number of elite tennis players aged 12-19 years, who underwent a lumbar spine MRI due to LBP.

1.2. STATEMENT OF THE PROBLEM

Whilst there is research investigating low back injuries and pain in young tennis players, it has been descriptive and/or cross-sectional and provided only limited insight into injury causation. For example, studies that have diagnosed morphological abnormalities in tennis players in an attempt to link them to LBP have captured moment-in-time views of the spine with no follow-up (Alyas, Turner, & Connell, 2007; Rajeswaran, Turner, Gissane, & Healy, 2014). Consequently, the mechanisms of LBP remain unclear, complicating evidence-based prevention for players and practitioners. This thesis therefore contributes to the existing base by directly examining lumbar spine abnormalities in the context of (a) lumbar vertebrae morphology, (b) service technique

and (c) workload over an extended period of time. In a departure from previous literature, this thesis investigated lumbar spine abnormalities longitudinally in order to enhance our understanding of these abnormalities in elite junior tennis players.

The aims of this project are:

- To determine the prevalence of lumbar spine abnormalities in asymptomatic male and female elite adolescent tennis players (Chapter 3)
- To investigate whether facet joint orientation is linked to pars interarticularis abnormalities (Chapter 3)
- To examine the relationship between serving kinematics and kinetics and the presence of lumbar spine abnormalities in asymptomatic elite adolescent tennis players (Chapter 4 & Chapter 5)
- To investigate the morphological changes in the lumbar spine and episodes of LBP among 25 elite junior tennis players over a 12-month period (Chapter 6)
- To compare the technical, workload and MRI results of these five players in the context of their LBP (Chapter 7)

1.3. JUSTIFICATION OF THE PROBLEM

This course of studies will comprehensively explore multiple proposed causes of LBP and their relationship with the onset of subsequent lumbar abnormalities and pain. To the knowledge of the researcher, no previous studies have evaluated the risk factors for LBP in tennis through such a multidisciplinary lens. Similarly, past research has stopped short of investigating modifiable risk factors in the context of longitudinal morphological changes.

The intent of this project is to promote a better understanding of player health, specifically LBP in adolescent tennis players as it remains a concerning health problem for young tennis athletes. The findings of this project have the potential to inform player development and the provision of guidelines to prevent future lumbar injuries. Collectively, it is envisioned that these novel data will promote improved lower back health among elite adolescent tennis players in the future.

1.4. HYPOTHESES

Hypotheses for Chapter 3 are:

- The majority of players will have an asymptomatic lumbar spine abnormality
- Bone marrow oedema and/or stress fractures at the pars interarticularis will be evident among a small number of asymptomatic players
- Asymptomatic male players will have more pars abnormalities than asymptomatic female players
- Asymptomatic players with larger facet joint angles will be more likely to have a pars abnormality.

Hypotheses for Chapter 4 are:

- Asymptomatic players with lumbar spine abnormalities will exhibit less dominant (right) side lumbar spine and pelvis rotation during the drive phase but greater non-dominant (left) side lateral flexion, lumbar spine rotation, pelvis rotation and anterior pelvis tilt during the forward-swing phase than players without abnormalities

- Male players will have more lateral impact positions as well as increased drive phase lumbar extension, lumbar lateral flexion and posterior pelvis tilt compared to female players, while the female serve will feature higher ball tosses, larger ball toss drop distances and peak front knee flexion compared to the male serve
- The kick serve will be characterized by greater lumbar lateral flexion and extension than the flat serve as well as a lower ball toss
- The timing of pelvis, trunk and ball toss kinematics will significantly differ between the serves of those with and without pars interarticularis abnormalities.

Hypotheses for Chapter 5 are:

- Lumbar forces will be positively associated with the presence of pars interarticularis abnormalities among all players
- Males will experience greater lumbar forces than females during the serve
- The kick serve will be characterised by higher peak lumbar kinetics than the flat serve
- The timing of peak lumbar kinetics in the serve will be differ between with and without lumbar pathology.

Hypotheses for Chapter 6 are:

- >90% of athletes will present with at least one pars interarticularis abnormality and there will be some instances of LBP among the group
- At least one in five of players will suffer an episode of LBP consistent with previous research

- Players diagnosed with pars bone marrow oedema (BMO) at baseline will develop low back pain in the following 12 months.

1.5. LIMITATIONS

- The natural variation in maturity status of participants and their anthropometric measurements could not be controlled and may complicate the interpretation of the results.
- The assumption that this sample was representative of high-performance players across Australia.
- Error introduced by movement of skin under the markers was not quantified, however the use of rigid marker clusters reduced this error (Eftaxiopoulou, Gupte, Dear, & Bull, 2013).
- The assumption was that players hit the ball with maximum effort for every serving trial.

1.6. DELIMITATIONS

This thesis was delimited to:

- Twenty-five participants within the Tennis Australia National Academy program.
- Results of this study pertain only to adolescent populations and cannot be reliably extended to adult tennis players.

1.7. DEFINITION OF TERMS

1.7.1. MORPHOLOGICAL TERMS

- Lumbar abnormality: a morphological defect within the lumbar spine that is considered “abnormal”
- Spondylolysis: a stress fracture at the pars interarticularis
- Spondylolisthesis: a vertebra slip – i.e. one vertebra slips forward (anterior) to the vertebra below

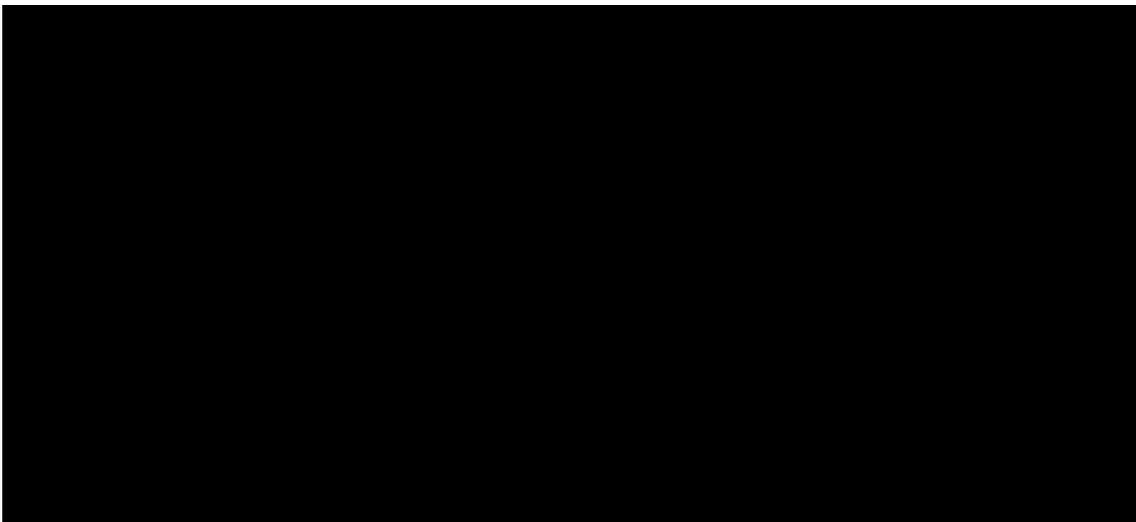


FIGURE 1.2. Visual representation of spondylolysis and spondylolisthesis. (Image from: Pediatric Orthopaedic Society of North America. “Spondylolysis and Spondylolisthesis.” American Academy of Orthopaedic Surgeons. September 2016. <http://orthoinfo.aaos.org/topic.cfm?topic=a00053>> Accessed August 27, 2020.)

1.7.2. MRI SCAN TERMS

- Section/slice thickness: The Magnetic Resonance Imaging (MRI) obtains “slices” of images in a particular direction. Thickness refers to the vertical height of these slices – i.e. axial image 3 mm slice thickness takes a 3 mm slice of the lumbar spine in the transverse plane

- Spacing: similar to slice thickness, spacing refers to how much “space” is between one slice and the next

1.7.3. TENNIS SERVE KINEMATIC TERMS

- Service box: there are two service boxes on either side of the net, to which players aim when serving. Players alternate serving to the service box in the deuce court and then the ad court
- Deuce court: when players stand on the right side of the baseline and the server aims to hit the ball into the deuce court service box
- Ad court: when players stand on the left side of the baseline and the server aims to hit the ball into the ad court service box
- Flat serve: a serve hit with maximal speed and negligible spin on the ball
- Kick serve: a serve hit with a combination of speed and spin with an aggressive out of bounce angle (kick) making the serve hard to return
- Serve type: refers to which type of serve is being used (e.g. flat vs kick serve)

1.7.4. TEMPORAL KINEMATIC TERMS

- Ball toss (BT): the moment at which the ball leaves the server’s hand
- Racquet high point (RHP): the point at which the racquet tip is at its vertical zenith prior to the initiation of a player’s drive toward impact
- Racquet low point (RLP): the point at which the racquet tip is at its lowest point (vertically) following RHP
- Impact: the point at which the racquet impacts the ball

1.7.5. PHASES BETWEEN TEMPORAL EVENTS

- Preparation phase: the time between BT and RHP
- Drive phase: the time between RHP and RLP
- Forward-swing phase: the time between RLP and Impact

CHAPTER 2
REVIEW OF THE LITERATURE

2.1. INTRODUCTION

Tennis is a major global sport with over 87 million participants worldwide (Baseline Tennis, 2017). It is played for personal fitness, leisure and as a competitive sport. Both males and females participate in the sport and it is not uncommon for these players to begin playing in childhood and continue to play throughout life (Bylak & Hutchinson, 1998). The goal of competitive tennis is to win more sets than the opponent (minimum of two sets depending on the format, (Carter & Crews, 1974) unlike many other sports, there are no time limits on match length. Consequently, professional matches can last less than an hour but in excess of five hours, highlighting the physicality of the sport.

The path to becoming a professional tennis player is highly competitive with currently over 6000 internationally ranked junior players and approximately 5000 professional tennis players (International Tennis Federation, 2019) competing year round (Roetert & Kovacs, 2019). These players often compete in 30 events and over 100 matches per year, with each match involving hundreds of shots (Whiteside & Reid, 2017) and considerable accumulative loading on hard and soft tissue over time. If not managed carefully, players can be prone to overuse injury. As a result, research has explored both external (dose of work completed by player, irrespective of physical characteristics) and internal loads (the physiological and psychological response to the external load) in the context of musculoskeletal injury (Halsen, 2014; Wallace, Slattery, & Coutts, 2009) in tennis (Gescheit, Cormack, Reid, & Duffield, 2015).

The service motion is the most explosive and load bearing stroke in tennis. It is also the most crucial stroke in tennis as it is the only shot players have full control over, thus when

executed well, can allow the serving player to win a point with immediate effect. From a mechanical perspective, the serve necessitates repeated trunk counter rotation, lateral flexion and hyperextension which places the lumbar spine under significant stress (Congeni, McCulloch, & Swanson, 1997; Cyron & Hutton, 1979; Ellenbecker, Pluim, Vivier, & Sniteman, 2009; Kelsey, 1980; Roetert, Ellenbecker, & Reid, 2009). As a result, low back pain is a common complaint amongst both junior and professional tennis players and is ranked as one of the most common injuries in tennis (Campbell et al., 2014; Campbell et al., 2013; Gescheit et al., 2019; Gescheit et al., 2017; Hjelm, Werner, & Renstrom, 2012; Hutchinson, Laprade, Burnett, Moss, & Terpstra, 1995). Unpublished data from Tennis Australia's National Academy found that between 2008 and 2015, the number of lumbar spine medical consults increased by 1413% with 711 lumbar spine consults in 2015 alone. Further, the number of serious lumbar spine injuries (those who were followed up with a positive MRI) increased 10-fold and were evident in players between 12 and 19 years of age. The pervasiveness of low back pain isn't limited to adolescent players though, with Grand Slam tennis tournament data revealing that low back pain is among the most common complaints in touring male and female professionals (Gescheit et al., 2017). It's apparent that lumbar spine pathologies have been ubiquitous in tennis for years and actionable insight to limit their prevalence has largely eluded researchers and practitioners.

2.2. EPIDEMIOLOGY OF INJURIES IN TENNIS PLAYERS

2.2.1. THE INCIDENCE OF TENNIS INJURIES

In tennis, injury incidence rates are typically measured by either athletic exposure; where injuries are calculated per exposure to tennis (e.g. match, training session) (Hutchinson

et al., 1995; Kerr et al., 2015; Sell, Hainline, Yorio, & Kovacs, 2012), or by duration of tennis play; where injury rates are calculated relative to a specific time frame (e.g. per 1000 hours of tennis play) (Orchard, James, & Portus, 2006; Silva, Takahashi, Berra, Cohen, & Matsumoto, 2003).

Injury data in adult, professional tennis players can be difficult to interpret. Several reports have investigated injury incidence in professional tennis players, however methods have varied, complicating any injury profile comparisons between sex/tournament/age/body regions. For example, one group of researchers investigated injury incidences at the U.S. Open between 1994 – 2009. They concluded that males were more likely to be injured during tournaments compared to females (Sell et al., 2012). This however is contrary to more recent reports that have suggested professional female tennis players are more likely to be injured during a tournament compared to males (Gescheit et al., 2017; McCurdie, Smith, Bell, & Batt, 2017). The same problem exists when comparing junior injury data. That is, some reports describe elite junior males to be more susceptible to injury during competition (Hjelm et al., 2010; Jayanthi, O’Boyle, & Durazo-Arvizu, 2009), yet others claim the opposite to be true (Kovacs, Ellenbecker, Kibler, Roetert, & Lubbers, 2014; Silva et al., 2003). In summary, the link between sex and injury prevalence in junior tennis players is currently inconclusive and therefore suggests that sex may not be a crucial factor in injury risk. With that in mind, other risk factors must be considered such as age and playing level when considering injury.

There is also variation in the methods used to calculate injury incidence between studies. For example, Sell et al. (2012) calculated injury incidence for the U.S. Open per 1000

match exposures which is consistent with a selection of other studies (Hutchinson et al., 1995; Kerr et al., 2015; Sell et al., 2012). However, McCurdie et al. (2017) reported injury incidence per 1000 sets played during Wimbledon championships between 2003 – 2012. Both methods have known limitations. Calculating injury incidence per 1000 match exposures neglects factors that are associated with injury such as duration, intensity and number of sets played, while the use of the number of sets per match neglects the duration and competitiveness of games (e.g. 6-7 vs 6-0). Consequently, Gescheit et al. (2017) has more recently advocated the use of a smaller denominator such as 10,000 games played to standardize the calculation of injury incidence.

Injury rates in elite tennis vary from 4.7 – 55.6 injuries per 1000 athletic exposures to 0.6 – 3.0 injuries per 1000 hours of playing exposure (Hjelm et al., 2012; Hutchinson et al., 1995; Kerr et al., 2015; Pluim, 2006; Sell et al., 2012). These rates are comparable to other sports such as swimming, cricket and long distance running (Orchard et al., 2006; Ristolainen, Heinonen, Waller, Kujala, & Kettunen, 2009) however contact sports like rugby union (160.6 injuries per 1000 playing hours) generally have higher incidence rates (Lopez et al., 2017; Orchard, Wood, Seward, & Broad, 1998; Starling, Readhead, Viljoen, & Lambert, 2019; Theilen, Mueller-Eising, Bettink, & Rolle, 2016; Toohey, Drew, Finch, Cook, & Fortington, 2019) (Gabbett, 2000). In this context, injury comparisons within any single sport must consider and standardise the age, sex and playing standard of different cohorts.

2.2.2. MECHANISMS OF TENNIS INJURY

Overuse injuries are common in tennis, owing to the repetitive loading during play (Gescheit et al., 2017). These overuse injuries tend to dominate the upper body as opposed to the lower body, which is more susceptible to acute injuries (Abrams, Renstrom, & Safran, 2012). The ankle and thigh are the most frequently injured segments in the lower body (Abrams et al., 2012) as a result of the acute and intense acceleration/deceleration demands and frequent changes in direction (Kibler & Safran, 2005). The elbow and shoulder are the segments most commonly injured in the upper body on account of to the repetitive overhead movements such as the serve, which are executed at high velocities (Kibler & Safran, 2005). Previous research has described a positive relationship between movement velocity and the load applied to the body (Elliott, Fleisig, Nicholls, & Escamilla, 2003) so meaning that faster movers might be at a higher risk of injury. Lastly, the load created by wielding a tennis racquet and the various court surfaces and ball types add to complex epidemiology of injuries in tennis.

2.2.3. TYPES OF TENNIS INJURY

Muscle injuries are the most common injuries for both male and female professional tennis players during grand slam tournaments and require the highest in-event treatment (Gescheit et al., 2017; McCurdie et al., 2017; Sell et al., 2012). Following muscle injuries, tendon and joint sprains are the next most common injuries for males and females respectively (Gescheit et al., 2017). However, injury type is poorly understood in elite junior tennis players, with injury regions (such as shoulder, low back etc.) rather than injury types, being the primary research focus. Among the limited research that has been

performed, strains and sprains have registered as the most common injury types for junior tennis players (Hutchinson et al., 1995; Kibler & Safran, 2005).

2.2.4. REGIONS OF TENNIS INJURY

Unlike other sports (e.g. swimming and running), injuries in tennis exist throughout the body (Abrams et al., 2012). Previous research has found that the lower body accounts for the most injuries in tennis (31-67%) followed by the upper body (20-49%) and then trunk (3-21%) (Abrams et al., 2012; Pluim, 2006; Sallis, Jones, Sunshine, Smith, & Simon, 2001; Winge, Jørgensen, & Nielsen, 1989). However, these reports have varied in sample size, age, sex and skill level of the players, thus drawing conclusions pertaining to common injury regions is difficult.

There is some consistency in injury regions between Grand Slam injury reports for professional tennis players. Injuries to the knee, shoulder and ankle/foot feature prominently (Gescheit et al., 2017; McCurdie et al., 2017; Sell et al., 2012) although both the Australian Open and Wimbledon tournaments reported high incidences of lumbar injuries and in-event treatment for the lumbar spine (Gescheit et al., 2017; McCurdie et al., 2017).

In the junior game, the findings for injury incidence by region are mixed. For example, Winge et al. (1989) reported that upper extremity injuries were most common (45.7%) followed by injuries to the lower extremity (39%) and then trunk (11%). On the contrary, Reece (1986) concluded that more than half (59%) of the 176 injuries sustained in junior players at the Australian Institute of Sport involved the lower limbs, with the remaining

injuries being equally distributed between the upper extremities and trunk. Yet, another study by Hutchinson et al. (1995) revealed the lower back to be the second most frequently injured region, behind the thigh among junior male players competing at the USTA championships. Most recently however, a report by Gescheit et al. (2019), found lumbar injuries to be the most frequent injury among junior male and female tennis players over a four year period, suggesting that lumbar spine is the most common body region injured in elite junior tennis players today.

2.3. LOW BACK PAIN IN TENNIS

In tennis, low back pain and subsequent injuries are pervasive among players (Ruiz-Cotorro, 2006) and if not carefully managed, can cause significant time out of the game. These pathologies are pervasive in both the adult and junior games. For example, in professional tennis, Sward et al. (1990) reported that 50% of professional male tennis players had experienced LBP for at least one week (Swärd et al., 1990), whilst the lumbar spine ranks among the most common medically consulted body regions at the Australian Open (Gescheit et al. (2017)).

Studies have also reported that the lumbar spine was the most common site of injury for both junior elite males (20.5% of injuries) and females (22.2% of injuries) and that nearly 40% of adolescent tennis players whom reported a lumbar injury resulted in missed training and/or competition (Hjelm et al., 2010). These findings are supported by a recent report by Gescheit et al. (2019) who concluded that lumbar spine injuries are the most frequent injuries for both male and female junior tennis athletes. Additionally, other studies involving select groups of elite Australian juniors, have revealed that 1 in 5

players reported disabling LBP over a 12 month window with each incidence resulting in an average of 34 days of missed training (Campbell et al., 2013). Further, between 2008 – 2010, of the players who missed training owing to LBP, almost 40% had bone marrow oedema at L4 and L5 and for players diagnosed with a symptomatic pars abnormality and the average return to play time was ~160 days. Interestingly, these episodes of LBP have been relatively rare among female players (Figure 2.1). The reasons for this however, are unclear.

This prevalence of lumbar spine injuries in both elite junior and adult tennis players highlights the opportunity for not only improved prevention but also further research to better understand the risk factors for LBP in elite tennis players.

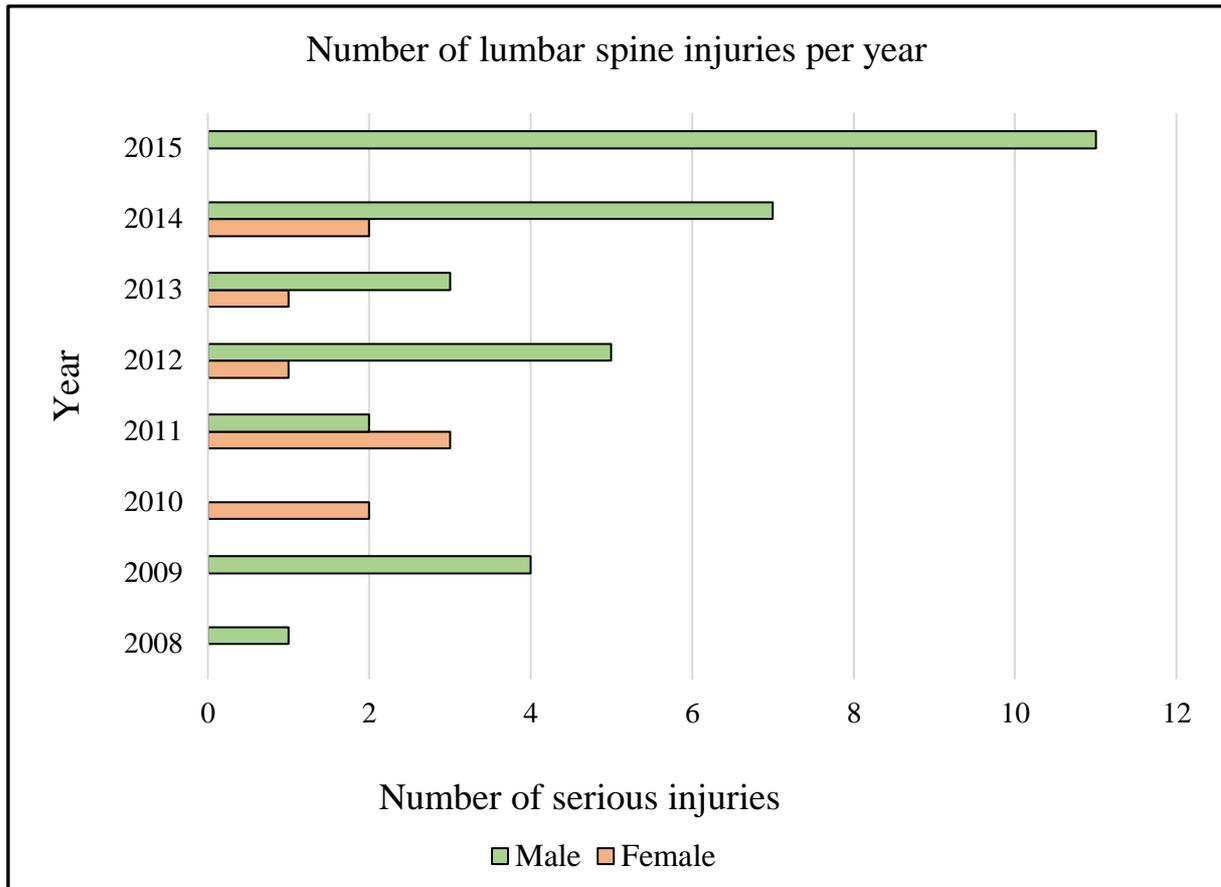


FIGURE 2.1. Number of serious lumbar injuries each year. Unpublished data - *Tennis Australia*

2.3.1. NON-MODIFIABLE RISK FACTORS FOR LOW BACK PAIN IN TENNIS

2.3.1.2. THE SPINE

The spine is a complex formation of 33 bones broken into five sections; cervical (7 vertebrae), thoracic (12 vertebrae), lumbar (5 vertebrae), sacral (5 vertebrae) and coccygeal (4 vertebrae). These bones develop in size and mass (bone deposition and resorption) throughout childhood and adolescence and eventually reach epiphyseal fusion (bone growth is complete, no presence of growth plate) between 20-30 years of age (Weaver et al., 2016). In the lumbar spine, each vertebra consists of a vertebral body (the majority of the bone), four facet joints (two superior and two inferior) which join one vertebra to another and a vertebral disc that sits in-between. The combination of the facet

joints and discs allow three-dimensional movement of the spine while the disc provides cushioning between the vertebrae to reduce vibrations and enhance shock absorption in the spinal column during daily activities (Brzuszkiewicz-Kuźmicka, Szczegielniak, & Bączkiewicz, 2018; Wosk & Voloshin, 1985). The pars interarticularis is a small segment of bone that connects the facet joints to the rest of the vertebra (see Figure 2.2).

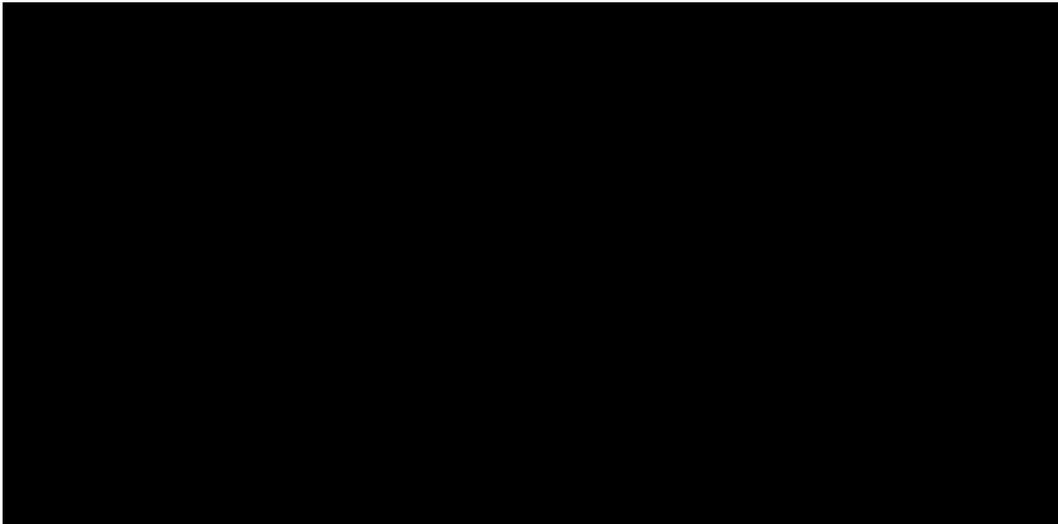


FIGURE 2.2. Lumbar vertebra anatomy

The spine permits rotation in three planes; flexion/extension (forward/backward in the sagittal plane), lateral flexion (side to side bending in the coronal plane) and axial rotations (twisting movement in the transverse plane). In the wider general population, repetitive three-dimensional movement of the spine (bending, twisting, lifting, pushing, pulling etc.) and heavy physical work, have been identified as risk factors for LBP (Van Tulder et al., 2006). Though the plane of movement most associated with spinal stress remains controversial. For example, some authors suggest that the combination of repetitive flexion, extension or rotation of the lumbar spine is most detrimental (Leone, Cianfoni, Cerase, Magarelli, & Bonomo, 2011) while others propose that lumbar flexion

elicits the greatest stress on the spine, regardless of whether other rotations occur concurrently (Chaudhry, Ji, Shenoy, & Findley, 2009).

2.3.1.3. LUMBAR SPINE ABNORMALITIES

A number of studies have investigated the lumbar pathology in elite tennis players. Alyas et al, (2007) explored the lumbar spine of 48 asymptomatic adolescent male and female tennis players and found that facet joint arthropathy, disc pathology and pars defects were the most common abnormalities. In addition, a follow-up study with a larger sample size, identified that facet joint arthropathy, disc pathology and pars defects were the most common abnormalities in asymptomatic elite adolescent tennis players (Rajeswaran et al., 2014). However, both of these articles report moment-in-time abnormalities and do not report if and how these abnormalities change over time. Therefore, whether these asymptomatic abnormalities contribute to the onset of LBP or not remains unknown.

Another interesting line of investigation involves the relationship between facet joint orientation and pars injuries (Boden et al., 1996; Don & Robertson, 2008; Fujiwara et al., 2001; Kalichman, Guermazi, Li, Hunter, & Suri, 2010; Kalichman, Suri, Guermazi, Li, & Hunter, 2009; Masharawi et al., 2007; Noren, Trafimow, Andersson, & Huckman, 1991; Wang & Yang, 2009), which has not been previously examined in tennis or among adolescent athletes. As facet joint orientation has been associated with spondylolisthesis (where one vertebra slips forward over another, see Figure 2.3), it is possible that facet joint orientation may influence spondylolysis (a stress fracture within the pars) in young tennis players. That is, those with more coronal facet joint angles might be more susceptible to a pars abnormality due to the greater joint surface area during flexion and

extension (Figure 2.4). Given that serving produces high spinal loads (Abrams, Harris, Andriacchi, & Safran, 2014), the combination of these loads with a greater transverse articular facet angle (more coronally facing facet joints) could explain the high number of pars abnormalities in young tennis players, though this hypothesis remains untested.

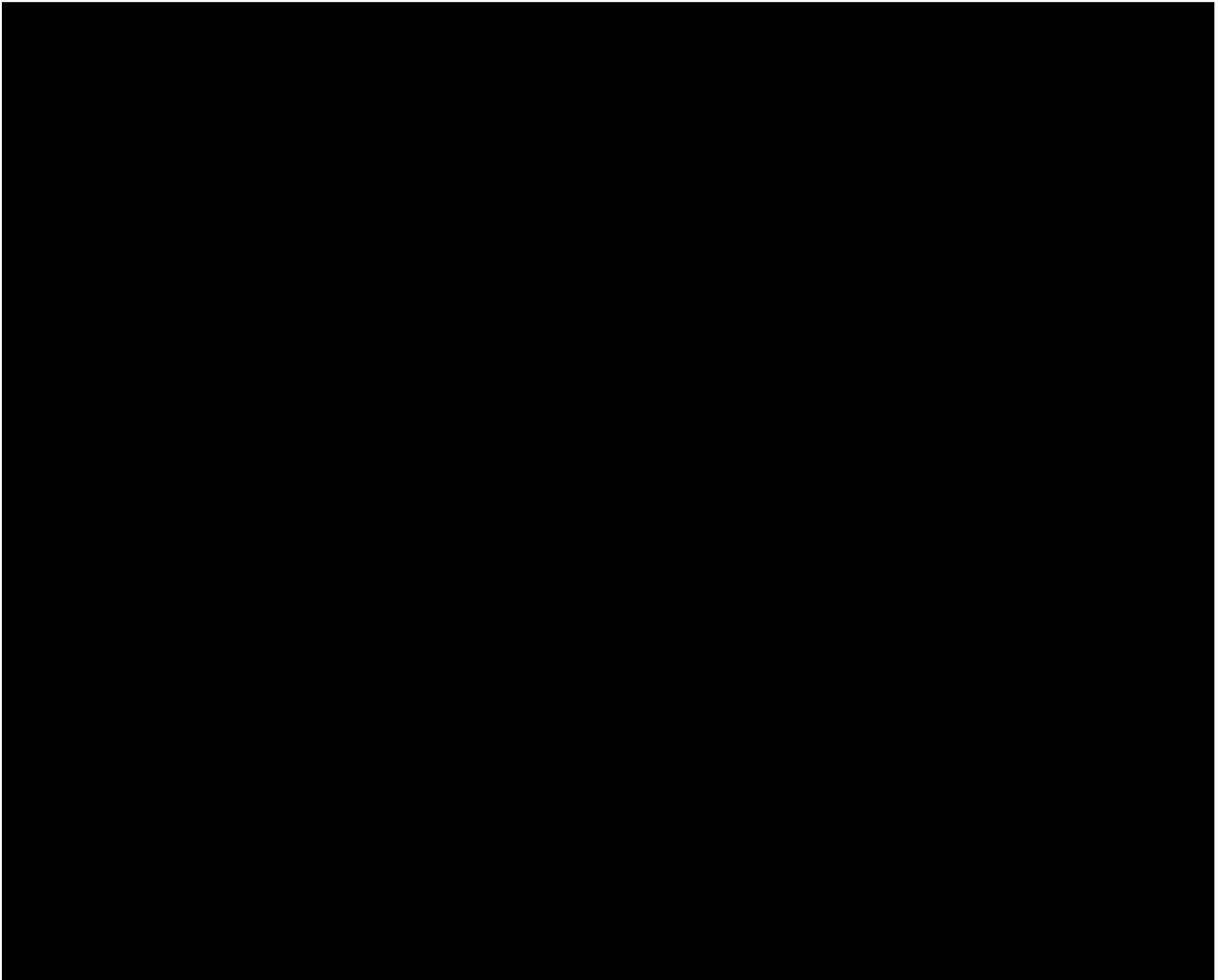


FIGURE 2.3. Meyerding Grading system for Spondylolisthesis. Grading is dependent upon degree of the displacement of the top vertebra (the surface of the bottom vertebra is divided into four spaces which ultimately determines spondylolisthesis grade). Image sought from Meyerding (1931).

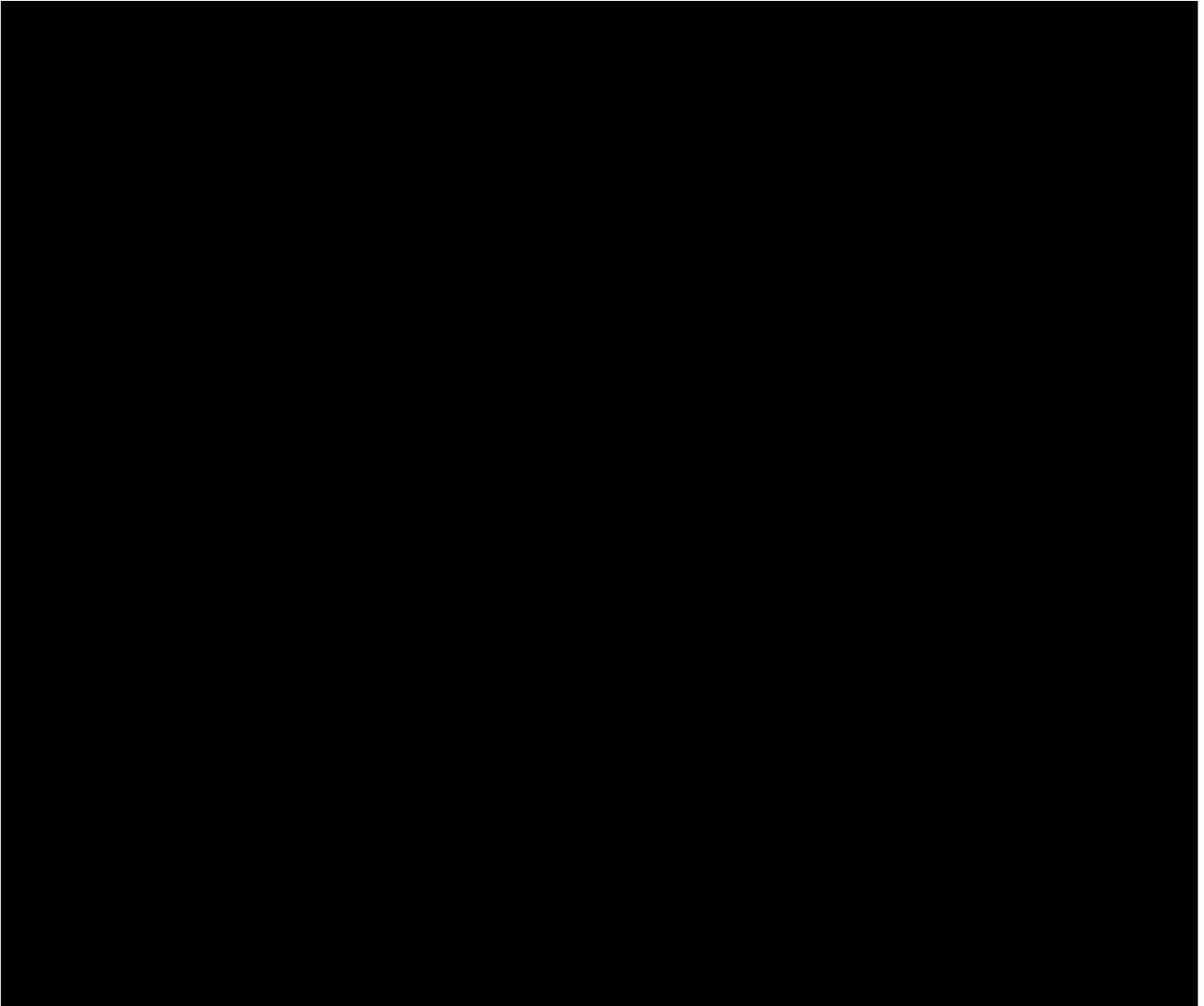


FIGURE 2.4. (A) Sagittally oriented lumbar facet – facilitates flexion and extension of the spine. (B) coronally oriented lumbar facet – results in a greater joint surface area during flexion and extension. Repetitive flexion/extension may result in a pars defect seen in the bottom right picture. Image sought from (Masharawi et al., 2007)

In other sports, like cricket, lumbar spine abnormalities have been prospectively identified as risk factors in LBP (Kountouris, Portus, and Cook (2012), giving rise to calls

for MRI to feature as part of regular screening protocols (Ranson et al. (2008)). This has been supported by recent work showing strong links between graded BMO severity and resultant LBP (Sims et al. (2019)). With this in mind, it is logical for tennis research to build upon previous literature exploring lumbar spine abnormalities in tennis and explore the link between these lumbar abnormalities, in particular BMO, and the onset of LBP in elite junior tennis players.

2.3.1.4. AGE

A typical adolescent tennis athlete will be recruited to elite training environments at approximately 11-12 years. This transition often coincides with several changes such as; increased time spent on court training, possible strength and conditioning intervention, new coaching styles and changes in equipment, all factors which could be harmful to junior athletes if not carefully planned.

In addition to this, adolescent athletes will be maturing physically, which heightens the need to manage the training process carefully. For example, Adirim and Cheng (2003) suggest that adolescents are especially prone to injury due to their growing cartilage being more vulnerable to stresses, which might explain their high prevalence of lumbar injuries. Further, sudden growth spurts are known to pose a risk to lumbar pain owing to changing length tension relationships (Motley, Nyland, Jacobs, & Caborn, 1998), while the lead up to adolescents arriving at peak bone mineral density presents as a period of bone weakness (Faulkner, Davison, Bailey, Mirwald, & Baxter-Jones, 2006). It then follows that repetitive stress on the lumbar spine in a young athlete who is undergoing (or about to

undergo) high growth might elevate his/her chances of sustaining a low back injury (DiFiori et al., 2014).

2.3.2. MODIFIABLE RISK FACTORS FOR LOW BACK PAIN

2.3.2.2. SERVE TECHNIQUE

A good tennis serve is crucial as it is the only stroke in tennis that a player has full control over (Đurović, Lozovina, Pavičić, & Mrduljaš, 2008; Kovacs & Ellenbecker, 2011a; Roetert et al., 2009; Sweeney, Reid, & Elliott, 2012). There are four key points of the serve that are typically used during serve analyses: ball toss or ball release (when the ball is released), racquet high point (RHP) or “trophy position” (where the tip of the racket is at its zenith, this usually occurs around the same time as peak pelvic obliquity and/or ball zenith), racket low point (RLP) (where the tip of the racket is at its lowest point and the hitting arm’s shoulder is externally rotated) and impact (where the ball comes in contact with racquet string-bed) (Figure 2.5) . These points can be used to define three main phases: “ball toss” which is ball toss to RHP; “drive” phase which is from RHP to RLP and “forward-swing” which is RLP to impact. Strangely, post-impact mechanics are rarely analysed.

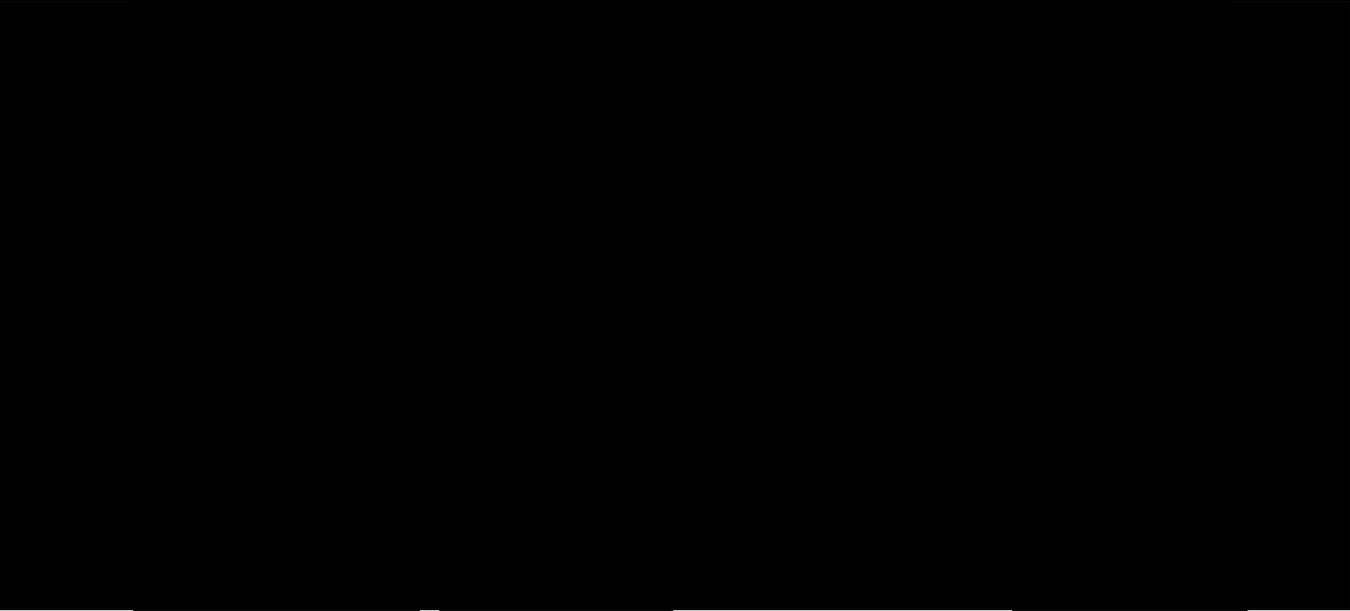


FIGURE 2.5. Key serve events

Traditionally, serves have been classified as first and second serves, with the first being a flatter and faster ball and the second being a slower ball though with greater spin (Knudson, 2006). Commonly, second serves are hit with slice (a combination of top and side spin, that results in the ball curving through the air to land wide) but more so, kick (where there is maximal amount of topspin applied to the ball that results in greater out of bounce height). Indeed it is the kick serve that has been scrutinized among tennis coaches, health professionals and within the literature as a cause for LBP in tennis athletes due to the large loads placed on the posterior elements of the spine (Abrams et al., 2012; Congeni et al., 1997; Cyron & Hutton, 1979; Ellenbecker et al., 2009; Kelsey, 1980; Roetert et al., 2009). In addition to this, the current body of literature lacks research reporting kick serve mechanics in females and lacks research comparing male and female first and second serves. Whilst the reason for this is unknown, it is possible that it relates to males being taught the kick serve earlier and practicing it more pervasively than females. Therefore, adolescent females are less experienced at this type of serve.

Broadly speaking, there has been limited research into serving mechanics and their relationship with LBP. Even at the most superficial level though given that prior research

has revealed (flexion) loads as low as 500 N causing damage to the neural arch of the vertebra, the spinal loads of nearly 3000 N (Abrams et al., 2014) tolerated during the serve seem problematic for the developing lumbar spine. Further, differences in lumbar spine mobility of adolescent tennis players with and without LBP have been revealed (Campbell et al. (2014)), with the LBP group experiencing less extension range, left lateral flexion and right rotation in the lumbar spine when completing end-range lumbar mobility assessments. Differences in serve kinematics in elite junior tennis players with and without LBP have also been observed. Campbell et al. (2014) revealed that during the drive phase, those with LBP had; greater right lateral pelvic tilt, significantly less lower lumbar region right rotation and significantly smaller pelvis/shoulder separation angles. Then, during the forward-swing phase, the LBP group had a greater lower lumbar rotation (Campbell et al., 2014). Interestingly, this study also concluded that upper lumbar mobility was comparable between the pain and non-pain groups, though those in the pain group had reduced lower lumbar mobility. The rigidity in the lower lumbar spine of the pain group was linked to either individual technique or high compression loads. Indeed, the spine becomes more resistant to bending, shear and torsion when under large compression loads (Janevic, Ashton-Miller, & Schultz, 1991) and therefore, this could be a possible explanation for the lower mobility and higher lumbar forces in players with LBP. Elsewhere, juniors with a history of LBP have also been observed to experience significantly greater and potentially injurious left lateral force (four times their body weight) than players without pain during the drive phase of the flat and kick serves (Campbell et al., 2013).

In sum, prior research investigating LBP in tennis has found relationships between serve biomechanics and LBP in junior tennis players. However currently, it is unknown whether the movements associated with LBP are adaptive or maladaptive to LBP as these studies have been cross-sectional. Currently, there is no research investigating the relationship between serving technique and those with asymptomatic lumbar abnormalities. Such research would be beneficial in order to uncover whether the aforementioned serve mechanics lead to LBP. Lastly, since male adolescents are more likely to sustain a serious lumbar injury and tend to learn and practice the kick serve, a comparison in serve mechanics between elite male and female junior tennis players will provide insight in to the relationship between sex, serve type and LBP.

2.3.2.3. WORKLOAD

In tennis, preparation for tournaments is difficult compared to other sports as players compete year-round and there are multiple junior tennis tournaments both nationally and internationally. In America, there are between 48 and 64 national tournaments organized each year with players playing up to 10 matches during a tournament depending on their progress (Jayanthi et al., 2009). This imposes considerable stress on the adolescent body and emphasizes the importance for managing player workloads.

According to Halson (2014) and Borresen and Lambert (2009), there are several different measurements of workload. These include internal workload and external workload (Mujika, 2017). External workload is an objective measurement that quantifies what the player has completed in either training or a match, for example; the duration of the session or number of strokes hit. Internal workload is defined as the assessment of biological

stress imposed on the body during a training session or match, for example; Rating of Perceived Exertion (RPE) or heart rate. It is important to measure both internal and external workload as these measurements can differ between athletes (Halson, 2014).

Various relationships have been found in other sports between external workload and injury (Barile, Limbucci, Splendiani, Gallucci, & Masciocchi, 2007; Dennis, Farhart, Goumas, & Orchard, 2003; Dennis, Finch, & Farhart, 2005; Fleisig et al., 2011; Gabbett, 2004; Gabbett & Jenkins, 2011; Orchard, James, Portus, Kountouris, & Dennis, 2009). Specifically, high workloads or short “spikes” in workload (typically when an athlete plays multiple matches over a short period) have been strongly associated with injury (Windt & Gabbett, 2017), while imprudent workload prescription has also been implicated (Dennis et al., 2003; Dennis et al., 2005; Orchard et al., 2009). The use of mathematical ratios of workload, namely Acute Chronic Workload Ratio (ACWR) where acute load is computed over 1 week and chronic load is computed over 4 weeks, have also been used to successfully model the likelihood of injury in cricket (Hulin et al., 2014) and rugby (Hulin, Gabbett, Lawson, Caputi, and Sampson (2016). Interestingly, in rugby, Hulin et al. (2016) found that players who had a high chronic workload were more resistant to injury when undergoing moderate-low and moderate-high workload ratios compared to those with low chronic workloads. He also unearthed that those with high chronic workloads were less resistant to injury when undergoing a very high acute:chronic workload ratio (Hulin et al., 2016). Despite these encouraging associations, there remains conjecture as to whether ACWR is the best method for assessing workload due to the fact that all days contributing to the chronic workload are weighted equally (Menaspa, 2017). Specifically, using ACWRs for predicting injury can be especially

troublesome due to the fact that a player's workload from 1 and 28 days ago are weighted equally when considering injury risk. Accordingly, the work of Williams, West, Cross, and Stokes (2017) suggested that exponentially weighted moving averages (EWMA), which weigh recent training sessions more heavily, may be more accurate for analysing chronic workloads. Subsequent research has further validated the sensitivity of the EWMA approach in detecting increases in injury risk at higher acute:chronic workloads.

Currently, relatively little research has been conducted to understand the interaction between workload and injury in tennis. This is surprising given that load is commonly referenced as a risk factor, especially for LBP. Promisingly, Myers, Aguilar, Mexicano, Knudson, and Kibler (2019) recently revealed that an ACWR value of >1.5 was associated with injury in junior tennis players. Although the ACWR has been criticized for its lack of sensitivity in the chronic workload calculation, this is an important first step in understanding this load-injury dynamic and sets the tone for future research to investigate tennis workloads using EWMA approaches.

2.4. CONCLUSION

Low back pain in young tennis players is pervasive and can be burdensome, both physically and psychologically, for those affected. To improve the prevention of this disabling condition, it is clear that prospective research exploring key risk factors for LBP, including morphology, sex, serve mechanics and workload, is required.

CHAPTER 3

LUMBAR SPINE ABNORMALITIES AND FACET JOINT

ANGLES IN ASYMPTOMATIC ELITE JUNIOR TENNIS

PLAYERS

Publication statement:

This chapter is currently under review in Sports Medicine – Open.

3.1. ABSTRACT

Objective. To describe the prevalence of lumbar spine abnormalities and the relationship between facet joint orientation and pars interarticularis abnormalities in elite adolescent tennis players.

Methods and Materials. Lumbar spine MRI images of 25 elite junior tennis players were obtained and distributed between five radiologists for analysis. Descriptive comparisons and confidence intervals were used to describe the prevalence of the abnormalities. A generalized linear regression model was conducted to investigate the relationship between both pars abnormalities and sex (independent variables) and resultant facet joint angle.

Results. 16 (64%) of 25 players were found to have at least one lumbar spine abnormality. Pars abnormalities affected 36% of players with grade 1 abnormalities (bone marrow odema only) being the most common severity. Bone marrow oedema (BMO) was found in 24% of players with half of the occurrences being bilateral. Disc herniation, disc degeneration and facet joint degeneration was diagnosed in 20%, 44% and 24% of players respectively. Six players (24%) were diagnosed with spina bifida and lastly, one player (4%) was diagnosed with spondylolisthesis. Females had significantly larger facet joint angles across L3/4, L4/5 and L5/S1 compared to males ($p < 0.01$). Further, those who had pars abnormalities had significantly greater facet joint angles compared to those who did not ($p < 0.001$).

Conclusion. This study was the first to link lumbar facet joint angles with pars abnormalities in elite adolescent tennis players. Disc degeneration, pars abnormalities, including bone marrow oedema (BMO) and facet joint degeneration were common findings amongst elite adolescent tennis players.

Keywords: MRI, lumbar spine, tennis, injury

3.2. INTRODUCTION

Low back pain is a symptom that arises due to either acute or repetitive micro-trauma, or can be due to a combination of both in young athletes (Kujala, Taimela, Erkinntalo, Salminen, & Kaprio, 1996). Despite the insidious nature of LBP in tennis players, empirical evidence linking risk factors with causation are limited. For example, prior research has found that pars abnormalities as well as facet joint arthropathy, disc degeneration and disc herniation are among the most common abnormalities in asymptomatic adolescent tennis players (Alyas et al., 2007; Campbell et al., 2014; Rajeswaran et al., 2014). Recent reports document that approximately 60% of players aged 11 – 19 years who presented with low back pain (LBP) were diagnosed with a symptomatic pars abnormality (unpublished data, Tennis Australia 2016) and suffered a mean time loss of ~160 days before returning to play. Most of the injured players were male, pointing to an apparent sex-based predisposition. Given the pervasive nature of LBP in tennis, particularly among adolescent players at a critical stage of their development, the importance of prospective research to understand the role of proposed morphological risk factors is essential.

Morphological components of the vertebrae have been explored as a potential risk factor

in LBP (Berlemann, Jeszenszky, Bühler, & Harms, 1999; Boden et al., 1996; Don & Robertson, 2008; Fujiwara et al., 2001; Grobler, Robertson, Novotny, & Pope, 1993; Kalichman et al., 2009; Masharawi et al., 2007). Facet joints with a more coronal appearance and larger facet joint angle have been associated with pars abnormalities (Don & Robertson, 2008; Masharawi et al., 2007). Intuitively, there seems scope to consider the possibility that coronally oriented facet joints will strongly relate to pars abnormalities in younger athletic populations. Further, the high spinal loads experienced in serving (Abrams et al., 2014), when coupled with more coronally facing facet joints, may explain the high number of pars abnormalities in young tennis players, though this hypothesis remains untested.

To the knowledge of the authors, no previous study has compared the lumbar abnormalities of asymptomatic male and female tennis players at a key risk age, between 11 and 16 years old. Further, literature exploring lumbar abnormalities in junior tennis players is currently limited to male players and thus little is known about female lumbar morphology. Therefore, the purpose of this study was to describe the prevalence of common lumbar abnormalities in male and female elite adolescent tennis players. The findings of this study will serve as baseline information for a prospective study investigating lumbar pain and subsequent lumbar injuries in elite adolescent tennis players. We hypothesise that the majority of players will be diagnosed with an asymptomatic lumbar abnormality but that males will be diagnosed with more abnormalities, specifically pars abnormalities, than females.

3.3. METHODS

3.3.1. PARTICIPANTS

Magnetic resonance imaging (MRI) scans of the lumbar spines of twenty-five (male: 14, female: 11) right-handed elite adolescent tennis players aged 13 ± 1.7 years (range 11 – 17 years), who were part of a National Tennis Academy, were obtained as part of an annual screening protocol between March and May 2017. All participants were free of low back pain, any current performance inhibiting injury or illness at the time of scanning and were excluded if they had reported 7 or more consecutive days of LBP during the last 6 months or had experienced LBP with an accompanying positive MRI (which then resulted in modified workload). All players were right-handed which means that abnormalities described as right sided will be synonymous with the dominant side.

Ethical approval was obtained from the Victoria University Human Research Ethics Committee while participants provided voluntary informed consent and assent prior to any involvement in the study.

3.3.2. IMAGING TECHNIQUE

All MRIs were carried out using 3-T Siemens Verio and Vida scanners (Siemens Erlangen Germany). The following standard sequences were performed: Sagittal T2, TR 4880ms, TE 43ms, FOV 260mm, Matrix 384 x 384, Slice thickness 3.5mm, 4.2mm separation. Sagittal STIR, TR 4020ms, TE 53ms, FOV 300mm, Matrix 384 x 384, Slice thickness 3mm, separation 3.75mm. Sagittal T1, TR 550ms, TE 11ms, FOV 260mm,

Matrix 768 x 768, slice thickness 3.5mm, separation 4.2mm. Axial T2 TR 3380ms, TE 87ms, FOV 240 x 240mm, Matrix 448 x 444, slice thickness 4mm separation 4.4mm. Sagittal T1 3D fat saturated VIBE, TR 7ms, TE 2.5ms, FOV 200 x 200mm, Matrix 256 x 256, slice thickness 2mm. Parasagittal T1 fat saturated VIBE images were reformatted through the lumbar pars interarticularis at 1mm thickness.

3.3.3. DATA COLLATION

Given the number of abnormalities to be evaluated, an inter-rater reliability was established.

Five MRI scans that included abnormalities of interest were sourced externally and provided to the radiologists to become familiar with the grading systems provided by the lead researcher. These scans were de-identified scans of other patients of the clinic that had undergone a lumbar spine scan. The scans chosen included abnormalities that the radiologists were required to grade in the study. Five experienced musculoskeletal radiologists assessed these five scans for familiarization of the abnormalities that would be assessed for the project. The five scans were assessed for the presence and severity of: pars abnormalities (including spondylosis), BMO, disc herniation, nerve root compression, canal stenosis, foraminal stenosis, disc degeneration, annular fissure, modic changes, Schmorls nodes, Scheuermann's disease, facet joint orientation, facet joint degeneration, facet synovial cysts, spondylolisthesis and spina bifida occulta. Each abnormality was graded using a peer-reviewed grading system within the literature unless deemed irrelevant (whereby Yes/No was used to indicated presence of an abnormality) (Table 1). Once the radiologists had discussed and mutually agreed upon the grading

systems and the specific grades corresponding to the five scans they reviewed together, they then participated in a reliability study.

Thirty de-identified lumbar spine scans were obtained from a clinician external to the study (working at the clinic where the radiologists are based) that included specific lumbar spine abnormalities (pars abnormalities, BMO and disc herniation) to use for the reliability study. Each radiologist was asked to grade: pars abnormalities, BMO, disc herniation and facet joint orientation for every scan (all radiologists assessed the 30 scans). These abnormalities were chosen for the reliability study as they are believed to be the most difficult (or potentially variable) to grade due to the large number of grading categories and attention to detail required to correctly diagnose each abnormality. The remaining abnormalities (Table 1) were deemed straightforward to grade and identify and therefore were determined unnecessary for the reliability study.

Agreement on ratings was measured with Fleiss's kappa for abnormalities with a categorical grading and with the intra-class correlation (ICC) for abnormalities graded on a continuous scale. Excellent agreement was found in the reliability results for pars abnormalities (ICC: 0.95), BMO (ICC: 0.93) and facet joint angles (ICC: 0.86). For disc herniation, where gradings were categorical, the kappa was 0.51. Lower values for agreement when using the kappa statistic are expected in comparison to the ICC, as kappa ignores any ordinality in ratings and high values of agreement are less likely as the number of rating categories increases (McHugh, 2011). General guidance suggests values between 0.41 and 0.6 as "moderate" reliability for kappa and 0.6 to 0.74 for the ICC (Hallgren, 2012). In our study, the observed agreement between raters was 83%.

Together, these results suggest that there was good reliability among raters in the most difficult categories of abnormalities.

Once reliability was established, the MRI scans from the sample of elite players (n=25) were randomly distributed between the radiologists (five scans each) by an independent researcher for detailed review. For these scans, the radiologists assessed all lumbar abnormalities featured in Table 1 using the nominated grading system.

Amongst a number of lumbar spine abnormalities, this study analysed both pars abnormalities and BMO severity. Pars abnormalities refer to morphological changes to the pars interarticularis as described by Ang et al. (2016). This grading system describes pars abnormalities as follows: normal (grade 0), a stress reaction (grade 1), an active incomplete fracture (grade 2a), a chronic incomplete fracture (grade 2b), an active complete fracture (grade 3) or a chronic complete fracture (grade 4). Three of these gradings (Grade 1, 2a and 3) include BMO, however the severity of the BMO not described. Therefore, we have also included a BMO grading system by Sims et al. (2019) in this study to describe the severity of BMO found on the MRI images. The severity of BMO is calculated from the sagittal STIR sequence. The intensity value of bone marrow at the site of oedema is measured using the region of interest tool. This value is then divided by the value of normal bone marrow within the vertebral body at the same level of the pars. This product value is referred to as the BMO “ratio”. Sims et al. (2019) states that a ratio close to 2 reflects early stage asymptomatic lumbar bone stress. A ratio between 2 and 3 signifies clinically significant BMO which is likely to be symptomatic

CHAPTER 3 LUMBAR SPINE ABNORMALITIES AND FACET JOINT ANGLES IN ASYMPTOMATIC ELITE JUNIOR TENNIS PLAYERS

and a ratio of 3 or more is likely to represent the later stages of symptomatic lumbar bone stress injury.

TABLE 3.1. List of abnormalities and the corresponding grading systems used in this study.

Abnormality	Grading System used	Classifications
Pars Abnormality	Ang et al., (2016)	Grade 0 = Normal, Grade 1 = a stress reaction, Grade 2a = an active incomplete fracture, Grade 2b = a chronic incomplete fracture, Grade 3 = Active complete fracture, Grade 4 = Chronic complete fracture
Bone Marrow Oedema	Sims et al., (2019)	The severity is calculated from the sagittal STIR sequence. The intensity value of bone marrow at the site of oedema is measured using the region of interest tool. This value is then divided by the value of normal bone marrow within the vertebral body at the same level of the pars. This is referred to as the BMO “ratio”
Disc Herniation	(Mysliwiec, Cholewicki, Winkelpleck, & Eis (2010)	Grading is dependent upon the direction and the magnitude of the disc protrusion. The grading depends on the magnitude of the distance the protrusion extends posteriorly (Grading is between 1-3), and then follows with the mediolateral location of the protrusion (Grade A-C).
Nerve root compression	Pfarrmann et al., (2004)	Grade 0 = Normal, Grade 1 = Contact, Grade 2 = Deviation or Grade 3 = Compression
Canal Stenosis	Guen, Joon, Hee, Kyoung-Jin, & Heung (2011)	Grade 0 = Normal, Grade 1 = Mild, Grade 2 = moderate, Grade 3 = Severe
Foraminal Stenosis	Park et al., (2012)	Grade 0 = Normal, Grade 1 = Mild, Grade 2 = Moderate, Grade 3 = Severe
Disc Degeneration	Pfarrmann, Metzendorf, Zanetti, hodler & Boos (2001)	Grade 0 = Normal through to Grade 5 = Severe
Annular Fissure	Yes/No answer with location (Right, Posterior/Central or Left)	"Yes" or "No"
Modic Endplate Changes	Modic, Masaryk, Ross, & Carter (1988)	“Type 1” (decreased signal intensity on T1-weight images, increased signal intensity in T2-weighted images), “Type 2” (increased signal on T1-weighted images and a slightly hyperintense signal on T2-weighted images) or “Type 3” (decreased signal intensity on both T1 and T2-weighted images)
Schmorl’s nodes	Yes/No answer	"Yes" or "No"
Scheuermans disease	Yes/No answer	"Yes" or "No"
Facet Joint Orientation	Noren, Trafimow, Andersson & Huckman (1991)	(Adapted method from Noren et al) Facet joint orientation was measured using a midsagittal line through the vertebral body and the intersecting lines passing over the endpoints of each facet (see Figure 1).
Facet Joint Degeneration	Weishaupt, Zanetti, Boos, & Hodler (1999)	Grade 0 = Normal, through to Grade 3 = Severe
Facet Synovial Cyst	Yes/No answer with location (Left/Right and Infraspinal/Extraspinal)	"Yes" or "No" with "Right" or "Left"
Spondylolisthesis	Meyerding (1931)	Grade 0 = No spondylolisthesis, Grade 1 = 1-25% vertebral slip, Grade 2 = 26-50% vertebral slip, Grade 3 = 51-75% vertebral slip, Grade 4 = 76 – 100% vertebral slip or Grade 5 = >100% vertebral slip
Spina Bifida Occulta	Yes/No answer	"Yes" or "No"

3.3.4. STATISTICAL ANALYSIS

Because this is a descriptive study, descriptive comparisons are mostly presented in the results and discussion. Age characteristics of the sample were summarised with the mean and standard deviation. The prevalence of abnormalities was summarised by the percentage occurrence and 95% confidence intervals. Qualitative comparison of sex differences was also performed where appropriate. A generalized linear regression model was conducted to determine the relationship between, 1) those with pars abnormalities and facet joint orientation angle and 2) between sex and facet joint orientation angle. All analysis was performed with RStudio software (version: 0.99.903, RStudio: integrated Development for R. RStudio, inc., Boston, MA).

3.4. RESULTS

Sixteen out of 25 players (64%, 95% CI: 43% to 81%) (10/14 male, 6/11 female) were found to have at least one abnormality (Table 2).

TABLE 3.2. A list of the participants and their respective lumbar spine abnormalities detected. Grey = Diagnosed, White = Not Diagnosed

Participant	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
Pars interarticularis*	Grey			Grey	Grey	Grey	Grey													Grey	Grey	Grey			Grey
BMO					Grey		Grey													Grey	Grey	Grey			Grey
Disc Herniation						Grey								Grey			Grey		Grey		Grey				
Nerve root compression																									
Canal Stenosis														Grey										Grey	
Foraminal stenosis				Grey		Grey																			
Disc Degeneration	Grey	Grey		Grey	Grey	Grey		Grey	Grey					Grey			Grey							Grey	Grey
Annular fissure														Grey											
Modic changes																									
Schmorls nodes																									
Scheuermann’s disease					Grey																				
Facet joint degeneration	Grey	Grey			Grey			Grey						Grey											Grey
Facet Synovial Cysts																									
Spondylolisthesis				Grey																					
Spina Bifida	Grey	Grey															Grey				Grey				Grey

*Pars interarticularis abnormality

3.4.1. PARS ABNORMALITIES

A total of 9 out of 25 (36%, 95% CI: 19% to 57%) players had pars abnormalities (7 male, 2 female) at L4 and L5. None were present between L1 and L3. Two players had abnormalities at L4 and seven players had abnormalities at L5. Three players had isolated right-side pars abnormalities while the remaining six players had bilateral pars abnormalities. No players had isolated left side pars abnormalities. Seven instances of grade 1 (a stress reaction) pars abnormalities were detected, two instances of grade 2a (an active incomplete fracture), one instance of grade 2b (a chronic incomplete fracture) and five instances of grade 4 pars abnormalities (chronic complete fracture). No instances of grade 3 pars abnormalities (active complete fracture) were detected in this study.

3.4.2. BMO

Six out of 25 players had BMO (5 males, 1 female) (24%, 95% CI: 10% to 46%), all at L4 or L5. Two male players had BMO at L4, one of which had BMO present on the dominant side at the pedicle (posterior side) and the pars while the other had BMO on the non-dominant side at the pedicle (posterior and anterior sides) as well as at the pars. The player with BMO on the dominant side had a BMO ratio above 2, however the player with BMO on the non-dominant side had a BMO ratio of less than 2 (Table 3). Three males had BMO (1 dominant side, 2 bilateral) at L5 at the pars, the pedicle (posterior side) and extending into the vertebral body. Two of the three males also had BMO (bilateral) at the pedicle on the anterior side, transverse process and the superior articular process while the other male had BMO (dominant side) at the inferior articular process. Of the male players with bilateral L5 BMO, one had a BMO ratio exceeding 3 on both sides, the other male had a ratio exceeding 2 on the dominant side and a BMO ratio less

than 2 on the non-dominant side. The male who had BMO at L5 on the dominant side had a BMO ratio exceeding 2. One female had bilateral BMO at L5 which was only present at the pars and had a BMO ratio of less than 2.

TABLE 3.3. A list of the participants with bone marrow oedema (BMO) and their respective BMO details

Participant	Sex	Ratio Right pars	Ratio Left pars	Diagnosis	Level
1	Male	4.4	3.6	Bilateral	L5
2	Male	2.1	1.1	Right	L4
3	Male	2.6	0.9	Right	L5
4	Male	2.2	1.5	Bilateral	L5
5	Male	1.2	1.7	Left	L4
6	Female	1.7	1.8	Bilateral	L5

3.4.3. DISC ABNORMALITIES

3.4.3.2. DISC HERNIATION

Five of the 25 players (4 male, 1 female) (20%, 95% CI: 8% to 41%) had disc herniation either at L4/5 or L5/S1. One female and one male had disc herniation at L4/5 with gradings of 1A and 1C respectively. Three males demonstrated disc herniation at L5/S1 with two gradings of 1A and one grading of 1C. All abnormalities demonstrated a small bulge into the lumbar canal with three bulges being central and one being lateral.

3.4.3.3. NERVE ROOT COMPRESSION

No instances of nerve root compression were found in this cohort.

3.4.3.4. CANAL AND FORAMINAL STENOSIS

Two of the 25 players (male: 0, female: 2) (8%, 95% CI: 1% to 28%) had canal stenosis. One player had a grade 1 canal stenosis at L4/5 and the other player had grade 1 stenosis at both L4/5 and L5/S1. Two out of 25 players had foraminal stenosis (male: 1, female: 1) (8%, 95% CI: 1% to 28%), both at the L5/S1 level. The male and female player had a grade 1 and a grade 3 foraminal stenosis respectively.

3.4.3.5. DISC DEGENERATION

Eleven out of the 25 players (5 male, 6 female) (44%, 95% CI: 25% to 65%) had some degree of disc degeneration within the lumbar spine with 30 instances of grade 2 disc degeneration. Five, five and seven instances of grade 2 degeneration were found at L1/2 (two males and 3 females), at L2/3 (2 males and 3 females) and at level L3/4 (4 males and 3 females) respectively. At level L4/5, 6 instances of grade 2 degeneration were detected in 4 males and 2 females. Lastly, 4 males and 3 females were detected with grade 2 disc degeneration at L5/S1. Whilst there were some players who had multi-level disc degeneration (7 players: 4 male, 3 female), there were 4 players (1 male, 3 female) who had single level of disc degeneration.

3.4.3.6. ANNULAR FISSURES

One female player had a posterior/central annular fissure (4%, 95% CI: 0% to 22%) at level L4/5.

3.4.4. ENDPLATES

No modic changes or Schmorls nodes were found within this cohort.

3.4.5. SCHEUERMANN'S DISEASE

Only one male (4%, 95% CI: 0% to 22%), was found to have Scheuermann's disease.

3.4.6. FACETS

3.4.6.2. FACET DEGENERATION

Six of the 25 players (3 male, 3 female) (24%, 95% CI: 10% to 46%) had grade 1 facet joint degeneration with a total of 16/125 (12.8%) facet joints affected. No degeneration was detected at the L1/2 or L2/3 joints in any player. Five instances of facet degeneration occurred at L3/4 across 3 players. One male and one female had bilateral facet degeneration while there was one case of right facet joint degeneration in a female player. Seven instances of facet degeneration were found at L4/5 in 4 players (1 male, 3 female), with three cases of bilateral degeneration and one case left facet joint degeneration in a female player. Lastly, five instances of facet degeneration were found at L5/S1 in 3 players (2 male, 1 female). A single male and female had bilateral facet degeneration while the other male had degeneration of the right facet joint.

3.4.6.3. FACET ORIENTATION

One hundred and fifty facet joint angles were calculated for this study using methods established by Noren et al. (1991) (Figure 1). The average facet joint angles at each level are represented in Table 4.

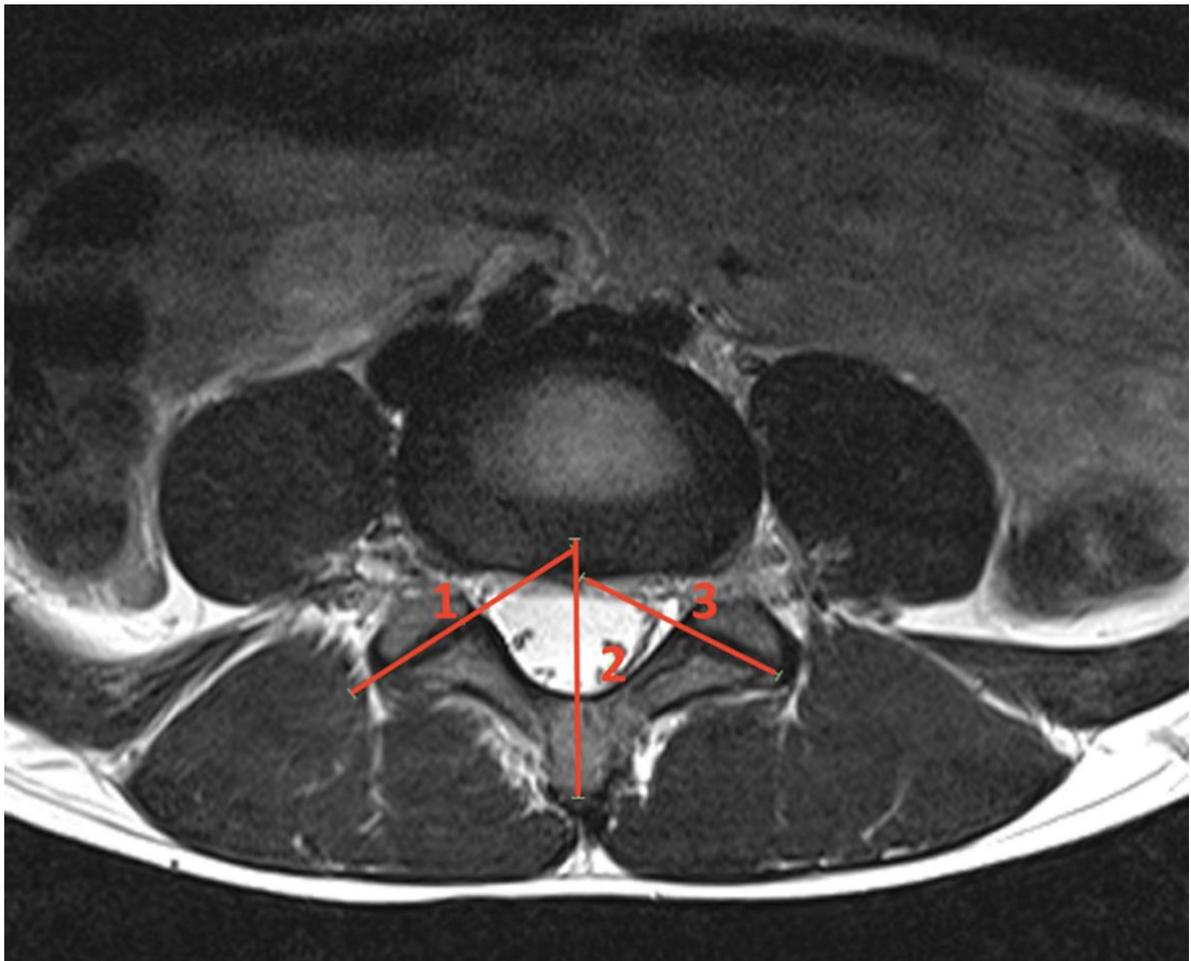


FIGURE 3.1. Facet joint angle of a 12-year old male. Line 2 in the picture above represents the midline for the Cobb angle (this is drawn down the center of the spinous process). Lines 1 and 3 pass through the facet joints being measured. The angle for the right facet joint (between line 1 and line 2) is 54° and the left facet joint angle (between lines 2 and 3) is 60° .

TABLE 3.4. Mean facet joint angles measured using grading system used in Noren et al. (1991)

Facet joint	Male		Female	
	Pars Abnormality	No Pars Abnormality	Pars Abnormality	No Pars Abnormality
Right Facet Angle - L3/4	41.9 ± 10	37.4 ± 9.4	40.9 ± 12.9	41.5 ± 10.7
Left Facet Angle - L3/4	42.9 ± 9.5	35.5 ± 5.2	49.7 ± 6.6	41.9 ± 13.2
Right Facet Angle – L4/5	53.6 ± 10.7	44 ± 7.8	54.2 ± 9.8	51.0 ± 6.1
Left Facet Angle – L4/5	55.5 ± 5.8	47.2 ± 7.0	64.2 ± 5.4	52.3 ± 9.4
Right Facet Angle – L5/S1	47.2 ± 12.0	45.6 ± 6.1	59.5 ± 9.2	51.2 ± 14.3
Left Facet Angle – L5/S1	51.2 ± 11.2	48.6 ± 8.0	64.7 ± 10.4	51.8 ± 9.1

Results from the generalized linear regression model found that those with pars abnormalities had significantly larger facet joint angles compared to those without pars abnormalities ($p < 0.001$). Further, females had significantly larger facet joint angles compared to males ($p < 0.01$).

3.4.7. SYNOVIAL CYSTS

No synovial cysts were found in this cohort.

3.4.8. SPONDYLOLISTHESIS

One female player had spondylolisthesis (4%, 95% CI: 0% to 22%) of grade 1 severity.

3.4.9. SPINA BIFIDA

Five of the 25 players (4 male, 1 female) (20%, 95% CI: 8% to 41%) had spina bifida.

All instances were at S1 with no other level affected.

3.5. DISCUSSION

Adolescent tennis players commonly demonstrate asymptomatic lumbar abnormalities on MRI, though there is little research available about the nature of these abnormalities and their relationship with future LBP. Adolescent tennis players commonly sustain LBP (Alyas et al., 2007; Rajeswaran et al., 2014), however junior male players have been found to be more susceptible to LBP than female players according to unpublished data from Tennis Australia. Whilst it is well established that asymptomatic lumbar spine abnormalities are prevalent in young tennis players, the link between such abnormalities and the risk of developing LBP is currently unclear. As a result, it would be wise to exercise caution when interpreting lumbar spine MRI results in players with LBP. The discussion below will critique the results with clinical and practical implications detailed.

3.5.1. PARS ABNORMALITIES

Abnormalities of the pars interarticularis are known to be the greatest cause for LBP within the Tennis Australia National Academy. Within this study, pars abnormalities were the second most common abnormality found in this sample, affecting 36% of players. As hypothesized, the incidence of pars abnormalities was lower in female players than in male players (18.2 vs 50% respectively) although the overall incidence rate of pars abnormalities in our sample was slightly higher than previous studies. Alyas et al. (2007) reported 9 pars abnormalities in their sample of 33 (mean age: 17 ± 1.7 years, 18

junior male and 15 female) players (27% incidence). Our study's higher prevalence rate may relate to the younger age of our cohort (mean age: 13 ± 1.7 years) with the ossification of the neural arch not yet complete (Maquirriain, 2006). Interestingly, the majority of the pars abnormalities were bilateral ($n = 6$), with a few on the dominant side only ($n = 3$). This suggests that perhaps the preferred hitting arm doesn't have a significant impact on the injury site in tennis players whereby for comparison, cricket players predominantly suffer pars injuries on the side contralateral (or non-dominant) to the bowling arm (Ranson, Burnett, & Kerslake, 2010; Ranson, Kerslake, Burnett, Batt, & Abdi, 2005).

As alluded to above, 7/14 male players had pars abnormalities as compared to 2/11 females. Grade 1 followed by grade 4 were the most frequent type of pars abnormalities among the males whilst the two females had an even distribution of grade 1 and 4 abnormalities. Interestingly, the affected males tended to be older (12 – 16 years of age) than the females (11 – 12 years). The timing of the introduction of the kick serve into regular practice for these adolescents, might be a possible explanation for the disparate incidence between sexes. That is, the repetitive hyperextension involved in the kick serve has been speculated as a potential cause for pars injuries, particularly when combined with rotation and side flexion. The pars interarticularis provides conduit between the superior and inferior facet joints and as a result is simultaneously sheared, stretched and subjected to large loads during extension when serving (Abrams et al., 2014; Leone et al., 2011).

Those who had grade 1, or grade 2a pars abnormalities had BMO (6 players: 5/14 male, 1/11 female). Four of the six players had at least one side (dominant or non-dominant) with a BMO ratio of 2 or more (Table 3), indicating that these players have clinically relevant BMO and should be monitored (Sims et al., 2019). The remaining two players (players 5 and 6 in Table 3) had BMO values of less than 2 and thus have a “normal” amount of BMO according to Sims et al. (2019). Arguably though, these players should still be monitored for the following months given some of their signal intensity ratios are closer to a value of 2. The signal intensity ratios are currently used as a guideline to prevent bone stress injuries in elite cricket players. Whether these guidelines are also applicable to junior tennis players will be tested throughout the remainder of this prospective study.

Indeed, those who had BMO were older (aged between 12 – 16 years), compared to those who did not have BMO but had chronic pars fractures (who were aged 11 – 13 years). A possible explanation for this could be that the transition from club tennis to elite academy training is characterised by greater volume and intensity of ball striking, which can heighten the stress on the lumbar spine. Academy players are often scouted as young as 10 years of age and recruited around the age of 11 and 12 years. The transition into elite training can also coincide with new coaching, possible strength and conditioning interventions and changes in equipment. These variables could all contribute as potential risk factors for developing pars abnormalities.

Another potential consideration in the onset of these injuries is puberty. Typically, puberty commences slightly earlier in females than males (9-13 years and 10-14 years

respectively) (Bitar, Vernet, Coudert, & Vermorel, 2000). Given that the pars abnormalities were evident among younger females, the onset of puberty may be linked to a heightened susceptibility of pars abnormality.

Bone mineral density (BMD) and bone growth may also play part in BMO findings. It is known that BMD peaks following peak height velocity (Rauch, Bailey, Baxter-Jones, Mirwald, & Faulkner, 2004), however this is not instantaneous. Thus, it is hypothesized that there is a period of time between peak height velocity and BMD when the bone is weaker and vulnerable (Faulkner et al., 2006). Spinal vulnerability following bone growth coupled with increased training loads, suggests that junior tennis players could be at a higher risk of low back injuries.

3.5.2. DISC DEGENERATION

Disc degeneration was the most common finding in this study. Disc degeneration was found in 44% of players – slightly higher than another examination of adolescent tennis players (39% prevalence)(Alyas et al., 2007) but lower than a similar study analysing disc degeneration in elite tennis players with a mean age of 18 years, which reported a prevalence of 62% (Rajeswaran et al., 2014). The work of Rajeswaran et al. (2014) revealed a higher incidence of disc degeneration among males, which contrasts with our study where disc degeneration was more common in female players. Once more, the difference in the age demographic, and therefore physical development stage and training regimen, of the respective cohorts may account for this difference.

3.5.3. FACETS

Facet joint arthropathy was a common finding amongst this cohort (third most common), affecting 24% of players and approximately 12.8% of all facet joints. Facet joints are a load bearing conduit between the vertebrae in the spine. Therefore, it was not surprising that facet joint arthropathy was a common finding given the spine is subjected to significant load during the tennis serve (Abrams et al., 2014). More specifically, during the windup phase of the serve, the lumbar spine is subjected to significant stress due to the center of mass of the upper body sitting behind the body during lumbar extension (Chow, Park, & Tillman, 2009). Given this extension loading, it follows that the facet joint degeneration was primarily found at L4/5 and L5/S1, similar to previous work (Alyas et al., 2007; Rajeswaran et al., 2014). That the degeneration was mostly bilateral was surprising as previous research has found that highly skilled players with greater lateral flexion have greater asymmetric loads on the lumbar spine during the serve, implying that facet joint degeneration may favor one side (Chow et al., 2009).

At L3/4 and L4/5, the facet orientation was reasonably symmetrical. However, this was not the case at L5/S1, with considerable asymmetry across all players. Interestingly, whilst there was a natural increase in facet angle from L3/4 to L5/S1, more than half of the facet angles at L4/5 were greater than those at L5/S1. The facet joint angles in this study were consistent with the work of Noren et al. (1991) despite that study's participants having a mean age of 32 years and there being some suggestion of facet joint angles decreasing with age (Morimoto et al., 2018).

Our study found that those with pars abnormalities had greater facet joint angles (approximately 4 ± 2.9 degrees larger) compared to those without pars abnormalities. A possible reason for this could be due to the additional strain the pars is under when the facet joints are more coronally oriented as there is greater joint surface area during flexion/extension of the spine (Masharawi et al., 2007). Tennis requires repetitive flexion and extension of the lumbar spine and thus over time could lead to pars abnormalities in tennis players. Furthermore, this could especially affect young tennis players whose spines have not yet fully developed. However, our study also found that females had significantly greater facet joint angles compared to males, although more males had pars abnormalities compared to females (7/14 male, 2/11 female). That females have greater facet joint angles and a low incidence of pars abnormalities is intriguing. Our findings partially support the research of Don and Robertson (2008) who, whilst having a broader range of ages (13 – 84 years), found that those who had larger facet joint angles had a pars abnormality.

3.5.4. DISC HERNIATION

This study used an objective measurement system established by Mysliwiec, Cholewicki, Winkelpfeck, and Eis (2010) whereby disc herniations were classified using their position relative to other anatomical landmarks. Disc herniation was found in 20% of the players (1/11 female, 4/14 male). This prevalence is marginally lower than previous tennis studies (Alyas et al. (2007): 39% and Rajeswaran et al. (2014): 31%) but higher than in asymptomatic non-athletes: 16%. (Salminen, Erkintalo, Pentti, Oksanen, & Kormanen, 1999) It has been documented that flexion in combination with axial loading places

enormous pressure on the annular fibrosis (Chaudhry et al., 2009) and can contribute to disc herniation.

3.5.5. SYNOVIAL CYSTS

No synovial cysts were found in this cohort, which contrasts with other studies in junior tennis players (Alyas et al., 2007; Rajeswaran et al., 2014). Whilst the etiology of these cysts is unclear (Khan & Girardi, 2006), it has been reported that there is a strong association between synovial cysts, degenerative spondylolysis and facet joint arthropathy (Banning, Thorell, & Leibrock, 2001; Hsu, Zucherman, Shea, & Jeffrey, 1995; Trummer et al., 2001) and they are known to manifest where axial rotation demands are high. The absence of cysts among this study's 25 adolescent players may relate to (a) only mild facet joint degeneration being found and (b) cysts being most common in later stages of life (Khan & Girardi, 2006).

3.5.6. SPINA BIFIDA

Spina bifida was present in four males and one female in this study and was one of the most common lumbar spine abnormalities detected on MRI. This finding was something we did not expect and raises questions about the prevalence of spina bifida in this population. An Australian report explored the effectiveness of folic acid supplementation in neural tube deficiencies (NTD, of which spina bifida is one) in pregnant women (AIHW, 2008). A 25% decline in prevalence of births with spina bifida occurred between 1998-2003 which is approximately the time of birth for many of the participants in this study (AIHW, 2008). Yet, this report also found that Australia has one of the highest prevalence rates of spina bifida compared to other developed countries (AIHW, 2008).

Our study had a couple of limitations. Firstly, this study had a convenience sample drawing on the limited number of elite adolescent tennis players in the available academy without previous lumbar pathology. This limits the generalizability of the findings. Secondly, we are unable to control for the participants' current extra-curricular activities and their influence on lumbar morphology, notwithstanding that the players' current playing level was standardized.

The use of MRI is a valuable tool for determining lumbar spine pathologies that may contribute to the development of low back pain. This study found that disc degeneration, pars abnormalities, including BMO and facet joint degeneration were common findings amongst elite adolescent tennis players. Since all players were asymptomatic, this study highlights the need to exercise caution when using MRIs to assist in diagnosing junior tennis players with LBP owing to the abundance of abnormalities that exist asymptotically.

Males had a higher prevalence of pars abnormalities, BMO and disc herniation compared to females, although females had a higher prevalence of disc degeneration and facet joint arthropathy. Those players with a pars abnormality had larger facet joint angles than players with a normal pars. This finding could be useful in determining athletes at risk of pars stress fracture injury. A further study will be conducted at 12-months following these MRI scans and will use statistical analysis to interpret any morphological changes over this period of time. This study will also monitor which participants become symptomatic and which remain asymptomatic.

CHAPTER 4

LUMBAR SPINE ABNORMALITIES IN ELITE

ADOLESCENT TENNIS PLAYERS: A LINK WITH

SERVE KINEMATICS?

Publication statement:

This chapter is currently under review in Sports Medicine – Open.

4.1. ABSTRACT

Objectives: The purpose of this study was to compare the flat and kick serve kinematics of asymptomatic elite adolescent tennis players with and without lumbar spine abnormalities.

Design: Cross-sectional.

Methods: Twenty-four players (nine of which had confirmed lumbar spine abnormalities) carried out a series of flat and kick serves, whilst marker trajectories were recorded by a 3D motion capture system. Pelvis and lumbar spine kinematics as well as key temporal events were compared between sex and serves using a mixed-effects model.

Results: Males had significantly greater posterior pelvis tilt than females during the drive phase of both flat (M: $-7.1 \pm 5^\circ$, F: $4 \pm 5.5^\circ$) and kick serves (M: $-8.6 \pm 5.1^\circ$, F: $2.1 \pm 5.8^\circ$). Independent of serve type, males also impacted the ball ~15cm further into the court than females, while all players contacted flat serves significantly further forward (~17cm). There were no effects for abnormality in the magnitude of pelvis and trunk kinematics, however the temporal sequencing of these kinematics was different. Players with pathology entered peak right knee flexion and peak lumbar right lateral flexion earlier than players without pathology. Lastly, the timing of pelvis rotation was highly variable among females but not males. **Conclusion:** Pelvis and ball toss kinematics vary with Sex and serve type but not necessarily abnormality in the elite adolescent serve. Crucially, the temporal sequence of the way in which players reach trophy position during the serve appears linked to the presentation of lumbar spine abnormalities in this adolescent playing cohort.

4.2. INTRODUCTION

Low back pain (LBP) is highly prevalent in tennis, particularly at the youth level (Hjelm et al., 2012). Gescheit et al. (2019) reported that the lumbar spine was the most often injured body region amongst elite junior tennis players. It's also a pervasive problem in professional tennis, with Grand Slam tournament data from the Australian Open revealing that lumbar pain is among the most common complaints of touring male and female professionals (Gescheit et al., 2017). Magnetic resonance imaging (MRI) has shown as many as 95% of asymptomatic players to have lumbar spine abnormalities, generally at the L4/L5 and L5/S1 levels (Alyas et al., 2007; Rajeswaran et al., 2014). Clearly, lumbar spine pathologies are ubiquitous in tennis and actionable insight to limit their occurrence has largely eluded the sport.

One of the proposed mechanisms of low back pain in tennis is the mechanics of the serve. The serve is the most important stroke in the game (Kovacs & Ellenbecker, 2011b; Roetert et al., 2009) and its repeated high-speed three-dimensional rotation of the spine (Congeni et al., 1997; Ellenbecker et al., 2009; Kelsey, 1980; Roetert et al., 2009) has been widely implicated in lumbar injury. In particular, the kick serve has been shown to produce the highest forces on the back (Abrams et al., 2014). It is introduced to players as young as 13 years of age (Myer et al., 2015), albeit more commonly among male adolescents. However, in contrast, other research has reported that the flat serve is characterized by higher lateral flexion moments in tennis players with low back pain and has suggested that the loading during the flat serve is linked to LBP in elite junior tennis players (Campbell et al., 2013). Historically, males have had a higher prevalence of

lumbar injuries compared to females (Gescheit et al., 2019; Reece, 1986). That males learn the kick serve earlier and therefore experience those extreme loading conditions sooner might explain the higher lumbar injury incidences. To the knowledge of the authors, the work of Campbell et al. (2014) and Campbell et al. (2016) remain the only studies to examine the influence of serve type (flat serve vs kick serve) on lumbar kinetics in elite adolescent males with and without low back pain. As this research was cross-sectional in nature and compared the serve mechanics of healthy players and players who have previously suffered LBP, it is unclear whether the observed differences in the serve action were adaptive or maladaptive to low back pain. Intriguingly, research has not considered the female serve in the context of lumbar spine injury, which seems an oversight given they appear to sustain fewer lumbar spine abnormalities/injuries.

Given the prevalence and impact of LBP in junior tennis players, particularly males, the current study aimed to compare the effect of serve type, sex and the presence of lumbar spine pars abnormalities on the kinematics and temporal sequencing of the serve in adolescent players. Our first hypothesis was that players with abnormalities would exhibit less dominant (right) side lumbar spine and pelvis rotation during the drive phase but greater non-dominant (left) side lateral flexion, lumbar spine rotation, pelvis rotation and anterior pelvis tilt during the forward-swing phase than players without abnormalities. Our second hypothesis states that males were proffered to have increased lateral impact position, drive phase lumbar extension as well as lumbar lateral flexion and posterior pelvis tilt while the female serve was anticipated to feature larger ball toss zeniths, ball toss drop distances and peak knee flexion. Our third hypothesis was that the kick serve would see greater lumbar lateral flexion and extension compared to the flat serve as well

as a smaller ball toss. Our final hypothesis expected the timing of pelvis, trunk and ball toss kinematics to significantly differ between the serves of those with and without lumbar spine abnormalities.

4.3. METHODS

4.3.1. PARTICIPANTS

Twenty-four (male: 14, female: 10) elite adolescent tennis players aged 13 ± 1.65 years (range: 11 – 17 years) were recruited from the Tennis Australia National Academy. Participants were excluded if they had had a previous bout of severe LBP (severe LBP defined as seven or more days missed training and/or competition due to LBP, similar to Ranson et al. (2008)) with an accompanying MRI diagnosing a lumbar injury, were ill, had a performance inhibiting injury, or experienced low back pain during testing. All players had recently undergone an MRI scan as part of an academy screening protocol which focused on the lumbar spine (L1/L2 to L5/S1). Based on their MRI screening results, participants were assigned to a group of those with pars abnormalities; (male: 7, female: 2, in this study we included those with either a pars stress fracture and/or bone marrow oedema designated by ‘P’ from here on), or those without these abnormalities (male: 7, female: 8, designated by ‘NP’ from here on). Ethics approval was obtained from the Victoria University Human Research Ethics Committee and participants provided voluntary written informed consent and assent prior to their involvement in the study.

4.3.2. PROCEDURE

A dynamic capture space (approximately 2 m x 2 m x 2 m) was calibrated at the baseline using a 12-camera Vantage opto-reflective motion capture system (Vicon Motion

Systems Ltd, Oxford, UK; 250 Hz). A global reference frame was set at the centre mark on the baseline with positive X pointing towards the net, positive Y pointing directly leftward (along the baseline) when facing the net, and positive Z pointing directly upward. Prior to testing, participant height and mass were recorded as well as racquet parameters (mass, center of mass (COM), and three dimensional moment of inertia). Retroreflective markers (12.7 mm diameter) and rigid plates with markers attached were then affixed to the participant's skin (over specific positions or anatomical landmarks on the lower body, trunk and upper body) using double-sided tape and rigid sport tape. Once the markers were attached, participants completed a self-directed warmup followed by a series of subject-specific calibration trials. Participants completed a series of serves aiming for a 1m x 2m target area bordering the "T" on the deuce court. Participants performed "Flat" serves (FS) followed by "Kick" serves (KS) at maximal intensity. Successful serves were defined as those that landed in the target area. Serving continued until three successful FS and KS were completed, adhering to prior established methods (Campbell et al., 2014; Reid, Whiteside, & Elliott, 2011).

4.3.3. DATA PREPARATION AND MODELLING

The data was processed and gaps in marker trajectories filled using Vicon Nexus Software (Vicon Motion Systems Ltd, Oxford, UK). Trajectories were treated using a fourth order low-pass Butterworth filter at 15 Hz following a residual analysis and visual inspection of the data. Filtered anatomical, racquet and ball data were modelled using a customised direct kinematic model (Crewe, Campbell, Elliott, & Alderson, 2013a; Wu et al., 2002; Wu et al., 2005). The segment parameters for the upper body, thorax and lumbar spine

were defined based on previous research (de Leva, 1996; Pearsall, Reid, & Livingston, 1996; Pearsall, Reid, & Ross, 1994).

The dependent variables of interest included selected lumbar spine, pelvis, racquet and ball toss kinematics relevant to tennis serve performance and/or injury in past research (Campbell et al., 2014; Reid et al., 2011). Selected ball toss kinematics (ball toss height, three-dimensional impact position) were also measured relative to players' height and will be described accordingly in the text. Kinematics were reported in the drive and forward-swing phase of the serve (Whiteside, Elliott, Lay & Reid., 2015) and the temporal sequencing was described relative to serve impact.

4.3.4. STATISTICAL ANALYSIS

For each variable of interest, the mean kinematics of the three successful serves (per serve type) were used for analysis (Table 1). A mixed effects model identified serve kinematic differences between the three comparison groups (P vs NP, male vs female, FS vs KS). As multiple comparisons were conducted, the alpha value was adjusted *a priori* to 0.01 to reduce the risk of Type 1 error (Fleisig, Nicholls, Elliott, & Escamilla, 2003; Reid, Whiteside, & Elliott, 2010; Whiteside, Elliott, Lay, & Reid, 2013).

4.4. RESULTS

4.4.1. THE EFFECT OF LUMBAR ABNORMALITIES ON SERVE KINEMATICS

The pelvis and trunk kinematics that characterised the serves of the P and NP groups were comparable. Lumbar right lateral flexion was the most disparate between the two groups

– with higher flexion in the NP group ($p=0.03$). Ball toss kinematics and racquet-head velocity were also comparable between P and NP groups.

4.4.2. THE EFFECT OF SEX ON SERVE KINEMATICS

Posterior pelvis tilt during the drive phase was significantly greater in males than females in both the flat ($\sim 11^\circ$ difference) and kick serves ($\sim 10^\circ$ difference, $p<0.01$, Table 1). Peak right (back) and peak left (front) knee and hip extension angular velocities were also comparable.

Serve impact position was further forward in both the flat (male: 57 cm, female: 42 cm) and kick (male: 40 cm, female: 25 cm) male serves ($p<0.01$, Table 2). Differences in the vertical displacement of the ball toss were also observed with peak relative ball toss height significantly higher in the female serve ($p<0.01$), leading to significantly larger ball drop distances (~ 27 cm for flat and kick).

4.4.3. THE EFFECT OF SERVE TYPE ON SERVE KINEMATICS

Serve type had no effect on the lumbar spine kinematics during the drive phase but some differences emerged during the forward-swing phase of the serve. All players flexed their lumbar spines more in the kick serve forward-swing. The flat serve, conversely, was characterized by greater anterior pelvis tilt and less pelvis obliquity (left down, $\sim 3^\circ$ difference, $p<0.01$). The extension angular velocity profile of the lower limbs was interesting between serves, with higher magnitudes of front and back hip extension angular velocities ($7^\circ/s$ and $10^\circ/s$ respectively) ($p<0.01$) observed in the flat serve but the front knee extension more dynamic in the kick serve ($p<0.01$, Table 2).

At impact, both in absolute and relative terms, the kick serve was impacted significantly further across the body and the kick serve was hit significantly further into the court. Peak forward and vertical racquet velocities were ~5 m/s and ~1 m/s faster in the flat serve respectively ($p < 0.01$).

4.4.4. TEMPORAL KINEMATICS

A temporal comparison between key serve events revealed differences between the P and NP groups. Peak right knee flexion and the commencement of pelvis left rotation (in almost all cases) preceded racquet high point (RHP) in the P serve (Figure 1A and 1B). Peak right lumbar lateral flexion also occurred earlier in the P group (Figure 1A). This resulted in a substantially longer time lag between peak right lumbar lateral flexion and racquet high point in the P group (Figure 1A). The initiation of pelvis left rotation was highly variable in the female serve but stable among the male serve (Figure 1A).

TABLE 4.1. Peak lumbar and pelvis kinematics for those with and without pars abnormalities.

	Pars				No Pars			
	Flat		Kick		Flat		Kick	
	Female	Male	Female	Male	Female	Male	Female	Male
Drive Phase								
Lumbar Extension (°)	-4.9 ± 4.5	-14.4 ± 9.0	-5.4 ± 3.2	-14.7 ± 9.6	-9.7 ± 8.7	-7.9 ± 8.4	-9.6 ± 8.2	-7.8 ± 9.3
Lumbar Right Lateral Flexion (°)	-6.1 ± 3.3	-3.9 ± 5.3	-6.3 ± 2.6	-3.9 ± 4.7	-1.8 ± 4.3	0.9 ± 3.8	-2.6 ± 4.2	1.5 ± 4.1
Lumbar Right Axial Rotation (°)	0.5 ± 2.8	-1.1 ± 2.0	0.7 ± 3.1	-1.3 ± 2.1	0.6 ± 2.1	-0.2 ± 1.8	0.9 ± 2.2	-0.4 ± 2.0
Trunk Extension (°)	-43.9 ± 1.0	-20.5 ± 6.2	-41.9 ± 1.5	-21.1 ± 6.1	-22.4 ± 13.7	-25.0 ± 7.0	-22.9 ± 15.8	-26.9 ± 8.4
Trunk Right Lateral Flexion (°)	20.4 ± 9.1	25.1 ± 11.0	21.2 ± 8.5	24.7 ± 12.0	29.2 ± 7.5	26.2 ± 9.7	29 ± 8.1	26.8 ± 10.3
Trunk Right Axial Rotation (°)	-31.6 ± 5.1	-25.5 ± 15.9	-28.5 ± 3.2	-24.1 ± 14.9	-23.3 ± 9.0	-25.1 ± 10.7	-23.8 ± 10.2	-22.4 ± 11.4
Pelvis Right Rotation (°)	-74.7 ± 4.1	-94.9 ± 26.3	-75.6 ± 7.8	-105.3 ± 24.4	-96 ± 13.7	-102.5 ± 12.1	-97.4 ± 15.2	-109.9 ± 14.5
Pelvis Posterior Tilt (°)^	7.7 ± 1.5	-8.6 ± 4.7	4.6 ± 3.7	-10.3 ± 5.1	3.1 ± 5.7	-5.5 ± 5.0	1.5 ± 6.1	-7 ± 4.6
Pelvis Obliquity (Right Down) (°)	3.3 ± 2.8	8.5 ± 4.0	3.6 ± 1.1	9.3 ± 4.1	7.1 ± 3.5	9.5 ± 7.0	7.1 ± 2.6	10.4 ± 8.2
Lumbar Extension Angular Velocity (°/s)	-189.5 ± 69.0	-179.8 ± 112.8	-156.8 ± 31.6	-190.9 ± 134.2	-253.3 ± 106.2	-150.1 ± 42	-237.4 ± 117.2	-186.3 ± 76.6
Lumbar Right Lateral Flexion Angular Velocity (°/s)	183.9 ± 22.1	200.5 ± 137.9	177 ± 27.8	203.5 ± 203.0	224.5 ± 60.1	146.8 ± 42.1	210.3 ± 61.7	165 ± 61.6
Lumbar Right Axial Rotation Angular Velocity (°/s)	58.4 ± 36.5	54.4 ± 29.1	50.2 ± 26.9	54.8 ± 33.8	62.8 ± 31.9	59.3 ± 27.1	63.8 ± 35.5	53.1 ± 19.9
Forward-Swing Phase								
Lumbar Flexion (°)*	10.4 ± 1.4	2.6 ± 3.7	11 ± 1.7	3.3 ± 3.3	5.8 ± 4.6	6.3 ± 4.4	7.6 ± 4.7	6.6 ± 4.4
Lumbar Left Lateral Flexion (°)	-19 ± 1.5	-16.1 ± 5.2	-18.8 ± 2.4	-16.8 ± 5.2	-16.9 ± 6.2	-11.4 ± 5.2	-17.3 ± 5.5	-10.7 ± 5.2
Lumbar Left Axial Rotation (°)	-1.6 ± 2.4	-1.5 ± 2.3	-2.1 ± 1.7	-1.7 ± 2.4	-0.7 ± 2.3	-1 ± 1.7	-1 ± 2.4	-1.3 ± 2.1
Trunk Flexion (°)	5 ± 10.3	16 ± 8.0	5.7 ± 5.2	15.6 ± 12.1	7 ± 19.5	6.6 ± 12.9	13.5 ± 17.5	5.9 ± 11.3
Trunk Left Lateral Flexion (°)	-37 ± 10.6	-38.5 ± 8.0	-36.5 ± 9.6	-40.1 ± 8.6	-36.1 ± 8.9	-35.4 ± 11.7	-34.6 ± 10.7	-37 ± 8.8
Trunk Left Axial Rotation (°)	-9 ± 3.2	-9.5 ± 8.4	-7.7 ± 5.7	-11.6 ± 7.6	-6.4 ± 13.4	-6.1 ± 10.4	-10.2 ± 12.2	-6.5 ± 8.5
Pelvis Left Rotation (°)	-4.2 ± 12.5	-9.2 ± 14.5	-8.8 ± 14.7	-24.5 ± 16.5	-2.3 ± 10.2	-4.8 ± 12.8	-13.5 ± 10.8	-28.7 ± 20.7
Pelvis Anterior Tilt (°)*	31.4 ± 6.1	22.7 ± 7.9	28.7 ± 8.3	18.4 ± 8.4	33.7 ± 3.6	28.5 ± 9.9	27.7 ± 6.9	22.6 ± 10.5
Pelvis Obliquity (Left Down) (°)*	-27 ± 4.1	-31.8 ± 6.7	-30.6 ± 1.6	-33.2 ± 7.1	-21.6 ± 9.4	-27.9 ± 3.4	-25.9 ± 6.5	-32 ± 5.0
Lumbar Flexion Angular Velocity (°/s)	258.9 ± 44.8	289.5 ± 132.6	268.6 ± 57.7	316.4 ± 152.4	463 ± 360.1	241.3 ± 103.2	335.3 ± 72.8	227.3 ± 75.1
Lumbar Left Lateral Flexion Angular Velocity (°/s)	-177.4 ± 78.6	-124.1 ± 91.0	-218.5 ± 71.6	-162.7 ± 130.1	-198.8 ± 88.8	-152.1 ± 44.2	-204 ± 113.9	-134.4 ± 32.9
Lumbar Left Axial Rotation Angular Velocity (°/s)	-84.3 ± 26.7	-93.9 ± 56.0	-90.5 ± 40.4	-84.7 ± 56.3	-125.9 ± 129.4	-79.9 ± 75.0	-83.7 ± 33.1	-73.9 ± 41.8

“*” Significant main effect for serve type (P<0.01). “^” Significant main effect for sex (P<0.01).

TABLE 4.2. Peak lower limb and ball toss kinematics

	Pars				No Pars			
	Flat		Kick		Flat		Kick	
	Female	Male	Female	Male	Female	Male	Female	Male
Front Knee Angle (°)	77.3 ± 8.2	68.2 ± 5.7	78.9 ± 9.5	67.7 ± 5.7	66.6 ± 9.6	67.9 ± 8.0	65.9 ± 8.9	69.2 ± 8.8
Back Knee Angle (°)	73.0 ± 9.6	79.9 ± 10.2	76.2 ± 11.8	79.3 ± 10.8	77.8 ± 8.5	73.1 ± 11.7	75.4 ± 11.2	73.6 ± 14.3
Front Hip Extension Angular Velocity (°/s)*	-186 ± 12.7	-193.9 ± 76.1	-198.9 ± 19.4	-199.2 ± 69.6	-255.2 ± 54.6	-214.1 ± 72.3	-236.3 ± 53.8	-203.7 ± 66.4
Front Knee Extension Angular Velocity (°/s)*	-452.5 ± 28.8	-462.4 ± 161.3	-505.8 ± 37.3	-507.2 ± 158.2	-581.9 ± 129.1	-478.7 ± 98	-633.5 ± 147.4	-520.7 ± 121.7
Back Hip Extension Angular Velocity (°/s)*	-175 ± 34.5	-230.2 ± 84.8	-173.5 ± 8.9	-236.3 ± 90.1	-291.3 ± 68.6	-240.1 ± 87.9	-262.7 ± 86	-231.6 ± 85
Back Knee Extension Angular Velocity (°/s)	-510.1 ± 41.1	-599.6 ± 149.7	-510.8 ± 32.8	-596.8 ± 123.9	-615.7 ± 107.4	-557.4 ± 90.4	-634.1 ± 136	-568.2 ± 94.9
Left Hip Joint Centre Velocity (m/s)*	1.4 ± 0.1	1.4 ± 0.4	1.4 ± 0.1	1.5 ± 0.4	1.6 ± 0.2	1.4 ± 0.2	1.6 ± 0.3	1.5 ± 0.3
Right Hip Joint Centre Velocity (m/s)	1.6 ± 0.1	2 ± 0.4	1.8 ± 0.1	2.2 ± 0.4	2 ± 0.2	2 ± 0.2	2.1 ± 0.4	2.2 ± 0.3
Racquet Velocity X (m/s)*	32.1 ± 2	37.3 ± 5.3	30.4 ± 1.2	32.8 ± 4.2	34.7 ± 3.2	37.2 ± 5.9	30.3 ± 3.5	31.5 ± 4.9
Racquet Velocity Y (m/s)	6.5 ± 1.5	8.3 ± 1.8	8.8 ± 2.1	8.4 ± 2.5	5.6 ± 2.3	6.9 ± 1.9	5.9 ± 2.3	7.7 ± 2
Racquet Velocity Z (m/s)*	23.3 ± 1.6	24.7 ± 2.1	22.8 ± 0.9	24.2 ± 2.9	24 ± 3.1	24.3 ± 3.8	22.8 ± 3.3	23 ± 3.7
Toss Height (cm)	319 ± 16.0	317.1 ± 33.5	319.1 ± 8.0	315.9 ± 23.0	331.6 ± 20.7	306.5 ± 26.1	332.8 ± 25.2	305.1 ± 25.2
Relative Toss Height (ratio)^	0.51 ± 0.01	0.55 ± 0.06	0.51 ± 0.01	0.55 ± 0.05	0.48 ± 0.04	0.54 ± 0.06	0.48 ± 0.05	0.55 ± 0.06
Lateral Impact Position (cm)*	-24.3 ± 6.0	-31.1 ± 18.8	-38.8 ± 11.2	-44.5 ± 24.1	-4.5 ± 25.5	-19.8 ± 21.1	-25.1 ± 22.0	-43.0 ± 24.1
Relative Lateral Impact Position (ratio)*	-0.15 ± 0.04	-0.18 ± 0.1	-0.24 ± 0.08	-0.25 ± 0.13	-0.03 ± 0.16	-0.12 ± 0.12	-0.16 ± 0.13	-0.26 ± 0.14
Forward Impact Position (cm)*^	-38.0 ± 13.6	-53.2 ± 31.8	-31.1 ± 11.1	-42.4 ± 12.2	-42.8 ± 10.8	-61.6 ± 11.9	-23.9 ± 13.7	-38.1 ± 16.1
Relative Forward Impact Position (ratio)*^	-0.24 ± 0.09	-0.3 ± 0.18	-0.2 ± 0.08	-0.24 ± 0.06	-0.27 ± 0.07	-0.37 ± 0.08	-0.15 ± 0.09	-0.23 ± 0.09
Impact Height (cm)	243.8 ± 5.8	257.7 ± 18.2	242.0 ± 8.0	257.0 ± 19.5	246.0 ± 11.2	250.9 ± 15.8	246.9 ± 12.5	249.1 ± 16.2
Relative Impact Height (ratio)	0.66 ± 0.02	0.69 ± 0.06	0.67 ± 0.01	0.68 ± 0.02	0.65 ± 0.02	0.66 ± 0.02	0.65 ± 0.02	0.67 ± 0.03
Drop Distance (cm)^	75.2 ± 10.9	59.4 ± 36.4	77.1 ± 3.7	58.9 ± 26.7	85.6 ± 20.8	55.6 ± 24.8	85.9 ± 26.3	56.0 ± 22.0

“*” significant main effect for serve (p<0.01). “^” significant main effect for sex (p<0.01).

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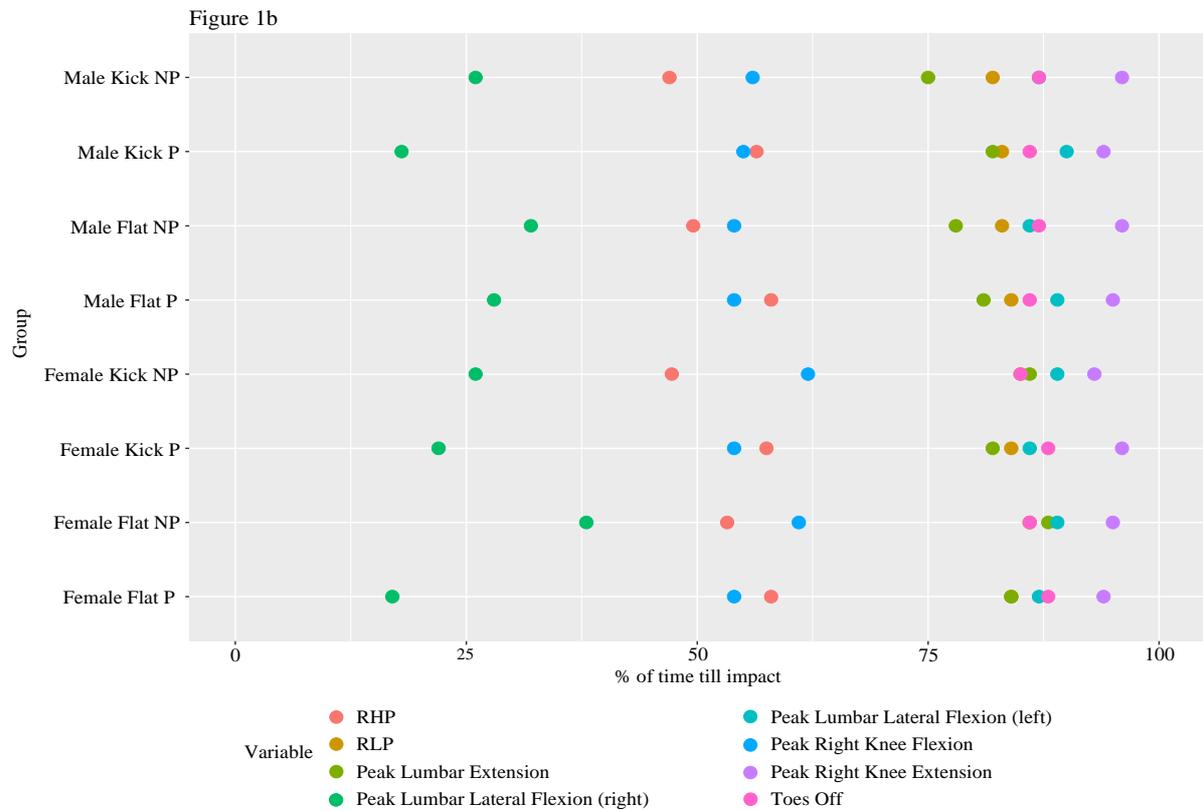
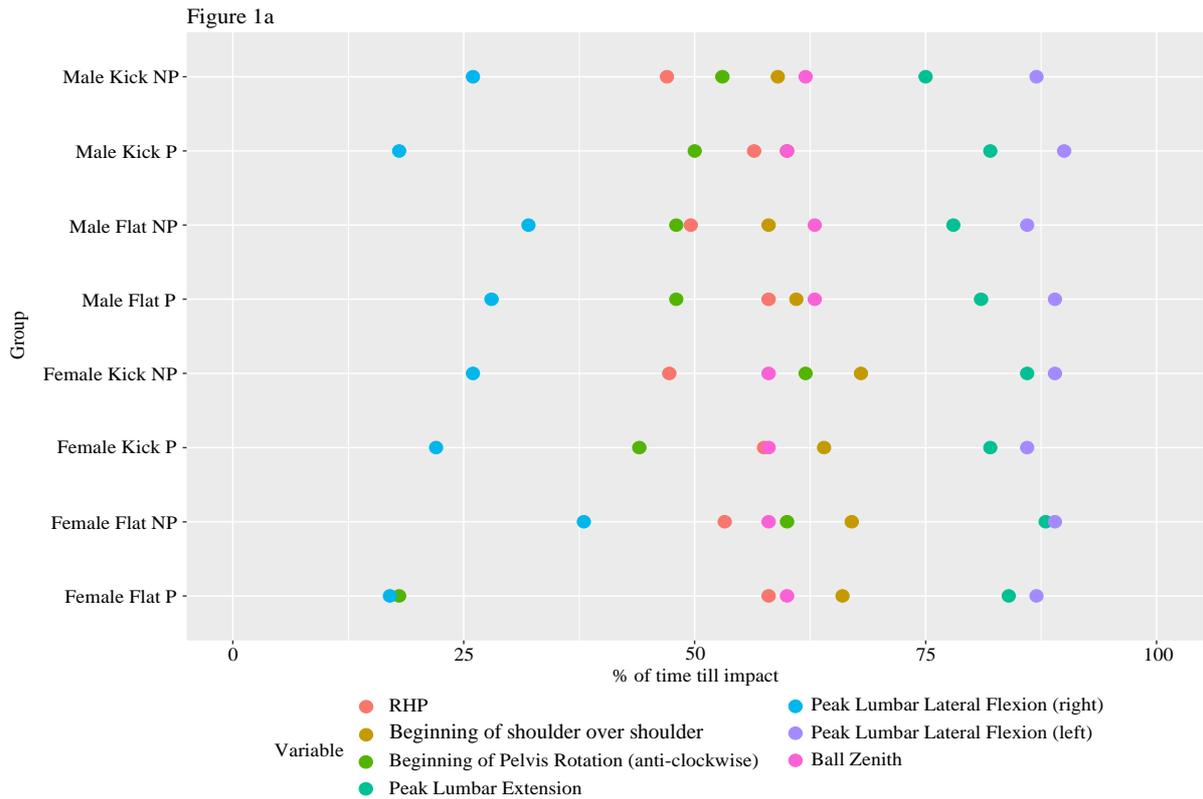


FIGURE 4.1. Charts 1a and 1b display the timing of key serve events throughout the serve as a percentage of time. 0% represents when ball toss occurs, 100% represents when racquet/ball impact occurs

4.5. DISCUSSION

This is the first study to investigate the relationship between lumbar spine abnormalities in asymptomatic elite adolescent players and serve kinematics. This study was also novel in its comparison of the effect of sex and serve type on the kinematics of the adolescent serve.

4.5.1. THE RELATIONSHIP BETWEEN LUMBAR ABNORMALITIES AND SERVE KINEMATICS

Surprisingly, the lumbar spine kinematics were comparable in magnitude, independent of pathology. Consequently, our first hypothesis (that stated: players with abnormalities would exhibit less dominant (right) side lumbar spine and pelvis rotation during the drive phase but greater non-dominant (left) side lateral flexion, lumbar spine rotation, pelvis rotation and anterior pelvis tilt during the forward-swing phase than players without abnormalities) was rejected. These findings contrast with previous research that has inferred a link between serve kinematics and low back pain among adolescent male tennis players (Campbell et al., 2014). Despite being informed by previous research (Campbell et al., 2014), our hypothesized reduction in lumbar and pelvis rotation in both the drive and forward-swing phases of those without pathology was not substantiated. Unexpectedly, lumbar left lateral flexion, lumbar and pelvis left rotation and pelvis anterior tilt were also comparable in the forward-swing phase. Whilst discrete kinematics are valuable in determining peak/moment-in-time differences, there are shortcomings of analyzing these values in isolation. For example, while there were no observed differences in peak lumbar kinematics, the timing of the kinematics did vary considerably between groups. These variations might prove instructive for coaches when identifying

players at risk of lumbar pathology (Cazzola, Pavei, & Preatoni, 2016). Specifically, coaches can observe the timing of lower limb and pelvis rotation leading into RHP to identify players who might be at risk of pars interarticularis abnormalities. Furthermore, the variation in age and skill level likely contributed to our findings. That is, the younger participants in our study displayed large amounts of variation in their kinematics, potentially indicative of still maturing technique. Similar kinematics may belie differential kinetics or electromyography, as has been shown in different types of adult serves (Chow, Shim, & Lim, 2003).

4.5.2. THE RELATIONSHIP BETWEEN SEX AND SERVE KINEMATICS

As expected, there were kinematic differences between the junior male and female serve. Peak posterior pelvis tilt was $\sim 11^\circ$ greater in male players during the drive phase of both the flat and kick serve. Most females adopted a more upright trunk posture during the ball toss (between $\sim 3\text{-}4^\circ$ more trunk extension), a probable by-product of these female players maintaining a neutral or anteriorly tilted pelvis during the drive phase compared to males. This trunk alignment tended to coincide with more pronounced peak front knee flexion, which saw female players assume a squat-like or more vertical (up-down) serve than male players.

Males made serve impact significantly further into the court on the flat (~ 16 cm) and kick (~ 15 cm) serves, even when held relative to their standing stature. The forward impact location of the adolescent male flat serve was similar to past research (Reid et al., 2010; Reid et al., 2011) that has found junior and adult players to impact the ball $\sim 52\text{-}58$ cm forward of the front toe. The adolescent female players in the current study however

tossed the ball up to 20cm closer to the baseline than previous descriptions of the adolescent female serve (Whiteside et al., 2013). It is possible that this was linked to the adoption of the abovementioned upright trunk position during the drive phase, which in turn likely contributes to reduced shoulder-over-shoulder rotation.

Interestingly, males impacted the flat serve 25 cm and kick serve 44 cm to the left of their front toe, which is substantially higher than some elite adult players (Reid et al., 2011). If we assume that the average standing height of male player in past research is 183 cm, then the difference in relative lateral impact position (adults: 0.19 ; adolescents: 0.26) is even more extreme. Although speculative, we expect that this leftward positioning of the ball relates to a combination of the heightened need to impart spin to the ball to clear the net as well as introduction of the kick serve at this age. Importantly, for players to position themselves in this way, there's likely to be compensation elsewhere. For example, pelvis obliquity (where the right hip was vertically higher than the left) was much higher than reported in other elite junior populations (Campbell et al., 2014). This appeared to result in players' bodies being rotated laterally, potentially explaining why players in this study impacted the ball further across their body compared to similar previously studied populations (Campbell et al., 2014). This type of alignment of the body could prove injurious if unconstrained and is worth coaches and health professionals monitoring.

4.5.3. THE RELATIONSHIP BETWEEN SERVE TYPE AND SERVE KINEMATICS

Flat and kick serve kinematics were notably different, largely supporting our third hypothesis. The kick serve displayed increased lumbar flexion and pelvis obliquity (left

down), suggesting that players adjust their sagittal plane lumbar kinematics and pelvis position to achieve laterally displaced impacts. Similar to the observed differences in impact position based on sex, serve type also significantly alters the relationship between ball and racquet at impact. As with previous research in the adult game (Reid et al., 2011), players in this study made flat serve impact significantly further forward (51cm vs kick: 34cm) and with higher horizontal velocity.

Interestingly, in contrast to previous work in elite tennis players (Chow, Carlton, et al., 2003), peak vertical racquet velocity was significantly higher for the flat serve. Conversely previous work has established that vertical racquet velocities are higher for second serves in order to impart topspin on the ball (Chow, Carlton, et al., 2003). A combination of comparatively smaller player heights and inexperience, as these junior players were likely only recently introduced to the kick serve, present as the most likely explanations of this finding.

4.5.4. TEMPORAL KINEMATICS

The timing of key serve events was different between the P and NP groups, upholding our final hypothesis. Specifically, peak right lumbar lateral flexion, pelvis left rotation as well as peak front knee flexion occurred prior to RHP in players with pathology indicating possible early initiation of leg drive. Indeed, this difference in sequencing coupled with their earlier engagement of peak right knee flexion meant that the RHP of players with pathology was substantially different to those without pathology. The importance of RHP to the serve's rhythm has been emphasized previously (Reid et al., 2010), and the lower

(8 cm) ball zenith of the P group afforded them less time to self-organise in order to impact the ball.

Female players with pathology reached peak lumbar extension and peak lumbar left lateral flexion earlier than players without pathology. This is likely related to their reduced lumbar extension and commencement of pelvis rotation prior to RHP. As the lumbar spine is extended during the drive phase, increasing the duration of time spent in lumbar extension may be deleterious due to the amount of stress placed on the spine in this position (Congeni et al., 1997).

Sample size was a limitation in this study due to the strict criteria and limited number of elite adolescent athletes available. This study also recruited players who reported as pain free at the commencement of the study.

4.6. CONCLUSION

The magnitude of discrete pelvis and lumbar spine kinematics, during the drive and forward-swing phases of the flat and kick serve, did not discriminate between elite adolescent players with and without lumbar abnormalities. Various kinematic differences were however observed between the male and female adolescent serve, which is interesting given that low back injury is more prevalent in male players. Significantly, in a departure from previous work, this study investigated and observed differential timing in the lower limb, pelvis and lumbar spine kinematics in the serves of players with and without lumbar abnormalities. Our data suggests that players with pathology might be initiating leg drive earlier than those without pathology. This provides some initial

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evidence suggesting that the way in which players arrive into RHP in their serves may be a risk factor in low back pain.

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CHAPTER 5

IS LUMBAR LOADING LINKED TO PARS

ABNORMALITIES IN ELITE ADOLESCENT TENNIS

PLAYERS?

Publication statement:

This chapter is currently under review in Sports Medicine – Open.

5.1. ABSTRACT

Objectives: The purpose of this study was to compare the serve kinetics of asymptomatic elite adolescent tennis players with and without lumbar spine abnormalities.

Design: Cross-sectional.

Methods: Twenty-four players carried out a series of flat and kick serves, whilst marker trajectories were recorded by a 3D motion capture system. Lumbar spine kinetics and key temporal events were compared between; those with and without pathology, sex and serves using a mixed-effects model.

Results: Lumbar spine kinetics were comparable between those with and without pathology as well as between sex. However, the temporal sequence of peak kinetic forces revealed that players with pathology entered RHP and peak posterior force later than those without (9% and 5% of serve later respectively, $p < 0.01$). Males also entered peak lumbar posterior force later than females (3% of serve later, $p < 0.01$). Lastly, the flat serve was characterized by greater peak lumbar extension moments compared to the kick serve ($p < 0.01$).

Conclusion: Lumbar kinetics do not appear to differ between sex and those with and without pathology, although greater lumbar extension moments are evident in the flat serve. Importantly though, the timing of RHP and peak lumbar posterior force is linked with the presence of lumbar spine abnormalities and thus might influence the presentation of low back pain.

5.2. INTRODUCTION

A well-executed serve is crucial in tennis as it is the only stroke that allows players to hit a winning point with immediate effect. Advancements in research and technology has allowed players to achieve greater ball speed for both serves and groundstrokes. However, increases in movement velocity has been linked to increases in kinetic loading (Elliott et al., 2003), which if high enough, could ultimately lead to tissue damage and or pain. Even though both racquet head and ball velocities have increased in groundstrokes (Landlinger, Stöggl, Lindinger, Wagner, & Müller, 2012), the serve still remains the most powerful stroke in tennis and thus it follows that the serve, which is highly dynamic, may lead to low back pain (LBP).

Low back pain is prevalent among elite junior tennis players (Gescheit et al., 2019) and often results in a significant amount of time away from training and tournaments (Campbell et al., 2013; Hjelm et al., 2010). It is among the most frequent reasons for visiting a tournament physician in professional tennis players at the Australian Open (Gescheit et al., 2017) and appears to be a problem throughout some players' careers. Since adolescence is a critical stage during an athlete's physical maturation, serious injuries to the lumbar spine can ultimately result in permanent withdrawal from the sport, and worse, long term damage to the spine. It is essential that further research is conducted in order to inform coaches and health practitioners of the risk factors associated with LBP in tennis.

The serve, has been speculated to be linked to the onset of LBP and spinal injury (Abrams et al., 2012; Abrams, Sheets, Andriacchi, & Safran, 2011; Campbell et al., 2013). The

serve necessitates fast trunk movements that can institute large spinal loads (Abrams et al., 2012) and result in stress on the posterior spinal structures. From a mechanical perspective, the kick serve necessitates greater lateral flexion and extension of the lumbar spine, movements that have been associated with lumbar injuries (Congeni et al., 1997; Cyron & Hutton, 1979; Ellenbecker et al., 2009; Kelsey, 1980; Roetert et al., 2009). However, the flat serve has been found to necessitate larger lateral flexion moments in tennis players with LBP and is thought to be linked with LBP (Campbell et al., 2013). Based on this, it is unclear whether one serve places the spine at greater risk of injury more than another.

Some studies have reported preliminary insights into the relationship between the serve and LBP in adolescent tennis players. Campbell et al. (2014) found that during the drive phase (between racquet high point (RHP; otherwise known as ‘trophy position’) and racquet low point (RLP)), adolescent players with LBP (age = 16.6 ± 1.4 years) had greater right lateral pelvis tilt, significantly less lower lumbar region right rotation and significantly smaller pelvis/shoulder separation angles (Campbell et al., 2014). Additionally, those with pain had greater lower lumbar rotation during the forward-swing phase, suggesting that those with pain had less lumbar mobility. Another study by Campbell et al. (2013) investigated the loading in the lumbar spine during first and second serves in the same group of players. It was found that the players who had LBP used significantly greater left lateral force of four times body weight throughout the drive phase in both flat and kick serves (Campbell et al., 2013). However, it is unclear whether these findings are adaptive or maladaptive to LBP and therefore whether the tennis serve is a risk factor for LBP remains uncertain.

Owing to the high prevalence and severity of LBP in elite adolescent tennis players, this study aimed to explore the relationship between sex, serve type and the presence of lumbar pars abnormalities and lumbar serve kinetics. Our first hypothesis is that lumbar forces will be associated with lumbar abnormalities. Our second hypothesis states that males will have greater lumbar forces compared with females. Our third hypothesis states that the kick serve will have larger loading on the lumbar spine compared with the flat serve. Finally, our fourth hypothesis states that there will be significant differences in the temporal sequence of peak kinetics between those with and without pathology.

5.3. METHODS

5.3.1. PARTICIPANTS

Twenty-four elite male (n=14) and female (n=10) tennis players aged 13.0 ± 1.6 years (range: 11 – 17 years) from a national tennis academy volunteered to take part in this study. To ensure appropriate participants were recruited, an exclusion criteria was established whereby participants who had a previous bout of severe LBP (severe LBP defined as seven or more days missed training and/or competition due to LBP, similar to Ranson et al. (2008)) during the last six months, were ill, had a performance inhibiting injury or experienced LBP during testing were excluded from the study. All participants had recently undergone a magnetic resonance imaging (MRI) scan which captured the lumbar spine from L1/L2 – L5/S1. Participants were then separated into either a ‘pars group’ (male: 7, female: 2) and ‘no pars group’ (male:7, female: 8) (P and NP respectively). Those in the P group were those who were found to have either bone marrow oedema or a stress fracture in the pars interarticularis, whereas those in the NP

group were those who did not have these MRI findings. Ethics approval was obtained from the Victoria University Human Research Ethics Committee and participants provided voluntary informed consent and assent prior to their involvement in the study.

5.3.2. PROCEDURE

A dynamic capture space (~ 2 x 2 x 2 m) was calibrated at the baseline of an indoor tennis court using a 12-camera opto-reflective motion capture system (Vicon Motion Systems Ltd, Oxford, UK; 250 Hz). A global reference frame was set at the centre mark on the baseline with positive X pointing towards the net, positive Y pointing directly leftward (along the baseline) when facing the net, and positive Z being the cross product pointing directly upward.

Prior to testing, participants' height and mass were recorded as well as their racquet parameters (mass, centre of mass (COM), and three-dimensional moment of inertia). Retro-reflective markers (12.7 mm diameter) and customized rigid plates (with markers attached) were attached to specific anatomical landmarks or positions on the lower body, upper body and trunk. The markers and plates were affixed used double-sided tape and rigid sports tape. Once the markers were attached to the participant, they were then given the opportunity to complete a self-directed warm-up which was then followed by subject specific static calibration trials.

Participants then completed a series of flat and kick serves to a target area (1 m x 2 m) bordering the 'T' at the deuce court. Participants were instructed to hit flat serves at maximal intensity aiming for the target area. Once three successful flat serves were achieved (successful serves were those that landed in the target area), the participant then

moved on to hit kick serves, as per prior established protocols (Campbell et al., 2014; Reid et al., 2011).

5.3.3. DATA PREPARATION AND MODELLING

The data was labelled, treated and processed using Vicon Nexus Software (Version 2.7.0., Vicon Motion Systems Ltd, Oxford, UK). Trajectories were filtered using a 15 Hz fourth-order low-pass Butterworth filter following a residual analysis and visual inspection of the data. Filtered anatomical, racquet and ball data were modelled using a customized and mathematical direct kinematic model (Crewe et al., 2013a; Wu et al., 2002; Wu et al., 2005). The segment parameters for the upper body, thorax and lumbar spine (i.e. length, mass, moment of inertia, radius of gyration, center of mass) were defined using information from previous research (de Leva, 1996; Pearsall et al., 1996; Pearsall et al., 1994).

The dependent variables of interest included selected normalized lumbar spine kinetics shown to be relevant to tennis serve performance and/or injury in past research (Campbell et al., 2013). These kinetics will be reported relative to the drive and forward-swing phase of the serve as previously defined by Whiteside et al. (2015). All kinetics analyzed and discussed will be related to the lumbar spine and normalized to individual body mass.

5.3.4. STATISTICAL ANALYSIS

Influential data points were identified using Cook's distance (Cook, 1979). An observation was considered influential if it had a Cook's distance greater than 0.02 and was excluded from the data.

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A mixed effects model was then used to determine significant differences within the data. Due to conducting multiple comparisons, the alpha value was adjusted a priori to 0.01 in order to reduce the risk of Type 1 errors (Elliott et al., 2003; Reid et al., 2010; Whiteside et al., 2013)

5.4. RESULTS

5.4.1. THE EFFECT OF PARS ABNORMALITIES ON SERVING KINETICS

A comparison between those with and without pars abnormalities revealed that lumbar forces and moments were comparable between the two groups during both the drive and forward-swing phase of the serve (Table 1). However, there was some evidence that those with pars abnormalities had greater peak lumbar right lateral flexion moments during the kick serve ($p < 0.02$).

5.4.2. THE EFFECT OF SEX ON SERVING KINETICS

There was no difference in lumbar kinetics between males and females (Table 1).

5.4.3. THE EFFECT OF SERVE TYPE ON SERVING KINETICS

Comparisons for lumbar kinetics between flat and kick serves found that the flat serve was characterized by greater lumbar extension moments (7.0 Nm/kg) compared to the kick serve (6.1 Nm, $p < 0.01$) (Table 1). Additionally, there was some evidence that players tended to have greater lumbar left lateral flexion moments during the flat serve compared with the kick (~0.1 Nm/kg difference, $p < 0.02$).

5.4.4. TEMPORAL SEQUENCE OF SERVE KINETICS

A comparison between key serve events revealed significant differences between the P and NP groups. Those in P group reached RHP and peak lumbar posterior force later compared with those in the NP group (57% vs 49% and 80% vs 75% of serve respectively, $p<0.01$) (Figure 1A). Peak lumbar posterior force appeared to occur earlier in females compared with males (75% vs 79% of serve respectively, $p<0.01$) as well as in the kick serve compared with the flat serve (76% vs 79% of serve respectively, $p<0.01$) (Figure 1B). Lastly, there appeared to be a trend for males to undergo peak lumbar left lateral flexion moment later (19% of serve) than females ($p<0.02$) (Figure 1C). In contrast, females underwent peak lumbar left axial rotation moment later than the males (7% of serve) ($p<0.02$).

TABLE 5.1. Peak lumbar kinetics for those with and without pars abnormalities.

	Pars				No Pars			
	Flat		Kick		Flat		Kick	
	Female	Male	Female	Male	Female	Male	Female	Male
Drive Phase								
Lumbar Anterior Force (N/kg)	18.8 ± 4.3	18 ± 19.0	22.9 ± 11.0	21.8 ± 19.1	22.5 ± 18.1	19.2 ± 11	24 ± 16.7	27 ± 17.4
Lumbar Posterior Force (N/kg)	-18.8 ± 6.1	-11.4 ± 12.5	-21.7 ± 8.9	-18.7 ± 16.9	-18.6 ± 14.3	-17.4 ± 10.4	-22.8 ± 11.6	-23.7 ± 15.4
Lumbar Compression Force (N/kg)	-10.1 ± 11.5	-4.9 ± 7.5	-13.6 ± 15.8	-6.9 ± 10.9	-7.4 ± 8.7	-4.5 ± 4.1	-8.3 ± 8.0	-9.2 ± 9.7
Lumbar Distraction Force (N/kg)	20.8 ± 6.9	16.5 ± 7.6	25.7 ± 11.3	18.5 ± 10.4	19.4 ± 7.0	17.9 ± 5.0	19.1 ± 4.5	20.3 ± 5.4
Lumbar Right Lateral Force (N/kg)	12.6 ± 3.5	7.2 ± 7.1	16.4 ± 9.4	7.9 ± 7.8	14.3 ± 9.0	12.8 ± 8.7	13.7 ± 7.0	12.5 ± 9.0
Lumbar Left Lateral Force (N/kg)	-11.6 ± 3.1	-9.8 ± 5.7	-16.3 ± 5.4	-12.8 ± 12.5	-16.1 ± 8.9	-15.3 ± 8.5	-18.3 ± 10.4	-16.6 ± 9.5
Lumbar Extension Moment (Nm/kg)*	-5.2 ± 2.0	-3.6 ± 3.1	-6.2 ± 3.4	-5 ± 3.9	-5 ± 3.1	-5.3 ± 2.8	-7.6 ± 4.1	-5.6 ± 3.5
Lumbar Flexion Moment (Nm/kg)	6.1 ± 3.6	4.6 ± 4.6	6.1 ± 2.1	5.7 ± 4.4	6.7 ± 4.6	6.5 ± 4.6	7.2 ± 3.4	6.5 ± 3.2
Lumbar Left Axial Rotation Moment (Nm/kg)	-3.9 ± 1.5	-3.4 ± 4.3	-4.5 ± 1.8	-3.6 ± 3.4	-4.2 ± 3.4	-4.9 ± 3.7	-5 ± 2.4	-4.2 ± 2.7
Lumbar Right Axial Rotation Moment (Nm/kg)	3.8 ± 1.4	3.2 ± 2.3	4.8 ± 2.2	4.4 ± 3.6	4.9 ± 3.1	5.5 ± 3.7	4.6 ± 2.4	5.5 ± 3.2
Lumbar Right Lateral Flexion Moment (Nm/kg)	5.7 ± 2.2	5.4 ± 3.0	9 ± 7.0	6.8 ± 4.4	6 ± 2.2	6.7 ± 3.4	5.5 ± 1.9	6 ± 2.7
Lumbar Left Lateral Flexion Moment (Nm/kg)	-3.7 ± 2.5	-2.1 ± 4.2	-4.8 ± 5.6	-3.2 ± 4.6	-3.3 ± 2.7	-3.4 ± 3.0	-2.8 ± 1.8	-2.6 ± 3.3
Forward-swing Phase								
Lumbar Anterior Force (N/kg)	13.1 ± 2.8	16.3 ± 14.2	18.3 ± 6.6	24.3 ± 21.8	24.3 ± 11.8	21.6 ± 16.2	28.1 ± 14.0	21.7 ± 14.2
Lumbar Posterior Force (N/kg)	-14 ± 5.6	-19.6 ± 18.9	-16.3 ± 7.1	-24.6 ± 21.2	-26.4 ± 19.4	-19.7 ± 11.6	-21.8 ± 10.7	-21.1 ± 11.2
Lumbar Compression Force (N/kg)	-14.5 ± 4.1	-16.8 ± 9.8	-15.9 ± 4.2	-17.1 ± 11.7	-19.6 ± 10.2	-16 ± 8.1	-17.9 ± 7.0	-17.2 ± 8.6
Lumbar Distraction Force (N/kg)	10.6 ± 3.0	11.4 ± 9.7	16 ± 12.7	11.5 ± 8.8	12.7 ± 8.3	13.4 ± 9.3	15.6 ± 8.7	14.8 ± 8.5
Lumbar Left Lateral Force (N/kg)	-18.5 ± 5.6	-15.7 ± 11.1	-19.7 ± 9.0	-19.8 ± 13.1	-21.6 ± 12.1	-15.6 ± 6.7	-22.3 ± 11.5	-18 ± 7.8
Lumbar Right Lateral Force (N/kg)	13.3 ± 4.8	14.3 ± 13.6	10.1 ± 2.5	12.1 ± 9.7	15 ± 12.0	13.7 ± 9.2	16.1 ± 10.1	13.1 ± 7.9
Lumbar Flexion Moment (Nm/kg)	4.2 ± 1.2	5.8 ± 4.0	5.8 ± 1.6	7.3 ± 4.8	8.7 ± 4.2	6.6 ± 4.0	9.1 ± 3.5	7.1 ± 4.3
Lumbar Extension Moment (Nm/kg)	-7.6 ± 7.7	-7 ± 3.9	-6.8 ± 2.3	-9 ± 5.5	-9.9 ± 6.1	-10.6 ± 6.0	-10.3 ± 4.7	-9.3 ± 4.9
Lumbar Left Lateral Flexion Moment (Nm/kg)	-3.8 ± 1.5	-6.4 ± 5.2	-3.1 ± 0.8	-5.1 ± 3.5	-4.3 ± 3.1	-5.1 ± 4.1	-5.9 ± 3.9	-4.6 ± 2.1
Lumbar Right Lateral Flexion Moment (Nm/kg)	6.4 ± 2.7	7 ± 5.1	5.2 ± 1.1	6.4 ± 4.2	6.7 ± 3.8	6.6 ± 4.2	6.7 ± 3.6	6 ± 2.7
Lumbar Left Axial Rotation Moment (Nm/kg)	-2.8 ± 1.3	-4.4 ± 3.2	-2.8 ± 1.0	-3.2 ± 2.2	-4.3 ± 2.7	-2.7 ± 1.2	-4.5 ± 2.9	-3 ± 1.9
Lumbar Right Axial Rotation Moment (Nm/kg)	2.4 ± 1.5	3 ± 1.7	2.6 ± 1.0	3.1 ± 1.6	3.2 ± 2.0	2.9 ± 1.9	3.7 ± 1.9	3.2 ± 1.6

“**” significant main effect for serve type.

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Figure 1A

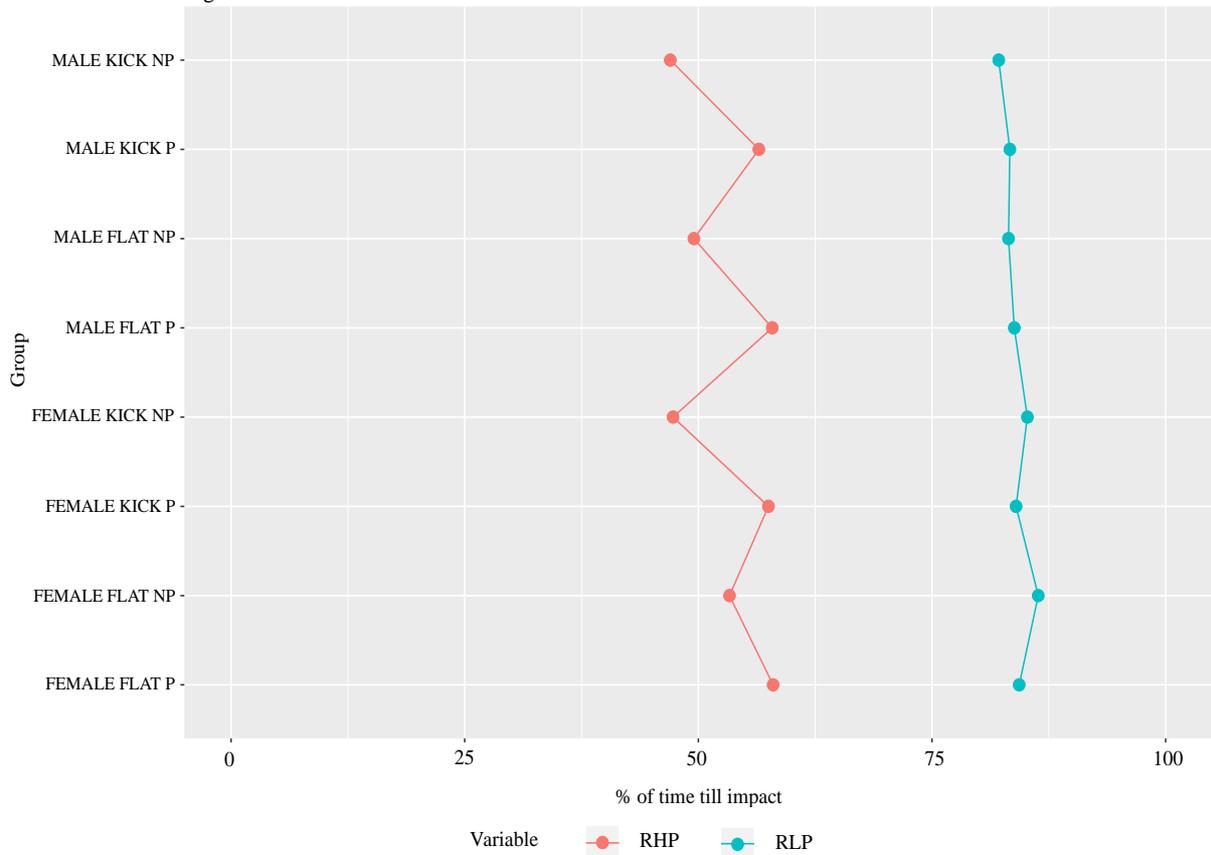
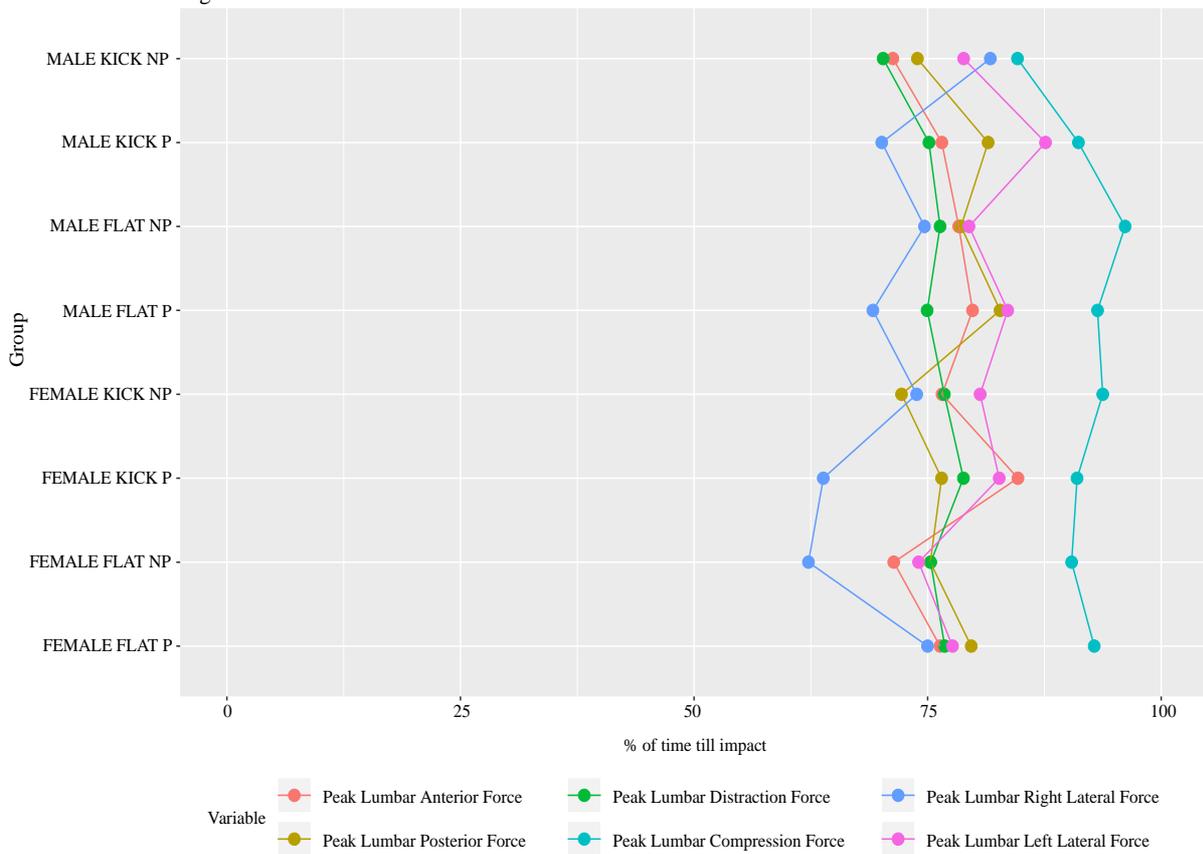


Figure 1B



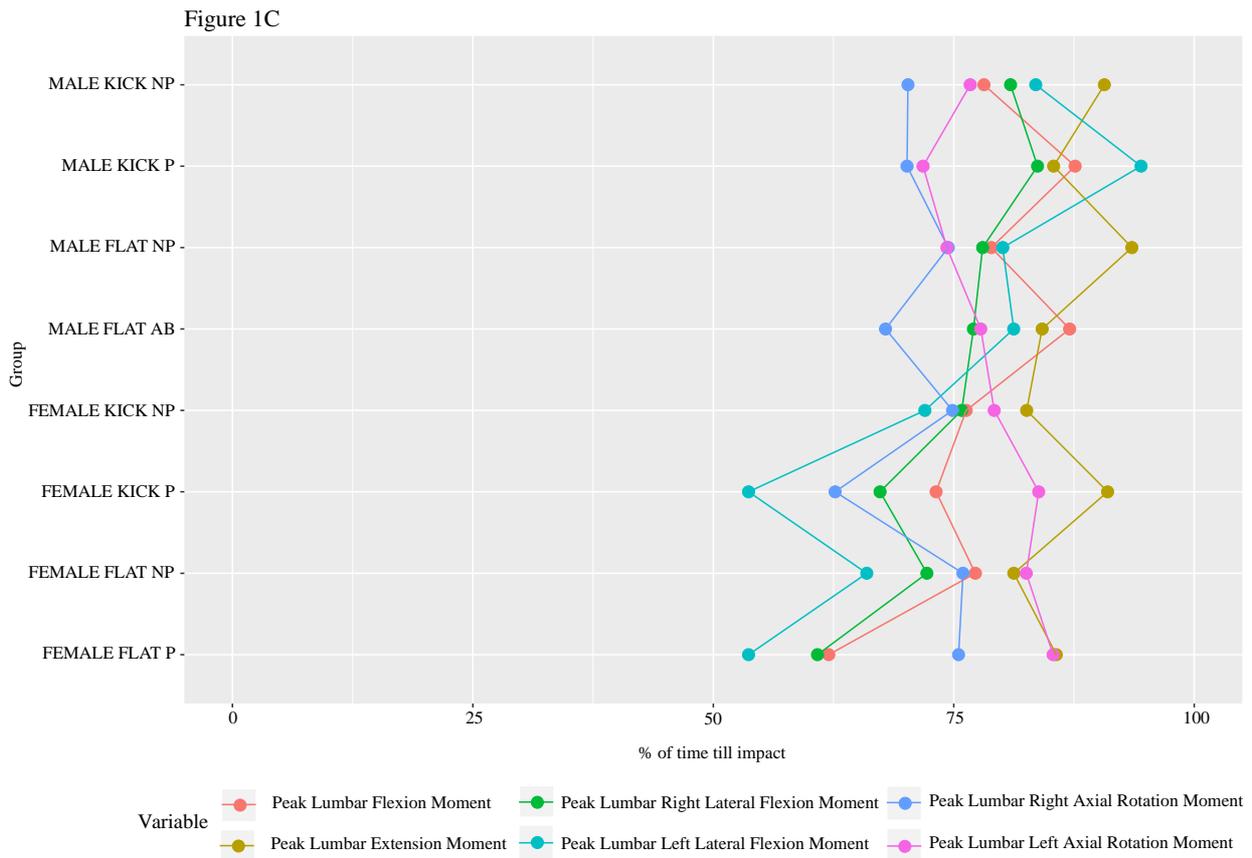


FIGURE 5.1. 1A, 1B and 1C are charts displaying when key serve temporal events occur throughout the serve as a percentage of time. Figure 1A shows the timing of racquet highpoint and racquet low point between groups. Figure 1B shows the timing of peak lumbar forces and key serve events. Figure 1C shows the timing of peak lumbar moments and key serve events. 0% represents when ball toss occurs, 100% represents when racquet/ball impact occurs

5.5. DISCUSSION

5.5.1. THE EFFECT OF PARS ABNORMALITIES ON SERVING KINETICS

Surprisingly, trunk and lumbar kinetics were comparable between participants with and without pathology and therefore our first hypothesis (that pars interarticularis abnormalities would be associated with greater lumbar loading) was rejected. It appears that peak forces and moments alone give little indication of lumbar spine abnormalities or pain in tennis players. A similar study carried out by Campbell et al. (2013) using

players with and without pain found that those with pain experienced significantly greater left lateral force during the drive phase of both flat and kick serves, compared to those without pain. Interestingly, the participants in our study used considerably more lateral flexion force (between -14.5 N/kg – -19.8 N/kg versus 2.6 N/kg – 4.1 N/kg in the study by Campbell et al. (2013)) however, no significant differences were found. Our values however, were similar to those found in cricket. Crewe, Campbell, Elliott, and Alderson (2013b) reported that fast bowlers (whom have very similar trunk movements to the tennis serve) had peak mediolateral forces between -13 – 16 N/kg which was comparable to our -19.8 – 18.3 N/kg. Since Crewe et al. (2013b) suggested that their lumbo-pelvic loading values were likely to cause lumbar injuries in fast bowlers, and the fact that our values are much higher than Campbell's who found lateral force to be linked to lumbar pain, it is peculiar that this study had no findings related to lumbar abnormalities. In addition, Bayne et al., (2016) found that there were significant differences in flexion/extension forces between injured and non-injured elite junior fast bowlers, suggesting that these differences likely influenced injury in the fast bowlers. In our study, the P group were diagnosed with either a lumbar stress fracture to the pars interarticularis and/or lumbar bone marrow oedema (BMO). It is known that tissue damage is a result of forces applied to tissue which are greater than that of which it can tolerate (Adams, McMillan, Green, & Dolan, 1996) and therefore it is possible that the reported values in our study could in fact be damaging other tissues which can lead to lumbar pain (such as discs and/or ligaments) rather than creating pars stress fractures. Ultimately, it appears that peak kinetic values in isolation are not helpful in understanding low back pain in tennis. Hence, we explored temporal sequences of the serve and the differences between abnormality, sex and serve type groups.

5.5.2. THE EFFECT OF SEX ON SERVING KINETICS

There were no significant differences in the peak kinetic values between males and females and therefore our second hypothesis was not supported. In the Australian National Tennis Academy, previous adolescent lumbar injuries have almost exclusively been in male players with few females sustaining LBP (unpublished data, Tennis Australia). Therefore, it is surprising to find that the lumbar spine loading is similar between males and females. Whilst differences have been found in other serve kinematics (Connolly, Middleton, & Reid, 2019), it appears that lumbar spine loading is comparable between males and females for both flat and kick serves.

5.5.3. THE EFFECT OF SERVE TYPE ON SERVING KINETICS

Finally, a comparison of peak lumbar kinetics between serve types found that the flat serve was characterized by greater peak lumbar extension moments ($p < 0.01$) compared to the kick serve and thus our third hypothesis was partially supported. Only one lumbar kinetic variable was found to differ between the flat and kick serve which was surprising given past research has confirmed that the kick serve places enormous load on tennis players' backs (Abrams et al., 2014) and speculates that it plays a key role in lumbar injuries (Sheets, Abrams, Corazza, Safran, & Andriacchi, 2011). Greater peak lumbar extension moments were found for the flat serve during the forward-swing phase, presumably to impact the ball further into the court. Additionally, peak extension moments occurred just after RLP (Figure 1A and 1C) and thus these higher values were likely a result of players transitioning from lumbar extension to flexion rapidly which is essential for impacting the ball further into the court. This contrasts with a kick serve

whereby lateral flexion and shoulder tilt are more important movements in order to impact the ball more laterally. Our finding is consistent with previous work by Campbell et al. (2013) who reported that lumbar extension moments were significantly higher during the flat serve compared with the kick serve. This finding was also Campbell's only finding in relation to lumbar loading between serve types, indicating that perhaps lumbar loading does not differ greatly between serve types at a junior level.

5.5.4. TEMPORAL SEQUENCE OF SERVE KINETICS

Analysis of the temporal sequencing of peak kinetic values found several differences in the timing of peak kinetic events between those with and without pathology and thus our fourth hypothesis was substantiated. Those with pathology reached RHP significantly later than those without pathology ($p < 0.01$) however entered RLP around the same time. This may have influenced the significantly later occurrence of peak posterior force in the P group ($p < 0.01$) due to RHP involving considerable lumbar extension. Other research has found that those with pars abnormalities have entered peak lateral flexion (right) and then begun pelvis rotation (anti-clockwise) before those without pathology and also before reaching RHP (unpublished data, Tennis Australia). With this in mind, the spine is then placed in a vulnerable position for longer in those with pathology with possible high posterior lumbar loading. It has been well established that hyperextension of the spine stresses the lumbar spine (Chosa, Totoribe, & Tajima, 2004) and therefore additional time spent in this position, combined with the repetitiveness of serving could potentially be damaging on the spine and may in part explain the cause for these abnormalities. A comparison between male and female temporal sequences revealed that males reached peak posterior lumbar force later than females ($p < 0.01$). With the

abovementioned extended time spent in lumbar extension, males might be subjecting themselves to more spinal damage than females.

This study was limited to a small sample size due to the strict inclusion criteria. This study was also limited to the population described and therefore may not be generalizable to other ages. Lastly, whilst this study explored peak kinetic values, we did not measure how long the athletes spent at or near those peak values. Future research might benefit from investigating the effect of impulse during lumbar loading and its effect on lumbar spine abnormalities.

5.6. CONCLUSION

Peak lumbar forces and moments were comparable between participants with and without pathology and cannot be linked to the presence of lumbar abnormalities. Similarly, no differences in lumbar loading was found between males and females. Lumbar extension moments were found to be greater during the flat serve as oppose to the kick serve, however no other observations were found between serve types. Importantly though, the timing of peak posterior force was significantly later in the serve for those with pathology and hence was found to be linked to the presence of lumbar abnormalities. Whilst peak lumbar kinetics provide little insight into the presence of lumbar spine abnormalities, the timing of peak forces appears linked to the presence of lumbar pathology and possibly LBP. Thus, the use of time series data in future investigations may provide a beneficial alternative to peak kinetic values when exploring lumbar spine kinetics in sport.

CHAPTER 6

**ARE PARS ABNORMALITIES LINKED TO LOW BACK
PAIN IN ELITE ASYMPTOMATIC ADOLESCENT
TENNIS PLAYERS?**

6.1. ABSTRACT

Objective: The purpose of this study was to investigate the morphological changes in the lumbar spine and episodes of low back pain among 25 elite junior tennis players over a 12-month period.

Materials and Methods: Lumbar spine MRI scans (L1/2 to L5/S1) of 25 elite asymptomatic adolescent tennis players (male = 14, female = 11) were obtained at the beginning of the study (baseline scan, T=0 months) and the follow-up scan was collected 12 months following baseline (T=12 months) for 18 players (7 withdrew from the study, male: 3, female: 4). Descriptive comparisons and confidence intervals were used to describe the prevalence of the abnormalities.

Results: 19 (76%) of 25 players were found to have at least one lumbar spine abnormality during the 12 months. Disc pathology was the most common form of abnormality (17/25, 68%), followed by degenerative changes (13/25, 52%) and pars abnormalities (12/25, 48%). Five participants went on to experience LBP following their baseline MRI. These participants had either disc pathology, degenerative changes or pars abnormalities or a combination of these abnormalities.

Conclusion. This study was the first to longitudinally analyse lumbar spine abnormalities and LBP in elite junior tennis players. Degenerative changes were the most prevalent abnormalities among this cohort, followed by disc pathology and pars abnormalities. It is difficult to be certain which abnormalities influenced low back pain in our study, particularly with so few participants experiencing LBP, although we suspect pars/bone marrow oedema (BMO) abnormalities are likely the most associated with LBP. Further research is required to determine what proportion of players with BMO go on to develop

LBP and whether early intervention can prevent the need for a longer period of rehabilitation later.

6.2. INTRODUCTION

Low back pain (LBP) is extremely common in elite junior tennis players (Campbell et al., 2014; Campbell et al., 2013; Campbell et al., 2016; Gescheit et al., 2017; Hjelm et al., 2010). Injury rates of 4.7 – 55.6 injuries per 1000 athletic exposure and 0.6 – 2.3 injuries per 1000 hours of playing exposure have been reported in tennis (Hjelm et al., 2012; Hutchinson et al., 1995; Kerr et al., 2015; Sell et al., 2012), with low back injuries ranking among most prevalent (Gescheit et al., 2017; Hjelm et al., 2010; Hutchinson et al., 1995). In training settings, they are similar pervasive with Campbell et al. (2014) reporting that 37.5% of elite junior male tennis players had missed training due to stress reactions within the lumbar spine. Further, Hutchinson et al. (1995) and Hjelm et al. (2012) found that LBP was reported in 16% to 33% of injured junior tennis players. These findings were reinforced by unpublished data from Tennis Australia who found that lumbar spine consultations in elite adolescent tennis players increased 10-fold (over 1000%) between 2005 and 2015, making the lumbar spine the most commonly consulted body region for adolescents in their programs.

In an attempt to explore possible risk factors for LBP in junior tennis players, prior research has investigated the presence of morphological abnormalities on the lumbar spine. Alyas et al. (2007) and Rajeswaran et al. (2014) found that >85% of asymptomatic elite adolescent tennis players had at least one lumbar spine abnormality. Similarly, Chapter 1 revealed that pars abnormalities were present in 36% of their sample of elite

junior tennis players, and also showed a possible link between the magnitude of facet joint angles and pars abnormalities. These studies, while offering some valuable insight, are limited by their cross-sectional nature and failure to observe changes in lumbar spine morphology over time.

Since players are subjected to long periods of time out of play following lumbar injuries, particularly following injuries to the pars interarticularis, the continued manifestation of LBP in tennis cannot be overlooked. Therefore, the aim of this study was to investigate the morphological changes in the lumbar spine and episodes of LBP among 25 elite junior tennis players over a 12-month period. We hypothesised that: 1) >90% of athletes will present with at least one abnormality; 2) at least one in five of players would suffer an episode of LBP consistent with previous research, 3) that players diagnosed with pars bone marrow oedema (BMO) at baseline will develop low back pain in the following 12 months.

6.3. METHODS

6.3.1. PARTICIPANTS

Twenty-five asymptomatic elite adolescent tennis players (male = 14, female = 11) aged 11 – 17 years (13 ± 1.7 years), in the Tennis Australia National Academy participated in this study. All participants were free from a current performance inhibiting injury, illness or LBP at the time of scanning. All players were right-handed which means that abnormalities described as dominant side will be synonymous with the right-side.

Ethical approval was obtained from the Victoria University Human Research Ethics Committee while participants provided voluntary informed consent and assent prior to any involvement in the study.

6.3.2. IMAGING TECHNIQUE

All Magnetic Resonance Imaging (MRI) was carried out using 3-T Siemens Verio and Vida scanners, Erlangen Germany. The following standard sequences were performed. **Sagittal:** Sagittal T2, TR 4880ms, TE 43ms, FOV 260mm, Matrix 384 x 384, Slice thickness 3.5mm, 4.2mm separation, Sagittal STIR, TR 4020ms, TE 53ms, FOV 300mm, Matrix 384 x 384, Slice thickness 3mm, separation 3.75mm, Sagittal T1, TR 550ms, TE 11ms, FOV 260mm, Matrix 768 x 768, slice thickness 3.5mm, separation 4.2mm. **Axial:** Axial T2 TR 3380ms, TE 87ms, FOV 240 x 240mm, Matrix 448 x 444, slice thickness 4mm separation 4.4mm, Sagittal T1 3D fat saturated VIBE, TR 7ms, TE 2.5ms, FOV 200 x 200mm, Matrix 256 x 256, slice thickness 2mm. Parasagittal T1 fat saturated VIBE images were reformatted through the lumbar pars interarticularis at 1mm thickness.

6.3.3. DATA COLLECTION

Lumbar spine MRI scans (L1/2 to L5/S1) of each participant were obtained at the beginning of the study (baseline scan, T=0 months) and the follow-up scan was collected 12 months following baseline (follow-up scan, T=12 months). The MRI scans were randomly distributed between five experienced radiologists for analysis following an inter-observer reliability test (as per Chapter 3). The radiologists assessed the lumbar spine abnormalities listed in Chapter 3, Table 3.1. Information of the timing and severity of episodes of LBP were recorded by experienced tennis physiotherapists using an athlete

management system (AMS). Significant LBP was defined as the player visiting the academy physiotherapist reporting LBP which resulted in modified training or rest.

Radiological findings were divided into four groups based on their reported MRI findings: disc pathology (DP), pars abnormalities/BMO (PB), degenerative changes and other (DO) and no abnormalities (NIL). The pathologies for each group are described in Table 1. Whilst it could be argued that the PB group be collapsed within the other groups, it was decided that pars abnormalities and BMO be treated independently owing to their severity and frequency. Disc degeneration was assigned to fall under the DP group in this study as the changes were deemed directly relevant to disc pathology.

Seven participants (male: 3, female: 4) withdrew from the study prior to the follow-up scan due to either leaving the tennis academy prior to T=12 or not wishing to participate in the follow-up MRI scan. The baseline scan data of these participants will still be reported (e.g. x/25 players) although these participants' data will not feature in the follow-up scan results (e.g. x/18 players).

TABLE 6.1. MRI findings included in each participant group

Group	MRI findings contain 1 or more of:
Pars abnormalities/BMO	<ul style="list-style-type: none"> • Pars Abnormalities • Bone Marrow Oedema • Spondylolisthesis
Disc pathology	<ul style="list-style-type: none"> • Disc Herniation • Annular Fissure • Disc Degeneration • Schmorls nodes • Nerve Root Compression
Degenerative changes/other	<ul style="list-style-type: none"> • Facet joint degeneration/synovial cysts • Canal Stenosis • Foraminal Stenosis • Modic Changes • Scheuermann's Disease • Spina Bifida
No abnormalities	Clear MRI scan, no abnormalities found

6.3.4. STATISTICAL ANALYSIS

For this descriptive study, mean and standard deviations were calculated for age. Confidence Intervals (CI) were calculated to consider the prevalence of the lumbar abnormalities in elite junior tennis players in a broader setting using RStudio software (version: 0.99.903, RStudio: integrated Development for R. RStudio, inc., Boston, MA). Cook's distance was utilized to remove influential data points from the facet joint

orientation data (Cook, 1979) followed by a generalized linear regression model to determine potential links between facet joint orientation and the presence of pars abnormalities.

6.4. RESULTS

6.4.1. PREVALENCE OF RADIOLOGICAL ABNORMALITIES

The prevalence of lumbar spine abnormalities in 25 elite junior tennis players over 12 months is depicted in Figure 1 and Table 2. At baseline, there were 25 participants, however, only 18 participated in the follow-up scan. Excluding the four participants (ID: 11, 12, 13, 18, Table 2) who were clear at baseline but later withdrew prior to the second scan, 19/21 (90.5%) individual players presented with an abnormality at either baseline, follow-up or both, 16/21 (64%) players presented with an abnormality at baseline (T=0, Table 2) and 15/18 (83%) players had a lumbar abnormality at follow up (T=12).

6.4.2. PAIN

The prevalence of LBP among this cohort is depicted in Table 2. In this study, five out of 25 athletes developed LBP (i.e. they visited the academy physiotherapist reporting LBP which resulted in modified training or rest) following their baseline scan. Consequently, our second hypothesis was supported. Two of these participants were clear of abnormalities at baseline (#3 and #15, Table 2), whilst the other three participants presented with either one or a combination of pars/BMO, degenerative changes and disc pathology. All participants who experienced LBP between T=0 and T=12 were pain-free when undergoing the follow-up MRI scan. Lastly, one player (#5, Table 2) who reported

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LBP and was diagnosed with pars abnormalities at baseline, was excluded from the follow-up scan due to withdrawing from the study before T=12.

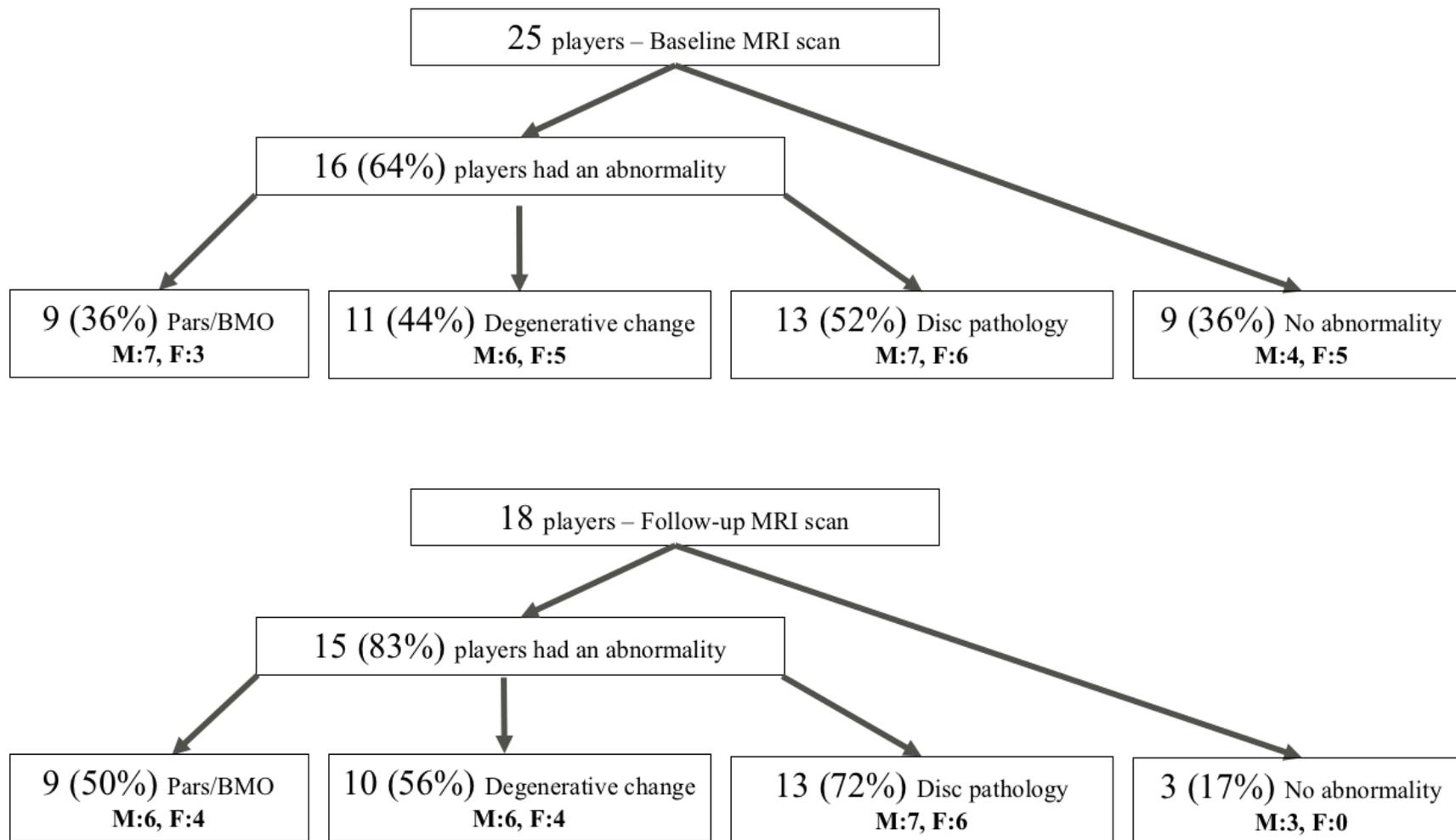


FIGURE 6.1. Flow charts displaying MRI findings from the baseline and follow-up scans.

TABLE 6.2. A list of the participants and their respective lumbar spine abnormalities detected at the baseline (B) and follow-up (F) scan. ID = participant ID, NP = No pain, P = pain, M = male, F = female. Dark grey = Present, White = Absent, Light grey = withdrew from study

ID	1	2	4	6	7	8	9	10	11	12	13	14	16	17	18	20	21	23	24	25	3	5	15	19	22
Pain	NP	P	P	P	P	P																			
Sex	M	M	F	M	M	F	F	M	F	F	F	F	F	M	M	M	M	M	F	F	M	M	F	M	M
Scan	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F	B F
PB	Dark	Dark	Dark	Dark	Dark	Dark	White	White	Light	Light	Light	Dark	White	White	Light	Dark	Dark	White	White	Dark	White	Dark	Light	White	Dark
DO	Dark	Dark	Dark	Dark	Dark	Dark	White	Dark	Light	Light	Light	Dark	White	Dark	Light	Light	Dark	White	Dark	Dark	White	Dark	Light	White	Dark
DP	Dark	White	Light	Light	Light	Dark	Dark	Dark	Light	Light	Dark	White	Dark	Dark	White	Dark	Light	White	Dark						
NIL	White	White	White	White	White	Light	White	Dark	Light	Dark	Light	White	Dark	White	Dark	Light	White	Dark	Dark	White	Dark	Dark	Light	Dark	White

6.4.3. PARS/BONE MARROW OEDEMA

6.4.3.2. BASELINE

At baseline, 9 participants (36%, 95% CI: 19% – 57%) (male: 7, female: 2) were diagnosed with a pars abnormality at either L4 and L5, with 6 of those participants having BMO (male: 5, female: 1, Table 3). The participants were diagnosed with either bilateral and/or isolated dominant side abnormalities while no isolated non-dominant side abnormalities were found. Mostly stress reactions (pars abnormality without a fracture, grade 1) and chronic complete fractures (grade 4) were found at the pars followed by two instances of active incomplete fractures (grade 2a) and one chronic incomplete fracture (grade 2b). No active complete fractures (grade 3) were detected at baseline.

6.4.3.3. FOLLOW-UP

At follow-up, 9 participants (50%, 95% CI: 29% – 71%) (male: 6, female: 3) were diagnosed with pars abnormalities at L4 and or L5, with 6 of those participants having BMO (male: 4, female: 2). Six of these 9 participants were also in the PB group at baseline (Table 2). Four bilateral, 6 isolated dominant side and 1 isolated non-dominant side pars abnormalities were observed. Two participants had multi-level pars abnormalities. Five stress reactions, one active incomplete fracture, two cases of chronic incomplete fractures and three instances of chronic complete fractures were diagnosed. Similar to the baseline scan, no active complete fractures were detected at follow-up.

At follow-up, three new cases of pars abnormalities presented, i.e. three participants who did not have a pars abnormality at baseline though had one at follow-up. One female participant (#15, Table 2) had a clear MRI at baseline though experienced LBP between

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T=0 and T=12 and presented with a stress reaction at L4 at follow-up but she was painfree. The other two participants (#2 and #14, Table 2) developed either a stress reaction or dominant side active incomplete fractures at L4 though did not suffer LBP.

Of the 6 participants who had BMO at follow-up, only one (mentioned above) female participant suffered from LBP in the previous 12 months. This participant had a BMO ratio of 2.0 on the dominant side pars (Table 3). At the baseline and follow-up scans, 9 and 3 (additional) participants presented with a pars abnormality respectively, leading to a total of 12 participants presenting with a pars abnormality between T=0 and T=12. Further, 10 of these participants presented with some degree of bone marrow oedema.

TABLE 6.3. Bone marrow oedema (BMO) gradings at baseline and follow-up scans and instances of low back pain. Grey = BMO was not present for this scan OR, the participant withdrew. BS = Bone stress, AIF = active incomplete fracture

Participant	Diagnosis	Baseline scan				Follow-up scan				Pain between T=0 and T=12
		Level	Ratio dominant-side Pars	Ratio Non-dominant-side Pars	Bone stress vs fracture	Level	Ratio Dominant-side Pars	Ratio Non-dominant-side Pars	Bone stress vs fracture	
5	Bilateral	L5	4.4	3.6	BS					Yes
7	Right	L4	2.1	1.1	BS	L4	2.9	1.2	BS	No
20	Right	L5	2.6	0.9	AIF					No
21	Bilateral	L5	2.2	1.5	BS	L5	2.7	2.2	BS	No
22	Left	L4	1.2	1.7	AIF					Yes
25	Bilateral	L5	1.7	1.8	BS					No
2	Right					L4	3.5	1.4	BS	No
6	Left					L4	1.4	2.4	BS	No
15	Bilateral					L4	2	1.3	BS	Yes
16	Right					L4	2.7	0.5	AIF	No

6.4.3.4. PAIN

Overall, 3/12 participants who were diagnosed with pars/BMO abnormalities at either baseline or follow-up, developed LBP during this study. Two male participants in the PB group at baseline developed LBP during the following 12 months. One participant was diagnosed with bilateral stress reactions at L5 whilst the other was diagnosed with an active complete fracture on the left pars at L4 and a chronic complete fracture on the right pars at L4 (participant 5 and 22 respectively, Table 2). The former participant (#5) had developed LBP shortly after the significant but asymptomatic BMO was seen on his baseline scan. He withdrew prior to the follow-up scan. The latter participant suffered LBP though this had resolved prior to the follow-up scan. Additionally, at follow-up, his active incomplete fracture had transformed into a chronic incomplete fracture and he was diagnosed with an additional chronic incomplete fracture diagnosed at his L5 right pars. Lastly, one female participant (#15, Table 2) who was clear at baseline developed LBP during the following 12 months and was diagnosed with a stress reaction at L4 at follow-up.

6.4.4. DISC PATHOLOGY

There was a high prevalence of disc pathology at baseline (52%) and follow-up (72%) (Figure 1) in this study, despite the players being pain-free at the time of recruitment.

6.4.4.2. BASELINE

Thirteen participants were in the DP group at baseline (52%, 95% CI: 32% – 72%) (male: 7, female: 6). Disc degeneration was the most common diagnosis in this group followed by disc herniation and annular tears.

6.4.4.3. FOLLOW-UP

At follow-up, 13 participants (72%, 95% CI: 46% – 89%) were in the DP group. Nine of these participants were diagnosed with DP at baseline while the remaining four developed DP following the baseline scan. Additionally, two participants who had DP at baseline withdrew from the study prior to T=12, whilst another two participants who presented with DP at baseline, did not present with DP at follow-up (both participants had disc herniations at baseline which had resolved).

In total, 17/25 participants presented with DP between T=0 and T=12 months. Disc degeneration and disc herniation were the most common abnormalities followed by one instance of an annular fissure. No instances of Schmorl's nodes or nerve root compression were diagnosed. As mentioned above, due to the high prevalence of disc pathology in the participants of this study (including those that did and did not experience LBP), pursuing further analysis to draw links between DP and LBP was not thought to be appropriate.

6.4.5. DEGENERATIVE CHANGES/OTHER PATHOLOGY

Thirteen of 25 participants were diagnosed with degenerative changes (or other) during this study. The most common abnormalities diagnosed were facet joint degeneration and

spina bifida followed by spinal canal and foraminal stenosis and Scheuermann's disease.

No instances of modic changes were observed.

6.4.5.2. BASELINE

Eleven participants were in the DO group at baseline (44%, 95% CI: 25% - 65%) (6 male, 5 female). All participants were diagnosed with one or more of the following abnormalities: facet joint degeneration, canal stenosis, foraminal stenosis, modic changes, scheuermann's disease and spina bifida.

6.4.5.3. FOLLOW-UP

Three of 12 participants in the DO group at baseline did not participate in the follow-up. Two of these participants withdrew from the study and the remaining participant's abnormality (foraminal stenosis) had resolved during the course of the study. At follow-up, 10 participants (55.6%, 95% CI: 31% - 78%) (6 male, 4 female) belonged to the DO group. Eight of these participants were in the DO group at baseline, presenting with the same abnormalities 12 months on. Of the two male participants that presented with DO at follow-up only, (#7 and #22, Table 2), one participant presented with grade 2 facet joint degeneration at level L4/5 on the non-dominant side and the other participant presented with grade 1 canal stenosis at L5/S1.

Similar to disc pathology, degenerative changes were pervasive in this cohort, irrespective of those who did and did not experience LBP. Therefore, undertaking further analysis linking DO to LBP was deemed unsuitable.

6.4.6. NIL ABNORMALITIES

6.4.6.2. BASELINE

Nine participants were clear at baseline (36%, 95% CI: 19% – 57%) (4 male, 5 female).

Four of the participants with no abnormalities withdrew from the study while another two participants remained clear at follow-up. The remaining 3 participants developed abnormalities following baseline.

6.4.6.3. FOLLOW-UP

Three of 18 participants were clear at follow-up (16.7%, 95% CI: 4% - 42%) (3 male), including one participant whose abnormality had resolved from baseline. This abnormality was a disc herniation (1A) at L5/S1.

6.4.6.4. PAIN

Of the 9 participants who were clear of abnormalities at baseline, 3 developed LBP during the study. One male participant (#3, Table 2) was clear of abnormalities at both baseline as well as follow-up, though experienced LBP in between these scans. One female (#15, Table 2) was clear of abnormalities at baseline though developed LBP following this scan and was diagnosed with lumbar abnormalities at follow-up. Lastly, one male participant was diagnosed with lumbar abnormalities at baseline though despite developing LBP between baseline and follow-up was declared clear of lumbar abnormalities at follow-up.

6.5. DISCUSSION

It is common for elite adolescent tennis players to present with asymptomatic lumbar abnormalities, though the link between these abnormalities and low back pain (LBP)

remains unclear. To the authors' knowledge, this is the first prospective study investigating lumbar spine abnormalities and the occurrence of LBP in elite adolescent tennis players over the course of 12 months. The discussion below will critique the results with regard to clinical and practical implications.

6.5.1. PARS/BONE MARROW OEDEMA ABNORMALITIES

Overall, pars abnormalities (with or without BMO) were the third most common abnormality found in this sample, affecting 48% (12/25) of participants and being found mostly in males compared to females (8/14 vs 4/11 instances respectively). The overall prevalence is higher than the 27-30% reported by other studies who have also investigated lumbar abnormalities in elite junior tennis players (Alyas et al., 2007; Rajeswaran et al., 2014). A potential reason for the higher prevalence of pars abnormalities in this study could be the lower mean age for our cohort (13 years). Tennis athletes are scouted as young as 10 years of age and therefore when entering academy structures may undergo several changes in training approach (e.g. new coaching, equipment modification, increased volume and intensity) whilst going through significant growth periods with bony structures not yet fully developed (Maquirriain, 2006). Furthermore, these young players may exhibit still-developing or suboptimal technique, which can predispose athletes to back injuries, particularly in a sport like tennis that involves such high speed and repetitive joint rotations (Harvey & Tanner, 1991).

In this study, pars abnormalities were almost exclusively diagnosed as bilateral or isolated on the dominant side, with only one abnormality diagnosed in the non-dominant pars at follow-up. At baseline, pars abnormalities diagnosed were mostly bilateral, however at

follow-up, there were more dominant side pars abnormalities (dominant side: 6, bilateral: 4). This suggests handedness may play a role regarding the sites at which pars abnormalities develop, as is commonly seen in cricket (Crewe, Elliott, Couanis, Campbell, & Alderson, 2012). In cricket, pars abnormalities typically present on the non-dominant side, which is thought to be linked to the lateral flexion on that side (Crewe et al., 2012). The fact that this trend is not observed in tennis suggests that the biomechanical causes for pars injuries between the sports might differ. Although in order to conclude whether handedness influences the site of pars abnormalities in tennis, further research is required involving left-handed tennis players.

For this study, we also expected that pars abnormalities would be associated with LBP, however this hypothesis was only partially supported. Two participants from the PB group at baseline suffered LBP during the study. Although one of these participants (#5, Table 2) was also in the DC and DO group, it seems reasonable to propose that his pain was related to the BMO in pars on both sides. Interestingly, more broadly, the BMO gradings did not seem to predict which players would go on to experience LBP. Sims et al. (2019) recommended that a BMO grading of >2.0 required a period of rest or reduction in training load (4 weeks) before rescanning, while a grading of >3.0 may reflect later stages of pre-symptomatic lumbar spine bone stress injury and require additional rest. One participant at baseline presented with bilateral BMO with gradings >3.0 and went on to experience LBP (#5, Table 3), yet the other participant who experienced LBP following baseline had BMO gradings of <2.0 (participant #22). Additionally, a third participant at baseline (participant #25) had similar BMO gradings to participant 22, though did not

report LBP. This demonstrates the challenge in the early detection of bone stress injuries and that regular MRI scans may be beneficial for predicting bone stress injuries.

Interestingly, one participant at follow-up presented with a BMO grading of 3.5 at the dominant-side pars (participant #2) though did not report LBP. In comparison, participant #15 reported LBP prior to the follow-up scan though only presented with a BMO value of 2.0 on the dominant-side pars. In summary, predicting whether a player will develop LBP due to a pars bone stress injury on the basis of their BMO ratio is not currently possible. Further research exploring BMO ratios prior to, and following periods of high tennis workloads might better inform sports clinicians of BMO behavior and its response high workloads in tennis. Additionally, monitoring players who are known to have higher BMO gradings (e.g. >2.0) will enable clinicians to make more informed decisions if and when symptoms arise. This is especially relevant as other sports have found it can take up to 100 days for symptoms to arise following BMO detection (Sims et al., 2019).

6.5.2. DISC PATHOLOGY

Disc degeneration was the most commonly diagnosed abnormality within this study, affecting 68% (17/25) of participants. This finding is consistent with previous work identifying that disc degeneration is common among young children (Kjaer, Leboeuf-Yde, Sorensen, & Bendix, 2005; Salminen et al., 1999). However, it is important to note that disc degeneration is also common within non-athlete adolescents and therefore the prevalence of disc degenerative changes among our cohort cannot be solely attributed to playing tennis at an elite level. Further, given the emergence of spinal abnormalities at

young ages, it can be difficult to identify what a ‘normal’ spine may look like on an MRI (Kjaer et al., 2005).

With this in mind, it is hard to know the extent to which degenerative disc changes play part in the onset of LBP. Most clinicians do not consider disc degeneration alone to be responsible for LBP, especially as both asymptomatic adults and children have been diagnosed with disc degeneration (Boden, Davis, Dina, Patronas, & Wiesel, 1990; Jensen et al., 1994). In elite populations, the link between LBP and degenerative disc disease is also debatable. Some authors have suggested that the increased physical activity and subsequent loading on the spine leads to athletes being more susceptible to experiencing degeneration compared to normal populations (Ong, Anderson, & Roche, 2003). However, the mixed results of research attempting to link LBP to certain spine abnormalities such as reduced disc height (Lundin, Hellström, Nilsson, & Swärd, 2001) highlight the complexity of identifying morphological risk factors of LBP.

6.5.3. DEGENERATIVE CHANGES/OTHER

Degenerative changes affected 52% (13/25) of participants in the study, representing the second most common diagnosis among the sample, particularly among males (8 males, 5 females). Facet joint degeneration (FJD) was the most common degenerative diagnosis, affecting 28% of players and 50% of the DO group. These proportions are considerably lower than the 90% of players being diagnosed with FJD in past research (Rajeswaran et al., 2014). At baseline, 6 participants had FJD, though during the following 12 months only one other unique participant developed FJD. A possible explanation for the lower mean age of our cohort (13 years) compared to the previous studies (17-18 years) (Alyas

et al., 2007; Rajeswaran et al., 2014) as the prevalence of FJD is known to increase with age (Eubanks, Lee, Cassinelli, & Ahn, 2007; Kalichman & Hunter, 2007). Additionally, as facet joints play an important role in load transmission (Kalichman & Hunter, 2007), stabilizing the spine during flexion and extension and limiting axial rotation (Kalichman & Hunter, 2007), it is plausible that accumulation of serving repetition over time, which involves highly repetitive axial lumbar spine rotation under load (Abrams et al., 2014), may increase the incidence of FJD as players age.

The most commonly affected levels for FJD were L4/5 and L5/S1, which is expected (Alyas et al., 2007; Rajeswaran et al., 2014). The affected levels were mostly bilateral rather than isolated, which leads us to conclude that perhaps (on the contrary to our pars/BMO conclusion) the preferred hitting arm does not have impact on which side the FJD occurs. Lastly, it is difficult to ascertain whether FJD is linked to the cases of LBP in this study despite other evidence suggesting that the relationship is strong (Allegrì et al., 2016). Therefore, in adolescent tennis players, FJD may not be inextricably linked to the onset of LBP.

6.5.4. NIL ABNORMALITIES

Abnormality-free MRIs were observed among 10 participants (40%: 9 at baseline and 3 at follow-up); two of which were abnormality-free throughout. Seventy-six percent of the study's participants were therefore found to have an abnormality at either baseline or follow-up. Instructively, at baseline, 64% of players presented with asymptomatic abnormalities suggesting that clinicians should exercise caution when using MRI to assist with diagnosis, to avoid diagnosing false-positives.

6.6. LIMITATIONS

Our study had a low sample size due to the limited number of available domestic elite junior tennis players. Further, a significant portion of the available players had previously experienced LBP or were injured at the time, further reducing the eligible pool of prospective participants. This limits the generalizability of our findings. Secondly, we did not control for training history or extra-curricular activities and the influence they may have had on lumbar spine morphology. Lastly, participants who experienced LBP did not undergo an MRI scan when their pain presented. This limits our understanding of which abnormalities (if any) at follow-up are most likely associated with the onset of LBP.

6.7. CONCLUSION

Lumbar spine abnormalities are common in elite junior tennis players. In our prospective 12-month study, 76% of participants were diagnosed with at least one lumbar spine abnormality at either baseline or followup. Disc pathology was the most common form of abnormality, followed by degenerative changes and pars abnormalities. Five participants went on to experience LBP following their baseline MRI. These participants had either disc pathology, degenerative changes or pars abnormalities or a combination of these abnormalities or no abnormalities. Therefore, it is difficult to be certain which abnormalities influenced low back pain in our study, particularly with so few participants experiencing LBP, although we suspect pars/BMO abnormalities are likely the most associated with LBP. Several players demonstrated varying ratios of BMO in one or more

CHAPTER 6 ARE PARS ABNORMALITIES LINKED TO LOW BACK PAIN IN ELITE ASYMPTOMATIC ADOLESCENT TENNIS PLAYERS?

pars, both at baseline and follow-up. Further research is required to determine what proportion of players with BMO go on to develop LBP and whether early intervention can prevent the need for a longer period of rehabilitation later. Lastly, several participants had different abnormalities at follow-up compared to baseline, highlighting the complex interplay between morphology, maturation and tennis performance.

CHAPTER 7

**A LONGITUDINAL ASSESSMENT OF ELITE JUNIOR
TENNIS PLAYERS WITH LOW BACK PAIN: A CASE
SERIES**

7.1. ABSTRACT

Background: The purpose of this case report was to investigate the potential mechanisms of LBP (including BMO, serve technique and player workload) that arose in five elite adolescent tennis players over a 12-month period.

Methods: Two lumbar spine MRI scans were taken 12 months apart on five tennis players to ascertain the presence of abnormalities. All players entered in RPE and session duration daily into an athlete management system app, whereby total daily workloads were calculated. Lastly, a 12 camera 3D motion capture system was used to capture lumbar spine serving kinematics and kinetics. Players who visited the academy physiotherapist for a LBP consult which resulted in modified training or rest were considered for this study.

Results: Two of the 5 players presented with BMO in the MRI scans at baseline, one presented with BMO at follow-up only and the other two remained clear of lumbar spine abnormalities for the entire study. All players had a spike in workload that exceeded an ACWR ratio of 1.5 within the 14 days prior to the onset of LBP. Lastly, the magnitude and timing of discrete peak serving kinetics and kinematics were similar between the players, although the timing of the peak lumbar posterior force was most varied amongst the group.

Conclusion: Trends in workload appeared more linked to the onset of LBP than the presence of BMO or individual serving mechanics in this cohort. Due to the multifactorial nature of LBP however, it remains difficult to identify players at risk of LBP using the contemporary approaches to workload quantification, biomechanical analysis and MRI.

7.2. INTRODUCTION

Lumbar spine injuries in adolescent tennis athletes are extensive (Gescheit et al., 2019; Hjelm et al., 2012; Hutchinson et al., 1995) and if not managed properly, can be detrimental to an athlete's career. Low back injuries are one of the most frequent reasons for visiting a health practitioner in professional tennis (Gescheit et al., 2017), indicating that current low back pain (LBP) prevention and management should be improved. In particular, injuries to the pars interarticularis (part of the lumbar vertebrae) often result in up to 4-5 months of missed training and competition among young developing athletes (unpublished data, Tennis Australia).

Researchers have explored potential risk factors for LBP in adolescent tennis players such as lumbar spine abnormalities (Alyas et al., 2007; Rajeswaran et al., 2014) and hitting biomechanics (Campbell et al., 2014; Campbell et al., 2013; Campbell et al., 2016). However, these studies have been cross-sectional and/or accessed samples with a history of LBP, limiting the extent to which their findings can be directly linked to the onset of LBP.

To date, there has been no prospective research investigating the potential causes for LBP in elite adolescent tennis players. Further, the onset of LBP in tennis players is unpredictable and thus conducting research that relies on injuries occurring within a specified period of time is extremely difficult. This study, which was performed as part of a series of studies exploring LBP in adolescent tennis athletes, therefore focuses on the specific serving mechanics, workload and scan results of the participants who experienced LBP over the study's 12-month data collection period. This case report

approach was considered most meaningful and practical given that only five players experience pain. The purpose of the study therefore became to compare the technical, workload and MRI results of these five players in the context of their LBP.

7.3. METHODS

7.3.1. DEMOGRAPHIC INFORMATION

Five athletes (male: 4, female: 1, age: 15 ± 0 years, height: 178 ± 9 cm, mass: 66 ± 6 kgs, Table 1) who had experienced LBP during a 12-month prospective research project were recruited for this study. Low back pain was defined as having pain to the lower back that resulted in a visit to a physiotherapist and disrupted the athlete's training/competition (i.e. the players modified their training or rested). All players were between the ages of 14-16 years of age (Table 1).

7.3.2. MAGNETIC RESONANCE IMAGING

All players had undergone two lumbar spine MRI scans 12 months apart (during March – May in 2017 (baseline) and 2018 (follow-up)) as part of annual sport-specific screening protocols. These MRI scans captured levels L1/2 to L5/S1 using both sagittal and axial images. Experienced radiologists then noted whether a pars abnormality (as defined by Ang et al. (2016)) was present at any of the lumbar levels.

TABLE 7.1. Participant demographics. BS = Bone stress at the pars interarticularis, AIF = Active incomplete fracture at the pars interarticularis, IIF = Inactive incomplete fracture at the pars interarticularis.

ID	Age	Sex	Height (cm)	Mass (kg)	MRI 1 - Abnormality at the Pars?	MRI 2 - Abnormality at the Pars?	Did workload exceed a ratio of >1.5 during the study?	Workload "spike" ratio	Time between ACWR exceeding 1.5 and LBP occurrence (days)?	Date of Pain	Number of days between MRI 1 and LBP	Did pain resolve prior to follow-up MRI scan?
1	16	Male	185	71.0	No	No	Yes	1.9	19	2/10/2017	144	Yes
2	16	Male	181	69.4	BS	NIL	Yes	1.5	13	24/04/2017	20	NA
3	14	Female	165	56.3	No	BS	Yes	1.9	23	19/10/2017	167	Yes
4	16	Male	172.5	61.9	No	No	Yes	1.6	1	23/09/2017	178	Yes
5	15	Male	186.5	70.5	AIF	IIF	Yes	2.3	15	23/09/2017	136	Yes

7.3.3. WORKLOAD

All players recorded external workload (the duration of the session in minutes) and internal workload (Rating of Perceived Exertion (RPE)) for each session they performed over a 12-month period (for both competition and training) using an Athlete Management System (AMS) app on their phone (Figure 1). Using this information, overall daily workload was calculated by multiplying the two measures together; session duration multiplied by RPE (rating out of 10)) (Foster et al., 2001). This workload measurement was then used to calculate Acute Chronic Workload Ratios (ACWR) using an Exponentially Weighted Moving Average (EWMA) model to identify when players may

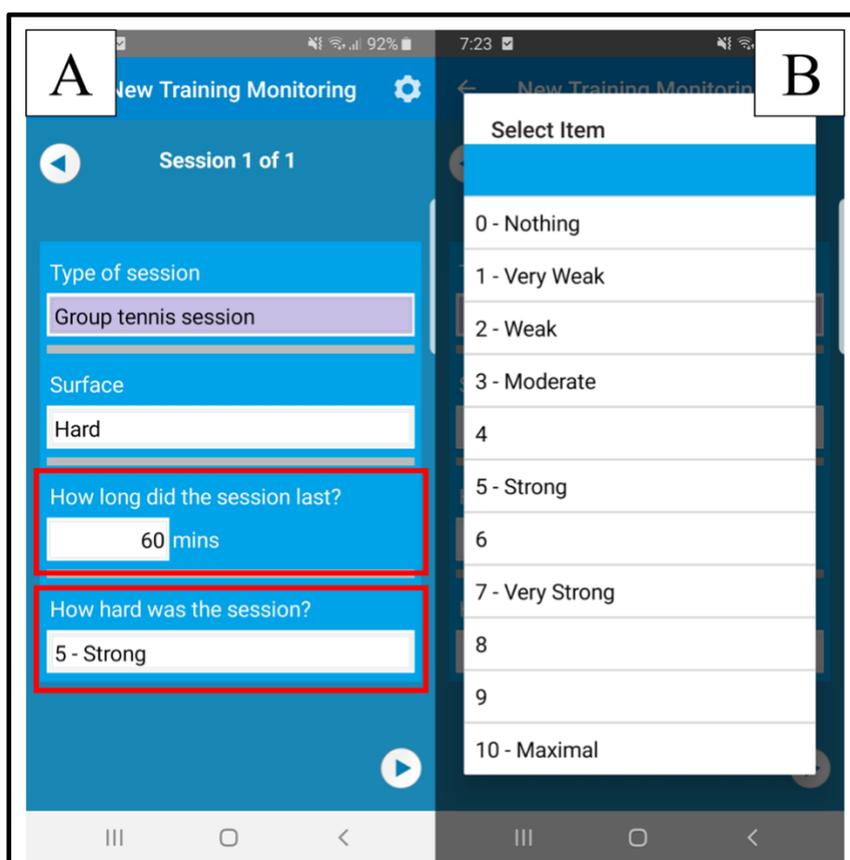


FIGURE 7.1. Screenshots of the Athlete Management System (AMS) app displaying how the athletes entered their workload data. In figure 1A the red boxes outline where athletes entered duration and RPE information. Figure 1B displays the dropdown

be at risk of injury using established methods (Murray, Gabbett, Townshend, & Blanch, 2017).

7.3.4. BIOMECHANICS

A serve analysis was carried out using a 12-camera opto-reflective motion capture system (Vicon Motion Systems Ltd, Oxford, UK; 250 Hz) in order to measure the kinematics and kinetics of the players' serves. A dynamic capture space (2 m x 2 m x 2 m) was calibrated on an indoor tennis court (hard surface). Participants' height, mass and racquet parameters (mass, center of mass (COM) and three dimensional moment of inertia) were recorded prior to attaching retro-reflective markers (12.7 mm) and customized rigid marker clusters to skin over relevant anatomical landmarks and segments on the lower body, upper body and trunk (Figure 2). Once all markers were attached, the participants completed a self-directed warm-up which was then followed by subject-specific calibration trials. The participants were then asked to perform a series of flat and kick serves at 'maximal intensity' to a target area (1 m x 2 m) bordering the "T" on the deuce court. Flat serves were performed first, followed by kick serves. Consistent with previous research, three successful flat and kick serves (those that landed in the target area) were used for analysis (Campbell et al., 2014).

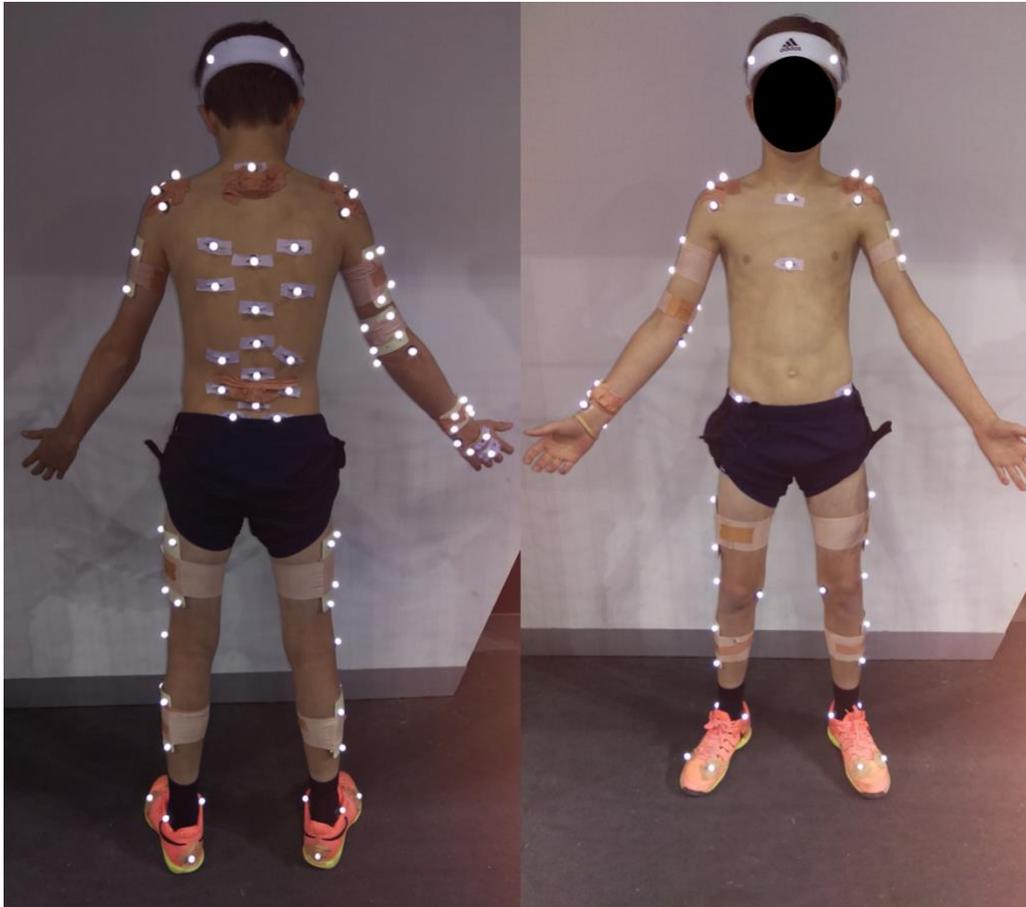


FIGURE 7.2. Retro-reflective markers attached to the skin

The data was processed using Vicon Nexus Software (Vicon Motion Systems Ltd, Oxford, UK) and gaps in marker trajectories were filled using the “Spline” fill function for individual markers and the “Rigid” fill function for markers that were placed on rigid structures (such as a customized marker plate). Marker trajectories were filtered using a fourth order low-pass Butterworth filter at 15 Hz following a residual analysis and visual inspection of the data. Filtered trajectory data were modelled using prior established kinematic and kinetic models (Crewe et al., 2013a; Wells, Donnelly, Elliott, Middleton,

& Alderson, 2018; Wu et al., 2002; Wu et al., 2005), and segment parameters for the upper body, thorax and lumbar spine were defined based on previous research (de Leva, 1996; Pearsall et al., 1996; Pearsall et al., 1994).

The dependent variables of interest were selected lumbar spine, pelvis, racquet and ball toss kinematics and kinetics identified as relevant to serve performance and/or LBP in past research (Campbell et al., 2014; Campbell et al., 2013; Reid et al., 2011). Kinematics were reported relative to the drive phase and forward-swing phase (Whiteside et al., 2015) and the temporal sequencing was held relative to serve impact.

7.4. RESULTS

7.4.1. MAGNETIC RESONANCE IMAGING

Three of the five participants (ID: 1, 3 & 4) were clear of abnormalities at the pars interarticularis at baseline (T=0) (Table 1). Participant 2 was diagnosed with bilateral bone stress at L5 while participant 5 was diagnosed with an active incomplete fracture (including bone marrow oedema (BMO)) at the left L4 pars and a chronic incomplete fracture (did not include BMO) at the right L4 pars.

At follow-up (T=12 months), participants 1 & 4 remained clear of pars abnormalities while participant 2 chose to withdraw from the study prior to the follow-up scan. Participant 3 was diagnosed with bone stress at L4 and participant 5 was diagnosed with chronic incomplete fractures (no bone stress) at L4 (left and right pars) and L5 right pars.

7.4.2. WORKLOAD

Participants tended to experience LBP 1-23 days following their ACWR being ≥ 1.5 (Table 1). The spike ACWR values were; 1.9, 1.5, 1.9, 1.6 and 2.3 for participants 1 – 5 respectively (Table 1). Participant 3 and 4 however, did not record workload data for an average of 10 days during the chronic period leading up to LBP. This resulted in a decreased in ACWR followed by a rise that was likely overestimated due to the lack of reporting (Table 1).

CHAPTER 7 A LONGITUDINAL ASSESSMENT OF ELITE JUNIOR TENNIS PLAYERS WITH LOW BACK PAIN: A CASE SERIES

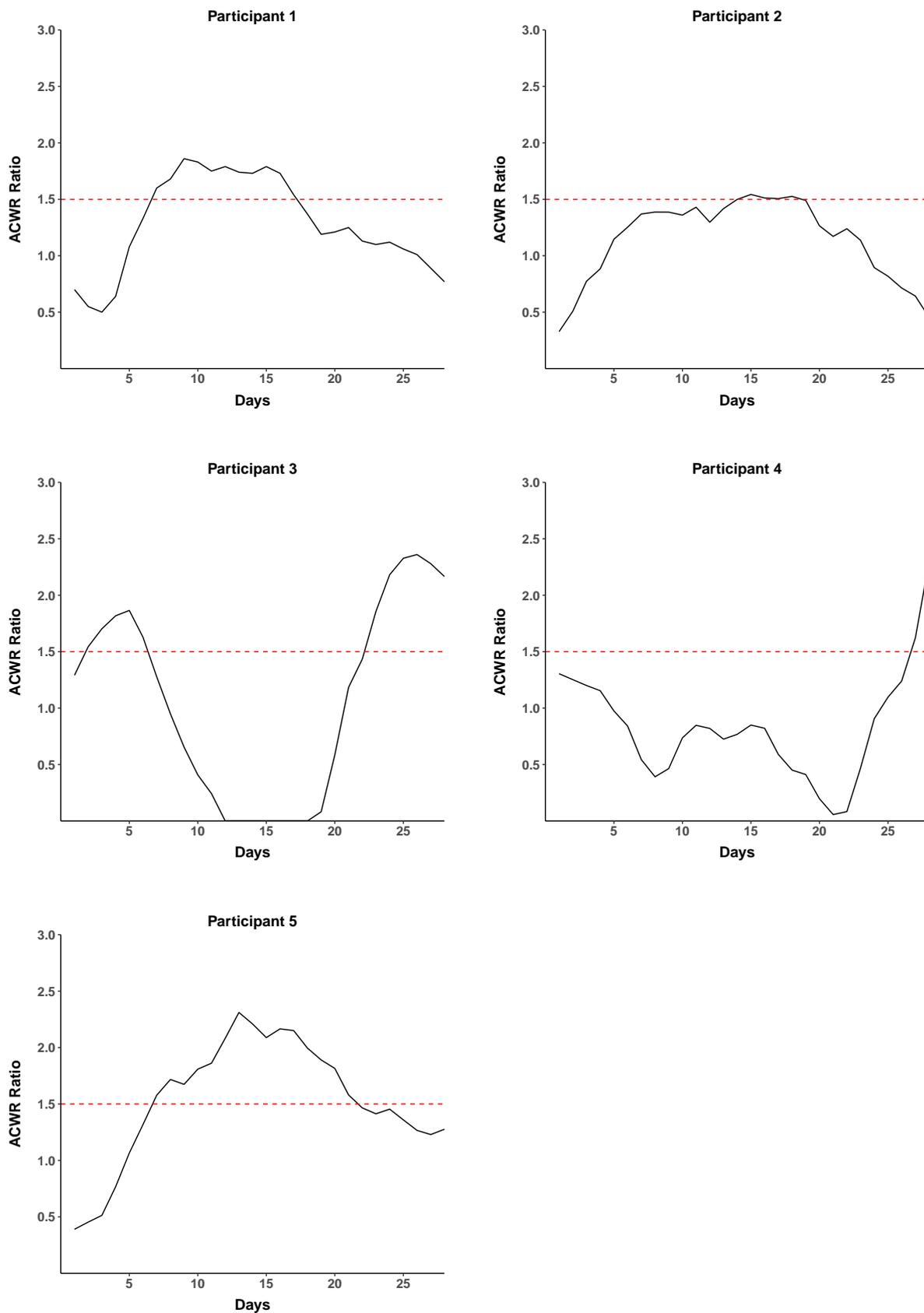


FIGURE 7.3. Acute Chronic Workload Ratios (ACWR) for the 28 days prior to onset of LBP for each participant

7.4.3. BIOMECHANICS

Serving biomechanics were generally comparable between the five players with a few exceptions. Player 4 and 5 tended to have lower peak magnitudes for trunk axial rotation, lumbar left lateral flexion and pelvis anterior tilt though higher pelvis rotation (left). Similarly, players 2, 4 and 5 presented with lower lumbar left lateral force during the drive phase. Temporal kinematics were broadly similar although player 2 commenced pelvis rotation (left) much earlier than the other players, and, players 3 and 4 had very idiosyncratic timing for peak lumbar posterior force for both flat and kick serves

TABLE 7.2. Serving biomechanics for both flat and kick serves.

Athlete	1		2		3		4		5	
Serve Type	Flat	Kick								
Kinematics (°)										
Lumbar Right Axial Rotation	-1.0 ± 0.5	-1.7 ± 0.3	-4.5 ± 0.1	-4.5 ± 0.2	-1.0 ± 0.7	-0.8 ± 0.6	2.0 ± 0.3	2.1 ± 0.2	0.5 ± 0.2	0.2 ± 0.1
Trunk Axial Rotation	-38.1 ± 2.7	-31.8 ± 0.2	-31.0 ± 1.0	-32.1 ± 1.1	-35.2 ± 0.5	-35.7 ± 0.7	-12.9 ± 0.9	-9.2 ± 3.2	-10.7 ± 1.8	-11.5 ± 1.4
Pelvis Obliquity Right Down	14.6 ± 1.4	17.8 ± 0.2	6.9 ± 0.8	7.2 ± 0.1	8.5 ± 1.1	8.2 ± 0.6	7.5 ± 0.5	9.8 ± 1.8	7.5 ± 1.6	7.3 ± 1.7
Lumbar Left Lateral Flexion	-11.0 ± 0.7	-11.0 ± 0.3	-21.0 ± 0.3	-22.5 ± 0.5	-15.1 ± 0.2	-16.4 ± 0.5	-9.0 ± 0.1	-7.6 ± 0.3	-19.7 ± 0.4	-20.4 ± 0.8
Pelvis Anterior Tilt	37.8 ± 1.7	36.7 ± 1.3	27.5 ± 0.9	22.1 ± 1.0	34.7 ± 0.6	29.5 ± 2.2	12.8 ± 1.5	2.9 ± 0.4	11.1 ± 1.5	7.9 ± 3.6
Pelvis Rotation (left)	8.6 ± 0.5	-9.1 ± 7.0	6.5 ± 0.7	-12.3 ± 2.4	-1.0 ± 5.1	-11.7 ± 3.7	-20.6 ± 2.1	-54.8 ± 3.0	-26.8 ± 2.8	-39.5 ± 3.2
Kinetics (N/kg)										
Lumbar Left Lateral Force	-24.7 ± 6.6	-23.6 ± 7.2	-9.7 ± 1.5	-7.1 ± 1.9	-29.4 ± 5.0	-30.9 ± 7.6	-8.2 ± 0.6	-14.9 ± 1.7	-13.9 ± 4.3	-16.1 ± 6.8
Temporal Kinematics (%)										
Racquet High Point	62.3 ± 1.5	60.3 ± 2.1	61.7 ± 0.6	57.3 ± 1.2	63.0 ± 0.0	57.3 ± 1.5	53.3 ± 0.6	49.3 ± 1.5	71.3 ± 0.6	66.0 ± 0.0
Peak Right Knee Flexion	53.3 ± 0.6	57.0 ± 1.0	42.7 ± 6.4	41.7 ± 3.8	60.0 ± 2.0	60.3 ± 2.1	49.7 ± 1.5	47.3 ± 3.1	60.0 ± 1.7	60.0 ± 1.0
Peak Lumbar Lateral Flexion (right)	27.0 ± 3.0	30.7 ± 2.1	21.0 ± 1.7	13.0 ± 8.7	33.0 ± 1.0	38.0 ± 3.6	35.7 ± 3.8	45.3 ± 16.3	13.3 ± 12.2	17.7 ± 0.6
Pelvis rotation (left)	33.7 ± 2.9	58.0 ± 4.0	12.7 ± 1.5	8.7 ± 1.2	64.0 ± 2.6	69.3 ± 1.5	44.3 ± 2.5	60.3 ± 4.0	69.0 ± 2.6	74.0 ± 0.0
Temporal Kinetics (%)										
Peak Lumbar Posterior Force	91.3 ± 9.9	84.7 ± 14.4	89.7 ± 2.1	97.0 ± 1.0	64.3 ± 24.3	55.7 ± 38.1	66.0 ± 36.7	73.7 ± 0.6	92.3 ± 6.7	77.3 ± 1.2

7.5. DISCUSSION

This study describes the technical, workload and MRI characteristics of five elite adolescent tennis players who experienced LBP during a 12-month period. All players underwent a baseline MRI scan to commence the study and, except for one player (#2, Table 1), completed a follow-up MRI scan 12 months later. The three-dimensional motion of the players' serves and self-reported daily workloads were recorded in an attempt to better understand the dimensionality of LBP.

7.5.1. MAGNETIC RESONANCE IMAGING

Previous work has related bone stress at the pars interarticularis to LBP, however the findings of this study do not necessarily support this. At baseline, only 2/5 participants presented with BMO and went on to experience LBP in the following months. However, in chapter 3, six participants were diagnosed with BMO at baseline (including the two participants mentioned above) though the remaining four participants diagnosed with BMO did not go on to experience LBP following their baseline scan. This is interesting as it indicates that BMO is potentially not a major risk factor for LBP in elite junior tennis players like it is in other sports (Sims et al., 2019). A possible reason for this could be the greater variation and diversity in loading across the vertebra in tennis (serving followed by groundstrokes) as opposed to fast bowlers who perform the same movement repeatedly and thus load the vertebra in the same place each time.

It is particularly difficult to determine the relationship between the MRI findings and the onset of LBP due to the scans being 12 months apart. Participants 1 and 4 both

experienced LBP midway through the study and therefore it is possible that BMO had arisen prior to the onset of pain though disappeared prior to the follow-up scan (Voormolen et al., 2006). A study by Kountouris et al. (2018) scanned junior elite fast bowlers every four weeks throughout a cricket season and found that BMO precedes bone stress injuries. Kountouris et al. (2018) found that two thirds (67%) of fast bowlers who developed a bone stress injury were diagnosed with BMO at baseline and thus highlighted a clear link between the presence of BMO and bone stress injuries. However, he also found a high prevalence of BMO in fast bowlers that remained injury-free. The findings in chapter 6 challenge the common belief the BMO leads to LBP in tennis players. Chapter 6 revealed that a total of 9/25 elite junior tennis athletes were diagnosed with BMO at either baseline or follow-up though only three of these athletes were symptomatic between the two scans. With this in mind, it is possible that the participants who presented with BMO at follow-up only, might still be yet to develop LBP. Based on what we found in this study (those with BMO developed low back pain in up to 4 months following their MRI scan, Table 7.1) and what has been presented by Sims et al. (2019), it is possible that the players who developed BMO at follow-up might experience LBP in the next 12-16 weeks. Thus, the relationship between BMO and LBP in tennis remains unclear.

It is also possible that other lumbar spine morphology such as disc pathology and/or degenerative changes might also be associated with some LBP pathology. However, due to the high prevalence of these abnormalities in junior tennis players (as seen in chapter 3), it is difficult to draw any firm conclusions. In view of these findings, the value in conducting annual MR scans remains unclear and as it is difficult to determine the relevance of the MRI findings to LBP episodes several months later. A possible solution

to this would be conducting more frequent MRI scans, although this would require significant financial investment.

7.5.2. WORKLOAD

All players in this study had a significant “spike” in workload approximately 14 days prior to experiencing LBP. Recent work by Myers et al. (2019) has shown that tennis players’ workload the week prior to pain is instrumental in assessing injury risk. They found that an ACWR of >1.5 in the current week increased injury risk by 2-4 times for the following week. This threshold is slightly lower than that reported by Hulin et al. (2016) who suggested that an ACWR of >2.0 inferred a 17% injury risk for the current week and 12% injury risk for the following week. In the current study, two players (#3 and #4) experienced ACWRs of >1.5 in the 7 days prior to LBP, three players experienced ACWRs of >1.5 in the 14 days prior to LBP and all players experienced ACWRs of >1.5 in the 28 days prior to LBP. While these results support the work of Myers et al. (2019), the data for player 3 and 4 may be misleading as their failure to report external load for an average of 10 days resulted in missing data prior to the onset of LBP. Consequently, the observed spike in ACWR for these players may be inflated and serves as a reminder that although load monitoring holds some value for tennis players, it is less so if players are not consistent in their reporting. Other methods such as imputing workload data may be useful for retrospective analysis if only a small portion of workload data is missing, however this might become a challenge for coaches and sports scientists if they’re monitoring workload daily. Overall however, the remaining 3 players all experienced workload spikes of 1.5 or higher within the 2 weeks prior to LBP. Therefore it appears

possible that the increase in ACWR might have influenced the onset of LBP in some players as suggested by Myers et al. (2019).

Interestingly, players 2-5 experienced ACWRs of >2.0 once prior to experiencing LBP while player 1 underwent three ACWRs of >2.0 all prior to the month in which he sustained LBP. This highlights the yoyo-like characteristics of tennis practice and workloads within a 28-day period. This also emphasizes the likelihood of practice thresholds being very individual and the concept of “how much is too much?”, as entertained by the recent International Olympic Committee consensus statement on load and injury in sport (Schwellnus et al., 2016; Soligard et al., 2016). Nevertheless, ACWR and other load management techniques hold promise for future research and in the ongoing care of athletes.

7.5.3. BIOMECHANICS

Players with LBP had comparable mechanics with only a few minor differences for peak mechanics between serve types as well as a few differences in temporal sequences between players (Table 2). For example, the temporal sequencing of peak lumbar posterior force was noticeably different for participant 3 (occurred at 65% of the way into the serve) compared to the other participants (~90%, Table 2). This inter-subject variation in serve mechanics and lack of an obvious trend tying all of the service actions together highlights the individuality of serve performance as well as the need to consider other factors like serve repetition to assist our understanding of the interaction between technique and LBP.

Our results also differed to previous research. For example, in this study, three participants had peak right trunk rotation values between 31° – 38.1° (irrespective of serve type), which is consistent with asymptomatic players in a similar study by Campbell et al. (2014) but considerably more than players who had a history of pain (16.8° – 19.7°). It is also important to note that the other two players who developed LBP actually registered lower peak trunk rotation values than either group in the Campbell et al. (2014) study. These findings combined with higher magnitudes of peak pelvis obliquity in all players, contrast previous suggestions that lumbar mobility is reduced in junior tennis players with LBP (Campbell et al. (2014)). In view of this, it is possible that some players (such as player #1 who has greater pelvis obliquity and anterior tilt) are either hypermobile, or lack lumbo-pelvic movement control which could in turn contribute to injury onset (Roussel et al., 2009).

Based on our data here, it seems as though lumbar spine imaging, serve biomechanics and workload monitoring on their own provide insufficient information to forecast, with the confidence needed in elite sport, who is likely to suffer LBP and when. Taken together though, these inputs might assist in flagging some players who may be at risk of injury. For example, if a player has BMO, it makes sense to monitor his/her external load ACWR for spikes that might aggravate the BMO. Better yet, would be to take a more proactive management approach that involved prescribing the workload to which the athlete would be exposed.

7.6. LIMITATIONS

With so few tennis players experiencing LBP over the 12 months period, generalizable outcomes or recommendations are difficult. Secondly, while the participants were generally compliant with reporting daily workload, there were several days missing and this may have affected the computation of ACWR. This study did not consider or control for extra-curricular activities and the effect they might have on LBP. Lastly, the timing of the biomechanical analysis of the serve was pre-determined and may have limited the extent to which the action was representative of that which the player used in the lead up to his/her pain episode.

7.7. CONCLUSION

This study explored the link between the onset of LBP and lumbar spine morphology, workload and serving biomechanics. The relationship between the presence of pars abnormalities, specifically BMO, and LBP remains unclear and warrants further investigation. There appears some merit in using EWMA workload measurements to identify players at risk for LBP as all players experienced spikes in load during the 14 days prior to their LBP episode. Lastly, it was difficult to find a relationship between specific serving kinematics and kinetics to the onset of LBP and it would seem necessary to develop an appreciation of serve volume (the repetition of these kinematics/kinetics) in the context of LBP in the future.

CHAPTER 8
GENERAL DISCUSSION

This dissertation aimed to explore the risk factors for LBP in elite adolescent tennis players. Specifically, this thesis investigated underlying lumbar spine abnormalities, spine biomechanics and player workload, and their respective links to LBP. This final chapter will summarise the findings of each experiment and discuss their implications in the context of LBP in tennis. Additionally, the methodology of these studies will be critically evaluated and suggestions for future research will be provided.

8.1. MAIN FINDINGS

Chapters 3 and 6 examined the underlying lumbar spine morphology in elite adolescent tennis players. Chapter 3 posed two questions: 1) “what lumbar spine abnormalities exist in elite asymptomatic junior tennis players?” and 2) “is facet joint orientation linked with the presence of pars abnormalities?”. This chapter found that 68% (17/25) of players had at least one abnormality at baseline, with facet joint degeneration, disc degeneration and pars abnormalities being the most prevalent. Further, six players presented with BMO, with some diagnoses being classified as clinically significant and/or representative of the later stages of symptomatic bone stress injury (Sims et al., 2019). Since all players were asymptomatic, this study highlights the risk in a singular reliance on MRI for diagnosing injuries. In response to the second question, we found that facet joint orientation showed a relationship with the presentation of pars abnormalities at baseline.

More broadly, Chapter 6 posed the question; “what lumbar spine abnormalities are present/not present 12 months following a baseline scan?”. Consistent with chapter 3, this study observed a large majority (78%, 14/18) of players to present with an asymptomatic lumbar spine abnormality. This thesis confirms that lumbar spine abnormalities were

pervasive in junior tennis players, both male and female, with most abnormalities remaining asymptomatic. Only 5/25 participants experienced LBP between the two MRI scans, further emphasizing that the presence of lumbar spine abnormalities are not always linked to LBP. Clinicians have speculated that pars abnormalities are the biggest contributor to LBP in tennis players, though during the course of this study, only two participants who had pars abnormalities at baseline, went on to experience LBP and the remaining seven who had BMO at baseline remained pain-free. This suggests that the presence of pars abnormalities (specifically BMO) is a common response to low back stress yet not necessarily a precursor to pain or injury. Worth noting though is that the sample size for this study was small, therefore further research should explore the behavior of BMO longitudinally in junior tennis players with a larger sample size. Lastly, consistent with chapter 3, the link between facet joint orientation and pars abnormalities was again explored in this chapter, although this time we did not find a strong association between the two variables. Therefore, the link between facet joint orientation and pars abnormalities remains unclear in elite junior tennis players.

Chapters 4 and 5 explored the relationship between serve mechanics (kinematics and kinetics) and the presence of pars abnormalities. Following on from previous research investigating serve mechanics and players with LBP (Campbell et al., 2014; Campbell et al., 2013) and in the knowledge that pars abnormalities can lead to significant time away from competition, these chapters posed questions such as “are pars abnormalities linked to serving kinematics?” and “are pars abnormalities linked to serving kinetics?”. Previous research had established links between serve mechanics and a history of LBP among athletes however the extent to these mechanics were adaptive or maladaptive remained

unknown (Campbell et al., 2014; Campbell et al., 2013). Therefore, chapters 4 and 5 aimed to identify whether serve mechanics were linked with pars abnormalities (a known risk factor for LBP) to prevent LBP from occurring.

Chapter 4 found that the magnitude of peak lumbar spine kinematics and ball toss kinematics were not linked to pars abnormalities, yet the timing of peak lumbar kinematics were. Accordingly, past research efforts linking LBP to peak lumbar kinematics may have examined kinematics/movements that were an adaptation to pain. Similarly, Chapter 5 showed that the timing but not magnitude of peak lumbar spine kinetics were associated with the presence of pars abnormalities. The results of these two chapters collectively indicate that the way in which players approach RHP during the serve is linked to the presence of pars abnormalities. Chapter 4 and 5 found that the timing of the lower limbs and trunk movement were found to be key differentiators between those with and without pars interarticularis abnormalities. Early initiation of leg drive was suspected to have been the cause for earlier pelvis rotation in those with pars abnormalities and is something that coaches can easily observe during daily practice without the intervention of technology. Collectively, these findings highlight the importance of previously overlooked aspects of the service motion (the timing and not just magnitude of key serve events) in the context of better understanding the link between technique and injury.

Chapter 7 used a case series approach to examine the lumbar spine imaging, serving biomechanics and external load among players who experienced LBP over a 12-month time window. Given the low sample size (only five players developed LBP), the intent

was to describe commonalities in lumbar morphology, technique and load in the lead up to the onset of pain so as to better inform current practice. The MRI findings in the five players with LBP differed considerably. Two players had BMO at baseline and one more player developed BMO during the study so that it was present at the 1- month follow-up scan. However, the remaining two players did not have BMO at any point during the study with one of these players having abnormality-free results at both scans. On account of these mixed imaging results among these five players whom experienced LBP, clinicians are ill-advised to solely focus on the presentation of BMO when evaluating the at-risk nature of tennis players' lumbar spines.

From the standpoint of workload, all players experienced spikes in ACWRs of ≥ 1.5 or higher in the two weeks leading up to the onset of LBP, which has previously been flagged as a risk for injury in the tennis literature (Myers et al., 2019). However, the players in this study experienced ACWR spikes of this magnitude multiple other times that didn't result in LBP so practitioners need to guard against oversimplifying the nature of any load-injury relationship. Further research needs to focus on monitoring load in a large number of junior tennis players and ensure the reporting compliance of participants.

Lastly, the serving biomechanics of players with LBP were observed to be highly variable which reaffirms that there is not a single biomechanical characteristic – even in the kick serve - that can be inextricably linked to pain.

8.2. THEORETICAL IMPLICATIONS

Two distinct theoretical implications were evident in this thesis: 1) asymptomatic lumbar abnormalities are pervasive in junior tennis athletes and seldom result in LBP, and 2) the presence of BMO is not as strongly associated with LBP as previously thought. Chapters 3 and 6 concluded that lumbar spine abnormalities are frequent among elite junior tennis players yet only five players (20%) experienced LBP. Consequently, it appears that annual MRI scans alone provide limited support in prognosticating who will experience LBP as has been found in older populations (Borenstein et al., 2001).

Chapter 6 explored the relationship between the presence of BMO and the onset of LBP. It was found that the relationship was more tenuous than previously reported. For example, players with no BMO, low-grade BMO and high-grade BMO all experienced LBP. Additionally, of the nine players who were diagnosed with BMO (either at baseline or follow-up), only three experienced LBP. These findings highlight the risk of “false positives” and confirm that lumbar morphology can change dynamically in elite adolescent tennis players.

8.3. METHODOLOGICAL IMPLICATIONS

The purpose of this section is to analyse the methodology of each chapter and its efficacy for answering each hypothesis.

Chapter 3 employed the use of MRI and five radiologists to determine the presence of pars abnormalities. The use of MRI proved valuable in determining the prevalence of abnormalities due to its high sensitivity. As highlighted in chapters 3 and 6 though, the use of MRI scanning for the purposes of diagnosing injury needs to be done with caution.

Due to the high sensitivity of MRI, false-positives (MRI findings that are not related to the patient's pain) are common, thus clinical assessments prior to scanning are important. Further, all five radiologists underwent inter-rater reliability tests for grading abnormalities prior to chapter 3 and 6. Whilst the inter-rater reliability was excellent, intra-observer agreement testing would also have been useful in describing the reliability of each radiologist.

Chapter 4 and 5 employed the use of a 12-camera, three-dimensional motion-capture system (Vicon). The Vicon system was a valid method for answering the hypotheses from chapters 4 and 5, though isn't a practical tool for use in the daily environment. This thesis was able to show that the timing of key serve events were linked to the presence of pars abnormalities, though ongoing monitoring of as much would likely require the use of equipment other than Vicon.

As in chapter 3, chapter 6 used MRI to surveil the presence of abnormalities 12 months following the baseline scans. This chapter also explored the abnormalities present (particularly BMO) in participants with LBP. This approach was useful in understanding the behavior of lumbar spine abnormalities, though more regular scanning as has been conducted in other research would have yielded potentially greater insight. For example, conducting MRI scans every 4 weeks would assist with understanding the time-course of change in BMO, and in the context of the current study, may have unearthed different links between BMO and LBP. Alternatively, scanning players at the time of LBP in addition to annual or frequent MRI scans might assist in linking lumbar abnormalities to LBP.

Chapter 7 was a case series study that explored the lumbar morphology, serving biomechanics and workload of players who developed LBP between their first and second MRI. MRI data was collected from the baseline and follow up scans for those who experienced LBP and any presence of pars interarticularis abnormalities and/or BMO was noted. Serving biomechanics collected for chapter 4 and 5 were used for this study to observe similarities in serving mechanics between participants who experienced LBP. Lastly, all players entered in RPE and session duration daily into an athlete management system app over the 12-month course of this project, whereby total daily workloads were calculated. Whilst the data used in this study was useful, insight into the relationship between lumbar spine morphology, serving biomechanics and player workload is limited due to having a small sample size.

8.4. PRACTICAL IMPLICATIONS

The findings of this thesis suggest that the causes of LBP are multi-factorial. This thesis found that 19/25 players were diagnosed with a lumbar abnormality (many participants had multiple), yet only five went on to experience LBP. This highlights the importance of clinical assessment prior to involving MRI. Similarly, given that the presence of BMO was only linked to LBP in 3/12 cases in our sample, MRI scanning for BMO (as is often performed in tennis currently) should not be done at the expense of experienced clinical judgement.

A second major practical implication of this thesis is the observed link between temporal serving mechanics and different spinal abnormalities, especially of the pars. This line of

research has not been pursued previously, with biomechanists preferring to investigate links to the magnitude of peak kinetics and kinematics. Knowing that the timing of lower limb and pelvis movement are linked to pars interarticularis abnormalities, coaches and scientists can observe and correct the timing of leg drive (tripe extension of the ankle, knee and hip) and pelvis rotation (axial rotation) leading into RHP (trophy position). Consequently, these revelations about the timing of the serve might allow coaches to consider analytical approaches to identify service actions that might put athletes at risk of lumbar abnormalities which may in turn become symptomatic.

8.5. STRENGTHS OF THIS THESIS

This thesis had a number of key strengths, including the following novel contributions: 1) it being the being the first research to prospectively investigate lumbar spine abnormalities in junior tennis players over 12 months, 2) its examination of the link between the presence of BMO and the onset of LBP in tennis players, and 3) its identification of links between pars abnormalities and serving mechanics. This thesis was also strengthened by the reliable and novel use of a peer-reviewed MRI grading system that allowed for lumbar abnormalities to be examined like never before in junior tennis athletes.

8.6. FUTURE DIRECTIONS

The findings from this thesis propose a number of interesting ideas for future research. Firstly, this thesis demonstrated how BMO is a prevalent abnormality in junior tennis players. Whilst BMO did not appear linked to LBP (chapter 6), due to its dynamic nature, it is possible that BMO did present and resolve within the 12-month time-frame in some

players. Future research may benefit from conducting more regular MRI scans to better understand the exact time course of change of BMO among a larger group of tennis players, as has been done in cricket. Accordingly this would allow researchers to more forensically examine the characteristics of BMO (severity, timing etc) that becomes symptomatic and potentially establish BMO ratios that link to LBP (Sims et al., 2019).

Another question that requires further examination is: are facet joint angles linked to pars abnormalities? The link between pars abnormalities and facet joint angles remains unclear, thus future research should explore this in a larger sample of tennis players.

In relation to serving mechanics, future work should continue to consider different analytical approaches. For example, additional context around the rate at which forces are developed (i.e. through impulse) and its effect on net loading of the lumbar spine should be contemplated. Lastly, the quantification of workload in elite tennis is extremely difficult given the coordinative complexity of the game's skills, especially in younger players. Whilst this thesis observed that players whom experienced LBP did so following a spike in workload, the relationship was tenuous and future research should employ sensor or computer vision technology to address issues of poor reporting compliance and improve the resolution of the load measures (Campbell et al., 2016).

8.7. CONCLUDING REMARKS

This thesis explored risk factors for LBP in elite adolescent tennis players using a multidisciplinary approach. Firstly, this thesis explored the lumbar abnormalities present in asymptomatic elite junior tennis players using MRI. At the same time of scanning, serving mechanics were examined using a 3-D motion capture system that quantified lumbar spine kinematics and kinetics. Then, the training workloads of all players and occurrences of LBP were monitored for 12 months, prior to a follow-up scan. As with the first scan, the follow-up MRI scanned for the presence of abnormalities. This thesis had two main conclusions: 1) the presence BMO was not strongly linked with LBP, and 2) the timing of the tennis serve appears linked to pars abnormalities.

The first finding is particularly important as it challenges the common belief in tennis that the presence of BMO will lead to LBP. Indeed, the findings suggest that the presence of BMO may be adaptive to training loads or technique changes and not necessarily a high risk for LBP. However, more research is needed to fully understand the extent to which BMO interacts with LBP. Our second finding is one that has potential inform the daily training environment. We found that the timing of the serve is linked to pars abnormalities and more specifically, the timing of RHP and the key events in the drive phase are linked to pars interarticularis abnormalities. This finding is important as it is modifiable and can be detected by coaches in the daily training environment.

In conclusion, this thesis was the first to prospectively analyse elite junior tennis players in order to better understand the risk factors for LBP. The findings of this thesis can be

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used by clinicians, coaches and sports scientists both enhance injury prevention methods and improve tennis serving techniques to alleviate risk to the adolescent lumbar spine.

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CHAPTER 10
APPENDICES

APPENDIX A – INFORMATION STATEMENT FOR PARENT/GUARDIAN

INFORMATION TO PARENTS OF PARTICIPANTS INVOLVED IN RESEARCH

Your child is invited to participate

Your child is invited to participate in a research project entitled “A multidisciplinary approach to understanding low back pain in elite adolescent tennis players”.

This project is being conducted by a student researcher Molly Connolly as part of her PhD study at Victoria University under the supervision of Damian Farrow from the Institute of Sport, Exercise and Active Living.

Project explanation

We want to investigate why low back pain occurs in elite adolescent tennis players and why the rate of low back pain is increasing in this population. Between 2008 and 2015, lumbar injuries in the Australian National Academy tennis players increased 10-fold. In 2015 alone, eight male adolescents suffered from lumbar injuries and most of these injuries (all but one), were either a stress fracture in the lumbar spine (Burton et al.) or a stress reaction. These injuries cost a total average of 165 days, or just over 5 months, out of play for these players. Our research team wants to analyse the possible risk factors that may predispose a player to low back pain. Specially we would like to analyse the structure of the lumbar spine, serving technique, workload and the general well-being of players as potential risk factors for low back pain. By doing so, we will be able to manage, though most importantly predict and prevent low back injuries in future.

You have been approached because you have an adolescent child who is Victorian and is ranked in the top 20 for their age group state-wide. Your child is an ideal participant for our project if they do not currently suffer from low back pain, have not suffered from a previous lumbar stress fracture/bone stress and are able to train at the required level appropriate for this study. We anticipate that at least 35 participants will be involved in this study.

What will your child be asked to do?

There will be three parts to your child's involvement in this study.

Part 1

Your child will be asked to attend a 1-hour technique analysis testing session in August (2018). The testing session will be located at the TA National Training Centre and will involve the use of a 3D motion analysis system. We will attach small reflective markers to specific points of the body using double sided tape and sports tape, as this is what the cameras track when moving. VICON (3D motion analysis system) does not use conventional filming though instead records the movement of the markers attached to your child's body. It appears as a black screen with a series of silver dots, meaning your child will be unidentifiable (see figure 1). The markers will be placed in non-invasive positions and the researcher attaching them will talk to the participant while attaching them, so the participant is aware of where they're being attached. There will be a female researcher present to attach markers to the female participants and a male researcher present to attach marker to the male participants. The females will be required to wear a crop top and shorts and the males will be required to remove their shirts for the testing session and wear shorts in order for the cameras

to detect markers on your child's back and hips. Your child will then be asked to perform a self-directed warm-up on the tennis court, followed by a series of serves. Your child will be asked to hit approximately 30 serves targeting a marked area around the "T" on the other side of the net. These serves will be recorded by the 3D cameras. Lastly, testing will take place on the indoor courts at the National Training Centre meaning the area will not be secluded. We will put up black sheets along the netting that divides the courts, however should your child feel uncomfortable about participating in the session, they are able to withdraw from the study. This will not impact their relationship with the researchers or Tennis Australia.

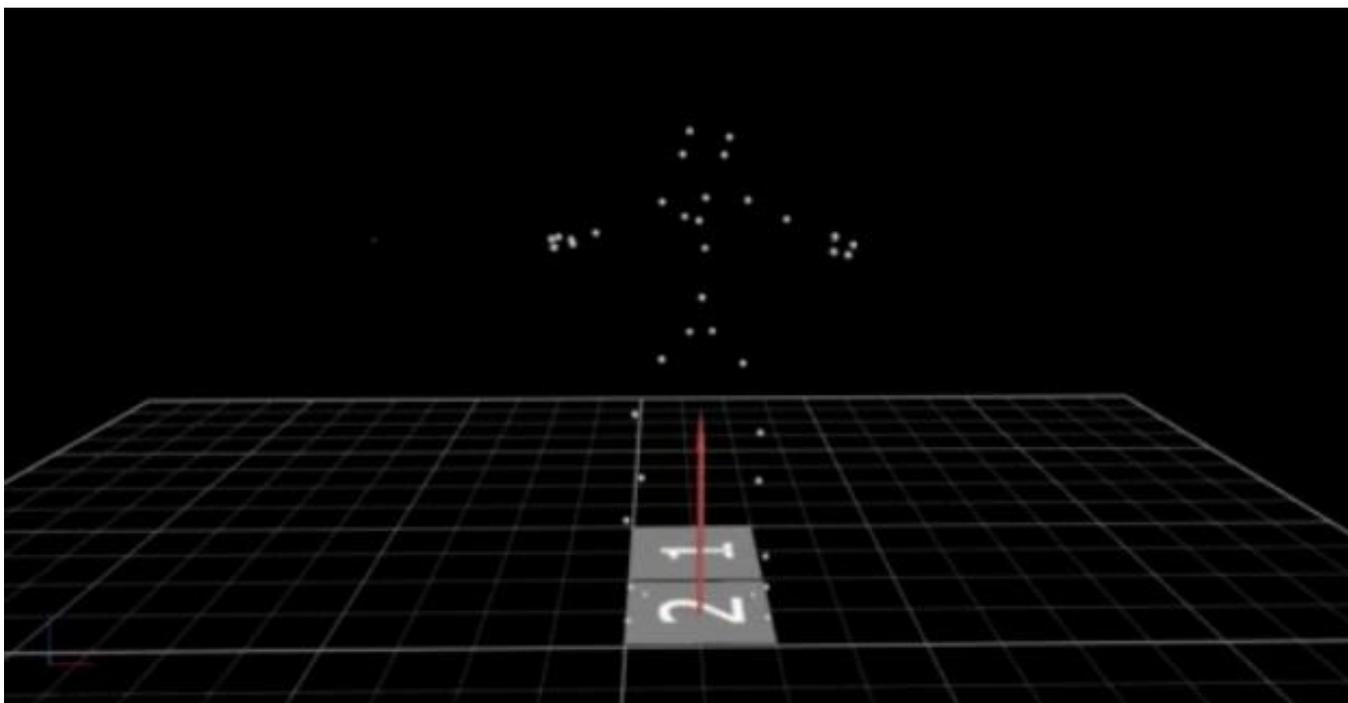


Figure 1: This is similar to what our screen will look like (the dots will be placed in slightly different places).

Part 2

Your child will be asked to undergo three Magnetic Resonance Imaging (MRI) scans across the period of one year. These scans will be conducted at Victoria House Imaging in Prahran and scan sessions will take approximately 20 minutes to complete. Additionally, the MRI scans will be at no cost. A potential risk your child might face when undergoing an MRI scan can include: claustrophobia. Should your child become claustrophobic when entering the MRI, they are able to decline partaking in the MRI scans. There are criteria that must be adhered to prior to undergoing a MRI scan (such as removing jewellery), however these criteria will be explained in detail to you and your child when arriving for your appointment by a qualified radiologist. Please know that MRI does not use ionising radiation like CT scans, and therefore does not pose health risks to younger children and adolescents.

Part 3

The final part to the study will involve your undergoing a one-time musculoskeletal screen at the National Tennis Centre, Melbourne. This will be conducted by our Physiotherapist and will take approximately 20 minutes. In addition to this, we will ask your child to download our Athlete Management System (AMS) app to record their workload daily for the period of one year. The student researcher (Molly Connolly) will explain in detail how to log workload using the app. Lastly the student researcher will send out a weekly email to ask whether any soreness has been experienced throughout the previous week and if so, where on the body the soreness was experienced and if it affected training. Molly will also on occasion ask for height updates throughout the year.

What will your child gain from participating?

We can promise no direct benefits for your child, however back pain is emerging in this population and we predict that the outcomes of this study will allow us to prevent and manage low back pain in our future athletes.

How will the information my child gives be used?

- Your child's results remain confidential and will not be shared with other participants. No material that could identify your child will be used in any written reports. Your child's results will be kept on the Tennis Australia private server which is password protected and the chief investigator's external hard drive (which will be stored in a locked cabinet) which only the investigators of this project will have access to this information. The information collected will also be stored on Victoria University's online secure platform (Research Drive) in which only members of the research team have access to. The information will be stored for a period of seven years, after which, all material will be securely destroyed. Should your child wish to gain access to this data, they can contact the chief researcher and it will be provided to them.
- We have permission from Tennis Australia to use data currently being entered into the Athlete Management System (AMS). Specifically, workload, soreness, height and wellness data, and routine MRI screening information will be used to retrospectively correlate findings with low back pain and/or abnormalities. This data will be deidentified when used in reports and any form of presentation/publishing.
- There may be a delay of several months between data collection, reports and publication of the results. At the completion of the project a summary of the results will be available to interested participants and/or their parents or guardians. Your child's individual results will also be available to them upon their request.
- We expect to publish scientific papers in appropriate peer-reviewed journals and conferences in future. We will only report group averages and therefore there will be no details in the publication that would allow others to identify your child.

Photos and Videos

- Photographic images and videos will be taken during testing sessions to enable us to collect the required information. These images may be used for seminar and/or conference presentations and journal publications to demonstrate the testing methodology that was conducted as part of the study. Not all of the images will be used for this purpose. If you and your child agrees to participate in this study, you are agreeing for your child to be photographed or videoed. Any images used in future publications will have identifying features blurred and will not allow identification of the participants of whom they're obtained from.

What are the potential risks of participating in this project?

The risks in this project are minimal because the project involves minimal intervention. The intervention of the technique analysis will require efforts from the participant that are no greater than what they currently perform in the daily training environment and therefore will not create a higher risk of injury. However, similar to the risk posed in their daily training environment, there is the chance of sustaining an injury. Should a participant become injured, they will be excluded from the technique analysis testing and treated by a first-aid trained member of the research team. The participants will have access to health professionals (physiotherapists, doctors, strength and conditioning experts etc.) who will be able to advise and help them manage their injury. There is also the risk of becoming claustrophobic (as outlined above) when undergoing the MRI scan. Again, if your child feels uncomfortable at any stage throughout this project they are able to withdraw from the project and it will not affect their relationship with Tennis Australia or the research team.

How will this project be conducted?

This project will involve the undergoing the MRI scans in August (2017), April (2018) and August (2018) and technique analysis in August (2018). Technique information will be related to the scan results to determine the possibility of technique influencing abnormalities at the lumbar spine. Other information such as workload, soreness and height will also be analysed to determine patterns in workload and their influence on the lumbar spine.

Who is conducting the study?

Victoria University and Tennis Australia

Dr. Damian Farrow
Sport Scientist at Victoria University
Victoria University – Institute of Sport, Exercise and Active Living
E: Damian.Farrow@vu.edu.au, Ph: (+61) 408 445 701

Dr Machar Reid
Sport Scientist at Tennis Australia
Victoria University – Institute of Sport, Exercise and Active Living
E: mreid@tennis.com.au, Ph: (+61) 401 077 441

Molly Connolly, BAppSci (Hons)
PhD Student at the Institute of Sport Exercise and Active Living
E: mconnolly@tennis.com.au, Ph: (+61) 408 377 346

Any queries about your participation in this project may be directed to the Chief Investigator listed above. If you have any queries or complaints about the way you have been treated, you may contact the Ethics Secretary, Victoria University Human Research Ethics Committee, Office for Research, Victoria University, PO Box 14428, Melbourne, VIC, 8001, email researchethics@vu.edu.au or phone (03) 9919 4781 or 4461.

APPENDIX B – INFORMATION STATEMENT FOR CHILD/PARTICIPANT



INFORMATION TO PARTICIPANTS INVOLVED IN RESEARCH

You are invited to participate

You are invited to participate in a research project entitled “A multidisciplinary approach to understanding low back pain in elite adolescent tennis players”.

This project is being conducted by a student researcher Molly Connolly as part of her PhD study at Victoria University under the supervision of Damian Farrow from Victoria University (ISEAL).

Project explanation

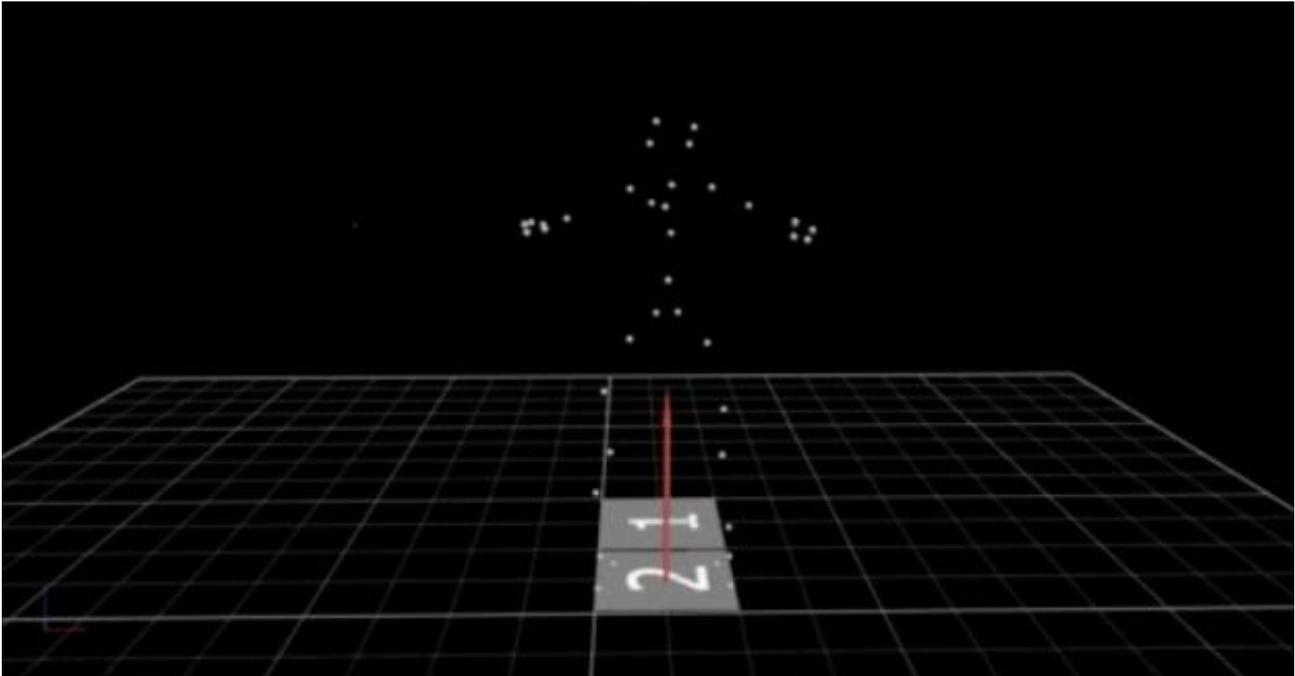
We want to look at why low back pain occurs in young tennis players and why the number of players with low back pain is increasing. Even at the elite level, players still suffer low back injuries. Low back injuries have increased in the last few years, especially at the high level. We would like to better understand these injuries and how we can prevent them. Specifically, we would like to look at the structure of your spine, your serving technique, how often and how hard you train, and how you feel each day, as possible reasons players may get low back pain. By doing so, we will know how and why players get low back injuries.

You have been approached because you are a Victorian player ranked in the top 20 for your age state-wide. You are an ideal participant for our project if you do not currently suffer from low back pain or have suffered from a previous fracture/stress on your spine. We anticipate that at least 35 participants will be involved in this study.

What will I be asked to do?

Part 1

You will be asked to attend a 1-hour tennis session located at Tennis Australia’s National Training Centre in August (2017). In these sessions, we will attach small markers (reflective dots) to your body using sticky tape and sports tape. These dots will be placed on you as this is what the cameras use to tell where you are when we film you. The cameras we use are not normal cameras, they will record the dots on your body. Our computer screen will look black with silver dots on it (see Picture 1), so people will not know who is on the screen. The researcher attaching the markers will talk to you while attaching them, so that you know where they’re being attached. There will be a male researcher to attach the markers to males and female researchers to attach markers to female participants to make sure you feel comfortable. Females will be required to wear a crop top and shorts and the males will need to remove their top and also wear shorts, so that the cameras can see the dots on your back and hips. You will then be asked to perform a warm-up on the tennis court however you like, followed by some serves. There will be a target area marked area around the “T” on the other side of the net, which we will want you to aim for when you are serving. You will be asked to perform up to 30 serves while being filmed by 3D cameras. Lastly, testing will be on the indoor courts next to other courts. We will put up black sheets on the net at the side of the court so those on the next court cannot see you during testing. Though if you do not feel comfortable participating next to other courts, you can withdraw from the study. This is fine and will not affect your relationship with the researchers or Tennis Australia.



Picture 1: This is what our screen will look like when filming you serve (though there will be more markers, and in different spots on the body)

Part 2

You will be asked to undergo three Magnetic Resonance Imaging (MRI) scans over one year. These scans will take around 20 minutes to complete. There is a chance you may feel claustrophobic (where you feel uncomfortable being in a small space). If you do become claustrophobic when entering the MRI, you are able to say no to undergoing the MRI scans. There are a few procedures before undergoing a MRI scan (such as taking off jewellery), however these rules will be explained to you in detail to you when you arrive for your appointment by a qualified radiologist.

Part 3

The final part to the study will involve you coming to the National Tennis Centre in Melbourne to undergo a one-time musculoskeletal screen at the National Tennis Centre, Melbourne. This will be conducted by our Physiotherapist and will take approximately 20 minutes. In addition to this, we will ask you to download our Athlete Management System (AMS) app to record your workload every day for one year (or until you've completed your final scan). The student researcher (Molly Connolly) will explain to you how to record workload using the app. Lastly the student researcher will send out a weekly email to ask whether you have been sore in the last week and if so, where on the body you were sore and if it affected your training. Occasionally, Molly will also ask for your height.

What will I gain from participating?

We cannot promise you any benefits however, by participating you are helping us collection information to help players like you with low back pain.

How will the information I give be used?

- Your personal information and testing information will be kept secret and will not be available for other participants or tennis staff to see. The only people who can look at your information will be the researchers and yourself. The researchers will store your information for 7 years and after that, the information will be destroyed.

- The information from your MRI scans and also the reports you put into AMS will be used for the project. Your results will be kept secret, so no-one other than the researchers and medical staff will know who was in the study or what the results were.
- It may take a little while to finish looking at your information and find results. However, if you would like to see your results, you can ask the Chief investigator and they will give them to you.
- We will use the information to educate others by publishing the results. The information will be in public speeches, books and articles on the internet. However, the information used will not use your name or identifiable information.
- We might take photos or videos during testing. Not all of these will be used in articles, though if they do, your face will be blurred so no-one recognises you. If you agree to be in this study, you are agreeing for us to use your pictures.

What are the potential risks of participating in this project?

The risks of this project are very small. The only risk is that you might accidentally hurt yourself during testing, which is the same level of risk as training or competition. We will not put you at higher risks of injury than normal day-to-day life. If you do get hurt, there will be a first-aid member, doctor and physiotherapist you can see to help you get better.

How will this project be conducted?

This project will use information from your scans in August (2017), April (2018) and August (2018). We will relate you scan results to your technique analysis results. Workload, soreness and height information that you give us will also be looked at during this time to see if there are patterns in workload and low back pain.

Who is conducting the study?

Victoria University and Tennis Australia

Dr. Damian Farrow
 Sport Scientist at Victoria University
 Victoria University – Institute of Sport, Exercise and Active Living
 E. Damian.Farrow@vu.edu.au, Ph: (+61) 408 445 701

Dr Machar Reid
 Sport Scientist at Tennis Australia
 Victoria University – Institute of Sport, Exercise and Active Living
 E: mreid@tennis.com.au, Ph: (+61) 401 077 441

Molly Connolly, BAppSci (Hons)
 PhD Student at the Institute of Sport Exercise and Active Living
 E: mconnolly@tennis.com.au, Ph: (+61) 408 377 346

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APPENDIX C – INFORMED CONSENT FORM FOR PARENT/GUARDIAN

CONSENT FORM FOR PARTICIPANTS INVOLVED IN RESEARCH

INFORMATION TO PARTICIPANTS:

We would like to invite your child to be a part of a study “A multidisciplinary approach to understanding low back pain in elite adolescent tennis players”. The project will aim to (a) enhance our understanding of the frequency of low back injuries in elite pain-free tennis players, (b) determine the relationship between serving technique and the structure of the lumbar spine and (c) understand how hard and how often your child trains and the effect this has on the structure of the lumbar spine. This project requires your child to undergo a 1 hour technique testing session, three MRI scans and record workload via an app for 12 months. The risk your child faces being involved in this study is the possibility of sustaining an injury during testing, although this risk is no greater than the risk of sustaining an injury in training or match play. There will be a medical team provided should your child sustain an injury. This project also involves your child participating in a program of magnetic resonance imaging scans – the details of which, including the risks, are presented in the information sheet. Finally, throughout the study, your child will be asked to record self-reported soreness, workload and height information on a secure online platform.

CERTIFICATION BY SUBJECT:

I, _____ (parent/guardians name)

of _____ (parent/guardians suburb)

certify that I am at least 18 years old and that I am voluntarily giving my consent for

my child _____ (participant/child's name)

to participate in the study titled: “A multidisciplinary approach to understanding low back pain in elite adolescent tennis players” being conducted at Victoria University and Tennis Australia by: Damian Farrow.

Yes No (please tick)

I certify that the objectives of the study, together with any risks and safeguards associated with the procedures listed hereunder to be carried out in the research, have been fully explained to me by Molly Connolly and that I freely consent to my child's participation involving the below mentioned procedures:

- Attending one 1-hour technique analysis sessions at Tennis Australia's National Training Centre
- MRI scans across a period of 1 year
- Self-reporting soreness, workload and height data on a secure online platform

I agree for my child to be filmed for research

Yes No (please tick)

I agree for this film to be used in presentations for teaching purposes and for scientific presentations

Yes No (please tick)

I certify that I have had the opportunity to have any questions answered and that I understand that I can withdraw from this study at any time and that this withdrawal will not jeopardise myself or my child in any way.

I have been informed that the information I provide will be kept confidential.

Signed: _____ Date: _____

Any queries about your participation in this project may be directed to the researcher

Dr. Damian Farrow
Sport Scientist at Victoria University
Victoria University – Institute of Sport, Exercise and Active Living
E. Damian.Farrow@vu.edu.au, Ph: (+61) 408 445 701

Dr Machar Reid
Sport Scientist at Tennis Australia
Victoria University – Institute of Sport, Exercise and Active Living
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APPENDIX D – INFORMED CONSENT FORM FOR CHILD/PARTICIPANT

CONSENT FORM FOR PARTICIPANTS INVOLVED IN RESEARCH

INFORMATION TO PARTICIPANTS:

We would like to invite you to be a part of a study “A multidisciplinary approach to understanding low back pain in elite

adolescent tennis players”. The project will aim to (a) help us understand why there are so many low back injuries in

elite tennis players, (b) see if there is a link between serving technique and the structure of your spine and (c) understand how hard and how often you train and how that affects the structure of your spine. This project requires you to undergo a 1 hour serving session. There is a risk you could get injured, although this risk is no bigger than in training or match play. There will be doctors and physiotherapists available for you if you get injured. This project also involves you participating in three magnetic resonance imaging scans – the details of these scans are written in the information sheet. Finally, throughout the study, you will be asked to record soreness, workload and height information for us.

CERTIFICATION BY SUBJECT:

I, _____ (participant/child) give assent to participate in the study “A multidisciplinary approach to understanding low back pain in elite adolescent tennis players” being conducted at Victoria University and Tennis Australia by: Damian Farrow.

Yes No (please tick)

I certify that the objectives of the study, together with any risks and safeguards associated with the procedures listed hereunder to be carried out in the research, have been fully explained to me by Molly Connolly and that I freely consent to participation involving the below mentioned procedures:

- Attending one 1-hour technique analysis sessions at Tennis Australia’s National Training Centre in 2017
- MRI scans over 1 year
- Reporting soreness, workload and height data

I agree to be filmed for research.

Yes No (please tick)

I agree for this film to be used in presentations for teaching purposes and for scientific presentations.

Yes No (please tick)

I certify that I have had the opportunity to have any questions answered and that I understand that I can withdraw from this study at any time and that this withdrawal will not jeopardise me in any way.

I have been informed that the information I provide will be kept confidential.

Signed: _____ Date: _____

Any queries about your participation in this project may be directed to the researcher

Dr. Damian Farrow
Sport Scientist at Victoria University
Victoria University – Institute of Sport, Exercise and Active Living
E. Damian.Farrow@vu.edu.au, Ph: (+61) 408 445 701

Dr Machar Reid
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APPENDIX E – PERMISSION TO USE DATA COLLECTED BY TENNIS

AUSTRALIA



Tennis Australia
Batman Avenue
Victoria Australia
Private Bag 6060
Richmond Victoria 3121
T +61 3 9914 4000
F +61 3 9650 2743
www.tennis.com.au

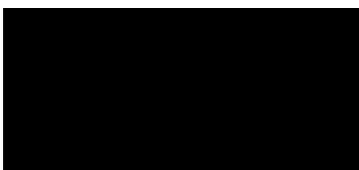
TO WHOM IT MAY CONCERN

12 November 2016

Dear Sir / Madam,

This letter is to confirm the proposed project entitled 'A multidisciplinary approach to understanding low back pain in elite adolescent tennis players.' is part of a Doctoral thesis being undertaken by Ms Molly Connolly under the supervision of Drs Damian Farrow and David Whiteside at the Institute of Sport, Exercise and Active Living (ISEAL), Victoria University in collaboration with Tennis Australia. Tennis Australia supports the proposed research and has agreed to provide the data required for its completion.

Sincerely



Machar Reid
Director, Research and Development
Tennis Australia

APPENDIX F – ETHICS APPROVAL LETTER

Tuesday, June 27, 2017 at 1:47:19 PM Australian Eastern Standard Time

Subject: Quest Ethics Notification - Application Process Finalised - Application Approved

Date: Monday, 30 January 2017 at 3:16:47 PM Australian Eastern Daylight Time

From: quest.noreply@vu.edu.au

To: Damian.Farrow@vu.edu.au

CC: Molly Connolly, MReid@Tennis.com.au

Dear DR DAMIAN FARROW,

Your ethics application has been formally reviewed and finalised.

- » Application ID: HRE16-290
- » Chief Investigator: DR DAMIAN FARROW
- » Other Investigators: DR MACHAR REID, MS Molly Connolly
- » Application Title: A multidisciplinary approach to understanding low back pain in elite adolescent tennis players.
- » Form Version: 13-07

The application has been accepted and deemed to meet the requirements of the National Health and Medical Research Council (NHMRC) 'National Statement on Ethical Conduct in Human Research (2007)' by the Victoria University Human Research Ethics Committee. Approval has been granted for two (2) years from the approval date; 30/01/2017.

Continued approval of this research project by the Victoria University Human Research Ethics Committee (VUHREC) is conditional upon the provision of a report within 12 months of the above approval date or upon the completion of the project (if earlier). A report proforma may be downloaded from the Office for Research website at: <http://research.vu.edu.au/hrec.php>.

Please note that the Human Research Ethics Committee must be informed of the following: any changes to the approved research protocol, project timelines, any serious events or adverse and/or unforeseen events that may affect continued ethical acceptability of the project. In these unlikely events, researchers must immediately cease all data collection until the Committee has approved the changes. Researchers are also reminded of the need to notify the approving HREC of changes to personnel in research projects via a request for a minor amendment. It should also be noted that it is the Chief Investigators' responsibility to ensure the research project is conducted in line with the recommendations outlined in the National Health and Medical Research Council (NHMRC) 'National Statement on Ethical Conduct in Human Research (2007).'

On behalf of the Committee, I wish you all the best for the conduct of the project.

Secretary, Human Research Ethics Committee

Phone: 9919 4781 or 9919 4461

Email: researchethics@vu.edu.au

**APPENDIX G – INFORMATION DOCUMENT DETAILING ABNORMALITY
ANALYSIS GUIDELINES FOR RADIOLOGISTS**

(Note: some of the descriptions in this chapter were directly copied from the relevant journal articles as this was handed to the radiologists for familiarization of the grading systems)

APPENDIX

PARS INTERARTICULARIS

Pars abnormality grading	Hollenberg (2002)
Grade 0	"Patients without signal abnormalities of the pars interarticularis"
Grade 1	"Patients with T2 signal abnormalities of the pars interarticularis with or without signal changes in the adjacent pedicle or articular process. These were cases with stress-related marrow oedema but no spondylolysis"
Grade 2	"Patients with T2 signal abnormalities and thinning, fragmentation, or irregularity of the pars interarticularis visible on T1- and/or T2-weighted images"
Grade 3	"A visible complete unilateral or bilateral spondylolysis (i.e., cortical disruption) with associated abnormal T2 signal"
Grade 4	"Was reserved for the cases of complete spondylolysis without abnormal T2 signal"

ABNORMALITY - (Hollenberg 2002)

GRADE 1

Grade 1 pars interarticularis stress injury. Sagittal short repetition time (TR)/echo time (TE) and fat-suppressed long TR/TE images of a 16-year-old football player with right side low back pain are shown. A: T1-weighted images demonstrate an intact right L5 pars interarticularis with mildly decreased signal about the pars and pedicle (arrow). B: T2-weighted images demonstrate



increased T2 signal (marrow edema) of the right L5 pars interarticularis, pedicle, and articular process (arrows). The finding of isolated marrow edema makes this a Grade 1 injury.

GRADE 2



Grade 2 pars interarticularis stress injury. Sagittal short repetition time (TR)/echo time (TE) and fat-suppressed long TR/TE images of a 17-year-old basketball player with lumbar pain are shown. A: T1-weighted images demonstrate an intact but irregular left L3 pars interarticularis with mildly decreased T1 signal and irregular cortex (arrow). B: T2-weighted images demonstrate increased T2 signal (marrow edema) of the left L3 pedicle and pars interarticularis (arrow). The finding of both marrow edema and cortical irregularity makes this a Grade 2 stress injury.

GRADE 3



Grade 3 pars interarticularis stress injury. Sagittal short repetition time (TR)/echo time (TE) and fat-suppressed long TR/TE images of a 16-year-old soccer player with low back tenderness that is

worse on the left are shown. A: T1-weighted images demonstrate a complete spondylolysis of the left L5 pars interarticularis (arrow). B: T2-weighted image demonstrates increased T2 signal (marrow edema) of the left L5 pars, pedicle, and articular process (arrows). A gap can be seen at the site of the spondylolysis. The finding of complete spondylolysis and marrow edema on the T2-weighted images makes this a Grade 3 stress injury.

GRADE 4



Grade 4 pars interarticularis stress injury. Sagittal short repetition time (TR)/echo time (TE) and fat-suppressed long TR/TE images of a 13-year-old athlete who sustained a fall and presented with left sacral pain and lumbar pain with extension are shown. A: T1-weighted images demonstrate a complete spondylolysis of the left L5 pars interarticularis (arrow). B: T2-weighted images demonstrate no abnormal T2 signal of the bone. The spondylolysis can be clearly seen (arrow). Mildly increased T2 signal in the spondylolysis defect may represent fluid or granulation tissue. The finding of complete spondylolysis without marrow edema on T2-weighted images makes this a Grade 4 stress injury.

DISC HERNIATION – (Mysliwiec 2010)

The size and location of disc herniation are measured at the level of maximal extrusion in reference to a single intra-facet line drawn transversely across the lumbar canal, to and from the medial edges of the right and left facet joint articulations (Fig. 1).

To further qualify location of the disc herniation, the lesion is described as A, B, or C to more exactly locate the position that is routinely, but less accurately, reported as central, lateral or far lateral. Three points are placed along the intra-facet line, dividing it into four equal quarters (Fig. 2).

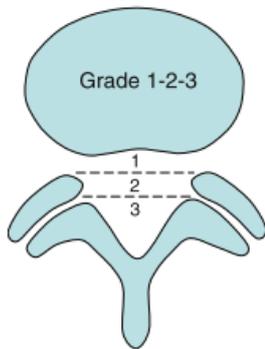


Fig. 1 Grading the disc herniation for size. Grade 1 lesions have little impact and grade 3 have the most impact on nerve compression

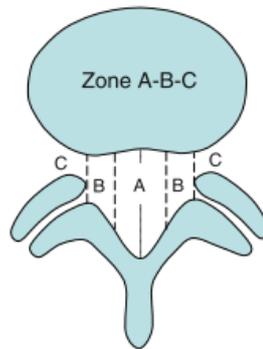
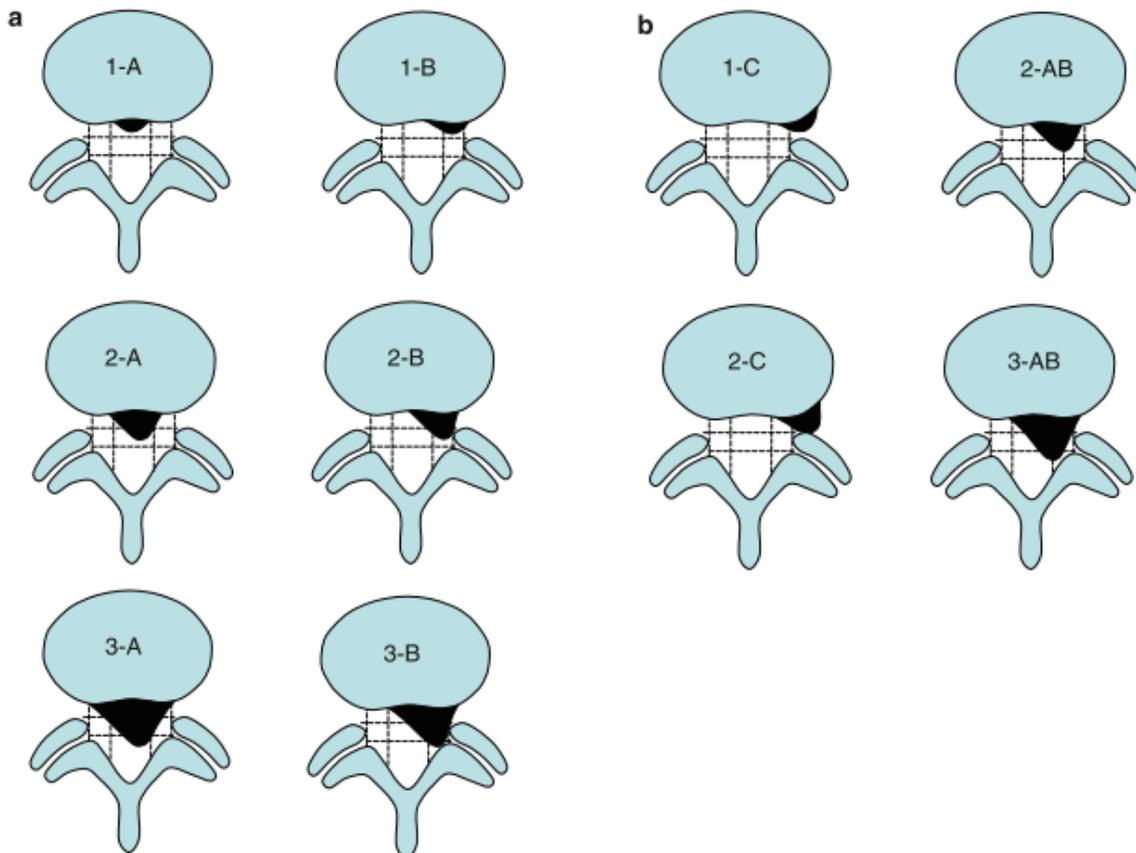


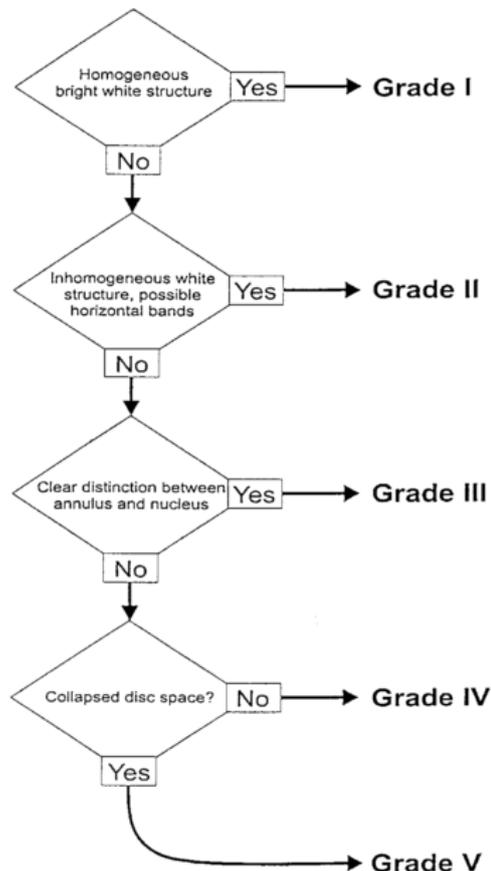
Fig. 2 Zoning the disc for location. Lesions have more impact in tighter zone-B and -C

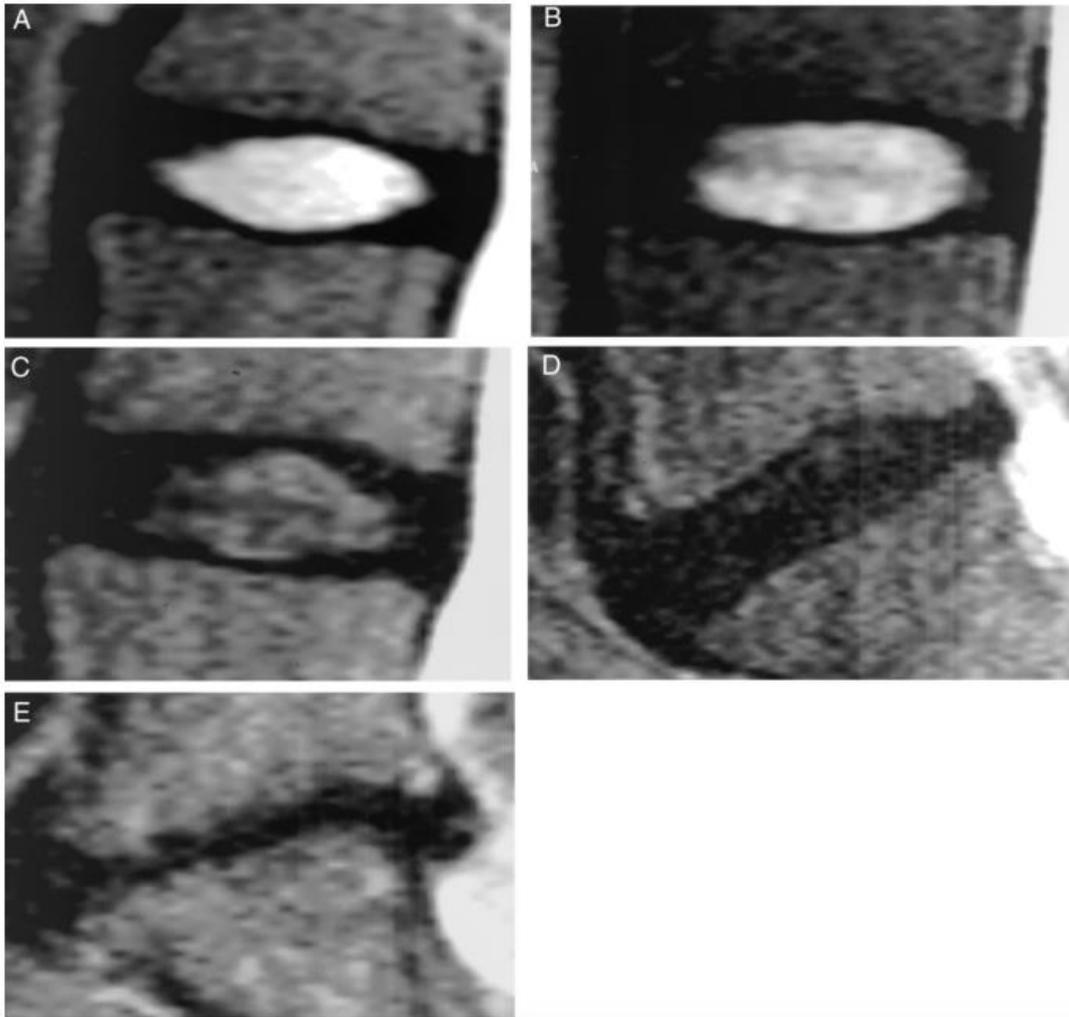


DISC DEGENERATION – (Pfirrmann 2002)

This grading system was performed using T2W sequence.

Grade	Structure	Distinction of Nucleus and Anulus	Signal Intensity	Height of intervertebral disc
I	Homogenous, bright white,	Clear	Hyperintense, isointense to cerebrospinal fluid	Normal
II	Inhomogeneous with or without horizontal bands	Clear	Hyperintense, isointense to cerebrospinal fluid	Normal
III	Inhomogenous, gray	Unclear	Intermediate	Normal to slightly decreased
IV	Inhomogeneous, gray to black	Lost	Intermediate to hypointense	Normal to moderately decreased
V	Inhomogenous, black	Lost	Hypointense	Collapsed disc space





A–E: Grading system for the assessment of lumbar disc degeneration.

Grade I - (A): The structure of the disc is homogeneous, with a bright hyperintense white signal intensity and a normal disc height.

Grade II - (B): The structure of the disc is inhomogeneous, with a hyperintense white signal. The distinction between nucleus and annulus is clear, and the disc height is normal, with or without horizontal gray bands.

Grade III - (C): The structure of the disc is inhomogeneous, with an intermediate gray signal intensity. The distinction between nucleus and annulus is unclear, and the disc height is normal or slightly decreased.

Grade IV - (D): The structure of the disc is inhomogeneous, with a hypointense dark gray signal intensity. The distinction between nucleus and annulus is lost, and the disc height is normal or moderately decreased.

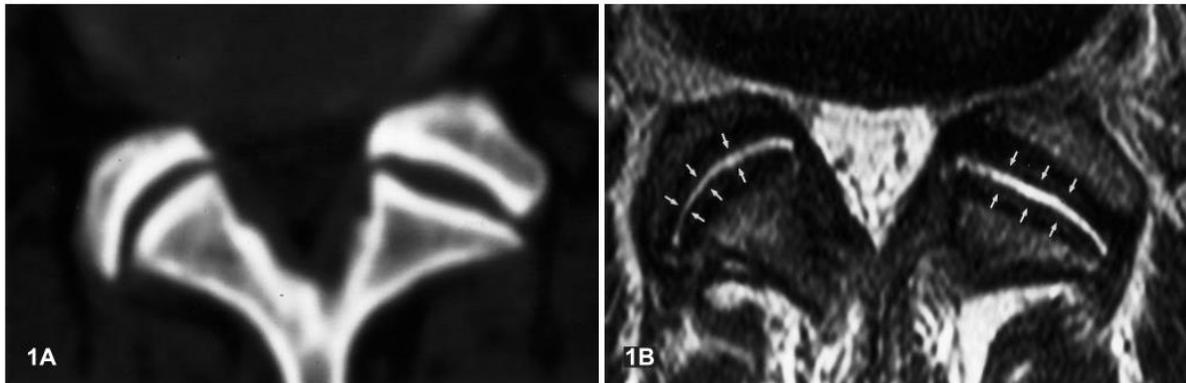
Grade V - (E): The structure of the disc is inhomogeneous, with a hypointense black signal intensity. The distinction between nucleus and annulus is lost, and the disc space is collapsed. Grading is performed on T2-weighted midsagittal (repetition time 5000 msec/echo time 130 msec) fast spin-echo images.

FACET JOINT ARTHROPATHY

DEGENERATION- (Weishaupt 1999)

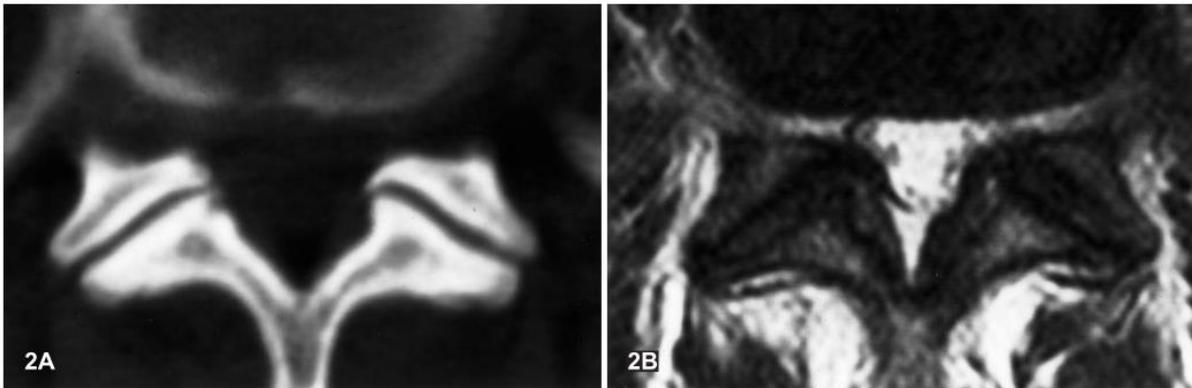
Grade 0	Normal facet joint space (2–4 mm width)
Grade 1	Narrowing of the facet joint space (<2 mm) and/or small osteophytes and/or mild hypertrophy of the articular process
Grade 2	Narrowing of the facet joint space and/or moderate osteophytes and/or moderate hypertrophy of the articular process and/or mild subarticular bone erosions
Grade 3	Narrowing of the facet joint space and/or large osteophytes and/or severe hypertrophy of the articular process and/or severe subarticular bone erosions and/or subchondral cysts

Grade 0



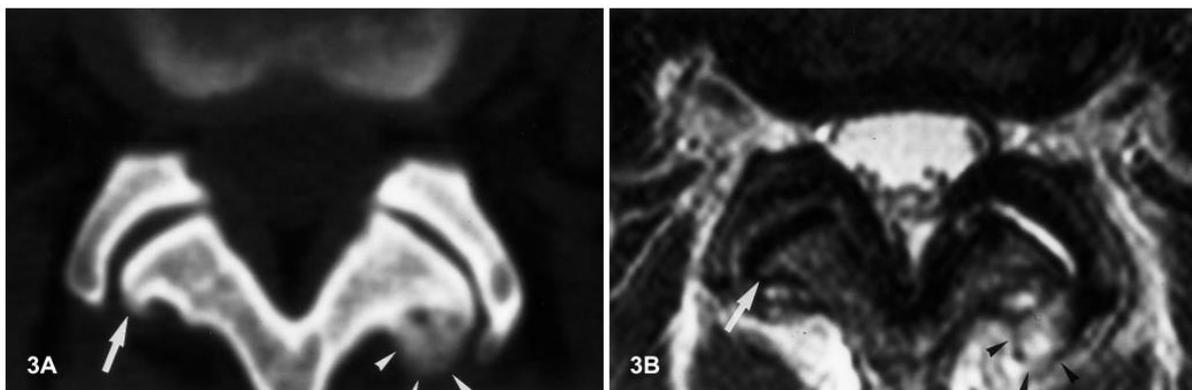
A 52-year-old man with normal L4/5 facet joints. **A:** CT demonstrates normal facets joints on both sides. **B:** Axial T2-weighted turbo spin-echo image [3800/96 (TR/TE)]. Cartilage (*arrows*) is visualized as layer of intermediate signal intensity between the articular cortices of the superior and inferior articular processes.

Grade 1



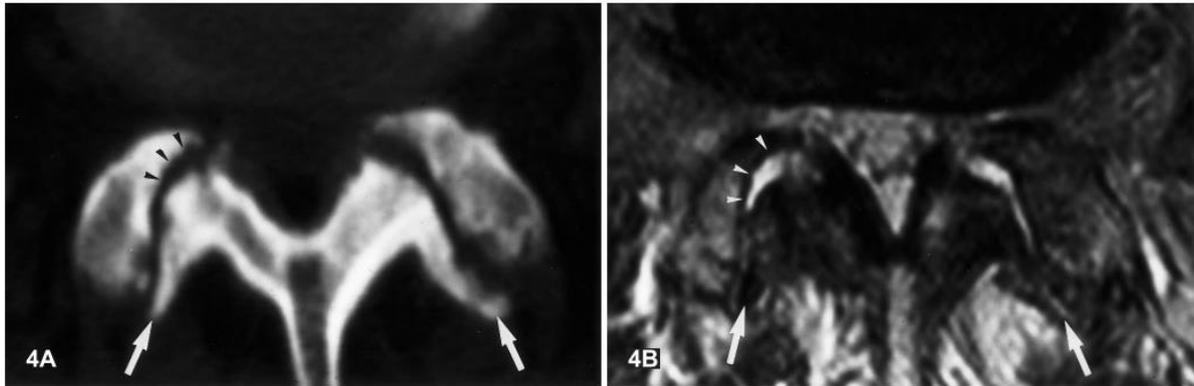
A 48-year-old man with grade 1 osteoarthritis of the L5/S1 facet joints. **A:** both articular joint spaces are narrowed on the CT. The facets are slightly hypertrophied on both sides. **B:** The corresponding axial T2-weighted MR image shows familiar findings.

Grade 2



A 46-year-old woman with grade 1 and grade 2 osteoarthritis of the L4/5 facet joints. **A:** CT scan demonstrates grade 1 osteoarthritis of the right facet joint with small osteophyte (*arrow*). On the left side grade 2 osteoarthritis with narrowing of the facet joint and moderate osteophytes is present (*arrowheads*). **B:** Axial T2-weighted MR image also reveals a small osteophyte (*arrowheads*) is visible on the left. Cartilage is replaced by fluid.

Grade 3



A 50-year-old woman with grade 3 osteoarthritis of the L5/S1 facet joints. **A:** Severe degeneration of the facet joint with joint space narrowing, hypertrophy of the articular processes, large osteophytes (*arrows*), and subarticular bone erosions (*arrowheads*) is demonstrated on the CT scan. **B:** Axial T2-weighted MR image also reveals severe degeneration of both facet joints with joint space narrowing, hypertrophy of the articular processes, large osteophytes (*arrows*), and subarticular bone erosions (*arrowheads*).

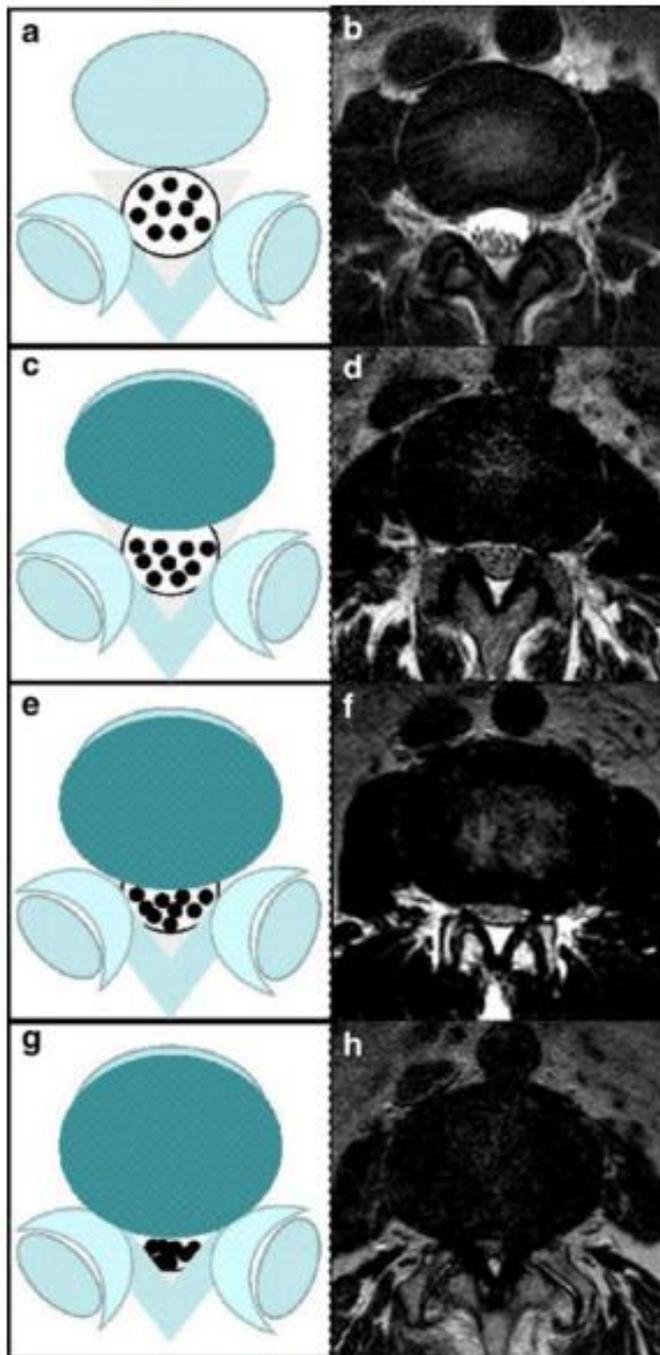
ENDPLATES – (Modic 1988)

Type	Characteristics
Type 1	Decreased signal intensity on T1-weighted images and an increased signal intensity on T2-weighted images
Type 2	Increased signal on T1-weighted images and an Iso- or slightly hyperintense signal on T2-weighted images
Type 3	Decreased signal intensity on both T1- and T2-weighted images, which appears to correlate with extensive bone sclerosis on plain radiographs

CANAL STENOSIS - (Guen 2011)

This grading system was performed using a T2W sequence.

Grade	Characteristics
Grade 0	Defined as no Lumbar Central Canal Stenosis (LCCS) as the anterior CSF space was not obliterated
Grade 1	Defined as mild LCCS, in which the anterior CSF space was mildly obliterated, but all cauda equine could be clearly separated from each other
Grade 2	Defined as moderate LCCS, in which the anterior CSF space was moderately obliterated and some of the cauda equine were aggregated, making it impossible to visually separate them
Grade 3	Defined as severe LCCS, in which the anterior CSF space was obliterated so severely as to show marked compression of the dural sac, and none of the cauda equine could be visually separated from each other, appearing instead as one bundle



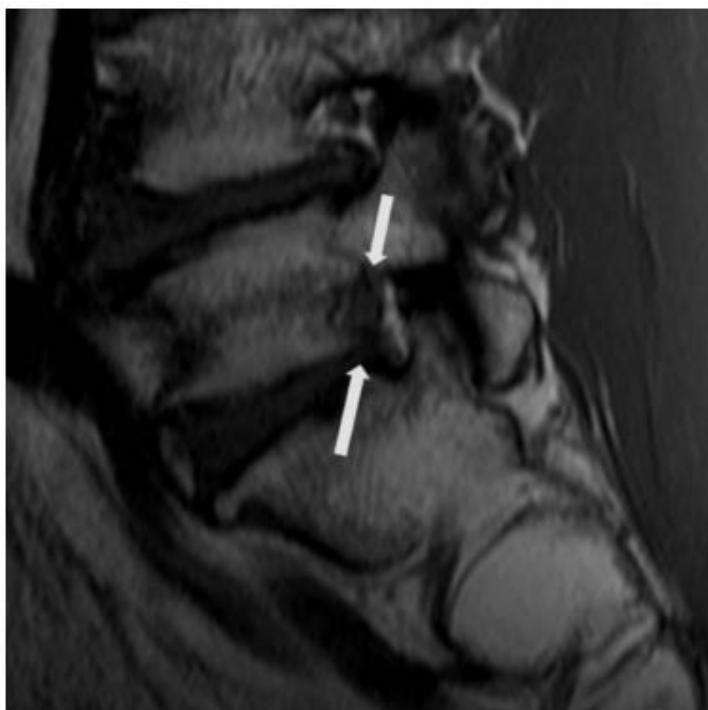
Lumbar central canal stenosis (LCCS) is defined when anterior CSF space is obliterated and is divided into four grades: grade 0, no LCCS (**a, b**); grade 1, mild stenosis with clear separation of each cauda equine (**c, d**); grade 2, moderate stenosis with some cauda equina aggregation (**e, f**); grade 3, severe stenosis with the entire cauda equina as a bundle (**g, h**). Diagrams on left and T2-weighted axial images on right side of each LCCS grade are illustrated grading

FORAMINAL STENOSIS – (Park 2012)

Grade	Characteristics
Grade 0	Absence of foraminal stenosis
Grade 1	Mild foraminal stenosis showing perineural fat obliteration surrounding the nerve root in 2 opposing directions without morphologic change of the nerve
Grade 2	Moderate foraminal stenosis showing perineural fat obliteration surrounding the nerve in 4 directions without morphologic change of the nerve
Grade 3	Severe foraminal stenosis showing nerve root collapse or morphologic changes\

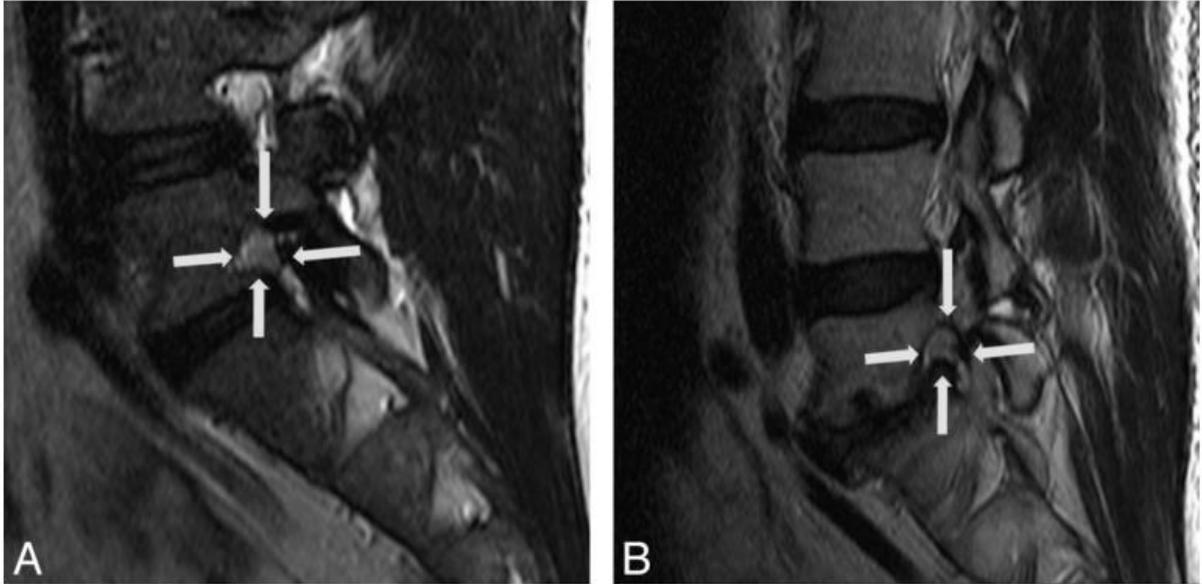
T1W sequence used. When unsure of grading, T2W sequence was used.

Grade 1



The Lee system grade 1. T1-weighted image of a 54-year-old man shows narrowing of the vertical width of the neural foramen and decreased intervertebral disk space in the left L5–S1 (arrows). Partial perineural fat obliteration is noted, but deformity of the nerve root is not seen.

Grade 2



A and B, The Lee system grade 2. T2-weighted images of a 39-year-old man and a 64-year-old man show narrowing of the vertical and transverse width of neural foramina in the left L5–S1 and the right L5–S1. Decreased intervertebral disk space, thickened ligamentum flavum, and disk protrusions are seen (arrows). Perineural fat obliteration is also seen, but nerve root deformity is not noted.

Grade 3



The Lee system grade 3. T1-weighted image of an 82-year-old woman revealing marked narrowing of the vertical and transverse width of neural foramina at L5–S1. Decreased intervertebral disk space, thickened ligamentum flavum, and disk protrusions are seen (arrows). The nerve root is collapsed and deformed compared with another nerve root at a different level.

NERVE ROOT COMPRESSION – (Pfirrmann 2004)

Grade	Characteristics
Grade 0	No compromise of the nerve root is seen. There is no evident contact of disc material with the nerve root, and the epidural fat layer between the nerve root and the disc material is preserved
Grade 1 (contact)	There is visible contact of disc material with the nerve root, and the normal epidural fat layer between the two is not evident
Grade 2 (deviation)	The nerve root is displaced dorsally by disc material
Grade 3 (compression)	The nerve root is compressed between disc material and the wall of the spinal canal; it may appear flattened or be indistinguishable from disc material

Grade 0

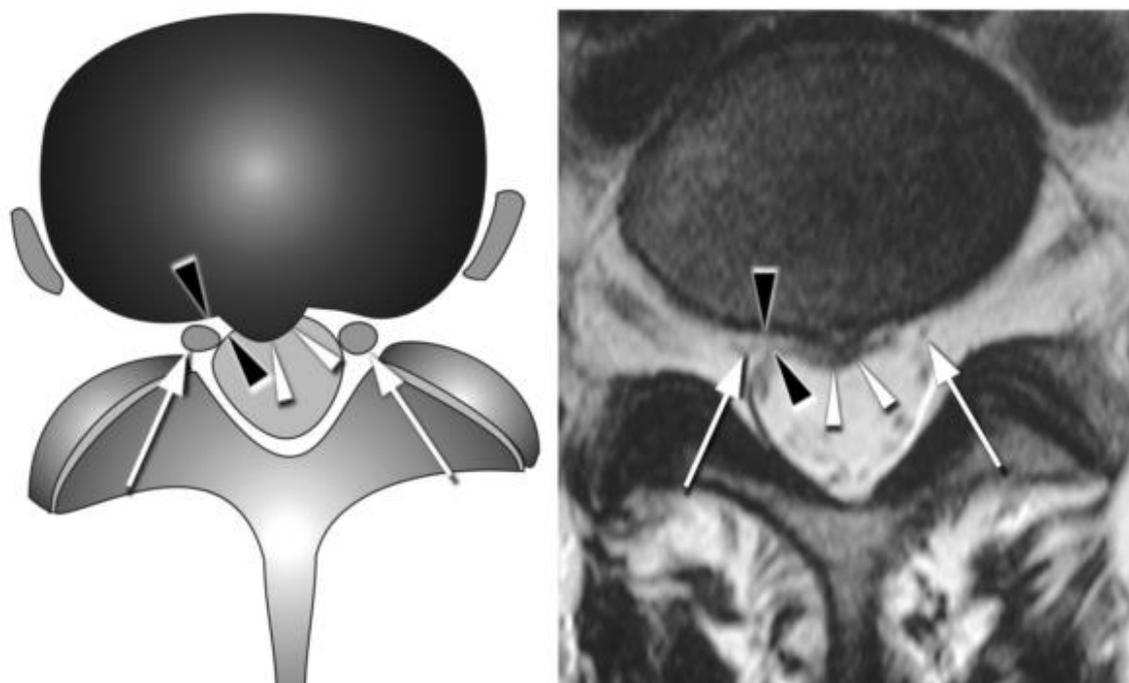


Figure 1. Diagram (left) and transverse T2-weighted fast spin-echo (SE) (repetition time msec/echo time msec = 4,000/122) MR image (right) show no compromise of the nerve root. A normal epidural fat layer (black arrowheads) is visible between the nerve root (arrows) and the disk material (white arrowheads).

Grade 1

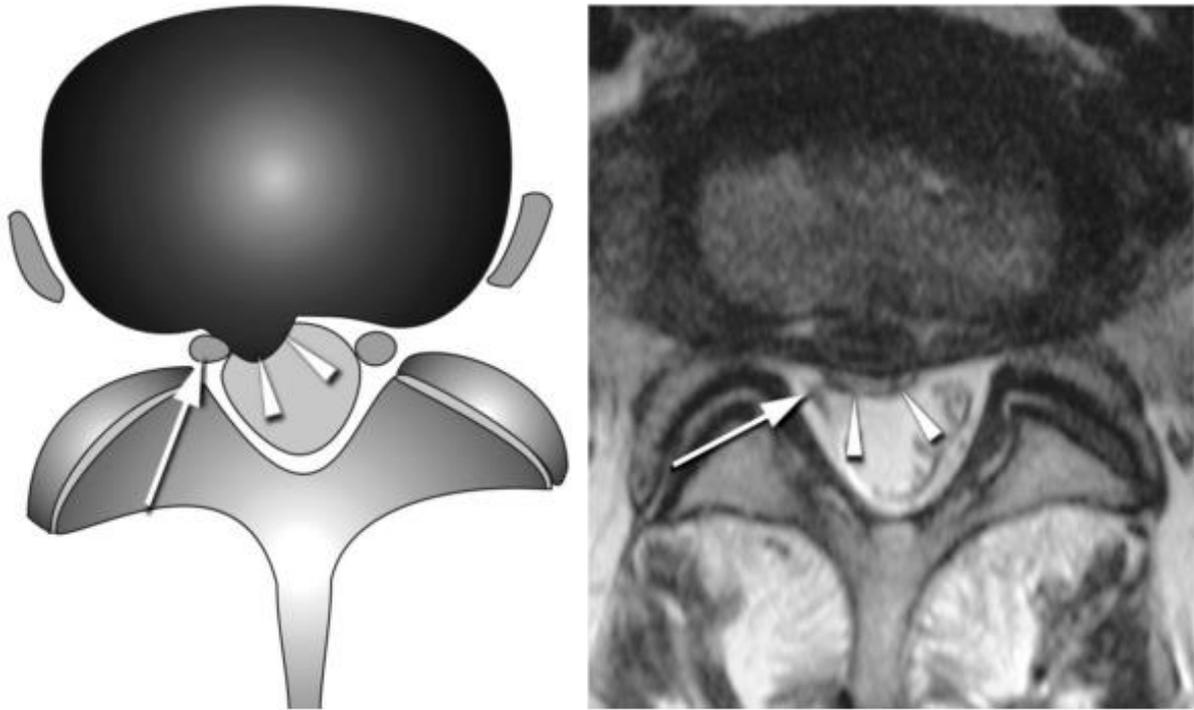


Figure 2. Diagram (left) and transverse T2-weighted fast SE (4,000/122) image (right) show contact of disk material (arrowheads) with the right nerve root (arrow). No epidural fat layer is visible between the nerve root and the disk material. The nerve root is in the normal position and is not dorsally deviated.

Grade 2

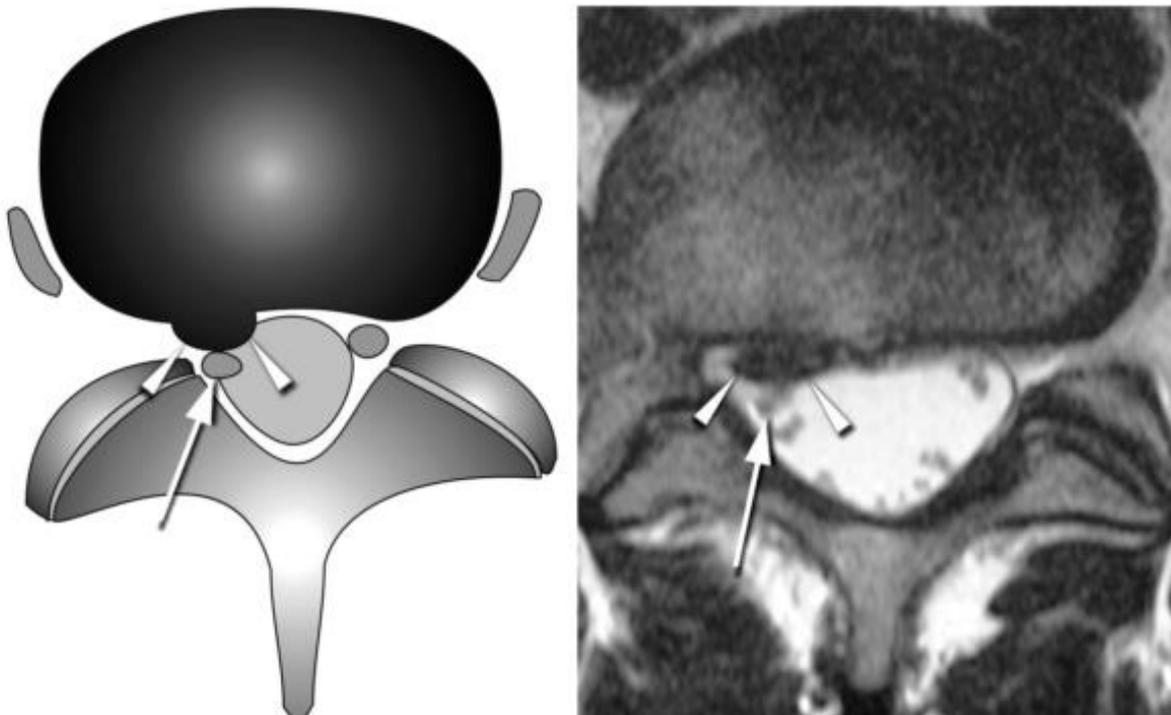


Figure 3. Diagram (left) and transverse T2-weighted fast SE (4,000/122) image (right) show dorsal deviation of the right nerve root (arrow), caused by contact with disk material (arrowheads).

Grade 3

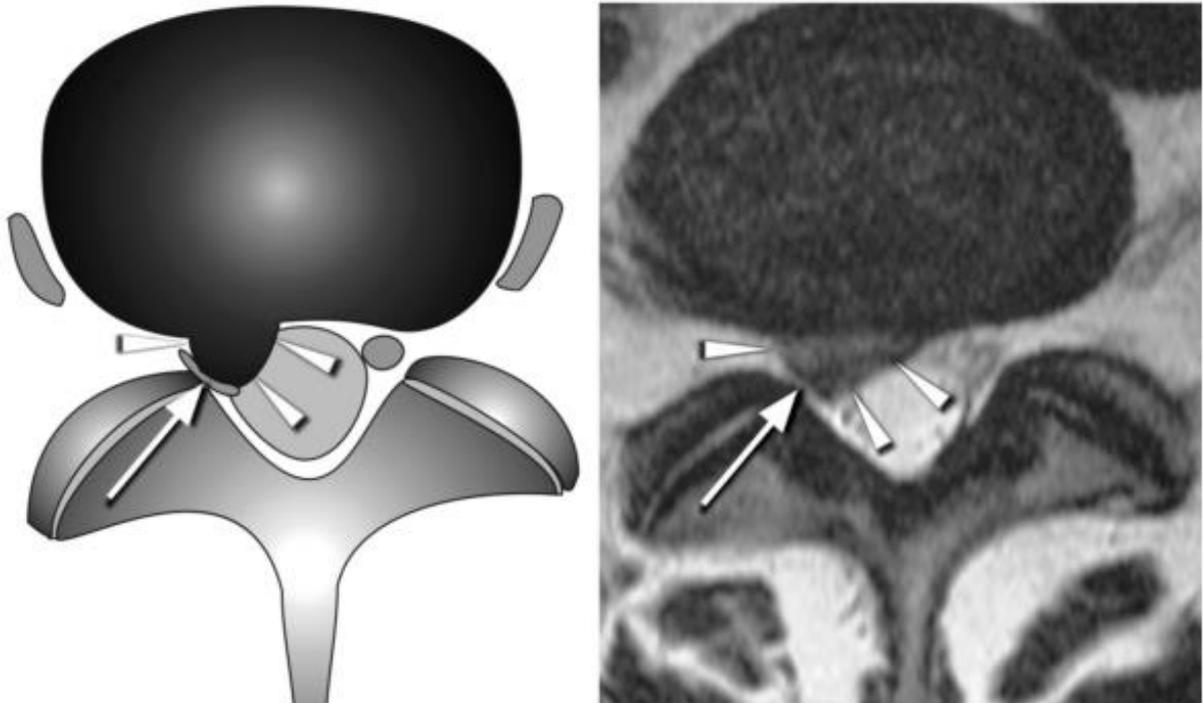


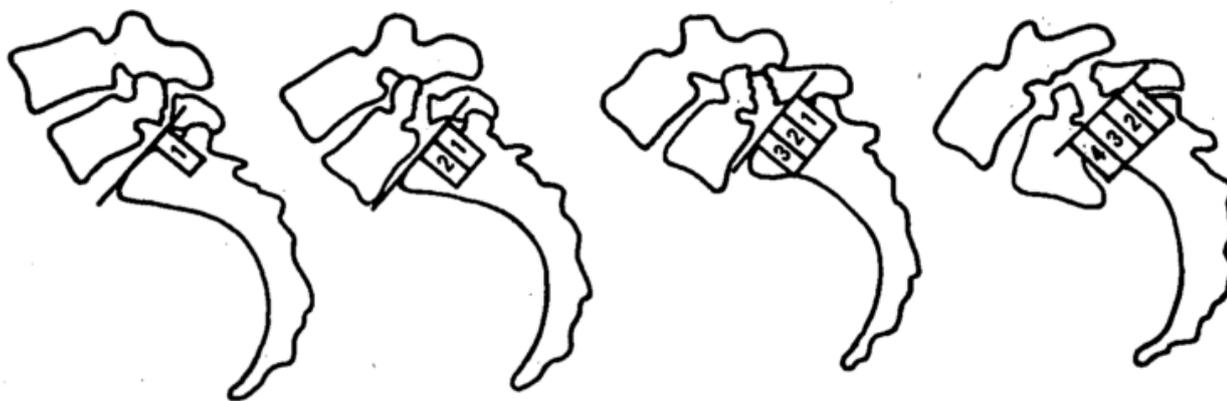
Figure 4. Diagram (left) and transverse T2-weighted fast SE (4,000/122) image (right) show compression of the right nerve root (arrow) between disk material (arrowheads) and the wall of the spinal canal. The nerve root appears flattened and is indistinguishable from disk material.

SPONDYLOLISTHESIS – (Meyerding 1932)

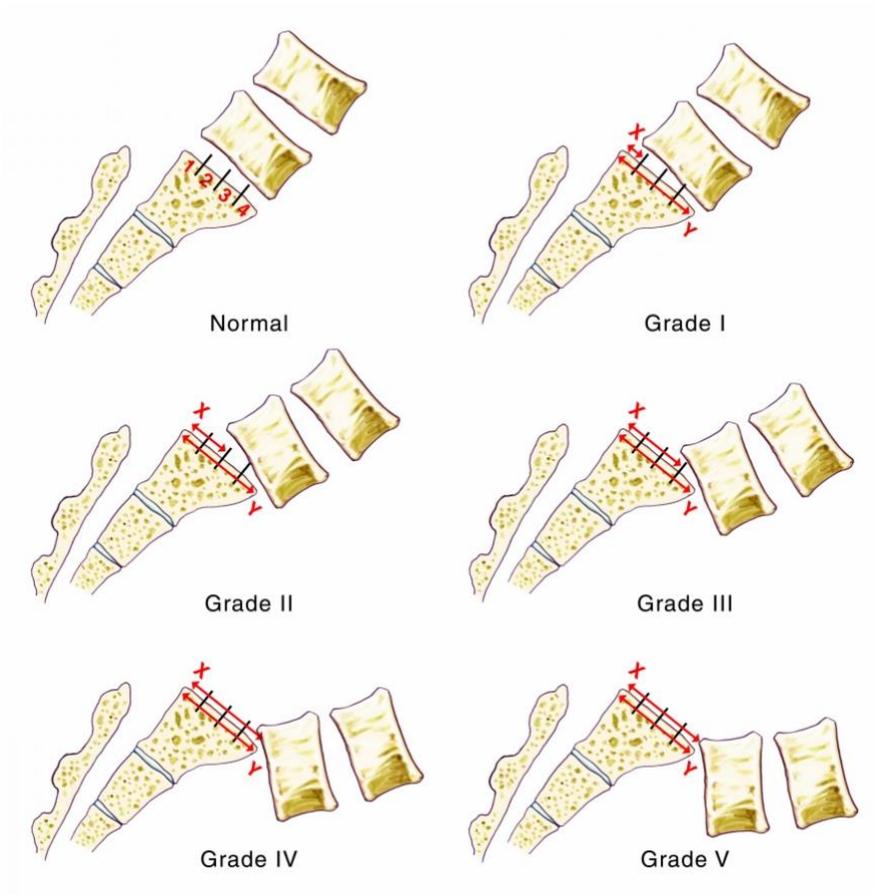
(This is slightly modified by myself (Grade 0))

Spondylolisthesis grading is based on the percentage that a vertebral body has slipped forward over the vertebral body below.

Grade	Characteristics
Grade 0	No slippage
Grade 1	1-24%
Grade 2	25-49%
Grade 3	50-74%
Grade 4	75-99%
Grade 5	Complete slip (100%) - Spondyloptosis



(Meyerding H.W. 1932)



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