

IS THE KICK SERVE LINKED WITH PARS ABNORMALITIES IN ELITE ADOLESCENT TENNIS PLAYERS?

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The purpose of this study was to compare lumbar kinetics in elite adolescent tennis players with and without pars abnormalities. A 3D motion capture system was used to capture lumbar spine kinetics while 24 right-handed elite adolescent tennis players (male = 14, female = 10, 9 players with pars abnormalities) performed kick serves. Players without pathology were found to have significantly greater peak lumbar posterior force (7.3 N/kg greater), left lateral flexion force (4 N/kg greater), right lateral flexion force (3.4 N/kg greater), flexion moment (2 Nm/kg greater) and left rotation moment (1.2 Nm/kg greater) during the drive phase. Interestingly, no differences were observed for peak kinetic values during the forward-swing phase. Those with pars abnormalities had lower lumbar loading overall compared with those without pars abnormalities.

KEY WORDS: biomechanics, tennis, injury, lumbar spine

INTRODUCTION: Low back pain (LBP) is pervasive among elite junior tennis players (Gescheit et al., 2019; Hjelm, Werner, & Renstrom, 2012; Hutchinson, Laprade, Burnett, Moss, & Terpstra, 1995). Between 2008 – 2015, lumbar spine consultations in junior tennis athletes increased 10-fold at the Tennis Australia National Academy (unpublished data, Tennis Australia). Further, most consultations were for male players, inferring that males are more susceptible to LBP than females. The prominence of LBP in tennis extends right through the development pathway to professional players where LBP is still ubiquitous.

The tennis serve, whilst the most important stroke in the sport, has been scrutinized as a potential risk factor for LBP (Abrams, Renstrom, & Safran, 2012; Abrams, Sheets, Andriacchi, & Safran, 2011). Preliminary work has explored the relationship between LBP and the serve and has found that players with LBP have less lumbar mobility and significantly greater peak lumbar left lateral force compared to players without LBP (Campbell, O'Sullivan, Straker, Elliott, & Reid, 2014; Campbell, Straker, O'Sullivan, Elliott, & Reid, 2013). The extent to which this is adaptive or maladaptive remains unclear though and thus further research in asymptomatic junior tennis athletes is needed to explore potential risk factors for LBP.

An improved understanding of lumbar spine loading in asymptomatic players with lumbar spine abnormalities would allow health practitioners and coaches to better understand the relationship between serving technique and LBP. The aim of this study was to investigate the link between lumbar spine loading and the presence of lumbar spine abnormalities. We hypothesize that players with lumbar spine abnormalities will demonstrate greater lumbar loads.

METHODS: Twenty-four (male: 14, female: 10) right-handed elite adolescent tennis players (mean age: 13.0 ± 1.7 years), who had undergone a lumbar spine MRI as part of annual screening were recruited for this study. The participants were then divided into two groups; Pars (P) those who were diagnosed with an abnormality at the pars Interarticularis (n = 9), and No Pars (NP) those who did not have a pars abnormality (n = 15). Participants were not split based on gender as recent research has shown that no differences in lumbar kinetics exist between males and females (Connolly, Middleton & Reid, *in review*). Participants were excluded if they had a current performance inhibiting injury, were ill, or experienced pain at the time of testing. Ethical approval was obtained from the Victoria University Human Research Ethics Committee and participants provided voluntary informed consent and assent prior to any involvement in the study.

All data were collected on indoor hard courts at the Tennis Australia National Academy in Melbourne. Prior to testing, participant height, mass and racquet parameters (mass, centre of mass (COM), swing-weight, spin-weight and polar moment of inertia) were recorded. Retroreflective markers (12.7 mm diameter) and clusters on rigid plates were attached to the skin on relevant segments and anatomical landmarks (lower body, trunk and upper body, racquet and ball) using double-sided tape. Marker trajectories were recorded using a 12-camera opto-reflective motion capture system (Vicon Motion Systems Ltd, Oxford, UK; 250 Hz). Once markers were attached, players completed a self-directed warm up followed by subject-specific calibration trials. Participants completed a series of serves aiming toward a 1 m x 2 m target area on the deuce court at the "T". The players performed maximal effort kick serves (KS) with successful serves being defined as those that landed in the target area. Serving continued until three successful KS were completed in accordance with prior established methods (Campbell, O'Sullivan, Straker, Elliot & Reid, 2014; Campbell, Straker, O'Sullivan, Elliot & Reid, 2013).

The data were processed and treated using Vicon Nexus software (Vicon Motion Systems Ltd, Oxford, UK, V 2.7.0) and a cubic spline was used to fill gaps within marker trajectories. Trajectories were filtered using a 15 Hz low-pass Butterworth filter following a residual analysis and visual inspection of the data. Markers on the left arm, ball and racquet were used to identify the frame for ball release and impact as well as four key time points within each serve to define phases as per previous research (Whiteside, Elliott, Lay, & Reid, 2013); ball release/toss (BT), racquet high point (RHP), racquet low point (RLP) and impact. A Cook's distance test was carried out to remove influential data points followed by a Shapiro-Wilk test to test for normality. The Shapiro-Wilk test revealed that the data was not normally distributed and thus a non-parametric test (Mann-Whitney U test) assessed for differences between players with abnormalities. As multiple comparisons were conducted, the alpha value was adjusted *a priori* to 0.01 to reduce the risk of Type 1 error (Whiteside et al., 2013). 95% confidence intervals and effect sizes were also calculated between abnormality groups (Cohen, 1988).

RESULTS: The magnitude of peak lumbar kinetics for the kick serve were generally comparable between the P and NP groups (Table 1). The NP group tolerated greater left lateral forces (4 N/kg, $p=0.01$) compared with the P group during the drive phase. No differences in peak lumbar kinetics were found during the forward-swing phase. Lastly, moderate effect sizes were found for peak lumbar posterior and left lateral force as well as peak lumbar flexion moment during the drive phase.

Table 1 Peak lumbar kinetics for the Pars and No Pars group.

| | Pars | No Pars | p-values | Confidence Intervals | Effect size |
|------------------------------------|--------------|--------------|----------|----------------------|-------------------|
| Drive Phase | | | | | |
| <i>Peak Lumbar Forces (N/kg)</i> | | | | | |
| Anterior | 18.4 ± 12.0 | 23.6 ± 13.4 | 0.12 | -1.22 - 10.96 | 0.42 |
| Posterior | -16.5 ± 11.7 | -23.8 ± 13.7 | 0.04 | -12.6 - -0.3 | 0.58 [^] |
| Distraction | 19.7 ± 9.7 | 19.5 ± 4.9 | 0.23 | -1.22 - 4.95 | 0.03 |
| Compression | -6.5 ± 8.9 | -8.7 ± 8.8 | 0.12 | -6.06 - 0.87 | 0.25 |
| Left Lateral | -12.0 ± 8.5 | -16.0 ± 7.2 | 0.01* | -8.17 - -0.98 | 0.52 [^] |
| Right Lateral | 9.8 ± 8.7 | 13.2 ± 7.9 | 0.04 | 0.16 - 7.82 | 0.42 |
| <i>Peak Lumbar Moments (Nm/kg)</i> | | | | | |
| Flexion | 5.0 ± 2.7 | 7.0 ± 3.4 | 0.03 | 0.19 - 3.57 | 0.68 [^] |
| Extension | -5.3 ± 3.7 | -6.8 ± 4.0 | 0.11 | -3.04 - 0.55 | 0.40 |
| Right Lateral Flexion | 6.9 ± 4.4 | 5.8 ± 2.3 | 0.68 | 1.26 - 1.24 | 0.32 |
| Left Lateral Flexion | -3.7 ± 4.7 | -2.8 ± 2.6 | 0.86 | -1.24 - 1.38 | 0.26 |

| | | | | | |
|------------------------------------|--------------|--------------|------|---------------|------|
| Right Rotation | 4.1 ± 2.2 | 5.0 ± 2.8 | 0.27 | 0.50 - 2.02 | 0.37 |
| Left Rotation | -3.4 ± 2.6 | -4.6 ± 2.6 | 0.05 | 2.42 - -0.02 | 0.48 |
| Forward-Swing Phase | | | | | |
| <i>Peak Lumbar Forces (N/kg)</i> | | | | | |
| Anterior | 19.9 ± 14.8 | 25.4 ± 14.4 | 0.19 | -1.76 - 13.29 | 0.38 |
| Posterior | -18.0 ± 10.7 | -21.4 ± 10.7 | 0.36 | -8.76 - 2.82 | 0.32 |
| Distraction | 14.0 ± 11.5 | 15.3 ± 8.3 | 0.15 | -1.18 - 6.66 | 0.14 |
| Compression | -16.8 ± 10.2 | -17.9 ± 7.7 | 0.50 | -6.13 - 2.30 | 0.12 |
| Left Lateral | -17.7 ± 10.0 | -19.5 ± 8.8 | 0.72 | -6.08 - 2.92 | 0.19 |
| Right Lateral | 13.0 ± 9.5 | 15.1 ± 9.2 | 0.37 | -1.85 - 6.16 | 0.23 |
| <i>Peak Lumbar Moments (Nm/kg)</i> | | | | | |
| Flexion | 7.2 ± 4.4 | 8.2 ± 4.1 | 0.41 | -1.14 - 3.06 | 0.23 |
| Extension | -8.0 ± 4.2 | -9.8 ± 4.8 | 0.29 | -3.98 - 1.26 | 0.38 |
| Right Lateral Flexion | 6.4 ± 3.9 | 6.4 ± 3.1 | 0.82 | -1.36 - 1.70 | 0.01 |
| Left Lateral Flexion | -5.2 ± 3.8 | -5.3 ± 3.2 | 0.65 | -1.58 - 0.90 | 0.03 |
| Right Rotation | 2.9 ± 1.5 | 3.7 ± 2.0 | 0.22 | -0.32 - 1.4 | 0.41 |
| Left Rotation | -3.0 ± 1.6 | -3.7 ± 2.4 | 0.56 | -1.21 - 0.49 | 0.36 |

“**” Significant main effect for abnormality groups ($p < 0.01$). between pars and no pars groups.

“^” Represents a moderate or higher effect size between abnormality groups.

DISCUSSION: Similar to previous reports, this study found differences in kick serve kinetics between those with and without lumbar pathology. Campbell et al. (2013) reported that lumbar spine kinetics were comparable between those with and without LBP however found that peak left lateral forces were significantly higher in those with LBP during the drive phase. On the contrary, this study found that peak left lateral forces were significantly lower in the P group, even though the values from our study were much higher than previous reports (~3.5 N/kg versus ~18 N/kg). Similar lumbar kinetics have been reported in cricket fast bowlers (Crewe, Campbell, Elliott, & Alderson, 2013), with anterior-posterior force values ranging from ~19 N/kg (anterior force) to ~-30 N/kg (posterior force) and the magnitude of medio-lateral forces ranging from ~25 N/kg (right lateral flexion force) to ~-20 N/kg (left lateral flexion force). These magnitudes have been implicated in the high incidences of stress fractures in fast bowlers and, when coupled, are known to increase the risk of lumbar injury (Chosa, Totoribe, & Tajima, 2004).

The passive spinal structures (discs, ligaments, vertebrae) are responsible for limiting lumbar movement and are known to stiffen with repeated loading (Markolf, 1972). The purpose of this is to facilitate spinal stability during movements such as serving in tennis. However, loss in mobility has been shown to decrease force production and thus it is possible that the P group in this study have reduced spinal stability resulting in up to 7 N/kg less force (posterior force) (Table 1). The combination of less lumbar mobility/active range of motion (ROM) and repeated loading (resulting in subsequent increase in stiffness) might lead to more focal loading on the passive spinal structures resulting in an abnormality as opposed to loading these spinal structures over a greater ROM.

Lastly, from a physical maturity stand point, large lumbar extension loads are experienced when serving and as a result the pars interarticularis is simultaneously stretched and sheared (Leone, Cianfoni, Cerase, Magarelli, & Bonomo, 2011). With the ossification of the neural arch not yet complete (Maquirriain, 2006), these forces could provoke lifelong damage to the lumbar spine early during the players' development.

Many pars injuries develop from a rapid increase in workload (Barile, Limbucci, Splendiani, Gallucci, & Masciocchi, 2007). In tennis, athletes compete year-round with little time off and thus prudent workload prescription is essential for injury prevention, especially since the tennis serve has been flagged as a risk factor for lumbar spine injury. However, workload management can be tricky in tennis as not only do athletes compete year-round, if progressing deep into tournaments, athletes can find themselves competing daily in matches that can last

for multiple hours. Interestingly, pars injuries have been linked with large facet joint angles (more coronal) (Connolly et al., *in review*) that can inhibit lumbar flexion/extension to reduce mobility and potentially reduce posterior forces and flexion moments ($p=0.04$ and $p=0.03$ respectively). With reduced lumbar loading and increased tension on the pars, it is then possible that the pattern of bone loading for the P group is different during this crucial stage of bone mineral density development (Rauch, Bailey, Baxter-Jones, Mirwald, & Faulkner, 2004). Therefore, there is potential that the lower lumbar spine loading seen in our results may help explain the presence of lumbar abnormalities.

CONCLUSION: This study quantified the differences in kick serve kinematics between elite adolescent tennis players with and without an asymptomatic lumbar pars abnormality. The NP group had significantly greater peak lumbar left lateral force compared to the P group, though no other differences were detected. Future research would benefit from exploring time series data near peak kinetics so as to further inform LBP prevention and management strategies.

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