

Lumbopelvic motor control in elite tennis players with and without a history of low back pain

Contrôle moteur lombo-pelvien de joueurs de tennis élités avec et sans antécédent de lombalgie

S. Grosdent^{a,b}, D. Colman^{a,b}, C. Demoulin^{a,b}, J.-F. Kaux^{a,b}, N. Roussel^c, M. Vanderthommen^{a,b}

^a Department of Sport and Rehabilitation Sciences (EVAREVA), University of Liège, Quartier Blanc Gravier, B21, allée des Sports 2, 4000 Liège, Belgium

^b Department of Physical Medicine and Rehabilitation, Liège University Hospital Centre, Liège, Belgium

^c Department of Physiotherapy and Rehabilitation Sciences (Movant), University of Antwerp, Antwerp, Belgium

Abstract

Objectives. — The purpose of this cross-sectional study is to investigate the prevalence of impaired lumbopelvic motor control in elite tennis players with disabling episode of low back pain in the previous year.

Material and methods. — Thirty-five elite tennis players (9 males and 26 females, mean age 17.4 ± 2.6 years) from two Belgian Tennis Academies were asked to fill in questionnaires related to low back pain and to perform five lumbopelvic motor control tests (bent knee fall out test, knee lift abdominal test, sitting knee extension test, waiters bow and transversus abdominis test). A physiotherapist, blinded to the medical history of the participants, scored the performance of the players for each of the tests (0 = failed, 1 = correct) resulting in a total lumbopelvic motor control score ranging from 0 to 5.

Results. — Forty percent of players reported at least one past disabling episode of low back pain lasting more than five consecutive days in the previous year. Significant more altered lumbopelvic motor control tests were observed in players with a history of low back pain (score of 1.7 vs. 3.3 in those without a history of low back pain, $P < 0.001$). The between-group difference was particularly marked for the bent knee fall out test, the knee lift abdominal test and the transversus abdominis test ($P \leq 0.05$).

Conclusions. — The current study confirms that low back pain is common in tennis players and has pointed out impaired motor control of the lumbopelvic region in most tennis players with a history of low back pain.

Keywords:

Lumbopelvic region

Motor-control

Tennis

Low back pain

Abstract

Objectifs. — Cette étude transversale a pour objectif d'examiner la prévalence des altérations du contrôle moteur lombo-pelvien de joueurs de tennis élités avec un antécédent de lombalgie invalidante au cours de l'année écoulée.

Matériel et méthodes. — Trente-cinq joueurs de tennis élités (9 hommes et 26 femmes, âge moyen de $17,4 \pm 2,6$ ans) issus de deux académies de tennis belges ont complété des questionnaires relatifs à la lombalgie et réalisé cinq tests de contrôle moteur lombo-pelvien (le « bent knee fall out test », le « knee lift abdominal test », le « sitting knee extension test », le « waiters bow » et la capacité à contracter le muscle transverse de l'abdomen). Une kinésithérapeute expérimentée, ignorant les antécédents médicaux des participants, a évalué les performances de chacun des joueurs lors des cinq tests (0 = échec, 1 = réalisation correcte), aboutissant à un score de contrôle moteur lombo-pelvien compris entre 0 (échec aux cinq tests) et 5 (réussite aux cinq tests).

Résultats. — Quarante pour cent des joueurs ont rapporté au moins un épisode invalidant de lombalgie d'une durée minimale de cinq jours consécutifs au cours de l'année écoulée. Les joueurs avec antécédent de lombalgie présentent un score de contrôle moteur lombo-pelvien significativement inférieur à celui des joueurs sans antécédent de lombalgie (score de 1,7 vs 3,3 respectivement, $p < 0,001$). La différence entre les groupes était particulièrement marquée pour le « bent knee fall out test », le « knee lift abdominal test » et la capacité à contracter de manière isolée le muscle transverse de l'abdomen ($p \leq 0,05$).

Conclusions. — Cette étude confirme que les douleurs lombaires représentent des plaintes fréquentes chez les joueurs de tennis élités et a permis de mettre en évidence une perturbation du contrôle moteur lombo-pelvien chez la plupart des joueurs de tennis avec un antécédent de lombalgie au cours de l'année écoulée.

Keywords:

Région

Lombo-pelvienne

Contrôle moteur

Tennis

Lombalgie

1. Introduction

Tennis is played by millions of recreational players and is by far the most popular of all racket sports. Over the last 20 years, the number of players has grown significantly, with more than 200 nations being member of the International Tennis Federation [1]. At the professional level, tennis gets a high amount of media attention particularly for the major international tournaments organized all around the world. Tennis is a unilateral sport with high physical demands, requiring repetitive trunk multidirectional movements. The trunk, and more specifically the lumbar spine, is a common site of musculoskeletal injuries in elite junior and adult tennis players [2,3]. The lifetime prevalence for non-specific low back pain (LBP) ranges from 54 to 86% in elite tennis players [4,5]. Female tennis players seem to have a higher incidence of LBP than males [6] and low back/trunk injuries represent 22 to 26% of total injuries in female adolescent players [7].

LBP can interfere with tennis training and competition and is thereby a common reason for lost playing time by competitive professional and semi-professional athletes [8,9]. According to Vad et al. (2003), 40% of professional men tennis players from the ATP circuit report a past history of LBP having limited their performance for more than two weeks [8]. In a case series of 148 male professional tennis players, 38% reported having missed at least one event at some point in their careers because of LBP and 29% suffered from chronic LBP [9]. Subsequently, it not only leads to a less optimal performance or contractual and financial consequences, but it might also have long-term consequences for spinal health [10].

LBP is a complex and multifactorial condition with potential risk factors grouped broadly into biophysical, psychological and social dimensions [11]. The repetitive ballistic trunk movements required in tennis induce mechanical stress which might contribute to LBP injury [12,13]. Some studies reported that muscular (lateral flexion lumbar forces contralateral to the dominant arm [12]) and kinematic parameters (pelvis movement during the kick and flat serves [13]) differ between players with and without a history of LBP suggesting that mechanical factors may contribute to lumbar pain in elite junior tennis players. Neuromuscular imbalance of erector spinae (measured by surface-electromyography) has also been reported in tennis players with LBP in comparison with asymptomatic players [14], whereas flexibility and maximal voluntary trunk strength have not been linked with LBP [15]. Regarding bio-physical factors, some authors suggest that attention should be paid to sensorimotor control rather than strength impairments in athletes and in the general population suffering from LBP [16]. Indeed, although trunk muscle strength and endurance contribute to the stability of the trunk and the lumbopelvic region during athletic activities, this stability most depends on the sensorimotor control and intersegmental stability [17]. Some studies have also reported that poor sensorimotor control of the trunk predisposes athletes to sport injuries of the spine [18,19] and that significant changes, especially regarding the deep lumbopelvic stabilization muscles (the multifidus and the transversus abdominis), occur and result in changes of the lumbopelvic stabilization strategies in athletes with current or past LBP (e.g., hyperactivation of some superficial trunk muscles) [20,21]. These changes might alter neuromuscular control strategies for sudden trunk loading and thereby increase the risk of (recurrent) low back pain/injury [18]. Besides these “biophysical components”, influence of psychological and social risk factors should not be ignored [11]. Yet, these factors have not been extensively investigated. Mood and stress could predict LBP in various elite athletes (including

tennis players) [22] and the risk of chronicity was higher in athletes with high levels of stress [23].

Whilst altered movement control of the lumbopelvic region has been reported in different athletes (e.g., elite cricketers [24], elite Australian Football League players [25], dancers [26], soccer players [27]) with current or past LBP, research regarding this topic in tennis players is lacking. Therefore, the present study aimed to investigate lumbopelvic motor control (LMC) of young elite tennis players with and without disabling episode of LBP in the previous year. We hypothesized that elite tennis players with a history of LBP present an altered motor control of the lumbopelvic region in comparison with players without a history of LBP.

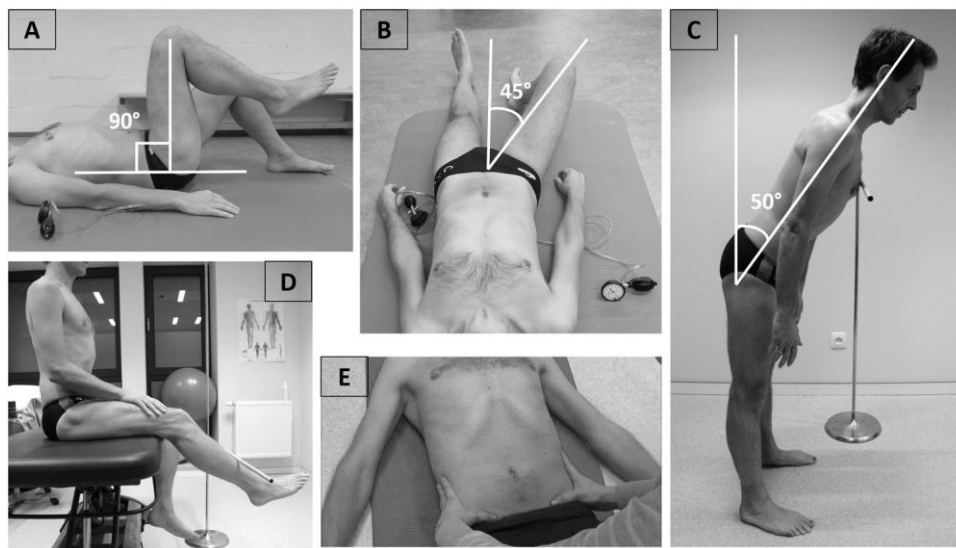


Figure 1. The battery of five lumbopelvic motor control tests. A. Knee lift abdominal test. B. Bent knee fall out. C. Waiter's bow. D. Sitting knee extension test. E. Ability to contract the transversus abdominis muscle.

2. MATERIALS AND METHODS

2.1 PARTICIPANTS

Elite competitive male and female tennis players from two Belgian Tennis Academies were invited to participate in this study. Inclusion criteria were: age between 15 to 25 years, ranking at the Association of Tennis Professionals (ATP), Women's Tennis Association (WTA) or International Tennis Federation (ITF).

Tennis players with a history of low back surgery, or with a past history of serious injury or operative treatment which could interfere with normal tennis training or competing at the time of the study were excluded.

Prior to participation, the purpose and procedure of the study were explained in detail to the participants and their parents (for participants under 18 years of age). The research protocol was approved by the relevant Hospital and Faculty Ethics Committee (Ref. number: B70720111330) and the necessary parental and athlete consent/assent was obtained.

2.2.PROCEDURES

A cross-sectional design was used to compare LMC in tennis players with and without a history of LBP. All participants were invited to participate to one assessment session organized in the fitness room of each Tennis Training Center. During this session, all participants were asked to fill in a general questionnaire and the NORDIC questionnaire. LMC was assessed by means of a battery of five LMC tests (Knee Lift Abdominal Test [KLAT], Bent Knee Fall Out Test (BKFO), Waiter's Bow [WB], Sitting Knee Extension Test [SKET] and Ability to contract the transversus abdominis muscle [cTrA]) (**Fig. 1**).

A general questionnaire was used to collect demographic information (age, weight, height and dominant upper limb) as well as information regarding sport practice (number of years in high-performance sport, hours of training per week, number of weekly training sessions). The presence and sites of musculoskeletal injury were assessed by means of the NORDIC questionnaire. The following anatomical sites were studied: neck, upper back, lower back, shoulders, elbows, wrists/hands, hips/thighs, knees/lower legs and ankles/feet. An injury was defined as any physical complaint that is the result of participating in tennis training or competition, leading to a player being unable to fully participate in future tennis training or match play. The NORDIC questionnaire was used to identifying participants with LBP [28].

The battery of LMC tests was based on previous research in general population and athletes [26,27,29]. This test battery included five field tests. Before each test, three pre-testing trials were conducted to familiarize the participants with the clinical tests. An experienced physiotherapist, blinded to the medical history questionnaires, scored (0 = failed, 1 = correct) the performance of each player on the five tests. For bilateral tests (KLAT, BKFO, SKET), unilateral failure led to a score of zero. This resulted in a "total lumbopelvic motor control score" (LMC score) ranging from 0 (failed at all tests) to 5 (maximal score) [27].

The KLAT and the BKFO tests were performed in a supine position. A Pressure Biofeedback Unit (Chattanooga Ltd Hixson, USA) was placed under the lumbar spine, as previously described [27], to objectify lumbar spine movements. In brief, the pressure of the Pressure Biofeedback Unit was inflated to 40 mmHg (baseline pressure). Participants were instructed to maintain the neutral spine position (preventing spinal movement) during a unilateral thigh movement (flexion for the KLAT, abduction/external rotation for the BKFO). The other leg was extended (BKFO) or bent (KLAT) and rested on the table. Maximal pressure deviation from the baseline during the movement was recorded (in mmHg) during each test. The test was considered as failed when a change in the pressure level > 8 mmHg (either up or down) occurred [27].

The WB and the SKET were executed in an upright standing and in an upright sitting neutral (i.e., in the midrange between anterior and posterior pelvic tilt) position, respectively. The participant was instructed to keep the neutral position (physiological lordosis) of the lumbar spine, while

performing a 50° trunk flexion at the hips for the Waiter's Bow or extending the knee to minus 10° extension or within the available range of motion for the SKET. The test was successful (score = 1) if the investigator observed that the stabilizing muscles of the trunk activated isometrically and managed to keep the spine in neutral position, while the participant bent forwards (Waiter's Bow) or extended the knee (SKET) [27].

The cTrA was assessed by the abdominal “drawing in” action in crook lying. For a correct test performance (score = 1), participants should be able to tension the transversus abdominis muscle (hollowing of the lower abdominal wall) for 10 s without excessive overflow to the upper abdominal wall, expansion of the internal oblique muscles, spinal or rib cage movement, as previously described [26]. Observation and palpation were used to evaluate the correct contraction of the transversus abdominis muscle. In case of compensations as mentioned above, the test was considered as failed.

2.3. STATISTICAL ANALYSES

Statistical analyses were performed with Statistica version 13.3 (StatSoftInc, Paris, France). A Shapiro-Wilk test was used to ensure the normality of the outcomes. In case of normally distributed variables, results are expressed as means $\pm$ SD and the independent samples Student's *t*-test was used to compare the tennis players with and without a history of LBP. If the Shapiro-Wilk test revealed that an outcome was not normally distributed, the nonparametric Mann-Whitney test was used to compare the tennis players with or without a history of LBP.

The Fisher's exact test was used to compare participants with and without a history of LBP regarding the categorical data (KLAT, BKFO, WB, SKET, cTrA). The significance level was set at 0.05.

The number of positive tests and pressure deviation during the KLAT and the BKFO in the two groups (tennis players with and without a history of LBP) were compared; the between-group differences were analyzed by the effect size (ES) *d*. The ES (*d*) is the difference of the means divided by the mean standard deviation of the groups. ES with $0.0 \leq d < 0.2$ is non-considered, $d \geq 2$ as small, $d \geq 0.5$ as moderate and $d \geq 0.8$ as large.

The sample size was estimated using power-based sample size calculations (power of 0.80 and α of 0.05). The planned sample size was estimated at 13 participants per group. The calculations were based on a study assessing LMC in athletes with and without a history of LBP [27]. We anticipated that approximately half of the tennis players would have experienced LBP in the year before the study and that differences in pressure deviation between participants with and without a history of LBP would be 2.5 mmHg. In order to account for possible drop out, 35 tennis players were recruited.

3. RESULTS

Thirty-five elite tennis players (9 males and 26 females, mean age, 17.4 ± 2.6 years) completed the study. Forty per- cent (6 males and 8 females) (named LBP group) reported at least one past disabling (preventing the player from training or taking part in competitions) episode of LBP lasting

more than five consecutive days in the previous year (range 5—90 days). At the time of the study, all players in the LBP group were training and competing.

Table 1 presents descriptive statistics of the two groups. Players with and without a history of LBP did not differ in age, body mass index, hours of tennis practice per week and duration of tennis practice.

Total LMC score, pressure excursion measured by means of the pressure biofeedback unit and number of tennis players with and without a history of LBP who failed the LMC tests are presented in **Table 2**.

The LBP group had a significantly more altered LMC tests compared to tennis players without a history of LBP (mean of 1.7 vs. 3.3 respectively; $P < 0.001$). The ES for the difference between the groups was large ($d = 1.6$), reflecting that the difference between the two means is greater than one standard deviation.

Differences between tennis players with and without a history of LBP were particularly marked for the BKFO ($P=0.006$), the KLAT ($P=0.03$) and the cTrA ($P=0.05$). In

contrast, no significant differences between groups were found for the SKET or the WB. Tennis players with a history of LBP had significantly higher changes in pressure than players without LBP for the BKFO.

Table 1 Descriptive statistics of tennis players with and without a history of LBP in the previous year.

	No LBP ($n = 21$)	LBP ($n = 14$)	<i>P</i> -value
Sex			
Women	18 (86%)	8 (57%)	0.07
Age (years)	18.0±2.8	16.6±1.9	0.10
BMI (kg/m ²)	20.0±2.5	21.3±3.0	0.18
Tennis activity (hours/week)	27.6±2.9	25.5±4.1	0.15
Duration of tennis practice (years)	9.6±3.6	8.6±2.8	0.38

Continuous variables are expressed in mean±SD, qualitative variables are expressed in absolute and relative frequencies, n (%). LBP: low back pain, SD: standard deviation, BMI: body mass index.

Table 2 Motor control assessment in tennis players with and without a history of LBP in the previous year.

	No LBP ($n = 21$)	LBP ($n = 14$)		
	Mean±SD	Mean±SD	<i>P</i> -value	ES
LMC Score (0-5)	3.3±0.8	1.7±1.1	< 0.001*	1.6
KLAT pressure deviation (mmHg)	8.7±3.4	11.6±7.2	0.16	0.5
BKFO pressure deviation (mmHg)	8.8±3.0	12.7±2.8	< 0.001*	1.4
	No LBP ($n = 21$)	LBP ($n = 14$)		
	n (%)	n (%)		<i>P</i> -value
KLAT failed	7 (33)	10 (71)		0.03*
BKFO failed	10 (48)	13 (93)		0.006*
WB failed	6 (29)	6 (43)		0.30
SKET failed	8 (38)	9 (64)		0.12
cTrA failed	5 (24)	8 (57)		0.05*

LBP: low back pain, KLAT: knee lift abdominal test, BKFO: bent knee fall out, WB: waiter's bow, SKET: sitting knee extension test, cTrA: transversus abdominis contraction, SD: standard deviation, LMC: lumbopelvic motor control, ES: effect size.

* $P \leq 0.05$.

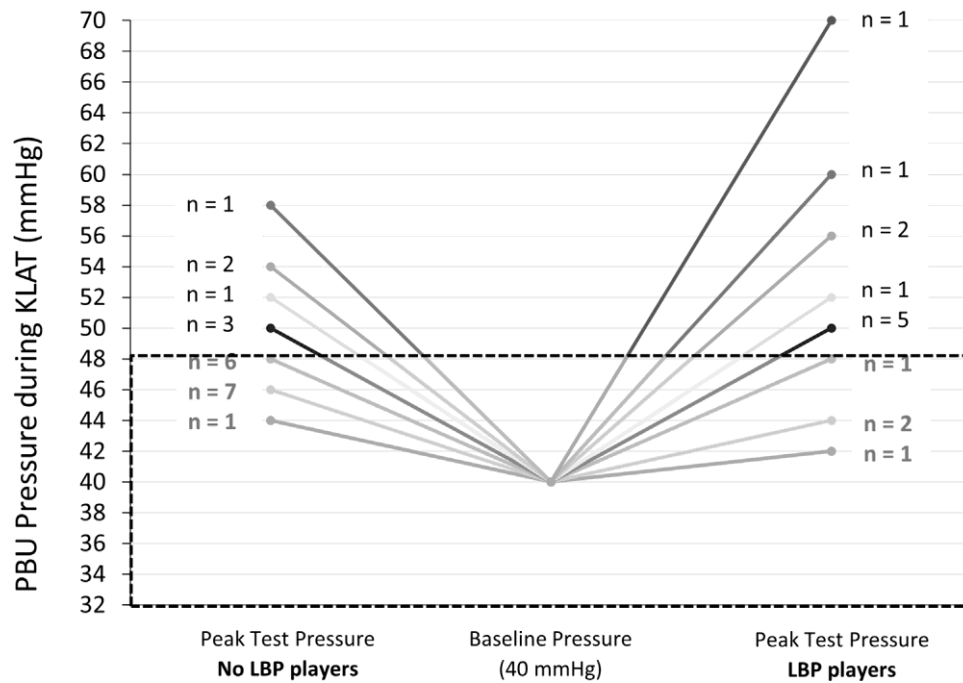


Figure 2 Changes in pressure (in mmHg) during the knee lift abdominal test (KLAT) for the 21 tennis players without a history of low back pain (no LBP players) and the 14 players with a history of low back pain (LBP players). Notes: PBU, pressure biofeedback unit.

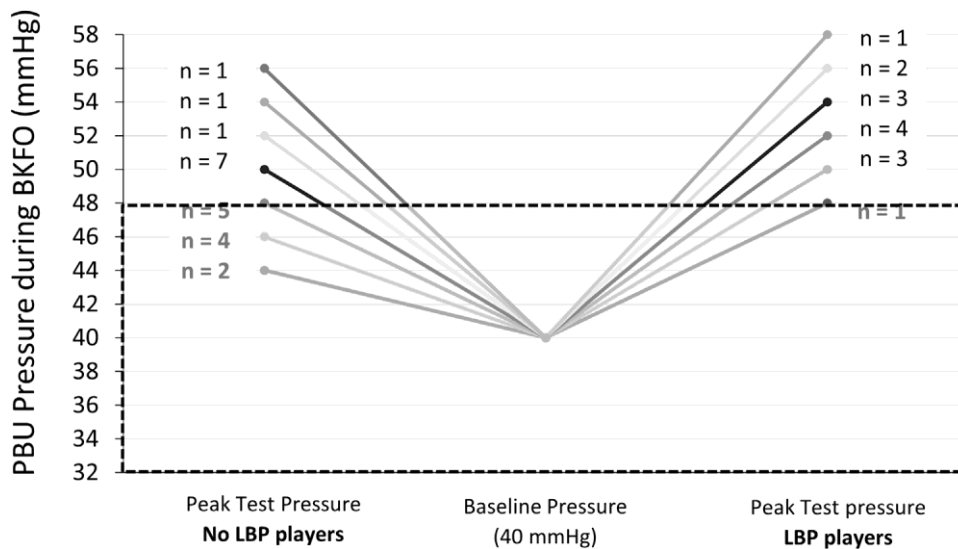


Figure 3 Changes in pressure (in mmHg) during the bent knee fall out (BKFO) for the 21 tennis players without a history of low back pain (no LBP players) and the 14 players with a history of low back pain (LBP players). Notes: PBU, pressure biofeedback unit.

For the KLAT, all the players demonstrated an increase in pressure. Regarding peak test pressure, 14 players (67%) without a history of LBP kept pressure in the tolerated range (between 32 and 48 mmHg) versus four players (29%) with a history of LBP (**Fig. 2**). For the BKFO, all the players also demonstrated an increase in pressure. Regarding peak test pressure, 11 players (52%) without a history of LBP kept pressure in the tolerated range (between 32 and 48 mmHg) versus one player (7%) with a history of LBP (**Fig. 3**).

4. DISCUSSION

The results of the present study confirmed that LBP is common in tennis players and that most tennis players with a history of LBP have altered motor control of the lumbopelvic region in comparison to players without a history of LBP.

Tennis practice involves repetitive trunk multidirectional movements and LBP among both recreational and professional tennis players can interfere with training, practice and competition [9]. In accordance with previous studies [8,9], we observed that 40% of the elite players suffered from disabling LBP during the previous year. Whereas acute injuries commonly occur in the lower extremity [30], two prospective studies of adolescent tennis players performed over a period of two to six years, showed that most chronic and recurrent pain involve the back [31,32].

Although risk factors of LBP are multi-factorial and as for the general population, they concern various bio-psycho- social aspects [11], delayed trunk muscle reflex responses were identified as a significant predictor of low back injury in athletes [18]. Spinal motor control appears to be an important issue in patients and athletes with recurrent LBP [17,33]. In the current cross-sectional study, LMC was assessed by means of a battery of five field tests. Previous research concluded that these tests allow to discriminate soccer players with and without a history of LBP [27]. On average, tennis players with a history of LBP were less optimal in the different tests than the players without a history of LBP. The large ES between the groups points out a substantial difference in movement control between group of players with and without a history of LBP. The cross-sectional design of the present study does not allow for making causative interpretations between motor control changes and LBP. However, previous studies discussed the cause-effect relationship between motor control and LBP. Research that introduced experimental nociceptive input and lesions suggest that many of the differences between patients and healthy individuals arise from the presence of pain and/or injury [34,35]. Lumbopelvic pain seems associated with a vast array of changes in activity of the trunk muscles [36]. Several studies indicate that changes in motor control persist after the resolution of symptoms in participants with a history of LBP, but no pain at the time of testing [18,26]. This implies that motor strategies do not resolve spontaneously after cessation of pain, at least in some individuals [36]. On the other hand, while delayed trunk muscles responses after mechanical perturbations can be elicited by experimentally induced pain [37], similar changes have been observed to precede LBP and increase LBP risk [18]. Motor control seems thus both a cause and an effect of pain and injury [38].

A large proportion of tennis players with a history of LBP was unable to adequately contract their transversus abdominis muscle, and these players demonstrated higher pressure deviations on the pressure biofeedback unit during the KLAT and the BKFO. Reduced ability to efficiently contract the TrA has been previously reported in elite cricketers [24], elite Australian Football League players

[25], dancers [26] and soccer players [27] with current or past LBP. Impaired control of TrA muscle in participants with LBP might result from changes in cortical excitability, as well as modification of the representation of the abdominal muscles on the motor cortex [33]. Indeed, Tsao et al. (2008) demonstrated, using transcranial magnetic imaging, a posterolateral shift in motor cortical representation of the TrA in a recurrent LBP sample [33]. The extent of this shift was related to the magnitude of the delay of activation of transversus abdominis muscle, which was itself related to the inability to achieve an isolated contraction of the TrA muscle [39].

The KLAT and the BKFO are dissociation tests, which investigate the players' ability to control movement of lumbopelvic region while performing simple movements in the hips. The unilateral leg movements performing during the KLAT and the BKFO are considered to be low load [40,41]. Previous studies showed that the reliability of the two tests is acceptable [19,41]. Jull et al. (1993) suggested that 'excessive' pressure changes during low load exercise reflect inability to maintain isometric contraction of the deep abdominal muscles, resulting in uncontrolled movement of the lumbar spine [40]. They did, however, not describe a cut-off of pressure changes. Using results of previous studies [19,27,29], we considered a change in the pressure level equal to or lower than 8 mmHg (either up or down) during the KLAT and the BKFO as successful completion. However, some other authors used a lower threshold than ours (i.e., 4 mmHg) to assess LMC of Swedish soldiers [41].

The mean change in pressure obtained for tennis players without a history of LBP for both KLAT (8.7 mmHg) and BKFO (8.8 mmHg) appears to be close to pressure variations obtained in cricket pace bowlers [29] and pre-professional dancers [19]. Regarding athletes with a history of LBP, a significant higher proportion of players failed the two tests. Moreover, tennis players with a history of LBP demonstrated a significantly higher pressure excursion on the pressure biofeedback unit during the BKFO than the asymptomatic players. Similar results were observed in pre-professional dancers [26] and elite soccer players [27]. The failure of the test could result from an inadequate coordination between the trunk muscles which control trunk rotation (oblique muscles of the abdomen, psoas and multifidus), and the hip muscles which, by their eccentric contraction, slow down the abduction/external rotation of the hip [42].

Previous studies showed that the ability to reduce lumbopelvic motion during limb movement tests may be influenced by gender [43–46]. Men demonstrated significantly greater and earlier lumbopelvic motion than women [44–46]. Possible explanations for these differences in lumbopelvic movement patterns between genders include differences in passive limb tissue stiffness, tissue extensibility, patterns of muscle recruitment, muscle strength, and anthropometric characteristics [43,44]. Although the percentage of women did not differ significantly between groups, most players without a history of LBP (86%) are females; thereby it might have influenced the LMC score. However, the average LMC score of tennis players without a

history of LBP in the present study (3.3 ± 0.8) is similar to average LMC score of male soccer players without a history of LBP assessed in a previous study (3.3 ± 1.3) [27].

To our knowledge, the present study is the first to report a difference in LMC between elite tennis players with and without a recent (last 12 months) disabling low back episode. Trunk muscles provide stability and balance when performing movements with the extremities and allow production, transfer, and control of force and motion to the terminal segment [47]. Therefore, motor control of the trunk and lumbopelvic region is essential during tennis training and competition. Interestingly, some players without a history of LBP also failed in some LMC tests. Further research is necessary to

determine whether correcting this dysfunction could reduce the occurrence of (new) LBP episodes in elite tennis players.

Although this study has reached its aims, the findings have to be seen in light of some limitations. First, it was conducted on a specific group of participants, i.e., on young elite tennis players.

Secondly, although the sample size in the current study was similar to other studies conducted on elite athletes [24,26] and is based on a sample size calculation, it might be considered as limited. Therefore, to generalize the results for larger groups, the study should have involved more players practicing at different levels (i.e., recreative adult players). Finally, considering the present study is based on a cross-sectional design, causative interpretations cannot be made.

5. CONCLUSION

The present study confirms that LBP is common in tennis players and has pointed out an impaired motor control of the lumbopelvic region in most tennis players with a history of LBP. The battery of five field tests used in this study had the potential to discriminate between tennis players with and without a history of LBP. Difference was particularly marked for three out of five LMC tests (cTrA, BKFO and KLAT). Further research should try to determine whether impaired lumbopelvic motor control is the origin or the consequence of the low back pain episodes and whether correcting this dysfunction could reduce the occurrence of (new) low back pain episodes in elite tennis players.

FUNDING

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

DISCLOSURE OF INTEREST

The authors declare that they have no competing interest.

ACKNOWLEDGEMENTS

The authors would like to express their gratitude to the tennis academies and players who participated in the study.

REFERENCES

- [1] International tennis federation: history of the ITF. [Accessed 2020 October 1]. Available from; <https://www.itftennis.com/about/organisation/>.
- [2] Gescheit D, Cormack S, Duffield R, et al. Injury epidemiology of tennis players at the 2011—2016 Australian Open Grand Slam. *Br J Sports Med* 2017;51(17):1289—94.
- [3] Di Giacomo G, Gasperis N, Costantini A. Tennis: epidemiology and injury mechanism. In: Volpi P, editor. *Arthroscopy and sport injuries: applications in high-level athletes*. Cham: Springer; 2016. p. 19—23.
- [4] Fett D, Trompeter K, Platen P. Back pain in elite sports: a cross-sectional study on 1114 athletes. *PLoS One* 2017;12(6):e0180130.
- [5] Fett D, Trompeter K, Platen P. Prevalence of back pain in a group of elite athletes exposed to repetitive overhead activity. *PLoS One* 2019;14(1):e0210429.
- [6] Johansson F, Gabbett T, Svedmark P, Skillgate E. External training load and the association with back pain in competitive adolescent tennis players: results from the SMASH cohort study. *Sports Health* 2022;14(1):111—8.
- [7] Rogers H, Taylor L. Adolescent female tennis players: injury prevalence and prevention. *ITF Coaching Sport Sci Rev* 2021;29(83):16—20.
- [8] Vad V, Gebeh A, Dines A, Altchek D, Norris B. Hip and shoulder internal rotation range of motion deficits in professional tennis players. *J Sci Med Sport* 2003;6(1):71—5.
- [9] Marks M, Haas S, Wiesel S. Low back pain in the competitive tennis player. *Clin Sports Med* 1988;7(2):277—87.
- [10] Fiani B, Jarrah R, Wong A, et al. Repetitive traumatic discopathy in the modern-era tennis player. *Cureus* 2020;12(8):e9783, <http://dx.doi.org/10.7759/cureus.9783>.
- [11] Hartvigsen J, Hancock M, Kongsted A, et al. What low back pain is and why we need to pay attention. *Lancet* 2018;391(10137):2356—67.
- [12] Campbell A, Straker L, O'Sullivan P, Elliott B, Reid M. Lumbar loading in the elite adolescent tennis serve: link to low back pain. *Med Sci Sports Exerc* 2013;45(8):1562—8.
- [13] Campbell A, O'Sullivan P, Straker L, Elliott B, Reid M. Back pain in tennis players: a link with lumbar serve kinematics and range of motion. *Med Sci Sports Exerc* 2014;46(2):351—7.
- [14] Renkawitz T, Boluki D, Grifka J. The association of low back pain, neuromuscular imbalance, and trunk extension strength in athletes. *Spine J* 2006;6(6):673—83.
- [15] Grosdent S, Demoulin C, Souchet M, Tomasella M, Crielaard J-M, Vanderthommen M. Trunk muscle profile in elite tennis players with and without low back pain. *J Sports Med Phys Fitness* 2015;55(11):1354—62.
- [16] Jull G, Richardson C. Motor control problems in patients with spinal pain: a new direction for therapeutic exercise. *J Manipulative Physiol Ther* 2000;23(2):115—7.
- [17] Hodges P. The role of the motor system in spinal pain: implications for rehabilitation of the athlete following lower back pain. *J Sci Med Sport* 2000;3(3):243—53.
- [18] Cholewicki J, Silfies S, Shah R, et al. Delayed trunk muscle reflex responses increase the risk of low back injuries. *Spine* 2005;30(23):2614—20.
- [19] Roussel N, Nijs J, Mottram S, Van Moorsel A, Truijen S, Stassijns G. Altered lumbopelvic movement control but not generalized joint hypermobility is associated with increased injury in dancers. a prospective study. *Man Ther* 2009;14(6):630—5.
- [20] Hides J, Stanton W, McMahon S, Sims K, Richardson C. Effect of stabilization training on multifidus muscle cross-sectional area among young elite cricketers with low back pain. *J Orthop Sports Phys Ther* 2008;38(3):101—8.
- [21] Hides J, Stanton W, Freke M, Wilson S, McMahon S, Richardson C. MRI study of the size, symmetry and function of the trunk muscles among elite cricketers with and without low back pain. *Br J Sports Med* 2008;42(10):509—13.
- [22] Galambos S, Terry P, Moyle G, Locke S, Lane A. Psychological predictors of injury among elite athletes. *Br J Sports Med* 2005;39(6):351—4.
- [23] Heidari J, Mierswa T, Kleinert J, et al. Parameters of low back pain chronicity among athletes: associations with physical and mental stress. *Phys Ther Sport* 2016;21:31—7.
- [24] Hides J, Stanton W, Wilson S, Freke M, McMahon S, Sims K. Retraining motor control of abdominal muscles among elite cricketers with low back pain. *Scand J Med Sci Sports* 2010;20(6):834—42.
- [25] Hides J, Hughes B, Stanton W. Magnetic resonance imaging assessment of regional abdominal muscle function in elite AFL players with and without low back pain. *Man Ther* 2011;16(3):279—84.

- [26] Roussel N, De Koning M, Schutt A, et al. Motor control and low back pain in dancers. *Int J Sports Med* 2013;34(2):138—43.
- [27] Grosdent S, Demoulin C, Rodriguez de La Cruz C, et al. Lumbopelvic motor control and low back pain in elite soccer players: a cross-sectional study. *J Sports Sci* 2016;34(11):1021—9.
- [28] Takekawa K, Gonçalves J, Moriguchi C, Coury H, Sato T. Can a self-administered questionnaire identify workers with chronic or recurring low back pain? *Industrial Health* 2015;53(4):340—5.
- [29] Olivier B, Stewart A, Olorunju S, McKinnon W. Static and dynamic balance ability, lumbo-pelvic movement control and injury incidence in cricket pace bowlers. *J Sci Med Sport* 2015;18(1):19—25.
- [30] Pluim B, Staal J, Windler G, Jayanthi N. Tennis injuries: occurrence, aetiology, and prevention. *Br J Sports Med* 2006;40(5):415—23.
- [31] Hutchinson M, Laprade R, Burnett Q, Moss R, Terpstra J. Injury surveillance at the USTA boys' tennis championships: a 6-yr study. *Med Sci Sports Exerc* 1995;27(6):826—30.
- [32] Hjelm N, Werner S, Renstrom P. Injury profile in junior tennis players: a prospective two year study. *Knee Surg Sports Traumatol Arthrosc* 2010;18(6):845—50.
- [33] Tsao H, Galea M, Hodges P. Reorganization of the motor cortex is associated with postural control deficits in recurrent low back pain. *Brain* 2008;131(8):2161—71.
- [34] Zedka M, Prochazka A, Knight B, Gillard D, Gauthier M. Voluntary and reflex control of human back muscles during induced pain. *J Physiol* 1999;520(2):591—604.
- [35] Hodges P, Holm A, Hansson T, Holm S. Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. *Spine* 2006;31(25):2926—33.
- [36] Hodges P, Ferreira P, Ferreira M. Lumbar spine: treatment of motor control disorders. In: Magee D, Zachazewski J, Quillen W, Manske R, editors. *Pathology and intervention in musculoskeletal rehabilitation*. Saint Louis, Missouri: Elsevier; 2016. p. 520—60.
- [37] Boudreau S, Farina D, Kongstad L, et al. The relative timing of trunk muscle activation is retained in response to unanticipated postural-perturbations during acute low back pain. *Exp Brain Res* 2011;210(2):259—67.
- [38] Van Dieën J, Reeves N, Kawchuk G, Van Dillen L, Hodges P. Motor control changes in low back pain: divergence in presentations and mechanisms. *J Orthop Sports Phys Ther* 2019;49(6):370—9.
- [39] Hodges P, Richardson C, Jull G. Evaluation of the relationship between laboratory and clinical tests of transversus abdominis function. *Physiother Res Int* 1996;1(1):30—40.
- [40] Jull G, Richardson C, Toppenberg R, Comerford M, Bui B. Towards a measurement of active muscle control for lumbar stabilisation. *Aust J Physiother* 1993;39(3):187—93.
- [41] Monnier A, Heuer J, Norman K, Ang B. Inter- and intra-observer reliability of clinical movement-control tests for marines. *BMC Musculoskelet Disord* 2012;13(1):263.
- [42] Comerford M, Mottram S. *Kinetic control: the management of uncontrolled movement*. Chatswood, NSW: Elsevier; 2012.
- [43] Gombatto S, Collins D, Sahrman J, Engsberg J, Van Dillen L. Gender differences in pattern of hip and lumbopelvic rotation in people with low back pain. *Clin Biomech* 2006;21: 263—71.
- [44] Scholtes S, Van Dillen L. Gender-related differences in prevalence of lumbopelvic region movement impairments in people with low back pain. *J Orthop Sports Phys Ther* 2007;37: 744—53.
- [45] Hoffman S, Johnson M, Zou D, Van Dillen L. Sex differences in lumbopelvic movement patterns during hip medial rotation in people with chronic low back pain. *Arch Phys Med Rehabil* 2011;92:1053—9.
- [46] Hoffman S, Johnson M, Zou D, Van Dillen L. Gender differences in modifying lumbopelvic motion during hip medial rotation in people with low back pain. *Rehabil Res Pract* 2012;2012:635312—7.
- [47] Kibler W, Press J, Sciascia A. The role of core stability in athletic function. *Sports Med* 2006;36(3):189—98.