

1    **Abstract**

2    Assessment of player's postural control following a lower limb injury is of interest to sports  
3    medicine practitioners due to its fundamental role in daily tasks and sporting activities. The  
4    aim was to longitudinally monitor professional rugby union players 'postural control during  
5    each phase of the rehabilitation programme (acute, middle, late) following a lower limb  
6    injury. Seven male rugby union players (height  $1.80 \pm 0.02$  m; mass  $100.3 \pm 11.4$  kg; age  $24$   
7     $\pm 4$  years) sustained a time loss, non-contact lower limb injury. Static postural control was  
8    assessed via sway path (m) and dynamic postural control was assessed via vertical postural  
9    stability index (VPSI). Group differences ( $p < 0.05$ ) were reported across the acute, middle,  
10   and late phase. Smaller magnitudes of sway path were observed for eyes-open sway path, and  
11   for the middle and late phase smaller magnitudes of VPSI ( $p < 0.05$ ) at the at end session  
12   compared to first session. Whereas larger magnitudes of VPSI were found between baseline  
13   and the last session ( $p < 0.05$ ). Large inter and intra-individual variation was apparent across  
14   the three phases of rehabilitation. Postural control improvements were identified during  
15   rehabilitation. However, postural control did not return to baseline, with altered kinetics  
16   throughout each rehabilitation phase.

17

18    **Keywords:** *RTP, static, dynamic, sway path and vertical postural stability index*

19

20    **Word Count: 4373**

21

## Introduction

22 In rugby union, lower limb injuries account for a higher injury incidence and burden  
23 compared to upper limb injuries<sup>1-3</sup>. The majority occur due to a non-contact mechanism,  
24 predominantly during running, change of direction (e.g., side stepping, cutting) and jump  
25 landing manoeuvres<sup>4,5</sup>. How such movements are executed ('movement pattern') as well as  
26 their ability to control their centre of mass ('postural control') may predispose players to  
27 injury<sup>5,6</sup>. Following a non-contact lower limb injury, athletes may alter their movement  
28 pattern, whilst maintaining pre-injury levels of performance<sup>7-9</sup>. Therefore, using performance  
29 metrics, such as jump height, alone likely overestimates an athlete's rehabilitation status<sup>10</sup>.

30 During rehabilitation, practitioners should focus on a player's injured structure,  
31 postural control, static vector alignment and movement pattern<sup>11</sup>. In particular, assessing an  
32 athlete's postural control during rehabilitation is important as it measures an athlete's ability  
33 to maintain, achieve or restore a state of balance during any posture or movement<sup>12</sup>.  
34 Maintaining postural stability requires the integration of sensory information and execution  
35 of appropriate motor responses<sup>13</sup>. However, practitioners typically overlook the importance of  
36 postural control assessments during rehabilitation, instead focussing on an athlete's  
37 movement pattern during the first landing of a drop jump and/or change of direction  
38 manoeuvres.

39 Postural control can be evaluated using both static and dynamic functional  
40 assessments<sup>14-16</sup>, thus allowing investigation of changes to a player's sensorimotor control  
41 throughout the course of their rehabilitation until their return to play (RTP). This  
42 multidimensional system (sensorimotor) encompasses the interaction of visual, vestibular,  
43 and somatosensory systems providing neural feedback via proprioception to the central  
44 nervous system (CNS) to maintain posture.

45           It is proposed that the rehabilitation process should be divided into three phases  
46 (acute, middle and late), to follow the progressive nature of rehabilitation and the associated  
47 kinetic assessments that are measured in each phase of RTP<sup>11</sup>. Early assessments of static  
48 postural control have been thought to highlight any sensorimotor disparities and ensure they  
49 are accounted for throughout the rehabilitation programme<sup>17-20</sup>. During the earlier stages of  
50 rehabilitation, assessments of postural control are typically measured through static postural  
51 control assessments, and are suggested to highlight any sensorimotor disparities (e.g., larger  
52 centre of pressure (CoP) sway path)<sup>17-20</sup>. Static postural control, however, has been suggested  
53 to not represent the functional demands of sports such as rugby union. Although, research has  
54 found athletes who demonstrate better postural control in the early stage of rehabilitation  
55 predict an athlete's recovery during the later phase of RTP<sup>20</sup> and static postural control is  
56 associated with more complex biomechanical assessments such as landing or jumping<sup>20-22</sup>.  
57 Static postural control is deemed the underpinning of all human movement, with it being  
58 related to athletes' progression throughout rehabilitation (during the middle-to-late  
59 rehabilitation phase) following injury.

60           Dynamic postural control is typically measured using the dynamic postural stability  
61 index (DPSI; Wikstrom et al. 2005). The DPSI quantifies the ability of an athlete to maintain  
62 control when transitioning from a dynamic to static state (e.g., landing a jump), and has been  
63 suggested to better represent the functional demands of multiplanar sports than solely  
64 assessing static postural control<sup>15</sup>. Specifically, the DPSI has been proposed to reflect  
65 player's ability to decelerate their centre of mass (CoM) upon landing<sup>22</sup>. Assessments of  
66 dynamic postural control provide practitioners with an objective measure of an athlete's  
67 response to perturbation of the CoM and provides additional insight that static assessments  
68 alone cannot provide. Furthermore, DPSI is thought to be associated with the peak vertical  
69 force that individuals must dissipate upon landing<sup>22</sup>. Higher DPSI scores have been found in

70 athletes at the point of RTP following a lower limb injury when assessed through uniplanar  
71 and multiplanar assessments compared to controls and uninjured limbs<sup>16,23</sup>. Moreover, higher  
72 vertical postural stability index (VPSI) scores have been found in runners with hip, thigh,  
73 knee, ankle and foot injuries compared to uninjured runners, suggesting it is the attenuation  
74 of vertical as opposed to anterior-posterior and medial-lateral forces that are affected  
75 following a lower-limb injury<sup>24</sup>. This may imply that there are central mediated changes  
76 following an injury, with the sensorimotor system being unable to adapt to the required  
77 vertical ground reactions forces experienced during landing and deceleration of the CoM.  
78 Furthermore, non-linear dynamics theory may provide additional support for these central  
79 mediated changes, as following an injury there are reduced degrees of freedom in an athlete's  
80 motor program to adapt to the change in the situation and neuromuscular feedback  
81 impairment results<sup>25</sup>.

82 To the authors knowledge, no study has longitudinally assessed postural control  
83 across the entire rehabilitation period through to point of RTP in any playing level.  
84 Prospective longitudinal investigation of professional athletes provides practitioners with a  
85 comprehensive understanding of athlete responses to medically supervised and guided  
86 rehabilitation following a lower limb injury. Therefore, the aim of this study was to  
87 longitudinally monitor professional rugby union players' postural control during three phases  
88 of the rehabilitation programme (acute, middle, late) following a lower limb injury. It was  
89 hypothesised that there would be initial deficits in players static and dynamic assessment of  
90 postural control compared to baseline, however over the phases of rehabilitation there would  
91 be improvements in static and dynamic postural control.

## 92 **Methods**

93           Participants: A season long prospective funded field-based applied study was  
94 conducted. The final cohort of injured players were seven male rugby union players (height  
95  $1.80 \pm 0.02$  m; mass  $100.28 \pm 11.38$  kg; age  $24 \pm 4$  years) from a professional rugby union  
96 team based in South Wales, UK. Each player had sustained a minimum of 14-days' time loss  
97 non-contact lower limb injury<sup>26</sup>. To overcome the fundamental issues of the small sample  
98 size due to resource constraints<sup>27</sup>, a compromise power analysis was calculated (GPower;  
99 version 3.1.9.7) ensuring power was met at 0.8, with an alpha level of 0.05 and medium  
100 effect size of 0.6. Effect sizes were computed to assess the relevance of differences between  
101 testing sessions. Ethical approval was obtained from the University Institutional Review  
102 Board.

### 103           Protocol

104           Phases of RTP: Pre-injury baseline data were collected for all players within the team  
105 (n = 37) during pre-season testing. Upon sustaining an injury all players received an  
106 individual rehabilitation plan following their professional medical diagnosis from the same  
107 practitioner who was the medical lead of the RTP program. The nature of this environment  
108 meant that we could control for 'safe' progression of players with supervised and guided  
109 rehabilitation. All players sustained a lower limb injury whilst playing for the professional  
110 team during the same playing season. The same medical lead of the RTP created individual  
111 rehabilitation programme for all players. Immediately following injury, players entered a  
112 RTP programme comprising three phases (acute, middle, late; Figure 1). The progression of  
113 players to the subsequent phase of RTP (acute to middle, middle to late, late to RTP) was an  
114 informed decision between relevant stakeholders (e.g., medical performance manager,  
115 medical lead of the RTP program and the strength and conditioning RTP coach). Postural  
116 control was assessed during each phase. Players were tested weekly to determine their  
117 readiness to progress to the next stage of the RTP programme, and this helped to determine

118 the first and last testing session within each rehabilitation phase. Subsequently, data recorded  
119 during the first and last session of each rehabilitation phase were used in analysis. All players  
120 received an individual rehabilitation programme designed by the medical lead of the RTP  
121 programme. The progression of players through the phases of RTP (Figure 1) was a shared  
122 decision between the medical performance manager, medical lead of the RTP programme and  
123 strength and conditioning RTP coach.

124 \*\*\*FIGURE 1 HERE\*\*\*

125 Injury detail: The knee was the most commonly injured body area (n = 4), followed  
126 by the hip (n = 2) and then the ankle (n = 1). Ligament injuries accounted for 57% of all  
127 lower limb injuries and muscle accounted for the remaining 43%. The mean and median  
128 duration of player's time in each phase can be found in Table 1. One physician from the team  
129 was responsible for recording and reporting all injury details. Injury records were checked for  
130 missing data by an independent researcher.

131 \*\*\*TABLE 1 HERE\*\*\*

132 Postural control assessments: In the acute phase, unilateral postural control was  
133 assessed under two conditions: eyes-open and eyes-closed<sup>28</sup>. Data were collected using a  
134 PASCO dual axis force plate (PS-2142; 1000 Hz). Each trial was 20 seconds in duration,  
135 interspersed with 30 second rest between trials. Players were instructed to stand on their  
136 injured limb, place their hands on their hips, flex (90 degrees) their contralateral non weight  
137 bearing limb at the knee and to look straight ahead for both conditions. Players were  
138 informed that should they come out of this starting position, they should regain it as soon as  
139 possible as the trial would not be stopped.

140 During the middle phase players performed a unilateral drop jump from 20 cm<sup>29</sup> onto  
141 a PASCO single axis force plate (PS-2141; 1000 Hz). Players were instructed to stand upright

142 with their hands on their hips, rolled off the injured leg to land on the injured leg, and once  
143 they hit the floor to jump as high as they can, whilst spending as little time as possible on the  
144 force plate. Players had 30 seconds rest between trials. Prior to data collection the force plate  
145 was calibrated according to manufacturer specifications, and prior to each test the force plate  
146 was zeroed.

147         During the late phase a lateral hurdle hop was performed on PASCO single axis force  
148 plate (PS-2141; 1000 Hz). Players were required to hop unilaterally over a 15 cm hurdle and  
149 immediately hop back to their initial starting position<sup>11</sup>. If testing their right leg, players were  
150 instructed to stand on their right foot to the left of the hurdle (on the left force plate), with the  
151 first hop being in a rightwards direction over the hurdle, and the second hop back in a  
152 leftwards direction to the original starting position. Each trial was interspersed with 1-minute  
153 rest periods.

154         Data analysis: All biomechanical data were processed using a customised written  
155 MATLAB script (Matlab R2019b). A 4th order, recursive low pass Butterworth filter with a  
156 cut-off frequency of 35 Hz for static postural control (acute phase) and 25 Hz for dynamic  
157 postural control assessments (middle and late phase) determined by residual analysis. Sway  
158 path was calculated for the acute phase static postural control assessments as the total  
159 distance of the CoP trajectory<sup>30</sup> during the final 5 seconds of each trial to measure the static  
160 phase of static postural control. Within our laboratory, the test-retest reliability and  
161 concurrent validity of three trials has been shown to have high agreement and measurement  
162 precision when assessing the final 5 seconds of static unilateral postural control<sup>31</sup>. Additional  
163 analysis found the concurrent validity to have small differences illustrating heteroscedasticity  
164 between Kistler laboratory-grade and PASCO force plates (Additional file 1). The test-retest  
165 reliability observed moderate intraclass coefficient correlation (ICC) were observed for sway  
166 path across the interval for eyes open (ICC 0.60-0.81) and eyes closed (ICC 0.62-0.95).

167 During the middle and late phases, dynamic postural control was assessed (Wikstrom  
168 et al. 2005) using vertical force to determine the VPSI. The VPSI is a measure of the  
169 fluctuation from body weight to standardise the vertical ground reaction force of the landing,  
170 describing the attenuation of force upon landing<sup>15</sup>. The second landing of dynamic postural  
171 control assessments were analysed. Within our laboratory, the validity of the VPSI was  
172 assessed against the DPSI score for both dynamical postural control assessments used in the  
173 middle and late phases (unilateral drop jump and lateral hurdle hop, respectively), with a  
174 small bias being observed between the separate calculations (Additional file 2). The mean  
175 test-retest reliability displayed excellent ICC for the drop jump (ICC 0.94 (0.86-0.97) and  
176 moderate ICC for hurdle hop (ICC 0.79 (0.51-0.91)).

177 Statistical analysis: Means  $\pm$  SD of all three trials for each participant were computed.  
178 For statistical analysis the Shapiro-Wilk test were used to test for normality, for all variables.  
179 Simple, last category contrast analysis was used to compare player's responses between pre-  
180 injury baseline and the last session of each of the RTP phases, as well as between the first and  
181 last session of each RTP phase (acute, middle, late). Relative change (RC; %) were calculated  
182 as the difference between the first and last session of each phase, relative to the last session.  
183 The RC was also calculated between pre-injury baseline and the last acute session. Due to the  
184 small sample size Hedges' *g* effect size (*g*) was used to determine the magnitude of change  
185 and interpreted as small (*g* = 0.2–0.5), medium (*g* = 0.51–0.8) and large (*g* > 0.8; Hedges  
186 1981). The coefficient of variation (CV) for each variable within each phase was calculated  
187 ( $CV\% = \frac{\text{standard deviation}}{\text{group mean}} \times 100$ ) to assess the dispersion of players' response relative to the  
188 mean, being independent of the unit which the variable was calculated from. All statistical  
189 analysis was performed using SPSS (v.27.0), significance was set at  $p < 0.05$ .

190

## Results



191 In the acute phase (Figure 1), a shorter sway path was observed for the eyes-open  
192 condition in the last session ( $0.19 \pm 0.06$  m) compared to the first session ( $0.17 \pm 0.04$  m;  
193  $F(1) 11.88, p 0.01, \eta^2 0.66, g 0.81$ ; Figure 2). For eyes closed a larger sway path were  
194 observed for the last session ( $0.52 \pm 0.13$  m) compared to baseline ( $0.41 \pm 0.10$  m;  $F(1)$   
195  $19.28, p 0.01, \eta^2 19.38, g 0.75$ ; Figure 2).

196 When looking at the CVs across the three testing sessions (pre-injury baseline, the  
197 first and last testing session), static postural control individual player dispersion of CVs  
198 widen across all testing sessions. During eyes open, CV increased from baseline (21%) to  
199 first session (33%) and then decreased in the last session (25%). The first testing session had  
200 the largest intra-variability (CV 33%) compared to pre-injury baseline and the last testing  
201 session (CV 21% and 27% respectively). During eyes closed assessments, the CV dispersion  
202 for inter- variability was lowest at baseline (20%) and, increases in the first (24%) and last  
203 (26%) testing session. The intra-variability for eyes closed assessment showed the last testing  
204 session to have the largest variability within the acute phase (CV 26%) compared to the  
205 initial two testing sessions (24% and 20% respectively).

206 \*\*\*FIGURE 2 HERE\*\*\*

207 A larger VPSI was observed at the end session compared to the pre-injury baseline for  
208 drop jump (middle phase;  $F(1) 47.99, p < 0.001, \eta^2 0.89, g 2.72$ ; Table 2). The difference  
209 between RTP first and last testing sessions found VPSI to reduce over the sessions ( $F(1)$   
210  $47.99, p 0.05, \eta^2 0.89, g 0.74$ ). In the drop jump, all players had lower VPSI at pre-injury  
211 baseline compared to the last testing session (Table 3). In contrast, two out of seven players  
212 (001 and 004) increased their VPSI between the first and last testing session. The CV  
213 dispersion is highest at baseline (25%) and, decreases in the first (18%) and last (20%) testing

214 session. The intra-variability was the largest at players pre-injury baseline (CV 25%)  
215 compared to the first and last testing session (CV 18% and 20% respectively).

216 \*\*\*TABLE 2 HERE\*\*\*

217 Similar to the middle phase, VPSI during the hurdle hop (late phase) was lower in the  
218 pre-injury baseline compared to the last session ( $F(1) 32.47, p 0.001, \eta^2 0.84, g 0.94$   
219 respectively; Table 2). Additionally, a smaller VPSI was observed between first and last  
220 testing sessions ( $F(1) 7.69, p 0.03, \eta^2 0.56, g 1.67$ ). The variability between players kinetic  
221 strategy for the lateral hurdle hop shows all of seven players increased VPSI between the  
222 baseline and last testing session (Table 3), with CV dispersion increasing from pre-injury  
223 baseline (41%) to first session (63%) and then decreasing in the last session (23%). The intra-  
224 variability between players movement pattern for the lateral hurdle hop was the smallest  
225 during the last testing session (CV 23%) compared to preinjury baseline and the first testing  
226 session (CV 41% and 63%).

227 \*\*\*TABLE 3 HERE\*\*\*

## 228 **Discussion**

229 The aim of this study was to characterise the longitudinal alterations in postural  
230 control throughout rehabilitation following lower limb injury in professional rugby union  
231 players. At a group level there was a significant difference ( $p < 0.05$ ) in players last testing  
232 session across each phase compared to pre-injury baseline, suggesting players had not  
233 returned to pre-injury postural control levels. However, players' postural control did improve  
234 over the course of rehabilitation as there were significant improvements between the first to  
235 last testing sessions of each phase. Inter-individual variation was relatively unchanged during  
236 early and middle rehabilitation but was reduced during the late rehabilitation phase.

237           Within the acute phase of rehabilitation at a group level a larger sway path was  
238 observed for the static postural control assessment for the eyes-closed condition between pre-  
239 injury baseline and end testing session. A larger sway path may suggest a reduced static  
240 postural control ability, and may be explained by the disruption in afferent signals from the  
241 mechanoreceptors following a lower limb injury<sup>32</sup>. Following an injury, mechanoreceptors  
242 have been found to inhibit postural control due to being unable to actively change the tension  
243 of the joint<sup>33-35</sup>. Moreover, it is important to consider the lack of familiarity in this task,  
244 leading to a decreased static postural control (as seen through larger sway path). Conversely,  
245 for eye-open conditions there were improvements between the first to end sessions of the  
246 acute rehabilitation phase. Whilst this study was the first to consider static postural control  
247 improvements in the acute phase of RTP, other studies have shown similar improvements in  
248 the late phase of RTP<sup>36</sup>. These improvements could be associated with rehabilitation targeting  
249 deficits into the somatosensory system specifically mechanoreceptors and proprioceptors<sup>37-39</sup>.  
250 Furthermore, these improvements may further suggest that the individualised rehabilitation  
251 programme each player undertook in the acute phase of RTP focused on targeting the  
252 relevant aspects of the efferent components of the neural system<sup>40</sup>. This may, therefore, allow  
253 improvements in the transmission of sensory information, regarding joint position,  
254 movement, and strain, through afferent pathways to the CNS<sup>41</sup>. These findings support  
255 previous research that has shown that increasing task complexity, such as eyes-closed  
256 conditions, leads to larger magnitudes of change in postural control (e.g., larger sway path)  
257 than simpler tasks, such as eyes-open conditions<sup>42</sup>.

258           Five out of seven players displayed a larger sway path in the last session of the acute  
259 phase compared to pre-injury baseline for eyes-closed assessments. Additionally, there is a  
260 larger dispersion in players' response during the last testing session compared to pre-injury  
261 baseline. This could suggest that individual player responses differ as the trial progresses and

262 suggests the strategy players employ to ensure their CoM remains above the base of support  
263 varies between players<sup>43</sup>. The larger dispersion between players may indicate that at the end  
264 of the acute phase of RTP, there are larger inconsistencies in the way players execute the  
265 static postural control trial. A possible explanation for this could be that following an injury  
266 the alterations occur to players sensorimotor system and their ability to control their CoM  
267 likely varies<sup>38,44</sup>. Within practice the assessment of dynamic postural control may provide  
268 practitioners with the tool to determine the aberrant landing mechanisms. This may suggest  
269 persistent deficits to players' neurosensory characteristics, through an inability to control and  
270 stabilise themselves on landing, and as a result of the inability to absorb and dissipate kinetic  
271 energy during impact<sup>16</sup>. During the middle and late phase, the assessment of dynamic  
272 postural control show group level differences between testing sessions. Improvements were  
273 evident across the two phases between the first and last testing session of each phase, during  
274 the middle phase five players and all players in the late phase decrease their VPSI. These  
275 findings likely suggest that across the middle and late phases, players have a greater  
276 capability to decelerate their CoM following reactive rebound uniplanar and multiplanar  
277 biomechanical assessments. This could imply that when comparing to the first testing session  
278 of the middle and late phase of rehabilitation the mechanoreceptors are able to actively  
279 change the tension of the joint prior to landing<sup>35,45</sup>. Findings could also infer an improvement  
280 in a players' landing strategy to ensure they are able to actively change the tension of the  
281 joint in order to react efficiently to the base of support displacements or to external  
282 mechanical stimuli.

283           During the middle phase, P1 demonstrated the opposite kinetic response when  
284 performing the uniplanar dynamic movement and P4 showed no improvement, which may be  
285 harmful as VPSI is an indicator of a player's interaction of neurophysiological,  
286 biomechanical, and motor control<sup>38</sup>. The difference in VPSI for P1 likely infers a control

287 strategy alteration, whereby there are changes in the sensorimotor system facilitating players  
288 inability to dissipate force upon landing. This implies that there are central mediated changes  
289 following an injury with the sensorimotor system being unable to adapt to the required  
290 landing and deceleration of the CoM<sup>25</sup>. Therefore, it may be suggested that practitioners should  
291 routinely assess a player's dynamic postural control throughout the latter phases of their RTP.

292 In line with previous research, this study offers further support that negative  
293 alterations occur in players ability to control and stabilise themselves upon landing following  
294 a lower limb injury. Despite improvement across the middle and late phase, there were still  
295 group level deficits in players VPSI at the end of both phases when compared to players pre-  
296 injury baseline movement pattern. At an individual perspective all players displayed an  
297 increase in VPSI at the end of both phases, however the magnitude of this increase varied  
298 between players. This increase is indicative of a poorer/worse VPSI score and may suggest  
299 alterations in the neurosensory characteristics, possibly reflecting an inability for players to  
300 absorb kinetic energy during impact<sup>16</sup>. Therefore, although rehabilitation during the middle  
301 phase seemingly improved a player's ability to decelerate their CoM following reactive  
302 rebound biomechanical assessments, players did not return to their pre-injury baseline score.  
303 This supports previous research that has observed deficits in biomechanical assessments at  
304 the point of RTP<sup>46-48</sup>, meaning players' control during landing linear movements were not  
305 adequately addressed in the middle and late phase of rehabilitation. As such, these findings  
306 suggest that there is a need to quantify landing kinetics during the later phase of RTP,  
307 ensuring that players' rehabilitation programmes aim to restore their ability to control their  
308 CoM on landing and are assessed through lateral biomechanical assessments and prior to the  
309 point they are cleared to RTP. Therefore, it is advisable that practitioners should look to use  
310 dynamic assessments of postural control throughout an athlete's rehabilitation as the findings  
311 reinforce that landing is a complex action, requiring dynamic resistance from structures to

312 withstand the forces experienced on landing and simultaneously enable rapid deceleration of  
313 the CoM. The aberrant landing mechanisms in this study suggest persistent deficits to  
314 players' neurosensory characteristics through an inability to control and stabilise themselves  
315 on landing, subsequently resulting in an inability to absorb and dissipate kinetic energy  
316 during impact.

317         This study is the first to characterise the group and individual postural control patterns  
318 throughout rehabilitation, showing the association injury has on the complex nature of  
319 players movement pattern and the varying degrees of freedom players have to execute the  
320 same movement following a lower limb injury. It appears that players sit along a continuum  
321 for movement pattern where their unique responses to cope with the consequences of a lower  
322 limb injury can be quantified. For example, there is variability in players responses to  
323 rehabilitation, although all players show deficits in VPSI at the end testing session during the  
324 dynamic postural control assessments there is varying magnitudes of differences. It could be  
325 inferred that despite similar magnitudes of VPSI between the middle and late phase, there is a  
326 larger dispersion in the late phase than the middle. Thus, this may imply that task complexity  
327 may influence the degeneracy that occurs<sup>49,50</sup>. Based on the self-organisation theory<sup>51</sup>  
328 individuals may use varying combinations of degrees of freedom to achieve the same  
329 outcome, in this case landing. For the middle phase, smaller dispersion are evident in VPSI  
330 compared to the late phase across all three testing assessments (pre-injury baseline, the first  
331 testing session, and the end testing session). These findings may suggest that as the  
332 complexity of assessment increases so does the variability between kinetic strategy. Once  
333 again it could be postulated that the kinetic strategy adopted is individual to each player<sup>52</sup>.  
334 Despite the individual nature of a player's response to RTP practitioners should routinely  
335 assess dynamic postural control, as VPSI may assist in the ability to detect changes in the  
336 sensorimotor system through prospective outcome-oriented investigations.

337 We acknowledge that there were several limitations in this study, first, although  
338 kinetic analyses were performed on the biomechanical assessments, only vertical ground  
339 reaction force was quantified during the acute, middle, and late phase of RTP, due to the  
340 ‘infield’ nature of the study. This limitation prevented the assessment of medio-lateral and  
341 anterior-posterior forces ( $F_x$  and  $F_y$ , respectively), that would have provided insight into the  
342 directional force that was being applied to the body. Secondly the results can only be applied  
343 to elite male rugby union players, meaning further research is required to examine if postural  
344 changes over the duration of the trial were observed in different populations. A final  
345 limitation of this study was the sample size due to resource constraints<sup>27</sup>, as this would have  
346 affected the power of the measurement and is therefore a likely reason for the moderate  
347 positive correlations being non-significant. However, as this is a prospective study, the lower  
348 sample size is typical due to the nature of data collection.

349 The study supports the existence of players independent response to rehabilitation  
350 following a lower limb injury, with alterations in players movement strategy sitting along a  
351 continuum, varying in the magnitude of change evident in the varying dispersion of player  
352 responses. In summary, the findings from the investigation highlight that in all phases of  
353 RTP, players alter their kinetic strategy to attain the same performance magnitudes from the  
354 first to end session. However postural control deficits are present at the end of each phase.  
355 Therefore, whilst players may not have returned to their pre-injury movement strategies, it is  
356 likely that they developed compensatory strategies to overcome this. This suggests that the  
357 prescribed rehabilitation programme fails to account for the factors that expose players to  
358 greater risk of injury occurrence. This suggests practitioners should incorporate investigation  
359 of static and dynamic postural control into their assessment of a player’s rehabilitation  
360 following lower limb injury. Due to the changes in the athlete’s motor program to adapt and  
361 subsequently receive neuromuscular feedback it may elicit players to a great risk of reinjury.

362           **Acknowledgements**

363   The Authors would like to thank Ospreys Rugby, Anthony Carter, Dr Leah Bitchell and  
364   Jennifer Baker for their help with data collection.

365           **References**

- 366   1.    Moore IS, Ranson C, Mathema P. Injury Risk in International Rugby Union: Three-  
367        Year Injury Surveillance of the Welsh National Team Isabel. *Orthop J Sport Med*.  
368        2015;3(7):1-9. doi:10.1177/2325967115596194
- 369   2.    Bitchell CL, Mathema P, Moore IS. Four-year match injury surveillance in male  
370        Welsh professional Rugby Union teams. *Phys Ther Sport*. 2020;42:26-32.  
371        doi:10.1016/j.ptsp.2019.12.001
- 372   3.    Williams S, Robertson C, Starling L, et al. Injuries in Elite Men’s Rugby Union: An  
373        Updated (2012–2020) Meta-Analysis of 11,620 Match and Training Injuries. *Sport*  
374        *Med*. 2022;52(5):1127-1140. doi:10.1007/s40279-021-01603-w
- 375   4.    Montgomery C, Blackburn J, Withers D, Tierney G, Moran C, Simms C. Mechanisms  
376        of ACL injury in professional rugby union: A systematic video analysis of 36 cases. *Br*  
377        *J Sports Med*. 2018;52(15):994-1001. doi:10.1136/bjsports-2016-096425
- 378   5.    Mohammadi F, Salavati M, Akhbari B, Mazaheri M, Khorrami M, Negahban H. Static  
379        and dynamic postural control in competitive athletes after anterior cruciate ligament  
380        reconstruction and controls. *Knee Surgery, Sport Traumatol Arthrosc*.  
381        2012;20(8):1603-1610. doi:10.1007/s00167-011-1806-4
- 382   6.    Jones HSR, Moore IS, King E, et al. Movement strategy correspondence across  
383        jumping and cutting tasks after anterior cruciate ligament reconstruction. *Scand J Med*  
384        *Sci Sport*. 2022;32(3):612-621. doi:10.1111/sms.14104



- 385 7. Read PJ, Michael Auliffe S, Wilson MG, Graham-Smith P. Lower Limb Kinetic  
386 Asymmetries in Professional Soccer Players With and Without Anterior Cruciate  
387 Ligament Reconstruction: Nine Months Is Not Enough Time to Restore “Functional”  
388 Symmetry or Return to Performance. *Am J Sports Med.* 2020;48(6):1365-1373.  
389 doi:10.1177/0363546520912218
- 390 8. Paterno M, Schmitt L, Ford K. Biomechanical measures during landing and postural  
391 stability predict second anterior cruciate ligament injury after anterior cruciate  
392 ligament reconstruction and return to sport. *Am J Sports Med.* 2010;38(10):1968-1978.  
393 doi:10.1177/0363546510376053.Biomechanical
- 394 9. Kotsifaki A, Korakakis V, Whiteley R, Van Rossom S, Jonkers I. Measuring only hop  
395 distance during single leg hop testing is insufficient to detect deficits in knee function  
396 after ACL reconstruction: A systematic review and meta-analysis. *Br J Sports Med.*  
397 2020;54(3):139-153. doi:10.1136/bjsports-2018-099918
- 398 10. King E, Franklyn-Miller A, Richter C, et al. Clinical and biomechanical outcomes of  
399 rehabilitation targeting intersegmental control in athletic groin pain: Prospective cohort  
400 of 205 patients. *Br J Sports Med.* 2018;52(16):1054-1062. doi:10.1136/bjsports-2016-  
401 097089
- 402 11. Pollock AS, Durward BR, Rowe PJ, Paul JP. What is balance? *Clin Rehabil.*  
403 2000;14(4):402-406. doi:10.1191/0269215500cr342oa
- 404 12. Willimas V, Nagai T, Sell TC, et al. Prediction of dynamic postural stability during  
405 single-leg jump landings by ankle and knee flexibility and strength. *J Sport Rehabil.*  
406 2016;25(3):266-272. doi:10.1123/jsr.2015-0001
- 407 13. Jonsson E, Seiger Å, Hirschfeld H. One-leg stance in healthy young and elderly adults:  
408 A measure of postural steadiness? *Clin Biomech.* 2004;19(7):688-694.

- 409 doi:10.1016/j.clinbiomech.2004.04.002
- 410 14. Wikstrom EA, Tillman MD, Smith AN, Borsa PA. A new force-plate technology  
411 measure of dynamic postural stability: The dynamic postural stability index. *J Athl*  
412 *Train.* 2005;40(4):305-309.
- 413 15. Heinert B, Willett K, Kernozek TW. Influence of Anterior Cruciate Ligament  
414 Reconstruction on Dynamic Postural Control. *Int J Sports Phys Ther.* 2018;13(3):432-  
415 440. doi:10.26603/ijsp20180432
- 416 16. Hewett TE, Myer GD, Ford KA, et al. Biomechanical measures of neuromuscular  
417 control and valgus loading of the knee predict anterior cruciate ligament injury risk in  
418 female athletes: A prospective study. *Am J Sports Med.* 2005;33(4):492-501.  
419 doi:10.1177/0363546504269591
- 420 17. Buckthorpe M, Della Villa F. Optimising the ‘Mid-Stage’ Training and Testing  
421 Process After ACL Reconstruction. *Sport Med.* 2020;50(4):657-678.  
422 doi:10.1007/s40279-019-01222-6
- 423 18. Paterno M V., Schmitt LC, Ford KR, Rauh MJ, Hewett TE. Altered postural sway  
424 persists after anterior cruciate ligament reconstruction and return to sport. *Gait*  
425 *Posture.* 2013;38(1):136-140. doi:10.1016/j.gaitpost.2012.11.001
- 426 29. Labanca L, Laudani L, Menotti F, et al. Asymmetrical Lower Extremity Loading Early  
427 after Anterior Cruciate Ligament Reconstruction Is a Significant Predictor of  
428 Asymmetrical Loading at the Time of Return to Sport. *Am J Phys Med Rehabil.*  
429 2016;95(4):248-255. doi:10.1097/PHM.0000000000000369
- 430 20. Ross SE, Guskiewicz KM, Yu B. Single-leg jump-landing stabilization times in  
431 subjects with functionally unstable ankles. *J Athl Train.* 2005;40(4):298-304.

- 432 21. Huurnink A, Fransz DP, Kingma I, de Boode VA, Dieën JH va. The assessment of  
433 single-leg drop jump landing performance by means of ground reaction forces: A  
434 methodological study. *Gait Posture*. 2019;73:80-85.  
435 doi:10.1016/j.gaitpost.2019.06.015
- 436 22. Wikstrom EA, Tillman MD, Chmielewski TL, Cauraugh JH, Naugle KE, Borsa PA.  
437 Discriminating between copers and people with chronic ankle instability. *J Athl Train*.  
438 2012;47(2):136-142. doi:10.4085/1062-6050-47.2.136
- 439 23. Meardon S, Klusendorf A, Kernozek T. Influence of Injury on Dynamic Postural  
440 Control in Runners. *Int J Sports Phys Ther*. 2016;11(3):366-377.
- 441 24. Wikstrom EA, Tillman MD, Chmielewski TL, Cauraugh JH, Naugle KE, Borsa PA.  
442 Dynamic postural control but not mechanical stability differs among those with and  
443 without chronic ankle instability. *Scand J Med Sci Sport*. 2010;20(1):e137-e144.  
444 doi:10.1111/j.1600-0838.2009.00929.x
- 445 25. Fuller CW, Molloy MG, Bagate C, et al. Consensus statement on injury definitions and  
446 data collection procedures for studies of injuries in rugby union. *Clin J Sport Med*.  
447 2007;17(3):328-331. doi:10.1097/JSM.0b013e31803220b3
- 448 26. Lakens D. Sample Size Justification. *Collabra Psychol*. 2022;8(1):1-28.  
449 doi:10.1525/collabra.33267
- 450 27. Troester JC, Jasmin JG, Duffield R. Reliability of single-leg balance and landing tests  
451 in rugby union; prospect of using postural control to monitor fatigue. *J Sport Sci Med*.  
452 2018;17(2):174-180.
- 453 28. King E, Richter C, Franklyn-Miller A, et al. Whole-body biomechanical differences  
454 between limbs exist 9 months after ACL reconstruction across jump/landing tasks.

- 455 *Scand J Med Sci Sport*. 2018;28(12):2567-2578. doi:10.1111/sms.13259
- 456 29. Prieto TE, Myklebust JB, Hoffmann RG, et al. Measures of postural steadiness:  
457 Differences between healthy young and elderly adults. *IEEE Trans Biomed Eng*.  
458 1996;43(9):956-966. doi:10.1109/10.532130
- 459 30. Mccarthy-Ryan MF. An Interdisciplinary Investigation Into Return To Play in  
460 Professional Rugby Union Following Lower Limb Injury. 2023. Cardiff Metropolitan  
461 Univeristy. Thesis. <https://doi.org/10.25401/cardiffmet.21828798.v1>
- 462 31. Courtney C, Rine RM, Kroll P. Central somatosensory changes and altered muscle  
463 synergies in subjects with anterior cruciate ligament deficiency. *Gait Posture*.  
464 2005;22(1):69-74. doi:10.1016/j.gaitpost.2004.07.002
- 465 32. Kapreli E, Athanasopoulos S, Gliatis J, et al. Anterior cruciate ligament deficiency  
466 causes brain plasticity: A functional MRI study. *Am J Sports Med*. 2009;37(12):2419-  
467 2426. doi:10.1177/0363546509343201
- 468 33. Negahban H, Mazaheri M, Kingma I, van Dieën JH. A systematic review of postural  
469 control during single-leg stance in patients with untreated anterior cruciate ligament  
470 injury. *Knee Surg Sport Traumatol Arthrosc*. 2014;22(7):1491-1504.  
471 doi:10.1007/s00167-013-2501-4
- 472 34. Lehmann T, Paschen L, Baumeister J. Single-Leg assessment of postural stability after  
473 anterior cruciate ligament injury: a systematic review and meta-analysis. *Sport Med -*  
474 *Open*. 2017;3(1):1-12. doi:10.1186/s40798-017-0100-5
- 475 35. Thomeé P, Währborg P, Börjesson M, Thomeé R, Eriksson BI, Karlsson J. Self-  
476 efficacy, symptoms and physical activity in patients with an anterior cruciate ligament  
477 injury: A prospective study. *Scand J Med Sci Sport*. 2007;17(3):238-245.

- 478 doi:10.1111/j.1600-0838.2006.00557.x
- 479 36. Grooms DR, Page SJ, Nichols-Larsen DS, Chaudhari AMW, White SE, Onate JA.  
480 Neuroplasticity associated with anterior cruciate ligament reconstruction. *J Orthop*  
481 *Sports Phys Ther.* 2017;47(3):180-189. doi:10.2519/jospt.2017.7003
- 482 37. Riemann BL, Lephart SM. The sensorimotor system, part I: The physiologic basis of  
483 functional joint stability. *J Athl Train.* 2002;37(1):71-79.
- 484 38. Hewett TE, Patterno M, Myer G. Strategies for Enhancing Proprioception and  
485 Neuromuscular Control of the Knee. *Clin Orthop Relat Res.* 2002;402(5):76-94.  
486 doi:10.1097/01.blo.0000026962.51742.99
- 487 39. Ingersoll CD, Grindstaff TL, Pietrosimone BG, Hart JM. Neuromuscular  
488 Consequences of Anterior Cruciate Ligament Injury. *Clin Sports Med.*  
489 2008;27(3):383-404. doi:10.1016/j.csm.2008.03.004
- 490 40. Pietrosimone BG, McLeod MM, Lepley AS. A theoretical framework for  
491 understanding neuromuscular response to lower extremity joint injury. *Sports Health.*  
492 2012;4(1):31-35. doi:10.1177/1941738111428251
- 493 41. Blosch C, Schäfer R, De Marées M, Platen P. Comparative analysis of postural control  
494 and vertical jump performance between three different measurement devices. *PLoS*  
495 *One.* 2019;14(9):1-16. doi:10.1371/journal.pone.0222502
- 496 42. Mackinnon CD. Sensorimotor anatomy of gait, balance, and falls. *Handb Clin Neurol.*  
497 2018;159(1):139-148. doi:10.1016/B978-0-444-63916-5.00001-X
- 498 43. Riemann BL, Lephart SM. The sensorimotor system, Part II: The role of  
499 proprioception in motor control and functional joint stability. *J Athl Train.*  
500 2002;37(1):80-84.

- 501 44. Kapreli E, Athanasopoulos S. The anterior cruciate ligament deficiency as a model of  
502 brain plasticity. *Med Hypotheses*. 2006;67(3):645-650.  
503 doi:10.1016/j.mehy.2006.01.063
- 504 45. Miles JJ, King E, Falvey EC, Daniels KAJ. Patellar and hamstring autografts are  
505 associated with different jump task loading asymmetries after ACL reconstruction.  
506 *Scand J Med Sci Sport*. 2019;29(8):1212-1222. doi:10.1111/sms.13441
- 507 46. Gore SJ, Franklyn-Miller A, Richter C, King E, Falvey EC, Moran K. The effects of  
508 rehabilitation on the biomechanics of patients with athletic groin pain. *J Biomech*.  
509 2020;99:109474. doi:10.1016/j.jbiomech.2019.109474
- 510 47. Schmitt L, Paterno M, Ford K, Myer G, Hewett T. Strength asymmetry and landing  
511 mechanics at return to sport after ACL reconstruction. *Med Sci Sport Exerc*.  
512 2015;47(7):1426-1434. doi:10.1249/MSS.0000000000000560.Strength
- 513 48. Newell KM. Time scales in motor learning and development. *Psychol Rev*.  
514 2001;108(1):57-82.
- 515 49. Newell KM, Liu YT. Collective Variables and Task Constraints in Movement  
516 Coordination, Control and Skill. *J Mot Behav*. 2021;53(6):770-796.  
517 doi:10.1080/00222895.2020.1835799
- 518 50. Newell KM. Coordination, control and skill. In: Goodman, D., Wilberg, R.B. and  
519 Franks, I.M., Eds., Differing Perspectives in Motor Learning, Memory and Control.  
520 Advances in Psychology; 1985:295–317
- 521 51. Preatoni E, Hamill J, Harrison AJ, et al. Movement variability and skills monitoring in  
522 sports. *Sport Biomech*. 2013;12(2):69-92. doi:10.1080/14763141.2012.738700
- 523

525 Table 1: Mean  $\pm$  SD and median (95% CI) of weeks spent in each phase

<b>RTP phase</b>	<b>Mean duration</b>	<b>Median duration</b>
Acute	7 $\pm$ 4	4 (11-3)
Middle	10 $\pm$ 5	10 (19-1)
Late	6 $\pm$ 2	6 (10-2)

526 Table 2: Mean ± SD group VPSI pre-injury baseline, initial and end testing session of the drop jump  
 527 and lateral hurdle hop [CV]. Effect size ( g ) and relative change (RC) between testing session  
 528 comparison

			<b>Testing Comparison</b>				
			Baseline – Last		First – Last		
			<i>g</i>	RC%	<i>g</i>	RC%	
<b>Middle phase -</b> Drop jump	RTP	Baseline	<b>9.69±2.29 [25%]<sup>+</sup></b>	2.72	82%		
		First session	<b>20.32±4.13 [18%]<sup>*</sup></b>			0.74	17%
		Last Session	17.51±3.44 [20%]				
<b>Late phase -</b> Hurdle hop	RTP	Baseline	<b>8.73±3.57 [41%]<sup>+</sup></b>	1.67	93%		
		First session	<b>25.45±16.02 [63%]<sup>*</sup></b>			0.94	42%
		Last Session	14.53±3.38 [23%]				

529 *Abbreviations:* *g*: effect size, RC: relative change, RTP: return to play. *Note:* Bold indicates  $p \leq$   
 530 0.05, or hedges  $g \geq 0.80$  \* **and dashed underlined** indicates significant difference between the first  
 531 and last session. + **and underlined** significant difference between pre-injury baseline and the last  
 532 session



533 Table 3: Mean  $\pm$  SD individual player VPSI for the middle phase (drop jump) and end phase (lateral  
 534 hurdle hop

		<b>Middle phase - Drop jump</b>	<b>Late phase – Hurdle hop</b>
001	Baseline	7.39 $\pm$ 0.98	6.42 $\pm$ 0.56
	RTP	First session	14.83 $\pm$ 1.56
		Last Session	16.06 $\pm$ 1.98
002	Baseline	6.00 $\pm$ 0.56	8.14 $\pm$ 0.98
	RTP	First session	20.60 $\pm$ 3.98
		Last Session	12.92 $\pm$ 1.21
003	Baseline	10.97 $\pm$ 1.43	10.89 $\pm$ 1.25
	RTP	First session	23.08 $\pm$ 0.76
		Last Session	22.02 $\pm$ 3.54
004	Baseline	10.48 $\pm$ 2.76	7.24 $\pm$ 1.43
	RTP	First session	16.33 $\pm$ 1.85
		Last Session	16.84 $\pm$ 2.12
005	Baseline	12.84 $\pm$ 1.01	4.40 $\pm$ 0.98
	RTP	First session	20.80 $\pm$ 2.09
		Last Session	15.90 $\pm$ 1.45
006	Baseline	10.34 $\pm$ 2.12	11.18 $\pm$ 1.69
	RTP	First session	27.38 $\pm$ 2.98
		Last Session	22.32 $\pm$ 1.12
007	Baseline	8.54 $\pm$ 0.67	9.82 $\pm$ 1.02
	RTP	First session	27.38 $\pm$ 3.17
		Last Session	16.98 $\pm$ 1.99
		13.60 $\pm$ 3.02	

535

## Figure Captions

536 **Figure 1** — Return to play testing timeline

537 **Figure 2** — Mean  $\pm$  SD and individual player responses in the acute phase (eyes open and  
538 eyes closed static postural control assessments) between baseline, and the first and last  
539 session of the acute phase for sway path intervals. \* Indicates significant difference between  
540 sessions ( $p < 0.05$ )