Symptom presentation and evolution in the first 48 hours after injury are associated with return to play after concussion in elite Rugby Union

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Abstract

*Background***:** Return to play in elite rugby is managed using a six-stage Graduated Return to Play (GRTP) process, which can result in clearance to play within one week of injury. We aimed to explore how symptom, cognitive and balance presentation and evolution during concussion screens two hours (Head Injury Assessment, HIA2) and 48 hours (HIA3) after injury were associated with time to return to play, to identify whether a more conservative GRTP may be appropriate.

*Methods***:** A retrospective cohort study was conducted in 383 concussed rugby players from elite men's rugby over three consecutive seasons**.** Players were classified as SHORTER or LONGER returns, depending on whether RTP occurred within seven days (allowing them to be considered to play the match one week after injury) or longer than eight days, respectively. Symptom, cognitive and balance performance during screens was assessed relative to baseline (normal or abnormal), and to the preceding screen (improving or worsening). Associations between sub- test abnormalities and RTP time were explored using Odds Ratios (LONGER vs SHORTER). Median days' absence were compared between players with abnormal or worsening results and those whose results were normal or improving.

Results: Abnormal symptom results during screens 2 hours and 48 hours after concussion were associated with LONGER return time (HIA2 OR 2.24, 95% CI 1.42
- 3.55; HIA3 OR 3.32, 95% CI 1.91-5.79). Worsening symptom number or severity from the time of injury to 2 and 48 hours post-injury were associated with LONGER return (HIA2 OR 2.53, 95% CI 1.38-4.65; HIA3 OR 3.38, 95% CI 1.11
- 10.29.
Median days' absence was greater in players with abnormal symptom results at both HIA2 and HIA3. Cognitive and balance performance were not associated with LONGER return, and did not affect median days' absence.

Conclusion: Symptom presentation and evolution within 48 hours of concussion were associated with longer RTP times. This may guide a more conservative approach to return to play, while still adhering to individualized concussion management principles.

Keywords: Brain injury, Concussion management, general return to play, medical management

1. Introduction

Concussion is the most common injury in elite Rugby Union, accounting for between 15% and 20% of all match injuries[1], with an incidence equating to approximately four concussions every five matches[1,2]. Given the potential for a second concussion, increased risk of other injury, and potential longer-term health implications of repeated head injuries[3,4], it is recognized and accepted that the return to play of a concussed player must be managed prudently [5-7].

In Rugby Union, a six-stage Graduated Return to Play (GRTP) process has been followed since 2011[5,6]. In this process, a concussed player completes 6 distinct stages, beginning with symptom limited daily activities (Stage 1). The player then progresses through four stages of training-based restricted activity during which symptoms are monitored, followed by a sixth and final stage where the player is cleared to return to play. At each stage, if symptoms do not worsen during exercise, players progress to the next more physically challenging stage, while if symptoms do worsen, players repeat that stage [5].

The GRTP thus imposes a minimum of 5 additional days of reduced intensity and supervised exercise training before returning to play. Given the typical weekly cycle of elite rugby, players who progress without interruption through the GRTP can be cleared to play in time for the match the week following their concussion. In an analysis of 3006 concussions in elite rugby in England, West et al reported that such next-match returns occur in 33% of all concussions [6].

The potential for a concussed player to return for the next match has been questioned and must be considered for player welfare reasons[8,9]. Some contact sports have recently increased the minimum return to play period. Mandating a stand down period carries with it risks of under-reporting and under-diagnosis of concussions[10], given that symptom endorsement, reliant on player disclosure, is the most sensitive component of the diagnostic process [11,12]and the likeliest means by which delayed concussion presentation would be identified.

Of interest is whether a more conservative approach to return to play can be adopted while still adhering to individualized concussion management principles. One potential approach to this issue is to use the outcome of clinical assessments conducted after head injury to stratify players within the GRTP such that some are delayed and thus ineligible to play in a match one week after injury.

Previous research has identified that the number and severity of acute and sub-acute symptoms predict return to normal function and play after concussion [13,14], with both symptom number and severity showing a dose-dependent relationship with RTP [14]. Balance performance as a predictor of post-concussion syndrome and delayed recovery is disputed ([15,16], albeit in youths rather than adult athletes, while cognitive and balance function are often assessed after concussion, but over longer periods [17] and immediate deficits have not been associated with RTP in a sporting context.

Rugby offers a unique opportunity to explore the association between symptom, cognitive and balance elements of the concussion diagnostic process and RTP, because in addition to the GRTP, the sport employs a multi-modal, three-point-in-time Head Injury Assessment (HIA), consisting of a screen at the time of injury (HIA1), and again in the early periods (HIA2 two hours post head impact and HIA3 48 hours post impact) after head injury[18]. The HIA screens are based on the Sideline Concussion Assessment Tool [19], and include a symptom checklist, cognitive sub-tests, and balance assessments. Concussion is diagnosed when any of these sub-tests results is abnormal compared to a previously assessed baseline test, but the association between performance in these sub-tests and return to play time is unknown.

Therefore, this research aimed to describe the clinical presentation of concussed players during the HIA process, with a view to identifying whether any sub-tests are associated with longer return to play time after concussion, such that a player would miss at least one match. We hypothesized that abnormal sub-tests results within the first three days after concussion would be associated with delayed return to play time.

2. Methods

380 cases of match concussion from three seasons (2018 to 2021) of club rugby in England were analyzed. This represents the highest level of national rugby in England, consisting of fully-professional elite adult players. The return to play time for all cases was known, calculated as days between the injury and the medical clearance to return to full contact training. Concussion was diagnosed as per the World Rugby operational definition, described previously[18]. Briefly, a player was identified as concussed during match play based on the observation of one of eleven Criteria 1 signs, or confirmed as concussed during the three-stage Head Injury Assessment (HIA) process. A concussion diagnosis is confirmed by a physician if any SCAT5 sub-test result is abnormal relative to a player's previously measured baseline performance in that sub-test, or if a doctor has a clinical suspicion of a concussion despite normal sub-test results. An abnormal sub-test can be overruled by the doctor's clinical judgment, however this overrule requires a written justification from the doctor completing the assessment.

Once diagnosed as concussed, players enter the previously described six-stage GRTP. Our first analysis approach was to classify players as either SHORTER or LONGER return to play (RTP) cases based on time loss. SHORTER cases were those who were medically cleared to return to full contact within 7 days of a concussion diagnosis (n = 148, median 6, range 4 to 7 days). LONGER cases were players whose medical clearance occurred eight or more days after injury (n = 235, median 15, range 8 to 253 days).

This distinction was made in order to identify players who would be eligible to return in time for the next match (SHORTER), or would be excluded for least one match by a delay in recovery (LONGER). This approach was prioritized because if differences exist between players who are cleared for the next match and those who miss at least one match, changes to policy to more conservatively manage players can be created within the practical context of the rugby environment.

We have used medical clearance as the return to play criteria, rather than return to match participation, since the latter is affected by numerous other factors that may

be unrelated to the concussion. These factors include other injuries, fixture variability where a team may not play for weeks after a concussion, and tactical selections that may confound the reported next-match selection.

We also explored the outcome if the time-period dividing SHORTER from LONGER was set to 12 days. This is the minimum possible period that would allow a player to play the second match after concussion. This analysis revealed no differences in outcomes compared to the use of a seven-day distinction, and thus the seven-day distinction was used for all subsequent analysis.

2.1. HIA sub-test performance as a predictor of RTP category

The results of the symptom checklist, cognitive sub-tests, and balance assessments within HIA1, HIA2 and HIA3 were analyzed in the concussed players. For each sub-test, two sets of analyses were conducted. First, the result of each sub-test was expressed as normal or abnormal relative the player's previously conducted baseline assessment. For symptoms and balance sub-tests, an abnormal result was recorded when a concussed player endorsed any number of symptoms more than at baseline, or made more balance errors during their HIA screen than in their baseline assessment. Cognitive sub-tests were abnormal when the concussed player scored lower during the HIA than in their baseline screen. The proportion of players in SHORTER and LONGER who produced abnormal sub-test results was then used to calculate an odds-ratio that an abnormal sub-test result would be found in LONGER compared to SHORTER.

The odds ratio, 95% confidence intervals (CIs) and corresponding p-value were calculated using a logistic regression with days absence (binary outcome: SHORTER or LONGER) as the dependent variable and sub-test pass/fail status as the independent variable. In addition, previous concussion history in the past year was included as a binary variable (yes or no), since concussion history, including in the previous twelve months, is correlated with RTP times [14] and delayed resolution of symptoms [20], which would be expected to affect progression through the GRTP.

A second analysis compared the actual numerical scores in each subtest during the HIA1, HIA2 and HIA3 diagnostic screens, with changes in these scores assessed over the course of the HIA process. The change in score in the cognitive subtests and balance errors from HIA1 to HIA2 to HIA3 was computed, along with a change in the number of symptoms endorsed and their reported severity. Sub-tests were classified as either better (or the same) or worse than in the preceding test, and an odds ratio that a player whose test result worsened would be a LONGER case compared to a SHORTER case was calculated. The odds ratios, 95% CIs and corresponding p-value were calculated using a logistic regression with sub-test improvement or worsening (better vs worse) as the independent variable, and previous concussion as an additional independent variable.

2.2. Days' absence as a function of sub-test result

A second analysis approach explored the number of days before a player was returned to play as a function of sub-test abnormalities. The result of each sub-test was categorized as a pass (better than or the same as the baseline or preceding HIA stage result) or a fail (worse than baseline or the preceding HIA stage result), and the median number of days to return was compared as a continuous variable between these two sub-groups, using a Kruskal-Wallis test.

For all sub-test analyses, the number of cases available for analysis varied because of missing screens or sub-tests within screens, and when there was a mismatch between the word-list used in screens. For the analysis of changes in median scores in each sub-test, we excluded cases with 'perfect' sub-test results (for example, zero symptoms endorsed, or zero cognitive or balance errors at HIA2 and HIA3) since high numbers of such zero-cases skews the calculated median towards zero. The number of cases available for each sub-test comparison are shown in the appropriate analysis.

When logistic regressions were not used to obtain odds ratios, we have also calculated effect sizes. Effect sizes are usually only recommended for data that are normally distributed [21,22]. As the data from the present study were not normally distributed, we were required to calculate the effect size from a Mann Whitney U

Test [21]. This was done using the formula r = Z / [square root of N], where r = point biserial correlation, Z = z score calculated by Mann Whitney U Test and N = sample size. Once r was obtained from this formula, the corresponding Cohen's effect size (d) and probability of superiority (PS) were obtained from Fritz et al [21]. PS is the percentage of occasions when a randomly sampled member of the distribution with the higher mean will have a higher score than a randomly sampled member of the other distribution.

2.3. Domain performance during HIA3

We examined how the failure of various combinations of domains within the HIA3 (symptom, balance or cognitive) was associated with membership of LONGER and SHORTER. Abnormal sub-tests within each domain were identified, and the proportion of abnormal domains within LONGER and SHORTER were compared using previously described methods.

Ethical approval for the Professional Rugby Injury Surveillance Project (PRISP) was provided by the Research Ethics Approval Committee for Health at the University of Bath (EP 16/17 200), which allows for the analysis of anonymized data from PRISP. All elite players provide informed consent for use of HIA data for research purposes.

3. Results

Players who had experienced a concussion in the previous twelve months were more likely to be LONGER return cases (OR 2.56, 95% CI 1.16-5.52). Players in LONGER were significantly more likely to have had a previous concussion in the past year than players in the SHORTER group (18 vs 10%, p=0.018). The SHORTER group consisted of 148 players. Median RTP was 6 days (range 4 to 7 days). LONGER cases (n = 235) had a median RTP of 15 days (range 8 to 253 days).

3.1. HIA2 presentation

Table 1 presents the results of HIA2 sub-tests, with players categorized as LONGER and SHORTER returns. Players with an abnormal symptom result at HIA2 were 2.24 times more likely to be in LONGER than SHORTER (68% in LONGER vs 48% in SHORTER, OR 2.24, 95% CI 1.42 to 3.55). When symptom endorsement increased from HIA1 to HIA2, players were, on average, 2.53 times more likely to be a LONGER case (46% worse in HIA2 than HIA1) than a SHORTER case (25% of players worse in HIA2 than in HIA1 (Odds Ratio (OR) 2.53, 95% CI 1.38 to 4.65)).

Players whose symptom severity scores were greater during HIA2 than at baseline were 2.35 times more likely to be in LONGER than in SHORTER (69% of LONGER vs 31% of SHORTER cases, OR 2.35, 95% CI 1.48 to 3.73).

The median improvement in Immediate Memory performance from HIA1 to HIA2 was significantly greater for SHORTER than LONGER (P = 0.048), though this performance change was not associated with an improved odds of being in SHORTER compared to LONGER (OR 1.83, 0.89 to 3.81). No cognitive or balance sub-test result was associated with being a LONGER return.

3.2. HIA3 presentation

Table 2 shows the result of the HIA3 sub-tests compared to baseline screens for LONGER and SHORTER cases. Abnormal symptom endorsement and symptom severity at HIA3 were associated with a greater likelihood of being LONGER than SHORTER. 36% of players in LONGER endorsed more symptoms at HIA3 than in baseline, compared to 14% in SHORTER (OR: 3.32, 95% CI: 1.91 to 5.79), while 36% of LONGER cases had a higher symptom severity in HIA3 than at baseline, compared to 15% of SHORTER cases (OR: 3.25, 95% CI: 1.87 - 5.67).

Players whose symptom endorsement increased from HIA1 to HIA3 were significantly more likely to be LONGER cases (15%) than SHORTER cases (5%, OR 3.38, 95% CI 1.11 to 10.29). When symptom endorsement increased from HIA2 to HIA3, there was no increase in the odds of being a LONGER case, though this situation of worsening symptom presentation at HIA3 was relatively rare, occurring in only 6% of LONGER and 3% of SHORTER cases (OR 1.83, 95% CI 0.57 to 5.92).

Similarly, an increase in symptom severity from HIA2 to HIA3 was not associated with greater likelihood of being a LONGER case (OR 1.52, 0.52 to 4.51).

For all cognitive and balance sub-tests, abnormal results relative to baseline and worsening results from HIA1 and HIA2 to HIA3 were not associated with a player being a LONGER return case (Table 2). The median change in Immediate Memory performance from HIA1 to HIA3, and the odds of being LONGER when Immediate Memory performance worsened from HIA1 to HIA3 tended towards significance (P = 0.08 and P = 0.05, respectively). Figure 1 depicts the adjusted odds ratios (95% CI) for abnormal or worsening sub-test results during HIA2 (left) and HIA3 (right) screens, relative to baseline or to the preceding sub-test result withing the HIA process, respectively.

3.3. Sub-test changes and median days' absence

Table 3 shows the median days' absence for players whose sub-test results were abnormal (relative to baseline) or worse (relative to the preceding sub-test within the HIA process), compared to players with normal or non-worsening sub-test results. Selected results for median days' absence as a function of abnormal or worsening compared to non-worsening performance are shown in Figure 2.

Median days' absence was greater when symptom endorsement at HIA2 was abnormal (10 days for abnormal HIA2 result vs 7 days for normal HIA2 result, P = 0.004). Abnormal symptom endorsement at HIA3 was similarly associated with a greater median days' absence (12 days vs 8 days, abnormal vs normal, P < 0.001). In players whose symptom endorsement increased from HIA1 to HIA2 and from HIA1 to HIA3, median days' absence was significantly greater than when players had no change or an improvement in symptom presentation between these stages of the HIA process.

3.4. Domain abnormality and RTP

Table 4 shows how different combinations of domain abnormalities predicted LONGER vs SHORTER return. Of the 235 LONGER and 148 SHORTER cases, 216 and 139 had complete HIA3 data, respectively.

173 players had no abnormal sub-test results during HIA3, and were equally distributed between LONGER (52%) and SHORTER (48%). 182 players had at least one abnormal sub-domain during HIA3, with 126 cases (69%) in LONGER and 56 cases (31%) in SHORTER. Abnormalities in one or more domains were significantly more likely in LONGER than SHORTER (one sample proportion test vs 50%: $p<0.001$). Within the abnormal domains, the LONGER group were over-represented for symptom domain (76%, $p<0.001$), *symptoms + cognitive* (81%, $p=0.013$), *symptoms + balance* (88%, $p=0.032$), and *all three domains* (83%, $p=0.001$). However, it should be noted that there were very small sample sizes for *symptoms + cognitive*, *symptoms + balance* and *all three domains*.

4. Discussion

This study aimed to explore whether early presentation at HIA1 (time of head impact), HIA2 (two hours post head impact), and HIA3 (48 hours post impact) were associated with return to play time in concussed rugby players, specifically whether players who were cleared to play in time for the next match differed from those who were not cleared within seven days.

4.1. HIA2 sub-tests and return to play

Our first important finding is that symptom presentation at HIA2 was associated with whether a player was likely to be a SHORTER or LONGER return case. This association was found for an abnormal symptom endorsement or severity, as well as for an increase in symptom endorsement from HIA1 to HIA2 (after adjusting for the abbreviated symptom list of HIA1), but not for cognitive or balance sub-test results at HIA2.

Previous research on the HIA1 off-field screen has found that symptoms were most sensitive in correctly identifying a concussed player, whereas cognitive and balance

sub-test abnormalities were significantly less likely in players who would later be diagnosed as concussed [11]. The present finding supports the similar importance of symptoms in the hours and days after the injury is sustained as an indicator of concussion severity, and it confirms previous findings that symptom number and severity are correlated with RTP in a dose-dependent fashion [14].

It is perhaps a result of the greater sensitivity of symptoms that this association exists, as it is notable that a significantly higher proportion of players endorse abnormal symptoms at HIA2 than produce abnormal cognitive and balance sub-tests (Table 1). In addition, cognitive and balance tests may be less likely to differentiate between early and later RTP because of ceiling effects [23], allowing similar test performance in players with different concussion severities. Finally, performance in vestibulo-ocular motor tasks have been associated with RTP after concussion [14,24], but such tasks are not presently included in Rugby's HIA process, so we cannot explore their association with RTP in the immediate aftermath of injury. Cognitive, balance and vision sub-tests may also be considered to have greater specificity compared to symptoms [25], and so concussions that present with specific outcomes (eg: balance problems may not be detected by these tests, whereas symptoms, being less specific, capture a wider range of disturbances.

4.2. HIA3 sub-tests and return to play

A similar association between symptom presentation and evolution, but not cognitive or balance sub-test results, was found for the HIA3 screen, performed two days after the injury (Table 2 and Figure 1). Further, median days' absence was significantly greater in players with symptom abnormalities at HIA3 and increased symptoms from HIA1 and HIA2 to HIA3 (Figure 2).

These findings may be in part a result of following the GRTP process, since the policy implemented by World Rugby in 2016 stipulates that a player should remain at Stage 1 of the GRTP if normal daily activities provoke symptoms [5,6]. This would cause players to be delayed at Stage 1, which would extend their RTP beyond our seven-day cut-off for next-match return. The significant odds of being in LONGER when endorsing symptoms at HIA3 (Table 2) would thus be created by compliance

with the GRTP process. However, doctors were permitted to apply discretion to progress players through the exercise stages of the GRTP even while symptomatic, provided exercise did not provoke an increase in symptom report[5]. We found that 31% of players in SHORTER had abnormal sub-test results during HIA3 (Table 4), suggesting that this discretionary progress through the GRTP did occur.

To explore this, we analyzed our cohort with a 12-day division between SHORTER and LONGER, since this cut-off would allow players with abnormal HIA3 sub-tests to delay the GRTP by up to five days, and still fall into a SHORTER category that is defined as clearance to play within 12 days. This analysis revealed no differences in any of the findings compared to a seven-day categorization of LONGER vs SHORTER. That is, the same symptom abnormalities at HIA2 and HIA3, and changes in symptoms from HIA1 to HIA2 and HIA3, were associated with LONGER return at twelve days. This suggests that the sub-test abnormalities we report as associated with return to play time are not solely the result of the GRTP process, but have clinical relevance for the player's RTP.

With respects to doctors who permit players to progress through the GRTP prior to full resolution of symptoms, it has previously been shown that clinical judgment during HIA1 off-field screens, where doctors overrule the presence of abnormal sub-test results, improves the overall accuracy of the HIA1 phase of the HIA process[11,26]. To support more conservative concussion management, guidance was previously given to doctors as to when they should avoid making such clinical judgment overrules [26]. The present findings support consideration of similar guidance to recommend that players who are symptomatic at HIA3 should delay the initiation of the exercise stages of the GRTP. This would achieve a more conservative return to play period, using player endorsed symptoms (individual management).

Whilst we do not know when doctors have applied their discretion to initiate the GRTP in symptomatic players, we are able to gain some insight into how players commence the GRTP and return to play by examining how often players in SHORTER and LONGER presented with one or more abnormal sub-domains at HIA3 (Table 4). The relative likelihood of being in LONGER increased as the number of abnormal domains increased (for one abnormal domain, RR = 1.79, for two

abnormal domains, RR = 3.22 and for three abnormal domains, RR = 5.00). This suggests that overrule decisions are less likely as the number of abnormal sub-tests increases, indicative that team physicians factor the magnitude of abnormalities into their RTP decisions. A more conservative approach may be to implement policies that prevent a player with any symptom or cognitive abnormalities at HIA3, as well as balance abnormalities in combination with abnormal symptoms, from beginning the GRTP.

Previous research has identified that the most consistent predictor of a slower post-injury recovery, defined as return to normal function, is the severity of acute and sub-acute symptoms [13,14], a finding we confirm here. In the sporting context, higher symptom severity scores and worse cognitive and balance performance during an assessment within 48 hours of injury were predictive of delayed recovery from concussion[27] defined as return-to-play clearance greater than 24 days.

Previous studies have also associated various pre-injury characteristics with concussion severity [13]. These include age[28,29], female sex [29-31] and elements of medical history such as headache [32-34], family history[35] and psychiatric history[35,36]. We cannot assess the influence of these factors in this study, since such characteristics are not available in the anonymised dataset on which this analysis was performed.

However, sex and young age are not considerations since our cohort is entirely adult males. We do not have medical histories to explore other medical factors, but we did assess the influence of prior concussion history, finding that players with a concussion in the preceding twelve months were 2.6 times more likely to return to play in the LONGER group. Guidelines for more conservative return to play may thus consider concussion history as a factor for delaying return to play.

It has been suggested that neurobiological and psychosocial factors interact to influence recovery after concussion [27], with initial injury severity predictive of acute outcomes, and psychosocial and psychological health variables important for prolonged recovery[37]. While we have not evaluated prolonged recovery, our findings support the notion that initial injury severity is associated with return to play within a

seven-day period, with the novel contribution that these changes are evident as early as the first two hours after injury, as well as up to two days after injury.

4.3. Limitations

There are some important limitations in the present study. As noted above, we cannot be certain when doctors have initiated the GRTP process by overruling abnormal sub-tests at the diagnostic phase of the HIA3, which has implications for the true effect of sub-tests results at HIA3 on RTP. Another limitation is that we have created two groups for return to play, because we wished to explore a specific question regarding players who are cleared and eligible for the earliest possible next-match return compared to those who are delayed by a week or more.

This results in analysis of RTP in two groups and does not distinguish between a player who is cleared early within LONGER (8 days, for example) and a player with a considerably longer RTP (28 days or more). However, we chose this binary approach to address a practical challenge faced by the sport, so that policy changes may be informed in order to govern the early-return scenarios that we capture by comparing SHORTER to LONGER cases. We did however assess RTP as a continuous outcome, and confirmed that symptoms, but not cognitive or balance abnormalities, are associated with longer RTP times.

Future studies may explore differences in players with significantly longer return to play times, but this was not possible in the present study, also because of limited sample size of very long RTP times and abnormal sub-tests. Future studies may also explore the medical histories of players in greater detail, to account for family and personal history of factors known to predict delayed recovery, to determine their influence of return to play decisions after sports concussions.

5. Conclusion

In conclusion, this study demonstrates that within the first two hours of a concussion, symptom endorsement and worsening symptom profiles compared to HIA1 are associated with a delay in return to play time that is sufficient to cause players to

miss at least one match. Similarly, during the HIA3 done 48 hours after injury, symptom endorsement and worsening symptom presentation are associated with longer return to play time. No cognitive or balance sub-test abnormalities or impairments are associated with delayed return to play time. These findings may provide sports with a means to adopt a more conservative concussion management approach by delaying the initiation of the GRTP or the return to play decision based on that initial presentation, while still maintaining a principle of individualized medical management of players.

Authors' contributions

RT and JB conceived of the research questions, performed the analysis and wrote the manuscript. EF and MR conceived of the research question and study and provided edits. SK, KS, MC, LS, RH and SW provided support on data generation and analysis and provided edits.

Competing interests

EF is the Chief Medical Officer of World Rugby, the body that runs the sport of Rugby Union globally. MR was former Chief Medical Officer of World Rugby. RT and LS employed as a consultant by World Rugby. JB receives research funding from World Rugby. KS, SK and RH are employed by the Rugby Football Union (RFU) that runs Rugby Union in England. SW was funded by WR and the RFU for collection of data used in this study. MC is employed by Premiership Rugby, the professional rugby competition in England.

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Table 1: Sub-test performance and relative distribution of abnormal HIA2 subtests in SHORTER and LONGER cases

	LONGER		SHORTER			LONGER		SHORTER			
	Cases for medians (n)	**Median (IQR)**	**Cases for medians (n)**	**Median (IQR)**	**P value (medians)**	**Cases for OR calculation**	**% fail/worse**	**Cases for OR calculation**	**% fail/worse**	**Adjusted OR (for prev_conc_1yr (yes or no)**	**Adjusted P value for OR**
Symptoms											
Symptoms at baseline (BL)	229	0 (0 ; 0)	144	0 (0 - 0)	0.925						
Change in symptoms from BL	204	-3 (-7; 0) *	133	0 (-4; 0)	<0.001	204	68%	133	48%	2.21 (1.39 - 3.50) †	0.001
Change in symptoms from HIA1	78	-2(-4; -1)	35	-2(-3; 0)	0.086	137	46%	82	25%	2.49 (1.36 - 4.58) †	0.003
Immediate Memory											
Performance relative to baseline	183	0(-2;4)	117	0(-2;3)	0.312	183	37%	117	40%	0.91 (0.56 - 1.47)	0.696
Change in IM performance from HIA1	87	1(-1; 4)	66	3 (0; 5)	0.059	97	33%	67	22%	1.80 (0.87 - 3.73)	0.115
Digits backwards											
Performance relative to baseline	197	0 (0 ; 0)	116	0 (0 ; 0)	0.505	197	10%	116	8%	1.11 (0.49 - 2.51)	0.801
Concentration											
Performance relative to baseline	197	0 (0 ; 0)	116	0 (0 ; 0)	0.709	197	10%	116	10%	1.03 (0.48 - 2.21)	0.937
Delayed recall											
Performance relative to baseline	88	0(-2; 0)	60	0(-2; 1)	0.314	183	40%	117	43%	0.89 (0.55 - 1.45)	0.650
Change in DR performance from HIA1	77	0(-1; 2)	58	1(0; 3)	0.208	96	34%	66	21%	1.89 (0.91 - 3.92)	0.089

Balance tests											
Double leg relative to baseline	205	0 (0 ; 0)	133	0 (0 ; 0)	0.638	205	1%	133	1%	0.36 (0.03 - 3.97)	0.405
Single leg relative to baseline	205	0 (-1 ; 0)	133	0 (0 ; 1)	0.673	205	26%	133	24%	1.16 (0.69 - 1.94)	0.572
Tandem stance relative to baseline	205	0(0; 1)	133	0 (0 ; 0)	0.709	205	19%	133	19%	0.96 (0.54 - 1.69)	0.91
Total balance errors relative to baseline	205	0(-1;2)	133	1(-1;2)	0.915	205	30%	133	26%	1.21 (0.74 - 2.00)	0.451

** p<0.05 median days' absence compared to players in SHORTER; † p<0.05 OR for being LONGER vs SHORTER for abnormal or worsening sub-test result.*

Abbreviations: DR = Delayed Recall; HIA = Head Injury Assessment; IM = Immediate Memory

Table 2: Sub-test performance and relative distribution of abnormal HIA3 subtests in SHORTER and LONGER cases

	LONGER		SHORTER			LONGER		SHORTER			
	Cases for medians (n)	**Median (IQR)**	**Cases for medians (n)**	**Median (IQR)**	**P value (medians)**	**Cases for OR calculation**	**% fail/worse**	**Cases for OR calculation**	**% fail/worse**	**Adjusted OR (for prev_conc_1yr (yes or no)**	**Adjusted P value for OR**
Symptoms											
Symptoms at baseline (BL)											
Change in symptoms from BL	216	0(-2; 0) *	136	0(0; 0)	<0.001	216	36%	136	14%	3.30 (1.89 - 5.75) †	<0.001
Change in symptoms from HIA1	66	1(-1;2) *	28	1(1; 2)	0.047	142	15%	78	5%	3.34 (1.10 - 10.15) †	0.034
Change in symptoms from HIA2	146	3(1; 6)	73	3(1;7)	0.888	198	6%	127	3%	1.82 (0.56 - 5.88)	0.317
Change in symptom severity from BL	216	0(-2; 0) *	136	0(0; 0)	<0.001	216	36%	136	15%	3.23 (1.85 - 5.62) †	<0.001
Change in symptom severity vs HIA2	146	6(2; 11)	73	5(2; 12)	0.523	198	6%	127	4%	1.43 (0.49 - 4.24)	0.513
Immediate Memory											
Performance relative to baseline	197	2(0; 4)	119	2(0; 4)	0.758	197	17%	119	20%	0.76 (0.42 - 1.35)	0.344
Change in IM performance from HIA1	94	3(0; 5)	64	4(1; 6)	0.096	104	16%	66	6%	3.03 (0.97 - 9.44)	0.056
Change in IM performance from HIA2	184	1(-1; 4)	121	2(0; 4)	0.544	200	28%	126	24%	1.10 (0.65 - 1.87)	0.731
Digits backwards											
Performance relative to baseline	208	0(0; 0)	124	0(0; 0)	0.344	208	6%	124	3%	2.18 (0.69 - 6.88)	0.184

Concentration											
Performance relative to baseline	208	0(0; 0)	124	0(0; 0)	0.406	208	7%	124	4%	1.94 (0.68 - 5.51)	0.215
Delayed recall											
Performance relative to baseline	88	0(-1; 1)	60	0(0; 1)	0.215	197	27%	119	19%	1.62 (0.92 - 2.83)	0.092
Change in DR performance from HIA1	75	1(0; 3)	56	2(1; 3)	0.232	103	17%	65	12%	1.37 (0.54 - 3.43)	0.507
Change in DR performance from HIA2	70	1(-1;3)	55	1(-1;2)	0.884	200	24%	126	22%	1.13 (0.66 - 1.95)	0.650
Balance tests											
Double leg relative to baseline	218	0(0; 0)	136	0(0; 0)	0.428	218	0%	136	0%	-	-
Single leg relative to baseline	218	0(0; 2)	136	1(0;2)	0.292	218	15%	136	13%	1.15 (0.62 - 2.15)	0.663
Tandem stance relative to baseline	218	0(0; 1)	136	0(0; 0)	0.473	218	9%	136	5%	1.75 (0.72 - 4.30)	0.216
Total balance errors relative to baseline	218	1(0; 2)	136	1(0; 2)	0.462	218	14%	136	12%	1.16 (0.60 - 2.24)	0.661

** p<0.05 median days' absence compared to players in SHORTER; † p<0.05 OR for being LONGER vs SHORTER for abnormal or worsening sub-test result.*

Abbreviations: DR = Delayed Recall; HIA = Head Injury Assessment; IM = Immediate Memory

Table 3: Median days absence after concussion for different sub-test results during HIA2 and HIA3

	Fail/Worse		Pass/Better				
	Count	**Median (IQR)**	**Count**	**Median (IQR)**	**P value**	**Calculated effect size (ES) – Cohen's d**	**Probability (%) of superiority (based on ES)**
Symptom endorsement at HIA2	203	10 (6; 17)	134	7 (6; 16)	0.010	0.2	56
Symptom endorsement at HIA3	98	12 (8; 22)	254	9 (6; 16)	< 0.001	0.4	61
Symptom change from HIA1 to HIA2	84	11 (8; 19)	135	9 (6; 16)	0.003	0.3	58
Symptom change from HIA1 to HIA3	25	18 (9; 39)	195	9 (6;16)	0.001	0.3	58
Symptom change from HIA2 to HIA3	15	11 (8; 20)	310	9 (6; 16)	0.382	0.1	53
Symptom severity change HIA2 to HIA3	16	9 (7; 16)	202	10 (6; 17)	0.559	0.0	50
IM at HIA2	116	9 (6; 17)	184	10 (6; 17)	0.618	0.0	50
IM at HIA3	58	10 (6; 18)	258	10 (6; 17)	0.913	0.0	50
IM change HIA1 to HIA2	47	10 (6; 18)	117	8 (6; 12)	0.091	0.1	53
IM change HIA1 to HIA3	21	11 (8; 18)	149	9 (6; 14)	0.072	0.1	53
IM change HIA2 to HIA3	84	10 (6; 19)	242	9 (6; 16)	0.233	0.1	53
DR at HIA2	122	9 (6; 13)	176	10 (6; 18)	0.332	0.1	53
DR at HIA3	76	10 (6; 16.5)	240	9 (6; 17)	0.442	0.0	50
DR change HIA1 to HIA2	47	10 (6; 15)	115	8 (6; 15)	0.178	0.1	53
DR change HIA1 to HIA3	25	10 (6; 12)	143	9 (6; 16)	0.806	0.0	50
DR change HIA2 to HIA3	75	9 (6; 16)	251	9 (6; 17)	0.582	0.0	50
Concentration at HIA2	32	9 (6; 17)	281	10 (6; 17)	0.573	0.0	50

Concentration at HIA3	20	11 (8; 17)	312	10 (6; 18)	0.359	0.1	53
Digits backward at HIA2	29	9 (6; 18)	284	10 (6; 17)	0.798	0.0	50
Digits backward at HIA3	17	10 (8; 17)	315	10 (6; 18)	0.381	0.0	50
DL balance at HIA2	4	8 (7; 13)	334	9 (6; 17)	0.926	0.0	50
DL balance at HIA3	1	32 (32; 32)	353	9 (6; 17)	0.181	0.1	53
DL balance change HIA2 to HIA3	1	32 (32; 32)	326	9 (6; 16)	0.173	0.1	53
SL balance at HIA2	85	10 (6; 16)	252	9 (6; 17)	0.391	0.0	50
SL balance at HIA3	51	11 (6; 23.5)	303	9 (6; 16)	0.098	0.1	53
SL balance change HIA2 to HIA3	73	10 (6; 18)	254	9 (6; 16)	0.725	0.0	50
TS balance at HIA2	63	10 (6; 24)	275	9 (6; 16)	0.302	0.1	53
TS balance at HIA3	26	11 (7; 19)	328	9 (6; 17)	0.202	0.1	53
TS balance change HIA2 to HIA3	25	9 (6; 18)	302	9 (6; 16)	0.995	0.0	50
Total balance errors at HIA2	95	10 (6; 16)	243	9 (6; 17)	0.285	0.1	53
Total balance errors at HIA3	46	11 (6; 23)	308	9 (6; 16)	0.135	0.1	53
Total balance errors change HIA2 to HIA3	66	9 (6; 16)	261	9 (6; 16)	0.802	0.0	50

* *p<0.005* median days' absence compared to players in SHORTER; † p<0.05 OR for being LONGER vs SHORTER for abnormal or worsening sub-test result.

Abbreviations: DL = Double Leg; DR = Delayed Recall; HIA = Head Injury Assessment; IM = Immediate Memory; SL = Single Leg; TS = Tandem Stance

Table 4: Comparison of proportion of HIA3 domain abnormalities in LONGER and SHORTER cases

	All cases		As % of domain criteria				
Domain abnormalities	**LONGER**	**SHORTER**	**LONGER**	**SHORTER**	**P-value: LONGER % compared to 50% (one-sample proportion)**	**Relative risk**	**RR with 95% CI**
No abnormal domains (all domains normal)	90	83	52%	48%	0.599	1.08	1.08 (0.80 – 1.46)
Any abnormality during HIA3	126	56	69% *	31%	<0.001	2.25	2.25 (1.64 – 3.08)
Symptoms only	38	12	76% *	24%	<0.001	3.17	3.17 (1.65 – 6.06)
Cognitive only	31	17	65% *	35%	0.038	1.82	1.82 (1.01 – 3.29)
Balance only	8	14	36%	64%	0.190	0.57	0.57 (0.24 – 1.36)
Symptoms and cognitive	13	3	81% *	19%	0.013	4.33	4.33 (1.23 – 15.21)
Symptoms and balance	7	1	88% *	13%	0.032	7.00	7.00 (0.86 – 56.90)
Cognitive and balance	9	5	64%	36%	0.295	1.80	1.80 (0.60 – 5.37)
All three domains	20	4	83% *	17%	0.001	5.00	5.00 (1.71 – 14.63)
Two abnormal domains	*29*	*9*	*76% **	*24%*	*0.005*	*3.22*	*3.22 (1.53 – 6.81)*
Only one abnormal domain	*77*	*43*	*64% **	*36%*	*0.01*	*1.79*	*1.79 (1.23 – 2.60)*

* *p<0.05* proportion of players with abnormal domain in LONGER vs expected equal (50%) distribution

Abbreviation: HIA = Head Injury Assessment

Figure 1: Adjusted odds ratios for selected sub-test outcomes in the HIA2 (left panel) and HIA3 (right panel) screens. Odds ratios are calculated using the proportion of players in SHORTER (< 8 days return to play) and LONGER (8 or more days return to play) who produced abnormal sub-test results, either relative to baseline or worsening compared to the preceding test. * *p<0.05, LONGER vs SHORTER for abnormal or worsening sub-test result.*
Abbreviations: BL = Baseline; DB = Digits Backward; DR = Delayed Recall; HIA = Head Injury Assessment; IM = Immediate Memory

Figure 2: Median days' absence as a function of sub-test results for selected sub-tests. Filled diamonds show players with abnormal or worsening sub-test performances relative to baseline or the preceding screen in the HIA process respectively, while open diamonds show players whose sub-test results were normal relative to baseline, or which improved compared to the preceding HIA screen. Counts for each sub-test are shown. * *p<0.05, compared to players with abnormal results for that sub-test.*
Abbreviations: BL = Baseline; DR = Delayed Recall; HIA = Head Injury Assessment; IM = Immediate Memory; SL = Single Leg

1.