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ORIGINAL ARTICLE

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Wheelchair rugby players maintain sprint performance but alter propulsion biomechanics after simulated match play

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Abstract

The study aimed to explore the influence of a sports-specific intermittent sprint protocol (ISP) on wheelchair sprint performance and the kinetics and kinematics of sprinting in elite wheelchair rugby (WR) players with and without spinal cord injury (SCI). Fifteen international WR players (age 30.3 ± 5.5 years) performed two 10-s sprints on a dual roller wheelchair ergometer before and immediately after an ISP consisting of four 16-min quarters. Physiological measurements (heart rate, blood lactate concentration, and rating of perceived exertion) were collected. Three-dimensional thorax and bilateral glenohumeral kinematics were quantified. Following the ISP, all physiological parameters significantly increased ($p \leq 0.027$), but neither sprinting peak velocity nor distance traveled changed. Players propelled with significantly reduced thorax flexion and peak glenohumeral abduction during both the acceleration (both −5°) and maximal velocity phases (−6° and 8°, respectively) of sprinting post-ISP. Moreover, players exhibited significantly larger mean contact angles (+24°), contact angle asymmetries (+4%), and glenohumeral flexion asymmetries (+10%) during the acceleration phase of sprinting post-ISP. Players displayed greater glenohumeral abduction range of motion (+17°) and asymmetries (+20%) during the maximal velocity phase of sprinting post-ISP. Players with SCI (SCI, $n = 7$) significantly increased asymmetries in peak power (+6%) and glenohumeral abduction (+15%) during the acceleration phase post-ISP. Our data indicates that despite inducing physiological fatigue resulting from WR match play, players can maintain sprint performance by modifying how they propel their wheelchair. Increased asymmetry post-ISP was notable, which may be specific to impairment type and warrants further investigation.

KEYWORDS
asymmetries, fatigue, upper-body kinematics, wheelchair sprinting

1 | INTRODUCTION

Wheelchair rugby (WR) is a popular Paralympic team sport originally developed for individuals with cervical spinal cord injury (SCI), known as tetraplegia.[1] However, wheelchair users with non-SCI impairments such as limb deficiency, neurological impairment (cerebral palsy), and neuromuscular disorders are now eligible to compete.[1] The sport is characterized by frequent and intermittent bouts of high speed and/or sprint propulsion relative to impairment severity. Thus, the ability to accelerate rapidly and attain a high maximal velocity are key indicators of WR performance.[2] That said, these activities rely on the small muscle mass of the upper limb, which impose a large mechanical demand on the shoulder and coincide with detrimental glenohumeral kinematics linked to reduced subacromial space.[2–5] WR match-play consists of four 8-min quarters separated by 2, and 5 min for half time. When the ball is out of play, the game clock is stopped exposing players to ~16-min of active propulsion per quarter, with only short periods of recovery. Earlier work by Sarro et al[6] noted both reduced distance traveled and average velocities in the second half of WR match play, indicating players may experience impaired sprint performance, particularly toward the end of the game due to fatigue. That said, the playing standards have changed considerably, data were presented only as two halves of a working clock of 66.8 min and only 8 players investigated. In contrast, to more recent evidence[2] incorporating larger cohorts of players ($n = 100$) where no significant difference in activity profiles over the course of match play were found. Thus, this topic warrants clarification.

The influence of fatigue on wheelchair propulsion biomechanics has been investigated during both steady-state and start-up propulsion at low speeds during daily wheelchair propulsion of experienced non-athletic individuals with SCI and non-wheelchair users.[7–10] Wheelchair users maintained prescribed submaximal speed and average power output when fatigued by compensating with larger push-rim contact angles and trunk forward flexion.[7–9] Moreover, during start-up propulsion in a fatigued state reductions are evident in push-rim contact time resulting in reduced applied forces.[11] These findings, suggest that fatigue-induced alterations in propulsion biomechanics are present[11] but may not be transferable to a sporting context since the constraints of these aforementioned studies were specific to the task of daily propulsion. It is well-known that wheelchair propulsion biomechanics vary according to speed and wheelchair design[3] and that the underlying mechanisms of fatigue vary based on the constraints of the task.[12] Hence further work is warranted to gain a thorough understanding of the biomechanical alterations induced by WR match play.

Recent evidence suggests the biomechanical characteristics of wheelchair sprinting in wheelchair sports athletes are adaptable to varied task constraints and shoulder pain thereby facilitating task performance (peak velocity and distance traveled).[3] WR players with cervical SCI typically have limited trunk function and display different physiological responses to simulated WR activities to those with non-SCI impairments.[13] Thus, distinct biomechanical alterations may exist between these groups because of WR match play and warrant investigation. WR players exhibit asymmetries in both kinetics during sprinting[14,15] and shoulder kinematics during submaximal propulsion[16] in a non-fatigued state, highlighting the value of quantifying asymmetries for sports performance and injury surveillance. From a theoretical perspective, asymmetries indicate the unequal distribution of forces. Thus, if simulated WR match play negatively amplifies these asymmetries even further, the uneven acute and chronic stress imposed by wheelchair sprinting throughout a game may be substantial. Therefore, the purpose of this study was to: (1) establish whether a sports-specific intermittent sprint protocol (ISP) decreases wheelchair sprint performance; and (2) examine alterations in the kinetic, joint kinematic, and the asymmetries therein, during wheelchair sprinting in elite WR players with or without SCI. It was hypothesized that peak velocity and distance traveled during sprinting would decrease and biomechanical modifications including reduced shoulder motion and greater kinetic and kinematic asymmetries would be present following WR match play.

2 | MATERIALS AND METHODS

2.1 | Participants

Fifteen male international WR rugby players (mean ± standard deviation: age: 30.3 ± 5.5 years; body mass: 65.5 ± 14.6 kg; years competing in WR = 7.8 ± 3.9 years) volunteered to participate in this study. Participants provided written informed consent for the study which was approved by the University's Ethical Advisory Committee. To achieve a statistical power of 80% with an alpha criterion level of 0.05 based on previous wheelchair sprinting kinematic data,[3] a minimum of 12 participants was required (G*Power, 3.1.9.2). Players were divided into two groups: Individuals with SCI ($n = 7$, level = C5-C7 World Wheelchair Rugby [WWR] classification range = 0.5–2.0) and those with non-SCI health conditions ($n = 8$) including double below-knee amputation ($n = 3$), cerebral palsy ($n = 1$), Roberts syndrome ($n = 1$), arthrogryposis multiplex congenita (AMC) ($n = 1$), polyneuropathy ($n = 1$) and osteogenesis imperfecta type II ($n = 1$), WWR classification range = 1.5–3.5.

2.2 | Experimental protocol

Players were asked to refrain from strenuous exercise, caffeine, and alcohol 24 h prior to their laboratory visit. All trials were performed on a dual roller wheelchair ergometer (Lode Esseda, m988900), which simultaneously collects spatio-temporal and kinetic parameters of wheelchair propulsion from each side and shows good agreement with that of instrumented measurement wheels.[17] Players completed the test in their own individualized rugby wheelchair used in training and competitions. Dimensions ranged with chair mass 17.4 ± 3.9 kg; wheel diameter 0.60 ± 0.01 m; rim diameter 0.54 ± 0.02 m and wheelbase 0.74 ± 0.04 m.

Following a 10-min warm-up (consisting of self-selected propulsion, dynamic stretching, 3-min bout of submaximal propulsion and 2-min rest), two 10-s sprints from a rolling start were completed (Figure 1), separated by a 5-min rest. A rolling start of 1 m s^{-1} was employed to minimize wheel slippage when propelling from a stationary position on the ergometer. Standardized verbal encouragement was provided throughout to maximize players' efforts during the sprint trials. These two sprints were repeated immediately after completing a sports-specific ISP, to examine propulsion following simulated WR match-play (Figure 1).

The ISP protocol began with a further warm-up (16-min) to replicate competitive match-play preparations followed by the main protocol (73 min in duration).[13] In brief, this consisted of four 16-min quarters which incorporated prescribed propulsion speeds at various percentages of participant's peak velocity (V_{max}) determined from the pre-ISP sprint trials (Figure 1). Each quarter, players performed 11 blocks of very low speeds (≤20% V_{max}), low speeds (21%–50% V_{max}) and moderate speed (51%–80% V_{max}) propulsion, for 35, 30 and 25 s, respectively. Throughout each quarter, submaximal propulsion was separated by 5 and 10 s sprints representing high (81%–95% V_{max}) and very high speed (≥95% V_{max}) propulsion. Speed zones were distributed across each quarter to replicate the interval nature of match-play and were based on relative speed profiles of WR players during international competitions.[2]

2.3 | Physiological and biomechanical measurements

After each participant voided their bladder, body mass was recorded to the nearest 0.1 kg using a digital platform scale (Detecto 6550 Wheelchair Scales). Before and immediately after the ISP, a capillary blood sample was taken from the earlobe to determine blood lactate concentration (BLa) (Biosen C-line, EKF Diagnostics). Heart rate (HR) (Polar Team^2, Polar Electro Oy) was recorded continuously throughout the testing session and was reported as an average for pre-ISP and post-ISP sprints. Rating of perceived exertion (RPE) was verbally reported immediately following each sprint using Borg's 6–20 RPE scale.[18]

Upper body kinematics were measured using a 10-camera Vicon motion analysis system (200 Hz: MX T40-S, Vicon Motion Systems Limited) alongside ergometry data during the Pre-ISP and Post-ISP protocols. Eighteen retro-reflective markers (14 mm, B&L Engineering) were placed on the anatomical landmarks of the trunk (seventh cervical vertebrae, eighth thoracic vertebrae, incisura jugularis and xiphoid process), and bilateral upper limbs (medial and lateral epicondyle) of the players according

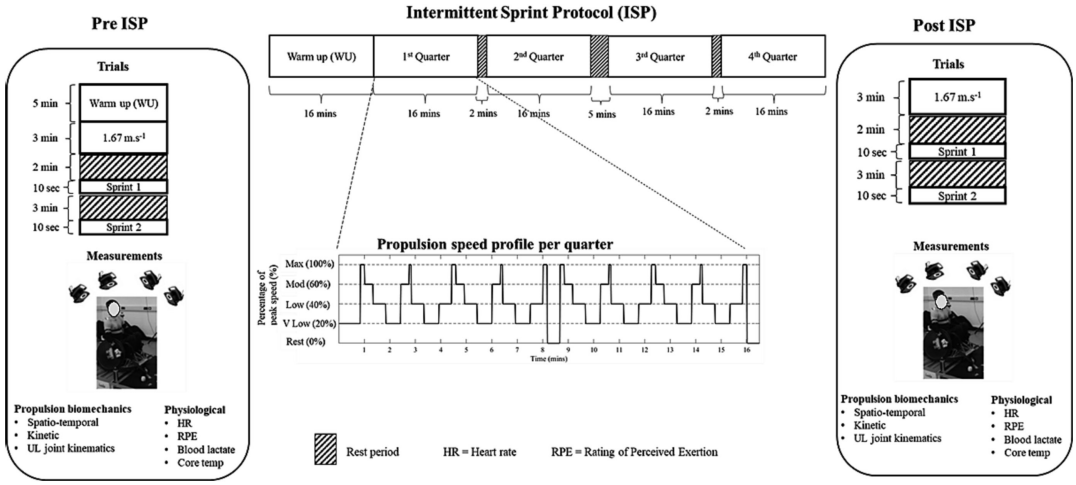

FIGURE 1 Experimental procedure. Physiological data and biomechanical measurements of submaximal and sprint propulsion were taken before and immediately after a wheelchair rugby specific intermittent sprint protocol (ISP). HR, heart rate; RPE, Rating of perceived exertion; UL, Upper limb.

to International Society of Biomechanics (ISB) recommendations.[19] Single markers were attached using sports adhesive spray and double-sided hypoallergenic tape. A four-marker cluster was placed on each upper arm using strapping. The acromion marker cluster (AMC) method was used to track scapular motion to enable the estimation of glenohumeral kinematics.[20] Previous work has established the validity and reliability of the AMC method for humeral elevation up to 90°[20,21] and as a reliable method during wheelchair propulsion.[16] The Optimal Common Shape Technique (OCST)[22] was used to account for soft tissue artifacts of the upper arms during a bilateral shoulder circumduction trial. In addition, glenohumeral joint centres were determined by the Symmetrical Centre of Rotation Estimation (SCoRE) method from the circumduction trial[23] in accordance with previous wheelchair sprinting research.[3] Where possible, markers remained in situ during the entire data collection and subject calibration were repeated Post-ISP.

2.4 | Data analysis

Sprint performance outcome parameters (peak velocity and total distance) and kinetic and kinematic data were extracted for both groups during two phases of the sprints, the acceleration phase represented by the first three pushes and the maximal velocity phase which included the propulsion cycle during which peak velocity was reached and one cycle on either side[3,15] (see Figure 2 for an illustration and parameters calculated). The start and end of each propulsion cycle was identified using an ergometer roller torque cutoff of 1 Nm based on previous studies.[3,15] Custom written MATLAB scripts (Matlab R2021a, The Mathworks Inc.) were used for all further data processing and analysis. Spatio-temporal and kinetic data derived from the dual roller wheelchair ergometer included bilateral contact angles, peak forces, and peak powers.[3,15] An eighth-order Butterworth filter with a cutoff of 10 Hz filtered force data. A fourth-order, low-pass Butterworth filter with a cutoff frequency of 7 Hz filtered motion analysis data. Filter cutoff frequencies were determined by residual analysis and in line with previous wheelchair sprinting research.[3,24] The orientation of the thorax (thorax to global) was calculated in accordance with ISB recommendation[19] and glenohumeral (humerus to scapular) joint motion was determined using a ZXY rotation sequence.[25] Discrete kinematic data selected for analysis were peak angles and range of motion (ROM) for thorax flexion, glenohumeral flexion, and abduction as these parameters have previously been associated with alterations with pain, propulsion speed, and fatigue.[3,8,26,27] Inter-limb asymmetries were calculated for the biomechanical parameters described above using the symmetry index (SI) (Equation 1).[14,15,28] The SI reports asymmetry as a percentage whereby, 0% denotes perfect symmetry.

$$SI = \frac{|\,Dom - NDom\,|}{Dom} \times 100 \quad (1)$$

Note: SI = Symmetry index, Dom = Value from the dominant limb, NDom = Value from the non-dominant limb.

2.5 | Statistical analysis

All statistical analyses were performed using the Statistical Package for Social Sciences (SPSS version 27, IBM Corporation). Separate two-way mixed analyses of variance were used to determine main effects for time (Pre_ISP, Post_ISP), impairment (SCI, Non-SCI), and a time × group interaction for each dependent variable. Data normality, homogeneity of variance, and sphericity were assessed by Shapiro–Wilk tests, Levene's test, and Mauchly's test of sphericity, respectively. Differences in how each group altered propulsion biomechanics parameters over time were identified as significant (time × group) interactions. The alpha level was set at $p < 0.05$. For parameters that had a significant interaction effect post hoc *t*-tests, with a Bonferroni correction, were performed for each participant group to establish where differences occurred. This enabled the change in propulsion biomechanics to be evaluated for each impairment group separately via paired *t*-tests. Independent *t*-tests examined group differences at each time point. Effect sizes were calculated using Partial eta squared (η^2) for the ANOVA outputs and Cohen's d was to determine the magnitude of the post hoc effects, which were classified as small ($d = 0.2$), moderate ($d = 0.5$) and large ($d = 0.8$).[29]

3 | RESULTS

3.1 | Physiological and sprint performance changes

Significant main effects for time (Post ISP-Pre ISP) indicated that BLa, HR, and RPE increased post-ISP compared to pre-ISP levels (Table 1). No significant main effect for group or interaction effect was observed for BLa, HR, and RPE. No significant main effect for time, impairment, or interaction effect was observed for any sprint performance outcome variable (V_{max} or distance)

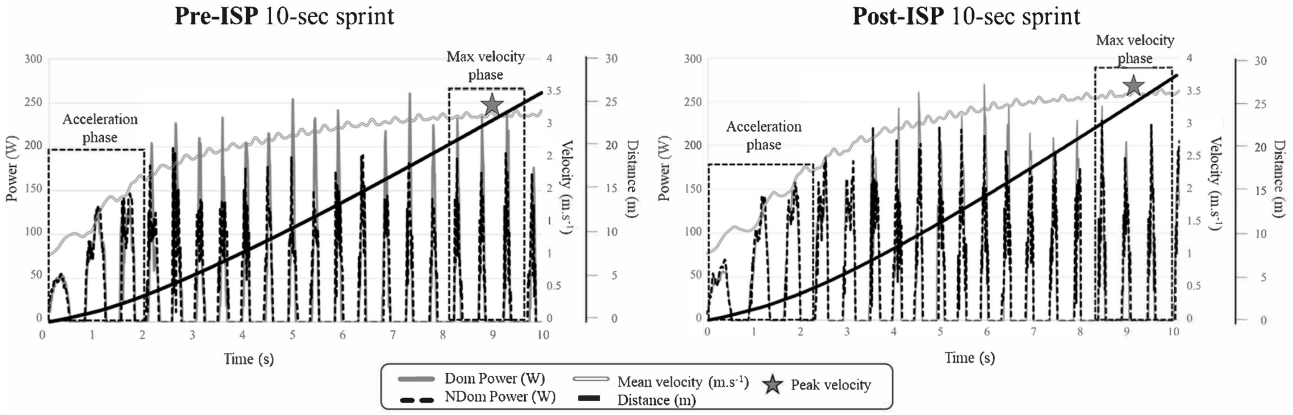

FIGURE 2 Representative mean velocity, distance, and power (dominant limb and nondominant limb) data of the whole 10-s sprint before the intermittent sprint protocol (Pre-ISP) and immediately following the ISP (Post-ISP). Highlighted view of the first three pushes (Acceleration phase) and the maximal velocity phase analyzed in this study.

during the acceleration phase or maximal velocity phase (Table 1).

3.2 | Propulsion biomechanical changes

Significant main effects for time (pre-ISP vs. post-ISP) were identified for nine biomechanical parameters of sprinting (Tables 2 and 3). Post ISP, players significantly reduced peak thorax flexion ($p = 0.016$, $\eta^2 = 0.373$, mean change = −5.1°) and glenohumeral (GH) abduction ($p = 0.001$, $\eta^2 = 0.565$, mean change −5.1°) but increased contact angles ($p < 0.001$, $\eta^2 = 0.650$, mean change −5.1°), contact angle asymmetries ($p = 0.044$, $\eta^2 = 0.277$, mean change −5.1°), and GH flexion asymmetries ($p = 0.041$, $\eta^2 = 0.283$, mean change = 1.5°) during the acceleration phase of sprinting. During the maximal velocity phase of sprinting players reduced peak thorax flexion ($p = 0.003$, $\eta^2 = 0.497$, mean change = −5.8°) and GH abduction ($p = 0.031$, $\eta^2 = 0.311$, mean change = −8.1°) but increased GH abduction ROM ($p = 0.007$, $\eta^2 = 0.444$, mean change = 16.9°) and abduction asymmetries ($p = 0.046$, $\eta^2 = 0.273$, mean change 20.2%) following the ISP.

Significant main effects for impairment (Table 3) showed players with non-SCI impairments displayed greater GH peak abduction during both the post-ISP acceleration phase and post-ISP max velocity phase of sprinting than those with SCI ($p = 0.018$, $d = 1.4$, mean difference = 12.1° and $p = 0.027$, $d = 1.3$, mean difference = 6°, respectively). Players with non-SCI displayed significantly greater glenohumeral flexion ROM ($p = 0.029$, $d = 1.3$, mean difference > 10.9°) and abduction peak angles ($p = 0.010$, $d = 1.6$, mean difference > 8.1°) during the pre-ISP and post-ISP maximal velocity phase of the sprint than those with SCI.

Significant interaction effects (Tables 2 and 3) and pairwise comparisons revealed players with SCI significantly increased asymmetries in both peak power ($p = 0.003$, $d = 1.9$, mean change = 6.9°) and GH abduction ROM ($p = 0.009$, $d = 1.5$, mean change = 15.1°) during the acceleration phase of sprinting post-ISP. However, neither parameter differed significantly in individuals with non-SCI ($p = 0.152$ and $p = 0.785$) for peak power and GH abduction ROM asymmetries, respectively. The different patterns of peak power and GH abduction asymmetries alterations between two players (SCI and non-SCI) are clearly shown in Figure 3.

4 | DISCUSSION

This novel study evaluated the ISP-induced physiological changes as well as performance and biomechanical alterations of wheelchair sprinting in elite WR players. Contrary to the first study hypothesis players maintained peak velocity and distance during wheelchair sprinting post-ISP. In support of the second hypothesis, players displayed alterations in kinetics, joint kinematics, and asymmetries of wheelchair sprinting following the ISP. Thus, this work provides insights into WR rugby players' wheelchair propulsion technique following simulated match play to maintain sprint performance.

Players significantly increased blood lactate concentration, RPE, and HR following the ISP, indicating the development of physiological fatigue. Yet, sprint performance was not impaired. Specifically, all players reported a high level of exertion (RPE >16) which was comparable to peak values reported during WR match play.[30] Furthermore, the increase in blood lactate values (pre- to post-ISP) exceeded those reported by athletes with greater physical capacity than the current study during wheelchair basketball

TABLE 1 Physiological and wheelchair sprint performance changes following the intermittent sprint protocol (ISP) overall ($n = 15$), those with SCI ($n = 7$) and non-SCI ($n = 8$). Data reported as mean(SD).

				ANOVA		
Variable	**Group**	**Pre ISP**	**Post ISP**	**Time**	**Imp.**	**Time × Imp.**
Physiological changes						
Blood lactate ($mmol\,l^{-1}$)	Overall	1.35 (0.54)	5.43 (2.87)	**<0.001**	0.174	0.088
	SCI	1.31 (0.53)	4.22 (2.22)			
	Non-SCI	1.39 (0.58)	6.49 (3.07)			
RPE	Overall	15 (2)	17 (2)	**0.001**	0.764	0.788
	SCI	15 (2)	17 (2)			
	Non-SCI	15 (2)	17 (2)			
Heart rate ($b\,min^{-1}$)	Overall	116 (21)	132 (21)	**0.027**	0.718	0.203
	SCI	113 (25)	139 (24)			
	Non-SCI	119 (17)	127 (18)			
Sprint performance changes						
Acceleration phase						
Peak Vel.($m\,s^{-1}$)	Overall	2.34 (0.28)	2.43 (0.39)	0.268	0.310	0.530
	SCI	2.19 (0.33)	2.23 (0.41)			
	Non-SCI	2.47 (0.15)	2.61 (0.28)			
Distance (m)	Overall	2.1 (0.5)	2.2 (0.6)	0.927	0.320	0.464
	SCI	2.1 (0.5)	2.0 (0.6)			
	Non-SCI	2.2 (0.3)	2.3 (0.6)			
Overall						
Peak Vel. ($m\,s^{-1}$)	Overall	3.79 (0.62)	3.79 (0.59)	0.991	0.087	0.227
	SCI	3.47 (0.65)	3.54 (0.67)			
	Non-SCI	4.07 (0.46)	4.00 (0.44)			
Distance (m)	Overall	41.4 (9.3)	42.3 (8.5)	0.510	0.172	0.995
	SCI	38.1 (12.4)	39.1 (8.9)			
	Non-SCI	44.2 (4.7)	45.2 (7.6)			

Note: Bold text indicates statistical significance $p < 0.05$.

Abbreviations: Imp, Impairment; RPE, rating of perceived exertion.

match play.[31] Given these significant physiological responses, the absence of sprint performance changes was partially unexpected. High-intensity intermittent sports, such as WR match play, have been associated with fatigue development which is typically reflected in a functional decline in the ability to express maximal force and power, possibly impairing sprint performance.[32] Indeed, Sarro et al.[6] reported reductions in distance traveled and average velocities in the second half of WR match play. However, the current findings support more recent work from our laboratory whereby activity profiles of elite WR players did not change during match play[2] and repeated sprint ability was maintained throughout simulated match play on an ergometer.[13] One possible explanation for these findings is the advancement of sports science support, training time, and higher standard of playing nation, competition and coaching compared to Sarro et al's[6] work in 2008. Thereby, leading to better physical capabilities and propulsion skills which facilitate players' ability to adapt to the demands of WR match play and maintain performance.

During the acceleration phase of sprinting, players propelled their wheelchairs with reduced peak thorax flexion and glenohumeral abduction but utilized larger contact angles, alongside greater contact angle asymmetries, and glenohumeral flexion asymmetries following the ISP. These kinematic alterations are likely interrelated. Increasing contact angles can facilitate the high push rim forces and power which are necessary to overcome the inertia of the wheelchair-user and maximally accelerate the wheel. Evidence suggests, wheelchair athletes typically achieve these favorable technique alterations by increasing thorax flexion and to a lesser extent shoulder flexion during the acceleration phase of sprinting when in an unfatigued state.[3,33] Furthermore,

TABLE 2 Spatiotemporal and kinetic changes during wheelchair sprinting following the intermittent sprint protocol (ISP) overall ($n = 15$), those with SCI ($n = 7$) and non-SCI (n = 8). Data reported as mean(SD).

Variables	Group	Wheelchair sprinting									
		Acceleration phase					Maximum velocity phase				
		Pre	Post	Time	Imp	Int.	Pre	Post	Time	Imp	Int.
Push freq. (Hz)											
	Overall	1.8(0.3)	1.8(0.4)	0.774	0.545	0.583	2.5(0.6)	2.3(0.5)	0.145	0.763	0.946
	SCI	1.8(0.3)	1.7(0.4)				2.5(0.8)	2.3(0.5)			
	Non-SCI	1.9(0.4)	1.9(0.5)				2.6(0.3)	2.4(0.6)			
Contact angle (°)											
Mean (°)	Overall	121.1(28.2)	145.2(35.7)	**<0.001**	0.053	0.237	101.8(21.0)	100.1(30.0)	0.896	0.058	0.062
	SCI	133.9(19.4)	164.6(31.0)				106.0(18.3)	118.4(28.7)			
	Non-SCI	110.0(31.1)	128.3(32.0)				98.2(23.8)	84.0(21.4)			
Asymmetry (%)	Overall	5.5(3.9)	9.8(8.5)	**0.040**	0.376	0.924	12.5(10.4)	10.5(12.7)	0.389	0.568	0.261
	SCI	4.0(2.4)	8.4(9.3)				12.3(9.3)	7.2(5.0)			
	Non-SCI	6.9(4.5)	11.0(8.3)				12.6(11.9)	13.4(16.7)			
Pk. Force (N)											
Mean (N)	Overall	121.9(37.7)	125.3(47.2)	0.758	0.155	0.918	79.1(23.4)	85.0(27.8)	0.188	0.285	0.054
	SCI	106.4(44.0)	111.0(50.1)				76.0(23.1)	73.2(19.3)			
	Non-SCI	135.5(27.1)	137.8(43.8)				81.7(24.9)	95.3(31.0)			
Asymmetry (%)	Overall	10.0(10.9)	7.2(5.1)	0.326	0.568	0.099	16.2(9.9)	17.4(10.4)	0.712	0.522	0.683
	SCI	6.5(4.2)	8.4(5.4)				18.4(9.0)	18.3(13.1)			
	Non-SCI	12.9(14.1)	6.2(5.0)				14.2(10.9)	16.7(8.1)			
Pk. Power (W)											
Mean (W)	Overall	188(70)	198(76)	0.552	0.319	0.849	284(137)	314(133)	0.169	0.159	0.406
	SCI	167(86)	181(98)				242(129)	253(83)			
	Non-SCI	206(53)	213(53)				321(142)	267(151)			
Asymmetry (%)	Overall	10.7 (4.6)	12.0(4.7)	0.348	0.844	**0.007**	18.6(9.3)	18.5(11.8)	0.943	0.264	0.764
	SCI	8.0(2.6)	14.9(3.3)				22.0(6.2)	20.8(14.8)			
	Non-SCI	13.0(4.9)	9.4(4.4)				15.7(10.9)	16.5(8.9)			

Note: Bold values indicate statistical significance $p < 0.05$.

Abbreviations: Imp, Impairment; SCI, spinal cord injury.

TABLE 3 Upper body kinematic changes during wheelchair sprinting following the intermittent sprint protocol (ISP) overall ($n = 15$), those with SCI ($n = 7$) and non-SCI ($n = 8$). Data reported as mean(SD).

		Acceleration phase					Maximum velocity phase				
Variables	**Group**	**Pre**	**Post**	**Time**	**Imp**	**Int.**	**Pre**	**Post**	**Time**	**Imp**	**Int.**
Thorax Pk. flexion (°)	Overall	37.8(9.4)	32.7(8.9)	**0.016**	0.118	0.717	40.6(13.1)	34.8(14.2)	**0.003**	0.196	0.991
	SCI	33.8(9.0)	29.4(6.8)				35.8(12.9)	29.9(13.1)			
	Non-SCI	41.4(8.7)	35.7(9.9)				44.9(12.6)	39.0(14.5)			
Flex/Ext. ROM (°)	Overall	21.4(6.8)	21.0(6.6)	0.861	0.068	0.255	9.9(3.1)	10.7(3.3)	0.483	0.769	0.681
	SCI	17.4(4.0)	18.9(6.3)				10.4(3.9)	10.7(2.4)			
	Non-SCI	24.9(7.0)	22.8(6.8)				9.5(2.3)	10.8(4.1)			
GH Pk. flexion (°)	Overall	28.6(11.6)	27.1(14.7)	0.801	0.076	0.378	31.5(13.9)	25.3(17.6)	0.297	**0.026**	0.9
	SCI	21.6(7.7)	24.7(5.6)				24.1(15.0)	18.7(7.9)			
	Non-SCI	34.7(11.2)	29.3(19.8)				38.0(9.5)	31.1(22.1)			
Flex/Ext. ROM (°)	Overall	56.5(14.4)	52.1(14.2)	0.179	0.109	0.42	59.8(21.7)	47.9(14.0)	0.084	**0.01**	0.319
	SCI	49.3(6.9)	47.6(7.1)				47.2(14.8)	42.1(5.3)			
	Non-SCI	62.8(16.7)	56.0(14.2)				70.7(21.3)	53.0(17.5)			
Asymmetry (%)	Overall	15.9(11.2)	25.3(13.7)	**0.041**	0.604	0.92	25.8(18.2)	22.7(22.0)	0.556	0.476	0.18
	SCI	14.2(11.7)	24.1(8.5)				33.7(21.8)	21.4(27.9)			
	Non-SCI	17.3(11.4)	26.4(17.7)				18.9(11.6)	23.9(17.3)			
GH Pk. abduction (°)	Overall	47.2(10.2)	42.1(10.9)	**0.001**	**0.018**	0.941	49.5(11.5)	41.4(15.5)	**0.031**	**0.027**	0.086
	SCI	40.8(9.1)	35.7(7.1)				46.3(11.6)	31.2(5.7)			
	Non-SCI	52.7(7.8)	47.8(10.8)				52.3(11.4)	50.0.4(16.0)			
Add/Abd. ROM (°)	Overall	25.5(8.9)	25.8(12.5)	0.975	0.665	0.298	26.1(7.6)	43.0(21.6)	**0.007**	0.851	0.86
	SCI	28.1(11.4)	25.7(12.7)				24.9(8.5)	42.8(25.0)			
	Non-SCI	23.3(6.0)	25.8(13.1)				27.1(7.2)	43.1(20.0)			
Asymmetry (%)	Overall	17.6(12.2)	22.2(10.1)	0.141	0.333	**0.011**	27.0(21.1)	47.2(25.6)	**0.046**	0.781	0.452
	SCI	10.2(9.4)	25.3(11.8)				21.9(14.6)	50.0(26.2)			
	Non-SCI	24.1(11.0)	19.5(8.1)				31.4(25.7)	44.9(26.6)			

Note: Bold values indicate statistical significance.

Abbreviations: Imp, Impairment; SCI, spinal cord injury.

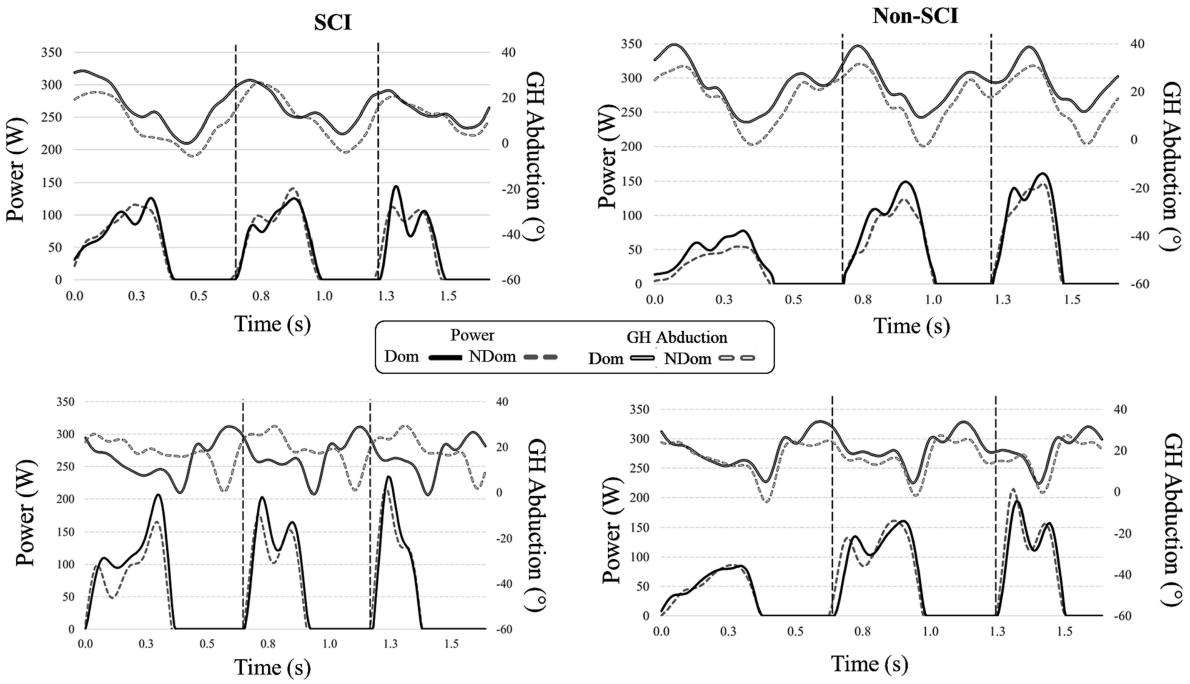

FIGURE 3 Typical example of the different alterations in peak power and glenohumeral (GH) abduction asymmetries following the intermittent sprint protocol (ISP) between two players (spinal cord injury [SCI] and non-spinal cord injury [Non-SCI] impairment). Power and GH abduction over the dominant arm and non-dominant arm displayed over the first three propulsion cycles of the Pre-ISP and Post-ISP sprint.

previous work investigating submaximal daily propulsion reported that individuals increased both trunk and shoulder flexion and push rim forces when fatigued.[8] In contrast, the current study suggests increases in contact angle and the maintenance of propulsion forces post ISP coincide with larger contact angle and glenohumeral flexion asymmetries during the acceleration phase of sprinting. Thus, these alterations may reflect a specific kinematic strategy to maintain acceleration performance whereby WR players shift toward the dominant hand to facilitate a larger contact angle to meet the force/power expression demand of accelerating the wheelchair.

During the maximal velocity phase of sprinting, players exhibited lower peak thorax flexion and glenohumeral abduction but increased both the ROM and asymmetries of glenohumeral abduction/adduction following the ISP. The large number of biomechanical alterations during both phases of sprinting alongside the absence of changes in sprint performance following the ISP, suggest that players can maintain the gross features of sprinting but propel their wheelchair differently. The decreased peak thorax flexion and glenohumeral abduction during the maximal velocity phase were comparable alterations to that observed during the acceleration phase. Previous studies indicate that lower glenohumeral abduction may increase subacromial space in which structures such as rotator cuff and biceps tendon reside, potentially reducing acute stress on these structures.[34,35] This alteration is comparable with that exhibited by athletes with greater shoulder pain during sprinting[3] and aligns with the protective response hypothesis.[36] This theory proposes that during tasks that may provoke painful/fatigue symptoms the nervous system searches for movement patterns that are less painful by constraining motion at the painful/fatigued joint/area.[36] Consequently, it appears WR players adapt to physiological fatigue, induced by an ISP by reducing shoulder abduction, thereby minimizing acute pain/perceived threat of pain/fatigue at the shoulder during sprinting. However, it should be noted that players did not change either contact angle and glenohumeral flexion asymmetries during the maximal velocity phase instead became more asymmetrical in glenohumeral abduction. This subtle alteration is likely due to the lower forces and smaller contact angle requirements during this phase of sprinting resulting from a higher wheel velocity and coupling difficulties between the hand and push-rim,[3] meaning large asymmetrical technique adjustments are not required.

Regarding impairment-specific alterations, during the acceleration phase of sprinting players with SCI significantly increased asymmetries in both peak power and glenohumeral abduction post ISP. Whereas these parameters were not significantly altered in those with non-SCI. Several explanations for this finding may exist.

The SCI group consisted of individuals with tetraplegia (levels C7 to C5) who possess less muscle mass because of impairment.[37] As a result, SCI players may be susceptible to earlier physiological fatigue leading to a greater duration of the ISP performed with adjusted propulsion biomechanics. Additionally, there are inherent differences in propulsion techniques between WR players with SCI and non-SCI. For instance, high-point players contact the push rim with the palm of their hands, while low-point players are more likely to adopt a backhanded technique and contact the push rim with the dorsum of their hands.[15] Thus, the use of the backhanded technique may be more susceptible to simulated match play induced alterations to kinetic and kinematic of sprinting. Finally, the lack of trunk function in the SCI group may lead to athletes relying more on inter-limb asymmetries to adapt wheelchair propulsion biomechanics. It is widely known that during movement tasks, such as wheelchair propulsion, individuals with low trunk muscle strength often compensate for lack of trunk control by increasing the recruitment of shoulder and arm musculature.[38,39] Contrary to this, we speculate that the non-SCI group may possess a wider range of options to achieve the movement task outcomes during the ISP. This was reflected by the large inter-individual differences in biomechanical alterations within the non-SCI group compared to those with SCI. Nevertheless, these findings indicate the need for coaches and researchers to consider players' impairment type when monitoring and developing sports propulsion skills under fatigue.

A notable finding of the current study was the propensity for players to propel with larger kinematic asymmetries during sprinting following the ISP. Greater kinematic asymmetry may unequally distribute the forces within the upper limbs thereby influencing chronic stress imposed by wheelchair propulsion. However, little empirical data are available to support this hypothesis. Although, recent evidence from our laboratory suggests that WR athletes with SCI possessed thicker supraspinatus tendons than those with non-SCI thereby implicating differences in chronic tendon adaptations between these groups.[40] Thus, the additional asymmetrical alterations reported in those with SCI compared to those without SCI may be one factor that contributes to these tendon differences.[40] Alternatively, increased asymmetries may be another aspect of the wider strategy utilized by WR players to protect the upper limb and maintain sprint performance following an ISP. Furthermore, these findings may indicate that each limb responds differently to physiological fatigue. That said, it should be clarified that the cause and consequence of these biomechanical alterations during sprinting cannot be stated with confidence.

4.1 | Limitations

An assumption of this work was that the ISP could induce fatigue resulting in a decline in forces generated by shoulder muscles. However, changes in muscle activity and/or strength were not assessed. Thus, no comment can be made regarding ISP-induced neuromuscular fatigue. Nevertheless, it should also be reiterated that the purpose of this research was to investigate sprint performance and biomechanical alterations following simulated WR match play. Despite efforts made to minimize markers falling off, unfortunately, due to excessive sweating in some players some markers did become detached and fell the markers fell off. Where this occurred the markers were replaced by the same investigator who had originally positioned the markers at the start of the protocol. Furthermore, the bilateral circumduction and anatomical trials were repeated post-ISP. Due to the use of glenohumeral kinematics rather than humerothoracic angles, caution is necessary when interpreting our findings. Although validated for typical upper limb movements during wheelchair propulsion,[21] the accuracy of the AMC method for fast sporting motions, such as wheelchair sprinting, remains unclear and warrants further investigation. In accordance with the wider literature the testing protocol examined propulsion biomechanics before and immediately after the ISP without quantifying any alterations during the ISP. Therefore, further work is necessary that investigates sprinting biomechanics throughout the ISP which would help establish if there is a pivotal period where sprint propulsion biomechanics change during the ISP or whether changes are gradual. Furthermore, the ISP was designed to reflect an entire match on court. However, due to laboratory constraints no ball handling and other technical skills relevant to on court match play were performed. That said, a strength of this study was that these biomechanical alterations were observed despite the constraints of sprinting on an ergometer. Finally, it should be noted that in competition for tactical purposes and if performance is deteriorating players would be substituted.

5 | PERSPECTIVES

Coaches, practitioners, and researchers should note that despite physiological fatigue, induced by the total duration of an ISP, elite WR players can maintain sprint performance outcomes (peak velocity and distance) by modifying how they propel their wheelchair. Overall, players appear to reduce peak thorax and glenohumeral abduction but increase kinetic and kinematic asymmetries following simulated match play. Therefore, WR practitioners should consider quantifying these

biomechanical parameters when monitoring and developing sports propulsion skills under fatigue. Furthermore, given specific alterations in asymmetries differ between impairment types and phases of sprinting any applications of this work should account for a player's impairment type.

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CONFLICT OF INTEREST STATEMENT

There are no conflicts of interest associated with this research.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on reasonable request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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