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CASE REPORT

A case report of shoulder fatigue imbalance in wheelchair rugby: implications to pain and injury

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The purpose of this case–control study was to examine potential agonist–antagonist fatigue imbalance during wheelchair rugby activity. A 16-channel NORAXON electromyography (EMG) system 1400A with telemetry was used to assess EMG activity in a 39-year-old male with a C6 complete spinal cord injury (American Spinal Injury Association Impairment Scale (AIS) A), 17.5 years post injury. Mean amplitude and median frequency were determined for push-phase agonists (anterior deltoid and pectoralis major) and antagonist (posterior deltoid) across four training sessions at a community fitness and Paralympic training facility. Unlike continuous wheelchair pushing, acute muscle imbalances between agonists and antagonists (that is, push and recovery muscles) were not demonstrated. Wheelchair sports such as rugby may reduce risk of shoulder pain and overuse injury due to intermittent activity rather than continuous pushing. The current study is one of the first to document sport fatigue through electromyography during intermittent, live play rather than clinical conditions (that is, continuous pushing).

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INTRODUCTION

Wheelchair sport is a growing component of rehabilitation and post-rehabilitation services for individuals with spinal cord injuries.[1] Electromyography (EMG) analysis has been used in wheelchair sport to identify sport-specific prime movers and onset of fatigue in attempts to build effective training and rehabilitation methods for participants.[2] To date, most EMG studies have examined sport participants in a clinical setting, assessing continuous, cyclical exercise that is atypical of many wheelchair sports. Although these data have helped to identify prime movers during the push and recovery phases of forward wheelchair motion, they are limited in the ability to examine fatigue and its associated ramifications under actual training and gameplay conditions (for example, start/stop, braking and so on).

Fortunately, EMG technology and analyses have been extended to non-laboratory settings, allowing the assessment of wheelchair sport mechanics during actual therapeutic exercise and training.[2,3] This advancement has substantiated the contribution of agonist–antagonist 'imbalance' to shoulder joint pain and injury risk. Stated differently, continuous wheelchair pushing results in muscle fatigue of wheelchair push-phase agonists (that is, shoulder flexors) without concomitant muscle fatigue in recovery-phase antagonists (that is, shoulder extensors). As a result, unnatural joint motion and stresses occur at the shoulder joint, increasing risk of pain and injury.[3,4]

It is clear that continuous pushing, a common rehabilitation and post-rehabilitation therapeutic exercise, leads to agonist–antagonist fatigue imbalance both acutely and chronically; however, the effects of alternative therapeutic exercises on fatigue imbalance, such as wheelchair sport, are just starting to be explored. It is unknown if wheelchair sport activity leads to greater, similar or lesser imbalance than typical therapeutic exercise (that is, continuous pushing). Therefore, the purpose of this study was to examine agonist–antagonist imbalance during actual wheelchair rugby training. Findings from the current study will identify if wheelchair rugby is similar to, or distinct from, traditional rehabilitation and post-rehabilitation therapeutic exercise (that is, continuous pushing) in the ability to reduce shoulder joint paint and injury in persons with spinal cord injury.

MATERIALS AND METHODS

Participant

A wheelchair rugby player was recruited through a Paralympic Training site. Rugby was chosen because the sport is typical of intermittent wheelchair sport. The athlete was a 39-year-old male with a complete C6 spinal cord injury (AIS Grade A). He was 17.5 years post injury and trained 12 h per week. He had a 0.5 player classification and was left-side dominant with a seating tilt of 70°. This player was studied because the limited active musculature allowed researchers to examine fatigue under the greatest potential push and recovery imbalance (that is, limited synergist assistance from the triceps). Institutional Review and player consent were obtained before data collection. 'We certify that all applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during the course of this research.'

Instruments

A 16-channel Noraxon EMG system 1400A (Scottsdale, AZ, USA) with telemetry (Telemyo DTS) was used. Electrodes were placed on the dominant side of the body guided by the SENIAM project. All electrodes were placed parallel to the muscle fibers following standard skin preparation that included shaving, mild skin abrasion and alcohol rubs.

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EMG processing

Raw EMG signals were digitally filtered using a band-pass filter of 20 and 500 Hz, to minimize movement artifacts. Signals were subsequently rectified and low-pass filtered using a fourth order low-pass Butterworth filter with a cutoff frequency of 5 Hz. Fast Fourier transfer analysis with analytic windows of 0.5 s width were utilized to estimate mean power frequency slope with respect to time. EMG data were normalized against maximal voluntary isometric contractions (MVC) performed at the beginning of each day. Since each EMG channel had multiple firings (bursts), individual files were visually inspected to a 20% threshold above baseline signal (ensemble averaging). During training, the short-time Fourier transform algorithm was used to analyze scores.

Procedures

EMG data were collected during five rugby sessions across 2 consecutive days. The case subject was participating in a pre-determined rugby camp, which enabled the researchers to examine EMG activity under extensive sport training (that is, 15 total hours). Training day 1 consisted of MVC tests and two 3-h training sessions separated by lunch. Training day 2 consisted of the MVC tests followed by three 2-h training sessions separated by lunch and dinner. Each 3-h training session consisted of an hour of team strategy, an hour of sport-specific skill development and an hour of rugby activity (that is, gameplay). EMG activity was collected and evaluated only during gameplay because dynamic EMG measurement requires that the activity conditions be similar.[5] Gameplay was divided into 8-min work bouts (that is, 8-min scrimmages). We assessed EMG activity for 60 s at the start of gameplay and repeated the assessment at the conclusion of each 8-min scrimmage. The participant was allowed to rest and data collection was delimited to actual court time.

The participant practiced the MVC test for shoulder flexors (that is, anterior deltoid (AD) and pectoralis major (PM)) and shoulder extensors (that is, posterior deltoid (PD)) on the evening before training day 1. The following morning, researchers completed standard skin preparation procedures and placed sensors on these three muscles. The MVC included a 5-s resting baseline followed by a 5-s maximal contraction. Two MVC trials on the AD, PD and PM were administered with 1-min of rest between. On training day 2, standard skin preparation and sensor placement were made before MVC testing of the same muscles.

Analysis

Joint analysis of the EMG spectrum and amplitude was used to assess agonist–antagonist imbalance. (5) This analysis allows the therapist or physician to simultaneously examine mean amplitude and median frequency to detect muscle fatigue (increase in mean amplitude and decrease in median frequency) and to identify muscles that compensate for fatigue through increased force generation (increase in mean amplitude and median frequency). To maximize external validity of findings, we examined imbalance under two conditions: (1) across each individual gameplay session (that is, change in EMG activity from the initial gameplay minute to the final minute), and (2) within each individual training session (that is, from the initial scrimmage minute to the last for each 8-min interval). We chose to examine imbalance across each session because competitive and recreational sport typically run start to finish, regardless of fatigue. We also chose to examine imbalance within individual training sessions because this information is likely more meaningful for therapists and physicians who use intervals of therapeutic exercise (for example, an 8-min sport session) for specific outcomes. An algorithm was used to quantify mean amplitude (http://www.mathworks.com/matlabcentral/fileexchange/9223-simpleeda-emg/content/simpleEMG.m).

RESULTS

The participant rested the majority of one training session; therefore data across 4 (rather than 5) training sessions are presented. Specific to data analysis across training sessions, EMG activity reflected agonist–antagonist balance rather than imbalance (Table 1). Our data reflected nearly identical muscle fatigue or force decreases (leading to fatigue) exhibited between the antagonist, PD and the shoulder flexor muscles (AD, PM) across gameplay sessions. Only in session 4 did agonist–antagonist EMG activity vary, a change that may be explained by the extensive on-court time of the participant (that is, ~15 h of training).

When examining imbalance within individual training sessions, there was a symbiotic relationship between agonists and antagonists. Table 2 reflects EMG activity for session 3 and demonstrates the increased force generation by the PD when fatigue was exhibited in the AD or PM. In addition, the AD increased force production when the PD fatigued. These data may demonstrate that agonists and antagonists compensate for opposing muscle fatigue to maintain joint stability and force generation during wheelchair sport. This finding supports the potential of intermittent wheelchair sport activity (rugby in this case) to reduce risk of acute muscle imbalances that lead to pain and injury typically seen in continuous wheelchair pushing.

DISCUSSION

In the current study, a wheelchair rugby participant demonstrated fatigue in shoulder joint agonist (that is, push-phase) and antagonist (that is, recovery phase) muscles during sport participation. As a result, acute muscle fatigue imbalance was not present either across or within training sessions, reducing risk of acute shoulder pain and injury compared with traditional continuous pushing modalities.[3] Findings from the current study, unique in that fatigue data were collected under actual gameplay conditions, support the use of wheelchair sport as an alternative to traditional rehabilitation and post-rehabilitation exercise. Additional empirical evaluations of wheelchair sport are warranted but the lack of fatigue imbalance typically demonstrated by continuous pushing is promising.

Table 1. EMG results across individual training sessions

Session		*PM*		*AD*		*PD*	
		MDF	*AMP*	*MDF*	*AMP*	*MDF*	*AMP*
1	Initial	60	134	93	232	72	71
	Final	58	80	87	270	41	60
	Outcome	Force decrease		Fatigue		Force decrease	
2	Initial	61	62	84	199	64	34
	Final	59	65	78	223	58	102
	Outcome	Fatigue		Fatigue		Fatigue	
3	Initial	55	62	83	219	55	132
	Final	64	24	93	206	62	117
	Outcome	Muscle recovery		Muscle recovery		Muscle recovery	
4	Initial	63	66	89	210	68	234
	Final	57	70	92	210	67	119
	Outcome	Fatigue		No change		Force decrease	

Abbreviations: AD, anterior deltoid; AMP, mean amplitude (mV); MDF, median frequency (Hz); PD, posterior deltoid; PM, pectoralis major. Initial reflects the first gameplay minute and final represents the last gameplay minute of each hour-long training session.

Table 2. EMG activity during training session 3

	PM			*AD*			*PD*		
Collection	*MDF*	*AMP*	*Outcome*	*MDF*	*AMP*	*Outcome*	*MDF*	*AMP*	*Outcome*
Initial	55	62		83	219		55	132	
2	60	47	Recover	88	239	[a]Force ↑	66	143	[a]Force ↑
3	59	26	Force ↓	89	217	Recover	69	168	[a]Force ↑
4	62	22	Recover	95	239	[a]Force ↑	70	109	Recover
5	64	24	Recover	93	206	Force ↓	62	117	Fatigue

Abbreviations: AD, anterior deltoid; AMP, mean amplitude (mV); MDF, median frequency (Hz); PD, posterior deltoid; PM, pectoralis major. Data were collected approximately every 8 min.
[a]Muscle compensation.

Compared with previous work in the field, muscle fatigue in the AD is consistent with the literature. However, fatigue in the antagonist PD and limited fatigue in the PM is distinct from findings on typical rehabilitation and therapeutic exercise. This outcome may explain why imbalance was not present. Continuous pushing typically does not cause fatigue in antagonists musculature, which in turn results in altered joint mechanics and ultimately joint pain and injury. However, the PD muscle demonstrated fatigue both across training sessions (from initial minute to final minute) as well as within training sessions. This finding supports the use of intermittent sport activity as a viable therapeutic exercise and rehabilitation alternative to continuous pushing.

Experienced wheelchair users can create pushing movements through various techniques; therefore, individuals compensate for fatigue through varying muscle synergies during push and recovery phases. Table 2 demonstrates how agonists and antagonists were able to compensate for fatigued muscles. As an agonist demonstrated fatigue, the PD increased force generation. This finding is consistent with wheelchair sport studies in that individuals modify their general movement patterns following the onset of prime mover fatigue. The intermittent activity and the stopping/starting activity typical of many wheelchair sports allow this varied force production to occur. However, during continuous pushing, the shoulder flexors are constantly activated to maintain movement, but the shoulder extensors are not required to generate braking forces, ultimately leading to agonist fatigue without concomitant antagonist fatigue (that is, imbalance). The need to stop a chair and change direction during gameplay likely explains the coupled agonist–antagonist fatigue demonstrated in the current study. Although the mechanisms that explain this symbiotic relationship are needed, the initial findings are promising in regards to morbidity experienced by persons with spinal cord injury, namely shoulder joint pain and injury.

Since multiple factors contribute to fatigue, these findings should not be extrapolated to persons with lower injury levels. The current case participant demonstrated less fatigue than a teammate with triceps function (data not published). This outcome could be due to the current subject's gameplay strategy (for example, more blocks and less offensive sprints) or his reduced ability to produce force-development byproducts that cause fatigue. Also, we did not report data of other push-phase agonists (for example, biceps brachii) or antagonists. These muscles likely increased force production across training sessions as demonstrated by fatigue in shoulder joint movers (Table 1, session 2). This finding is consistent with shoulder fatigue compensation by arm musculature during continuous pushing. Finally, a limitation of the current study was that data was collected during a multi-player training camp and the participant was forced to rest for some of the 8-min scrimmages during a session. Data were not reported for rest times but future research should examine continuous, uninterrupted training activity.

In summary, the purpose of this study was to document agonist–antagonist imbalance between push-phase and recovery-phase muscles during actual wheelchair sport activity. Our findings indicate that fatigue is present in wheelchair pushing agonists (that is, AD and PM) and antagonists (that is, PD) across and within wheelchair rugby training. Fortunately, the typical push-recovery muscle imbalance seen during continuous pushing was not present in the intermittent rugby activity. This finding reflects the potential of certain wheelchair sports to reduce risk of shoulder pain and overuse injuries through sport rehabilitation. Future research should examine EMG responses to controlled intermittent exercise stimuli to test the current findings.

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COMPETING INTERESTS

The authors declare no conflict of interest.

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