A Description of Sprint Performance and Neuromuscular Characteristics of Elite Athletes with Cerebral Palsy

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Cerebral Palsy (CP)

Aetiology

- Common congenital condition (2.5 every 1000)
- Non- progressive damage to 3 areas of immature brain that control movement and co-ordination
- Movement impairments: spasticity, dyskinesia, ataxia

Central drive

- Exercise performance is impaired due to suboptimal central drive, including lowered muscle activation on the affected side
- 33-50% lower muscle activation on affected side

Fatigue in CP

- Literature shows a flattened fatigue profile in CP
- Spasticity, increased stiffness and collagen, co-activation, Type I fibre predominance and weakness limit capacity
- Results in the inability to reach performances high enough to show fatigue
- Consequently, mean and frequency EMG show flattened responses

Reddihough et al, 2003; Rose et al, 2005; Elder et al, 2003; Stackhouse et al, 2005

Exercise performance in CP

Deficits

- Significant reductions in strength, speed, anaerobic and aerobic capacity
- Sedentary, paediatric and adolescent samples

Benefits

- Large increases in all areas due to various interventions
- Up to 69% improvement in strength in hemiplegic individuals

Athletes

- Elite athletes train at very high volumes for many years
- This sample: 6-15 hours/week for ~8 years
- Intriguing to discover whether athletes respond in similar manner as untrained adolescent samples used in literature

Reid et al, 2010

Aim and Hypothesis

Aim

- To describe sprint cycling performance and neuromuscular characteristics in elite athletes with CP, compared to well-trained sprint-matched able-bodied (AB) athletes

Hypothesis

- Athletes with CP will demonstrate lower power output and a maintenance of EMG activation over the trial (flat fatigue profile), similar to the response found in untrained, paediatric samples

Methods: Wingate cycle test

utput

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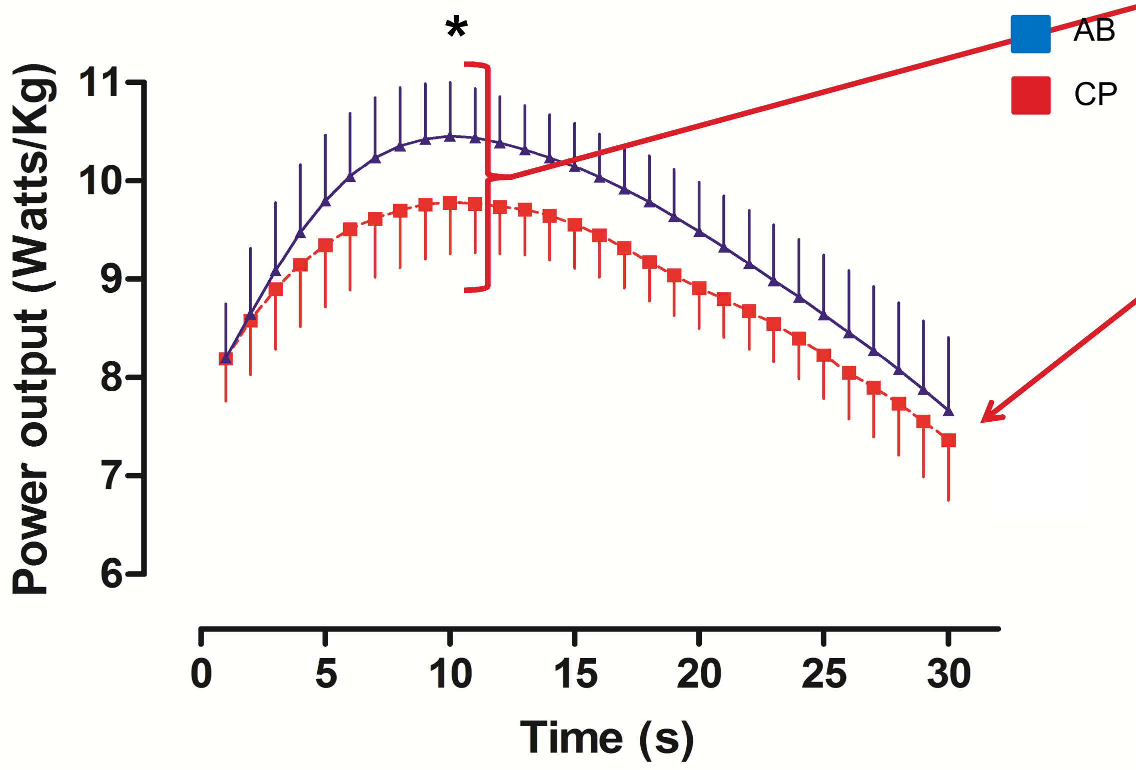
Cerebral Palsy (CP)	Able Bodied (AB)
N = 5 (3= T38 : 2= T37)	N = 16
Age: 21.6 ± 4.2 years	Age: 23.4 ± 1.1 years
elite sprint track athletes	performance matched athletes
100m: 12.2 s	100m: 12.3 s

Muscles tested (bilateral):

Erector spinae, Gluteus medius, Biceps Femoris, Gastrocnemius, Vastus lateralis

Results: Power output and fatigue



Power Output (W/kg) *

10.4 ± 0.5 vs. 9.8 ± 0.5

Fatigue Index (%)

27 ± 6.9 vs. 25 ± 7.3

Despite differences in power output, similar fatigue profile

* P<0.05, main group effect

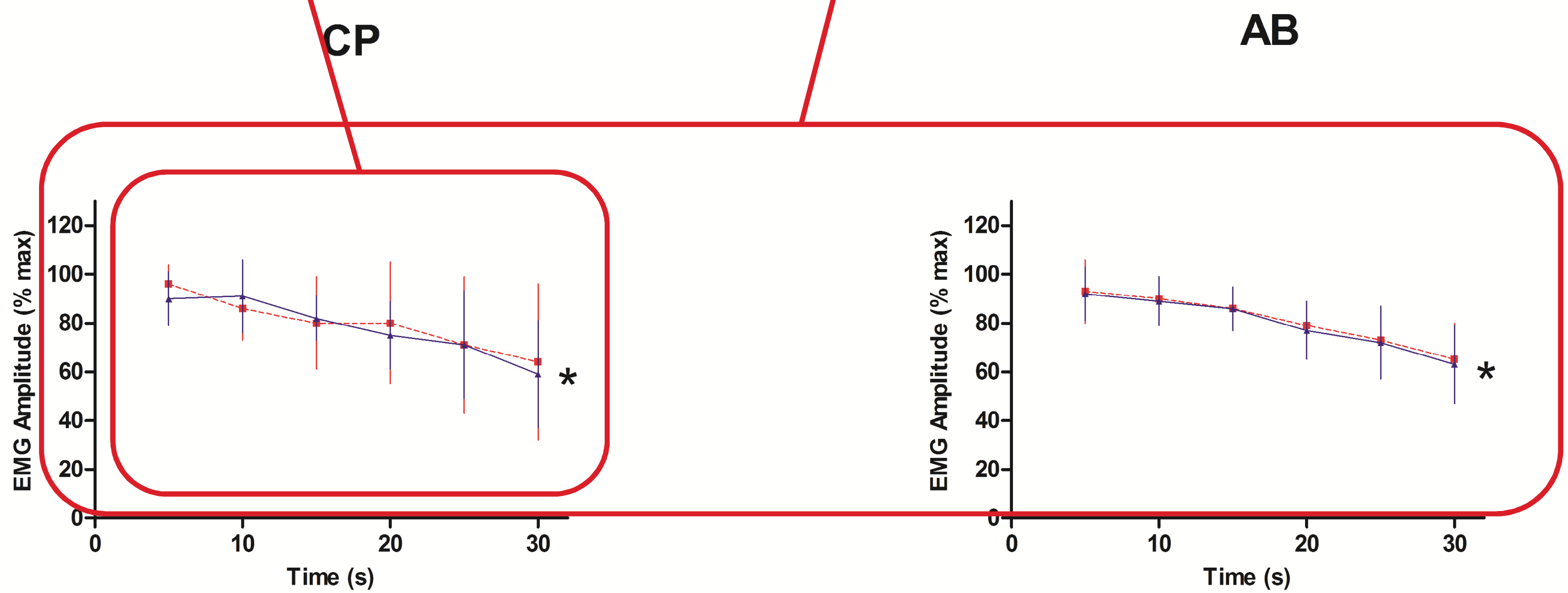
Results: Mean normalised EMG

No difference between sides in response to fatigue in CP

Similar response to fatigue in four muscles in CP and AB

Decrease in mean EMG

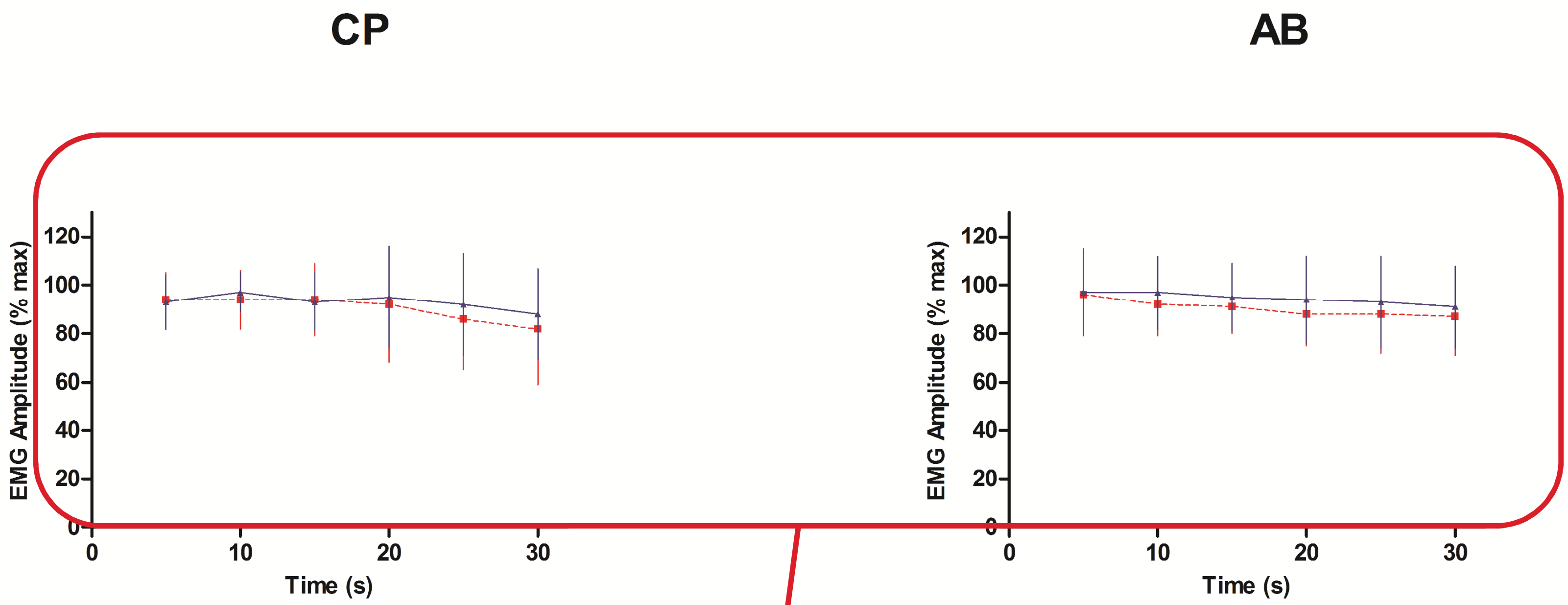
Biceps Femoris



* P<0.05, main time effect

Results: EMG cont.

Vastus Lateralis



Similar response to fatigue in VLO muscles in CP and AB

Maintained EMG

Results: Frequency analysis

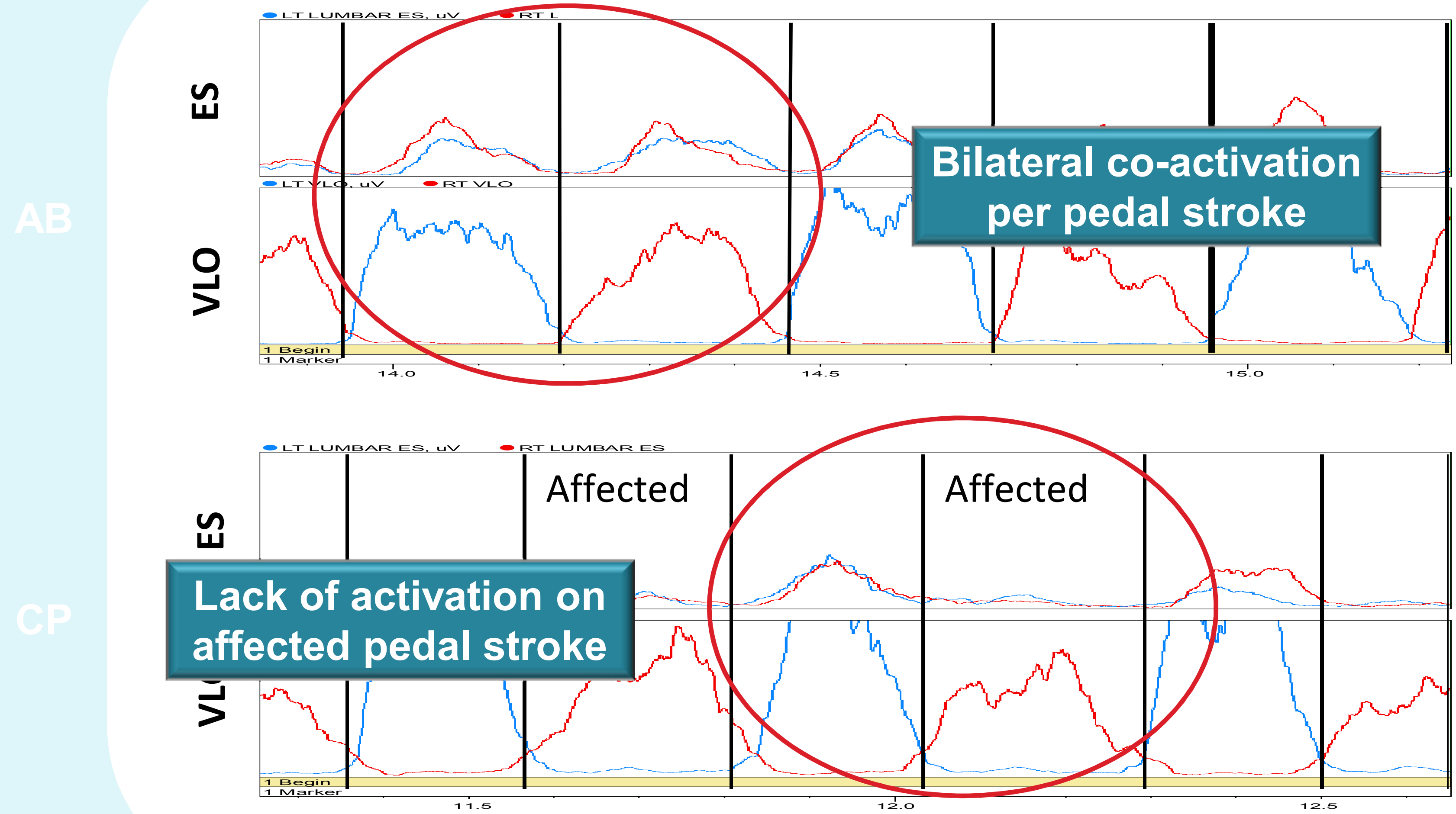
Muscle	CP		AB	
	Affected	Non affected	Non dominant	Dominant
Erector spinae	↓ *	↓ *	↓ *	↓ *
Gluteus medius	↓ *	↓ *	↓ *	↓ *
Biceps femoris	↓ *	↓ *	↓ *	↓ *
Gastrocnemius	↓ *	↓ *	↓ *	↓ *
Vastus lateralis	↓ #	↓ *	↓ *	↓ *

* P<0.05, main time effect; # P<0.05, group x side x time interaction

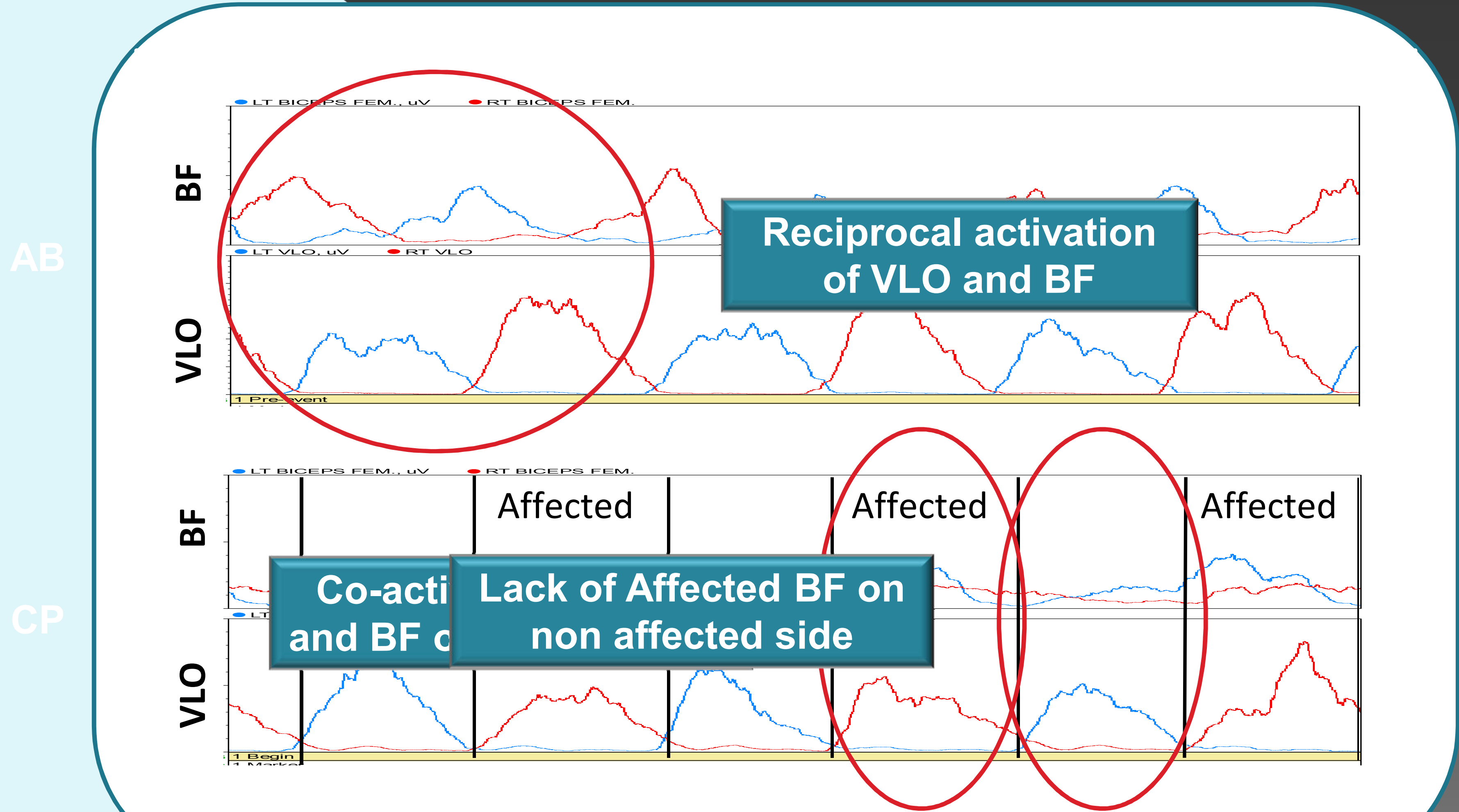
Similar decrease from pre- to post-fatigue in CP and AB

VLO: Affected side decreases more than NA and AB sides despite constant mean EMG

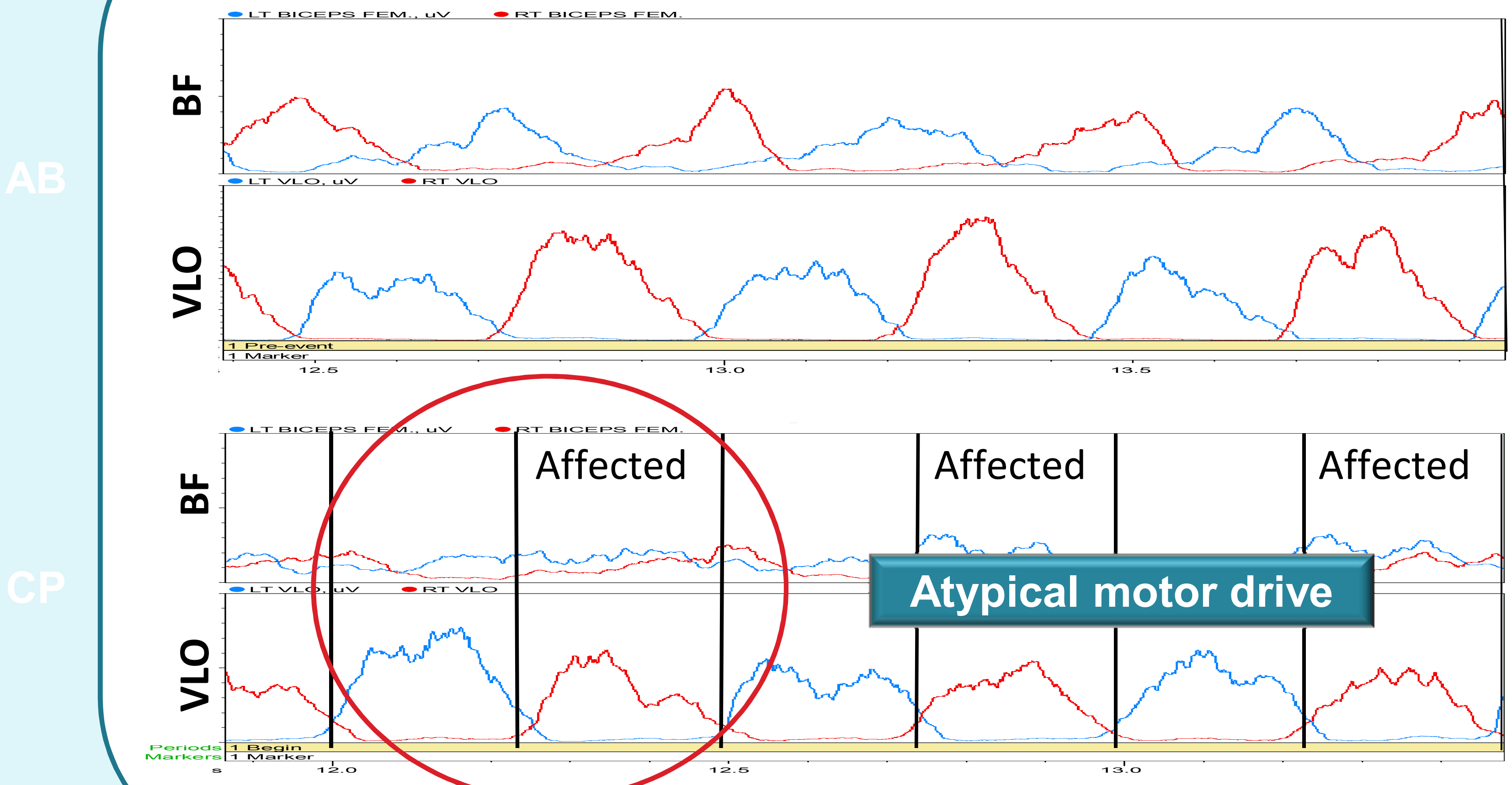
Irregularities identified in supporting muscle groups in CP



Irregularities identified in power muscle groups in CP



Irregularities identified in power muscle groups in CP cont.



Conclusions and further direction

No difference in neuromuscular fatigue, despite lower PO

This is contrary to existing literature which indicates a flat fatigue profile in CP, due to Type I muscle fibre predominance

Interesting to speculate: 1) athletes have a mild form of CP or 2) long-term high-level training may have changed Type I predominance

We identified irregularities in supporting and power muscles, associated with CP

Studies using running are required to further investigate findings of current study

Thank you!

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