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Control of muscle activation in the lower limbs during leg cycling after a motor complete spinal cord injury.

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CONTROL OF MUSCLE ACTIVATION IN THE LOWER LIMBS DURING LEG CYCLING AFTER A MOTOR COMPLETE SPINAL CORD INJURY

By

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B.A., University of Louisville, 2020

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ABSTRACT

CONTROL OF MUSCLE ACTIVATION IN THE LOWER LIMBS DURING LEG CYCLING AFTER A MOTOR COMPLETE SPINAL CORD INJURY

Jae Fish

April 12, 2024

Spinal cord injury (SCI) affects the central nervous system (CNS) causing muscle paralysis and autonomic dysfunctions throughout the body. Muscle activation after SCI is essential to facilitate functional recovery and may be produced via remaining input from supraspinal centers, the central pattern generators (CPG), or muscle stretch reflex responses. In this study, assisted leg cycling is evaluated as a tool to identify the neural structure control of muscle activation after motor complete SCI.

Eight individuals with a complete or sensory incomplete SCI between C2 and T5 participated in the study. Participants completed leg cycling trials with and without supraspinal intention and at five different cadences. Surface electromyographic (EMG) signals were recorded bilaterally from the lower limb muscles and analyzed for EMG entrainment. The results show significant relationships between leg cycling cadence and supraspinal input with EMG entrainment in some, but not all muscles.

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INTRODUCTION

Spinal cord injury (SCI) is a neurological condition that affects sensory and motor functions below the level of injury and autonomic systems throughout the whole body. On average in the United States, there are 17,000 new cases of SCI anually (Morrison et al., 2018). Cervical level SCIs are the most prevalent and result in the most impairments of both sensory and motor function. Thirty percent of traumatic SCIs are between the C1 and C4 vertebrae levels (Barbiellini Amidei et al., 2022). Forty percent of SCIs result in complete loss of sensory and motor functions below the injury with the highest percentage being at the cervical level (Tien et al., 2021).

Injury to the cervical region of the spinal cord is classified as tetraplegia because it affects sensory and motor function in both upper and lower limbs. This often affects abilities to perform activities of daily living (ADL) and can cause decreases in quality of life (QOL) (Tien et al., 2021). Individuals with tetraplegia are also at an increased risk for multiple secondary health conditions including circulatory and respiratory problems, autonomic dysreflexia, and postural hypotension when compared to paraplegics (Brinkhof et al., 2016). Injury to the thoracic, lumbar, or sacral region of the spinal cord is classified as paraplegia and affects sensory and motor functions in the lower limbs. Higher level thoracic SCIs can result in complications similar to cervical level injuries.

Those living with either tetraplegia or paraplegia are less likely to participate in regular exercise. Only forty percent of individuals with SCI engage in the

recommended amount of physical activity (Dolbow et al., 2012). Low participation in physical activity increases the risk of cardiovascular disease and obesity in individuals with SCI (Maher et al., 2017).

As of today, there is no cure for SCI, only rehabilitation techniques to improve sensory and motor functions and QOL. The goal of this study is to determine if leg cycling can be used to identify the neural structures control of muscle activation in the muscles of the lower limbs after motor complete SCI at the cervical and upper thoracic region.

LITERATURE REVIEW

CLASSIFICATION OF SPINAL CORD INJURY

The severity of a SCI is determined by The American Spinal Injury Association (ASIA) classification of impairment. To determine the classification of a SCI, an examination is done evaluating the sensory and motor functions at each dermatome (from C2 to S4-5) and myotome (From C5 to T1 and L2 to S1). The dermatomes are the area of the skin innervated by the sensory axons, and the myotomes are the collection of muscle fibers innervated by the motor axons (Rupp et al., 2021). This examination is called the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) and includes the ASIA Impairment Scale (AIS).

Neurological injuries are classified as either complete or incomplete based on whether there is sensory and motor function present at the most caudal sacral segments (S4-S5) (Rupp et al., 2021). The neurological level of injury (NLI), which refers to the most caudal segment of the spinal cord with normal sensory and motor function, is also determined during the examination (Kirshblum et al., 2011). The AIS grades the degree of impairment (A= Complete, B= Sensory Incomplete, C= Motor Incomplete with motor function in less than half key muscles below the NLI, D= Motor Incomplete with motor function in more than half key muscles below NLI, E= Normal). AIS A represents no preservation of sensory or motor function, whereas AIS B-D represent varying levels of preservation of sensory or motor

functions. Injuries with no sacral sparing may have dermatomes and myotomes caudal to the sensory and motor levels with partially preserved functions; this is referred to as Zones of Partial Preservation (ZPP) (Rupp et al., 2021). Further neurological examination can provide insight into the capabilities of the neural structures within the spinal cord after injury.

CENTRAL NERVOUS SYSTEM CONTROL OF MUSCLE ACTIVATION

It is important to understand how injury changes to the neural structures of the spinal cord and central nervous system (CNS). After a SCI, the spinal cord processes input and generates outputs differently (Edgerton et al., 2001). The neural structures responsible for muscle activation during locomotion in a non-injured individual are primarily supraspinal centers and spinal circuits (Field-Fote et al., 2017). The supraspinal centers control locomotion by processing sensory information from the peripheral nervous system and by producing motor output in response through descending tracts in the spinal cord (Field-Fote et al., 2017).

The motor cortex and basal ganglia plan and initiate voluntary movement (Jahn et al., 2008; Leiras et al., 2022) . The structures of the brainstem including the mesencephalic locomotor region (MLR), subthalamic locomotor region (SLR), and cerebellar locomotor region (CLR) control movement initiation, speed, and termination, as well as postural control during locomotion (Jahn et al., 2008; Leiras et al., 2022; Pernia-Andrade et al., 2021). Descending information from the brainstem reaches the locomotor networks in the spinal cord via corticoreticulo-spinal tract and the corticorubro-spinal tracts (Martin, 2022). The corticospinal tract

projects directly from the primary motor cortex, located in the frontal lobe, to the spinal cord (Martin, 2022). The supraspinal contributions are impaired after SCI and hinder the ability to voluntarily activate muscles and modulate locomotion due to the lack of descending control.

MUSCLE TYPES & PROPERTIES

Spinal circuits produce muscle activation via sensory feedback detected by afferents found in muscle spindles. There are two main components to sensory afferents: 1) group Ib Golgi tendon organ (GTO) afferents, and 2) muscle spindle afferents II and Ia (Oliver et al., 2021). GTOs are type Ib sensory afferents that detect changes in load on the muscle tendons and produce a reflex response that results in relaxation of the previously contracted muscle fibers. Type II muscle spindle afferents signal positional changes in the muscle spindle. Type Ia afferents detect the velocity of stretch in the muscle spindles to produce a reflex response that causes contraction of the previously stretched muscle fiber. This can initiate Central Pattern Generator (CPG) activity to produce a reciprocal pattern (Barbeau et al., 1999). CPGs can be found in the brainstem; they play an important role in respiration, chewing, swallowing, and cardiovascular functions, and in the spinal cord to produce locomotion (Steuer & Guertin, 2019).

CPGs are neural circuits within the spinal cord that produce rhythmic motor patterns and can respond to Type Ia muscle spindle afferent feedback (Field-Fote et al., 2017). The CPGs are not only responsible for generating repetitive patterns, but also for receiving, interpreting, and predicting the appropriate sequence of

actions during locomotion (Edgerton et al., 2004). After a SCI, CPGs have been found to control muscle activation speed and direction while walking in the absence of supraspinal input (Roy et al., 2012). Movement frequency is associated with the CPGs ability to distribute a coordinated drive to the motoneuron pools and respond to the rate of muscle stretch (Dzeladini et al., 2014). Although CPGs have been studied mostly in animal models, there is compelling evidence and it is widely accepted that humans have CPGs (Steuer & Guertin, 2019).

The automaticity property of the spinal cord seems to persist after complete a SCI (Roy et al., 2012). The uninjured human spinal cord has properties of automaticity, the ability to function without conscious control (Edgerton et al., 2004). The automaticity of the spinal cord to control posture and locomotion emerges from the interaction between the CPGs and sensory input. For the systems to work in synergy, they both must have intrinsic activation and inhibition patterns to coordinate motor outputs (Edgerton et al., 2004). After a SCI, the interaction between the CPGs and peripheral inputs is essential for control of posture and locomotion due to the absence of supraspinal control.

Walking and leg cycling are both locomotor tasks where lower limb muscles are typically activated by the supraspinal centers so they can contribute to the movement. The muscles of the lower extremities include mono- and bi-articular muscles, which have parallel processes but different roles and organization of neural control (Van Ingen Schenau et al., 1995). Mono-articular muscles produce movement only about one joint, whereas bi-articular muscles produce movement

about two adjacent joints (Zagrodny et al., 2018). Leg cycling involves the use and coordination of these muscles to produce cycling movements.

It's been found that the CNS activates the mono-articular muscles during the period where they can contribute to the pedal force which coincides with the period during the muscle is shortening (Van Ingen Schenau et al., 1995). The bi-articular muscles are able to adjust the timing of activation based on the environmental constraints to produce force during the period in which they can contribute to pedal forces (Rouffet et al., 2009). Mono- and bi-articular muscles are activated based upon when they can contribute to the pedal forces and the coordination of their activations is controlled by muscle synergies. Muscles synergies distribute neural drives to motoneurons innervating a group of muscles to control the movement (Barroso et al., 2014). CPGs are responsible for coordinating and distributing the neural drive to the muscle synergies (Barroso et al., 2014). Therefore, if the CPGs have the ability to coordinate and stimulate muscle synergies after a motor complete SCI, muscles should be activated during the time they are shortening (mono-articular muscles) and can contribute to the movement (bi-articular muscles).

BODY WEIGHT SUPPORTED TREADMILL TRAINING

One method used for functional recovery after a SCI is stepping on a body weight supported treadmill (BWST). Patients are put into a harness, which is attached to the cylinders above the treadmill providing body weight support, and trainers manually assistance at the trunk and lower limbs to facilitate stepping on

the treadmill. The Neuro Recovery Network (NRN) has shown that manually facilitated stepping on a BWST improves locomotive function by improving gait speed, distance, and balance in individuals with incomplete SCIs (Buehner et al., 2012). By manipulating stepping frequency and load, increased muscle activation of the lower limbs can be achieved.

Two factors influence locomotion, the stretch reflex activity and load on the body (Dietz et al., 1998). In individuals with motor complete SCI, limb loading can lead to increases in EMG amplitudes of the lower extremity muscles (Harkema et al., 1997). When increasing load to the Type Ib afferents during walking, reflex responses will produce muscle activation of the lower limb muscles independent of input from the supraspinal centers. This demonstrates the reflex control of muscle activation of the lower limb muscles during stepping after SCI.

It has been shown that EMG amplitudes, in the muscles of the lower limbs, increased at faster treadmill speeds in individuals with thoracic level motor complete SCIs (Beres-Jones, 2004). An increase in walking speed leads to an increase in velocity of muscle stretch. The increase in muscle stretch velocity is detected by Type Ia afferents. An increase in muscle stretch velocity has been observed in individuals with complete SCI while stepping at faster speeds due to decreased time between EMG burst (Beres-Jones, 2004). The CPG activity and sensory input allow for the rapid changes in stepping speed and limb load (Edgerton et al., 2004). The similarities in mechanical properties between walking and leg cycling indicate leg cycling may be another tool to access the neural structures of the CNS after motor complete SCI.

FUNCTIONAL ELECTRICAL STIMULATION CYCLING

Leg cycling with functional electrical stimulation (FES) is another common practice in SCI rehabilitation. It has been shown to increase muscle mass, bone density, and QOL as well as reduce spasticity after a SCI (Szecsi et al., 2014). FES during leg cycling uses electrodes to stimulate the muscles of the lower limbs at the time the muscles are supposed to contract and is used to replace the CNS control of muscle activation. The vastus lateralis (VL), vastus medialis (VM), and rectus femoris (RF) in non-injured individuals will be activated during the knee extension phase (0°-90°; 1 revolution equals 360°) of leg cycling and the hamstring muscles (biceps femoris; BF) will be activated during the knee flexion phase (0°-270°) (Hug & Dorel, 2009). The agonist and antagonist muscles are contracted in a reciprocal pattern during leg cycling similar to other forms of locomotion.

Although FES confers significant health benefits, it may have limitations. Muscle activation is not regulated or generated by CPGs or reflex responses and proprioceptive feedback is limited with FES-evoked muscle activation (Ibitoye et al., 2014). FES reverses the size principle of motor unit recruitment, where the larger motor units are recruited before the smaller motor units (Gregory & Bickel, 2005). The unnatural recruitment of motor units and early onset of fatigue with FES-evoked muscle activation limits the ability of the spinal cord to independently induce muscle contraction (Ibitoye et al., 2014). Leg cycling with FES is a beneficial rehabilitation method to increase muscle mass, enhance metabolism, and

increase QOL after SCI (Davis et al., 2008). However, the unnatural FES-evoked muscle activation may inhibit the ability to assess the neural structure control of muscle activation. So using leg cycling without FES will allow us to understand the mechanisms associated with the activation of the lower limb muscles during locomotor tasks after SCI.

PASSIVE LEG CYCLING

Passive leg cycling can be a helpful intervention for individuals who have insufficient motor control and are unable to engage in active leg cycling. Animal and human studies suggest there are neurological and cardiovascular benefits to passive leg cycling (Nardone et al., 2017; Phadke et al., 2019). Neuroplasticity allows the brain to grow and reorganize neural networks after an injury. Graziano et. al., found that passive cycling exercise increases plasticity within the somatosensory cortex and promotes cortical reorganization after spinal cord injury in complete thoracic transected adult rats. This study proposes there are interactions between the supraspinal centers and the exercised limbs that do not rely on intact sensorimotor pathways (Graziano et al., 2013). This relationship between the supraspinal centers and the spinal cord after SCI has also been observed with humans.

In human studies, improvements in spasticity, H-reflex, and cortical inhibition have been associated with passive leg cycling in individuals with motor complete SCIs (Phadke et al., 2019). It is important to further investigate the reflex responses evoked by passive leg cycling on muscle activation and the translation

to locomotion. Limited studies show cardiovascular benefits of passive leg cycling in humans, but Ballaz et. al. report increased blood flow velocity in individuals with SCIs after acute passive leg cycling (Ballaz et al., 2007). Ensuring proper blood flow is reaching the lower limbs is important to prevent tissue damage and skin breakdown. With more understanding of how leg cycling can be used for recovery of motor functions, new rehabilitation techniques for SCI can be developed.

PURPOSE, AIMS, AND HYPOTHESIS

Individuals with complete SCIs have no sensory or motor function below the neurological level of injury. The spinal circuits must adapt to produce movement. Supraspinal input seems to be unable to produce or influence muscle activation after injury. The CPGs, through muscle spindle afferent feedback, may be able to produce muscle activation through the spinal cord circuit independent of the supraspinal centers. Evaluating the timing of muscle activation at various cadences can help us determine the roles in which the CPGs and reflex responses play during leg cycling. The goal of this study is to determine whether leg cycling can be used to identify the neural structures that control muscle activation in the muscles of the lower limbs after motor complete SCI at the cervical and upper thoracic region.

Study Aim #1: To investigate the control operated by the supraspinal centers on activation of the lower limb muscles after a motor complete SCI by comparing muscle activation between leg cycling exercises performed with and without voluntary intent.

Hypothesis #1: Due to the impaired supraspinal input after motor complete SCI, there will be no difference in EMG activity in the lower limbs between leg cycling exercises performed with and without voluntary intent in SCI participants.

Study Aim #2: To investigate the contribution of the CPGs and reflex responses on activation of the lower limb muscles after a motor complete SCI by comparing EMG data across five different cadences.

Hypothesis #2: Leg cycling at faster cadences will increase the type 1a afferent feedback to produce a reflex response and increase EMG entrainment in individuals with motor complete SCI.

METHODS AND MATERIALS

SUBJECTS

Eight individuals (1 female and 7 male) volunteered to take part in the study. All participants (n=8) were diagnosed with a complete (AIS A) or sensory incomplete (AIS B) spinal cord injury based on the American Spinal cord Injury Association's (ASIA) International Standards for the Neurological Classification of Spinal Cord Injury (ISNCSCI) and ASIA impairment scale (AIS). All SCI participants had no motor function below the level of injury prior to this study. The mean time since injury was 14±6 months. Participants were classified as tetraplegic (n=6) or paraplegic (n=2) and all with neurological levels of injury between C2-T5.

Participants	Level	AIS	Age	Sex	Time Since Injury (Months)
R047	C2	A	37	M	14
R183	C5	A	34	M	13
R356	C4	A	38	M	15
R394	T2	A	40	M	5
R420	C5	A	43	M	17
R481	T5	B	25	F	6
R485	C5	B	26	M	2
R486	C7	A	32	M	17

Table 1: Subject demographics.

METHODS AND MATERIALS

EXPERIMENTAL PROTOCOL

Each participant attended one experimental session at the Kentucky Spinal Cord Injury Research Center in Louisville, Kentucky. A cycle ergometer was used for all trails (Monark type 881E, Stockholm, Sweden). For all conditions participants sat in a stationary chair with an upright trunk position. The chair was positioned at a comfortable distance from the leg cycle. Experimenters assisted participants with maintaining proper leg alignment and with leg drive to maintain leg cycling cadences. Experimenter's hands were placed at the knee and underneath the pedal to provide assistance. Participants' shoes were secured to the pedals with toe clips. Participants continued to cycle until 10 consecutive cycles were recorded without visual indication of muscle spasms. A metronome was used to help examiners maintain cadences. This was repeated during both protocols. A two minute rest period was given in between each trial. The order of trials was randomized to control for an order effect.

Protocol #1: Supraspinal intention condition (Aim #1)

Participants were instructed to assist examiners as much as possible to pedal at a cadence of 30rpm until 10 consecutive revolutions were recorded. This was considered active leg cycling (voluntary supraspinal intention). Participants again were instructed to pedal at a cadence of 30rpm until 10 revolutions were recorded except participants were instructed to not attempt to voluntarily contract their lower limbs (Hoover et al., 2023). This was considered passive leg cycling (no voluntary supraspinal intention). To

ensure no supraspinal input was influencing muscle activation, participants were verbally given math problems while passive cycling.

Protocol #2: Cadence condition (Aim #2)

Participants were instructed to assist examiners as much as possible to pedal at a cadence of 15rpm until 10 consecutive revolutions were recorded. This was repeated for each cadence (15, 22, 30, 37, and 45rpm). Participants were instructed to attempt to voluntarily contract their lower limbs during all trials. (see Figure 1).

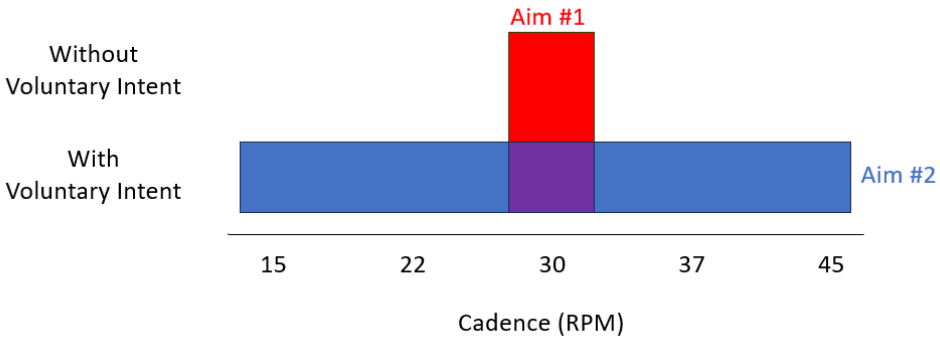


Figure 1: This graph shows the experimental design. The EMG activity of lower limb muscles was recorded at each cadence (15, 22, 30, 37, and 45 rpms). The EMG activity for passive (without voluntary intent) and active (with voluntary intent) leg cycling was also recorded at a cadence of 30 rpms.

METHODS AND MATERIALS

DATA COLLECTION

Bipolar surface EMG was recorded using the Delsys Trigno system. Wireless bipolar electrodes located on eight different muscles transmitted and recorded EMG signals to the Delsys Trigno digital system. Delsys wearable Trigno Avanti Sensors were used for all muscles. Electrodes were placed on the surface of the skin and located on the muscle belly parallel to the muscle fibers (Rouffet et al., 2009). Skin was prepared and electrodes were secured to the surface of the skin with adhesive tape. Tegaderm was placed over the electrodes to minimize electrode movement. (see Figure 2)

Bilateral recorded muscles:

1. Rectus Femoris (RF)
2. Vastus Lateralis (VL)
3. Vastus Medialis (VM)
4. Hamstring (Biceps Femoris) (HAM)
5. Gastrocnemius Lateralis (GL)
6. Gastrocnemius Medialis (GM)
7. Soleus (SOL)
8. Tibialis Anterior (TA)

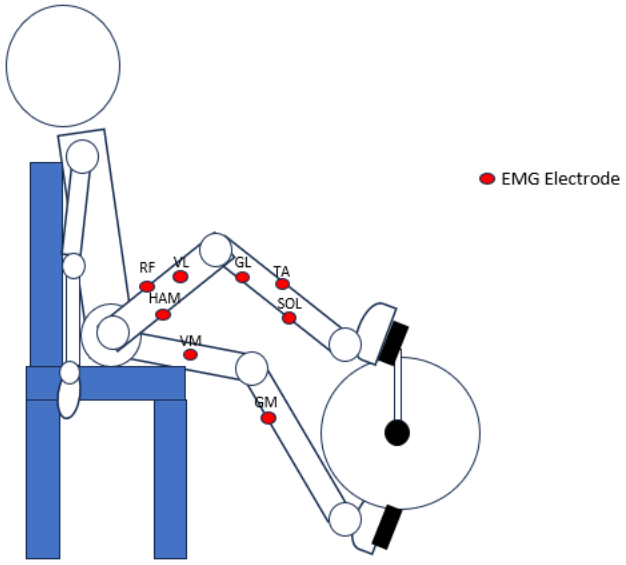


Figure 2: This figure shows the experimental set-up. The participant sat in a stationary chair with feet secured to the pedals of the cycle ergometer. Wireless electrodes were placed on the selected muscles (Rectus Femoris, Vastus Lateralis, Vastus Medialis, Hamstring (Biceps Femoris), Gastrocnemius Lateralis, Gastrocnemius Medialis, Soleus, and Tibialis Anterior) and recorded EMG activity.

METHODS AND MATERIALS

DATA PROCESSING

The surface EMG was sampled at 2,000 Hz. First the signals were bandpass filtered at 20 Hz – 500 Hz. Next a moving root-mean-squared (EMG_{rms}) with a 400 ms window size was used. Time was normalized to study EMG activity variations within the pedal cycle. A gyroscope attached to the femur defined the start and end of the pedal cycles using the femur's angular velocity. Average EMG was then calculated from the EMG_{rms} signals. Amplitude was than normalized for the EMG_{rms} for each muscle for each condition. This process was repeated separately for each muscle.

For each muscle, Delsys-Visual3D software was used to identify if a burst in the EMG signal (EMG_{burst}) occurred while cycling. If an EMG_{burst} was present, time was normalized to examine the EMG activity within a pedal cycle. A pedal cycle was defined as when the left limb began in extension and returned to extension. If the EMG_{burst} coincided with the pedaling frequency, with an amplitude of at least 5uV for at least five consecutive cycles, it was identified as EMG entrainment ($EMG_{entrain}$). Entrainment is defined as the phase relationship between a stimulus frequency and an oscillating frequency (Kriellaars et al., 1994). This process was repeated for each condition. (see Figures 3 & 4)

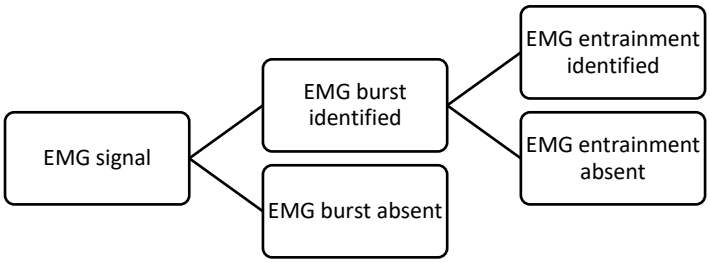


Figure 3: This diagram displays the data analysis process for each muscle during each condition. The EMG signals were examined for the presence of EMG_{burst}. If an EMG_{burst} was identified, it was then examined for the presence of $EMG_{entrain}$.

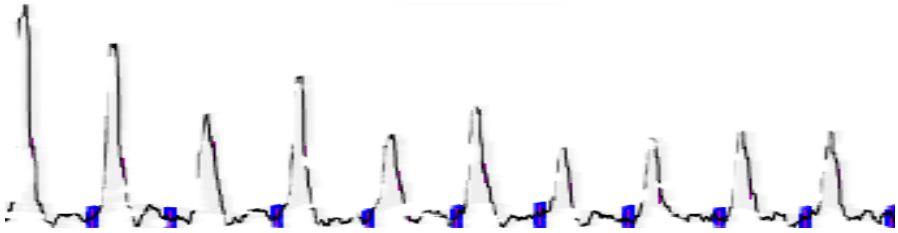
1.

EMG Burst

2.

No EMG Entrainment

3.



EMG Entrainment

Figure 4: This picture shows example graphs of an EMG_{burst} (1), No $EMG_{entrain}$ with event labels (2), and $EMG_{entrain}$ with event labels (3). The blue marker indicates the event of the end of one pedal cycle.

METHODS AND MATERIALS

STATISTICAL MEASURES

The EMG activity for each muscle of the lower limbs was recorded during the trials for the supraspinal intention conditions and the cadence conditions. To determine whether these variables are associated, a chi-square test was used. A chi-square test of independence was performed using categorical variables (see Table 2) with the $EMG_{entrain}$ for each muscle bilaterally during the active (voluntary supraspinal intention) and passive (no voluntary supraspinal intention) leg cycling conditions. In this test, the dependent variable was supraspinal intention and the independent variable was $EMG_{entrain}$.

A chi-square test of independence was also performed using categorical variables (see Table 2) with the $EMG_{entrain}$ for each muscle bilaterally during the 15, 22, 30, 37, and 45rpm cadence conditions. The dependent variables used were the five different cadences and the independent variable was the $EMG_{entrain}$. If statistical significance was found, a Bonferroni method statistic was used for a post-hoc analysis to identify which cadence conditions were significantly different from one another. SPSS Statistics software was used to analyze the data and Microsoft Excel was used to create visual graphical representations of the data. A p-value of .05 was used as the threshold for statistical significance for all tests. A Chi-square symmetrical measure was done for all tests to determine the effect size of the p-value.

Key for Categorical Variables

Side:	Left = 1 Right = 2
Muscle: (Used for VAS and GAS only)	VL/GL = 1 VM/GM = 2
Condition: **Cadence:**	15rpm = 1 22rpm = 2 30rpm = 3 37rpm = 4 45rpm = 5
Supraspinal Intention:	Passive = 1 Active = 2
EMG Burst:	No = 0 Yes = 1
EMG Entrainment:	No = 0 Yes = 1

Table 2: Categorical variables and values used for chi-squared tests for independence for the supraspinal intention and cadence conditions.

RESULTS

SUPRASPINAL INTENTION

$EMG_{entrain}$ was observed across all muscles during the passive (no voluntary supraspinal intention) and the active (voluntary supraspinal intention) leg cycling conditions (see Table 3). When comparing each side of the body there was a significant relationship in $EMG_{entrain}$ for the active (voluntary supraspinal intention) condition at the right Soleus muscle only, (N=5; p=.039; effect size=.516), but not the left, (N=2; p=.590; effect size=.135) (see Figure 5). There was no significant difference in total muscle $EMG_{entrain}$ between the active (N=47) and passive (N=40) leg cycling conditions, p>.05.

		EMG Entrainment (count)						
		Vasti (VL/VM)	Rectus Femoris	Hamstring	Tibialis Anterior	Soleus	Gastrocnemius (GL/GM)	Total
Side	Condition							
Left	Active	4	1	7	2	2	4	20
	Passive	4	3	6	1	3	5	22
Right	Active	7	2	3	1	*5	9	27
	Passive	9	1	1	1	1	5	18

Table 3: The table displays the number of muscles with $EMG_{entrain}$ for each condition (Active and Passive leg cycling) for each side of the body (Right/Left). The total for each row is calculated in the last column of the table. * indicates significant relationship (p>.05).

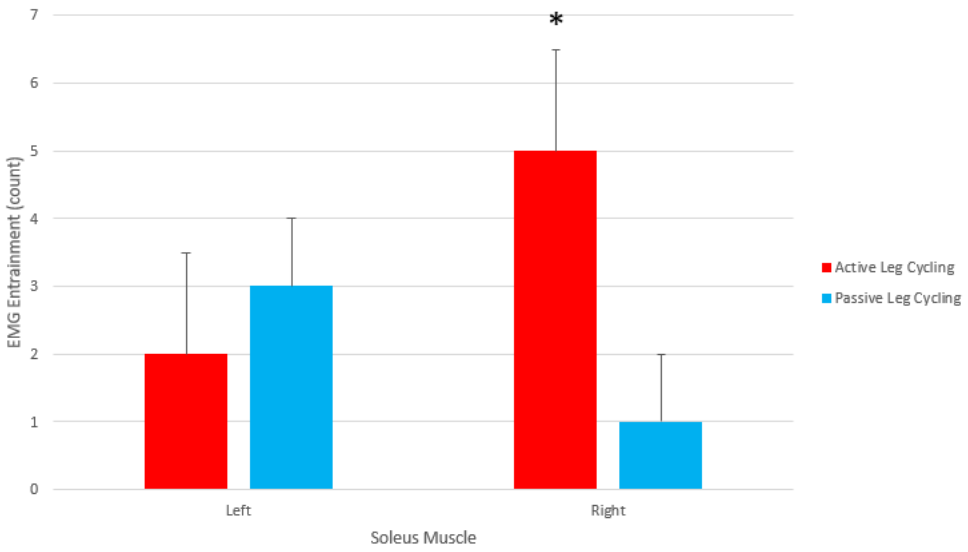


Figure 5: This graph shows the $EMG_{entrain}$ counts for the right and left soleus muscle for active and passive leg cycling. * indicates the significant relationship between $EMG_{entrain}$ of the right soleus muscle and active leg cycling. The standard error is indicated by the error bars.

RESULTS

CADENCE

$EMG_{entrain}$ was identified across most muscles during each leg cycling cadence (15, 22, 30, 27, & 45rpm) except for the Tibialis Anterior muscle during the 15rpm cadence (see Table 4). The Hamstring muscle (N=40; p=.021; effect size=.379) and Gastrocnemius muscles (N=72; p=.010; effect size=.288) were the only muscles that showed a significant increase in $EMG_{entrain}$ during the 45rpm cadence and a significant decrease in $EMG_{entrain}$ during the 15rpm cadence (see Figure 6). Comparing sides of the body revealed a significant relationship in $EMG_{entrain}$ and the 45rpm cadence at the left Gastrocnemius muscles (p=.008; effect size=.416), but not the right (p=.406; effect size=.224) (see figure 7). No evidence suggests that an increase in cadence would result in a significant decrease in $EMG_{entrain}$.

		EMG Entrainment (count)						
		Vasti (VL/VM)	Rectus Femoris	Hamstring	Tibialis Anterior	Soleus	Gastrocnemius (GL/GM)	Total
Side	**Condition**							
Left								
	15 RPM	6	1	3	0	0	*2	12
	22 RPM	7	2	5	1	2	6	23
	30 RPM	5	1	7	2	2	4	21
	37 RPM	6	3	7	1	3	9	29
	45 RPM	7	2	7	1	2	*11	30
Right								
	15 RPM	7	0	0	0	4	6	17
	22 RPM	7	1	1	1	5	7	22
	30 RPM	7	2	3	0	5	9	26
	37 RPM	8	0	3	2	5	7	25
	45 RPM	7	1	4	2	5	11	30
Total								
	15 RPM	13	1	**3	0	4	**8	29
	22 RPM	14	3	6	2	7	13	45
	30 RPM	12	3	10	3	7	13	48
	37 RPM	14	3	10	3	8	16	54
	45 RPM	14	3	**11	3	7	**22	60

Table 4: The table displays the $EMG_{entrain}$ count for each muscle during each cadence for each side of the body (Right/Left) and combined total. The total for each row is calculated in the last column of the table. * indicates the significant relationship between $EMG_{entrain}$ of the left gastrocnemius muscle and increase in cadence. ** indicates the significant relationship between $EMG_{entrain}$ of the hamstring and the gastrocnemius muscles and an increase in cadence.

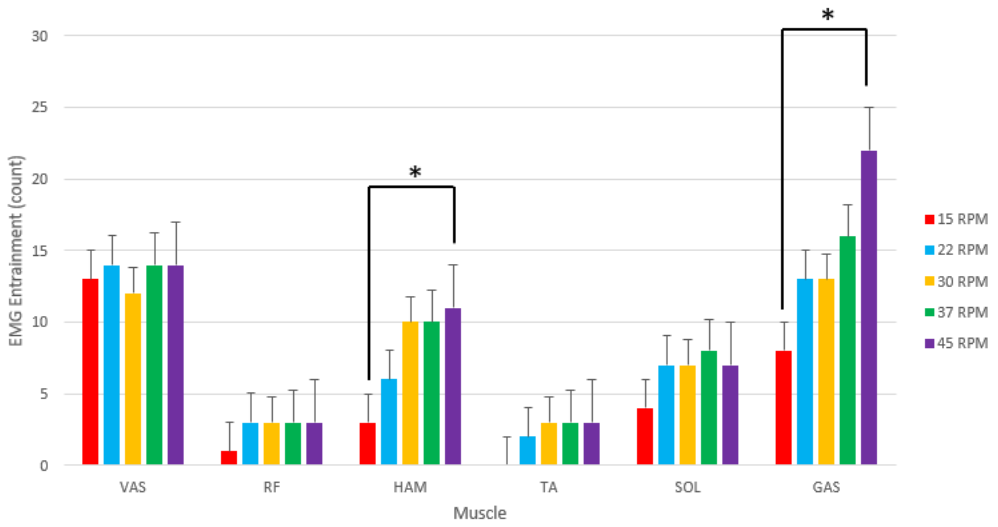


Figure 6: This graph shows the relationship between $EMG_{entrain}$ in the hamstring and gastrocnemius muscles and increases in cycling cadence. There is a positive increase in $EMG_{entrain}$ between 15rpm and 45rpm indicated by *. The standard error is indicated by the error bars.

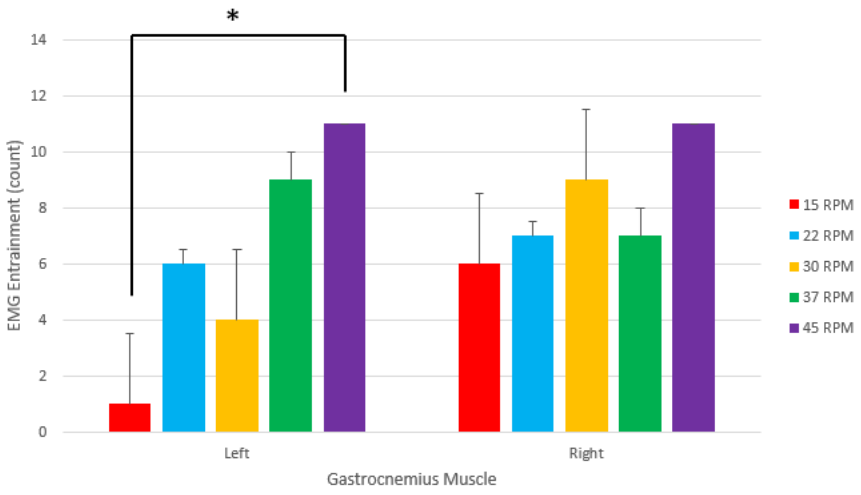


Figure 7: This graph shows the relationship between $EMG_{entrain}$ in the left and right gastrocnemius muscles and increases in cycling cadence. There is a positive increase in $EMG_{entrain}$ between 15rpm and 45rpm indicated by * for the left side. The standard error, if present, is indicated by the error bars.

DISCUSSION

The results from this study show the possibility of a potential remaining relationship between supraspinal input and muscle activation of the lower limbs during leg cycling after motor complete SCI. When comparing the sides of the body, the results from this study show supraspinal input may have an unequal influence among the muscles. The right soleus muscle showed a significant increase in EMG entrainment during active leg cycling compared to passive leg cycling, while the left soleus did not. However, no significant data showed supraspinal intention while leg cycling resulted in an increase in total muscle activation when compared to leg cycling with no supraspinal intention. This confirms the hypothesis for Aim 1 that due to the impaired supraspinal input after motor complete SCI, there will be no difference in EMG activity in the lower limbs between leg cycling exercises performed with and without voluntary intent in SCI participants.

Other evidence from this study identifies the relationship between EMG entrainment and the increase in leg cycling cadence. This suggests leg cycling at a faster cadence, which increases the muscle stretch velocity and thus the afferent feedback from Type Ia fibers to the spinal cord, leads to greater muscle activation of the lower limb muscles even with the loss of motor function. This supports the hypothesis for Aim 2 that leg cycling at faster cadences will increase

the Type Ia afferent feedback to produce a reflex response and increase EMG entrainment in individuals with motor complete SCI.

SUPRASPINAL INTENTION

Motor planning begins in the supraspinal centers and is communicated via the spinal cord to produce movement. After a motor complete SCI, the supraspinal input below the level of injury is heavily impaired. It would be expected that the loss in supraspinal input would result in the inability to produce muscle activation. However, when comparing active and passive leg cycling, muscle activation was produced during both conditions. Similar results were found in a study done with individuals with a complete SCI (AIS A and B) who were participating in BWST training. EMG activity was present, but no change in EMG activity was seen when participants were asked to passively or actively attempt to step (Dobkin et al., 1995). If the supraspinal centers are not producing muscle activation during locomotion, the CPGs and reflex centers in the spinal cord are likely responsible for the EMG activity being produced.

The results of this study do show the trend in the possibility in a relationship between supraspinal intention and EMG entrainment. When comparing the sides of the body at the soleus muscle, the right soleus muscle showed a significant increase in EMG entrainment during active leg cycling compared to passive. This relationship had a large effect size of .516 on a scale of 0-1.0. Although this relationship was seen in only one muscle, the association

between supraspinal input to the right soleus and EMG entrainment is strong. This may be a result of zones of partial preservation, discussed below, and should be further investigated with a larger sample size to determine whether this relationship exists in other muscles.

CADENCE

After SCI, sensory regulation of locomotion can occur from reflex pathways to motor neurons (i.e. afferent feedback) or via the CPGs in the spinal cord. The speed of locomotion dictates the velocity at which a muscle is being stretched and thus influences the rate of Type Ia muscle afferent feedback. Results from this study shows the trend in increased EMG entrainment with the increase in leg cycling cadence in the hamstring and gastrocnemius muscles. This suggests that this relationship between the increase in cadence and EMG entrainment is likely due to the increase in afferent feedback.

In a study using BWST, changes in length of the vastus lateralis muscle during passive stepping did not modulate EMG activity (Dobkin et al., 1995). If a reflex response was responsible for the muscle activation of the vastus muscle, EMG activity likely would have occurred when the muscle was being stretched (swing phase of a step), but EMG activity was present when the muscle contributed to the movement (stance phase of a step). In this case, it cannot be determined whether reflex responses were absent due to additional afferent feedback from body weight load on muscles. There is likely a combination of reflex responses and CPG activity modulating locomotion.

Although not scientifically proven, it is widely accepted that CPGs exist in humans. CPGs are neural circuits located in the spinal cord that produce rhythmic motor patterns seen in locomotion. In this study when the pedaling frequency repeatedly coincided with EMG activity, it was considered entrainment. EMG entrainment trends showed the hamstrings and gastrocnemius muscles increased significantly with the increase in cadence. The effect size for the hamstrings (.379) and the gastrocnemius (.288) shows a moderate strength in their relationships with cadence and EMG entrainment. The vastus, rectus femoris, and tibialis anterior muscles showed similar counts of EMG entrainment across different cadences, although not significant. This supports the likelihood of a combination of reflex responses and CPG contributing to EMG entrainment during locomotion after a motor complete SCI.

When comparing the sides of the body, there was a significant relationship at the left gastrocnemius muscle of the increase in cadence and EMG entrainment. This relationship showed a moderate to large effect size of .416. The same relationship was not seen in the right gastrocnemius muscles. There is limited research available addressing the difference in the EMG activity between sides of the lower limbs after SCI. Based on the injury, there may be some spared pathways to one side of the body but not the other. However the classification of SCI is determined by the neurological level of injury (NLI) which is defined as the most caudal segment of the spinal cord with remaining sensation and motor function on both sides of the body (Kirshblum et al., 2011). This is discussed further in the following section.

ZONE OF PARTIAL PRESERVATION

Within this study, some muscles had a greater response to changes in supraspinal input and/or cycling cadence (i.e. SOL, HAM, & GAS muscles). A possible explanation to this is if participants had any zones of partial preservation (ZPP) identified during their ASIA examination. ZPP is a term that refers to dermatomes and myotomes below the NLI with partially preserved functions (Rupp et al., 2021). ZPP is used only with injuries with the absence of sensory or motor functions at the most caudal segment of the spinal cord. When the ASIA examination is scored to determine the AIS, a ZPP greater than three levels below the NLI can be a defining factor as to whether the injury is classified as a B (sensory incomplete) or C (motor incomplete) (Rupp et al., 2021). In this study, all participants were classified as either an AIS A or B. But, it is possible that ZPPs may be present even if they were not significant enough to change the injury classification.

When examining for the presence of ZPPs, motor function is tested on a 0-5 scale with 0 being total paralysis and 5 being normal function (Rupp et al., 2021). To receive a score of 1, there must be a visible or palpable muscle contraction. Using a leg cycling model with EMG, as in this study, may be a useful tool to better predict whether any ZPPs remain after injury that may be present but not strong enough for a visible muscle contraction. A previous study with individuals with a sensorimotor complete cervical SCI showed a positive relationship between the magnitude of motor ZPPs and the possibility for functional recovery (Jaja et al., 2021). Further evidence has shown that in

individuals with motor complete SCI, a majority of functional recovery is likely to occur within the ZPP (Fawcett et al., 2007). If ZPPs can be identified, targeted therapies may increase the possibility for recovery within the ZPP.

STUDY LIMITATIONS

The predictive value of the results in this study may be limited by the small sample size of individuals with cervical and upper-thoracic motor complete SCIs. A larger sample size may lead to more meaningful outcomes.

The EMG analysis may also be influenced by cross-talk between muscles, because EMG electrodes were placed on the surface of the muscle, and EMG activity from neighboring muscles could have influenced the EMG recordings. Smaller muscle groups have been more susceptible to cross-talk interference.

This study did not have access to participants' original ASIA examinations. Although the AIS for each participant was available, the ZPPs (if present) were not available and could not be considered.

CONCLUSION AND FUTURE DIRECTIONS

This study shows that a leg cycling model can be used to identify the neural structures responsible for motor control after a motor complete SCI. A leg cycling model gives the ability to manipulate variables contributing to afferent feedback and supraspinal input. Future studies are needed to determine whether a leg cycling model can be used to further investigate the roles of afferent feedback and supraspinal input on muscle activation and to identify the presence of ZPPs. The results found in this study, and future studies, provide a greater understanding of the control of muscle activation and aid in developing new therapeutic methods for sensory and motor function recovery after a SCI. The leg cycling model could also have therapeutic benefits to motor function recovery and should be investigated.

REFERENCES

Ballaz, L., Fusco, N., Cretual, A., Langella, B., & Brissot, R. (2007). Acute peripheral blood flow response induced by passive leg cycle exercise in people with spinal cord injury. *Arch Phys Med Rehabil*, *88*(4), 471-476. https://doi.org/10.1016/j.apmr.2007.01.011

Barbeau, H., McCrea, D. A., O'Donovan, M. J., Rossignol, S., Grill, W. M., & Lemay, M. A. (1999). Tapping into spinal circuits to restore motor function. *Brain Res Brain Res Rev*, *30*(1), 27-51. https://doi.org/10.1016/s0165-0173(99)00008-9

Barbiellini Amidei, C., Salmaso, L., Bellio, S., & Saia, M. (2022). Epidemiology of traumatic spinal cord injury: a large population-based study. *Spinal Cord*. https://doi.org/10.1038/s41393-022-00795-w

Barroso, F. O., Torricelli, D., Moreno, J. C., Taylor, J., Gomez-Soriano, J., Bravo-Esteban, E., Piazza, S., Santos, C., & Pons, J. L. (2014). Shared muscle synergies in human walking and cycling. *J Neurophysiol*, *112*(8), 1984-1998. https://doi.org/10.1152/jn.00220.2014

Beres-Jones, J. A. (2004). The human spinal cord interprets velocity-dependent afferent input during stepping. *Brain*, *127*(10), 2232-2246. https://doi.org/10.1093/brain/awh252

Brinkhof, M. W., Al-Khodairy, A., Eriks-Hoogland, I., Fekete, C., Hinrichs, T., Hund-Georgiadis, M., Meier, S., Scheel-Sailer, A., Schubert, M., Reinhardt, J. D., & Swi, S. C. I. S. G. (2016). Health conditions in people with spinal cord injury: Contemporary evidence from a population-based community survey in Switzerland. *J Rehabil Med*, *48*(2), 197-209. https://doi.org/10.2340/16501977-2039

Buehner, J. J., Forrest, G. F., Schmidt-Read, M., White, S., Tansey, K., & Basso, D. M. (2012). Relationship between ASIA examination and functional outcomes in the NeuroRecovery Network Locomotor Training Program. *Arch Phys Med Rehabil*, *93*(9), 1530-1540. https://doi.org/10.1016/j.apmr.2012.02.035

Davis, G. M., Hamzaid, N. A., & Fornusek, C. (2008). Cardiorespiratory, metabolic, and biomechanical responses during functional electrical stimulation leg exercise: health and fitness benefits. *Artif Organs*, *32*(8), 625-629. https://doi.org/10.1111/j.1525-1594.2008.00622.x

Dietz, V., Wirz, M., Curt, A., & Colombo, G. (1998). Locomotor pattern in paraplegic patients: training effects and recovery of spinal cord function. *Spinal Cord*, *36*(6), 380-390. https://doi.org/10.1038/sj.sc.3100590

Dobkin, B. H., Harkema, S., Requejo, P., & Edgerton, V. R. (1995). Modulation of locomotor-like EMG activity in subjects with complete and incomplete spinal cord injury. *J Neurol Rehabil*, *9*(4), 183-190. https://www.ncbi.nlm.nih.gov/pubmed/11539274

Dolbow, D. R., Gorgey, A. S., Moore, J. R., & Gater, D. R. (2012). Report of practicability of a 6-month home-based functional electrical stimulation cycling program in an individual with tetraplegia. *The Journal of Spinal Cord Medicine*, *35*(3), 182-186. https://doi.org/10.1179/2045772312y.0000000007

Dzeladini, F., van den Kieboom, J., & Ijspeert, A. (2014). The contribution of a central pattern generator in a reflex-based neuromuscular model. *Front Hum Neurosci*, *8*, 371. https://doi.org/10.3389/fnhum.2014.00371

Edgerton, V. R., Leon, R. D., Harkema, S. J., Hodgson, J. A., London, N., Reinkensmeyer, D. J., Roy, R. R., Talmadge, R. J., Tillakaratne, N. J., Timoszyk, W., & Tobin, A. (2001). Retraining the injured spinal cord. *The Journal of Physiology*, *533*(1), 15-22. https://doi.org/10.1111/j.1469-7793.2001.0015b.x

Edgerton, V. R., Tillakaratne, N. J. K., Bigbee, A. J., De Leon, R. D., & Roy, R. R. (2004). PLASTICITY OF THE SPINAL NEURAL CIRCUITRY AFTER INJURY. *Annual Review of Neuroscience*, *27*(1), 145-167. https://doi.org/10.1146/annurev.neuro.27.070203.144308

Fawcett, J. W., Curt, A., Steeves, J. D., Coleman, W. P., Tuszynski, M. H., Lammertse, D., Bartlett, P. F., Blight, A. R., Dietz, V., Ditunno, J., Dobkin, B. H., Havton, L. A., Ellaway, P. H., Fehlings, M. G., Privat, A., Grossman, R., Guest, J. D., Kleitman, N., Nakamura, M., . . . Short, D. (2007). Guidelines for the conduct of clinical trials for spinal cord injury as developed by the ICCP panel: spontaneous recovery after spinal cord injury and statistical power needed for therapeutic clinical trials. *Spinal Cord*, *45*(3), 190-205. https://doi.org/10.1038/sj.sc.3102007

Field-Fote, E. C., Yang, J. F., Basso, D. M., & Gorassini, M. A. (2017). Supraspinal Control Predicts Locomotor Function and Forecasts Responsiveness to Training after Spinal Cord Injury. *Journal of Neurotrauma*, *34*(9), 1813-1825. https://doi.org/10.1089/neu.2016.4565

Graziano, A., Foffani, G., Knudsen, E. B., Shumsky, J., & Moxon, K. A. (2013). Passive exercise of the hind limbs after complete thoracic transection of the spinal cord promotes cortical reorganization. *PLoS One*, *8*(1), e54350. https://doi.org/10.1371/journal.pone.0054350

Gregory, C. M., & Bickel, C. S. (2005). Recruitment patterns in human skeletal muscle during electrical stimulation. *Phys Ther*, *85*(4), 358-364. https://www.ncbi.nlm.nih.gov/pubmed/15794706

Harkema, S. J., Hurley, S. L., Patel, U. K., Requejo, P. S., Dobkin, B. H., & Edgerton, V. R. (1997). Human lumbosacral spinal cord interprets loading during stepping. *J Neurophysiol*, *77*(2), 797-811. https://doi.org/10.1152/jn.1997.77.2.797

Hoover, C., Schuerger, W., Balser, D., McCracken, P., Murray, T. A., Morse, L., Parr, A., Samadani, U., Netoff, T. I., & Darrow, D. P. (2023). Neuromodulation Through Spinal Cord Stimulation Restores Ability to Voluntarily Cycle After Motor Complete Paraplegia. *J Neurotrauma*. https://doi.org/10.1089/neu.2022.0322

Hug, F., & Dorel, S. (2009). Electromyographic analysis of pedaling: A review. *Journal of Electromyography and Kinesiology*, *19*(2), 182-198. https://doi.org/10.1016/j.jelekin.2007.10.010

Ibitoye, M., Estigoni, E., Hamzaid, N., Wahab, A., & Davis, G. (2014). The Effectiveness of FES-Evoked EMG Potentials to Assess Muscle Force and Fatigue in Individuals with Spinal Cord Injury. *Sensors*, *14*(7), 12598-12622. https://doi.org/10.3390/s140712598

Jahn, K., Deutschlander, A., Stephan, T., Kalla, R., Wiesmann, M., Strupp, M., & Brandt, T. (2008). Imaging human supraspinal locomotor centers in brainstem and cerebellum. *Neuroimage*, *39*(2), 786-792. https://doi.org/10.1016/j.neuroimage.2007.09.047

Jaja, B. N. R., Badhiwala, J., Guest, J., Harrop, J., Shaffrey, C., Boakye, M., Kurpad, S., Grossman, R., Toups, E., Geisler, F., Kwon, B., Aarabi, B., Kotter, M., Fehlings, M. G., & Wilson, J. R. (2021). Trajectory-Based Classification of Recovery in Sensorimotor Complete Traumatic Cervical Spinal Cord Injury. *Neurology*, *96*(22), e2736-e2748. https://doi.org/10.1212/WNL.0000000000012028

Kirshblum, S. C., Burns, S. P., Biering-Sorensen, F., Donovan, W., Graves, D. E., Jha, A., Johansen, M., Jones, L., Krassioukov, A., Mulcahey, M. J., Schmidt-Read, M., & Waring, W. (2011). International standards for neurological classification of spinal cord injury (revised 2011). *J Spinal Cord Med*, *34*(6), 535-546. https://doi.org/10.1179/204577211X13207446293695

Kriellaars, D. J., Brownstone, R. M., Noga, B. R., & Jordan, L. M. (1994). Mechanical entrainment of fictive locomotion in the decerebrate cat. *J Neurophysiol*, *71*(6), 2074-2086. https://doi.org/10.1152/jn.1994.71.6.2074

Leiras, R., Cregg, J. M., & Kiehn, O. (2022). Brainstem Circuits for Locomotion. *Annu Rev Neurosci*, *45*, 63-85. https://doi.org/10.1146/annurev-neuro-082321-025137

Maher, J. L., McMillan, D. W., & Nash, M. S. (2017). Exercise and Health-Related Risks of Physical Deconditioning After Spinal Cord Injury. *Topics in Spinal Cord Injury Rehabilitation*, *23*(3), 175-187. https://doi.org/10.1310/sci2303-175

Martin, J. H. (2022). Neuroplasticity of spinal cord injury and repair. *Handb Clin Neurol*, *184*, 317-330. https://doi.org/10.1016/B978-0-12-819410-2.00017-5

Morrison, S. A., Lorenz, D., Eskay, C. P., Forrest, G. F., & Basso, D. M. (2018). Longitudinal Recovery and Reduced Costs After 120 Sessions of Locomotor Training for Motor Incomplete Spinal Cord Injury. *Arch Phys Med Rehabil*, *99*(3), 555-562. https://doi.org/10.1016/j.apmr.2017.10.003

Nardone, R., Orioli, A., Golaszewski, S., Brigo, F., Sebastianelli, L., Holler, Y., Frey, V., & Trinka, E. (2017). Passive cycling in neurorehabilitation after spinal cord injury: A review. *J Spinal Cord Med*, *40*(1), 8-16. https://doi.org/10.1080/10790268.2016.1248524

Oliver, K. M., Florez-Paz, D. M., Badea, T. C., Mentis, G. Z., Menon, V., & de Nooij, J. C. (2021). Molecular correlates of muscle spindle and Golgi tendon organ afferents. *Nat Commun*, *12*(1), 1451. https://doi.org/10.1038/s41467-021-21880-3

Pernia-Andrade, A. J., Wenger, N., Esposito, M. S., & Tovote, P. (2021). Circuits for State-Dependent Modulation of Locomotion. *Front Hum Neurosci*, *15*, 745689. https://doi.org/10.3389/fnhum.2021.745689

Phadke, C. P., Vierira, L., Mathur, S., Cipriano, G., Jr., Ismail, F., & Boulias, C. (2019). Impact of Passive Leg Cycling in Persons With Spinal Cord Injury: A Systematic

Review. *Top Spinal Cord Inj Rehabil*, *25*(1), 83-96. https://doi.org/10.1310/sci18-00020

Rouffet, D. M., Mornieux, G., Zameziati, K., Belli, A., & Hautier, C. A. (2009). Timing of muscle activation of the lower limbs can be modulated to maintain a constant pedaling cadence. *J Electromyogr Kinesiol*, *19*(6), 1100-1107. https://doi.org/10.1016/j.jelekin.2008.11.014

Roy, R. R., Harkema, S. J., & Edgerton, V. R. (2012). Basic Concepts of Activity-Based Interventions for Improved Recovery of Motor Function After Spinal Cord Injury. *Archives of Physical Medicine and Rehabilitation*, *93*(9), 1487-1497. https://doi.org/10.1016/j.apmr.2012.04.034

Rupp, R., Biering-Sorensen, F., Burns, S. P., Graves, D. E., Guest, J., Jones, L., Read, M. S., Rodriguez, G. M., Schuld, C., Tansey-Md, K. E., Walden, K., & Kirshblum, S. (2021). International Standards for Neurological Classification of Spinal Cord Injury: Revised 2019. *Top Spinal Cord Inj Rehabil*, *27*(2), 1-22. https://doi.org/10.46292/sci2702-1

Steuer, I., & Guertin, P. A. (2019). Central pattern generators in the brainstem and spinal cord: an overview of basic principles, similarities and differences. *Rev Neurosci*, *30*(2), 107-164. https://doi.org/10.1515/revneuro-2017-0102

Szecsi, J., Straube, A., & Fornusek, C. (2014). A biomechanical cause of low power production during FES cycling of subjects with SCI. *Journal of NeuroEngineering and Rehabilitation*, *11*(1), 123. https://doi.org/10.1186/1743-0003-11-123

Tien, N. L. B., Thanh, V. V., Hanh, K. T. H., Anh, P. G., Huyen, L. T. M., Tu, N. T., Mai, D. T. N., & Toi, P. L. (2021). Quality of Life and Activities of Daily Living among Patients with Complete Cervical Spinal Cord Injury and Surgical Treatment in Vietnam. *Int J Environ Res Public Health*, *18*(18). https://doi.org/10.3390/ijerph18189703

Van Ingen Schenau, G. J., Dorssers, W. M., Welter, T. G., Beelen, A., De Groot, G., & Jacobs, R. (1995). The control of mono-articular muscles in multijoint leg extensions in man. *The Journal of Physiology*, *484*(1), 247-254. https://doi.org/10.1113/jphysiol.1995.sp020662

Zagrodny, B., Ludwicki, M., Wojnicz, W., Mrozowski, J., & Awrejcewicz, J. (2018). Cooperation of mono- and bi-articular muscles: human lower limb. *J Musculoskelet Neuronal Interact*, *18*(2), 176-182. https://www.ncbi.nlm.nih.gov/pubmed/29855439

APPENDIX

Abbreviations

ADL	Activity of Daily Living
AIS	American Spinal Injury Association Impairment Scale
AIS A	Complete Injury
AIS B	Sensory Incomplete Injury
AIS C	Motor Incomplete Injury with Motor Function in Less Than Half Key Muscles Below the Neurological Level of Injury
AIS D	Motor Incomplete Injury with Motor Function in More Than Half Key Muscles Below the Neurological Level of Injury
AIS E	Normal Function
ASIA	American Spinal Injury Association
BF	Biceps Femoris
BWST	Body Weight Supported Treadmill
CLR	Cerebellar Locomotor Region
CNS	Central Nervous System
CPG	Central Pattern Generator
EMG	Electromyography
EMG_{burst}	Electromyography Burst

$EMG_{entrain}$	Electromyography Entrainment
EMG_{RMS}	Electromyography Root-Mean-Squared
FES	Functional Electrical Stimulation
GAS	Gastrocnemius Muscles
GL	Gastrocnemius Lateralis
GM	Gastrocnemius Medialis
GTO	Golgi Tendon Organ
HAM	Hamstring
ISNCSCI	International Standards for Neurological Classification of Spinal Cord Injury
MLR	Mesencephalic Locomotor Region
NLI	Neurological Level of Injury
NRN	Neuro Recovery Network
QOL	Quality of Life
RF	Rectus Femoris
RPM	Revolutions Per Minute
SCI	Spinal Cord Injury
SLR	Subthalamic Locomotor Region
SOL	Soleus
TA	Tibialis Anterior
VAS	Vasti Muscles
VL	Vastus Lateralis

VM	Vastus Medialis
ZPP	Zone of Partial Preservation

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