

Determining the impact of in-bed leg cycling on spinal cord excitability during the acute phase following spinal cord injury

Jean-Marc Mac-Thiong

jean-marc.mac-thiong@umontreal.ca

Hopital du Sacre Coeur de Montreal <https://orcid.org/0000-0003-4524-3893>

Xavier Thibault

Andréane Richard-Denis

Université de Montréal <https://orcid.org/0000-0003-3638-4416>

Gerardo Tibamoso Pedraza

Dorothy Barthelemy

David Magnuson

University of Louisville <https://orcid.org/0000-0003-3816-3676>

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Abstract

Study Design: Prospective exploratory interventional study.

Objective: This study evaluates the impact of in-bed leg cycling on spinal cord excitability during the acute phase following acute traumatic spinal cord injury (SCI).

Setting: Level 1 trauma center specialized in SCI care in Montreal, Canada.

Methods: Eighteen participants with traumatic SCI underwent a single 30-minute session of in-bed leg cycling during acute hospitalization, with motor assistance as needed. Using soleus H-reflex testing, H-reflex and M-wave responses were recorded pre- and post-session to assess the change in spinal cord excitability.

Results: Changes in H-reflex parameters were observed after cycling even when performed passively. There were three main phenotypes but also large variability with regard to spinal cord excitability in response to cycling. Most individuals (9/18) had decreased excitability after cycling, while 6/18 individuals had increased excitability. A third group (3/18) did not exhibit any significant response to cycling. Spinal cord excitability never increased after cycling when performed 2 weeks or more after the SCI.

Conclusions: Performing in-bed leg cycling during the acute phase following traumatic SCI appears to modulate spinal cord excitability, but responses are highly individualized.

INTRODUCTION

Following the acute phase of the spinal shock in spinal cord injury (SCI), disinhibition associated with dysfunction of upper motor neurons can lead to spasticity in up to 60% of individuals during acute care [1]. Spasticity and spinal hyperreflexia result from the loss of presynaptic inhibition due to the SCI [2, 3], and are considered as signs of maladaptive plasticity (i.e. associated with poor neurofunctional recovery). They also often require treatment, cause pain, and/or impair function and quality of life [4, 5].

Preclinical studies suggest that activity-based (or exercise) therapy can reduce spasticity and spinal hyperreflexia, potentially by encouraging adaptive over maladaptive plasticity, even when performed passively [6, 7]. Activity-based therapy (e.g. cycling, locomotor training, etc.) after SCI stimulates recovery through a relearning process that involves task-specific repetitive neuromuscular activation below the level of the SCI [8, 9]. There is preclinical evidence that there is an early window of opportunity within days/weeks of a SCI for which activity-based therapy is most beneficial to harness adaptive plasticity and promote recovery [10, 11]. Rehabilitation training is mostly effective when performed early to circumvent the decline expression of plasticity-associated genes that are up-regulated directly following SCI, the degradation of neuronal function, and formation of a glial scar in the later stages [12]. Conversely, immobilization or disuse early after the SCI produces chronic spinal circuit hyper-excitability and limits recovery in animals [13, 14].

Passive cycling has been shown to suppress the soleus H-reflex in healthy individuals, indicating its potential to modulate spinal cord excitability through activity-dependent mechanisms [15]. The H-reflex, the electrical analogue of the mechanically induced spinal stretch reflex, provides a measure of spinal cord excitability from the responsiveness of alpha motor neurons to an electrical afferent input. When associated with functional electrical stimulation, passive cycling has the potential to decrease H-reflex response and reduce spasticity in humans with chronic SCI [16].

Despite the potential for activity-based therapy to modulate spinal cord excitability – and therefore harness adaptive plasticity – early after an acute SCI, it has never been assessed in humans. The aim of this study is to examine the effect of in-bed leg cycling on spinal cord excitability through soleus H-reflex testing within days of an acute traumatic SCI.

METHODS

Participants Three healthy volunteers were recruited as controls to undergo the same cycling protocol as for SCI individuals, in order to perform passive cycling. Eighteen individuals with an acute traumatic SCI participated in the study during their acute hospital stay at a single Level 1 trauma center specialized in SCI care between July 1st and August 31st 2024. The protocol was approved by the institutional review board. All participants provided informed consent. Inclusion criteria were: 1) acute traumatic SCI, 2) any severity of SCI with American Spinal Injury Association Impairment Scale (AIS) grade A, B, C, or D, 3) neurological level of injury (NLI) between C1 and L1, 4) blunt (non-penetrating) trauma, 5) spinal surgery performed ≤ 48 h of the injury. Exclusion criteria were: 1) condition limiting participant's ability to engage in cycling (e.g. pelvis or lower extremity injury or deformity, etc.), 2) medical contraindication to cycling (e.g. hemodynamic instability, cardiac ischemia, etc.), 3) moderate or severe traumatic brain injury, 4) pre-existing neurological condition (e.g. cerebrovascular disease, myasthenia gravis, Parkinson's disease, etc.).

Data collection Medical charts were used to collect all baseline information including age and sex. The neurological status at the time of cycling was assessed from the International Standards in Neurological Classification of SCI (ISNCSCI) to determine the NLI, AIS grade, and motor score. The timing of assessment after the trauma and the concomitant use of baclofen for spasticity was also collected.

In-bed leg cycling All participants completed a single 30-minute continuous session of in-bed leg cycling. They did not undergo another session of activity-based therapy during their acute hospitalization. During the cycling session, participants remained in their hospital bed in a semi-reclined position, with a 30° torso inclination (Fig. 1). Participants wore running shoes to prevent against harmful shear and pressure on the heels during pedaling, while the thighs were secured by cuffs inter-connected by an elastic band to prevent the knees from flaring out during pedaling. The legs were positioned in leg supports mounted onto the pedals of a motorized ergometer with an adjustable frame (Hi-Lo Stand with APT-5 ergometer, Tzora Active Systems, Beachwood, OH, USA). The ergometer was positioned so that the knee was flexed at 30° when the pedal reaches the 3 o'clock position. If the participant is unable to

engage into active cycling due to the paralysis of the lower extremities (i.e. all individuals with AIS grade A, B or C lesions), the ergometer was then activated in its passive (motor-driven) mode until reaching a target cadence of 40 revolutions per minute. Participants were encouraged to engage in active pedaling as much as possible and as tolerated, and motor assistance was titrated to reach the target cadence. Controls were instructed not to provide any active effort from lower extremities, in order reproduce passive cycling as much as possible.

Soleus H-reflex testing Soleus H-reflex testing was performed immediately before and after the cycling session while in bed. Participants remained in a semi-reclined position at a 30° torso inclination (same as during cycling) with legs resting fully extended on the bed, out of the pedals and without shoes. The soleus H-reflex was evoked on the right side (N.B.: all participants exhibited symmetrical impairment in the lower extremities) by applying 1 ms electrical pulses in the popliteal fossa using a DS7AH HV Current Stimulator (Digitimer, UK) and 3M 2330N surface electrodes (3M, USA). Stimulation intensity started at 3 mA and was increased by 2 mA increments until reaching 25 mA, and then by 5 mA increments until reaching a maximal stimulation of 75 mA. Each stimulation was delivered once with a 10-second interval between pulses. Electromyographic (EMG) activity was recorded from the right soleus muscle at 2,000 Hz using the Noraxon Ultium EMG Sensor System (Noraxon, AZ, USA) with Red Dot electrodes (3M, USA) according to SENIAM guidelines. The equipment (and manipulations from the assessor) was kept out of sight of participants during H-reflex testing. The assessor noted when the H-reflex was visible, while participants indicated verbally the minimal intensity for which the stimulation was felt, with the option to stop the experiment anytime if pain or discomfort occurred. The M-wave and H-reflex were recorded during reflex stimulation to assess neuromuscular function. Hmax and Mmax correspond to the maximal amplitudes of the H-reflex and M-wave, respectively, and are reached by progressively increasing the intensity of stimulation. Hmax and Mmax reflect the maximal activation of the reflex (afferent) and direct (efferent) motor pathways, respectively. Hmax/Mmax provides insight into the excitability of spinal reflex circuits modulated by the central nervous system, therefore reflecting spinal cord excitability. In addition, the H-reflex latency (time from stimulus to onset of H-wave) and H-reflex threshold (minimum stimulus required to elicit H-reflex) are also reported. H-reflex latency indicates the reflexive activation of motor neurons via Ia afferent fibers, providing a measure of the spinal reflex pathway excitability. In addition to descriptive statistics, these parameters were compared before vs. after cycling using non-parametric sign tests with a level of significance of 0.05.

RESULTS

Participants The three controls consisted in a 24-year-old female, a 23-year-old male, and a 19-year-old female performing a session of passive cycling. Twenty individuals with SCI were initially approached, and all consented to participate in the study. However, one individual was excluded due to imminent transfer to a rehabilitation facility the same day, such that the time available to complete the study was insufficient. Another individual dropped out after 6 minutes of cycling due to pain, and was not considered in the final analysis. The final study sample for analysis consisted of 18 male participants (Table 1) with a mean age of 51.7 ± 20.2 years (range: 19–81 years), who all completed the full 30-

minute cycling session and H-reflex testing before and after cycling. The timing of the cycling session was 11.1 ± 10.3 days after the trauma (range: 1–41 days). There were 6 participants with AIS grade A (4 cervical and 2 thoracic NLI), 6 with grade B (5 cervical and 1 thoracic NLI), 5 with grade C (all with cervical NLI), and 1 with C4 grade D SCI.

Table 1
Characteristics of participants and controls

Patient ID	Age	Sex	AIS* grade	NLI [†]	Total motor score	Timing of H-reflex testing after injury	Baclofen received
1	64	M	B	T4	38	16	No
2	71	M	A	T3	0	31	No
3	37	M	C	C5	18	41	No
4	61	M	C	C2	36	15	Yes
5	47	M	A	T6	50	10	Yes
6	76	M	D	C4	81	14	Yes
7	81	M	A	C5	11	6	Yes
8	50	M	A	C5	19	12	No
9	33	M	B	C4	16	1	No
10	24	M	B	C4	19	11	No
11	79	M	C	C5	2	5	No
12	70	M	C	C5	54	5	No
13	22	M	B	C8	48	5	No
14	32	M	A	C6	24	3	No
15	48	M	B	C6	28	5	No
16	19	M	C	C7	45	13	No
17	54	M	A	C3	8	3	No
18	62	M	B	C4	37	4	No
Control 1	24	F	-	-	-	-	-
Control 2	23	M	-	-	-	-	-
Control 3	20	F	-	-	-	-	-
* American Spinal Injury Association Impairment Scale							
† Neurological level of injury							

H-reflex testing Individual parameters for H-reflex testing are presented in Table 2. A systematic decrease in Hmax/Mmax was observed after passive cycling for all three controls (31.4%, 31.8% and

17.1% decrease). These observations correspond to a respective decrease in H-reflex amplitude by 22.0%, 19.9% and 1.6% and increase in M-wave amplitude by 13.8%, 17.5% and 18.7% after cycling. H-reflex latencies (≤ 0.002 seconds variation for each control) and H-reflex thresholds (≤ 2 mA variation for each control) remained relatively stable following cycling.

Table 2
H-Reflex data before and after cycling session of participants and controls

Patient ID	Before cycling session			After cycling session			Change in Hmax/Mmax (%)
	H-reflex latency (seconds)	H-reflex threshold (mA)	Hmax/Mmax	H-reflex latency (seconds)	H-reflex threshold (mA)	Hmax/Mmax	
1	0.032	19	0.06	0.033	13	0.04	-37.0
2	0.035	13	0.30	0.036	9	0.25	-15.4
3	0.031	25	0.68	0.031	21	0.68	-0.4
4	0.035	15	4.49	0.036	13	0.73	-83.7
5	0.032	11	0.16	0.031	9	0.11	-30.8
6	0.039	23	0.19	0.040	23	0.13	-30.6
7	0.034	15	0.19	0.035	15	0.31	64.8
8	0.033	13	0.45	0.032	9	0.58	29.0
9	0.036	21	0.83	0.036	21	0.67	-18.9
10	0.029	23	0.42	0.029	25	0.57	36.0
11	0.038	15	0.08	0.034	13	0.06	-22.1
12	0.039	7	0.09	0.039	15	0.70	657.8
13	0.033	9	0.35	0.033	9	0.19	-46.0
14	0.040	9	0.17	0.041	9	0.18	7.9
15	0.030	25	0.50	0.030	25	0.76	50.6
16	0.038	13	0.39	0.038	15	0.40	2.8
17	0.036	15	0.18	0.038	13	0.28	54.9
18	0.028	21	0.86	0.035	30	0.27	-68.6
Control 1	0.032	11	0.31	0.032	9	0.22	-31.5
Control 2	0.032	5	0.65	0.032	5	0.44	-31.9
Control 3	0.028	23	0.52	0.030	25	0.43	-17.1

All participants required motor assistance during cycling, ranging from full assistance for individuals with AIS grades A and B SCI to minimal assistance for the individual with AIS grade D SCI. All individuals tolerated the H-reflex stimulation protocol, and there was no adverse event associated with the protocol. There was no statistical difference before vs. after cycling in Hmax ($p = 0.4$), Mmax ($p = 0.5$), H-reflex latency ($p = 0.2$) and H-reflex threshold ($p = 0.5$).

Overall, Hmax/Mmax was similar statistically before (mean: 0.58 ± 1.01 ; median: 0.32; range: 0.06–4.49; interquartile range: 0.17–0.49) vs. after (mean: 0.31 ± 1.62 ; median: 0.30; range: 0.04–0.76; interquartile range: 0.18–0.65) cycling ($p = 0.8$). However, the individual change in Hmax/Mmax following cycling varied substantially from a decrease by 83.7% in participant #4 to an increase by 657.8% in participant #12 (Fig. 2). Hmax/Mmax decreased by at least 10% after cycling in 9 individuals (2 AIS grade A, 4 AIS grade B, 2 AIS grade C, 1 AIS grade D), and increased by at least 10% in 6 individuals (3 AIS grade D, 2 AIS grade B, 1 AIS grade C). The change in Hmax/Mmax after cycling was less than 10% in only 3 participants (1 AIS grade A, 2 AIS grade C).

Specifically for AIS grade A participants, Hmax/Mmax increased after cycling in all individuals with a cervical NLI and decreased after cycling in all individuals with a thoracic NLI. There was no clear trend observed for participants with AIS grade B or C lesions, as opposite changes in Hmax/Mmax were observed among some participants despite having the same AIS grade and NLI. One individual with a C4 AIS grade B lesion (participant #10) had a 36.0% increase in Hmax/Mmax, while the two others with the same lesion (participants #9 and #18) had a decrease in Hmax/Mmax by 18.9% and 68.6%, respectively. Of the 3 individuals with a C5 AIS grade C lesion, Hmax/Mmax increased by 657.8% in participant #12, while it decreased by 22.1% in participant #11 and remained stable (-0.4%) in participant #3. In the individual with mild AIS grade D SCI (C4 NLI with motor score of 81), Hmax/Mmax decreased by 30.6% after cycling, which is in the same range at the decreases in Hmax/Mmax observed in the healthy controls.

Of the 9 individuals who performed cycling within a week of the injury, Hmax/Mmax decreased by at least 10% in 4 individuals (C4 AIS grade B, C8 AIS grade B, C5 AIS grade C, C4 AIS grade B), increased by at least 10% in 4 individuals (C5 AIS grade A, C5 AIS grade C, C6 AIS grade B, C3 AIS grade A), and remained stable in 1 individual (C6 AIS grade A). However, Hmax/Mmax did not increase in any of the 5 individuals performing the cycling 2 weeks or more after the injury. Of note, all 4 individuals with a cervical AIS grade A lesion were tested within 2 weeks of the injury, and had an increase in Hmax/Mmax after cycling. Of the 9 participants who performed cycling more than one week following the injury, Hmax/Mmax increased in only 2 individuals (11 days for participant #10 and 12 days for participant #8).

Among the 4 participants taking baclofen to alleviate their spasticity, Hmax/Mmax decreased by 30% or more in 3 individuals (C2 AIS grade C, T6 AIS grade A, C4 AIS grade D), and increased by 64.8% in the other individual (C5 AIS grade A). Of the 10 participants younger than 60 years old, Hmax/Mmax decreased by at least 10% in 3 individuals (T6 AIS grade A, C4 AIS grade B, C8 AIS grade B), increased by 10% or more in 4 individuals (C5 AIS grade A, C4 AIS grade B, C6 AIS grade B, C3 AIS grade A), and

remained within a 10% increase in 3 individuals (C5 AIS grade C, C6 AIS grade A, C7 AIS grade C). For those older than 60 years old, Hmax/Mmax decreased by at least 10% in 6 individuals (T4 AIS grade B, T3 AIS grade A, C2 AIS grade C, C4 AIS grade D, C5 AIS grade C, C4 AIS grade B), and increased by 10% or more in 2 individuals (C5 AIS grade A, C5 AIS grade C).

DISCUSSION

Our study reports the changes in spinal cord excitability due to early leg cycling performed during the acute phase after traumatic SCI, as early as 1 day following the SCI. It sheds new light on the possible impact of initiating early acute activity-based therapy on spinal cord excitability and its potential to harness adaptive neuroplasticity after traumatic SCI, that have been demonstrated previously in preclinical studies. We used H-reflex testing to assess spinal cord excitability because it is recognized as a relevant tool to study plasticity of the nervous system [17, 18]. Our study shows that after acute traumatic SCI, modulation of spinal cord excitability can be achieved from early acute cycling – as soon as 1 day after the injury –, even if performed passively in the presence of severe (or complete) paralysis of the lower extremities. This finding suggests that the afferent input (proprioceptive and plantar pressure) may be sufficient to modulate spinal cord excitability, in agreement with animal studies of early acute activity-based therapy showing that even limited sensory feedback may be sufficient to improve motor function [19]. However, it remains to be determined from future studies whether providing a full daily program of early acute activity-based therapy in the form of in-bed cycling would translate into adaptive plasticity and improved recovery.

Our findings with previous studies of chronic SCI [16] showing that spinal cord excitability systematically decreases when it is assessed months or years after SCI. Our study shows that there are three main phenotypes and large variability with regard to spinal cord excitability in response to leg cycling during the acute phase days to weeks after traumatic SCI. Most individuals (9/18) had decreased excitability after cycling, although a large proportion of individuals (6/18) had increased excitability after cycling. A third group (3/18) did not exhibit any significant response from cycling. Observing different phenotypes in response to cycling within days/weeks after the SCI – as opposed to the systematic decrease when performed months/years after the SCI – suggests that starting activity-based therapy early vs. later after a SCI may result in different neuroplasticity and recovery processes, in agreement with preclinical studies showing that neurofunctional recovery is influenced by the timing at which activity-based therapy is introduced after the SCI [10, 11].

Surprisingly, some participants with the same SCI severity had completely different responses to cycling, further suggesting that the impact of cycling remains unpredictable during the acute phase, particularly for individuals with AIS grade B and C lesions (see Fig. 2 for participants with C4 AIS grade B and C5 AIS grade C lesions). However, the response to cycling was more consistent for complete AIS grade A SCI since all 4 participants with a cervical lesion exhibited increased spinal cord excitability after cycling – including 3 with more than 10% increase –, and the 2 participants with a thoracic lesion exhibited decreased excitability. The presence of preexisting degenerative cervical myelopathy can potentially

influence spinal cord excitability and the response to cycling since the 3 participants with more than 10% increase in Hmax/Mmax had underlying cervical spinal stenosis on MRI contributing to the SCI. Alternatively, the discrepancy between complete cervical and thoracic lesions could partly be explained by different pathophysiological processes involved in cervical vs. thoracic injuries considering that complete thoracic SCI are typically associated with trauma of higher energy and poorer potential for recovery [20].

As for mild AIS grade D SCI, our study does not provide definite conclusions, as there was only one participant with such injury. Nevertheless, the decrease in spinal cord excitability observed in this participant after cycling was in the same range as that observed in healthy controls. This suggests that individuals with the mildest injuries are more likely to exhibit decreased spinal cord excitability after early acute cycling, which is usually observed in normal individuals after cycling. Accordingly, our findings in healthy controls are consistent with the H-reflex attenuation normally observed during either passive or active cycling in healthy individuals [21].

We did not observe any clear tendency in the response to cycling based on age or on the concomitant use of baclofen. The impact of the timing of cycling session on the direction or magnitude of change in spinal cord excitability was not consistent throughout the entire cohort. However, there was no occurrence of increase in spinal cord excitability in any of the 5 individuals (1 AIS grade A, 1 AIS grade B, 2 AIS grade C, 1 AIS grade D) performing the cycling session 2 weeks or more after the SCI, which is similar to the observations in individuals with chronic SCI for whom spinal reflex excitability was reduced with activity-based therapy [22–25]. Conversely for the 13 individuals who performed their cycling session within 2 weeks of the injury, 7 had increased spinal cord excitability after cycling, 5 had decreased spinal cord excitability, and 1 did not respond to cycling. Of note, all 4 individuals with a complete AIS grade A cervical SCI performed cycling within 2 weeks of the injury and had an increase in spinal cord excitability. Further study is needed to determine whether performing cycling sessions earlier after the injury increases the probability of increasing spinal cord excitability, particularly for individuals with complete lesions for whom the spinal shock could be more severe and prolonged. Whether increasing spinal cord excitability early after the SCI is beneficial for accelerating the resolution of the spinal shock, harnessing adaptive plasticity and/or promoting recovery remains unknown, and cannot be answered from the current study.

Limitations The conclusions are limited by the small number of participants, and further study is required to identify predictors of changes in spinal cord excitability due to cycling. In addition, the response to a single cycling session cannot directly be translated into the outcomes with a full program of activity-based therapy for which sustained daily sessions of cycling would be performed. It is also possible that the impact of cycling on spinal cord excitability varies along the course of activity-based therapy. Conducting a study involving a full program of activity-based therapy is therefore needed to determine the relationship between the direction (increase vs. decrease) and magnitude of change in spinal cord excitability, plasticity and recovery.

Conclusion

Overall, this study shows that spinal cord excitability may be modulated by in-bed leg cycling after acute spinal cord injury, even when performed passively. Performing in-bed leg cycling within 2 weeks of a traumatic SCI had variable impact on H-reflex (increase, decrease or no effect). When performed more than 2 weeks after the injury, H-reflex was more likely to be decreased after cycling. Further study is required to assess the relationship between initiating in-bed leg cycling during acute care to provide early acute activity-based therapy, neuroplasticity and recovery.

Declarations

DECLARATION OF INTERESTS

XT received a scholarship from the PRogramme d'Excellence en Médecine pour l'Initiation en Recherche (PREMIER).

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ETHICAL APPROVAL

All experimental procedures were approved by the *Comité d'Éthique de la recherche du CIUSSS du Nord-de-L'Île de Montréal* ("Mobilisation précoce suite à une lésion médullaire", study #2020 – 1901, approved on March 12th, 2020). Participants provided written informed consent prior to enrollment in the study.

AUTHOR CONTRIBUTIONS

XT was involved in data curation, formal analysis, investigation, methodology, validation, writing original draft, and reviewing & editing manuscript.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are not publicly available. Data are, however, available from the authors upon reasonable request and with permission from the Ethics committee.

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Figures



Figure 1

In-bed leg cycling Participants remain in bed in a semi-reclined position with their torso inclined at 30° during cycling. The legs are placed in leg supports mounted on the pedals of the ergometer, while the thighs are secured by cuffs interconnected by an elastic band.

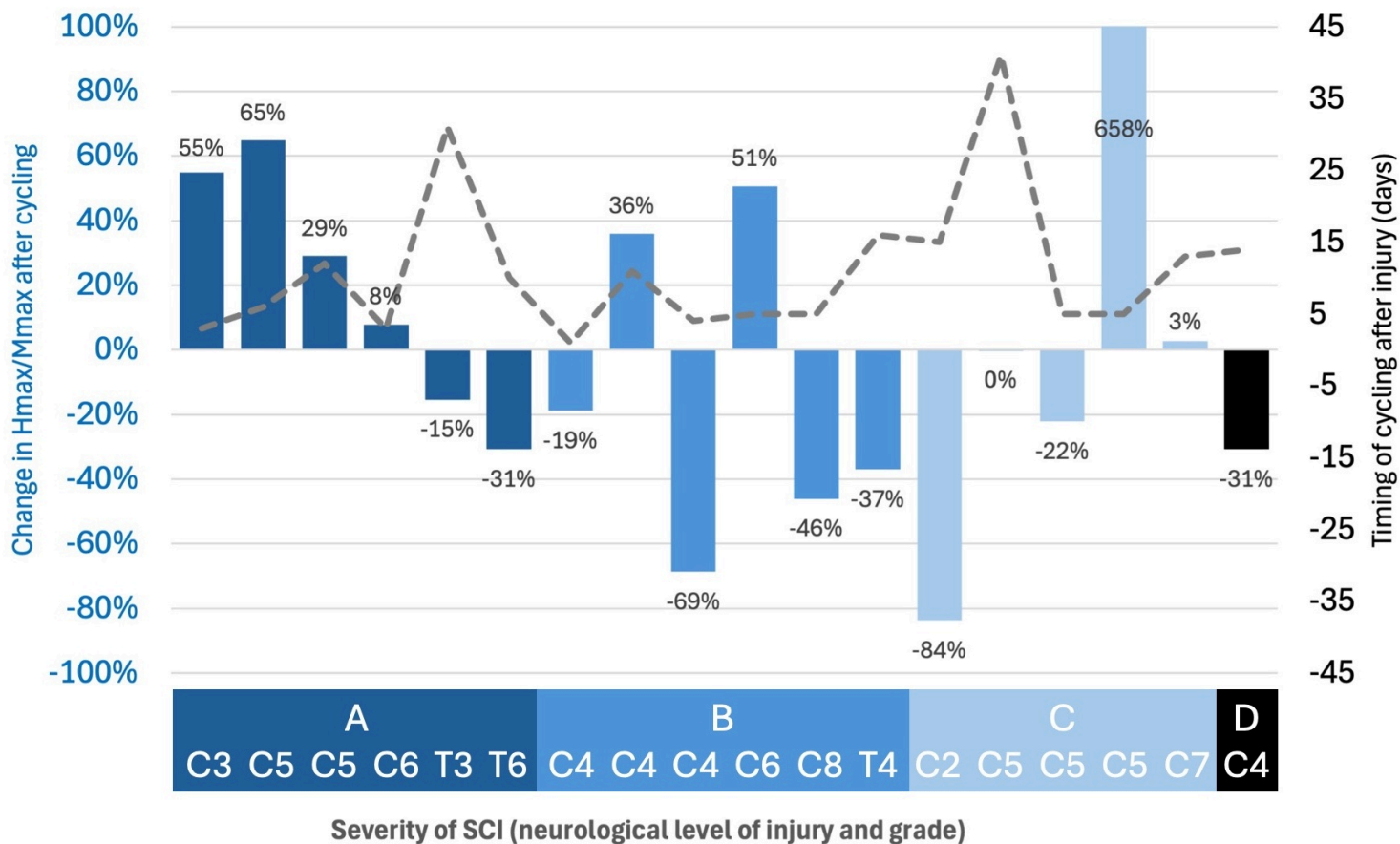


Figure 2

Modulation of spinal cord excitability from cycling The change in Hmax/Mmax following the cycling session is presented for each participant with SCI, based on the severity of SCI. The timing for cycling for each participant is also presented (dash line).