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ORIGINAL REPORT

PREVALENCE OF DISCOMPLETE SENSORIMOTOR SPINAL CORD INJURY AS EVIDENCED BY NEUROPHYSIOLOGICAL METHODS: A CROSS-SECTIONAL STUDY

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Objectives: To assess the prevalence of residual trans-lesion connectivity in persons with chronic clinically complete spinal cord injury (discompleteness) by neurophysiological methods.

Participants: A total of 23 adults with chronic sensorimotor complete spinal cord injury, identified through regional registries the regional spinal cord registry of Östergötland, Sweden.

Methods: Diagnosis of clinically complete spinal cord injury was verified by standardized neurological examination. Then, a neurophysiological examination was performed, comprising electroneurography, electromyography, sympathetic skin response and evoked potentials (sensory, laser and motor). Based on this assessment, a composite outcome measure, indicating either strong, possible or no evidence of discomplete spinal cord injury, was formed.

Results: Strong neurophysiological evidence of discomplete spinal cord injury was found in 17% (4/23) of participants. If also accepting "possible evidence", the discomplete group comprised 39% (9/23). The remaining 61% showed no neurophysiological evidence of discompleteness. However, if also counting reports of subjective sensation elicited during neurophysiological testing in the absence of objective findings, 52% (12/23) showed indication of discomplete spinal cord injury.

Conclusion: Evidence of discomplete spinal cord injury can be demonstrated using standard neurophysiological techniques in a substantial subset of individuals with clinically complete spinal cord injury. This study adds to the evidence base indicating the potential of various modes of cross-lesional sensorimotor functional restoration in some cases of chronic clinically complete spinal cord injury.

LAY ABSTRACT

Spinal cord injuries are usually classed as complete or incomplete. A complete injury implies that no residual function exists below the neurological level of injury. This status is determined by standardized neurological examination and is thought to correlate with spinal cord function in each individual with spinal cord injuries. However, studies have indicated that, in some people with complete spinal cord injury, there may be residual function that is not detected by such testing. The aim of the current study was to examine whether residual function, which is not detected by such testing, exists, and, if so, how common it is among people with complete spinal cord injuries (based on clinical testing) in a chronic stage (>2 years since injury). A battery of neurophysiological tests was used. Signs of "subclinical" residual function were found in 17–39% of 23 participants. This finding may lead to improvements in rehabilitative outcomes for people with complete spinal cord injury.

Spinal cord injury (SCI) causes sensorimotor deficits at and below the neurological level of injury (NLI), including paralysis and altered or lost sensory function. Autonomic functions are usually also impaired to some extent, affecting respiratory, circulatory, bladder, bowel and sexual functions (1). In addition, complications such as excessive spasticity and neuropathic pain are common (2, 3) and associated with decreased quality of life (4). There is no known unambiguous correlation between the degree of neurological deficits and the presence of such complications.

Currently, no methods are available in clinical routine practice for repairing SCI. However, as the concept of neuroplasticity has gained acceptance (5, 6), interest in exploring therapeutic neuro-modulation has increased.

SCI is classified as complete or incomplete, using the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) (7), which is based entirely on clinical sensorimotor examination. Based on the clinical assessment, SCI is sub-classified into 5 levels of varying residual infra-lesion neurological function; American Spinal Injury Association Impairment Scale (AIS) A–E (where A is complete functional transection,

and E is complete restitution of infra-lesional sensory and voluntary motor function). In some individuals with SCI of grade AIS A, there exists a "zone of partial preservation" (ZPP), defined as the most caudal segment with *some* sensory function, determined by pin-prick and light touch testing below NLI, without sacral preservation of sensory and voluntary motor function (7, 8).

A high prevalence (30–78%) of residual anatomical continuity was noted in post-mortem studies on clinically complete traumatic SCI (9, 10). It is likely that such anatomical continuity corresponds to a functional but subclinical neurological connectivity across the injury site in some cases. However, other pathways for such connectivity have also been proposed, e.g. the sympathetic trunk and/or the vagus nerve (11–14).

For residual subclinical trans-lesion connectivity, the term discomplete spinal cord injury (dSCI) was proposed (15). Various methods for detection of this phenomenon have since been used, and both motor and sensory discompleteness have been proposed.

For assessment of motor dSCI, electromyography (EMG) was the first method used (16). EMG recordings below the NLI can also be combined with transcranial magnetic stimulation (TMS) of the brain motor cortex (17, 18) to find signs of subclinical trans-lesion motor conduction.

Sensory dSCI has been studied using somatosensory-evoked potentials (SEP, 17–19), consisting of somatosensory peripheral stimulation (typically electrical nerve stimulation) under simultaneous registration of activity in the somatosensory cortex, using scalp electrodes. Functional magnetic resonance imaging (fMRI) appears to have a higher sensitivity for detecting remaining sensory connectivity in patients with clinically complete SCI (25–54% (19–21)), compared with SEP (13–25% (22, 23)):

In one study (23), 8/12 (67%) people with SCI and neuropathic pain perceived a change in perception below the NLI using adjuvant-enhanced thermal stimulation, compared with 0/12 (0%) for the group without neuropathic pain. Thus, a correlation was postulated between sensory dSCI and neuropathic pain after SCI.

In summary, evidence for the concept of dSCI has accumulated. However, most of these studies have been small, and methods and quality of control conditions have varied. The current study aimed to determine the prevalence of motor and/or sensory dSCI among persons with chronic clinically complete SCI, by strict application of current clinical diagnostic criteria, and use of multiple, standardized neurophysiological methods.

MATERIALS AND METHODS

Participants

A descriptive cohort study design was chosen. Potential participants were identified from an internal registry of patients with SCI at the Department of Rehabilitation Medicine, Linköping University Hospital, Linköping, Sweden. Medical records were screened for eligibility. All participants living in the region and fulfilling the study criteria (see below) were contacted by one of the authors (CW). Persons <2 years after SCI were excluded from the study, as prior studies have shown spontaneous conversion from complete to incomplete injuries in some cases (24).

In total, 24 participants fulfilling the criteria provided written informed consent and participated in the study. Ethical approval was obtained from the local Ethics Committee prior to recruitment, Linköping Regional Board of Ethics (dnr: 2016/433-31. The study was performed in accordance with the Declaration of Helsinki.

Inclusion criteria

Inclusion criteria were all of: acquired chronic (>2 years) sensorimotor complete (AIS A) SCI; NLI T12 or rostral; and resident of the regional county resident of Östergötland, Sweden.

Exclusion criteria

Exclusion criteria were any of: clinically detectable voluntary motor or sensory function more than 5 levels below the NLI; severe comorbidity (e.g. terminal cancer, psychosis or other diseases likely to preclude full participation); age >80 years; contraindications to TMS (e.g. presence of a ventriculo-peritoneal shunt or epilepsy.); and inability to communicate in Swedish or English.

Clinical evaluation

Each of the 24 participants was clinically evaluated by the one of the authors (CW), using a semi-structured interview and a standardized neurological examination (according to the ISNCSCI protocol (7)), including rectal examination, confirming an AIS A grade. The interview guide is shown in Appendix S1[1], and is based in part on the ISCOS Core (25) and Pain (26) International data-sets. This was confirmed in 23 participants. However, 1 participant had sacral sparing of sensibility, indicating an incomplete injury, and was therefore excluded from the study.

Basic descriptive data

Basic descriptive data pertaining to study participants are shown in Table I. Importantly, all participants were confirmed AIS A SCI, i.e. clinically complete. Furthermore, it was confirmed that all assessments of indications of dSCI were made below the NLI, and in cases with a ZPP, also below this zone. Presence or absence of neuropathic below-lesion pain and/or spasticity was also noted and quantified using a numerical rating scale (NRS), as such problems might correlate with presence or absence of dSCI.

Neurophysiological evaluation

The neurophysiological protocol comprised a battery of standard tests, each performed and evaluated according to clinical routine (signal quality and reference values) at the Department of Clinical Neurophysiology, Linköping University Hospital. No blinding or randomization was performed. To minimize the effect of interrater bias, each modality was performed by the same investigator in every patient, except for LEP, where 2 different examiners performed the testing. No medication was changed during the study.

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[1]http://www.medicaljournals.se/jrm/content/?doi=10.2340/16501977-2774

Table I. Basic descriptive data pertaining to study participants

Participant number	NLI	ZPP	Age, years	Sex	Time since injury, years	Traumatic	Aetiology	Neuropathic pain (last 14 days)	Spasticity (last 14 days)
1	C6	–	55	M	5	Yes	Falling	Daily	Daily
2	T4	T5	28	M	8	Yes	Vehicle accident	Daily	Daily
3	C4	C6	68	M	39	Yes	Falling	None	Daily
4	C4	–	51	M	17	Yes	Vehicle accident	Daily	Daily
5	T8	T9	39	M	5	Yes	Gunshot	Daily	Daily
6	T2	T4	59	M	35	Yes	Vehicle accident	Daily	Daily
7	T3	–	36	M	19	Yes	Vehicle accident	Daily	Often
8	C8	–	68	M	46	Yes	Diving	None	None
9	T12	L2	79	M	18	Yes	Falling	None	Daily
10	T5	T6	62	M	36	Yes	Vehicle accident	Daily	Daily
11	T2	T5	64	M	33	Yes	Vehicle accident	Often	Often
12	T3	T6	46	F	4	No	Myelitis	Daily	Daily
13	C4	C7	38	M	14	Yes	Vehicle accident	Daily	None
14	T11	–	50	M	28	Yes	Vehicle accident	Daily	None
15	C6	C7	59	M	9	Yes	Vehicle accident	Daily	Daily
16	T11	L1	73	M	52	Yes	Falling	Daily	None
17	T11	L2	22	F	9	Yes	Falling	Daily	Often
18	T8	T10	55	M	21	No	Infection	Daily	None
19	T5	T7	20	M	18	Yes	Falling	None	None
20	T7	T11	76	M	5	No	Bleeding	Daily	None
21	C4	C6	53	M	13	Yes	Gunshot	Daily	Daily
22	T4	T9	52	M	5	No	Cancer	Often	Daily
23	T2	T4	32	F	29	No	Infection	Often	Often

M: male; F: female; NLI: neurological level of injury (the most caudal level of intact sensory and motor function according to ISNCSCI); ZPP: zone of partial preservation (the most caudal segment of some sensory function, as determined by pin-prick and light touch testing below NLI without sacral preservation of sensory and voluntary motor function); Daily: present every day during the last 14 days; Often: present during at least 4 out of the last 14 days; None: not present at any time during the last 14 days.

Electroneurography. In the right upper extremity, motor and sensory nerve conduction tests of the median and ulnar nerves were performed using electroneurography (ENeG) (Synergy Carefusion, Carefusion, Middletown, WI, USA). For motor nerve stimulation, a hand-held bipolar stimulating-electrode (Natus Neurology, Middleton, WI, USA) and for sensory assessments, a digital ring electrode (Alpine Biomed/Natus Neurology, Middleton, WI, USA) were used respectively. The presence of peripheral nerve dysfunction unrelated to the SCI may affect assessment. The most common such interference is median nerve dysfunction due to carpal tunnel syndrome. To avoid this confounder, the ulnar nerve was also examined in the upper extremity. (However, this was not performed for the first 4 patients due to a procedural mistake. In the lower extremities, motor nerve conduction tests of the tibial and peroneal nerve, and sensory nerve conduction tests of the sural nerves were performed bilaterally (the equipment used was as described above, except that recording for the sural nerve was made with surface electrodes.) After analysis of signal quality and cursor settings, values of response latency, response amplitude and conduction velocity were graded "normal", "abnormal" or "absent".

Electromyography. Recordings were made using electromyography (EMG) with concentric needle electrodes (Ambu, Neuroline 38 × 0.45 mm electrodes, Cephalon A/S, Nørresundby, Denmark). In the right upper extremity, recordings were obtained from the abductor digiti minimi (ADM) and the abductor pollicis brevis (APB).

In both lower extremities recordings were made from the abductor hallucis (AH), the tibialis anterior (TA), the gastrocnemius (GCM) and the vastus lateralis (VL) muscles. In 3 participants the right leg could not be tested, due to amputation or similar causes.

For each muscle examined, 3 aspects were assessed: (*i*) spontaneous electrical muscle activity (fibrillations/positive waves and other spontaneous activity, such as complex repetitive discharges); (*ii*) motor unit potentials (MUP; amplitude, duration, number of phases); (*iii*) interference (capacity for activation at high frequency). All parameters were graded, using a semi-quantitative scale: 0 (missing)/N (normal), 1 (slightly increased), 2 (moderately/strongly increased), 3 (very strongly increased) or N/A (not applicable).

Sympathetic skin response. The effects on sympathetic skin response (SSR) of auditory (examiner suddenly and unexpectedly clapping hands), electrical (sudden and unexpected impulse of 20–40 mA on a region with intact sensation on the upper arm) and/or touch stimuli (a stroking motion of approximately 3 cm/s with the examiner's palm against the participant's upper arm) were assessed using standard surface electrodes (as for ENeG, described above) in extremities (both palms and dorsum of the feet) connected to a Natus machine. Changes in impedance secondary to altered perspiration were recorded from the aforementioned electrodes, giving an indication of autonomic nervous system function. Responses were graded as: clearly present (2), probably present (1), or not present (0).

Somatosensory-evoked potentials. Responses to electrical stimulation of the median (unilaterally) and tibial (bilaterally) nerves were recorded as somatosensory-evoked potentials (SEP). Using standard surface EEG electrodes (White Silver Electrode ACCE120100, Cephalon A/S, Nørresundby, Denmark), responses were recorded over the somatosensory cortex (electrode position C3' or C4' according to the 10–20 electrode positioning system) contralateral to stimulation with electrodes connected to a Natus machine. Standard response potentials were evaluated, i.e. N20 for median- and P40 for tibial nerve stimulation. Responses were graded as: clearly present (2), probably present (1), or not present (0).

Laser-evoked potentials. Laser-evoked potentials (LEP) were elicited using a neodymium: yttrium–aluminium–perovskite (Nd; YAP) laser (Stimul1340, DEKA Ltd, Calenzano, Italy) with laser light stimulating the dorsum of one hand and each foot, sequentially.

In healthy individuals, intensity is increased to achieve a slightly painful stinging/prickling sensation. If the participant could not sense stimulation of a lower energy level, 2.5 J was applied, which is generally considered safe, given the stimulation parameters (wavelength 1,340 nm, pulse duration 10 ms, spot diameter 4 mm) with repeated short 1.5–2.5 J bursts. LEPs were simultaneously recorded in a manner similar to SEP (electrode positions T3/T4 for the N1- and Cz for the N2-potential, according to the 10–20 system). First, electroencephalography (EEG; Fz, Cz, Pz, T3, T4, A1, A2 and both mastoids; M1, M2) and electrooculography (EOG; forehead and lateral to the eyes) surface electrodes (same mark as for SEP) were applied. The responses were recorded using a 64-channel amplifier (SynAmps RT, Compumedics Neuroscan, Charlotte, NC, USA) and analysed with neuroimaging software (CURRY 7; Compumedics Neuroscan and MATLAB 2017B; Mathworks Inc., Natick, MA, USA). The LEP recording was made using a standard technique aiming to define primarily the N2 and P2 potentials from the signal of the Cz electrode referenced to the mean of M1 and M2. N2 and P2 potentials were defined as the largest negative and positive peak, respectively, in the post-stimulus interval 0–500 ms. Peak latencies and amplitudes of the N2-P2 complex were then assessed by one of the authors (SA). Sequences in which the participant was blinking were manually excluded from analysis.

Responses were graded as: clearly present (2), probably present (1), or not present (0).

Motor-evoked potentials. Motor-evoked potentials (MEP) were obtained using transcranial magnetic stimulation (TMS; Magstim 200, The Magstim Company Ltd, Whitland, UK) over the primary motor cortex, bilaterally, with simultaneous EMG recording (Nicolet Biomedical, EMG surface electrode, Cephalon A/S, Nørresundby, Denmark) connected to a Natus machine. Recordings were made from the AH in each leg and the APB in the right hand. MEPs from TMS over the lower cervical and upper sacral spinal cord/nerve roots below the NLI were also determined in a similar manner. Responses were graded as: clearly present (2), probably present (1), or not present (0).

Subjective sensations. Throughout the neurophysiological assessment, it was also noted whether or not the participants experienced any physical sensations from the stimulation performed. If so, they were asked to localize and characterize the sensation.

RESULTS

The results of the neurophysiological assessments of study participants are shown in overview in Table II and are also presented separately in more detail for each modality below, and Appendix S2[1] and Appendix S3[1].

Electroneurography

On ENeG, normal (or close to normal) amplitudes and latencies were found in most or all nerves studied in approximately one-third of participants. In approximately the same proportion of participants, responses were highly abnormal or missing in most or all muscles. The rest showed a more mixed pattern of moderately abnormal responses mixed with some intact nerves and some completely unresponsive nerves (Appendix S2[1]). As expected, a higher proportion of nerves in the upper extremities had normal amplitudes and latencies, compared with nerves in the lower extremities. In total, 85 nerves were evaluated in the upper extremities (58% normal, 39% abnormal in latency and/or amplitude, and 2% absent) and 135 in the lower extremities (19% normal, 41% abnormal, 41% absent).

Electromyography

On EMG, spontaneous activity was seen in the majority of muscles below the NLI in most participants (Appendix S3[1]). In most cases in which spontaneous activity was absent, the muscle was atrophied. However, in a few muscles in 3 participants (P 5, 12 and 17) an interesting pattern was observed, consisting of: no spontaneous activity, in combination with some physical sensation from the same muscle, with no signs of atrophy.

Similarly, in one participant (participant 7), there was slight spontaneous activity (less than in other muscles in the same participant), in combination with physical sensations from that muscle, and no signs of atrophy.

Sympathetic skin response

A positive SSR in the lower extremity was seen bilaterally in one participant (participant 17). In 4 partici-

Table II. Overview of main neurophysiological results as related to cross-lesional connectivity

Participant number	NLI	ZPP	Age	ENeG	EMG	SSR	SEP	LEP	MEP	Total
1	C6	–	55							
2	T4	T5	28							
3	C4	C6	68							
4	C4	–	51							
5	T8	T9	39		2					2
6	T2	T4	59					2		2
7	T3	–	36	1						1
8	C8	–	68							
9	T12	L2	79							
10	T5	T6	62							
11	T2	T5	64							
12	T3	T6	46		2					2
13	C4	C7	38			1				1
14	T11	–	50							
15	C6	C7	59							
16	T11	L1	73							
17	T11	L2	22	2	2					2
18	T8	T10	55							
19	T5	T7	20							
20	T7	T11	76							
21	C4	C6	53			1				1
22	T4	T9	52			1				1
23	T2	T4	32			1				1

M: male; F: female; C: cervical; T: thoracic, L: lumbar; NLI: neurological level of injury (definition in Table I); ZPP: zone of partial preservation (see Table I). ENeG: electroneurography; EMG: electromyography; SSR: sympathetic skin response; SEP: sensory-evoked potentials; LEP: laser-evoked potentials; MEP: motor-evoked potentials; 1 denotes "possible" signs of discomplete; 2 denotes "strong" signs of discomplete. In the "total" column; 1 denotes "possible" signs of discomplete for at least 1 modality, and 2 denotes "strong" signs of discomplete for at least 1 modality.

pants (13, 21, 22 and 23), possible SSRs were seen in the lower extremities (unilaterally in participant 13 and bilaterally in the others).

Somatosensory-evoked potentials

For SEP, no responses were detected on stimulation of either lower extremity in any participant. Responses were detected on stimulation of the upper extremity in most participants (18/23), as expected. For the 5 participants with absent upper extremity SEPs, 4 had a high NLI C4-C6. For the remaining participant (P9, NLI Th12), it was unclear whether a SEP response from the upper extremity was present.

Laser-evoked potentials

For LEP, in one participant (P6) a positive response was detected on stimulation of the right lower extremity, well below his NLI (T2 with ZPP T4). In all other participants, no responses were detected on stimulation of the lower extremities. Responses were detected on stimulation of the upper extremities in most participants (12/17) with NLI C7 or lower, as expected. Of those 5 participants with NLI C7 or lower who *did not* have a LEP response from the upper extremities, all reported a prickling sensation during stimulation of the upper extremities, but none of them reported any sensation during laser stimulation of the lower extremities.

In individuals with NLI C6 or higher (6 participants), no responses were detected on laser stimulation of the hands. One of those participants felt a prickling sensation in both hands, and one felt a slight prickling in the right hand, but not the left hand. The remaining 4 participants reported no sensation upon laser stimulation of the hands.

Motor-evoked potentials

For MEP, in 21/23 participants, responses in the upper extremities were evoked from cortical stimulation. No response was seen in any lower extremity in any participant upon TMS of the primary motor cortex. In 9 patients, responses were seen in the lower extremities from cervical or lumbar stimulation, in 10 patients, no responses were detected on spinal stimulation. (Four participants did not receive spinal TMS due to presence of metallic instrumentation at the site of stimulation.)

Subjective sensations

During the neurophysiological assessments, subjective sensations were reported by 10/23 participants (43%). Six participants reported some sensation during 1 modality of testing (P3, 5, 6, 13, 15 and 22). Two participants reported sensation during 2 modalities

Table III. Self-rated mean intensity of spasticity and neuropathic pain (last 14 days, with treatment)

Group	Pain intensity	Spasticity intensity
Discomplete (strict criteria)	4.25 (2–6) $n=4$	1.75 (0–4) $n=4$
Discomplete (liberal criteria)	6 (2–10) $n=9$	2 (0–4.5) $n=9$
Non-discomplete (strict)	6 (0–10) $n=19$	0 (0–4.5) $n=18$*
Non-discomplete (liberal)	4 (0–10) $n=14$	0 (0–2) $n=13$*

Pain and spasticity intensity, rated on a numerical rating scale (0–10). Participants were asked: "How much does spasticity/neuropathic pain affect activities in your daily life?". Data are given as median (range).
*For spasticity intensity, data were missing for 1 participant, who was excluded from that analysis. Means and SD were also calculated and are shown in the Appendix S4[1].

(P7 and 11). Two participants reported sensations during 4 modalities (P12 and 17). These participants had a sensory score of 0 points in the corresponding dermatomes upon standardized clinical testing, prior to the neurophysiological assessments.

As can be seen in Table III, no statistically significant difference in median intensity was found between the dSCI group and the rest of the cohort (using the strict criteria to define the dSCI group,) regarding neuropathic pain or spasticity.

The analysis was repeated with the participants fulfilling the more liberal criteria also included in the dSCI group, also yielding non-significant results for neuropathic pain and spasticity. Analyses were performed using Mann–Whitney *U* test.

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DISCUSSION

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As mentioned in the introduction, several observations have sparked studies of how to enhance remaining neurological function after spinal cord injury. In that vein, studies using TMS in combination with peripheral nerve stimulation according to the paired associative stimulation (PAS) paradigm have indicated a potential for treatment-induced improvement in motor function in the upper and lower extremities (27–29). In addition, several studies have explored brain-computer interfaces (BCI) for restoration of neurological function after SCI (30–32).

In pioneering work based on EMG studies, Dimitrijevic (16, 33–35) proposed the concept of brain motor control assessment (BMCA). BMCA entails activating muscles above the NLI in order to increase the general excitability of the neuromuscular system (15, 36). The finding that some persons with motor complete (AIS A or B) SCI could exert some infra-lesion volitional motor control, albeit indirect (such as suppression of reflexes), on functionally paralysed muscles was the basis for the notion of dSCI proposed by these authors. Despite mounting evidence for the concept, current clinical assessment of SCI does not include assessment of dSCI. Neither is it included in the ISNCSCI, and thus does not influence standard rehabilitation strategies. However,

with the recent development of BCI and related methods of neuromodulation as potentially revolutionizing additions to rehabilitation, the detection and characterization of dSCI may become highly relevant.

The current study assessed the neurophysiological indications of trans-lesional sensorimotor connectivity in people with clinically complete, chronic SCI. Our findings provide additional support for the existence of dSCI in a significant minority of persons deemed to have complete SCI by current clinical classification. The estimated prevalence of dSCI among the participants with AIS A lesions was 17%, (4/23) when applying strong criteria, and 39%, (9/23) when applying liberal criteria ("possible" discomplete). If adding reports of any subjective sensation provoked by the sensory neurophysiological tests (being more intense than stimuli used to assess sensation according to standard clinical protocol), 52% (12/23) of participants showed some indication of dSCI (Fig. 1).

As a matter of course, neurophysiological registration within the ZPP is not informative as there is already, by definition, clinical evidence of some incompleteness within the ZPP. However, all registration in this study was performed well below the most caudal segment of ZPP (pertaining to the lower extremities). These findings indicate residual neuronal conduction beyond what was indicated by clinical assessment (ISNCSCI).

Thus, our findings confirm that ISNCSCI is insufficient for detection of neurological function in SCI. The results of the current study are also in line with previous research, indicating the presence of dSCI in a substantial subset of people with clinically complete SCI. Although the frequency of dSCI varies between study populations and methods utilized for its detection, the higher incidence estimate in the current study is supportive of recent fMRI studies showing a prevalence of sensory dSCI of approximately 50% (20, 21). Our study did not employ techniques such as capsaicin sensitization of peripheral nerve endings (23). The addition of such measures might have yielded an even higher proportion of discomplete SCI.

The principal modes of estimation vary between the methods used. For EMG, SEP, MEP and SSR, semi-quantitative assessments (strong-, possible- or no indication of discomplete SCI) were made by visual inspection of the recorded signals. For ENeG, quantitative evaluations of amplitudes, velocities and latencies were made. For LEP, the laboratory standard amplitude and latency cut-offs were used. Taken together, a strength of the current study is that a wide variety of methods were used to study possible remaining spinal connectivity. However, a possible weakness is that the study did not include sensory fMRI, a method that recent studies indicate to be a sensitive means to detect remaining connections (19–21).

Information regarding the presence or absence of excessive spasticity or neuropathic pain was obtained, as it has been proposed that dSCI may influence these phenomena. This study did not show any difference between the groups (dSCI vs non-dSCI) regarding pain or spasticity, regardless of which cut-off was used to define the groups (see Results). It is possible that the study lacked the statistical power to shed light on this question. It may also be that no such connections exist. Further, larger-scale studies are needed to address this important aspect.

CONCLUSION

Evidence of dSCI from a battery of neurophysiological methods assessing infra-lesional sensorimotor function was found in 17–52% of 23 people with chronic AIS A SCI. The prevalence estimates varied depending on choice of stringency criteria (see Fig. 1). This study lends support to the concept of discompleteness and, furthermore, indicates the feasibility of diagnosing dSCI by standard neurophysiological methods. It is notable that SEP and MEP failed to disclose indications of dSCI in this study, pointing to the need for further studies to define which methods to use, possibly in combination, in order to establish a reliable protocol for identifying and characterizing dSCI in clinical practice. Based on these results, the widely available method of EMG seems to be
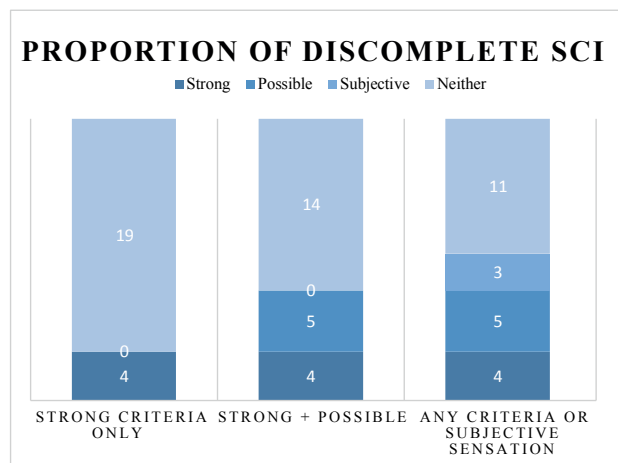

PROPORTION OF DISCOMPLETE SCI

■ Strong ■ Possible ■ Subjective ■ Neither

Fig. 1. Proportion of discomplete spinal cord injury (SCI) depending on criteria used (*n* = 23). "Strong" (*dark blue*) includes only the 4 participants with strong signs of cross-lesional connectivity (denoted 2 in Table I). "Possible" denotes the 5 participants with possible neurophysiological signs of cross-lesional connectivity (denoted 1 in Table I). "Subjective" refers to the 3 participants who reported some subjective physical sensation during neurophysiological testing, but were lacking objective signs. "Neither" (*lightest shade of blue*) refers to those participants who did not report any subjective sensation during testing, while also lacking objective signs.

a strong candidate for such a protocol. The identification of dSCI may prove to be of great clinical importance for the application of neuromodulation (such as navigated TMS) in neurological rehabilitation.

The authors have no interest of interest to declare.

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