Special article

Profiling motor control in spinal cord injury: Moving towards individualized therapy and evidence-based care progression

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Summary: This article, based on the keynote address at the 5th National Spinal Cord Injury Conference in Toronto, addresses methods to neurophysiologically characterize patients after spinal cord injury and proposes how those methods could be used to individualize therapeutic interventions and monitor their efficacy over the course of neurorehabilitation.

Keywords: Spinal cord injuries, Neurorehabilitation, Evidence based, Outcomes, Neurorecovery, Assessment, Neurophysiological

Introduction

The goal of neurorehabilitation in spinal cord injury is the recovery of function. Every clinician, however, has experienced the rather loose relationship between the therapeutic interventions we employ and the clinical effects they have, with some patients responding with impressive recovery and others without so much. Setting aside the treatment of secondary medical complications and the teaching of compensatory strategies to patients, we assume that the neurorehabilitation interventions we provide to enhance functional recovery, whether they are surgical, pharmacological, or rehabilitative, will work to affect the residual and probably re-organized nervous system that remains after spinal cord injury. Yet, most of our studies in neurorehabilitation rely on rather crude characterizations of that residual, re-organized nervous system and we measure the impact of our interventions using those same gross characterizations or by measuring aspects of the functional outcomes we hope our patients will obtain. We rarely actually investigate the functioning of the nervous system we hope to change for the better or how our therapies impact on that system.

Fundamentally, we understand rather little about the neurophysiological states our patients exist in, or can exist in, and we know even less about how those states

relate to functioning. A patient may not be able to move much, but this may be because of very weak muscular activation or because of excessive co-contraction of agonist and antagonist muscles. Two rather distinct neurophysiological states can, therefore, give rise to the same functional outcome, but it is unlikely that the therapeutic approaches designed to improve movement in one state will work equally well in the other. Furthermore, it is possible that our therapeutic interventions will transiently or permanently change the nervous system to perhaps drive it from one pathological state to another. It is for this reason that the timing, dosing, duration, sequencing, and/or combination of therapies, again whether they are surgical, pharmacological, or rehabilitative, will be critical to gaining the greatest clinical benefit. Making these clinical decisions is clearly the “medical art” of neurorehabilitation, and there are those clinicians whose track records with even the most difficult patients are impressive, but in the era of evidence-based medicine, we strive to rigorously study what we practice in the hopes of providing better therapeutic interventions in the future.

Most large clinical studies in spinal cord injury rehabilitation, whether randomized clinical trials or measurements of clinical programs (see Ref.¹), make use of relatively simple scales with few categories and rather large within-category variability. Some of these are behavioral scales that characterize patients’ abilities to interact with their surroundings and manage their

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own care and independence. The Functional Independence Measure (FIM)² and the diagnosis-specific measure, the Spinal Cord Independence Measure (SCIM),^{3,4} describe general patient function but do not sort out which patient ability is due to compensatory strategies and which might be due to neurological recovery. These scales inform us whether our interventions are ultimately useful to our patients but they do not tell us how to improve on them. Clinical exams, such as the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI),⁵ and the modified Ashworth scale for spasticity,⁶ more directly address nervous system function after injury but remain crude tools for revealing underlying neurophysiological states or state changes.

The ISNCSCI examination assesses a few sensory and motor pathways in that it tests voluntary muscle contraction and two modalities of sensory perception. Even so, patients are grouped into only five classification groups (A–E) based on those assessments. As with many such scales, the difference between the “best” patient in one category and the “worst” patient in the next category, the strongest “C” and the weakest “D” for instance, is less than that between patients at the ends of the spectrum within a category. Nevertheless, many clinical trials still only use a classification letter change as a “meaningful” outcome measure and fail to detect improvements of patients within a class.

Even if composite upper- or lower-extremity motor or sensory scores from the ISNCSCI examination are used as measures, there are limits to which patients with different functional capacities can be distinguished. Take, for instance, two patients whose last normal neurological injury level is C6 with motor scores of 18 (out of a possible 30) in the C7, C8, and T1 muscles on both sides combined. One patient could have motor scores of 3 (out of 5) at C7, C8, and T1 on both sides with weakened but still coordinated arm and hand function while the other patient could have a motor score of 0 at C7 bilaterally but a 4 at C8 bilaterally and a 5 at T1 bilaterally (same total score of 18), due, perhaps, to a central cord injury or concomitant C7 spinal nerve root injuries, and have good hand function despite great difficulty with extending the elbows. In this case the same upper-extremity composite motor score would be associated with two very different functional deficits. Of course, there is nothing in these upper-extremity examinations that definitively informs about motor or sensory completeness or incompleteness of the spinal cord injury (class A and B vs. C and D).

The Ashworth examination focuses on characterizing spasticity in the resting state but all clinicians have seen the patient who is relatively flaccid at rest but then develops hypertonia during movements as well as the patient who is hypertonic at rest and then becomes more fluid with voluntary activity. The extent to which spasticity affects motor control and movements may also differ from task to task. To fully appreciate the functional impact of spasticity, it needs to be able to be measured in some way during a variety of motor functions.

Sensorimotor task-specific evaluations, like measuring stepping, can start to separate out components of a behavior like gait and begin to provide insight as to which parts of a behavior are being affected by a given therapeutic intervention. Gait measures include the 6-minute walk (endurance) and the 10 m walk (speed) as well as stride length, stance width, footfall position, etc. which can be measured with devices like the GAITrite system⁷ and can provide continuous values that may be followed over time during an intervention like locomotor training. While these measures may be helpful with detecting therapeutic impact, they still do not necessarily distinguish “nice” gait from “nasty” gait. Two different patients could improve gait speed, one via adaptive neural plasticity and the re-establishment of normal motor control for walking, the other in spite of maladaptive neural plasticity that generates worse pain, spasms, altered kinematics, and inefficient energetics during walking. Therefore, specific functional measures still do not provide sufficient information about nervous system changes that occur either spontaneously after injury or as a result of our rehabilitation interventions.

Perhaps one of the best ways to assess and follow the neurophysiological state of the injured nervous system is to record its functional (motor) output during specific tasks or in response to specific sensory or supraspinal inputs. Comparing the input–output relationships in the injured spinal cord with the input–output relationships in uninjured “normals” allows one the ability to detect, if there are changes in the neurophysiological state of the injured spinal cord, whether those changes move towards or away from the normal state. Mechanically or electrophysiologically measuring spinal reflexes and their modulation, for instance, can be used to determine how normal (or how pathological) spinal neural circuit functioning is after injury or intervention. Soleus H-reflexes have been studied for some time^{8–10} but recently, transcutaneous spinal cord stimulation has been used to activate a collection of lumbar dorsal roots to generate monosynaptic motor reflexes simultaneously in multiple lower-extremity muscles.^{11,12}

Combining these methods with goniometric measurement triggering from locomotor training devices, such as the Lokomat, allows for the study of these reflexes at specific positions in the gait cycle,^{8,10} which reveals more about the dynamic control of spinal neural circuit function during the specific task of walking.

Probably the most useful of neurophysiological assessments of motor control is surface poly-electromyography (EMG). This method can be applied across a population of muscles involved in a sensorimotor task and multiple EMG features, such as pattern, timing, and relative amplitudes of muscle activity, can be assessed across a spectrum of motor tasks or in response to specific sensory or supraspinal inputs to the spinal neural circuitry, even if that circuitry is reduced and/or re-organized after injury. While there are some limitations to comparing across repeated recording sessions due to technical considerations like electrode placement and some difficulty in comparing absolute voltages recorded between individuals, the use of standardized electrode positions, and measuring relative amounts of activities across muscles within an individual can address some of these issues. Of great importance is that any one individual can be recorded from in one session during the performance or attempted performance of multiple movements, or even during the same movements performed at different speeds or while generating different amounts of force, thereby providing a unique “motor control profile” for that individual.

One of the best-reported poly-EMG protocols for assessing motor control in individuals with spinal cord injury is the Brain Motor Control Assessment (BMCA).^{13,14} In the BMCA, multi-muscle EMG is recorded during stereotyped maneuvers and tasks and the pattern and amplitude of that EMG is compared against the pattern and amplitude of EMG for that movement in an idealized “reference subject” who is derived from averaging several non-injured individuals. The BMCA was initially focused on the lumbosacral spinal segments controlling the lower limbs below a spinal cord injury,¹⁴⁻¹⁷ but now trunk and upper-extremity protocols have been put into use as well. The lower-limb BMCA has been validated in motor complete spinal cord injury^{18,19} and the voluntary movement portion of the BMCA protocol was evaluated in motor incomplete spinal cord injury comparing EMG activity in injured individuals to non-injured controls.^{20,21} Sherwood *et al.*,¹⁶ reported the presence of a supraspinal influence over reinforcement maneuvers, vibration responses, and withdrawal reflex suppression in 84% of 88 motor-complete subjects. Recently repeated, these neurophysiological profiles were found

in 64% of an additional 67 subjects with motor-complete spinal cord injury.^{18,19} These patients have been labeled “discomplete” by electrophysiological means despite being characterized as complete injuries by the ISNCSCI clinical examination.

The assessment of voluntary motor function in the BMCA uses an analytic tool that compares the multi-muscle activation patterns recorded from spinal cord injury subjects with prototypes collected from healthy subjects²⁰ for standard motor tasks. This voluntary response index (VRI) has shown validity and reliability as a measure of spinal cord injury severity and treatment effects on volitional control.²² Further, the index was used to differentiate weak voluntary motor function from spasm activation in clinically motor complete subjects.¹⁹ The VRI consists of two components, magnitude and a similarity index (SI), which characterizes the pattern of activity across the sampled muscles. SI values can range from 1 (same pattern of EMG activity as normals) to 0 (pattern of EMG activity not at all similar to normals) to a theoretical-1 (EMG patterns of activity completely opposite, in terms of agonist/antagonist activation, of normal). The relationship between the BMCA-derived similarity index has been shown to correlate with the lower extremity motor score from the ISNCSCI examination²¹ and, in a small sample of individuals, gait speed.²² The BMCA has also been used to qualitatively group neurologically impaired individuals into functional classes, relating the patterns of muscle activity seen during testing with the assistive devices they use for ambulation.²³ Most recently, the BMCA has been used to characterize the natural recovery of infra-injury motor control in the acute stages following spinal cord injury²⁴ and is under study over the time period of inpatient rehabilitation as a site specific study of the Spinal Cord Injury Model Systems research program at Shepherd Center. In summary, the BMCA provides a quantitative assessment of motor control below and across the level of spinal cord injury and can be used to relate that motor control to functional capacity and the evolution of motor recovery with treatment.

With the neurophysiological measures of poly-EMG and reflex testing, we finally have assessment tools to directly evaluate the residual, re-organized nervous system we hope to alter with our therapeutic interventions to ultimately generate functional recovery. We can use these measures to follow-up patients over time and determine if the neurophysiological states we are targeting with our therapeutic interventions are changing and if they are, in fact, becoming more normal. The BMCA may at first show a low similarity index

with high amplitude activity for a given task indicating abnormal patterns of activation (with co-activations or dysnergias) but later show an improved similarity index with less overall activity indicative of more normal motor control. Alternatively, the BMCA might initially show a high similarity index but low EMG amplitudes for a given task and later greater amplitudes of activity demonstrating strengthening of what was originally an appropriate motor control but hampered by low activity resulting in weakness. We might even use the BMCA to detect when an intervention designed to increase motor output might start causing the development of inappropriate patterns of activation (a lowered similarity index) at which time the therapeutic intervention should be altered to avoid replacing weakness with spastic dysfunction.

It is this idea that neurophysiological assessment could be used to track and guide therapeutic intervention that will permit the individualization of care for those with spinal cord injury. No two patients have the same injury or resultant neurophysiological state(s) and they will, therefore, likely respond to therapeutic interventions in different ways. While this individualized approach to treatment flies in the face of traditional clinical research design, it may be no less evidence driven, albeit on a case-to-case basis. With retrospective analysis of this approach, more generalized “rules” for rehabilitation may become obvious for different groups of patients or it might become clear that how therapeutic interventions are dosed, timed, or combined may lead to different paths of neurological recovery in different groups of patients, perhaps as defined by common motor control profiles from the BMCA.

It is also possible that these neurophysiological assessments could be used to make therapeutic decisions in real time. For instance, locomotor training parameters include variables like treadmill speed, body weight support, and, in the case of robotic therapy, specific mechanical controls like joint angles. Knowing how to change these variables over the course of locomotor training has been based on therapists’ experience and is not evidence-based, at least not in terms of determining what combination of training variable values generates the maximum training effect in the least time in any one individual with their own specific motor control profile. Imagine that one could test the lower extremity EMG patterns during stepping in the same way as they are evaluated during the BMCA and that a similarity index for the gait cycle could be determined. Therapists could then devote a locomotor training session instead to an electromyographic testing session where they could determine what combination or

combinations of treadmill speed, body weight support and joint angles generates the most normal EMG pattern for stepping in the individual patient in front of them. We could then hypothesize that training parameters chosen based on what generates the most normal motor control for stepping would also generate the greatest training effect. This may not turn out to be the case, but at least we would have worked to develop evidence-based criteria for how to or how not to progress locomotor training to change the nervous system and generate the best neurological recovery.

Periodically repeating this process over the course of locomotor training might also be wise. We might find that changing one training variable in one way generates the best motor control at one time point in locomotor training, but that at a subsequent evaluation, it is now another parameter that should be changed to again improve the motor control for stepping. These new approaches will take time to develop and test but they are the way in which we will be able to begin to link our interventions to the nervous system we are dealing with in spinal cord injury and the goals we have for our patients’ functional recovery.

Acknowledgement

This article is based on the author’s keynote address at the 5th National Spinal Cord Injury Conference in Toronto, Ontario, Canada in October 2012.

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