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SPECIAL COMMUNICATION

Upper Extremity Assessment in Tetraplegia: The Importance of Differentiating Between Upper and Lower Motor Neuron Paralysis

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Abstract

Scientific advances are increasing the options for improved upper limb function in people with cervical level spinal cord injury (SCI). Some of these interventions rely on identifying an aspect of paralysis that is not uniformly assessed in SCI: the integrity of the lower motor neuron (LMN). SCI can damage both the upper motor neuron and LMN causing muscle paralysis. Differentiation between these causes of paralysis is not typically believed to be important during SCI rehabilitation because, regardless of the cause, the muscles are no longer under voluntary control by the patient. Emerging treatments designed to restore upper extremity function (eg, rescue microsurgical nerve transfers, motor learning-based interventions, functional electrical stimulation) all require knowledge of LMN status. The LMN is easily evaluated using surface electrical stimulation and does not add significant time to the standard clinical assessment of SCI. This noninvasive evaluation yields information that contributes to the development of a lifetime upper extremity care plan for maximizing function and quality of life. Given the relative simplicity of this assessment and the far-reaching implications for treatment and function, we propose that this assessment should be adopted as standard practice for acute cervical SCI.

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Cervical spinal cord injury (SCI) imposes significant limitations in upper and lower extremity function and other body system functions. People with cervical level SCI desire improved use of their arms and hands,[1] believing that regained function will result in improved quality of life.[2] Scientific advances are increasing the options for improved upper limb function in people with tetraplegia. Some of these interventions rely on identifying an aspect of paralysis that is not uniformly assessed in SCI: the integrity of the lower motor neuron (LMN). The purpose of this article is to review the rationale and clinical procedure for differentiating upper motor neuron (UMN) and LMN paralysis in the upper extremities of people with cervical SCI. Emerging treatment and interventions for which this information is paramount will be reviewed, including a discussion of how such knowledge affects the decision-making process concerning upper extremity (UE) intervention. Differentiating between UMN and LMN damage in people with tetraplegia is an important step in identifying options for improved UE function. This noninvasive evaluation yields important information to develop a lifetime UE care plan for maximizing function and quality of life and should be a standard of care at all SCI rehabilitation programs.

Complexity of paralysis after SCI

SCI is commonly considered a condition of the central nervous system because of damage to the UMN. However, concurrent LMN damage can also result from direct trauma to the ventral horn cell, nerve root, or axon on exit from the intervertebral foramen. Damage to the UMN and LMN both result in muscle paralysis. The differentiation between these mechanisms of paralysis is not typically addressed during SCI rehabilitation because, regardless of the cause, the muscles are no longer under

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voluntary control by the patient. After the period of spinal shock after acute SCI, certain clinical characteristics of paralyzed muscles can indicate whether there is damage to the LMN (eg, hyporeflexia, hypotonia, muscle atrophy), with the absence of spasticity. Selective damage to the UMN leaves the LMN intact and is characterized by hyperreflexia, hypertonia, and spasticity in the affected muscles. Aside from clinical observation and examination, formal evaluation of the LMN via electrodiagnostic testing is not standardly performed in this population. However, early acute electrodiagnostic studies can help to identify incomplete injuries by assessing the presence of residual motor-evoked potentials[3] and the location of peripheral nerve injury by demonstrating conduction block across a damaged nerve segment.[4-6]

Clinical classification of SCI has largely been dependent on the measurement of voluntary muscle characteristics. To better understand the importance of differentiating UMN and LMN paralysis in the UE, it is important to review current SCI classifications. There are 2 accepted classification schemes for SCI. The most commonly used classification, the International Standards for the Neurological Classification of Spinal Cord Injury (ISNCSCI), was developed by the American Spinal Injury Association and International Spinal Cord Society and has been well described in the literature.[7] A more specific UE classification, the International Classification for Surgery of the Hand in Tetraplegia (ICSHT),[8,9] is used to identify candidates for UE surgical restoration, specifically through tendon transfers. This classification identifies the voluntary muscles available for surgical tendon transfer to replace another function lost from paralysis (table 1). The ISNCSCI and ICSHT differ in their strength rating criteria, requiring grades 3 and 4 muscle strength, respectively.

The ISNCSCI and ICSHT classifications provide a useful index of motor functioning in the UEs of individuals with tetraplegia. However, they fall short in their prognostic abilities for potential recovery and for guiding intervention because there is no provision for differentiating LMN from UMN paralysis. Muscles affected by LMN damage respond weakly or not at all to electrical stimulation of the motor point using surface electrodes. Therefore, electrical stimulation can be used diagnostically to distinguish between paralyzed muscles with intact versus damaged LMNs.[10-13] Figure 1A depicts a 3-level hierarchy of innervation after SCI: above the lesion, at the lesion, and below the lesion. Muscles above the spinal cord lesion are unaffected by UMN or LMN damage and are of normative strength. Muscles at the lesion share characteristics of UMN and LMN damage, and those below the lesion experience UMN paralysis but with intact LMNs. Coulet et al[13] define the lesion, or injured metamere, as variable in size because the severity of the UMN and accompanying LMN damage is variable.

The clinical picture of paralysis is often more complex than this 3-level hierarchy reflects. Figure 1B shows overlapping innervation patterns throughout the transition from normative to paralyzed muscle function. The addition of 2 mixed innervation zones helps clarify the clinical picture. Zone 1 is supralesional and represents normative neuromuscular function. This zone is also fully responsive to electrical stimulation; however, stimulated response is rarely tested in these muscles. Zone 2 represents a border zone of injury with a mixture of normative neuromuscular function combined with variable LMN or UMN damage. This zone is characterized by weak voluntary muscle contraction and muscles that are weakly responsive to electrical stimulation. Zone 3, as in figure 1A, represents the primary lesion site where UMN damage is likely accompanied by a significant degree of LMN damage. This zone is characterized by absent muscle contraction with muscles that are not responsive to electrical stimulation. Zone 4 represents another border zone of injury with a combination of UMN and LMN damage. This zone is characterized by absent muscle contraction with muscles that are weakly responsive to electrical stimulation. Zone 5 is infralesional and consists of intact LMNs, but they are not under voluntary control because of UMN damage. These muscles, although fully paralyzed, are fully responsive to electrical stimulation. The significance of these classification zones lies in their application in guiding appropriate interventions for improving UE function. Eligibility for certain interventions is zone dependent; therefore, early identification of LMN damage is critical. Further development of a supplemental classification, including the differentiation between UMN and LMN damage, would be a valuable complement to existing SCI classifications.

Table 1 SCI classifications

ISNCSCI	Key Movement (Muscle) Criteria: Grade 3	ICSHT Group	Key Muscle Criteria: Grade 4
C4 and higher		N/A	
C5	Elbow flexion (biceps, brachialis, brachioradialis)	0	Biceps
		1	Brachioradialis
C6	Wrist extension (ECRL, ECRB)	1	Brachioradialis
		2	ECRL
		3	ECRB
		4	PT
C7	Elbow extension (triceps)	4	PT
		5	FCR
		6	EDC
		7	EPL
C8	Finger flexion (FDS, FDP)	8	FDS
		9	FDS and FDP

Abbreviations: ECRB, extensor carpi radialis brevis; ECRL, extensor carpi radialis longus; EDC, extensor digitorum communis; EPL, extensor pollicis longus; FCR, flexor carpi radialis; FDP, flexor digitorum profundus; FDS, flexor digitorum superficialis; N/A, not applicable; PT, pronoator teres.

List of abbreviations:

ICSHT	**International Classification for Surgery of the Hand in Tetraplegia**
ISNCSCI	**International Standards for the Neurological Classification of Spinal Cord Injury**
LMN	**lower motor neuron**
SCI	**spinal cord injury**
UE	**upper extremity**
UMN	**upper motor neuron**

Early identification of LMN damage

There is evidence to suggest that LMN damage can be identified early after acute SCI and that it is permanent. Initial concerns regarding early identification of LMN damage were related to the timing of testing and the effect of spinal shock. How early after injury can electrical stimulation accurately identify LMN damage? Anterior horn cell body damage is evident in the peripheral