



Scientific Review

Electrophysiological recordings in patients with spinal cord injury: significance for predicting outcome

A Curt*¹ and V Dietz¹

¹Swiss Paraplegic Centre, University Hospital Balgrist, Zürich, Switzerland

The clinical assessment of the level, extent and severity of spinal cord injury (SCI) can be supplemented by electrophysiological recordings. These techniques also provide an early diagnosis of neurological deficits in patients with acute SCI and are of prognostic value even in uncooperative patients. Electrophysiological recordings (motor evoked potentials (MEP) and somato-sensory evoked potentials (SSEP)) are of similar significance in predicting functional outcome of ambulatory capacity, hand- and bladder function as the clinical examination according to the ASIA standards. EMG, neurographic and reflex recordings of acute SCI patients within spinal shock are even more sensitive in assessing an associated damage of the peripheral motor pathways (ie of motoneurons and nerve roots) than the clinical examination and allow the possibility of predicting the development of muscle tone or muscle atrophy. The evaluation of impairment of the autonomic nervous system after SCI by clinical examination is restricted. In contrast, recordings of the sympathetic skin response (SSR) can provide information about the extent and level of lesions of the spinal sympathetic nervous system which are related to autonomic dysfunction. Therefore, electrophysiological recordings supplementary to the clinical examination are helpful for planning and selecting the appropriate therapeutical approaches within the rehabilitation programme. Furthermore, they allow the prediction of functional outcome and the objective assessment of recovery of specific parts of the spinal and peripheral fibre tracts.

Keywords: spinal cord injury; clinical examination; electrophysiological recordings; functional outcome

Introduction

In patients with acute traumatic lesions of the spine the clinical examination is usually the first and most important diagnostic approach for the assessment of a spinal cord injury (SCI). The clinical examination delineates the level of the lesion, the extent of motor and sensory deficits, and initiates (neuro-) radiological examinations (X-ray, CT scan, MRI). The results of such examinations are essential in the choice of the best therapeutical approach (ie surgical and conservative procedures) and for planning the aims and determine the limitations of the rehabilitation programme.

This review intends to show how far electrophysiological recordings are able to broaden the clinical assessment of an acute SCI patient and its consequences. As electrophysiological recordings allow the objective assessment and distinguishing between lesions of different parts of the peripheral and central

nervous systems they are of supplementary value to the clinical examination and especially of importance in patients who are not able to cooperate.¹

The aim of this review is: (1) to illustrate the significance of electrophysiological recordings in the diagnostic and prognostic assessment of patients with acute SCI in relation to the clinical examination, (2) to describe the most reliable recordings for predicting the outcome of different functions in SCI patients, and (3) to monitor the extent of recovery of spinal cord function (by recording spinal impulse transmission) in relation to the degree of functional improvement during the rehabilitation programme. The indications and limitations of electrophysiological recordings in the diagnostic assessment of acute SCI patients and their value in comparison to the clinical examination are discussed.

Clinical examination

For an adequate assessment of the relevance of electrophysiological recordings in acute SCI patients

*Correspondence: A Curt, MD, University Hospital Balgrist, Forchstrasse 340, CH-8008 Zürich, Switzerland

the significance of some clinical examinations will be shortly reviewed. The clinical diagnosis of incomplete motor and/or sensory SCI lesion, such as sacral sparing, in patients with acute SCI provides a good prognosis for recovery of spinal cord function.^{2,3} Furthermore, the clinical diagnosis of specific post-traumatic spinal syndromes allows the estimation of the degree of neurological recovery in acute SCI.⁴ Best recovery is observed in the Brown-Séquard (90%), less in patients with central-cord (50%) and least in patients with anterior cord-syndrome (16%).^{5,6} However, these, rather gross, clinical differentiations are not sensitive enough to predict the recovery of special functions like locomotion, hand- or bladder function, which are of crucial importance for the functional outcome and future independence of patients with SCI.

One of the first standardized clinical protocols for the diagnosis of acute SCI was introduced by Frankel and coworkers.⁷ This internationally well recognized gradation, in combination with the level of lesion, describes the extent of neurological and functional deficits. Follow-up examinations revealed that no tetraplegic patient with a Frankel grade A lasting up to 72 h post-trauma and only about half of tetraplegic patients with a Frankel grade B achieved an ambulatory capacity 1 year after trauma.⁸ However, 87% of tetraplegic patients with a Frankel grade C were able to ambulate 1 year after trauma. Furthermore, the different prognostic value of light touch or pin prick sensation in incomplete SCI below the level of lesion could be documented. In about 70% of patients with acute SCI and loss of muscle function, but preserved pin prick sensation below the level of lesion, a functional relevant recovery of muscle strength occurred. However, such a recovery was seen in less than 20% of patients with preserved light touch sensation but lacking pin prick sensation in the respective dermatomes.^{2,9}

In 1992 the American Spinal Injury Association (ASIA) in cooperation with the International Medical Society of Paraplegia (IMSOP) introduced a standardized clinical examination protocol for SCI patients which aims to quantify the extent of motor and sensory deficit.¹⁰ The ASIA protocol has been recognized as a fast, simple and semiquantitative protocol for neurological examination which focuses on the special functions of SCI patients. The ASIA protocol determines the level of lesion and scores the preserved motor and sensory functions, differentiates between complete or incomplete (presence of any sensory or motor function in lowest sacral segment) lesion and describes a zone of partial preservation. Follow-up examinations using the ASIA protocol in tetraplegic patients have shown that more than 80% of functionally relevant neurological recovery is achieved 3–6 months post-trauma with a concomitant recovery of upper and lower limb function.¹¹ For later periods, up to 2 years post-trauma, only a minor neurological recovery can be expected without additional relevant motor functions. In 90% of

patients with complete tetraplegia more than 1 month post-trauma, no recovery of any new motor or sensory function does occur. In less than 10% of patients there was a decrease of the neurological level of lesion. By testing upper and lower limb motor scores according to the ASIA protocol in acute SCI patients it is possible to predict recovery of muscle strength and functional outcome, such as the ambulatory capacity.¹²

Electrophysiological recordings

Electrophysiological recordings have been used in the management and care of SCI patients since 1970 but are routinely performed only in a few SCI centres. These techniques supplement clinical and neuroradiological examinations and allow the differentiation between lesions of the spinal (ie ascending and descending fibre tracts) and the peripheral nervous system (eg radicular lesions, plexus, peripheral nerves). They are especially useful compared to clinical examination in uncooperative (due to drugs, language barrier, psychogenic paresis) and unconscious (due to head trauma, artificial ventilation) patients, as the electrophysiological recordings are less dependent upon the cooperation of the patient.¹³ The electrophysiological recordings can be separated into those relevant to central (spinal) or to the peripheral nervous system.

Recordings of spinal pathways

Somato-sensory evoked potentials (SSEP)

Using SSEP recordings the integrity of impulse transmission of somato-sensory nerve fibres through parts of the spinal (mainly dorsal column) and peripheral (peripheral nerve, plexus) nervous system can be tested. Spinal lesions at different levels can be separated from affection of sensory nerve fibres by combined recordings of the SSEP from central (conus medullaris, cervical spine, cortex) and peripheral (plexus) parts of the nervous structures. The recordings are not affected by spinal shock and can reliably be recorded even in sedated and unconscious patients.¹⁴

Motor evoked potential (MEP)

Following the introduction of painless transcortical magnetic stimulation by Barker and coworkers¹⁵ the integrity of the cortical and spinal motor tract fibres can be assessed even in awake SCI patients.¹⁵ MEP due to transcortical stimulation can be recorded from different proximal and distal muscles of the upper and lower limbs, and can be used to assess the level and extent of the SCI lesion. By combining magnetic stimulation of cortical and peripheral nervous structures (spinal roots, plexus, peripheral nerves), lesions of spinal and/or peripheral nerves underlying a muscle paresis can be differentiated.^{16,17}

Sympathetic skin response (SSR)

The sympathetic skin response (SSR) is a simple and non-invasive electrophysiological test to examine the common efferent pathways of the sympathetic nervous system. Pathways from the spinal cord to the sudomotor sweat glands of hands (palmar), feet (plantar) and the perineal skin region transmitted by pre- and post-ganglionic sympathetic nerve fibres can be evaluated. The SSR to supraspinal magnetic or electric stimulation can be recorded by conventional surface EMG disc electrodes applied to the relevant skin areas. This allows the assessment of lesions of the spinal and peripheral sympathetic nerve fibres subserving respective skin areas.¹⁸

Recordings of peripheral pathways

Electromyographic (EMG) and neurographic recordings

In SCI patients EMG and neurographic recordings from upper and lower limb muscles are required in order to assess accompanying peripheral nerve lesions in poly-traumatic SCI patients. In addition, damage of anterior horn cells and ventral nerve roots associated with a SCI (including conus medullaris or motor fibres of the cauda equina) can be evaluated.

The combination of motor and sensory neurographic recordings allows the differentiation between muscle paresis due to spinal anterior horn cell/ anterior nerve root lesions or peripheral nerve damage (plexus, peripheral nerve).¹⁹ In the latter disorder, both the peripheral sensory and motor nerve fibres are affected, whereas in spinal lesions only the motor nerve fibres are affected while sensory nerve fibres remain intact.²⁰

Reflex-recordings

By studying H-reflexes and F-waves it is suggested that the impairment of motoneurone excitability due to traumatic lesion, spinal shock and development of spasticity can be assessed.^{21,22}

The H-reflex (first description by Hoffman 1918) is an electrically induced monosynaptic reflex (corresponding to the tendon tap reflex), which includes the function of afferent, spinal-segmental and efferent pathways. The impulse volley evoked by submaximal electrical stimulation of afferent fibres (Ia) of a mixed peripheral nerve excites the α -motoneurons belonging to the same muscle where the stimulated afferent fibres originate by monosynaptic transmission. In contrast, F-waves represent late motor responses observed following supramaximal electrical stimulation of a peripheral nerve causing an antidromic activation of α -motoneurons. Therefore F-waves indicate preserved conduction along the efferent peripheral motor pathway and are of diagnostic value in proximal nerve lesions and are related to the excitability of the segmental motoneurone pool.²³

Prediction of functional outcome

Hand function

Patients with cervical SCI (30–40% of SCI patients) suffer from functional deficits of both upper and lower limbs. Impairment or loss of hand function needed for grasping movements and skilled motor tasks are among the most important factors restricting the future independence of these patients.²⁴ Therefore, an early prediction of the outcome of hand function in such patients is of crucial importance in planning the rehabilitation programme and thus the degree of self-independence to be expected. For studying functional outcome of hand function a simplified classification²⁵ into one of three most relevant types can be performed: (a) active hand function = voluntary activity of the intrinsic hand muscles (at least pulp- and lateral pinch), (b) passive hand function = loss of voluntary activity of intrinsic hand muscles. By using the tenodesis effect a passive closing and opening of the fingers is achieved and some gross grasping movements can be performed, and (c) no hand function = complete loss of intrinsic hand- and forearm muscle function without any active grasping function of the hand.

SSEP recordings by stimulation of upper limb nerves allows the assessment of the level of cervical SCI. In SCI patients with level of lesion C3–C6 the median SSEP was pathological in about half (52%), and ulnar SSEP in most (81%) of patients.²⁵ In lesions C6–T1 the median SSEP was less affected (<20%) whereas the ulnar SSEP was as pathological in more than 85% of patients. In the latter study, all patients with a loss of both median and ulnar SSEP achieved no hand function. Patients who recovered a passive hand function showed a pathological median SSEP in about half and pathological ulnar SSEP in more than 90% of cases. In contrast, patients with normal median and ulnar SSEP developed an active hand function in more than 90% of cases. SSEP recordings were of similar sensitivity ($P < 0.001$) as the ASIA scores of the sensory functions in predicting the outcome of hand function.

MEP recordings may also predict development of hand function of tetraplegic patients following acute cervical SCI.²⁶ In cervical SCI the MEP recordings of the abductor digiti minimi muscle were pathologically affected in most patients (about 90%) and significantly different from normal values (ANOVA, $P < 0.05$), whereas the MEP recordings of the biceps brachii muscle were less affected (in only about 20%). The MEP recordings of the abductor digiti minimi muscle were significantly ($P < 0.001$) correlated to the outcome of hand function, as this muscle is representative of voluntary activity of intrinsic hand muscles. Most patients (>90%) who lack MEP of both biceps brachii and abductor digiti minimi muscle following acute SCI developed no or only a passive hand function. Where abductor digiti minimi muscle MEP was absent no patient regained an active hand function. Furthermore, recordings of MEP from

proximal upper limb muscles (ie biceps and triceps muscle) predicted pushing and transfer abilities of the patients (active *vs* electrical wheelchair), which are most important for the activities of daily living and self-independence.

Neurographic recordings of the median and ulnar nerves in acute tetraplegic patients are also of relevance in predicting hand function and to differentiate between the development of a flaccid or spastic hand muscle paresis.²⁷ In cervical lesions (C6–T1) anterior horn cells and anterior nerve roots can be damaged resulting in a flaccid paresis of the intrinsic hand muscles. The combined neurographic examination of sensory and motor nerve fibres distinguishes between peripheral (plexus, peripheral nerve) and spinal cord lesions and enables estimation of the extent (complete or incomplete) of anterior horn cell damage by the reduction of the compound muscle action potential. By the examination of the median and ulnar nerves it can be determined whether one or both nerves are affected (representing intrinsic hand muscle function) and whether both hands are similarly affected. In a prospective study no tetraplegic patient with complete loss of the compound action potential of median and ulnar nerves regained an active hand function; all of such patients developed a flaccid paresis of the intrinsic hand muscles.²⁸ This is of crucial relevance for the development of hand function and for the decision at an early stage of the mode of occupational therapy (eg splinting procedures, gloves). Furthermore, the application of functional electrical stimulation (FES) to improve hand function or the indication for a muscle-tendon transfer operation is influenced by the outcome of hand function and the muscle tone which can be expected.²⁹

Ambulatory capacity

The tibial SSEP can be used to predict recovery of lower limb function and is related to outcome of ambulatory capacity. Earlier studies showed that a loss of tibial SSEP in patients with acute SCI indicates a poor recovery.³⁰ In contrast, in most patients (>80%) with an initially elicitable tibial SSEP post-trauma some ambulatory capacity recovered within 1 year post-trauma. In addition, these patients showed a significantly higher initial motor score and recovery of muscle function in follow-up examinations.³¹ For studying the prognostic value of electrophysiological recordings a gradation of the functional outcome of ambulatory capacity into four functionally relevant categories was applied.^{32,33} (a) full ambulatory capacity=no restriction in the activities of daily living and employment due to lower limb deficit; (b) functional ambulatory capacity=walking daily over a distance of more than 500 m without personal assistance. The use of one or two sticks and/or braces is allowed. The ambulatory capacity is regularly used in the activities of daily living and usable for employment; (c) therapeutical ambulatory capa-

city=standing and walking is only possible by aid of either a physiotherapist, two braces in parallel bars or with two sticks and the assistance of an accompanying person (the patient can perform these procedures only as a therapeutical approach); (d) no ambulatory capacity=not able to walk or stand. The patient may have some voluntary muscle activity in the lower limbs.

A recent study³³ demonstrated that no acute tetraplegic patient with a loss of *tibial SSEP* up to 2 weeks post-trauma recovers full ambulatory capacity. Only 20% of these patients achieved a functional or therapeutical ambulatory capacity, while the remaining 80% were still non-ambulatory 6 months post-trauma (Table 1). Most patients (70%) with elicitable but pathological tibial SSEP (prolongation of SSEP latency) developed a functional or therapeutical ambulatory capacity, although some patients (10%) regained a full ambulatory capacity. Most patients (>80%) with normal tibial SSEP latency developed a full ambulatory capacity.

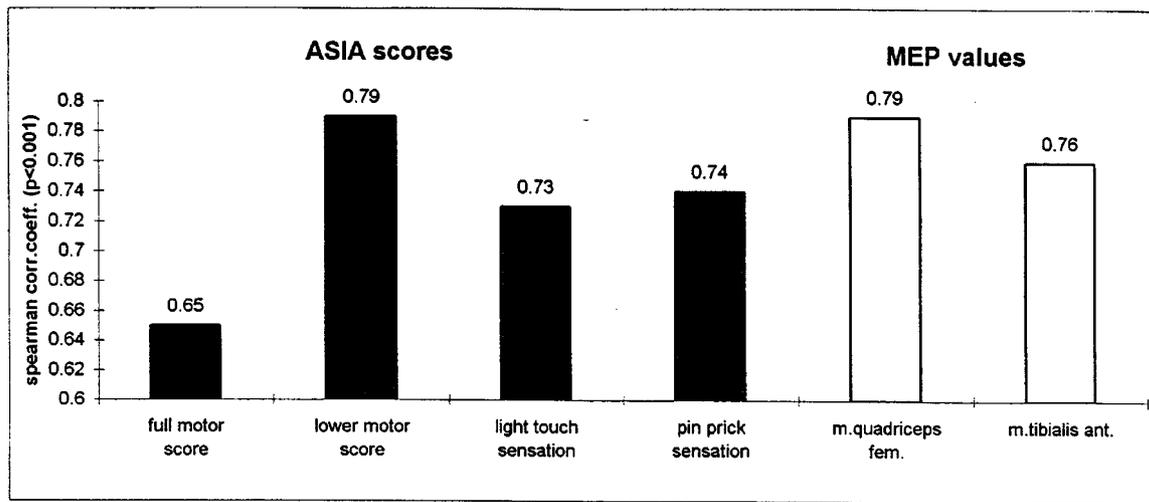
In patients with SCI and complete loss of lower limb muscle function a loss of *MEP* in the leg muscles was usually found.³⁴ In approximately 80% of patients with incomplete motor lesions a *MEP* could be recorded from the anterior tibial muscle. The latter patients had a significant slowing of spinal conduction velocity (a means of 32 m/s compared to a normal value of about 60 m/s) and the *MEP* amplitudes were reduced.³⁵ In most of the patients (70%) with acute SCI who subsequently recovered an ambulatory capacity post-trauma a *MEP* from the anterior tibial muscle was recorded. Most patients (about 80%) who achieved a full ambulatory capacity had normal *MEP* latencies of the anterior tibial and quadriceps femoris muscles. Only a few (<20%) acute SCI patients who suffered an initial loss of lower limb *MEP* finally achieved a full or functional ambulatory capacity.²⁶ The *MEP* values of lower limbs were similarly sensitive in predicting recovery of ambulatory capacity as the *ASIA* motor scores of the lower limbs (Table 2). Patients with an elicitable *MEP* in the lower limb within the first 4 days post-trauma showed the best recovery of motor function and achieved at least a functional ambulatory capacity.³⁶

Neurographic recordings of tibial and peroneal nerves can indicate lesions of the conus medullaris

Table 1 Relationship between initial tibial SSEP recordings and outcome of ambulatory capacity in acute tetraplegic patients (n=31)

Initial tibial SSEP result	Outcome of ambulatory capacity after 6 months			
	Full	Functional	Therapeutical	No
Normal latency	83%	17%	0%	0%
Pathological latency	10%	60%	10%	20%
Loss of SSEP	0%	7%	13%	80%

Table 2 Relationship between ASIA scores, lower limb MEP values and ambulatory capacity in acute tetraplegic patients ($n = 36$)



and the cauda equina. These results can be used to predict the development of a spastic or flaccid leg muscle paresis, which is of great influence for the particular rehabilitation programme (eg functional electrical stimulation (FES), development of spastic muscle contractions, locomotor training on a treadmill). In addition, patients with severe motor nerve fibre lesions tend to have a less favourable recovery rate of lower limb muscle function. In follow-up examinations any secondary development of motor nerve lesion due to spinal (myelomalacia, post-traumatic syringomyelia) or peripheral nerve damage (nerve entrapments) can be assessed at an early stage and enables the initiation of further diagnostic (neuroradiology) and therapeutical interventions.^{27,37}

Bladder function

Impaired bladder function is a major complication in patients with complete and incomplete SCI. An early diagnostic assessment and appropriate treatment of neurogenic bladder dysfunction is mandatory in order to improve life quality and expectancy of SCI patients.³⁸ The pudendal SSEP does not represent autonomic nerve fibres involved in bladder function.³⁹ The SSEP due to pudendal nerve stimulation includes somatic nerve fibres from S2–S4, and is related to somatic nerve function (external urethral sphincter, EUS). While the vesical detrusor muscle is innervated by parasympathetic nerve fibres within the pelvic nerve.⁴⁰

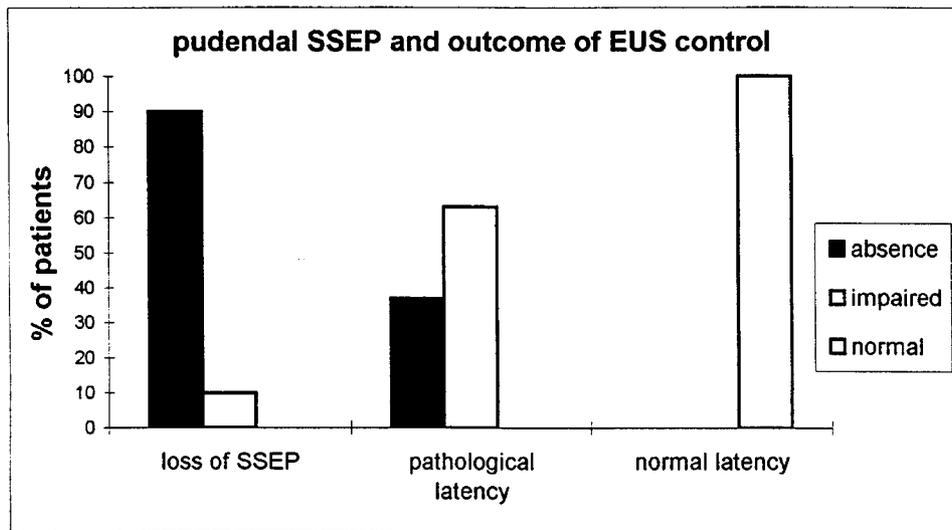
Elicitable *pudendal SSEP* are indicative of a good recovery of bladder function and are significantly related to the function of the external urethral muscle. In a prospective study⁴¹ most of the paraplegic (90%) and tetraplegic (about 70%) patients with acute SCI

and loss of pudendal SSEP showed a complete loss of voluntary EUS function 6 months after trauma (Table 3).³⁵ No patient of the latter groups achieved a normal EUS function. The pudendal SSEP recordings proved to be of similar value as the ASIA scores at predicting outcome of bladder function. However, it is important to note that both pudendal SSEP and ASIA scores cannot indicate or predict the kind of detrusor versicae dysfunction (eg upper *versus* lower motoneurone lesion, hyper-/hyporeflexive bladder) which has to be assessed by urodynamic examinations.³⁸

The *bulbocavernosus reflex* is a sacral polysynaptic reflex which can be elicited during spinal shock even though tendon reflexes of the lower limbs are abolished. Therefore the loss of the bulbocavernosus reflex during spinal shock in acute SCI patients suggests an impairment of the conus medullaris or cauda equina.⁴² Clinical and electrophysiological examinations of the bulbocavernosus reflex give indirect evidence about the function of the sacral somatic nerves S2–S4 which are involved in bladder function. An abolished bulbocavernosus reflex is usually associated with the development of bladder dysfunction due to a lower motoneurone lesion. However, like clinical and SSEP examinations, urodynamic characteristics of the detrusor muscle (urine retention, renal reflux) cannot be predicted by this reflex.⁴³ In addition, the examination of the bulbocavernosus reflex by needle EMG recordings assesses acute muscle denervation and, in follow-up examinations, signs of reinnervation.

The *anal reflex* as a typical sacral reflex is also elicitable during spinal shock. Again, as with the bulbocavernosus reflex the anal reflex is impaired by lesions of the conus medullaris, cauda equina and plexus sacralis.⁴³ Although the external anal

Table 3 Relationship between pudendal SSEP in acute paraplegic patients ($n = 39$) and outcome of external urethral sphincter function



sphincter muscle is innervated by the S2–S4 roots the activation of the external urethral sphincter muscle is different to that of the external urethral sphincter muscle.^{44,45} Therefore, EMG recordings of the anal sphincter muscle during urodynamic examinations are of limited value in assessing the behaviour and function of the external urethral sphincter muscle.⁴⁶

Autonomic dysfunction

In SCI descending tracts of the sympathetic nervous system within the spinal cord can be damaged which leads to a complete or incomplete disconnection of the supraspinal sympathetic centres from the peripheral effector organs (Figure 1).^{18,47} Complete tetraplegic patients develop different syndromes of sympathetic failure with orthostatic hypotension, disturbance of the circadian blood pressure regulation (diminished modulation of blood pressure but preserved heart frequency modulation during the night periods due to intact vagal innervation) and can suffer from autonomic hyperreflexia (AH).⁴⁸ In tetraplegic patients the loss of SSR in the hands was associated with the development of AH in 93% of patients. Disorders of the sympathetic nervous system were often missed clinically as 62% of patients with AH did not complain of any symptoms although severe blood pressure dysregulation was monitored (24 h blood pressure monitoring). In 27% of incomplete tetraplegic patients the clinical examination was not sufficient to assess a complete disconnection of the sympathetic nervous system, although these patients suffered from sympathetic failure.⁴⁹

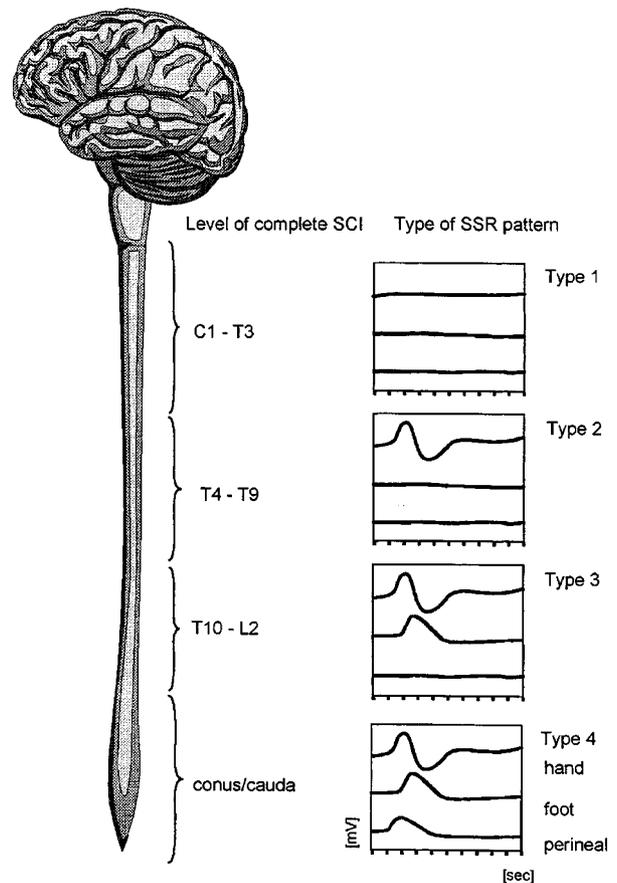


Figure 1 Loss of SSR in hand, feet and perineal skin area depending on the level of complete spinal cord lesion (type 1–3); preservation in lesions of the conus/cauda (type 4)

Development of muscle tone

In acute SCI tendon reflexes are abolished and the muscle tone is reduced below the level of lesion. This combination of symptoms in acute SCI is called 'spinal shock'. However, for this diagnosis any accompanying peripheral nerve lesions, which could be responsible for the abolished tendon reflexes and reduced muscle tone have to be excluded.⁵⁰

In contrast *H-reflexes* can be elicited during spinal shock, at a time when tendon reflexes of the lower limb are still lost.⁵¹ This confirms that the reflex arc is preserved and no accompanying peripheral nerve lesions below the level of lesion are present. These patients are prone to develop increased muscle tone and signs of spasticity.⁵²

By *F-wave recordings* the integrity of the efferent motor pathways can be assessed but they are influenced by spinal shock.⁵³ In about half of patients with acute tetraplegia during spinal shock F-waves of the median and ulnar nerves could not be recorded.⁵⁴ At 3 months post-trauma in most (80%) and 6 months post-trauma in all patients F-waves could be recorded. This recovery correlated with resolving spinal shock. While the F-wave latencies were not affected in spinal lesions, the F-wave persistence (number of elicitable F-waves to 20 repetitive stimulations) was found to be related to spinal shock.

Spinal cord recovery

By the ASIA protocol and the clinical scales of outcome (ambulatory capacity, transfer capabilities, bladder function) the functional recovery of patients after acute SCI can be assessed. However these scales are less sensitive in evaluating the extent of spinal cord recovery (ie improved spinal impulse transmission), as the functional and motor outcome can be improved due to alternative functional strategies and adaptations of the peripheral and spinal motor systems below the spinal cord damage. Especially in incomplete SCI patients the ASIA sensory and motor scores show an increment, which is related to the improvement of the patients' condition. In studies using SSEP and MEP recordings it could be shown that functional recovery is not in parallel with recovery of spinal impulse transmission.⁵⁵ Tibial SSEP in incomplete SCI patients at an acute, post acute (6 months after trauma) and chronic (>6 months after trauma) stage showed no improvement of pathological SSEP, although the patients showed a significant functional improvement. The same was true for MEP recordings: the prolonged latencies did not recover, while the amplitudes showed some increment (Figure 2). Therefore, only by the combination of clinical and electrophysiological examinations can functional and spinal cord recovery be distinguished. Both human and animal studies show that there is a very restricted recovery of impulse transmission within the spinal cord, which can be compensated by plastic processes within the spinal cord below the level of lesion and by

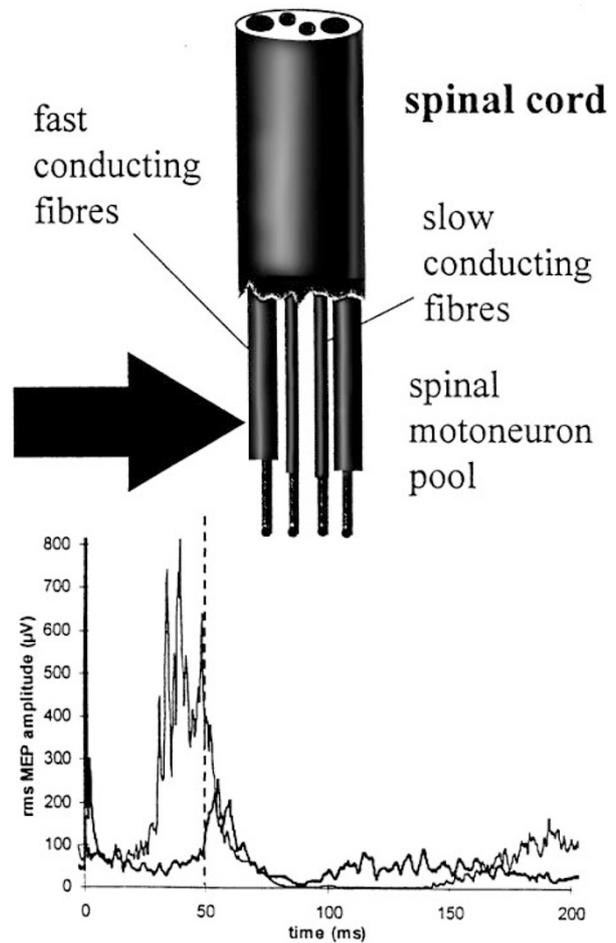


Figure 2 MEP recordings (normal value=thin line) in incomplete SCI (bold line) show a primary loss of fast conducting tract fibres with prolongation of MEP latency and reduction of amplitude

'extra-spinal' mechanisms.⁵⁶⁻⁵⁸ These observations may be important in the evaluation of new therapeutic strategies directed to spinal cord recovery.

Conclusion

The combination of clinical and electrophysiological recordings in acute SCI patients represent reliable predictors about the degree of recovery of upper and lower limb functions. Electrophysiological recordings are of similar sensitivity to the ASIA scores and provide additional information about the level and extent of lesions of the central (spinal) and peripheral parts of the nervous system (Table 4). In uncooperative patients, where the clinical examination is of restricted value, electrophysiological recordings are superior to the ASIA protocol. With the combination of appropriate examination techniques it is possible to predict the recovery of hand function, ambulatory capacity, bladder function and autonomic dysfunction, all of which are of relevance in SCI patients in respect to life

Table 4 Application of electrophysiological recordings to predict the outcome of functional deficits in patients with acute SCI

Functional outcome	Electrophysiological recording
Hand function	ulnar SSEP MEP abductor dig.min. muscle
Ambulatory capacity	tibial SSEP MEP tibial ant. muscle
Bladder function	pubdental SSEP bulbocavernosus reflex
Muscle paresis	EMG – neurographic recordings reflex recordings
Autonomic dysfunction	sympathetic skin response

quality and expectancy. Furthermore, the application of both clinical and electrophysiological recordings can help to assess spontaneous spinal cord recovery and to evaluate new therapeutic approaches.

Acknowledgement

This work was supported by grants from the International Institut Research in Paraplegia (No P 38/97), the ‘Swiss National Science Foundation’ (No 3200-052562.97) and the ‘Schweizerische Bankgesellschaft’ on behalf of a client.

References

- Grundy BL, Friedman W. Electrophysiological evaluation of the patient with acute spinal cord injury. *Critical Care Clinics* 1987; **3**: 519–548.
- Foo D, Subrahmanyam TS, Rossier AB. Posttraumatic acute anterior spinal cord syndrome. *Paraplegia* 1981; **19**: 201–205.
- Schrader SC, Sloan TB, Toleikis JR. Detection of sacral sparing in acute spinal cord injury. *Spine* 1987; **12**: 533–535.
- Dietz V, Young RR. The Syndromes of spinal cord dysfunction. In: Brandt Th *et al.* (eds). *Neurological Disorders: Course and Treatment*. Academic Press, San Diego 1996; **Chapter 58**, pp. 641–652.
- Stauffer ES. Neurologic recovery following injuries to the cervical spinal cord and nerve roots. *Spine* 1984; **9**: 532–534.
- Roth EJ, Lawler MH, Yarkony GM. Traumatic central cord syndrome: clinical features and functional outcomes. *Arch Phys Med Rehabil* 1990; **71**: 18–23.
- Frankel HL *et al.* The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and tetraplegia. *Paraplegia* 1969; **73**: 179–192.
- Maynard FM *et al.* Neurological prognosis after traumatic quadriplegia. *J Neurosurg* 1979; **50**: 611–616.
- Crozier KS, Graziani V, Ditunno JF, Herbison GJ. Spinal cord injury: Prognosis for ambulation based on sensory examination in patients who are initially motor complete. *Arch Phys Med Rehabil* 1991; **72**: 119–121.
- Ditunno JF, Young W, Donovan WH, Creasy G. The international standards booklet for neurological and functional classification of spinal cord injury. *Paraplegia* 1994; **32**: 70–80.
- Waters RL *et al.* Motor and sensory following complete tetraplegia. *Arch Phys Med Rehabil* 1993; **74**: 242–247.
- Waters RL, Rodney A, Yakura JS, Vigil D. Prediction of ambulatory performance based on motor scores derived from standards of the American Spinal Injury Association. *Arch Phys Med Rehabil* 1994; **75**: 750–760.
- Houlden DA, Schwartz ML, Klettke KA. Neurophysiologic diagnosis in incooperative trauma patients: confounding factors. *J Traum* 1992; **33**: 244–251.
- York DH *et al.* Utilization of somatosensory evoked cortical potentials in spinal cord injury. *Spine* 1983; **8**: 832–839.
- Barker AT *et al.* Magnetic stimulation of the human brain. *J Physiol* 1985; **369**: 3.
- Clarke CE, Modarres-Sadeghi H, Twomey JA, Burt AA. Prognostic value of cortical stimulation in spinal cord injury. *Paraplegia* 1994; **32**: 554–560.
- Tavy DLJ *et al.* Transcranial magnetic stimulation in patients with cervical spondylotic myelopathy: clinical and radiological correlation. *Muscle & Nerve* 1994; **17**: 235–241.
- Curt A, Weinhardt C, Dietz V. Significance of sympathetic skin response in the assessment of autonomic failure in patients with spinal cord injury. *J Autonomic Nervous System* 1996; **61**: 175–180.
- Parry GJ. Electrodiagnostic studies in the evaluation of peripheral nerve and brachial plexus injuries. *Neurol Trauma* 1992; **4**: 921–934.
- Krasilowsky G. Nerve conduction studies in patients with cervical spinal cord injuries. *Arch Phys Med Rehabil* 1980; **61**: 204–208.
- Fierro B, Raimondo D, Modica A. Analysis of F response in upper motoneurone lesions. *Acta Neurol Scand* 1990; **82**: 329–334.
- Liberson WT *et al.* ‘H’ reflexes and ‘F’ waves in hemiplegics. *Electromyogr Clin Neurophysiol* 1977; **17**: 247–264.
- Kimura J *et al.* Is the F wave elicited in a select group of motoneurons? *Muscle Nerve* 1984; **7**: 392–399.
- Welch RD, Lobleby SJ, O’Sullivan SB, Freed MM. Functional independence in quadriplegia: critical levels. *Arch Phys Med Rehabil* 1986; **67**: 235–240.
- Curt A, Dietz V. Traumatic cervical spinal cord injury: Relation between somatosensory evoked potentials, neurological deficit and hand function. *Arch Phys Med Rehabil* 1996; **77**: 48–53.
- Curt A, Keck ME, Dietz V. Functional outcome following spinal cord injury: Significance of motor evoked potentials. *Arch Phys Med Rehabil* 1998; **79**: 81–86.
- Curt A, Dietz V. Nerve conduction study in cervical spinal cord injury: Significance for hand function. *Neuro Rehabil* 1996; **7**: 165–173.
- Curt A, Dietz V. Neurographic assessment of intramedullary motoneurone lesions in cervical spinal cord injury: Consequences for hand function. *Spinal Cord* 1996; **34**: 326–332.
- Lantheim PA, Gregorio TL, Garber SL. Highlevel quadriplegia: An occupational therapy challenge. *Am J Occup Ther* 1985; **39**: 705–714.
- Taylor S, Ashby P, Verrie M. Neurophysiological changes following traumatic spinal lesions in man. *J Neurol Neurosurg Psychiatry* 1984; **47**: 1102–1108.
- Li C, Houlden DA, Rowed DW. Somatosensory evoked potentials and neurological grades as predictors of outcome in acute spinal cord injury. *J Neurosurg* 1990; **72**: 600–609.
- Crozier KS *et al.* Spinal cord injury: prognosis for ambulation based on quadriceps recovery. *Paraplegia* 1992; **30**: 762–767.
- Curt A, Dietz V. Ambulatory capacity in spinal cord injury: Significance of somatosensory evoked potentials and ASIA protocols in predicting outcome. *Arch Phys Med Rehabil* 1997; **78**: 39–43.
- Macdonell RAL, Donnan GA. Magnetic cortical stimulation in acute spinal cord injury. *Neurology* 1995; **45**: 303–306.
- Chang CW, Lien IN. Estimate of motor conduction in human spinal cord injury. *Muscle & Nerve* 1991; **14**: 990–996.
- Hirayama T *et al.* Clinical assessment of the prognosis and severity of spinal cord injury using corticospinal motor evoked potentials. In: Shimoji K, Kurokawa T, Tamaki T, Willis WD (eds) *Spinal Cord Monitoring and Electrodiagnosis*. Heidelberg, Springer, 1991, pp. 503–510.
- Boltshauser E, Isler W, Bucher HU, Friderich H. Permanent flaccid paraplegia in children with thoracic spinal cord injury. *Paraplegia* 1981; **19**: 227–234.

- 38 Rossier AB, Ott R. Bladder and urethral recordings in acute and chronic spinal cord injury patients. *Urol Int* 1976; **31**: 49–59.
- 39 Beric A, Light JK. Correlation of bladder dysfunction and lumbosacral somatosensory evoked potential S-wave abnormality in spinal cord-injured patients. *Neurourol Urodyn* 1988; **7**: 131–140.
- 40 Fowler CJ. Clinical significance of electrophysiological studies of patients with lower urinary tract dysfunction. *Neurourol Urodyn* 1992; **11**: 279–282.
- 41 Curt A, Rodic B, Schürch B, Dietz V. Recovery of bladder function in patients with acute spinal cord injury: Significance of ASIA scores and SSEP. *Spinal Cord* 1997; **35**: 368–373.
- 42 Nanninga JB, Meyer P. Urethral sphincter activity following acute spinal cord injury. *J Urol* 1980; **123**: 528–530.
- 43 Lucas MG, Thomas DG. Lack of relationship on conus reflexes to bladder function after spinal cord injury. *Br J Urol* 1989; **63**: 24–27.
- 44 Petersen I, Frankson C. Electromyographic study of the striated muscles of the male urethra. *Br J Urol* 1955; **27**: 148.
- 45 Pedersen E, Haring H, Klemar B, Torring J. Human anal reflexes. *J Neurol Neurosurg Psychiatry* 1978; **41**: 813.
- 46 Koyanagi T, Arikado K, Takamatsu T, Tsuji I. Experience with electromyography on the external urethral sphincter in spinal cord injury patients. *J Urol* 1982; **127**: 272–276.
- 47 Schürch B, Curt A, Rossier AB. The value of the sympathetic skin responses in the assessment of the vesico-urethral autonomic system. *J Urol* 1997; **157**: 2230–2233.
- 48 Nitsche B, Perschak H, Curt A, Dietz V. Loss of circadian blood pressure variability in complete tetraplegia. *J Human Hypertension* 1996; **10**: 311–317.
- 49 Curt A *et al.* Assessment of autonomic dysreflexia in patients with spinal cord injury. *J Neurol Neurosurg Psychiatry* 1997; **62**: 473–477.
- 50 Guttmann L. Spinal shock and reflex behaviour in man. *Paraplegia* 1970; **8**: 100–110.
- 51 Little JW, Halar EM. H-reflex changes following spinal cord injury. *Arch Phys Med Rehabil* 1985; **66**: 19–22.
- 52 Ashby P, Verrier M, Lightfoot E. Segmental reflex pathway in spinal shock and spinal spasticity in man. *J Neurol Neurosurg Psychiatry* 1974; **37**: 1352–1360.
- 53 Bischoff C, Schoenle PW, Conrad B. Increased F-wave duration in patients with spasticity. *Electromyogr Clin Neurophysiol* 1992; **32**: 449–453.
- 54 Curt A, Keck ME, Dietz V. Clinical value of F-wave recordings in traumatic cervical spinal cord injury. *Electroenceph Clin Neurophysiol* 1997; **105**: 189–193.
- 55 Dietz V, Wirz M, Curt A, Colombo G. Locomotor pattern in paraplegic patients: Training effects and recovery of spinal cord function. *Spinal Cord* 1998; **36**: 380–390.
- 56 Zhang Z, Guth L, Steward O. Mechanisms of motor recovery after subtotal spinal cord injury: Insights from study of mice carrying a mutation (WLD^S) that delays cellular responses to injury. *Experimental Neurology* 1998; **149**: 221–229.
- 57 Marino RJ, Herbison GJ, Ditunno JF. Pheripheral sprouting as a mechanism for recovery in the zone of injury in acute quadriplegia: a single-fiber EMG study. *Muscle & Nerve* 1994; **17**: 1466–1468.
- 58 Thomas CK, Broton JG, Calancie B. Motor unit forces and recruitment patterns after cervical spinal cord injury. *Muscle & Nerve* 1997; **20**: 212–220.