

REVIEW

International standards to document remaining autonomic function after spinal cord injury

MS Alexander¹, F Biering-Sorensen², D Bodner³, NL Brackett⁴, D Cardenas⁴, S Charlifue⁵, G Creasey⁶, V Dietz⁷, J Ditunno⁸, W Donovan¹¹, SL Elliott⁹, I Estores¹⁰, DE Graves¹¹, B Green¹², A Gousse⁴, AB Jackson¹, M Kennelly¹³, A-K Karlsson¹⁴, A Krassioukov⁹, K Krogh¹⁵, T Linsenmeyer¹⁶, R Marino⁸, CJ Mathias¹⁷, I Perikash⁶, AW Sheel⁹, G Shilero¹⁸, B Schurch⁷, J Sonksen¹⁹, S Stiens²⁰, J Wecht¹⁸, LA Wuermsers²¹ and J-J Wyndaele²²

¹University of Alabama, Birmingham, AL, USA; ²Rigshospitalet and University of Copenhagen, Copenhagen, Denmark; ³Case Western Reserve University, Cleveland, OH, USA; ⁴University of Miami, Miami, FL, USA; ⁵Craig Hospital, Englewood, CO, USA; ⁶Stanford University, Palo Alto, CA, USA; ⁷University Hospital Balgrist, Zurich, Switzerland; ⁸Thomas Jefferson University, Philadelphia, PA, USA; ⁹International Collaboration On Repair Discoveries (ICORD), University of British Columbia and Vancouver Coastal Health, Vancouver, British Columbia, Canada; ¹⁰James A Haley VA Hospital, Tampa, FL, USA; ¹¹Baylor College of Medicine, Houston, TX, USA; ¹²Shepherd Center, Atlanta, GA, USA; ¹³McKay Urology, Carolinas Healthcare, Charlotte, NC, USA; ¹⁴Sahlgrenska University Hospital, Gothenburg, Sweden; ¹⁵Aarhus University Hospital, Aarhus, Denmark; ¹⁶Kessler Institute for Rehabilitation, West Orange, NJ, USA; ¹⁷Imperial College, St Mary's Hospital, London, UK; ¹⁸James J Peters Veterans Affairs Medical Center, Bronx, NY, USA; ¹⁹Herlev Hospital, University of Copenhagen, Copenhagen, Denmark; ²⁰Veterans Affairs Puget Sound Health Care, Seattle, WA, USA; ²¹Mayo Clinic, Rochester, MS, USA and ²²University Hospital Antwerp, University Antwerp, Antwerp, Belgium

Study design: Experts opinions consensus.

Objective: To develop a common strategy to document remaining autonomic neurologic function following spinal cord injury (SCI).

Background and Rationale: The impact of a specific SCI on a person's neurologic function is generally described through use of the International Standards for the Neurological Classification of SCI. These standards document the remaining motor and sensory function that a person may have; however, they do not provide information about the status of a person's autonomic function.

Methods: Based on this deficiency, the American Spinal Injury Association (ASIA) and the International Spinal Cord Society (ISCoS) commissioned a group of international experts to develop a common strategy to document the remaining autonomic neurologic function.

Results: Four subgroups were commissioned: bladder, bowel, sexual function and general autonomic function. On-line communication was followed by numerous face to face meetings. The information was then presented in a summary format at a course on Measurement in Spinal Cord Injury, held on June 24, 2006. Subsequent to this it was revised online by the committee members, posted on the websites of both ASIA and ISCoS for comment and re-revised through webcasts. Topics include an overview of autonomic anatomy, classification of cardiovascular, respiratory, sudomotor and thermoregulatory function, bladder, bowel and sexual function.

Conclusion: This document describes a new system to document the impact of SCI on autonomic function. Based upon current knowledge of the neuroanatomy of autonomic function this paper provides a framework with which to communicate the effects of specific spinal cord injuries on cardiovascular, broncho-pulmonary, sudomotor, bladder, bowel and sexual function.

Spinal Cord (2009) 47, 36–43; doi:10.1038/sc.2008.121; published online 28 October 2008

Keywords: autonomic nervous system; spinal cord injury; human; international standards

Introduction

Currently, the International Standards for the Neurological Classification of Spinal Cord Injury (ISNCSCI) are used to

document impairments of motor and sensory function after spinal cord injury (SCI). These standards are now in the sixth edition. A major revision occurred in 1992 when the first internationally accepted standards were published.¹ Changes included a revised definition of complete and incomplete injuries, the modification of the Frankel scale to the ASIA Impairment scale, and the addition of sensory scores. Subsequently, a teaching package was developed consisting

Correspondence: Dr A Krassioukov, ICORD/Department of Medicine, University of British Columbia, 6270 University Blvd, Vancouver, British Columbia, Canada V6T 1Z4.

E-mail: krassioukov@icord.org

Received 19 June 2008; revised 29 August 2008; accepted 31 August 2008; published online 28 October 2008

of a reference manual² and videotapes. Both the ISNCSCI and the reference manual have since been revised, with the ISNCSCI reprinted in 2002³ and the reference manual in 2003.⁴ It is notable that the ISNCSCI increasingly have been used to document remaining spinal cord function in clinical trials related to SCI.

Despite success with the ISNCSCI, assessment of remaining autonomic functions after SCI is lacking. It is the goal of this document to provide a framework for the assessment of specific autonomic functions following SCI. Moreover, it is recommended that this assessment be part of the clinical evaluation of individuals with SCI.

Assessment of autonomic function in SCI

Autonomic dysfunctions, including abnormal blood pressure and heart rate control, and sweating and temperature dysregulation are common consequences of SCI in humans.^{5,6} Even though sympathetic activity is typically decreased, stimuli below the level of the lesion can induce widespread sympathetic activation resulting in potential problems such as profound vasoconstriction and blood pressure increase.⁷⁻⁹ There is relatively limited information on the relationship between the level and completeness of the SCI lesion and the degree of autonomic dysfunction.¹⁰ The complexity and organization of the autonomic nervous system and its involvement in the control of almost every bodily system makes it difficult to select appropriate clinical tests for individuals with SCI. However, experience in the clinical assessment of individuals with SCI is limited and uniform operational definitions of autonomic dysfunction are lacking. After SCI, changes in the autonomic nervous system regarding bladder, bowel and sexual function are also difficult to document through bedside examination. To address these problems, an international committee, designed to include representatives from the American Spinal Injury Association (ASIA), the International Spinal Cord Society (ISCoS) and other groups performing SCI research recommend these standards.¹¹

The goal of these standards is to describe the impact of SCI on particular organ system functions. These standards are not meant to provide recommendations for the treatment of specific organ systems. Rather, the addition of the autonomic standards will allow clinicians and researchers to appreciate possible autonomic dysfunctions and to be able to describe the effects of SCI on bowel, urinary bladder, sexual, cardiovascular, broncho-pulmonary, sudomotor and other autonomic functions.

This paper contains an overview of anatomical considerations relating to the autonomic nervous system. Four components of the autonomic classification system were reviewed (general autonomic, bladder, bowel and sexual functions) in order to provide definitions and instructions for completion of the Autonomic Standards Assessment Form (Appendix). A general anatomic diagnosis is utilized to document the overall impact of SCI on autonomic function. A classification chart is provided for the autonomic control of cardiovascular, broncho-pulmonary and sudomotor functions, including thermoregulation. A chart is provided for

the examiner to describe and grade the neurologic control of lower urinary tract, bowel and sexual responses. Finally, a chart is provided detailing a urodynamic evaluation that should be completed on all patients. This information is recorded on a single page document for each person with SCI (see Appendix).

General anatomic and physiologic considerations

Blood vessels, heart, respiratory tract, sweat glands, bowel, urinary bladder and sexual functions are under nervous system control: autonomic (involuntary), somatic (voluntary) or both. During micturition and defecation, there are coordinated actions of the external urethral and anal sphincters (both under voluntary cortical control by the somatic nervous system) and smooth muscles of the bladder and the bowel (both under involuntary control by the autonomic nervous system). Cortical control also plays an important role in sexual arousal, resulting in psychogenic erection and vaginal lubrication. However, reflex erection and lubrication can be achieved with peripheral stimulation, even when cortical influences are disrupted. On the other hand, many aspects of cardiovascular and sudomotor control are primarily dependent on the interplay of sympathetic and parasympathetic nervous system activity. SCI disrupts the descending spinal voluntary motor and involuntary autonomic pathways, resulting in dysfunctions of the cardiovascular and broncho-pulmonary systems, sudomotor, urinary bladder, bowel and sexual organs. The level and the severity of injury to these pathways results in a variety of autonomic dysfunctions depending on the altered supraspinal control of the sympathetic and parasympathetic nervous systems.¹²

Autonomic nervous system: anatomy and function

The autonomic nervous system commonly is subdivided into two major parts: the sympathetic and parasympathetic components.¹³ Most of the visceral organs are innervated by both components of the autonomic nervous system.^{13,14} The sympathetic and parasympathetic systems are integrated functionally with each other within the central nervous system and provide balanced regulation of innervated organs.

Certain cortical structures, and the hypothalamus, contribute to regulation of the autonomic circuits within the brainstem and spinal cord. These cerebral structures should be intact morphologically but there may be functional alterations following SCI.

Both divisions of the autonomic nervous system have two neuronal populations interposed between the central nervous system and target organs. The first neuron is called the preganglionic neuron, with the cell body within the gray matter of the brain or spinal cord. Axons of these neurons, called preganglionic fibers, travel within the ventral roots of the spinal cord or cranial nerves. These fibers synapse on the second group of neurons, called postganglionic. This group of neurons is located within the autonomic ganglia in the peripheral nervous system. The axons of these neurons, called postganglionic fibers, innervate the target organs.

Sympathetic preganglionic neurons reside in the spinal gray matter in the thoracic (T1–T12) and upper lumbar segments (L1–L2) of the spinal cord.^{13,15} The majority of sympathetic preganglionic neurons are localized within the lateral horns or intermediolateral nucleus of the spinal cord. A small proportion of sympathetic preganglionic neurons are found near the central canal of the spinal cord. Axons of the sympathetic preganglionic neurons exit through the ventral roots and synapse on postganglionic sympathetic neurons located in the spinal paravertebral ganglia (sympathetic chain ganglia) and prevertebral ganglia (the celiac, superior and inferior mesenteric ganglia). The postganglionic neurons then send their axons through the peripheral nerves to innervate the target organs, including the heart, blood vessels, respiratory tract, sweat glands, sexual organs and smooth muscles within the gut and bladder (Table 1).

Parasympathetic preganglionic neurons are located within the nuclei of four cranial nerves (CN III, VII, IX, X) in the brainstem and within the sacral spinal segments (S2–S4). Parasympathetic control of the cardiovascular system and the upper portion of the gastrointestinal tract is through the vagus nerve (CN X), which exits from the brain through the base of the skull and synapses with the sino-atrial node and the nerve cells within the enteric nervous system of the gut. There is no parasympathetic innervation of the peripheral vasculature except in the pelvic organs. Parasympathetic innervation of the bladder, reproductive organs and lower portion of the gut is provided by the sacral portion of the spinal cord (S2–S4).

Neural control of the cardiovascular system

Vessels in the upper portion of the body and the heart receive sympathetic innervation from the T1–T5 spinal sympathetic neurons, whereas the major vasculature beds in the gut and lower extremities are under the control of the more caudal T5–L2 spinal sympathetic neurons. In addition to sympathetic fibers, the sino-atrial node is also innervated by postganglionic parasympathetic fibers from the vagus nerve, providing tonic inhibition of the heart to lower heart rate.

Dual innervation of the heart and the segmental differences in sympathetic innervation to a variety of vascular beds are particularly important for the understanding of basal blood pressure and heart rate, as well as cardiovascular responses following cervical, mid-thoracic, or lower thoracic SCI.^{5,6}

Neural control of the broncho-pulmonary system

Similar to other organ systems, impairment in respiratory function is directly related to level and completeness of lesion following SCI.¹⁶ In individuals with an intact central nervous system respiration occurs through coordinated activity of the somatic nervous system (control of inspiratory and expiratory muscles) and the autonomic nervous system (bronchial tone and secretion). The diaphragm, is the major muscle of inspiration and is innervated by the phrenic nerve (C3–C5). Persons with cervical SCI usually maintain the ability to breathe spontaneously, but manifest greater reductions in vital capacity and more severe restrictive impairment than persons with thoracolumbar injury. Expiratory muscle function, especially the ability to generate

Table 1 Organization of innervation of major organs

<i>Organ</i>	<i>Sympathetic nervous system (T1–L2)</i>	<i>Parasympathetic nervous system vagus nerve (CN X) and (S2–4)</i>	<i>Somatic/motor</i>
<i>Heart</i>	T1–T5	Vagus nerve (CN X)	None
<i>Blood vessels</i>			
Upper body	T1–T5	Blood vessels in certain organs: salivary glands, gastrointestinal glands (CNX), and in genital erectile tissue (S2–S4)	None
Lower body	T5–L2		
<i>Lower urinary tract</i>			
Detrusor	T10–L2	S2–S4	
Bladder neck/internal urethral sphincter	T10–L2	None	
External urethral sphincter	T10–L2	None	S3–S5
<i>Gastro-intestinal tract</i>			
From esophagus to splenic flexure	T1–L2	Vagus nerve (CN X)	
From splenic flexure to rectum Internal anal sphincter	T1–L2	S2–S4	
External anal sphincter	T10–L2	S2–S4	S3–S5
<i>Reproductive organs</i>			
Penis	T10–L2	S2–S4	S1–S3
Vagina	T10–L2	S2–S4	S1–S3
Uterus and other reproductive organs	T10–L2	S2–S4, Vagus nerve (CNX)	S1–S3
Testicles and other reproductive organs	T10–L2	S2–S4	S1–S3
<i>Broncho-pulmonary system</i>	T1–T5	Vagus nerve (CN X)	C3–C8 T1–T12
<i>Sweat glands</i>	T1–L2	None	None

Abbreviations: C, cervical; CN, cranial nerve; L, lumbar; S, sacral; T, thoracic.

forceful cough for airway clearance, may be severely impaired in persons with cervical SCI and to a lesser extent in those with lower levels of injury because of paralysis of abdominal muscles and the lateral internal intercostals (T1–T12).

Autonomic control of the broncho-pulmonary system is principally governed by the parasympathetic nervous system. Sympathetic neurotransmission to the broncho-pulmonary system arising from the sympathetic chain at the T1–T6 level has historically been felt to have little functional significance in human airways.¹⁷ However, studies in persons with cervical SCI in whom sympathetic innervation to the broncho-pulmonary system is interrupted, reveal reduction in baseline airway caliber and exaggerated bronchial responsiveness.^{18–22}

Neural control of sweat glands

Similar to blood vessels, the sweat glands are predominantly under sympathetic control. Sweat glands in the upper portion of the body receive sympathetic innervation from T1–T5 spinal sympathetic neurons, whereas the glands of the lower part of the body are under the control of the T5–L2 spinal sympathetic neurons (Table 1). Supraspinal control of sweating, by regions in the hypothalamus and amygdala is now better defined in humans using neuroimaging studies.²³

Neural control of the lower urinary tract

Lower urinary tract (LUT) function is controlled by neural circuits in the brain and spinal cord that coordinate the activity of visceral smooth muscle in the urinary bladder and urethra with activity of striated muscle in the external urethral sphincter.^{24–26} LUT function involves central pathways (supraspinal and spinal) and peripheral pathways (pelvic parasympathetic, lumbar sympathetic and somatic pudendal nerve). Axons of Onuf's nucleus in the sacral cord segments innervate the external urethral sphincter through the pudendal nerve. The bladder receives sympathetic innervation through hypogastric nerves and sacral parasympathetic innervation through pelvic nerves. Afferent information from the LUT enters the spinal segments and synapses on interneurons that either make local segmental connections with motor pathways or send their axons to the brain. There are reciprocal intraspinal reflexes between the lumbar and sacral cord as well as supraspinal control, and there may be segmental reflexes between the LUT and colon.²⁷ Ascending pathways connect with structures in the brainstem, including the pons and periaqueductal gray matter of the midbrain to execute reflex functions, and with higher centers of the brain (cingulate and frontal gyri) mediating storage and conscious perception of sensations arising from the LUT.²⁸ Activation of the sympathetic circuits through spinal reflexes mediates detrusor muscle relaxation and bladder neck contraction, resulting in storage of urine. Activation of the sacral parasympathetic efferents results in detrusor contraction and promotes voiding. Normal volitional voiding is achieved with voluntary relaxation of the external urethral sphincter.

The LUT micturition reflex pathway has two modes of operation: storage and elimination. In infants these mechan-

isms function in a reflex manner to produce involuntary voiding; however, in adults urine storage and elimination are subject to voluntary control as a result of connections between the forebrain and brainstem. SCI damages the spinal tracts involved in central control of the LUT, often leading to simultaneous activation of parasympathetic neurons innervating the detrusor and somatic neurons innervating the external urethral sphincter. This abnormality causes varying degrees of detrusor–sphincter dysynergia. Dysynergia between the bladder and bladder neck can also be seen.

Neural control of the bowel

Similar to the lower urinary tract, bowel function requires the coordinated activity of the somatic and autonomic nervous systems. Colonic peristalsis is coordinated by a network of neurons linking the brain to the colonic mucosa. Cranial nerve X (vagus nerve) courses from the brainstem and innervates the gut to the splenic flexure of the colon. The inferior splanchnic nerve carries pelvic parasympathetic fibers from the sacral segments of the spinal cord to the splenic flexure, the left colon and rectum. The enteric nervous system includes the Auerbach's plexus (intramuscular myenteric), unmyelinated fibers and postganglionic parasympathetic cell bodies that mainly coordinate motility. Under the mucosa, Meissner's plexus relays sensory and local motor responses. The internal anal sphincter is a continuation of the circular muscle layer of the rectum under reflex control by the enteric nervous system and S2–4 spinal segments of the spinal cord. The external anal sphincter and pelvic floor are supplied by the mixed motor and sensory somatic pudendal nerve providing voluntary control. There is also evidence of sympathetic control of the external anal sphincter.

The bowel functions of storage, stool propulsion and defecation are dependent on coordinated control from the two components of the autonomic nervous system, the enteric nervous system and voluntary motor control of the skeletal muscles of the pelvic floor and external anal sphincter.^{29,30}

Neural control of sexual responses

Male and female sexual organs receive innervation from both components of the autonomic nervous system. Similar to the LUT, sympathetic innervation is provided through the hypogastric nerve and parasympathetic innervation through the pelvic nerve. Psychogenic erection and vaginal lubrication are thought to be regulated by the sympathetic nervous system in conjunction with the parasympathetic, whereas reflex erection and vaginal lubrication are thought to be activated by the parasympathetic nervous system.^{31–34}

Ejaculation is a neurologically more complicated phenomenon and relies on the coordination of the sympathetic (T11–L2) and parasympathetic nervous systems (S2–4) in addition to the somatic nervous system through the pudendal nerve (S2–5). Ejaculation and orgasm do not always coincide. The neurologic control of orgasm is less well understood; however, studies in SCI suggest that an intact sacral reflex arc is necessary for orgasm to occur.^{31,35}

The autonomic standards assessment form

Anatomical classification

An anatomical classification to describe the impact of SCI on urinary bladder, bowel and sexual function is recommended. Descriptive terms include supraconal, conal and cauda equina. Supraconal refers to those injuries occurring above the conus medullaris. In general, supraconal injuries cause an overactive or upper motor neuron pattern of damage affecting lower urinary tract, bowel and sexual functions. Conal injuries include those affecting the conus medullaris of the spinal cord and often cause a mixed lesion to lower urinary tract, bowel and sexual functions with a resultant overactive or acontractile picture. Cauda equina injuries include those affecting the cauda equina and generally cause an acontractile or lower motor neuron picture affecting lower urinary tract, bowel and sexual functions.

It should be noted that the anatomical classification for autonomic function is intended to be used in conjunction with the ISNCSCI. Thus, in addition to using the above terminology, the examiner should provide information on the completeness of injury as determined by the preservation of perianal and anal sensation and voluntary anal contraction as described in the ISNCSCI, and the type of reflex activity present, as described in the autonomic standards.

Classification of general autonomic function

It is recommended that a detailed description of remaining general autonomic function be provided as part of the neurologic assessment of individual patients. This information should be documented through checks in the appropriate boxes in the table entitled general autonomic function (see Appendix). The information is determined based on a combination of neurologic examination and clinical history.

The recognition and assessment of cardiac dysrhythmias includes documentation of bradycardia or heart rate under 60 beats per minute (b.p.m.) and tachycardia or heart rate over 100 b.p.m. Other dysrhythmias should also be documented.

Abnormalities of arterial blood pressure include supine hypertension (blood pressure greater than 140/90 mm Hg) and supine hypotension (systolic blood pressure less than 90 mm Hg). Orthostatic hypotension is a symptomatic or asymptomatic decrease in BP usually exceeding 20 mm Hg systolic or 10 mm Hg diastolic on moving from the supine to an upright position.³⁶ Symptoms include dizziness, headache or 'coat hanger' neck ache and fatigue.

Autonomic dysreflexia is a constellation of signs and/or symptoms in SCI at and above T6 in response to noxious or non-noxious stimuli below the level of injury defined by an increase in systolic blood pressure (\geq than 20 mm Hg above baseline), and which may include one of the following symptoms: headache, flushing and sweating above the level of the lesion, vasoconstriction below the level of the lesion and dysrhythmias. This syndrome may or may not be symptomatic and may occur at any period following SCI.

The autonomic components related to broncho-pulmonary control are not easily tested at bedside; however, it is

important to document the respiratory capacity of individuals with SCI. Therefore, the need for complete or partial ventilatory assistance should be documented under broncho-pulmonary control.

Temperature dysregulation or an elevation or decrease in body temperature without signs of infection may result from exposure to environmental temperature change and should be documented.

Finally, abnormalities of sudomotor function including hyperhidrosis or nonphysiological sweating in response to noxious/non-noxious stimuli, and hypohidrosis, diminished sweating below the level of the injury in response to a rise in temperature³⁷ should be documented.

Assessment of urinary bladder, bowel and sexual function

Information related to urinary, bowel and sexual function is recorded in the table entitled Lower Urinary Tract, Bowel and Sexual Function and is based on the clinical examination and history (see Appendix). Consistent with the ISNCSCI sensory scoring system, possible responses are graded. Responses include normal (2) when there is no change in neurologic control with respect to a specific function, reduced or altered (1) neurologic control with regards to a specific function, absent (0) neurologic control with regards to a specific function and unable to assess a specific function (not testable, NT).

For the LUT, it is strongly recommended that urodynamics be routinely performed following SCI. The committee recognizes that some persons with SCI will not yet have had urodynamics. Clinicians would benefit from being able to communicate information regarding the patient's ability to perceive sensations related to bladder filling and voluntary control of voiding. It is therefore recommended that the following assessment be used until a urodynamic evaluation can be performed (see appendix). Furthermore, the clinician should consult the International Data Sets in SCI for more information on recommended urologic assessments and procedures.^{38,39} Acknowledging the limitations of patient self-report, it is recommended that awareness of the need to empty the bladder (sensation) is first documented. Next, the ability to prevent leakage (continence) should be documented. The bladder emptying method should be recorded.

For bowel function, it is recommended that sensation of the need for a bowel movement is recorded. Continence of stool, that is, the ability to prevent an accidental bowel movement, should be recorded followed by documentation of the presence of voluntary sphincter contraction during anorectal examination.

For sexual function it is recommended that the presence of psychogenic genital arousal (penile erection or vaginal lubrication) is first recorded followed by documentation of reflex genital arousal. Ability to achieve orgasm is recorded. The presence of antegrade ejaculation is documented for males. The ability to sense menses (cramping, pain, etc.) relative to before injury is documented for females.

Urodynamic evaluation

The urodynamic assessment is based on the clinical examination and observations made during urodynamic studies. There are three sections to the assessment: sensation during filling, detrusor activity and sphincter function.

Sensation during filling has five possible choices and is dependent on specific information that can only be obtained through urodynamics. The following time points and associated data must be documented when performing urodynamics after SCI.^{39,40} The first sensation of bladder filling is the feeling during filling cystometry, when the patient first becomes aware of the bladder filling. The first desire to void is the feeling, during filling cystometry, that would lead the patient to pass urine at the next convenient moment, but voiding can be delayed if necessary. The strong desire to void is defined, during filling cystometry, as a persistent desire to void without the fear of leakage.

Normal bladder sensation can be determined by these three defined points noted during filling cystometry and evaluated in relation to the bladder volume at that moment and in relation to the individual's symptomatic complaints. Increased bladder sensation is defined, during filling cystometry, as an early first sensation of bladder filling (or an early desire to void) and/or an early strong desire to void, which occurs at low bladder volume and which persists. Reduced bladder sensation is defined, during filling cystometry, as diminished sensation throughout bladder filling. Absent bladder sensation means that, during filling cystometry, the individual has no bladder sensation.

Non-specific bladder sensations, during filling cystometry, may make the individual aware of bladder filling, for example, abdominal fullness or vegetative symptoms.

Assessment of detrusor function is the next variable that should be documented through performance of urodynamics. Four separate types of detrusor function are defined for purposes of documentation: normal detrusor function, detrusor overactivity, detrusor underactivity and acontractile detrusor. Normal detrusor function allows bladder filling with little or no change in pressure and no involuntary phasic contractions occur despite provocation. Normal detrusor function in the voiding phase is achieved by a voluntarily initiated continuous detrusor contraction that leads to complete bladder emptying within a normal time span, and in the absence of obstructions. Detrusor overactivity is a urodynamic observation characterized by involuntary detrusor contractions during the filling phase which may be spontaneous or provoked. Detrusor overactivity in the case of spinal cord injury is generally neurogenic detrusor overactivity and it is recommended that this term is used in lieu of 'detrusor hyperreflexia.' Idiopathic detrusor overactivity can also occur without a defined cause. Detrusor underactivity is defined as a contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or a failure to achieve complete bladder emptying within a normal time span. Finally, an acontractile detrusor is one that cannot be demonstrated to contract during urodynamic studies.

The final area that should be documented based on urodynamics is that of urethral sphincter function. Normal urethral closure mechanism maintains a positive urethral closure pressure during bladder filling even in the presence of increased abdominal pressure, although it may be overcome by detrusor overactivity. Normal urethral function during voiding is defined as a urethra that opens and is relaxed continuously to allow the bladder to be emptied at a normal pressure. Incompetent urethral closure mechanism is defined as one that allows leakage of urine in the absence of a detrusor contraction. Detrusor sphincter dyssynergia is defined as a detrusor contraction concurrent with an involuntary contraction of the urethral and/or periurethral striated muscle during voiding. Occasionally, the flow may be prevented altogether. Non-relaxing sphincter is defined as an obstruction during voiding that usually occurs in individuals with sacral or cauda equina lesions, such as meningomyelocele and is characterized by a non-relaxing, obstructing urethra resulting in reduced urine flow.

Conclusion

This document describes a new system to document the impact of SCI on autonomic function. All of the information is designed to be succinctly filled out on one document that should be used to communicate the neurologic effects of injury on autonomic function in all patients with SCI in conjunction with the ISNCSCI. The autonomic standards assessment is meant to complement information available in the respective International SCI Data Sets. Thus, the degree of detail in the autonomic standards is significantly less than that of the data sets, which are designed to serve as a complete patient record for persons with SCI.^{41,42} Based upon current knowledge of the neuroanatomy of autonomic function this paper provides a framework with which to communicate the effects of specific spinal cord injuries on cardiovascular, broncho-pulmonary, sudomotor, bladder, bowel and sexual function. An online training program for these standards is in development.

Acknowledgements

Members of the steering committee acknowledge funding and 'in kind' support from the, American Spinal Injury Association, International Spinal Cord Society, Aventis and Astra Tech. This document and documents with data sets are freely available on the ASIA and ISCoS websites. Steering Committee: Marcalee Sipski Alexander, Andrei Krassioukov, Fin Biering-Sorensen, Susan Charlifue, Volker Dietz, John Ditunno, Daniel E Graves, Michael Kennelly, Ralph Marino, Steven Stiens. Urology Domain: Michael Kennelly (Chair), Diana Cardenas, Graham Creasey, Angelo Gousse, Bruce Green, Todd Linsenmeyer, Brigitte Schurch, Jean-Jacques Wyndaele. Bowel Domain: Steve Stiens (Chair), Irene Estores, Klaus Krogh, Inder Perakash. Sexual Function Domain: Marcalee Sipski Alexander (Chair), Don Bodner, Nancy Brackett, Stacy Elliott, Amie Jackson, Jens Sonksen. General Autonomic Function Domain: Andrei Krassioukov (Chair), Ann-Katrin Karlsson, Ralph Marino, Christopher J Mathias,

Greg Schilero, Bill Sheel, Jill Wecht, Lisa Wuermsler. Other individuals who participated in the discussion and contributed to the present document were Victoria Claydon, Jean Gabriel Previnaire, Nancy Shinowara, Lynne Weaver and Darren Warburton.

References

- Ditunno JF, Young WS, Donovan WH, Bracken MB, Brown M, Creasey G *et al*. *International Standards for Neurological and Functional Classification of Spinal Cord Injury, Revised*. American Spinal Injury Association: Chicago, IL, 1992.
- Ditunno JF, Young WS, Donovan WH, Bracken MB, Brown M, Creasey G *et al*. *Reference Manual for the International Standards for Neurological and Functional Classification of Spinal Cord Injury*. American Spinal Injury Association: Chicago, IL, 1994.
- Marino RJ, Barros T, Biering-Sorensen F, Burns SP, Donovan WH, Graves DE *et al*. International standards for neurological classification of spinal cord injury. *J Spinal Cord Med* 2003; **26** (Suppl 1): S50–S56.
- Marino R, Maynard FM, Priebe M, Donovan WH, Ditunno JF, Burns S *et al*. *Reference Manual for the International Standards for Neurological Classification of Spinal Cord Injury*. American Spinal Injury Association: Chicago, IL, 2003.
- Krassioukov A, Claydon VE. The clinical problems in cardiovascular control following spinal cord injury: an overview. *Prog Brain Res* 2006; **152**: 223–229.
- Mathias CJ, Frankel HL. Autonomic disturbances in spinal cord lesions. In: Bannister R, Mathias CJ (eds). *Autonomic Failure, A Textbook of Clinical Disorders of the Autonomic Nervous System*. Oxford Medical Publications, Oxford University Press: Oxford, 2002, pp 839–881.
- Stjernberg L, Blumberg H, Wallin BG. Sympathetic activity in man after spinal cord injury. Outflow to muscle below the lesion. *Brain* 1986; **109** (Part 4): 695–715.
- Karlsson AK, Friberg P, Lonnroth P, Sullivan L, Elam M. Regional sympathetic function in high spinal cord injury during mental stress and autonomic dysreflexia. *Brain* 1998; **121**: 1711–1719.
- Gao SA, Ambring A, Lambert G, Karlsson AK. Autonomic control of the heart and renal vascular bed during autonomic dysreflexia in high spinal cord injury. *Clin Auton Res* 2002; **12**: 457–464.
- Low PA. Testing the autonomic nervous system. *Semin Neurol* 2003; **23**: 407–421.
- Sipski ML, Marino RJ, Kennelly M, Krassioukov AV, Steins SA. Autonomic standards and SCI: preliminary considerations. *Topics in Spinal Cord Rehabilitation* 2006; **11**: 101–109.
- Furlan JC, Fehlings MG, Shannon P, Norenberg MD, Krassioukov AV. Descending vasomotor pathways in humans: correlation between axonal preservation and cardiovascular dysfunction after spinal cord injury. *J Neurotrauma* 2003; **20**: 1351–1363.
- Krassioukov AV, Weaver LC. Physical medicine and rehabilitation: state of the art reviews. In: Teasell R, Baskerville VB (eds). *Anatomy of the Autonomic Nervous System*. Hanley & Belfus, Inc., Medical Publishers: Philadelphia, 1996, pp 1–14.
- Lefkowitz RJ, Hoffman BB, Taylor P. Neurohumoral transmission: the autonomic and somatic motor nervous system. In: Gilman AG, Rall TW, Nies AS, Taylor R (eds). *The Pharmacological Basis of Therapeutics*. Pergamon Press: New York, Oxford, Tokyo, Toronto, 2007, pp 84–121.
- Schramm LP, Strack AM, Platt KB, Loewy AD. Peripheral and central pathways regulating the kidney: a study using pseudorabies virus. *Brain Res* 1993; **616**: 251–262.
- Linn WS, Spungen AM, Gong H, Adkins RH, Bauman WA, Waters RL. Forced vital capacity in two large outpatient populations with chronic spinal cord injury. *Spinal Cord* 2001; **39**: 263–268.
- Barnes PJ. Neural control of human airways in health and disease. *Am Rev Respir Dis* 1986; **134**: 1289–1314.
- Grimm DR, Chandry D, Almenoff PL, Schilero G, Lesser M. Airway hyperreactivity in subjects with tetraplegia is associated with reduced baseline airway caliber. *Chest* 2000; **118**: 1397–1404.
- Grimm DR, Arias E, Lesser M, Bauman WA, Almenoff PL. Airway hyperresponsiveness to ultrasonically nebulized distilled water in subjects with tetraplegia. *J A Physiol* 1999; **86**: 1165–1169.
- Dicpinigaitis PV, Spungen AM, Bauman WA, Absgarten A, Almenoff PL. Bronchial hyperresponsiveness after cervical spinal cord injury. *Chest* 1994; **105**: 1073–1076.
- Schilero GJ, Grimm D, Spungen AM, Lenner R, Lesser M. Bronchodilator responses to metaproterenol sulfate among subjects with spinal cord injury. *J Rehabil Res Dev* 2004; **41**: 59–64.
- Schilero GJ, Grimm DR, Bauman WA, Lenner R, Lesser M. Assessment of airway caliber and bronchodilator responsiveness in subjects with spinal cord injury. *Chest* 2005; **127**: 149–155.
- Kanosue K, Sadato N, Okada T, Yoda T, Nakai S, Yoshida K *et al*. Brain activation during whole body cooling in humans studied with functional magnetic resonance imaging. *Neurosci Lett* 2002; **329**: 157–160.
- De Groat WC, Nadelhaft I, Milne RJ, Booth AM, Morgan C, Thor K. Organization of the sacral parasympathetic reflex pathways to the urinary bladder and large intestine. *J Auton Nerv Syst* 1981; **3**: 135–160.
- Yoshimura N, De Groat WC. Neural control of the lower urinary tract. *Int J Urol* 1997; **4**: 111–125.
- Torrens MJ, Morrison JFB. *The Physiology of the Lower Urinary Tract*. Springer: London, 1987.
- Stiens SA, Bergman SB, Goetz LL. Neurogenic bowel dysfunction after spinal cord injury: clinical evaluation and rehabilitative management. *Arch Phys Med Rehabil* 1997; **78**: S86–102.
- Mehnert U, Boy S, Svensson J, Michels L, Reitz A, Candia V *et al*. Brain activation in response to bladder filling and simultaneous stimulation of the dorsal clitoral nerve—an fMRI study in healthy women. *Neuroimage* 2008; **41**: 682–689.
- Janig W. Functional organization of the lumbar sympathetic outflow to pelvic organs and colon. In: Renaud LP, Polosa C (eds). *Organization of the Autonomic Nervous System: Central and Peripheral Mechanisms*. Alan R. Liss: New York, 1987, pp 57–66.
- Clinical practice guidelines: neurogenic bowel management in adults with spinal cord injury. Spinal Cord Medicine Consortium. *J Spinal Cord Med* 1998; **21**: 248–293.
- Sipski ML, Alexander CJ, Rosen R. Sexual arousal and orgasm in women: effects of spinal cord injury. *Ann Neurol* 2001; **49**: 35–44.
- Sipski ML, Arenas A. Female sexual function after spinal cord injury. *Prog Brain Res* 2006; **152**: 441–447.
- Sachs BD. Placing erection in context—the reflexogenic psychogenic dichotomy reconsidered. *Neurosci Biobehav Rev* 1995; **19**: 211–224.
- McKenna K. The brain is the master organ in sexual function: central nervous system control of male and female sexual function. *Int J Impot Res* 1999; **11** (Suppl 1): S48–S55.
- Sipski M, Alexander C, Gomez-Marin O, Spalding J. The effects of spinal cord injury on psychogenic sexual arousal in males. *J Urol* 2007; **177**: 247–251.
- Consensus statement on the definition of orthostatic hypotension, pure autonomic failure, and multiple system atrophy. The Consensus Committee of the American Autonomic Society and the American Academy of Neurology. *Neurol* 1996; **46**: 1470.
- Krassioukov AV, Karlsson AK, Wecht JM, Wuermsler LA, Mathias C, Marino RJ. Assessment of autonomic dysfunction following spinal cord injury: rationale for additions to the International Standards for Neurological Assessment. *J Rehabil Res Dev* 2007; **44**: 103–112.
- Biering-Sorensen F, Craggs M, Kennelly M, Schick E, Wyndaele JJ. International lower urinary tract function basic spinal cord injury data set. *Spinal Cord* 2008; **46**: 325–330.
- Biering-Sorensen F, Craggs M, Kennelly M, Schick E, Wyndaele JJ. International urodynamics basic spinal cord injury data set. *Spinal Cord* 2008; **46**: 513–516.
- Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U *et al*. The standardisation of terminology of lower urinary tract function: Report from the of the International Standardisation Sub-Committee Continence Society. *Neurourology and Urodynamics* 2002; **21**: 167–178.
- DeVivo M, Biering-Sorensen F, Charlifue S, Noonan V, Post M, Stripling T *et al*. International spinal cord injury core data set. *Spinal Cord* 2006; **44**: 535–540.
- Biering-Sorensen F, Charlifue S, DeVivo M, Noonan V, Post M, Stripling T *et al*. International spinal cord injury data sets. *Spinal Cord* 2006; **44**: 530–534.

Appendix

Autonomic Standard Assessment Form

Autonomic Standards Assessment Form

Patient Name: _____

Anatomic Diagnosis: (Supraconal , Conal , Cauda Equina)

General Autonomic Function

System/Organ	Findings	Abnormal conditions	Check mark
Autonomic control of the heart	Normal		
	Abnormal	Bradycardia	
		Tachycardia	
		Other dysrhythmias	
Unknown			
Unable to assess			
Autonomic control of blood pressure	Normal		
	Abnormal	Resting systolic blood pressure below 90 mmHg	
		Orthostatic hypotension	
		Autonomic dysreflexia	
Unknown			
Unable to assess			
Autonomic control of sweating	Normal		
	Abnormal	Hyperhidrosis above lesion	
		Hyperhidrosis below lesion	
		Hypohidrosis below lesion	
Unknown			
Unable to assess			
Temperature regulation	Normal		
	Abnormal	Hyperthermia	
		Hypothermia	
Unknown			
Unable to assess			
Autonomic and Somatic Control of Broncho-pulmonary System	Normal		
	Abnormal	Unable to voluntarily breathe requiring full ventilatory support	
		Impaired voluntary breathing requiring partial vent support	
Voluntary respiration impaired does not require vent support			
Unknown			

Lower Urinary Tract, Bowel and Sexual Function

System/Organ	Score
Lower Urinary Tract	
Awareness of the need to empty the bladder	
Ability to prevent leakage (continence)	
Bladder emptying method (specify) _____	
Bowel	
Sensation of need for a bowel movement	
Ability to Prevent Stool Leakage (Continence)	
Voluntary sphincter contraction	
Sexual Function	
Genital arousal (erection or lubrication)	Psychogenic
	Reflex
Orgasm	
Ejaculation (male only)	
Sensation of Menses (female only)	

2=Normal function, 1=Reduced or Altered Neurological Function
0=Complete loss of control NT=Unable to assess due to preexisting or concomitant problems

Urodynamic Evaluation

System/Organ	Findings	Check mark
Sensation during filling	Normal	
	Increased	
	Reduced	
	Absent	
	Non-specific	
Detrusor Activity	Normal	
	Overactive	
	Underactive	
	Acontractile	
Sphincter	Normal urethral closure mechanism	
	Normal urethral function during voiding	
	Incompetent	
	Detrusor sphincter dyssynergia	
	Non-relaxing sphincter	

Date of Injury _____

Date of Assessment _____

Examiner _____

This form may be freely copied and reproduced but not modified.

This assessment should use the terminology found in the International SCI Data Set (ASIA and ISCoS - <http://www.asia-spinalinjury.org/bulletinBoard/dataset.php>)