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Exercise Recommendations for Individuals with Spinal Cord Injury

Patrick L. Jacobs[1,2] and *Mark S. Nash*[1,3]

1 Department of Neurological Surgery, University of Miami School of Medicine, Miami, Florida, USA
2 Center of Excellence for Restoration of Function in Chronic SCI, Miami VA Medical Center, Miami, Florida, USA
3 Department of Orthopedics & Rehabilitation, University of Miami School of Medicine, Miami, Florida, USA

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Abstract

Persons with spinal cord injury (SCI) exhibit deficits in volitional motor control and sensation that limit not only the performance of daily tasks but also the overall activity level of these persons. This population has been characterised as extremely sedentary with an increased incidence of secondary complications including diabetes mellitus, hypertension and atherogenic lipid profiles. As the daily lifestyle of the average person with SCI is without adequate stress for conditioning purposes, structured exercise activities must be added to the regular schedule if the individual is to reduce the likelihood of secondary complications and/or to enhance their physical capacity. The acute exercise responses and the capacity for exercise conditioning are directly related to the level and completeness of the spinal lesion. Appropriate exercise testing and training of persons with SCI should be based on the individual's exercise capacity as determined by accurate assessment of the spinal lesion. The standard means of classification of SCI is by application of the *International Standards for Classification of Spinal Cord Injury*, written by the Neurological Standards Committee of the American Spinal Injury Association. Individuals with complete spinal injuries at or above the fourth thoracic level generally exhibit dramatically diminished cardiac acceleration with maximal heart rates less than 130 beats/min. The work capacity of these persons will be limited by reductions in cardiac output and circulation to the exercising musculature.

Persons with complete spinal lesions below the T_{10} level will generally display injuries to the lower motor neurons within the lower extremities and, therefore, will not retain the capacity for neuromuscular activation by means of electrical stimulation. Persons with paraplegia also exhibit reduced exercise capacity and increased heart rate responses (compared with the non-disabled), which have been associated with circulatory limitations within the paralysed tissues. The recommendations for endurance and strength training in persons with SCI do not vary dramatically from the advice offered to the general population. Systems of functional electrical stimulation activate muscular contractions within the paralysed muscles of some persons with SCI. Coordinated patterns of stimulation allows purposeful exercise movements including recumbent cycling, rowing and upright ambulation. Exercise activity in persons with SCI is not without risks, with increased risks related to systemic dysfunction following the spinal injury. These individuals may exhibit an autonomic dysreflexia, significantly reduced bone density below the spinal lesion, joint contractures and/or thermal dysregulation. Persons with SCI can benefit greatly by participation in exercise activities, but those benefits can be enhanced and the relative risks may be reduced with accurate classification of the spinal injury.

The human spinal cord is a complex association of upper and lower motor neurons that functions as a bidirectional conduit between the brain and its motor, sensory and autonomic targets. It also serves as a site for reflex integration between body sensors and their motor and autonomic effectors. Because spinal cord functions differ by level and structure, injury to or disease of spinal tracts results in varying types and degrees of dysfunction depending upon the specific neural structures affected.

The interruption of spinal cord functions by trauma affects 10 000 Americans annually, with an estimated 179 000 persons having survived their initial injury.[1-4] Thereafter, these individuals experience

unique physical, social and psychological changes throughout their lives, including diminished ability to perform and benefit from exercise conditioning.[5,6] The latter limitation is cause for concern as: (i) individuals with spinal cord injury (SCI) are usually young and physically active at the time of injury;[3] (ii) profound physical deconditioning is common after injury;[7-9] and (iii) physical deconditioning contributes to multisystem medical complications,[10-19] activity limitations[7,20,21] and accelerated aging.[6,7,22-26]

Many reviews and chapters published over the past decades have addressed the need for persons with SCI to adopt habitual exercise as part of a healthy lifestyle.[5,6,9,17,18,27-33] Exercise options contained in these reports have focused on use of muscle activities still under voluntary nervous system control, as well as sequenced contractions of muscles stimulated by electrical current. Outcomes of studies cited in these monographs provide credible evidence that exercise performed by persons with SCI enhances physical conditioning and reduces multisystem disease susceptibility. They further suggest that habitual exercise might reduce fatigue, pain, weakness, joint deterioration and incipient neurological deficits that appear as persons age with disability. As these deficits challenge the ability of those with SCI to perform essential daily activities first mastered after injury, their prevention will likely foster fullest health and life satisfaction for those aging with a disability.

Use of exercise as a healthy activity and lifestyle elective for those with SCI might appear fairly straightforward. To the contrary, exercise options available to those with SCI are more limited than those without disability,[33] their acute exercise and training responses less robust than those of persons without SCI,[28,34,35] and the risks of impudent activity greater and longer lasting.[33,36] This makes an understanding of neurological classification, exercise opportunities and risks of activity important if those with SCI are to benefit and not be harmed from exercise conditioning. This paper will review common medical problems experienced by persons with SCI that lend themselves to benefit from exercise conditioning, contemporary methods of injury classification, exercise options and established benefits of training, and risks imposed by inappropriate exercise recommendations for those with SCI.

1. Neurological Classification of Persons with Spinal Cord Injury (SCI)

If trauma to, or disease of, the spinal cord always resulted in its anatomical or physiological transection, classification of spinal cord dysfunctions following SCI would be relatively easy. However, the spinal cord is rarely severed unless penetrated by a bullet or severed by its bony covering during very high velocity impact. More often the cord remains anatomically intact but suffers contusion, infarction or mechanical deformation that interrupts its local or relay circuitry. It also can undergo secondary damage from inflammatory autodestruction, which explains the routine administration of high-dose corticosteroids within hours of an SCI.[37] Notwithstanding the cause of spinal damage, more than half of the survivors will experience varying degrees of motor, sensory or autonomic sparing at different spinal cord levels,[2,38,39] making classification of persons with SCI a challenging, yet important, science.[4]

To assist with accurate and consistent classification, uniform standards have been developed that allow persons with SCI to be systematically examined and classified, and to document changes in sensorimotor function that accompany the passage of time, clinical treatments or research interventions. The benchmark system used to classify persons with SCI is the *International Standards for Classification of Spinal Cord Injury* (revised 2000), which is written by the Neurological Standards Committee of the American Spinal Injury Association (ASIA), and endorsed as the recommended international standards by the International Spinal Cord Society (ISCoS; formerly the International Medical Society for Paraplegia, and hereinafter the 'ASIA Guidelines') [figure 1].[40] While a complete description of spinal column and spinal cord anatomy is beyond the scope of this review, the accurate description of an injury to a given spinal cord segment involves designation of a spinal column region

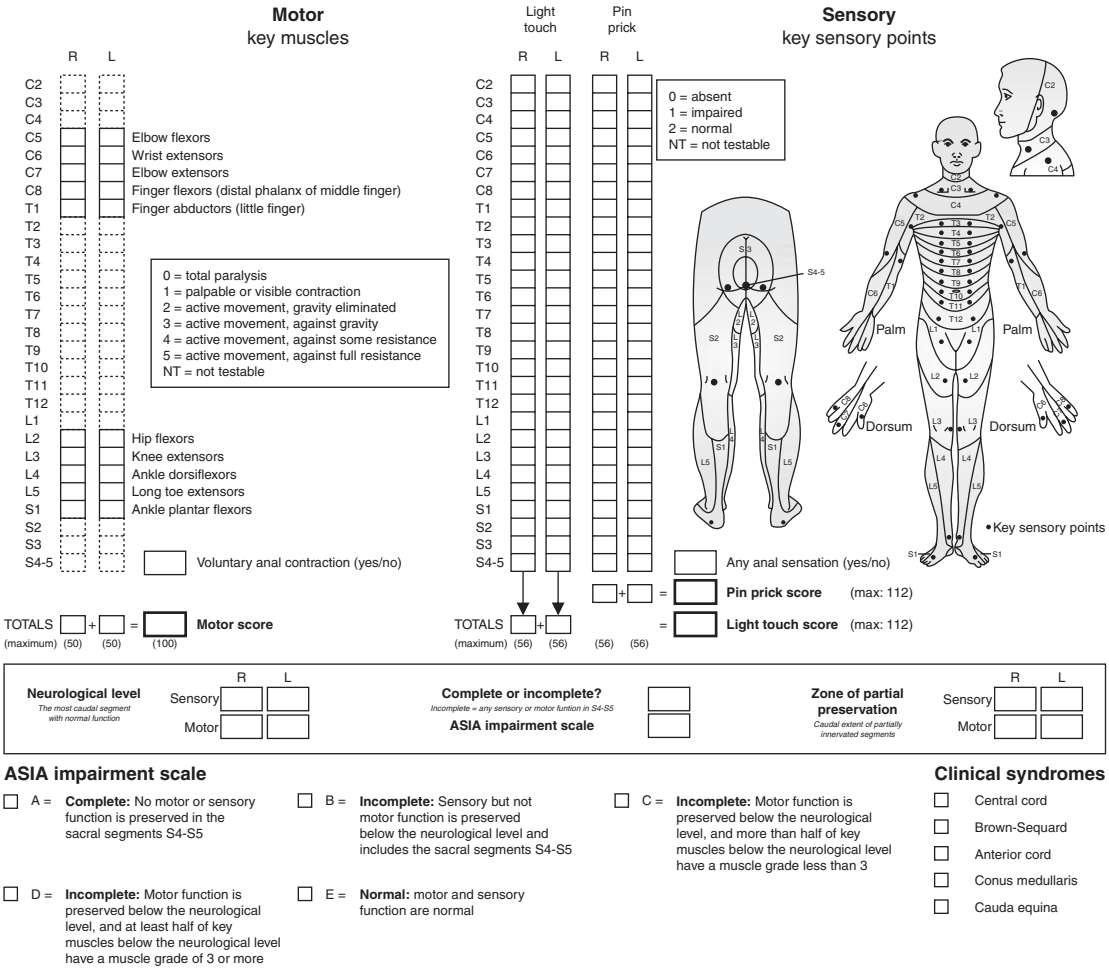

Fig. 1. American Spinal Injury Association (ASIA) classification of spinal cord injury (reproduced from the American Spinal Injury Association,[41] with permission).

(e.g. cervical, thoracic, lumber or sacral), a spinal nerve within that region, and the degree to which the injury is neurologically complete or incomplete. Should the injury result in a specific spinal cord syndrome this should also be designated.

1.1 Upper and Lower Motor Neuron Lesions

It is clinically important for those working with subjects having SCI to differentiate between upper and lower motor neuron injuries. The spinal cord is organised in such a way that either upper or lower motor neuron function, or both, can be disrupted by trauma or disease. The long tracts of the spinal cord are upper motor neurons. By convention, the lower motor neuron system begins in the anterior horn of the grey matter and exits the intervertebral foramen to join the returning sensory axons as a mixed nerve. The motor portion of this mixed nerve then travels to its target muscle(s). The returning sensory nerve fibres travel in this mixed nerve but, upon entry to the foramen, form a dorsal root that penetrates the cord in the dorsal root entry zone and synapses with the anterior horn cells of the anterior grey matter. This completes the motor-sensory reflex arc. Lesions of upper motor neurons result in 'decentralisa-

tion' of the nervous system, spastic paralysis and exaggerated sensorimotor reflexes below the injury. This means that motor, sensory and autonomic reflex activities are preserved, but no longer under command of the brain. The contrasting injury is a lower motor neuron lesion, which often accompanies SCI at the T_{10} level or lower and almost always occurs at, or caudal to, T_{12}. Persons with these lesions lose central nervous system control of sensorimotor functions, as well as sensorimotor reflex activity, which causes flaccid paralysis and areflexia ('denervation'). This explains the greater loss of lower extremity muscle mass in individuals with flaccid rather than spastic paralysis. Otherwise, injury to the cord at any site resulting in damage to the reflex arc can leave a denervated (areflexic) segment among neighbouring segments that remain spastic.

It is important to note that while spastic muscles are capable of contracting upon stimulation with transcutaneous electrical alternating currents, generally, flaccid muscles are not. This provides a means of discriminating upper from lower motor neuron lesions and also determining subject or patient candidacy for use of electrical prostheses that provide exercise or ambulation in persons with SCI.

1.2 Defining Spinal Cord Lesions: Plegias

Individuals with SCI are described as having lesions affecting either sensorimotor function of all limbs or the upper extremities alone. The former are said to have tetraplegia (preferred to 'quadriplegia'), which is defined as: "A term referring to impairment or loss of motor and/or sensory function in the cervical segments of the spinal cord due to damage of neural elements within the spinal canal. Tetraplegia results in impairment of function in the arms as well as in the trunk, legs and pelvic organs. It does not include brachial plexus lesions or injury to peripheral nerves outside the neural canal."[42,43] Paraplegia is defined as: "A term referring to impairment or loss of motor and/or sensory function in the thoracic, lumbar or sacral (but not cervical) segments of the spinal cord, secondary to damage of neural elements within the spinal canal. With paraplegia, arm functioning is spared, but, depending on the level of injury, the trunk, legs and pelvic organs may be involved. The term is used when referring to cauda equina and conus medullaris injuries, but not to lumbosacral plexus lesions or injury to peripheral nerves outside the neural canal."[42,43]

1.3 Neurological, Sensory and Motor Levels

Neurological level refers to the most caudal segment of the spinal cord with normal sensory and motor function on both sides of the body. Because the results of sensorimotor testing often differ by side, up to four different segments may be identified in determining the neurological level, i.e. R-sensory, L-sensory, R-motor and L-motor. In such cases it is strongly recommended that each of these segments be separately recorded and that a single 'level' not be used, as this can be misleading. When the term 'sensory level' is used, it refers to the most caudal segment of the spinal cord with normal sensory function on both sides of the body; the 'motor level' is similarly defined with respect to motor function. These 'levels' are determined by neurological examination of: (i) a key sensory point within each of 28 dermatomes on the right and 28 dermatomes on the left side of the body;[42] and (ii) a key muscle within each of ten myotomes on the right and ten myotomes on the left side of the body. Assessment of sensory and motor level is key in those with paraplegia having injuries from T_1 through T_8, whereas motor function is the determining factor in classification of tetraplegia.

1.4 Complete and Incomplete Spinal Cord Lesions

Confusion and imprecise language surround the definitions of neurological completeness and incompleteness. Historically, the Functional Classification scale of Frankel et al.[44] was used to assign a grade of SCI from A to E. These grades were assigned on the basis of sensorimotor sparing below the level of SCI. In contrast, the ASIA scale uses 'sacral sparing' as the criterion for determining neurological completeness.[39,42,45] This requires a test of motor function examining the presence of voluntary contraction of the external anal sphincter upon digital

examination. The ASIA definitions of complete and incomplete lesions are:

- 'Complete injury': a term describing absence of sensory and motor function in the lowest sacral segment.[42]
- 'Incomplete injury': a term describing partial preservation of sensory and/or motor functions below the neurological level *and* including the lowest sacral segment. Sacral sensation includes sensation at the anal mucocutaneous junction as well as deep anal sensation.[42]

Both ASIA and Frankel methods are useful for describing completeness of injuries, as an individual with infralesional sensory sparing (i.e. incomplete sensory loss; Frankel Grade B) can still be neurologically complete when using the ASIA guidelines. Also, the ASIA C classification is so broad as to sometimes limit useful classification of motor and functional abilities of patients with SCI that may be better understood when using Frankel grades.

Accurate descriptions of complete and incomplete lesions are often confounded by recovery of sensorimotor function within weeks or months after injury, which is not uncommon for some individuals initially presenting without preserved reflex or sensorimotor functions.[46] This recovery, however, will more likely occur in individuals with very low velocity injuries in which cord bleeding or anatomic disruption of neural elements are limited. Several mechanisms have been postulated to explain 'return of function' after SCI. The first involves a belief that a 'spinal shock' occurs immediately upon SCI in some individuals – a state of areflexia thought to result from hyperpolarisation of cord neurons accompanying injury-related loss of descending facilitation.[47] Thereafter, those with incomplete lesions often recover reflexes in a caudal to rostral sequence, although the extent of reflex recovery has questionable value in predicting eventual patterns or extent of motor recovery.[47] Otherwise, post-synaptic receptor up-regulation and synaptic growth have been postulated as explanations for neuronal recovery following SCI,[48,49] although the precise mechanisms promoting these changes remain incompletely understood. Notwithstanding the controversial explanations for resolution of 'spinal shock' and recovery of sensorimotor functions, the designation of 'neurological completeness' within the first year of injury leaves open the possibility for erroneous classification of a subject thereafter. This explains why many investigators use the first anniversary of injury as both a benchmark for neurological stability and a criterion for enrolment of subjects with SCI in research studies, especially when sensorimotor function is under study. Clearly, however, the use of the term 'complete' written on an initial trauma examination might ultimately have little bearing on whether recovery of reflexes or sensorimotor function might result. Thus, examination and classification at the beginning of exercise testing and training is needed.

1.5 SCI Syndromes

As the various spinal columns transmit different types of information, and because most persons surviving SCI do not experience complete severance of their cords, many different patterns of spared sensorimotor function are possible. The syndromes that ensue are usually described when defining the levels and completeness of injury. The major syndromes, their definitions and causations are listed in the following sections.

1.5.1 Anterior Cord Syndrome

An anterior cord syndrome is "a lesion that produces variable loss of motor function and of sensitivity to pain and temperature, while preserving proprioception".[42] An anterior cord syndrome results primarily in a profound motor loss below the level of injury. Destruction of the anterior portions of the white and grey matter of the spinal cord affects the corticospinal motor tracts and, to a lesser extent, sensory tracts that mediate light touch and pressure.

1.5.2 Central Cord Syndrome

A central cord syndrome is "a lesion occurring almost exclusively in the cervical region, that produces sacral sensory sparing and greater weakness in the upper limbs than in the lower limbs".[42] A person with a central cord syndrome often presents

with greater motor impairment of the upper than lower extremities if the damage or disease is primarily limited to the central grey and white spinal cord matter. Because upper extremity motor function is primarily activated by the medial corticospinal tracts, sensorimotor functions of the lower extremities are usually affected to a lessor degree. Central cord injury is common among individuals having congenitally narrow spinal canals and in those, especially aging persons, whose osteoarthritic pathology results in spinal canal stenosis.

1.5.3 Brown-Sequard Syndrome

A Brown-Sequard syndrome is "a lesion that produces relatively greater ipsilateral proprioceptive and motor loss and contralateral loss of sensitivity to pain and temperature".[42] The Brown-Sequard syndrome results from an anterior-posterior hemisection of the spinal cord often accompanying a penetrating wound that severs the neural elements. When the elements of only one lateral portion of the spinal cord are destroyed, the injury results in an unusual pattern of sensorimotor function with a loss of infralesional motor function, proprioception, fine touch and vibration discrimination on the same side of injury, but loss of pain, temperature, crude touch and deep pressure on the opposite side of injury. The syndrome is explained by decussation of motor tracts in the brain stem before they descend in the cord. In contrast, ascending sensory tracts of the anterolateral systems cross to the opposite side at (or near) the level in which the dorsal roots enter the cord. This explains why 'same side' motor deficits are common in patients with Brown-Sequard syndrome, but sensory preservation is greater on the contralateral side of injury.

1.5.4 Conus Medullaris and Cauda Equina Syndrome

A conus medullaris syndrome involves an "injury of the sacral cord (conus) and lumbar nerve roots within the spinal canal, which usually results in an areflexic bladder, bowel and lower limbs".[42] Sacral segments may occasionally show preserved reflexes (e.g. bulbocavernosus and micturition reflexes). A cauda equina syndrome involves "injury to the lumbosacral nerve roots within the neural canal resulting in areflexic bladder, bowel and lower limbs".[42]

2. Medical and Health Consequences of SCI

Persons with SCI face unique health challenges throughout their lives, and their injuries dissociate the normally well integrated homoeostatic responses of body systems known to accompany physical activity. Nervous system damage disrupts to varying degrees the necessary signal integration among motor, sensory and autonomic targets, and thus has a profound effect on fitness, exercise capacities and health. Depending on the level and type of cord lesion, persons with SCI are among the most physically deconditioned of all humans.[7,50] Not surprisingly, young persons with chronic SCI experience accelerated pathological states and conditions normally associated with physical deconditioning and premature aging, including: dyslipidaemias and heart disease;[23,24,51,52] arterial circulatory insufficiency[53-57] and clotting disorders;[58,59] bone and joint diseases;[60-62] and pain of musculoskeletal and neuropathic origins.[63-68]

Notwithstanding their physical limitations, many persons with SCI can still undergo exercise reconditioning. Those who retain upper extremity function can participate in a wide variety of exercise activities and sports,[6,69] and ambulate with the assistance of orthoses and computer-controlled electrical neuroprostheses.[70-76] Individuals with upper motor neuron lesions can pedal cycle ergometers by surface electrical stimulation of selected lower extremity muscle groups under computer control.[30,31,33] Many body organs and tissues acutely respond to exercise despite their decentralised or denervated states, and because many survivors of SCI experience complete sensory loss or significantly diminished nociceptive responses, electrically stimulated muscle contractions can often be utilised without pain.

2.1 Physical Deconditioning

Persons with SCI usually live sedentary lives,[7,9,68,77] which explains their poor physical fit-

ness and heightened risk of cardiovascular morbidity and mortality.[9,22,51,78] Nearly one in four healthy young persons with paraplegia fails to achieve levels of oxygen consumption ($\dot{V}O_2$) on an arm exercise test sufficient to perform many essential activities of daily living.[8] While those with paraplegia have far greater capacities for activity and more extensive choices for exercise participation than persons with tetraplegia, they are only marginally more fit.[7,11]

2.2 Musculoskeletal Decline

Altered structural and contractile properties of muscle after SCI limit the ability of totally paralysed and weakened muscle to sustained intense contractions for extended durations. Most studies of sublesional muscle after SCI in humans report fibres that:

- are smaller than those above the lesion and those of persons without SCI;[79-83]
- have less contractile protein;[84]
- produce lower peak contractile forces;[85,86]
- transform toward fast phenotypic protein expression;[81,87-90]
- increase myosin heavy chain isoforms;[90,91]
- decrease their resistance to fatigue.[19,79,84,92,93]

Muscle fibre cross-sectional area declines within 1 month of SCI,[80,89] while electrical stimulation of muscle paralysed for more than 1 year evokes forces only one-seventh to one-third those of subjects without SCI.[85,86,94] Physiological and contractile properties adopted after SCI further compromise the ability of muscle without motor sparing to optimally increase in size and metabolic activity after exercise reconditioning,[95] which has adverse consequences for cardiovascular, musculoskeletal and comorbid diseases to which those with SCI are highly susceptible.

Persons sustaining SCI develop muscles below the level of the lesion that are either hypertonic or atonic depending on the level and type of SCI. Hypertonia is the more common condition in which exaggerated rate-dependent stretch results in spastic contraction. This results from damage to the upper motor neurons alone, in which descending inhibition of reflex response in interrupted by cord injury. Hypertonia in these individuals can by worsened by a number of 'noxious' stimuli unrelated to muscle stretch, including urinary voiding, venous thrombosis, thermal dysregulation, occult fracture or infection.[96] In contrast, damage to lower motor neurons involving injury below T_{10} usually results in flaccid paralysis and loss of neuromuscular response to administration of alternating electrical currents.

Rapid bone demineralisation is expected during the first year after SCI, after which bone density levels continue to slowly decay. Increased urinary excretion of calcium and hydroxyproline,[97]and progressive rarefying of bone on radiographs are evident throughout this period. About one-third to one-half of bone mineral density is lost by 1 year after injury, with primary losses occurring in the supracondylar femur. The inevitable course of SCI leads to underhydroxylated and hypocalcific bone[98,99] with permanently heightened susceptibility to fracture, even following trivial or imperceptible trauma.[100] Joints experience similar deterioration[60] and heightened injury susceptibility brought on by cartilage atrophy[101] and joint space deformities.[102-104]

2.3 Cardiovascular Disease, Dyslipidaemia and Comorbidities

Epidemiological studies conducted in the early 1980s, and thereafter, reported emergence of cardiovascular disease as a major cause of death in persons with SCI.[1,3,24,105] While genitourinary complications accounted for 43% of deaths in the 1940s and 1950s, mortality from these causes were reduced to 10% of cases in the 1980s and 1990s.[24,106,107] Cardiovascular diseases currently represent the most frequent cause of death among persons surviving >30 years after injury (46% of deaths) and among persons >60 years of age (35% of deaths)[107,108]

Decline of cardiovascular function in persons aging with SCI mirrors that experienced by persons who age without SCI, although at an accelerated rate.[23] Asymptomatic cardiovascular disease also occurs at earlier ages after SCI[108,109] and its symptoms may be masked by interruption of ascending afferent pain fibres conveying warnings of impending heart damage or death.[108-110] Several major risk

factors commonly reported in persons with SCI have been linked with their accelerated course of cardiovascular disease; these include dyslipidaemia[7,22,78] and a sedentary lifestyle imposed by muscle paralysis and limited exercise options.[8,9] The cardiovascular disease risks of individuals without SCI are worsened by hyperinsulinaemia[22,111,112] and elevated percentages of body fat,[113,114] which are common among persons with SCI.[24]

An atherogenic lipid profile is commonly reported in persons with chronic SCI.[22,78,112-115] This lipid profile satisfies criteria for designation as 'dyslipidaemic', i.e. elevated total cholesterol (TC), triglyceridaemia and plasma low density lipoprotein-cholesterol (LDL-C) or depressed high density lipoprotein-cholesterol (HDL-C).[116] All have all been reported in sedentary persons with SCI, although elevated TC is not observed in all of these studies,[115,117] and LDL-C concentrations, while sometimes elevated, show patterns of elevation typical of those reported in the general population.[115] The most consistent finding of reports examining the lipid profiles of persons with SCI is a depressed blood plasma concentration of the cardioprotective HDL-C[22,78,114,115,118,119] whose levels are inversely associated with cardiovascular risk.[120] More than 40% of persons with SCI have HDL-C levels below the earlier criterion score for high cardiovascular risk (HDL >35 mg/dL). When combined with other known risks factors for vascular disease in persons with SCI (e.g. prevalent truncal obesity,[114] elevated body mass indices,[113] physical inactivity[8,9] reduced lean body mass,[95,113,121-123] diabetes,[124,125] metabolic syndrome X[126] and advancing age),[18] the risks of abnormal lipid profile on disease progression become magnified.

Insulin resistance occurring in a high percentage of persons with SCI was first reported more than two decades ago.[125] Since that time, others have confirmed this finding and included insulin resistance among the cardiovascular risks sustained by persons aging with SCI.[22,111,112,114,127] Almost half the persons with SCI live in a state of carbohydrate intolerance or insulin resistance.[22,125] A reason for prevalent insulin resistance in persons with SCI have not been firmly identified, although physical inactivity,[127] truncal obesity[113,114] and sympathetic dysfunction[111,112] have been suggested as causes. An association may also exist between their abnormal lipid profiles and insulin resistance. Such an association has been identified in persons without SCI having depressed serum HDL-C, as they are also especially prone to insulin resistance.[128-130] This risk profile closely matches that of persons with SCI, in whom isolated low HDL-C and insulin resistance are often comorbid.[51]

2.4 Cardiac Structure and Function

Individuals with chronic SCI experience various types of circulatory dysregulation depending on the level of their cord lesion.[96] When injury occurs above the level of sympathetic outflow at the T_1 spinal level, resting hypotension with mean arterial pressures of 70mm Hg are common.[131] In addition to challenging effective orthostatic pressure regulation, low pressures also instigate altered heart structure and function observed after SCI.[132] As size and architecture of the human heart are known to be influenced by peripheral circulatory volume and systemic pressures, withdrawal from normal activity levels and altered circulatory dynamics transform the structure of the heart and alter its pumping efficiency.[133] For those with tetraplegia, a chronic reduction of cardiac preload and myocardial volume coupled with pressure underloading causes the left ventricle to atrophy.[132,134] In contrast, long-term survivors of paraplegia are normotensive and have normal left ventricular mass and resting cardiac output, but experience a cardiac output comprised of elevated resting heart rate (HR) and depressed resting stroke volume (SV).[28,134] This lowered SV is attributed to decreased venous return from the immobile lower extremities or frank venous insufficiency of the paralysed limbs.[135,136]

2.5 Vascular Structure and Function

The volume and velocity of lower extremity arterial circulation is significantly diminished after SCI, with volume flow of about half to two-thirds that reported in matched subjects without paraly-

sis.[56,137] This circulatory 'hypokinesis'[138] results from loss of autonomic control of blood flow as well as diminished regulation of local blood flow by vascular endothelium.[56] The lowering of volume and velocity contribute to heightened thrombosis susceptibility commonly reported in those with acute and subacute SCI.[58] A contributing factor to thrombosis disposition appears to be a markedly hypofibrinolytic response to venous occlusion of the paralysed lower extremities,[139] a poor response likely attributable to low blood flow conditions in the paralysed lower extremities[54,56] or interruption of adrenergic pathways that regulate fibrinolysis in the intact neuraxis.[59]

2.6 Autonomic Dysregulation

Like those of the motor system, sympathetic nerve tracts descend in the spinal cord, albeit within the intermediolateral columns and not the corticospinal tracts, and exit with motor nerves in the thoracolumbar segments. As no sympathetic autonomic tracts exit the cord above the T_1 spinal level, individuals with cervical injuries often sustain decentralisation of their sympathetic nervous system. Loss of autonomic outflow to the adrenals is also observed in persons with paraplegia above the T_6 spinal level. Injury above the sacral cord segments abolishes central parasympathetic regulation of genitourinary organs (S2-4), which explains the common occurrences of neurogenic bowel and bladder after SCI, as well as erectile dysfunction in males.[140] Autonomic dysfunction that results from injury above the thoracolumbar levels of sympathetic nerve outflow is associated with cardiac and circulatory dysfunction,[34,132] clotting disorders,[58] altered insulin metabolism,[112,125] resting and exercise immunodysfunction,[65,141-151] orthostatic incompetence,[152] osteoporosis and joint deterioration,[60,153] and thermal dysregulation at rest and during exercise.[154-157] A blunted chronotropic response to exercise in persons with tetraplegia is well documented,[158-160] and usually yields peak HRs in the mid-120 beats/min range, similar in magnitude to persons without SCI who exercise under conditions of pharmacological adrenergic blockade. The absence of, or meagre catecholamine responses to, exercise in these individuals have been reported[161] and explains their suppressed HR responses to exercise. Thus, cardiac acceleration during exercise implies withdrawal of vagal tone and not increased sympathetic drive.

Few direct clinical comparisons can be made between persons having SCI above and below the highest level of sympathetic outflow at T_1 (i.e. between those persons whose motor status results in tetraplegia and paraplegia). Testing of subjects having inhomogeneous lesion levels usually leads to widely variable chronotropic, pressor, fuel, peripheral circulatory, thermal and work capacity responses. For those with paraplegia from T_2 to T_5 (or T_6), sparing of sympathetic efferents to the heart with resulting noradrenergic-mediated cardiac acceleration will be observed. A relatively normal response is observed below T_5 or T_6,[162] as central inhibitory control of the adrenals (normally innervated from T_6–T_9) is regained below these levels.[158]

3. Volitional Upper Extremity Exercise in Persons with SCI

3.1 Acute Physiological Responses to Exercise Stress

SCI blunt the expected physiological responses to exercise and diminish peak exercise capacity.[163] These limitations are associated with the level of SCI, and are explained by various factors. First, ascending levels of injury cause greater loss of muscle mass necessary to serve as prime movers and stabilisers of trunk position. This requires that the arms simultaneously generate propulsive forces and steady the trunk during exercise. Secondly, increasing levels of injury are associated with increasing adrenergic dysfunction, and at key spinal levels dissociate adrenal, cardiac and total sympathetic nervous system control from the brain. As the adrenergic and noradrenergic systems play key roles in cardiovascular regulation and fuel homeostasis during exercise, their loss attenuates the cardiovascular

and metabolic efficiencies achieved in persons with an intact neuraxis.

Many studies have observed a direct association among level of injury, peak workload attained and peak oxygen uptake ($\dot{V}O_{2peak}$) reached during arm crank testing. This association serves as the basis for sports classification used during paralympic and other sports events for persons competing with physical disability. Exercise performance after SCI is limited by circulatory dysregulation accompanying thoracic injuries.[138,156,159,164-166] Individuals with injuries below the level of sympathetic outflow at T_6 have a significantly lower resting SV and higher resting HR than persons without paraplegia.[156,159,167] The significant elevation of resting and exercise HR is thought to compensate for a lower cardiac SV imposed by pooling of blood in the lower extremity venous circuits, diminished venous return and cardiac end-diastolic volumes, or frank circulatory insufficiency.[138,168] Adjustments to the adrenergic systems after SCI may also regulate the excessive chronotropic response to work, as higher resting catecholamine levels and exaggerated catecholamine responses to physical work have been reported in individuals with paraplegia with middle thoracic (T_5) cord injuries. These exceed resting and exercise levels of both high level paraplegics and healthy persons without SCI.[155,169] Hypersensitivity of the supralesional spinal cord is believed to regulate this atypical adrenergic state and dynamic, which contrasts the downregulation of adrenergic functions observed in persons with high thoracic and cervical cord lesions.[155,161]

An exaggerated HR response is experienced during physical activity by persons with paraplegia,[158] which may be performance delimiting. This belief is consistent with reports in which subjects with paraplegia require higher levels of $\dot{V}O_2$ to perform the same work intensity as subjects without SCI[32,138,156,170] and may represent a limiting factor in the performance of activities of daily living.[21,156,171] As the sympathetic nervous system regulates haemodynamic and metabolic changes accompanying exercise, the elevated $\dot{V}O_2$ and HR response to work in individuals with paraplegia with injuries below T_5 may be due to adrenergic overactivity accompanying their paraplegia.[158,161,169]

3.2 Cardiorespiratory Testing

Assessment of cardiorespiratory fitness in persons with SCI requires specialised knowledge of both exercise testing procedures and the unique physiology that ensues SCI. Exercise testing modalities for these individuals commonly employ overground wheelchair propulsion on a treadmill or wheelchair rollers, cyclical arm ergometry and variations of electrically stimulated exercise. Exercise testing by wheelchair propulsion offers the advantage of comparison to many wheelchair-based daily tasks. However, wheelchair propulsion testing generally requires specialised equipment. Also, it is not known whether testing is best when using a single standardised wheelchair, in which the usual wheelchair-used interface is compromised, or having subjects use their habitual chair, in which some individuals may be advantaged by better equipment. These factors limit use of wheelchair ergometry as a research and clinical testing tool.

The exercise test mode most commonly used in the cardiorespiratory testing of persons with SCI is the arm crank ergometer. The lack of task specificity to wheelchair mobility has been considered a limitation to their usefulness. However, as these devices are generally found in the rehabilitation environment and allow standardised application of testing stresses, it represents the most frequently used means for clinical or fitness assessment of wheelchair users. Reliable test results require consistent positioning of the test subject and the ergometer. The test subject and the cranking ergometer should be positioned so that the axis of the crank arm is horizontally aligned with the subject's shoulder joint and the elbows are slightly flexed at the point of furthest reach. To ensure enhanced reliability of test results, care should be taken to replicate subject positioning and to use the same wheelchair and cushion. The latter may affect seating stability and exercise efficiency.

Cardiorespiratory exercise testing of persons with SCI follows the same general protocols as

testing in the general population. In most cases, the graded multi-stage tests proceed in 2- to 3-minute stages until volitional exhaustion. As $\dot{V}O_{2peak}$ is dependent on level of SCI, lower intensity loads are applied than those used in standard exercise testing. These workloads have been detailed in several publications,[172-175] although no particular advantage for a single testing strategy has been tested or established. However, accurate and valid results require that testing utilise metabolic monitoring to establish the $\dot{V}O_{2peak}$, as prediction equations normally used to estimate maximal responses in persons without disability have not been validated in those with SCI.

3.3 Endurance Training

In most cases, injury to the spinal cord renders the lower extremities without sufficient strength, endurance or motor control to support safe and effective physical training. This explains why most exercise training after SCI employs upper extremity exercise modes including arm crank ergometry, wheelchair ergometry and swimming. All of these training modes improve $\dot{V}O_{2peak}$ in those with SCI,[27,32,172,176-186] with the magnitude of improvement inversely proportional to level of spinal lesion. While it is possible for persons with low tetraplegia to train on an arm ergometer, special measures must be taken to affix the hands to an ergometer and their gains in $\dot{V}O_{2peak}$ fail to match those of their SCI counterparts with paraplegia.[187] Therefore, the level of injury is a key to predicting outcome from arm endurance training.[188,189] Guidelines for training after SCI have been published by several authorities,[32,52,172] although testing for superiority of training algorithms has not been performed. However, given that most persons with SCI are sedentary long after injury and the accepted dictum that training best benefits the most deconditioned individuals, these methods are nonetheless successful in achieving higher levels of fitness.

Benefits of arm conditioning are widely reported for persons with SCI, with many studies reporting significant increases in $\dot{V}O_{2peak}$ after training.[27,185-187,190,191] It is less clear whether the changes in $\dot{V}O_{2peak}$ result from central (HR, SV, cardiac output [$\dot{Q}$]) adaptations or increases in peripheral oxygen extraction (a-$\dot{V}O_2$ difference), or whether central adaptations take place at all. Only one endurance training study examining 16 weeks of intense arm exercise observed clear evidence of central cardiovascular benefit.[176] Notwithstanding the mechanisms contributing to increased $\dot{V}O_{2peak}$ after arm exercise training, many studies report individuals with paraplegia improve their $\dot{V}O_{2peak}$ by 10–20% following 8–12 weeks of training. These gains are inversely proportional to the level of SCI, as those with higher levels of injury attain lower peak exercise capacities on arm exercise test than those with lower-level SCI, and both groups lower than persons without disability tested by arm ergometry.

Endurance training recommendations for those with SCI do not vary dramatically from advice directed to the general population.[192-194] The American College of Sports Medicine recommended training frequencies, durations and intensities are contained in *Exercise Management for Persons with Chronic Diseases and Disabilities*.[192] Generally, three to five weekly exercise sessions of 20–60 minutes in duration and at an intensity of 50–80% $\dot{V}O_{2peak}$ is the recommended exercise prescription for persons with paraplegia using the following modes of exercise:

- arm cranking
- wheelchair propulsion
- swimming
- wheelchair sports
- circuit resistance training (CRT)
- electrically stimulated cycling
- electrically stimulated walking.

This reference suggests that target exercise intensities should produce HR responses equivalent to 50–80% of the individual's peak HR. As in the general population, excessive exercise volume has been associated with increase pain and injury. Thus, exercise frequency and duration should be carefully monitored and gradually increased with the input of certified exercise professionals familiar with the unique physiological responses of persons with SCI to exercise. As long-term compliance and injury

avoidance are major goals of training, erring on the conservative side of selected exercise durations and intensities are prudent and more important for persons training with a disability than those without.

3.4 Resistance Training for Persons with SCI

Far less is known about effects of resistance than endurance training in persons with SCI. This might seem counterintuitive, as muscle weakness has been reported to precipitate pain and dysfunction as persons with SCI age with their disability. Moreover, the most commonly reported symptom of upper extremity physical dysfunction among persons with SCI is pain of the shoulder joint and girdle.[66,195-200] While a single cause for shoulder pain has not been identified, deterioration and injury resulting from insufficient shoulder strength is one commonly cited source.[63,199-202] Pain that accompanies wheelchair locomotion and other wheelchair activities reportedly interferes with functional activities including upper extremity weight bearing for transfers, high resistance muscular activity in extremes of range of motion, wheelchair propulsion up inclines and frequent overhead activity.[196,198-200] Onset of pain is common during body transfer activities and severity increases as time following injury lengthens.[196] Given evidence that wheelchair locomotion is a major source of pain and dysfunction for persons with SCI, incorporation of resistance training into the healthcare plan of those with SCI appears both justified and essential.

Several investigators have studied effects of resistance exercise in persons with paraplegia. In a study of Scandinavian men (most of whom had incomplete low thoracic lesions) a weight-training programme emphasising triceps strengthening for crutch walking was undertaken for 7 weeks with modest but significant increases in $\dot{V}O_{2peak}$ observed following training. These endurance gains were accompanied by increased strength of the triceps brachii.[203] Another study[204] examined the effects of arm ergometry in subjects assigned to high-intensity (70% of their $\dot{V}O_{2peak}$) or low-intensity training (40% of their $\dot{V}O_{2peak}$) for 20 or 40 minutes per session, respectively. Strength gains were limited to subjects assigned high-intensity training, and occurred only in the shoulder joint extensor and elbow flexor muscles. Otherwise, no changes in shoulder joint abduction or adduction strengths were reported, and none of the muscles that move or stabilise the scapulothoracic articulation or chest were stronger following training. These results suggest that arm crank exercise is ineffective as a training mode for upper extremity strengthening because it fails to target the muscles most involved in performance of daily activities.

Conditioning of five paraplegic and five tetraplegic subjects three times weekly for 9 weeks was reported using a hydraulic fitness machine.[205] Exercises were limited to two manoeuvres: (i) chest press/chest row; and (ii) shoulder press/latisimus pull.[205] Significant increases in peak values of $\dot{V}O_2$ and power output measured by arm ergometry testing were observed at the conclusion of the study, although no testing was conducted that directly measured strength gain in any muscle groups undergoing training. Another study focused their training on strengthening of the scapular muscles, although this study focused solely on the scapular retractor muscles when comparing seated rowing and a standardised scapular retraction exercise, and did so only for concentric actions.[206] The authors found that higher levels of retractor activation were obtained during backward compared with forward wheelchair locomotion and suggested that rowing was effective for improving scapular retractor activity and cardiorespiratory fitness. A recent study observed reduced shoulder pain following a series of shoulder resistance exercises using elastic bands.[63]

As both endurance and resistance exercise benefit those without SCI, the effects of CRT[207] on various attributes of fitness, dyslipidaemia and shoulder pain have been studied in young and middle-aged subjects with paraplegia. This exercise programme incorporates periods of low-intensity high-paced movements interposed within activities performed at a series of resistance training stations. The CRT exercise programme adapted for persons with paraplegia consisted of three circuits of six resistance stations encompassing three pairs of agonist/

antagonist movements (e.g. overhead press and pull) and three 2-minute periods of free wheeling arm cranking performed between resistance manoeuvres. No true rest periods were allowed during the performance of CRT, with active recovery limited to the time necessary for the subject to propel the wheelchair to the next exercise station. Three weekly sessions were completed with each session lasting approximately 45 minutes. Young subjects undergoing 16 weeks of mixed resistance and endurance exercise increased their arm $\dot{V}O_{2peak}$ by an average of 29%, with accompanying strength gains of 13–40%, depending on the upper extremity site tested.[174] Subjects undergoing CRT lowered their total and LDL-C while increasing their HDL-C by nearly 10%.[175] Subjects aged over 40 years undergoing the same treatment for 12 weeks experienced significant gains in all of endurance, strength and anaerobic power, even though the latter was not specifically targeted by training (Nash MS, unpublished data). Shoulder pain present in these subjects before training was assessed by a pain instrument validated in the population and significantly reduced. Pain was eliminated in four of ten subjects.

In a separate study, the effects of circuit training performed on an adapted multi-exercise system could be replicated by use of elastic bands.[208] Evidence therefore supports health and fitness advantages of CRT over either endurance or resistance exercises alone for persons with paraplegia.

General recommendations for strength training exercise prescription include three sets of 8–12 repetitions per exercise movement for two sessions per week at moderate to high intensity using the following modes of exercise:

- free weights
- weight machines
- elastic tubing and bands.

4. Electrically Stimulated Exercise in Persons with SCI

4.1 Basis of Electrically Stimulated Movement

Many forms of electrically stimulated exercise are available for use by persons with SCI. These include site-specific stimulation of the lower extremities[82,209-212] and upper extremities,[65,213-218] leg cycling,[30,34,134,219-222] leg exercise with upper extremity assist,[222-225] lower body rowing,[50] electrically assisted arm ergometry,[226,227] electrically stimulated standing[76,217] and electrically stimulated bipedal ambulation either with[74-76,228,229]or without an orthosis.[55,70,72,173,230-232] Most forms of exercise require that the lower motor neuron system remain intact following injury, as direct activation of the peripheral nerve and not the muscle by electrical current ultimately results in muscle activation.[233] This reality excludes most individuals having cauda equina or conus medullaris syndromes from electrically stimulated exercise. It may also compromise the efficiency of a muscle having a denervated segment from injury to its anterior horn cells or spinal (Wallnerian) degeneration from injured adjacent spinal areas. Many applications of SCI target muscle strengthening of limb segments whose motor function is partially spared by injury,[76] while others use electrical current as a neuroprosthesis for the lower extremities[71,229] and upper extremities.[234-238]

Most electrical stimulation devices currently approved for use by the US FDA employ surface, not implanted, electrical stimulation. The most common uses of surface electrical stimulation for system exercise in those with SCI include electrically stimulated cycling, either with or without upper extremity assistive propulsion and electrically stimulated ambulation. Qualifications necessary to safely participate in exercise programmes have been described[71,72,233,239,240] and risks of participation are included in the section of this monograph addressing adverse events associated with exercise by those with SCI.

4.2 Leg Cycling Exercise

The simplest form of multi-limb segmental exercise for persons with SCI employs a cycling motion performed in a slightly recumbent seated position. This electrically stimulated cycling uses electrically activated contractions of the bilateral quadriceps, hamstrings and gluteus muscles under computer microprocessor command.[31] Control of pedal ca-

dence and muscle stimulation intensity is exerted by feedback provided from position sensors placed in the pedal gear.[241]

Training with electrically stimulated cycling is often preceded with electrically stimulated strengthening of the quadriceps muscles, which is necessary in cases of severe muscle atrophy or diminished muscle endurance.[209,221] Responses to electrically stimulated exercise are variable, ranging from poor to robust. As noted, poor muscle strength and endurance and altered muscle contractile properties normally accompany SCI. In general, these factors will slow success in training, especially in those individuals with longstanding paralysis, absent spasticity and flexor patters of spasticity. Electrically stimulated strength training programmes are capable of altering the strength, resistance to fatigue and contractile properties of the muscles undergoing the training. Dudley and associates,[209] demonstrated the effectiveness of a simple programme of electrically induced knee extensions, performed twice weekly over an 8-week period, dramatically reversed muscular atrophy of the quadriceps muscles.

Notwithstanding poor levels of muscle strength and endurance early in most training programmes and despite limited ability to exercise against intense workloads, enhanced levels of fitness,[224,242,243] improved gas exchange kinetics[244,245] and increased muscle mass[218] have been reported following exercise training using electrically stimulated cycling. For those with neurologically incomplete injuries, gains in lower extremity mass, as well as isometric strength and endurance under conditions of voluntary and electrically stimulated cycling have been reported.[218] Reversal of the adaptive left ventricular atrophy reported in persons with tetraplegia has also been observed, with near normalisation of pretraining cardiac mass.[134] This change may be associated with significantly improved lower extremity circulation following training,[246,247] which is also accompanied by a more robust hyperaemic response to experimental occlusion ischaemia.[55,56]

Attenuation of paralytic osteopaenia has been observed by several investigators[248,249] and an increased rate of bone turnover by another,[10] although the sites benefiting from training are the lumbar spine and proximal tibia, not the proximal femur.[249] Not all studies have found a post-training increase in mineral density for bones located below the level of the lesion.[250] Those that fail to do so have usually studied subjects with longstanding paralysis, in which attenuation or reversal of osteopaenia by any treatment has yet to be reported. Notwithstanding, a study examining the appearance of lower extremity joints and joint surfaces using magnetic resonance imaging reported no degenerative changes induced by cycling, and less joint surface necrosis than previously reported in sedentary persons with SCI.[60] Improved body composition favouring increased lean mass and decreased fat mass[251] and an enhancement of whole-body insulin uptake, insulin-stimulated 3-0-methyl glucose transport and increased expression of GLUT4 transport protein in the quadriceps muscle have been reported.[252] In a recent report, this training has resulted in improved profiles of insulin resistance.[253] When combined with simultaneous upper extremity arm ergometry, the acute cardiovascular metabolic responses to electrically stimulated cycling are more intense and the gains in fitness greater than observed with lower extremity cycling alone.[224,253]

4.3 Bipedal Ambulation

Complex electrical stimulation to achieve bipedal ambulation has been used as a neuroprosthesis for those with motor-complete injuries[71,72,228,239,254] and an assistive neuroprosthesis for persons with incomplete SCI who lack necessary strength to support independent ambulation.[255-263] Surface and implantable neuroprostheses for those without spared motor function have both been fabricated,[76,264] although the sole method currently approved by the FDA (Parastep-1®)[1] uses surface electrical stimulation of the quadriceps and gluteus muscles.[72,230] Muscle activation is sequenced by a microprocessor worn on the belt, with activation of step initiated by a finger-sensitive control switch located on a rolling

1 The use of trade names is for product identification purposes only and does not imply endorsement.

walker used by ambulating subjects. When pressed, the electrical stimulator sends a current to the stance limb that initiates contraction of the quadriceps and gluteus muscles. Contralateral hip flexion is then achieved by exploiting an ipsilateral flexor withdrawal reflex obtained by introducing a nociceptive electrical stimulus over the common peroneal nerve at the head of the fibula. This allows the hip, knee and ankle to move into flexion followed by extension of the knee joint by electrical stimulation to the quadriceps. As muscle fatigue occurs increasing levels of stimulation can be provided by a switch mounted on the handle of the rolling walker.

As in the case of electrically stimulated cycling, functional use of ambulation neuroprostheses by those with paraplegia are compromised by post-injury muscle weakness and poor endurance. These need to be addressed in subject preparation before ambulation training is initiated. Electrically activated knee extensions against progressively increased resistance (sandbag at ankle) is the standard training exercise. While not as commonly addressed, conditioning of the gluteal muscles with electrically produced hip bridging (in supine position) prior to bodyweight-supported gait training will enhance the response of the hip extensors to electrical stimulation. A single most effective protocol to satisfy requisite strength and endurance needs for upright ambulation training has not been reported. However, it is inadvisable to undertake electrically stimulated ambulation training until the ability to stand with electrical stimulation for at least 3–5 minutes has been demonstrated.

Once training is initiated, rates of ambulation are relatively slow and distances of ambulation relatively limited when using the system,[229] and the community use of these devices remains limited to a small percentage of training subjects. Despite the limitations of ambulation velocity and distance, ambulation distances of up to 1.6km (1 mile) have been reported after training in some subjects[229] and upper extremity fitness is enhanced following ambulation training.[71,173] Other adaptations to training include significantly increased lower extremity muscle mass,[265] improved resting blood flow[55] and an augmented hyperaemic response to an experimental ischaemic stimulus.[55] No change in lower extremity bone mineralisation following has been reported,[232] although most subjects participating in research trials of electrically stimulated exercise are beyond the duration of injury at which reversal of their neurogenic osteoporosis is expected.[232]

5. Risks of Exercise When Performed by Persons with SCI

Special attention is required when designing, instituting or performing exercise programmes for persons with SCI. Some of the risks encountered will be similar to those experienced by persons without paralysis, although complications such as general overuse may be exaggerated in persons with SCI, and their occurrence will likely compromise daily activities to a far greater extent than similar injuries arising in persons without SCI.

5.1 Autonomic Dysreflexia

Individuals having cord injuries at or above the T_6 spinal level are prone to episodes of autonomic hyperreflexia when exposed to noxious stimuli.[266] The neurological basis for these episodes involves loss of supralesional sympathetic inhibition that normally suppresses the unrestricted autonomic reflex accompanying such exposure. This allows the adrenals to release high concentrations of epinephrine (adrenaline) under reflex control and infralesional adrenergic targets to experience the full measure of reflex noradrenergic stimulation.[267] The most common stimuli evoking autonomic dysreflexia are bladder and bowl distention before their emptying. Other stimuli include venous thromboembolism, bone fracture, sudden temperature change, febrile episodes and exercise. The disposition to autonomic dysreflexia during exercise is especially heightened when an electrical current is used to generate muscle movement, or when exercising while febrile or during bladder emptying. Episodes of autonomic dysreflexia are characterised by hypertension and bradycardia, supralesional erythema, piloerection and headache. In some cases, hypertension can rise to the point where crisis headache results, and cere-

bral haemorrhage and death might ensue. Recognition of these episodes, withdrawal of the offending stimulus, and the possible administration of a fast-acting peripheral vasodilator may be critical in preventing serious medical complications. Prophylaxis with a slow calcium channel antagonist or α_1-selective adrenergic antagonist may be needed prior to exercise.[268-270] It is known that wheelchair racers have intentionally induced dysreflexia as an ergogenic aid by restricting urine outflow through a Foley catheter,[271] which represents a dangerous and possibly life-threatening practice.

5.2 Musculoskeletal Injury

Fracture and joint dislocation of the lower extremities is a risk of participation in exercise by those with SCI, and may be caused by asynergistic movement of limbs against the force imposed by either electrical stimulation or the device used for exercise.[36] This explains why these activities are contraindicated for individuals with severe spasticity or spastic response to the introduction of electrical current. Precautions to prevent overuse injuries of the arms and shoulders must be taken for those participating in upper extremity exercise.[201,206,272] As the shoulder joints are mechanically ill-suited to perform locomotor activities, but must do so in individuals using a manual wheelchair for transportation, these injuries may ultimately compromise performance of essential daily activities including wheelchair propulsion, weight relief and depression transfers.[273,274]

5.3 Hypotension

Small risks of post-exercise hypotension[131,275] are associated with lost vasomotor responses to orthostatic repositioning,[276] although these episodes can abate after upper limb training.

5.4 Thermal Dysregulation

Individuals with SCI often lack sudomotor responses below their level of injury and are thus challenged to maintain thermal stability.[154,277-279] These responses are less pronounced as the level of SCI descends,[280,281] and when exercising in an environment controlled for temperature and humidity.[278,282] Thus, attention should be paid to hydration and, if possible, limiting the duration and intensity of activities performed in intemperate environments.

6. Conclusions

Multisystem organ dysfunction is common after SCI. Depending on the level of injury, alterations of cardiac function, peripheral circulation, autonomic function, skeletal integrity, muscle composition and genitourinary functions all accompany an SCI. The fact that many of these pathological system changes are reversible permits better understanding of the roles played by central innervation and physical activity on organ system function in persons with intact neuraxes. As levels of injury, types of injury and the extent of organ system dysfunction vary among persons with SCI, careful attention must be paid to accurate classification of individuals before entry into clinical treatment or study. The use of homogeneous subject populations based on similar levels, types and durations of injury is especially important if conclusions concerning deconditioning and reconditioning in these persons are to be reached.

Despite these special needs and warnings, many persons with SCI benefit from sustained therapeutic and recreational exercise. Individuals with higher levels of cord injury may require electrical stimulation to perform exercise, which poses special restrictions on use and unique risks from participation. Qualified individuals safely improved cardiovascular and musculoskeletal functions. Positive benefits of training on bone density, regulation of orthostatic tolerance and affect have been reported in studies with limited numbers of subjects and require well controlled investigations for confirmation. Individuals with spared motor control of the upper extremity can perform arm or wheelchair exercises and participate in recreational sports. Greater emphasis needs to be placed on strengthening of the upper extremity to preserve shoulder and arm functions for performance of daily living as these individuals age with their paralysis. Risks of injury or illnesses associat-

ed with imprudent exercise must be managed to ensure that the desirable benefits of physical activity can be sustained. If carefully prescribed, exercise has the demonstrated ability to enhance the activity, life satisfaction and health of those with disability from SCI.

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Correspondence and offprints: Dr *Patrick L. Jacobs*, The Miami Project to Cure Paralysis, University of Miami School of Medicine, 1095 Northwest 14th Terrace, Miami, R-48, FL 33136, USA.
E-mail: pjacobs@miami.edu