Chapter Two

Factors That Influence Exercise Tolerance

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INTRODUCTION

Due to the improvements in medical care, individuals with spinal cord injury (SCI) are now mirroring the nondisabled population in life expectancy and cause of death. In a study by Geisler and Jousse (1), renal (kidney) failure remained the leading cause of death, at 30.8 percent of the population with SCI studied. A 1983 study on the survival rates of individuals with SCI done by Geisler, showed a higher mortality rate than the population at large. However, an improvement was noted in life expectancy of the individual with SCI. The 1983 study concluded that mortality rate was highest for the complete SCI lesions and lowest for the partial lesions. The authors of the 1983 study (3) attributed the increase in longevity to the fact that even a small amount of continuity in spinal cord function allowed the body a more normal homeostasis (internal balance). It was noted that the leading cause of death for paraplegics and incomplete tetraplegics was heart disease, while the leading cause of death for complete tetraplegia was respiratory disease. Another study done by Yekutiel in 1989 showed a higher incidence of hypertension and ischemic heart disease among those with SCI compared with age-matched controls: 34 percent versus 18.6 percent (2). Yekutiel found no correlation between level and extent of cord lesion or any other medical complications and cardiovascular disease.

A recent study by Bauman reported that cardiovascular disease was the leading cause of death in individuals with SCI (3). Cardiovascular disease appeared to occur at younger ages in the population with SCI than in the nondisabled population. The researchers concluded that this was due to the reduction in level of activity and adverse changes in body composition caused by the SCI. In addition, Bauman suggested that having a SCI had profound metabolic consequences that influenced the progression and severity of coronary artery metabolism. High density lipoprotein (HDL) cholesterol levels were markedly depressed and low density lipoprotein concentrations relatively elevated, in comparison to the nondisabled population. Other studies have found similar results for blood lipid profiles (3–5). These results are important as an inverse relationship exists between the HDL cholesterol level and coronary heart disease (6,7). Krum explored further by looking into the risk factors for cardiovascular disease in the Australian population with SCI (7). The risk factors investigated were serum cholesterol levels, blood pressure, cigarette smoking, glucose tolerance, physical activity, and family history. There was a low cardiac risk factor score in persons with SCI despite the reports of increased coronary heart disease incidence in these patients. Low cardiac risk scores were attributed to low diastolic blood pressure (DBP). A direct correlation was

found between the level of physical activity and HDL level.

EXERCISE AND SPINAL CORD INJURY

Concern for physical fitness has been a recognized phenomenon for the past several decades. Numerous studies validate the benefits of physical fitness on nondisabled individuals as well as on individuals with cardiopulmonary dysfunction and peripheral vascular disease. The documented responses to chronic aerobic exercise include, but are not limited to, cardiac muscle hypertrophy (enlargement), decreased recovery time for resting heart rate, decreased total cholesterol and HDL, decreased cardiac risk, increased self-esteem, and control of body weight (8–12). The positive response of the body to aerobic fitness in the nondisabled and circulatory-impaired populations is well documented (13), and led to the establishment of measurement parameters to monitor levels of fitness. The American College of Sports Medicine has established guidelines for fitness testing in the nondisabled population and certain special populations, including individuals with cardiovascular disease, pulmonary dysfunction, and diabetes mellitus (14).

Although physical fitness is important for individuals with SCI, many factors limit their ability to exercise, such as autonomic nervous system dysfunction and motor dysfunction. The ability of an individual with SCI to exercise is also complicated by prolonged bed rest and the sedentary lifestyle that follows discharge. Inactivity and bed rest result in complications, such as reductions in orthostatic tolerance, total blood volume, oxygen uptake, stroke volume, and cardiac output. These, in turn, result in a decreased exercise tolerance (15). The SCI population's ability to exercise is further limited by intrinsic factors caused by the disability.

The higher the level of injury in the spinal cord, the greater the number of skeletal muscles that are paralyzed. Less functioning muscle mass results in a lower voluntary exercise capability and lower absolute cardiopulmonary (aerobic) fitness level. In addition, immobilization of the lower limbs may precipitate venous stasis (stoppage or reduction of blood flow) and edema due to inactivity of the venous muscle pump. Aerobic exercise capacity in the population with SCI is also limited by diminished sympathetic outflow. Sympathetic stimulation is needed for cardiovascular reflex response to exercise. These reflexes increase blood flow to active skeletal muscle, providing more oxygen and fuel substrates while increasing the rate of metabolic end-product removal. These responses also include vasoconstriction of relatively inactive tissues and vasodilatation of skeletal muscle arterioles, and an increase in heart rate, myocardial contractility, and cardiac output (16). The loss of functional skeletal muscle and the diminished or nonexistent cardiovascular reflex during exercise can result in high fatigability of the active muscles of the arms. The relatively small muscle mass in the upper limbs fatigue quickly because of inadequate blood flow, limited aerobic energy supply, and the accumulation of metabolites in the muscle (16). All the above-stated factors discourage many individuals with SCI from engaging in a regular physical fitness program. Normal daily wheelchair activity is probably not sufficient to train the cardiopulmonary system or improve physical fitness (17,18). Activities of daily living (ADL) are shown to be stressful for individuals with spinal cord injuries, especially individuals with tetraplegia, as seen in their increase in heart rate during ADL (19). An individual with SCI who is sedentary becomes deconditioned, which can lead to functional deterioration. Exercise is necessary to break this cycle, decrease cardiovascular risk factors, and to maximize functional independence. Cardiovascular fitness has been shown to increase an individual's longevity, as with nondisabled persons, and to decrease the risk of heart disease, respiratory disorders, and coronary artery disease (12).

LIMITING FACTORS CONCERNING EXERCISE

Some road blocks exist when evaluating fitness for the population with SCI. Autonomic nervous system (ANS) and motor dysfunctions are primary limiting factors that alter the outcomes of testing and training procedures. The special concerns of the population with SCI are addressed in greater detail below, with the anticipated effects of exercise.

AUTONOMIC NERVOUS SYSTEM DYSFUNCTION

Sympathetic Outflow to the Heart

The sympathetic outflow to the heart is innervated between T3 and T5 (20). Therefore, individuals with

spinal cord injuries above T5 lose, or have impaired, sympathetic outflow to the heart. Cardioacceleration then becomes dependent upon withdrawal of vagal tone. While testing individuals at this injury level, an increase in workload does not necessarily result in an equivalent increase in heart rate. Thus, heart rate response should not be the sole determinant of exercise intensity for the population with SCI above the T5 level.

Sympathetic Outflow to the Adrenal Medulla

Epinephrine is released into the circulatory system in response to exercise. Sympathetic outflow to the adrenal medulla of the brain, the primary source of circulating epinephrine, is between T6 and T10 (20). The purpose of epinephrine is to accelerate adipose tissue lipolysis (fatty tissue decomposition), muscle glycogenolysis and hepatic glycogenolysis. "Therefore, any factor limiting adrenomedullary activation and subsequent elevation of circulating plasma epinephrine could ultimately influence exercise performance," (21). Above the T6 level, the sympathetic outflow to the adrenal medulla may be impaired or absent. This will result in abnormal releases of catecholamines (epinephrine and norepinephrine) during exercise. These are the "charge hormones."

Though individuals with higher level SCI do not have normal sympathetic responses, training changes their sympathetic responses. In a study conducted by Bloomfield in 1994, 7 subjects with SCI (C5–T7) were trained for 6 months with functional electrical stimulation (FES) cycle ergometry with plasma samples taken before and after the training period. Bloomfield found that resting norepinephrine decreased 37 percent (majority of those being paraplegics), with the resting epinephrine decrease in the paraplegics at 42 percent and in the quadriplegics at 18 percent. Acute FES cycle ergometry elicited an increase in norepinephrine of 55–844 percent, and a 35–350 percent increase in epinephrine above resting values, with heart rate responses ranging from 90 to 146 beats per minute (bpm). Since these increases cannot be attributed to adrenergic drive originating in the central nervous system, an alternative mechanism to the epinephrine response may be a spinal reflex driven by afferent signals from mechanoreceptors in contracting skeletal muscle.

Sympathetic Outflow to the Lower Limbs

Sympathetic outflow to the lower limbs controls vasoresponses during exercise. Spinal cord injuries above L1 result in impaired or absent vasoresponses in the lower limbs (20). This means that while the upper limb musculature is working, there will not be a resultant vasoconstriction in the inactive lower limb muscles. The inability of the musculoskeletal pump in the legs to be activated also contributes to venous pooling.

Hopman compared paraplegic and nondisabled subjects during arm exercises with eliminated muscle pump activity (22). During the onset of exercise, the group with SCI showed no leg volume changes indicating a lack of vasomotor responses in the calf muscle vessels because of the loss of sympathetic regulation below the lesion. During exercise, the lower limb volume of the group with SCI was significantly lower than that of the control group (confirming the fact that some individuals with SCI do not have the ability to redistribute blood in the lower limbs). The above-mentioned changes were correlated with the level, but not the completeness, of the spinal cord lesion. The amount of active muscle available, especially in the lower limbs, greatly influences the body's ability to redistribute blood and the amount of venous pooling in the lower limbs. The elevation in heart rate acts as a compensatory mechanism to maintain cardiac output and assure adequate blood perfusion in arm muscle. Studies have shown that by stimulating the muscle pump in the legs and by supporting the redistribution of blood by functional electrical stimulation, there are increases in cardiac output and stroke volume (23). Anti-gravity suits, which increase external pressure on the legs and splanchnic area, have shown to induce a central shift in lower body blood volume during arm exercise (24). Cardiac output is unchanged showing that the stroke volume has increased as a result of the supported redistribution of blood by the suit. Exercise in the supine position (lying face up) also shows a positive effect on the redistribution of blood in persons with tetraplegia during arm exercise (25). These techniques are discussed in greater detail in Chapters One and Three.

Thermoregulation

Individuals with SCI have higher core temperatures when exposed to the heat, and lower core temperatures when exposed to the cold. Therefore, an individual with SCI has a reduced ability to tolerate extremes in temperature and to perform aerobic exercise in hot or humid environments.

According to Sawka, the ability to thermoregulate is impaired in individuals with SCI because of 1) loss of

autonomic nervous system control for vasomotor and sudomotor responses in the areas of the insensate skin, 2) a reduced thermoregulatory effector response for a given core temperature, and 3) a loss of skeletal muscle pump activity from the paralyzed limbs (26). The absence of thermoregulatory sweating and vasoconstriction or vasodilation below the level of the lesion causes the body temperature of the client with SCI to be influenced by the temperature of the external environment (27). This may cause hyperthermia during exercise in a warm environment and hypothermia in cooler weather. When compared to nondisabled individuals exercising in the same environment, subjects with low paraplegia had a greater increase in oral and mean skin temperature, and a 14 percent increase in cardiac output. The subjects with SCI had to rely on dry heat exchange for thermal equilibrium; more blood had to be diverted to the skin to increase skin temperature (28).

Autonomic Dysreflexia

Autonomic dysreflexia (hyperreflexia, or exaggeration of reflexes) occurs in individuals with SCI lesions above the T6 level and is characterized by a nociceptive-induced reflex sympathetic discharge resulting in peripheral vasoconstriction (narrowing of the blood vessels), hypertension (HTN) and piloerecton (hair raising) distal to the lesion (29). The HTN results in stimulation of vascular baroreceptors activating the parasympathetic nervous system, causing vasodilatation (dilation of the blood vessels) above the lesion level. A sudden rise of blood pressure of greater than 40 mmHg over the basal pressure constitutes an emergency situation and appropriate measures to decrease blood pressure and remove the noxious stimulus must be performed. Some athletes with SCI deliberately induce autonomic dysreflexia, also known as "boosting," to enhance performance, most commonly done by overextending the bladder, applying tight leg straps, or sitting on pronounced objects. This increases catecholamine and norepinephrine levels, causing exaggerated blood pressure responses. On occasion, this was associated with significantly dangerous blood pressure elevations. This is considered an illegal activity during any sanctioned wheelchair/sports racing event. In addition, it is quite a dangerous method to use in order to positively affect racing performance.

Orthostatic Hypotension

Orthostatic hypotension is a sudden drop in blood pressure occurring during upright posture, whether standing or sitting. The hypotension is caused by a lack of muscular pumping of the vascular beds, which leads to peripheral blood pooling, impaired venous return, and diminished cardiac output (30). Injury above the sixth thoracic nerve results in an interruption of all spinal sympathetic efferent pathways. This interrupts the normal reflex control of the blood vessels via carotid sinus baroreceptors, although reflex control of heart rate via the vagus nerve persists. The disruption of vasomotor control below the level of lesion leads to inadequate vasoconstriction in the splanchnic (visceral) bed and nonexercising muscles, ultimately resulting in decreased venous return and the drop in blood pressure. External compression, such as an abdominal binder or support stockings causes stroke volume to increase; thus, orthostatic hypotension may be prevented (30).

Exertional Hypotension

Exercise hypotension occurs as a direct result of muscle paralysis and the absent vasoresponse in the lower limbs (31). Normal redistribution of blood flow and maintenance of blood pressure are prevented due to the interruption in the neural control of arterial smooth muscle in nonexercising muscles and in the splanchnic bed (32). Normally during exercise, vasodilatation occurs in active muscle, while there is a resultant vasoconstriction in inactive muscle. For the individual with SCI, this may not occur. Vasodilatation occurs in the active upper limb muscles, but there may be no compensated vasoconstriction occurring in the lower limbs to support an increase in blood pressure with increasing workloads. Coupled with decreased exercise stroke volume from inadequate venous return and vasodilatation in exercising muscle, these mechanisms may cause blood pressure to decrease during exercise.

As workloads increase, a normal blood pressure response in the nondisabled population is a gradual rise in systolic blood pressure and a stable diastolic blood pressure (14). It would be abnormal for the systolic blood pressure or the diastolic blood pressure to drop more than 10–20 mmHg during exercise. However, it is not unusual to see such a drop in blood pressure during exercise in the SCI population. Blood pressure should not drop below 70/40, as this may result in cardiac arrest (33). It is extremely important to closely monitor blood pressure at all times during exercise of this patient population. The client with tetraplegia is particularly at risk due to an already low resting blood pressure. During continuous maximal arm ergometry, exertional hypotension was seen in all subjects with

cervical injuries, 50 percent of the subjects with high thoracic injuries (T1 to T6), and 17 percent of the subjects with low thoracic injuries (T7 to T12) (32). Drory obtained similar results with 60 percent of the subjects with a cervical injury demonstrating exertional hypotension, as well as 50 percent of the subjects with a high thoracic lesion, 20 percent with low thoracic or lumbar lesions, and 16 percent of nondisabled controls (34).

Myocardial Atrophy

Compared to healthy nondisabled individuals, individuals with chronic SCI have left ventricular myocardial atrophy and diminished cardiac function. This is shown in decreases in cardiac output and stroke volume due to the chronic underloading of ventricular pressure, ventricular volume, and diminished venous return (35). Left ventricular mass has been shown to be greater in exercise-trained individuals with SCI using FES-induced lower limb cycle ergometry than in sedentary individuals, as a result of exercise-induced pressure and/or volume challenge on the heart (9). The individuals with SCI who exercised showed a 35 percent increase in left ventricular mass, as measured by echocardiogram, and an increase in left ventricular chamber size. The researchers concluded that these increases were due to pressure and volume overload imposed by the FES exercise.

MOTOR DYSFUNCTION

Muscle Paralysis

Injuries to the spinal cord usually result in some degree of muscular paralysis: 1) lower limb paralysis, 2) muscles involved with breathing, 3) trunk muscle paralysis, and/or 4) upper limb involvement. Lower limb muscle paralysis prevents many traditional methods of testing and training because the majority of the equipment used for the nondisabled population incorporates the use of the lower limbs. In addition, lower limb muscle paralysis results in a lack of skeletal muscle pump in the lower limbs. This leads to a decreased amount of circulatory blood volume.

The primary cardiorespiratory complications of the population with SCI include muscle paralysis of the diaphragm and accessory and intercostal muscles, which leads to decreased inspiratory efforts and chronic hypoventilation (36). The extent of ventilatory impairment is directly proportionate to the amount of inspiratory and expiratory muscle involvement (36). Lesions in the lower thoracic region of the spinal cord decrease the function of the abdominal musculature, causing a decrease in forced expiration (37). Abdominal and intercostal musculature paralysis and poor sitting posture can all decrease tidal volumes during exercise.

Expiratory muscle paralysis of the abdominals and intercostals decreases the cough mechanism, resulting in poor secretion clearance (38). Spasticity of the trunk musculature decreases rib cage expansion and further decreases inspiratory efforts. Not surprisingly, at rest persons with tetraplegia have been found to have less than 50 percent, and persons with paraplegia 75–80 percent of normal resting vital capacities and forced expiratory volumes (39). Exercise can affect these complications. Crane (37) has shown that persons with tetraplegia exercising over a 6-month period achieve an increase in their forced vital capacity (FVC). A reduction in FVC has been shown to be an important risk factor for respiratory complications such as pneumonia and atelectasis. No changes were noted in other pulmonary measurements after the exercise training program. Upper limb paralysis complicates testing and training even further due to an even smaller muscle mass to carry out the exercise program. The smaller upper limb muscles are quicker to fatigue than the larger lower limb muscles (31). For an individual to achieve true maximal oxygen consumption ($\dot{V}O_2$), one-half the normal muscle mass must be used during the exercise bout (33). Many times, this situation does not exist with individuals who have sustained a spinal cord injury.

The amount of available upper limb muscle to be used by the individual with SCI greatly influences exercise response. Exercise capacity was shown to be the lowest in persons with tetraplegia, followed by untrained females with paraplegia, untrained males with paraplegia, and trained males with paraplegia, in that order. Trained paraplegic males had the greatest exercise capacity as defined by maximal oxygen uptake (40). During wheelchair propulsion, persons with tetraplegia had a lower mean power output than other individuals with SCI (41).

Atrophy

Spinal cord lesions below the T12 level result in a lower motor neuron lesion, which may result in atrophy (muscle wasting/reduction in muscle bulk) of the lower limb musculature (42). With muscular atrophy, there is

a subsequent decrease in lean body mass and an increase in body fat percentage. As a result, metabolic rates may decrease secondary to this decrease in lean body mass, or an increase in body fat percentage. A decrease in metabolic rate leads to a decrease in caloric demand, which may lead to increased weight gain (33). Functional electrical stimulation to atrophied muscles may help slow the affects of disuse atrophy; yet the amount of time needed for significant results makes this modality unrealistic (42).

Spasticity

One-third of all individuals with SCI experience spasticity (44). Spinal cord lesions above the T12 level result in an upper motor neuron lesion, which may lead to significant levels of spasticity. Spasticity in the trunk musculature restricts inspiratory volumes by not allowing full rib cage expansion. Also, spasticity in the trunk, upper limbs, and lower limbs may affect movement control during exercise and may also lead to poor sitting balance, which again leads to decreased inspiratory volumes.

OTHER CONSIDERATIONS

Bone Density

Soon after injury, individuals develop osteoporosis below the neurological lesion. This indicates an imbalance between the synthesis and the resorption of bone (45). Other investigators showed evidence of increased bone resorption by urinary excretion of hydroxyproline, calcium, and phosphorus (46–48). Nash evaluated whether or not the mechanical forces incurred by the paralyzed skeletal system during exercise might prevent, retard, or accelerate the native course of arthro- and osteodegeneration of individuals with SCI (50). This is important due to the forces on the lower limb bones and joints that occur during rehabilitation, ambulation, and exercise. Nash evaluated 10 male individuals with C6–T9 SCI who completed a 1.5-year program of electrical stimulation cycle ergometry exercise 2–3 times per week. The investigators concluded that electrically stimulated exercise was safe for the bones, ligaments, and cartilage, and it did not foster bone or joint deterioration in individuals with chronic SCI. Leeds (51) found similar results as she trained male tetraplegics using functional electrical simulation cycle ergometry for 3 days a week for 6 months. However, there was no increase in the bone mineral density of the proximal femoral head (hip joint) after training. While the development of osteoporosis for the individual with SCI can not be entirely stopped, a person's level of activity affects how joint degeneration occurs. Wylie, in 1988, through radiographic assessment of joints and a survey of the level of activity, showed that only 38 percent of the active patients developed degenerative hip changes, as compared to 62 percent of the inactive group. Wylie also showed that moderate joint activity protects the shoulder joint from degenerative changes (52).

Immune System

Subjects with SCI may be a greater risk for sustaining infections due to changes in their immune system, especially those subjects with autonomic nervous system involvement (50). Exercise has been shown to have a beneficial effect on the immune system in some subjects with SCI, by increasing the natural killer cell number and cytotoxicity due to the significant catecholamine response following exercise (53,54), but if the individual is overtrained, the immune function may be inhibited (55).

BENEFITS OF EXERCISE

Individuals with SCI who are involved in regular exercise show psychological gains. A study by Shephard in 1991, showed that individuals with SCI who exercised had an improved state of mind, a reduction of anxiety and depression, and an increase of self-esteem and feelings of greater self-efficacy (56). Sociological gains included new experiences, new friendships, and a countering of stigmatization. The subjects had a perceived improvement in health and a reduced risk of many chronic diseases. Subjects had a greater likelihood of employment with less absenteeism and enhanced productivity.

As in nondisabled athletes, individuals with SCI who engage in a regular exercise program show higher oxygen consumption at maximum workloads than non-athletic individuals with SCI. Okuma showed a mean difference of 12.4 ml/kg/min of maximal oxygen uptake between wheelchair marathon competitors and non-athletic paraplegics (57). These same competitors had a significant reduction in their level of physical fitness during the off-season and still maintained a higher level than that of non-athletes. Hooker showed

that paraplegic road racers elicited significantly higher mean peak levels of power output and peak oxygen uptake than did untrained nondisabled males (58). After a 2-month exercise regimen, recreational wheelchair subjects showed significant increases in maximum oxygen uptake, functional endurance, and maximal workload (59,60). The benefits of exercise to individuals with SCI are innumerable. Many of them have been mentioned in this chapter along with the review of corresponding literature. Exercise should be an integral part of everyone's life, but especially of individuals with spinal cord injuries. Exercise improves the functional capacity of individuals with SCI and reduces some physiological complications of having a spinal cord injury. The guidelines needed to begin and carry out an exercise program for this population will be addressed in Chapter Three.

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