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Cardiovascular Disease and Spinal Cord Injury

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Abstract

Cardiovascular disease (CVD) has become an increasing concern for persons with spinal cord-injury (SCI) as it is the leading cause of death for those with SCI as well as the general population. Secondary complications of SCI such as autonomic dysreflexia and deep vein thrombosis; however, increase the risk for cardiovascular disease. As such, early diagnosis, prevention and treatment of cardiovascular disease should be a vital component in the ongoing medical care of persons with SCI. The focus of this article is to provide information regarding cardiovascular disease following spinal cord injury and address the prevalence, risk factors and treatment options that are unique for this population.

Keywords: cardiovascular disease, spinal cord injury, morbidity, cardiovascular system

Cardiovascular Disease and Spinal Cord Injury

Spinal cord injury (SCI) is a medical condition with significant functional, psychological, and socioeconomic considerations. Although there have been major advances in neurological treatment for SCI, the mortality rate for this condition remains high relative to ambulatory populations (DeVivo & Stover, 1995). In recent years, cardiovascular disease (CVD) has been demonstrated to be the leading cause of death for those with acute and chronic SCI (Garshick, Kelley, & Cohen, 2005; Popa et al., 2010). Increasing the risk of developing CVD for those with SCI include hypertension, hyperlipidemia, autonomic dysreflexia (AD), deep vein thrombosis (DVT) obesity and diabetes (Bauman, Kahn, Grimm, & Spungen, 1999b).

Contributing to cardiovascular morbidity is the more sedentary lifestyle of those with SCI due to the nature of their injuries and the impact of being non-weight-bearing (Washburn & Figoni, 1998). In addition, spinal cord injuries disrupt the normal autonomic cardiovascular control mechanisms decreasing functionality (Bauman et al., 1999b). The latter occurs as a result of a variety of physiologic changes related to cardiovascular control that are observed in SCI, including loss of normal regulation of the peripheral vasculature, autonomic dysreflexia (for those with T5 lesions or higher), and a higher prevalence of cardiac rhythm disturbances (Bauman et al., 1999b).

Cardiovascular Disease. At a rudimentary level, the cardiovascular system is comprised of the heart and blood vessels. The heart is essentially a muscular pump in the chest that receives and delivers blood throughout the body. As it beats, the heart delivers blood to the lungs to exchange oxygen. This oxygenated blood is then circulated back to the heart and pumped to all organs throughout the body. The blood vessels, consisting of the arteries, capillaries and veins, carry blood to all the tissues of the body and remove waste product.

Cardiovascular disease, heart and blood vessel disease, coronary heart disease, and heart disease are all conditions that identify complications with the cardiovascular system (American Heart Association, 2013). Cardiovascular disease typically develops after decades of natural wear and tear of the heart and arteries; however, is also facilitated by factors such as stress, fatty diet, and a sedentary lifestyle. Heart disease develops initially as atherosclerosis, which is the gradual buildup of fatty cholesterol plaque formation on the walls of the arteries. The plaque formations restrict blood flow through the arteries which cause the heart to work harder to replenish oxygenated blood. Plaques may break off the arterial wall and may result in a heart attack or stroke depending on where the insult occurs (Roger et al., 2012).

Statistics indicate that of the approximately 715,000 heart attacks occur annually in the U.S., and of these, an estimated 525,000 are a patients' first attack (Roger et al., 2012). Although statistics vary among ethnic and racial groups, coronary heart disease remains the number one killer in the US, claiming an estimated 385,000 lives annually (Kochanek, Xu, Murphy, Miniño, & Kung, 2011).

Cardiovascular disease costs in the United States are estimated at over $108.9 billion yearly (Heidenreich et al., 2011). Factors contributing to cardiovascular disease are largely preventable and include high blood pressure, high-density lipoprotein cholesterol, smoking, Type II diabetes, obesity, and physical inactivity (Dawber, Moore, & Mann, 1957; Ferrie et al., 2002; Kroese, 1977; Wolfram et al., 2006; Yusuf et al., 2004;). According to the Center for Disease Control (CDC, 2013), almost half of the population of the United States have at least one of these risk factors. As well, cardiovascular disease may manifest as various complications including coronary artery disease, autonomic dysfunction, and deep vein thrombosis.

Coronary artery disease (CAD). Characterized by the build-up of plaque in the coronary arteries, CAD is one of the leading contributory causes of death for those with SCI (Blackwell, Krause, Winkler, & Stiens, 2001). CAD accounts for high total cholesterol, LDL, and triglyceride levels, and is likely brought about by muscle loss and atrophy, lack of physical activity, and increased body fat among those with SCI. Morbidity from advanced coronary artery disease (CAD) is high in persons without a disability as well. For those with SCI, CAD frequently develops sooner than with non-disabled individuals (DeVivo, Stover, & Black, 1992; Garshick, Cohen, Garrison, & Tun, 2005).

Autonomic dysreflexia (AD). Potentially fatal, AD can cause hypertension and cardiac arrhythmias (Blackwell et al., 2001). AD occurs when there is an extreme sudden spike in elevated blood pressure frequently caused by bowel constipation or bladder distention; however, may also be caused by noxious stimuli. Complications may lead to stroke, seizure, or myocardial infarction. AD occurs in SCI cases whose lesions are above T6 and primarily in those with complete injuries more so than incomplete SCI. Approximately 95% of all reported cases of AD occurs with individuals during the chronic phase of SCI and predominantly those with cervical injuries (Popa et al., 2010). Statistics have reported its prevalence ranging anywhere between 48% to 90%. Prevention involves careful monitoring of blood pressure, an established regular bowel and bladder program, and making sure the individual is free of noxious stimuli.

Deep vein thrombosis (DVT). Often occurring in the legs, deep vein thrombosis (DVT) is the formation of blood clots in the deep veins, and can result in a pulmonary embolism, a potentially fatal condition should the clot travel to the lungs. Due largely to the non-weight bearing aspects of SCI, it is not uncommon for this population to experience one or more DVT episodes over their lifetime (Riklin et al., 2003). Frequently occurring during the initial year of

a spinal cord injury, Blackwell and colleagues (2001) state the greatest risk appears prior to six months post injury. Popa et al., (2010) assert that the risk of DVT is most common seven to 10 days after injury and this risk is reduced within 3 months post-injury. Nevertheless, DVT can occur during the chronic stages of SCI as well due largely to poor circulation from non-weight-bearing where the blood pools more easily in the lower extremities. Situations such as flying at high altitudes increases the likelihood for DVT and many physicians will often prescribe compression hose and low dose aspirin for their patients. Those with more serious DVT problems may be placed on blood thinners such as Coumadin for an indefinite period of time.

CVD and Spinal Cord Injury

Key contributors to the high risk of CVD for those with SCI include abnormal lipid profiles, diabetes, and obesity, which are more prevalent among individuals with SCI (Bauman, Adkins, Spungen, & Waters, 1999a; Demirel, Demirel, Tukek, Erk, & Yilmaz, 2001; LaVela, Weaver, Goldstein, Chen, & Miskevics, 2006; Lee, Myers, Hayes, Madan, & Froelicher, 2005). SCI is characterized by a disruption of the normal autonomic cardiovascular control mechanisms. Loss of autonomic control can result in a variety of physiologic changes related to cardiovascular control that are observed in SCI, including loss of normal regulation of the peripheral vasculature, autonomic dysreflexia (AD), and a higher prevalence of cardiac rhythm disturbances (Bauman et al., 1999b; Mallory,1994). The autonomic dysfunction that occurs with SCI also underlies several cardiovascular irregularities, including an accelerated decline in cardiovascular function with aging (Myers, Lee, & Kiratli, 2007).

The reduced physical function associated with SCI, an additional contributing factor to high cardiovascular morbidity and mortality, leads to a greater sedentary lifestyle and lower energy expenditure (Myers et al., 2007; Jacobs & Nash, 2004). Individuals were involved in regular physical activities pre-injury were able to a cardiovascular response to physical exertion; however, post injury there becomes a dramatic reduction of physiological exertion particularly to the lower extremities (Washburn & Figoni, 1998).

Claydon, Hol, Eng, and Krassioukov (2006) found abnormal cardiovascular responses to arm cycling exercise and transient post-exercise hypotension for those with cervical but not thoracic lesion SCI, citing it may be partially related to loss of descending sympathetic nervous control of the heart and vasculature following cervical level injuries. Loss of sympathetic tone also causes reduced myocardial preload and myocardial contractility resulting in reduced stroke volume and cardiac output which lead to cardiac atrophy. Other complications are left ventricular myocardial atrophy and diminished cardiac function that develop in the presence of chronic tetraplegia (Nash, Bilsker, Marcillo, Isaac, & Bothelho, 1991). These factors contribute significantly to a reduced capacity to adapt effectively to exercise, which result in early fatigue, general avoidance of physical exertion, and overall cardiac de-conditioning (Jacobs & Nash, 2004; Washburn & Figoni, 1998). The cardiac de-conditioning associated with chronic SCI may seriously compromise intervention readiness of the individual and the impact of interventions requiring even minimal physical exertion.

Prevalence of CVD following SCI

Abnormalities of the cardiovascular system have long been a major concern for individuals with chronic SCI. The prevalence of CVD among persons with SCI compared to ambulatory populations is higher, and as noted earlier, is the leading cause of death for both populations (Bauman, et al., 1992; Groah, Weitzenkamp, Sett, Soni, & Savic, 2001; Popa et al., 2010). Whiteneck and colleagues (1992) reported that cardiovascular disease was the leading

cause of death for individuals who had experienced a spinal cord injury at least 30 years prior. A more recent prospective study of mortality after chronic SCI indicated diseases of the circulatory system contributed most often to cause of death (Garshick et al., 2005).

Utilizing a sample of 545 participants with SCI at least 20 years post-injury, Groah and colleagues (2001) found in their study that the risk of developing CVD was correlated with a higher level lesion, severity, and advanced age. The authors delineated between three groups using Frankel ASIA scale: tetraplegia A, B, C; paraplegia A, B, C; and all D level injuries. Of the group, there were 458 recorded CVD events during the 20 year period comprised of coronary heart disease (24%), hypertension (21%), dysrhythmias (16%), peripheral vascular disease (15%), congestive heart failure (8%), valvular disease (7%) and atrial fibrillation (2%). Complete injuries accounted for 44% greater risk in developing CVD.

The level of spinal cord injury and its effects on cardiovascular function was analyzed by using meta-analyses by West, Mills, and Krassioukov (2012). The researchers found that cardiovascular dysfunction occurred more frequently with higher level injuries. Those individuals with cervical SCI exhibit lower blood pressure and heart rate. Additionally, the systolic blood pressure of those with cervical SCI was lower in the seated versus the supine position. The authors reviewed 98 studies using meta-analysis and relatedly found that those with high levels cervical injuries had a higher prevalence of orthostatic hypotension. Individuals with lower level injuries from T1-T5 had partial preservation of sympathetic function, while those with T5 injuries or lower had full supraspinal sympathetic control of the heart.

Other studies have reported lesser but nevertheless substantial prevalence rates of asymptomatic CVD in SCI populations ranging from approximately 25% to more than 50% (Janssen, Van Oers, & Van Kamp, 1997). In contrast among age-matched persons without SCI, the prevalence of CVD is typically reported to be in the range of 5% –10% (American Heart Association [AHA], 2011). When focusing on the relationship of mortality from cardiovascular causes, large cohorts of subjects with chronic SCI have been reported to possess both greater cardiovascular mortality rates and mortality occurring at earlier ages compared with persons without a disability. In an analysis of over 28,000 medical records of SCI patients occurring between 1973 and 1998 from two SCI health care systems, heart disease was the leading cause of mortality excluding first year of injury (DeVivo, Krause, Lammertse, 1999).

Davies and McColl (2002) conducted a study in which they investigated lifestyle risks of cardiovascular disease among those with SCI. The authors found that increased age and smoking presented a greater risk for people with SCI to have CVD. Those who smoked for a longer period of time were at greater risk for developing CVD than non-smokers with spinal cord injury.

Seeking to explain why those with SCI appear to be at an increased risk for developing cardiovascular disease, Matos-Souza et al., (2011) assessed 34 men with SCI and 31 without, all of whom were similar in demographic characteristics. The authors found that those participants who have SCI had left ventricular diastolic dysfunction, a pattern of left ventricle concentric remodeling (structural change to heart after injury to or disease of the myocardium), and impaired systolic function in a frequency greater than those without SCI. These problems to the heart may explain in part the prevalence of cardiovascular disease among individuals with SCI.

Treatment Options

Current data suggests that applicable intervention approaches be established to identify

risk factors for CVD and to decrease rates of cardiovascular morbidity and mortality in persons with SCI. Exercise has long been accepted as a means to increase cardiovascular health. For persons with SCI, physical inactivity is unfortunately common due to the nature of the injury and contributes to the risk of coronary heart disease. Exercises and assistive technology exercise equipment have been developed that individuals with SCI can utilize to maintain some degree of cardiovascular health.

Functional electrical stimulation (FES) exercise has been used for over a decade to stimulate paralyzed muscles and has been proven beneficial for cardiovascular exertion for those with SCI (Ditor et al., 2005). Ditor and colleagues, however, note that although beneficial, patients must be monitored for autonomic dysreflexia due to overstimulation, bone fractures, and dermal burns when using specific FES equipment. An alternative to FES is body-weight supported treadmill training (BWSTT). Ditor et al. (2005) studied the use of BWSTT on six patients with SCI (levels C4 to T12) over a four-month period. The authors found that after the training period, femoral artery compliance increased despite low heart rates of the patients. Given the risk of developing plaque on arterial walls, arterial stiffness, and blood clots among those with SCI, BWSTT is an encouraging alternative to functional electrically stimulated exercise. Arm and leg cycle FES assistive technology which at one time could only be found in rehabilitation hospitals, are now regularly sold for home use and may be a valid recommendation to any life care plan following appropriate therapist evaluation and recommendation. BWSTT; however, is a much more complicated piece of equipment that requires more specialized physical therapist and physician oversight.

Turiel and colleagues (2011) also studied BWSTT and its effects on cardiovascular function for those with SCI after a six week program. They found BWSTT produced a positive effect on left ventricle systo-diastolic function, coronary flow reserve, and endothelial dysfunction. The researchers also observed that BWSTT was more effective at improving heart rate, isovolumetric relaxation time (the interval between aortic valve closure and mitral opening), and E wave amplitude if the injury occurred less than 12 months prior to beginning the program.

The use of a harness or abdominal binder has also been shown to improve blood pressure rates for those with SCI. Krassioukov and Harkema (2006) compared 11 individuals with SCI to nine able-bodied subjects. For the able-bodied participants, the harness did not affect blood pressure or heart rate. However, the harness did significantly increase the diastolic blood pressure of subjects with SCI. In addition, the use of an abdominal binder for those with SCI was shown to assist in recirculating blood from the abdomen. Life care planner should additionally explore these options with relevant treating specialists to potentially minimize CVD complications.

Standing frames have typically been recommended for many persons with SCI in assisting with weight-bearing. Harkema, Ferreira, van den Brand, and Krassioukov (2008) studied the effects of standing locomotor training on heart rate and blood pressure using participants with a complete cervical lesion SCI. Improvements in the study participants after completion of the training program were noted through increased resting arterial blood pressure and stable blood pressure while standing with body weight support for one hour. Standing exercises, the authors concluded, are beneficial for those with SCI as they are more prone to secondary complications due to immobility.

Myers, Gopalan, Shahoumian, and Kiratli (2012) investigated the effectiveness of a multidisciplinary case management program targeting the reduction of CVD risk among 26 participants with SCI. In this study, participants were provided with individually-developed

exercise and nutritional plans. At regular intervals over a two year period, they were contacted for case management progress evaluations. Respondents were measured on their full lipid profile, blood pressure, diet, lifestyle (including tobacco use), and physical activity. The authors reported that the case management program produced moderate success at reducing risk factors for CVD for two year follow-up with 10 remaining respondents with SCI; most notably, insulin levels, body weight, and total cholesterol/HDL ratio were improved.

Popa et al., (2010) studied valvular dysfunction following spinal cord injury and noted such disturbances as being the leading cause of death for this population both in the acute and chronic stages of injury. They note the anatomy of neurogenic shock, atrial hypotension and bradycardia, autonomic dysreflexia, DVT, and orthostatic hypotension are all common for persons with high level thoracic and cervical SCI that are not generally found in the able-bodied individuals. Studies are cited (See Popa et al., 2010 for complete list) indicating cardiovascular dysfunction contributes to 40.5% of SCI deaths. In addition, of those with complete cervical injuries (ASIA A-B), 68% develop arterial hypotension, 35% requiring vasopressers, and approximately 16% eventually die of cardiac arrest. For individuals with a complete cervical injuries, an estimated 35% to 71% develop bradycardia, but relatively few develop hypotension. The authors cite 48% to 90% of individuals with SCI above T6 will experience autonomic dysreflexia, and 47% to 90% of cases will develop a DVT.

Popa et al., (2010) also discuss treatment options for valvular dysfunction, nonpharmaceutical options, including compression hose, avoiding alcohol and caffeine, eating smaller meals, elevated head position to 10° – 20° in bed, physical therapy, and elevation of the legs. For those with higher risk DVT, Doppler ultrasound or venograms are recommended to establish baselines and heparin for 7 to 10 days post-episode, followed by three months of oral anticoagulant such as Coumadin. Daily aspirin may be recommended by some physicians.

Summary

Cardiovascular diseases have progressively become a concern for those providing services for persons with SCI due largely to advances in medicine and enjoyed increased life expectancy for this population. For those with either acute or chronic SCI, cardiovascular diseases are the highest causes of morbidity and mortality. However, improved detection of CVD for those with SCI has the potential to reduce morbidity and mortality rates. Proper care and treatment, including a multidisciplinary approach may not only increase survival rates, but improve quality of life for individuals with SCI.

For the life care planner, consideration of appropriate exercise assistive technology to include in the life care plan becomes important. Home use FES and standing frame devices appear to have good empirical support in relation to minimizing CVD complications. Isometric exercises, stationary hand cycles and third wheel mobility hand cycles attached to one's wheelchair for outdoor use are all excellent equipment for cardiovascular exercise that should be staffed with the physical therapist and/or physiatrist depending on individualistic circumstances. Reducing the onset of early CVD complications continue to be studied for both SCI and non-SCI related populations. Proper diet and lifestyle must also be considered where needed in terms of smoking and substance abuse cessation programs where relevant, adding a nutritionist to the life care plan where relevant, and including any related cardiovascular health regular diagnostic tests such as Doppler ultrasound, CBC panels noting cholesterol levels, venogram(s), lipid and glucose levels all become important to monitor.

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