LITERATURE REVIEW

Cardiovascular Dysfunction in Spinal Cord Injury

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ABSTRACT

Cardiovascular functions ars altered by the impaired autonomic pathways in cervical and high thoracic spinal cord lesion, resulting in cardiovascular and thermoregulation dysfunction. The interruption of the motor and sensory functions in the high level spinal cord lesion also plays a role in lack of physical activity and reduce muscle metabolic mass, resulting in coronary heart disease and metabolic syndrome, thus premature mortality. Exercise program in these cases should be made specifically and individually related to the dysregulation of autonomic system and possibility impending autonomic dysreflexia.

INTRODUCTION

Spinal cord injury (SCI) is defined as an injury of the spinal cord which results in varying degree of motor and sensory impairment and autonomic dysfunction caudal to the level of injury.[1] It is classified by the American Spinal Injury Association (ASIA) using the International Standarts for Neurological Classification of Spinal Cord Injury (ISNCSCI). The loss of supraspinal control leads to a reduce in overall sympathetic activity below the level of injury and unopposed parasympathetic outflow through intact vagal nerve which commonly occurs in patients with spinal cord lesions at T-6 or higher. This condition may cause problems, such as hypotension (both supine and orthostatic), autonomic dysreflexia, and cardiac arrhythmias (including persistent bradycardia) in acute phase.[2] Cardiovascular dysfunction may be life-threatening and may exacerbate the neurological impairment due to SCI. One study found that all patients with cervical SCI ASIA A and B develop bradycardia, 68% develop arterial hypotension with 35% cases require vasopressors, and cardiac arrest occurs in 16% of them. From patients with cervical SCI with ASIA C and D, 35–71% develop bradycardia, but few have hypotension and require vasopressors. Thoracolumbar SCI related to bradycardia is encountered in 13–35% cases. The prevalence of autonomic dysreflexia varies between 48 to 90% of SCI above T6. Proper and comprehensive treatment of cardiovascular aspects in SCI should be performed immediately to prevent further complications which leads to mortality.[3]

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DISCUSSION

Alterations in Autonomic Nervous System After SCI

The control of cardiovascular function occurs

via a complex system of feedback loops that modulated by sympathetic and parasympathetic nervous system. Medial prefrontal cortex, insula, hypothalamus, and cuneiform nucleus project into cardiovascular nuclei within medulla oblongata.[3] Cardiac sympathetic impulses arise from T1–T4 spinal cord segments and parasympathetic impulses are carried out via vagus nerve. Disruptions of cardiovascular control following spinal cord injury are directly related to the level and degree of the injury. SCI above T1 interrupts the heart conducting system, thus the homeostasis is achieved via local and spinal reflex control. Injury between T1-T4 have partial innervation to the heart. If the level of SCI is below T4, normal cardiac response are maintained, but vascular tone and blood pressure control will be under local regulation. Sympathetic outflow to the splanchnic organs originates primarily from T5 to T9, thus injuries above T5 impairs the ability of splanchnic bed to vasodilate, reducing the ability of the blood to pool in the splanchnic circulation and may leads to autonomic dysreflexia due to lack compensatory response of blood pressure. Sympathetic response to events are not only regulated by the heart and vasculature, but also by the adrenal medulla that arise from T5 to T9 releasing hormones such as epinephrine. Injuries above T9 may impairs the sympathic response to exercise and stress.[1,2]

The initial response to cervical and high thoracic SCI is a short period of massive sympathetic stimulation and reflex parasympathetic activity that usually lasts for 3 to 4 minutes, mediated by alpha–adrenergic receptors. Patient may have reflexes of bradycardia or tachyarrhythmias and severe hypertension. The sympathetic stimulation is due to massive epinephrine and norepinephrine releasing from the suprarenal glands and disruption of cervical and high thoracic vasoactive neurons. After this immediate response to trauma, decrease in sympathetic activity occurs due to interruption of descending sympathetic pathways. Cardiac output and total peripheral resistance decrease, while central venous pressure remains unchanged. Patient is prone to bradycardia, hypotension, and hypothermia by lack of sympathetic activity and unopposed parasympathetic tone via vagal tone. Vagal stimulation may also depress cardiac function and impair ventricular filling by slowing atrioventricular conduction and altering the synchronicity of the atrioventricular contraction. The interruption of the communication between the supraspinal centers and peripheral sympathetic intermediolateral thoracic and lumbar neurons leads to spinal shock. It is a transitory condition of function and reflexes below the level of the injury which usually persist 4 to 6 weeks after injury. Appearance of bulbocavernous reflex, recovery of deep tendon reflexes or return of reflex detrusor functions are considered by different authors the endpoint of spinal shock.[1–3]

Other cardiovascular alteration after acute SCI include change in heart rate (HR) and cardiac output. The lower stroke volume is due to the decreased venous return from regions below the level of injury, which is lack of effective muscle pumping action and activation, and also lack of sympathetic vasoconstrictor tone. The elevated HR may compensate for the reduced stroke volume, but is not sufficient to compensate for the reduced sympatethic response. Orthostatic hypotension,that related to gravitation and rapid postural changes, occurs more frequent and worser in the morning on postural rising. Physical methods including repeated postural change on a tilt table or a high back reclining wheelchair, sleeping with bead head raised by 20 degrees, using compression wraps to the legs and abdominal binder using on sitting up position can be used to reduce orthostatic hypotension. Heavy meals may also induced orthostatic hypotension due to blood overflow to splanchnic circulation and can be minimized by consuming small frequent meals.[1]

Autonomic dysreflexia (AD) is a syndrome of massive imbalanced reflex sympathetic discharge in the thoracolumbar spinal cord, occurring in patients with SCI located above the splanchnic sympathetic outflow T5–T6, often triggered by the distension of pelvic viscera or other noxious stimuli. Viscerosensitive impulses below the level of injury are transmitted through intact peripheral sensory nerves, and release sympathetic reflex. Sympathetic hyperstimulation releases high

quantities of norepinephrine and dopamine, lead to massive vasoconstriction and arterial hypertension, but also cerebral vasodilatation. The brain perceives the hypertensive crisis throughout cervical baroreceptors. It generates inhibitory impulses that cannot be transmitted below the level of injury. Vasomotor centers from the medulla oblongata try to lower the arterial blood pressure by parasympathetic stimulation of the heart through nerve, generating severe bradicardia and also increasing parasympathetic activity above the level of lesion.[1,2] Classic signs and symptoms in AD include severe arterial hypertension, headache and visual impairment due to cerebral vasodilatation, cutaneous pallor and piloerection below the injury site, bradycardia, profuse sweating and cutaneous vasodilatation above the level of lesion, secondary to parasympathetic activity.[1] Arterial blood pressure can reach up to 300 mmHg, leading to retinal, intracerebral, or subarachnoid hemorrhage, pulmonary edema, myocardial infarction, seizures, confusion and death.[4] The appropriate tretment should be done immediately such as repositioning from lying supine to sit upright allowing orthostatic response, hemodynamic monitoring every 5 minutes over the next 2 hour following the resolution of acute AD, evacuating bowel and bladder, removing all constricting clothes and find the cause of noxious stimuli immediately.[1,2]

Physical Condition Related to Cardiovascular Dysfunction After SCI

Coronary heart disease and systemic atherosclerosis are common in chronic SCI with the prevalence of 30% to 50%. The risk factors including lack of physical inactivity and reduced muscle metabolic mass not only because of lack of motor function, but also due to lack of accessibility and fewer opportunities to engage in physical activity. This condition is associated with both the level of the SCI and clinical findings, and increases with increasing age, increasing rostral level of injury and the severity of the SCI (complete vs incomplete), and will lead to greater prevalence of obesity, lipid disorders, and metabolic syndrome, and latter will make premature mortality at earlier age than able-bodied. The prevalence of asymptomatic cardiovascular dysfunction after SCI was about 60% and it could appear due to the interruption of sensory pain fibers that normally describe cardiac ischemia and imminent cardiac dysfunction which may cause delayed treatment and poor prognostic in SCI related cardiovascular dysfunction. There is exercise-related vasodilation in working muscles while SCI have lack compensatory vasoconstriction in other muscles and organs below the level of injury. This results in an exercise-induced fall in blood pressure leading to critically low perfusion pressure in the working muscle and leads to physical exhaustion. During physical work, heat accumulates to a greater extent, may develop a paradoxical fall in blood pressure and rise in body temperature. The problem was the higher the level of injury, the less able the body is to regulate its temperature related on the injury level of the autonomic nervous system.

Research has shown that exercise after SCI can improve breathing ability, muscle strength and ability to ambulation and do work, circulation and immune system, body composition (more muscle, less fat), lipid profile, self-esteem and self-confidence, depression and anxiety, and also prevention of secondary conditions (pressure ulcers, urinary tract infections, respiratory infections).[5] The selection of an accessible and well-designed exercise mode for SCI group is important, either total or partial paralysis of the lower extremities limits regular use of ambulation, treadmills or other exercise modes that require weight bearing contraction of the lower extremities.[6] Body weight support is an option in SCI group, but requires some special equipment and/or several assistive personel. Upper extremity modes of exercise such as arm ergocycle, swimming, wheelchair locomotion (pushing, rowing, seated aerobics), or resistance activities are more reasonable and available exercise options. Functional electrical stimulation (FES) exercise is another option for tetraplegic group, and the combination between body weight support training with FES leads to increased metabolic and cardiorespiratory response in complete tetraplegic group. Cardiovascular deconditioning is reversible by doing exercise regularly, thus increasing vascular resistance in paralyzed legs.[7] The key

in selection of an exercise mode is the ability of the activity to hold the interest of the user on a regular basis. The compliance with long term exercise programs is influenced by the pleasure in exercise activities, availability of resources and reasonable cost.

In healthy person, the American College of Sports Medicine recommend to perform 30 to 60 minutes of physical activitiy with an intensity 50% to 80% of peak oxygen uptake on most days of the week (5-7 days/ week) may significantly improve physical fitness and to reduce cardiovascular disease risks. These guidelines do not consider that the upper extremities undergoing training will also be usefull for functional activities such as wheelchair propulsion.[5] Work performed by the upper extremity generally elicits higher heart rate (HR) response than lower extremity works. Person with SCI injuries above T1 level may have alter HR response to exercise. Moreover, it is difficult to attain the VO_2max based on the HR response to exercise. An optional measurement are focused on musculoskeletal fitness, such as time and distances, Borg scale (perceived exertion or RPE), or talk test to measuring exercise intensities in SCI groups. Karvonen method based on percentage of the HR reserve (HRmax-HRrest) with HR generally not exceed 120 bpm for those with injury above T1 is controversial in some study as it fails to consider the adrenergic dysfunction effects on peak HR during activity.[6] As in most studies, aerobic exercise two to three times weekly for 20 to 60 minutes each session in moderate intensity (40% to 59% HR reserve or 55 to 69% HR max) for a minimum of 6 weeks is sufficient to significantly increase cardiovascular fitness and exercise tolerance, and in some cases may improve the lipid profile in SCI group.[8]

Preservation of upper extremity function should be considered within exercise plan to avoid chronic fatigue and/or injury do not interrupt the consistency of activity and independent lifestyle.[6] To maintaining temperature dysregulation in SCI, it is important to exercising in a cool place, drinking water frequently, and being prepared with a spray bottle and a fan to prevent any body temperature increase.[6] Some types of exercise may worsen spasticity, thus doing stretch the spastic muscle groups before exercise and avoid the exercises tend to exacerbate or cause the spasms.[9] Pressure sores or abrasions can be caused by improper positioning for the exercise activity or by lack of cushioning, it is important to complete skin check routinely after doing exercise or activities and make adjustments as needed. The most important and life threatening condition is autonomic dysreflexia while not all that common during exercise, it could be a problem related to any noxious stimuli. Cardiovascular fitness is more difficult to reach with the higher level injury and cannot be gained in a hurry. It requires starting slowly and gradual increases in exercise intensity over a long period of time, at a level of moderate intensity that is more than that required for daily life. Another ways to increase cardiovascular endurance are by doing daily activities in a rhythmic manner and continously, maintaining the effort for at least twenty minute sessions three times a day.[9]

CONCLUSION

Cardiovascular dysfunctions in SCI result in cardiovascular and thermoregulation dysfunction. Lesions in high level spinal cord disrupts the motor and sensory functions cause physical inactivity that causes muscle metabolic mass degeneration and resulting in coronary heart disease and metabolic syndrome, thus premature mortality. Post-SCI exercises that are individually tailored, may improve breathing capacity, ambulation, circulation and immune system, muscle mass, lipid profile, psychological comditions, and prevent secondary diseases. Such challenging comprehensive exercise program that is executed by highly trained medical professionals is highly suggested. It is well-known that rapid Cardiovascular fitness is unfeasible in a person with SCI who has higher level injury. It requires long-term exercise with gradual increases in moderate exercise intensity that is required for daily life. Alternatively, performing daily activities in a rhythmic manner and continously regularly is also suggested.

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