Cardiovascular Complications during the Acute Phase of Spinal Cord Injury Executive Summary

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## 1.0 Executive Summary

Cardiovascular complications post SCI fall broadly into four categories; neurogenic shock, orthostatic hypotension, hemodynamic changes, and bradycardia and arrythmias. Cardiovascular complications are common following SCI due to secondary dysfunction of the autonomic nervous system (Malmqvist et al., 2015). Given cardiovascular control is heavily dependent on neural control from medullar and spinal cord circuits, it is highly likely that SCI patients will experience some cardiovascular dysfunction. Currently there is no consensus as to whether injury severity is related to cardiovascular dysfunction (West et al., 2013).

### Neurogenic Shock

Disrupted sympathetic and parasympathetic activity can lead to changes in arterial BP and HR. Neurogenic shock refers to the presence of bradycardia and arterial hypotension without a clear etiology (Furlan & Fehlings, 2008; Krassioukov, 2009; Popa et al., 2010). Bradycardia is defined as a HR of less than 60 beats per minute, and arterial hypotension is defined as a systolic BP below 90 mmHg and a diastolic BP of less than 60 mmHg (Popa et al., 2010; Wecht et al., 2013). Lower HR and lower BP is common among acute SCI patients due to reduced SNS activity. Initially post SCI, rates of neurogenic shock have been found to be anywhere between 7% and 24%, with 53% of cervical SCI patients presenting with neurogenic shock (Mallek et al., 2012; Ruiz et al., 2018). Treatments for neurogenic shock are primarily pharmacological. Vasopressor administration, in the form of pseudoephedrine, is the most commonly reported treatment, although there is a lack of strong evidence to support the continued use of pseudoephedrine as an intervention for neurogenic shock (Wood et al., 2014).

### Orthostatic Hypotension (OH)

OH is identified as a minimum decrease in systolic BP of 20 mmHg, or a minimum decrease of 10 mmHg in diastolic BP when patients are moved to an upright position from lying posture. Observed incidences of OH in the SCI population are between 60-70%, with those with cervical and upper thoracic injuries having slightly higher incidence rates. For the treatment of orthostatic hypotension, tilt tables in combination with functional electrical stimulation are the predominant non-pharmacological interventions. Combining tilt tables with FES has been shown to be effective for improving and raising BP in SCI populations significantly (Tesini et al., 2013; Elokda et al., 2000). Tilt tables alone have been shown to reduce BP further, so tilt tables should be used in combination with FES; otherwise, there is the risk of further reducing BP in patients.

### Hemodynamic Changes

Management of hemodynamic changes during the acute phase of SCI has focused on the effectiveness of early aggressive support on neurological outcomes. In general, hemodynamic management involves a multi-pronged approach which includes administering fluids, vasopressors, decompressive surgery, and pulmonary catheters. Two studies have shown that this type of aggressive treatment may be effective for improving neurological outcomes post SCI (Vale et al., 1997; Levi et al., 1993).

**Conclusions**

Incidences of cardiovascular complications post SCI are high, as vasculature control is provided by the T1 to L2 segments of the spinal cord. Proactively treating hemodynamic instability may improve neurological outcomes; treatment reacting to hemodynamic abnormalities post SCI has limited evidence and focuses on a combination of pharmacological and non-pharmacological treatments.