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Review Article

Management of Blood Pressure Disorders in Individuals with Spinal Cord Injury

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ABSTRACT

Spinal cord injury (SCI) often results in complex autonomic dysfunctions, particularly blood pressure (BP) disorders, due to impaired neural regulation. These include chronic hypotension, orthostatic hypotension (OH), and episodes of autonomic dysreflexia (AD), depending on the level and severity of the injury. This review explores the pathophysiology, clinical implications, diagnostic strategies, and management approaches for BP dysregulation in individuals with SCI. A multidisciplinary approach involving pharmacologic, non-pharmacologic, and rehabilitative strategies is essential to mitigate morbidity and enhance quality of life.

Keywords: Autonomic dysreflexia, Orthostatic hypotension, Spinal cord injury.

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INTRODUCTION

Spinal cord injury (SCI) affects over 27 million individuals globally, with an increasing prevalence due to improved survival rates. One of the most debilitating consequences of SCI is autonomic nervous system (ANS) dysfunction, especially related to cardiovascular regulation. Depending on the lesion's level and completeness, SCI can impair sympathetic outflow, leading to blood pressure instability. These disorders not only reduce quality of life but are also linked to increased cardiovascular morbidity and mortality.[1]

Pathophysiology of Blood Pressure Dysregulation in SCI

Blood pressure (BP) regulation is a tightly controlled process involving the ANS, which ensures adequate perfusion to vital organs in response to positional changes, physical activity, and stress. In individuals with spinal cord injury, especially those with cervical or upper thoracic lesions, this regulatory mechanism is severely disrupted. The resulting imbalance between sympathetic and parasympathetic output leads to various forms of BP instability, including orthostatic hypotension (OH), chronic hypotension, and autonomic dysreflexia (AD).

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Autonomic Nervous System Disruption

The autonomic nervous system is divided into sympathetic and parasympathetic divisions, which work in a coordinated fashion to maintain cardiovascular homeostasis. Sympathetic Outflow: Originates from the intermediolateral cell columns of the spinal cord segments T1 to L2. This system is primarily responsible for vasoconstriction, increasing heart rate, and raising blood pressure during stress or upright posture. Parasympathetic Control: Mainly mediated by the vagus nerve (cranial nerve X), which remains unaffected in SCI. It exerts a slowing effect on heart rate and has minimal influence on vascular tone.

In injuries above the T6 level, sympathetic nervous system control is disrupted, resulting in impaired vasomotor tone below the level of injury. The parasympathetic system, however, continues to function unopposed, especially in regulating the heart. This autonomic imbalance contributes significantly to the blood pressure dysregulation seen in SCI.[2]

Types of Blood Pressure Dysregulation in SCI

Orthostatic Hypotension (OH)

Orthostatic hypotension (OH) is defined as a sustained drop in systolic blood pressure of ≥20 mmHg or diastolic BP of ≥10 mmHg within 3 minutes of standing or sitting upright. In individuals with SCI, particularly those with high-level lesions, the normal sympathetic vasoconstrictive

response is impaired, leading to venous pooling in the lower limbs due to loss of vascular tone and muscle pump activity. Additionally, baroreceptor dysfunction and cardiovascular deconditioning from prolonged immobilization further exacerbate postural BP drops. Clinically, OH presents with symptoms such as dizziness, light-headedness, blurred vision, nausea, and syncope, significantly hindering rehabilitation efforts and reducing overall quality of life.[3]

Chronic Hypotension

Chronic hypotension in individuals with SCI, particularly those with cervical or high thoracic lesions, is characterized by a persistent resting systolic blood pressure below 90 mmHg. This condition arises from baseline sympathetic hypofunction, resulting in diminished vascular tone and systemic vascular resistance. Additionally, reduced cardiac output due to impaired stroke volume and heart rate responses, along with hypovolemia from decreased intake, diuresis, or renal dysfunction, further contributes to low BP. Clinically, chronic hypotension manifests as fatigue, reduced exercise capacity, cognitive impairment, and compromised cerebral perfusion, and may obscure signs of other cardiovascular issues or organ hypoperfusion, complicating overall clinical management.[4]

Autonomic Dysreflexia (AD)

Autonomic dysreflexia (AD) is a potentially life-threatening condition marked by sudden, severe hypertension, typically occurring in individuals with spinal cord injuries at or above the T6 level. It is triggered by stimuli such as bladder distension, bowel impaction, or skin irritation below the level of injury. These afferent signals ascend the spinal cord but are blocked by the lesion, provoking an unmodulated sympathetic reflex that causes widespread vasoconstriction and elevated blood pressure. The lack of inhibitory control from higher brain centers exacerbates the response, while the intact vagus nerve induces reflex bradycardia and vasodilation above the injury, resulting in symptoms like pounding headache, facial flushing, sweating, nasal congestion, and visual disturbances. If not promptly managed, AD can lead to serious complications including seizures, stroke, or death. The T6 level is significant because it marks the threshold above which supraspinal regulation of the splanchnic vascular bed—supplied by sympathetic fibers from T5–L2—is lost, increasing the risk for uncontrolled hypertensive episodes.

Understanding the distinct mechanisms underlying BP dysregulation in SCI is essential for timely recognition, accurate diagnosis, and effective management. The type of autonomic dysfunction depends heavily on the injury level and completeness, with cervical and upper thoracic lesions being most vulnerable. Clinicians must be vigilant in monitoring for signs of OH, chronic hypotension, and AD, as these not only reduce quality of life but can also lead to life-threatening complications if not promptly managed.[5]

CLINICAL EVALUATION AND DIAGNOSIS

Clinical evaluation and diagnosis of blood pressure dysregulation in individuals with SCI begin with a thorough history and symptom assessment. Key symptoms suggestive of BP instability include episodes of dizziness, headache, syncope, blurred vision, and excessive sweating, often linked to positional changes or specific triggers. It is essential to identify potential causes of AD, such as bladder distension, bowel impaction, or skin irritation. Blood pressure monitoring plays a critical role in diagnosis; 24-hour ambulatory blood pressure monitoring (ABPM) helps capture fluctuations and assess circadian patterns, while supine and upright BP measurements are vital for detecting OH. Additionally, BP should be monitored during known AD-inducing activities to identify reactive hypertensive episodes. Further investigations may include electrocardiogram (ECG) and echocardiography to assess cardiac function, tilt-table testing to evaluate orthostatic responses, and comprehensive autonomic function testing to characterize the extent of autonomic disruption.[6]

Management of Blood Pressure Disorders in Individuals with Spinal Cord Injury

General Principles

Effective management of blood pressure (BP) disorders in individuals with SCI requires a patient-centered and multidisciplinary approach. The first and foremost principle involves identifying and addressing the underlying causes contributing to dysregulated BP—whether they be related to infection, dehydration, bowel or bladder issues, or medication side effects. Management strategies must be tailored to the specific type of BP abnormality—OH, chronic hypotension, or AD—as each condition has distinct pathophysiological mechanisms and clinical implications. Equally important is patient and caregiver education, which plays a critical role in early recognition of warning signs and implementation of emergency protocols. Empowering patients to understand their condition and respond promptly can significantly reduce morbidity and improve quality of life.[7,8]

Management of Orthostatic and Chronic Hypotension

Non-Pharmacologic Approaches

Non-pharmacologic interventions form the cornerstone of initial management for both orthostatic and chronic hypotension in SCI. The use of compression garments, such as abdominal binders and thigh-high compression stockings, helps counteract venous pooling in the lower extremities and abdomen, thereby improving venous return and stabilizing BP. Patients are advised to change positions gradually—from lying down to sitting, and eventually to standing—to prevent sudden drops in BP. Physical maneuvers such as leg crossing, toe tapping, and arm tensing may be employed during symptoms to transiently increase BP. Maintaining adequate hydration and liberal salt intake (where not contraindicated) can expand intravascular volume and reduce hypotensive episodes. Additionally, tilt-table training has proven beneficial in improving orthostatic tolerance by retraining the autonomic nervous system and cardiovascular reflexes through progressive, supervised upright positioning.[9]

Pharmacologic Treatments

When non-pharmacologic strategies are insufficient, pharmacologic therapy may be introduced. Midodrine, an alpha-1 adrenergic agonist, is often the first-line drug as it promotes peripheral vasoconstriction and raises BP effectively. Fludrocortisone, a mineralocorticoid, enhances sodium retention, thus expanding plasma volume and improving BP stability. Droxidopa, a synthetic precursor of norepinephrine, helps restore sympathetic tone and is particularly effective in patients with neurogenic OH. Pyridostigmine, a cholinesterase inhibitor, augments cholinergic transmission at the autonomic ganglia and may provide additional benefit,

especially when combined with other agents. These drugs should be started at low doses and titrated carefully, with close monitoring of side effects, such as supine hypertension and electrolyte imbalances.[10-12]

Management of Autonomic Dysreflexia (Ad)

Immediate Treatment

AD is a medical emergency requiring prompt intervention. At the first sign of AD, the patient should be placed in an upright sitting position to harness gravitational effects in lowering BP. Identifying and removing the inciting stimulus is the next critical step. Common culprits include bladder distension, fecal impaction, tight clothing, or skin injuries. The urinary catheter should be checked for blockage or kinking, and bowel impaction should be assessed. Blood pressure must be monitored every 2 to 5 minutes until normalization is achieved.[13]

Pharmacologic Options

If the BP remains dangerously elevated despite removing the trigger, pharmacologic intervention becomes necessary. Nifedipine (bite-and-swallow form) provides rapid and effective BP control, making it suitable for outpatient settings. In emergency scenarios, topical nitroglycerin paste can be applied for quick vasodilation. For severe or refractory hypertension, intravenous agents such as hydralazine or labetalol may be administered in a hospital setting under close supervision. Care should be taken to avoid overshooting BP reduction, which may lead to hypotension and cerebral hypoperfusion.[14]

Prevention

Preventive strategies are crucial to minimizing the frequency and severity of AD episodes. A structured bladder and bowel care regimen, including regular catheterization and bowel programs, is fundamental. Avoiding noxious stimuli—such as pressure ulcers, ingrown toenails, and tight clothing—is equally important. Long-term prevention also involves educating patients and caregivers about potential triggers, warning symptoms, and appropriate steps to take during an episode. Wearing medical alert identification can help ensure appropriate treatment is provided during emergencies by non-specialist healthcare providers.[15]

Long-term Cardiovascular Monitoring

Individuals with SCI and BP instability are at an increased risk of developing long-term cardiovascular complications, including coronary artery disease, stroke, and heart failure. Therefore, routine cardiovascular screening is recommended. This includes periodic assessments of lipid profiles, fasting glucose levels, and electrocardiograms (ECG) to detect early signs of atherosclerosis or arrhythmias. Encouraging safe physical activity and participation in structured rehabilitation programs can significantly improve autonomic regulation, vascular tone, and overall cardiovascular health. Rehabilitation efforts should include tailored aerobic and resistance training regimens that accommodate the patient's level of injury while aiming to restore cardiovascular reflexes and functional independence.[16-21]

FUTURE DIRECTIONS AND RESEARCH

Emerging innovations in the management of blood pressure disorders in SCI are revolutionizing care. Neuromodulation therapies, such as epidural spinal cord stimulation, have shown promise in partially restoring autonomic cardiovascular control. Wearable technology, including continuous blood pressure monitors, enables real-time detection of BP fluctuations and early intervention. The integration of telemedicine allows for remote monitoring and personalized management plans, improving access and adherence. Additionally, pharmacogenomics is paving the way for tailored pharmacologic treatments based on individual genetic profiles, enhancing efficacy and minimizing side effects.

CONCLUSION

Blood pressure disorders in SCI patients are multifactorial and pose significant risks, if not properly managed. A structured, individualized approach—incorporating preventive care, non-pharmacologic techniques, and medication—is critical. Ongoing research and emerging technologies offer new hope for improving autonomic regulation and quality of life in this vulnerable population.

REFERENCES

1. McDaid D, Park AL, Gall A, Purcell M, Bacon M. Understanding and modelling the economic impact of spinal cord injuries in the United Kingdom. Spinal Cord. 2019;57(9):778–88.
2. Wulf MJ, Tom VJ. Consequences of spinal cord injury on the sympathetic nervous system. Front Cell Neurosci. 2023;17:999253.
3. Nightingale TE, Zheng MMZ, Sachdeva R, Phillips AA, Krassioukov AV. Diverse cognitive impairment after spinal cord injury is associated with orthostatic hypotension symptom burden. Physiol Behav. 2020;213:112742.
4. Use of droxidopa for BP augmentation after acute SCI. Acute Crit Care. 2021;36(2):217–24.
5. Lakra C, Swayne O, Christofi G, Desai M. Autonomic dysreflexia in spinal cord injury. Pract Neurol. 2021;21(6):532–8.
6. Evaluation and management of autonomic functions in patients with SCI. J Spinal Cord Med. 2025 Feb; [Review].
7. Kulkarni S, Jenkins D, Dhar A, Mir F. Treating lows: management of orthostatic hypotension. J Cardiovasc Pharmacol. 2024;84(3):303–15.
8. Krassioukov A, Linsenmeyer TA, Beck LA, Elliott S, Grimm DR, Johns JS, et al. Evaluation and management of autonomic dysfunctions after spinal cord injury. J Spinal Cord Med. 2021;44(4):631–83.
9. Philips AA, Krassioukov AV. Contemporary cardiovascular concerns after spinal cord injury: mechanisms, maladaptations, and management. J Neurotrauma. 2021;38(12):1685–702.
10. Kulkarni S, Jenkins D, Dhar A, Mir F. Treating lows: management of orthostatic hypotension. J Cardiovasc Pharmacol. 2024;84(3):303–15.
11. Zheng AC, Fong KT, Kline I, Curtis J, Raj SR. Real-world droxidopa or midodrine persistence in patients with neurogenic orthostatic hypotension. Auton Neurosci. 2020;225:102–9.
12. OpenHeart Editorial Team. Pyridostigmine in the management of orthostatic hypotension. Open Heart. 2025;12(1):e003106.
13. Kiser T, Lindberg R, Adeogba S. Management of acute autonomic dysreflexia: spinal cord injury guidelines 2022. UAMS Brain Injury Program. Published July 2022. Available from: https://pva.org
14. Lakra C, Swayne O, Christofi G, Desai M. Autonomic dysreflexia in spinal cord injury. Pract Neurol. 2021;21(6):532–8.
15. Krassioukov A, Warburton DE, Teasell R, Eng JJ. Autonomic dysreflexia in spinal cord injury: Clinical practice guidelines for management. Spinal Cord. 2021;59(12):1209–22.

16. Singh R, Roy S, Ghildiyal A, Verma S. Association of Anthropometry With Nerve Conduction Parameters of Median Nerve: A Cross-Sectional Study in a North Indian Medical University Hospital. Cureus. 2024 Jul 6;16(7):e63946. doi: 10.7759/cureus.63946. PMID: 39105004; PMCID: PMC11299048.
17. Tiwari R, Singh S, Bajpai M, Verma N, Verma S. Impact of Osteocalcin on Glycemic Regulation and Insulin Sensitivity in Type 2 Diabetes Mellitus Patients. Cureus. 2024 Oct 17;16(10):e71675. doi: 10.7759/cureus.71675. PMID: 39553160; PMCID: PMC11568420.
18. Tiwari R, Singh N, Singh S, Bajpai M, Verma S. Interplay of Adiponectin With Glycemic and Metabolic Risk Metrics in Patients With Diabetes. Cureus. 2024 Sep 30;16(9):e70543. doi: 10.7759/cureus.70543. PMID: 39479098; PMCID: PMC11524515.
19. Tiwari R, Verma S, Verma N, Verma D, Narayan J. Correlation of serum uric acid levels with certain anthropometric parameters in prediabetic and drug-naive diabetic subjects. Ann Afr Med. 2024 Jan-Mar;23(1):13-18. doi: 10.4103/aam.aam_40_22. PMID: 38358165; PMCID: PMC10922179.
20. Verma S, Tiwari R, Verma N, Singh S, Sharma A. Anthropometry and blood biomarkers of diabetes and their possible association with obesity and metabolic syndrome. J Diabetes Metab Disord. 2023 Oct 11;23(1):509-517. doi: 10.1007/s40200-023-01276-4. PMID: 38932840; PMCID: PMC11196461.
21. Solinsky R, Burns K, Tuthill C, Hamner JW, Taylor JA. Transcutaneous spinal cord stimulation and its impact on cardiovascular autonomic regulation after spinal cord injury. Am J Physiol Heart Circ Physiol. 2024;326(1):H116–22.

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