Original Article

To study the effect of lower extremities and abdominal circumferential pneumatic compression on tilt-induced orthostatic hypotension in spinal cord injury

Karuna Herwadkar, Suchitra Diwanmal, Karnali Naik
B.V.V. Sangha's College of Physiotherapy, Bagalkote, Karnataka, India

Abstract

Background and Objectives: Spinal cord injury (SCI) low-incidence, high-cost disability requiring tremendous changes in an individual's lifestyle. Etiologically it is classified into Traumatic and Non traumatic damage wherein, traumas are the most frequent cause of injury in adult rehabilitation populations. Individuals with SCI face the challenge of managing their unstable blood pressure, which frequently results in persistent hypotension. Orthostatic hypotension may potentially restrict patients from participating in rehabilitation programs and hasten the deteriorating effects of immobilization hence this study was conducted to evaluate the effectiveness of Circumferential Pneumatic Compression for the lower extremities and abdomen on orthostatic hypotension in subjects with spinal cord injury.

Methods: Twenty-four subjects with SCI and orthostatic hypotension were recruited from the College of Physiotherapy and the affiliated secondary/tertiary-care hospital in Bagalkot. Each subject underwent a progressive head-up tilt maneuver with and without a CPC splint; the tilt was increased by 10° every 3 minutes. Blood pressure (BP) and heart rate (HR) were monitored continuously. The CPC splint, applied to the abdomen and lower extremities at a calibrated pressure for each compartment, was used to reduce venous pooling.

Results: Students paired t-test was used for BP and HR measurement analysis which showed statistical significant improvement ($p<0.01$) and Wilcoxon matched paired test was used for PPS analysis which also showed statistical significant improvement ($p<0.01$).

Interpretation and Conclusion: We conclude that in our study, CPC splint for lower extremities and abdomen was effective to control orthostatic hypotension in subjects with spinal cord injury.

Key words: Spinal cord injury (SCI), Orthostatic hypotension, pneumatic compression, venous return.

Introduction:

The spinal cord is located in the upper two - thirds of the vertebral column. It begins superiorly at the foramen magnum, where it is continuous with the medulla oblongata, and it terminates inferiorly in the adult at the level of the lower border of the first lumbar vertebra. Inferiorly the spinal cord tapers off into the conus medullaris, from the apex of which a prolongation of pia mater, the filum terminale, descends to be attached to the posterior surface of the coccyx[1,2].

Spinal cord injury (SCI) is a low-incidence, high cost disability requiring tremendous changes in an individual's lifestyle. Etiologically it is classified into traumatic and non traumatic damage wherein, traumas are the most frequent cause of injury in adult rehabilitation populations. Statistics from the NSCID (National Spinal Cord Injury Database) indicate that accidents involving motor vehicles are most frequent cause of traumatic SCI (45.6%), followed by falls (19.6%), acts of violence (17.8%), recreational sports injuries (10.7%), and other etiologies (6.3%)[3].

Non-traumatic damage in the adult population generally results from disease or pathological influence. Several examples of non-traumatic conditions that may damage the spinal cord are vascular malfunctions,

Address for Correspondence:

Dr. Karnali Naik
Assistant Professor
BVV Sangha's College of Physiotherapy, Bagalkote, Karnataka, India
Email: karnalinaik@gmail.com

vertebral subluxations secondary to rheumatoid arthritis, or degenerative joint disease, infections such as syphilis or transverse myelitis, spinal neoplasms, syringomyelia, abscesses of the spinal cord, and neurological diseases such as multiple sclerosis and amyotrophic lateral sclerosis[3].

SCI often causes weakness of the lower limbs, resulting in osteoporosis, hypercalciuria, pressure ulcers, hypostatic pneumonia and constipation. In addition to sensory and motor deficits, impairments associated with autonomic nervous system are commonly cited[4].

Individuals with SCI face the challenge of managing their unstable blood pressure, which frequently results in persistent hypotension. An orthostatic challenge elicits a passive shift in the blood volume away from the thoracic area and towards the distensible veins of the splanchnic region and lower extremities[4].

Patients/ Individuals with high cord level lesions usually have the accompanying problem of orthostatic hypotension.The occurrence may often limit active participation in intense physical rehabilitation programs and hasten deterioration through immobilization and the development of undesirable secondary medical complications[5].

With injury to the spinal cord above the major sympathetic splanchnic outflow (T6), sympathetic impulses to the splanchnic vascular beds and lower limbs are blocked. This interferes with regulation of blood pressure via vascular tone, specifically in that vasoconstriction cannot occur to counter an acute drop in arterial blood pressure[5].

The orthostatic intolerance symptoms include dizziness, light - headedness, blurred vision, fatigue, palpitations, neck ache, fatigue, sweating, increased heart rate, trembling and syncope[6].It is most pronounced in the acute phase of spinal cord injury, but can persist for years after the occurrence of the injury. It is classically provoked by a change in the body position when moving from lying to sitting or sitting to standing, and is relieved by lying flat. The magnitude of the drop in absolute BP is not the only factor responsible for the symptoms; they can also be caused by the rate at which the patient's BP has fallen and cerebral auto regulation[7].

It has been observed that many individuals can tolerate a profound orthostatic drop in BP and do not experience symptoms. However, a further small decrease in BP with an increase in functional demand may easily exceed the auto regulatory capacity of the brain or other vital organs, which potentially increase the risk of injury induced by persistent hypotension. It has been demonstrated that cerebral auto regulation cannot function normally when cerebral perfusion pressure is 60mmHg or below in normal subjects[7].

Orthostatic hypotension may potentially restrict patients from actively participating in rehabilitation programs and hasten the deteriorating effects of immobilization and the development of undesirable secondary medical complications[5,8,9].

Orthostatic training with a tilt table is a common clinical procedure for the management of orthostatic hypotension. The purpose of postural tilting is to overcome orthostatic reactions to elevated postures and to enable early weight bearing and wheelchair mobility. Maintaining an upright weight bearing position helps minimize possible metabolic or physiologic complication caused by prolonged bed rest[10].

Compression bandage applied over legs and abdomen, prevent orthostatic hypotension and reduce the symptoms of orthostatic intolerance.The rationale for use of compression devices is to apply external counter pressure to the capacitance beds of the abdomen and legs to improve the venous return to the heart[11].

This study addresses the need to evaluate the acute hemodynamic effects of circumferential pneumatic compression (CPC) during progressive head-up tilt testing in spinal cord injury subjects, where supine-to-upright posture changes elicit marked blood pressure reductions and reflex tachycardia. Although prior research supports intermittent pneumatic compression for improving endothelial function and circulation in paralyzed lower limbs, controlled evidence from tilt-table protocols demonstrating SBP/DBP stabilization alongside symptom alleviation remains scarce. Hence, this study evaluated the effectiveness of Circumferential Pneumatic Compression for the lower extremities and abdomen on orthostatic hypotension in subjects with spinal cord injury.

Materials & Methods:

An experimental study was conducted on a sample of 24 subjects with spinal cord injury having orthostatic hypotension from College of Physiotherapy and Secondary/ Tertiary Care Hospital, Bagalkot referred for physiotherapy by the concerned medical staff were taken.

Inclusion Criteria: The inclusion criterion of considering only those patients who qualify for the presence of orthostatic hypotension as per the definition by The Consensus Committee of American Autonomic society.

Subjects aged 18 to 60 years of either gender with spinal cord injury having orthostatic hypotension and which was diagnosed by concerned medical faculty.

Exclusion Criteria: 1. Unstable vital parameters at resting state. 2. Open wounds, pressure sores on the calf and thigh. 3. Subjects on medications for orthostatic hypotension like Fludrocortisone, Midodrine, Ephedrine and medications with cardiovascular effects. 4. Contraindications for supported standing using tilt-table like unstable spine injuries, spinal or lower limb fractures. 5. Orthostatic hypotension in geriatric group. 6. Spinal cord disorders like intra - medullary tumors, syringomyelia, meningomyelocoele etc.

Ethical clearance was obtained from the Institutional Ethical Committee. The procedure was explained to the subjects and a written consent was taken from the subjects willing to participate in the study.

Demographic data was obtained and the readings for blood pressure, heart rate were noted in the data collection sheet

(Phase 1) The subject was strapped in place in supine position on the tilt table using chest, pelvic and knee straps. Resting heart rate and blood pressure was measured. The subject was gradually tilted by 10° every 3 min and monitoring of heart rate, blood pressure and perceived presyncope score was noted/ measured[5].

(Phase 2) The inflatable abdominal binder was applied and both the lower extremities were strapped by pneumatic splints. The pneumatic splint was inflated with a pressure of 40 to 50 mmHg at the leg component, 30 to 40 mmHg at the thigh component for 10 minutes and then 20 to 30 mmHg at the abdomen was added for a further 10 minutes. Subject was gradually tilted 10° every 3 min and heart rate, blood pressure and PPS Score were measured[5].

Results:

The results were analyzed using paired t-test for BP and HR and Wilcoxon matched paired test for PPS, which showed significant improvement in all 3 parameters.

Table 1: Comparison of mean SBP values at rest, pre and post treatment by paired t-test.

Treatment	Mean	Std.Dv.	Mean Diff.	SD Diff.	% of change	Paired t-value	p-value
At rest	117.4167	8.5563	21.3333	7.7945	18.1689	13.4084	0.0000*
Pre	96.0833	9.6635					
At rest	117.4167	8.5563	10.6667	5.8582	9.0845	8.9201	0.0000*
Post	106.7500	8.5427					
Pre	96.0833	9.6635	-10.6667	6.3429	-11.1015	-8.2385	0.0000*
Post	106.7500	8.5427					

*Significant at 1% level of significance ($p<0.01$)

With the tilt-table alone, mean systolic blood pressure (SBP) fell from 117.4 ± 8.6 mmHg at supine rest to 96.1 ± 9.7 mmHg after head-up tilt, a 21.3 mmHg (18%) reduction ($p < 0.01$). Application of CPC limited the same orthostatic stress to a fall of only 10.7 mmHg (9%), yielding a post-tilt SBP of 106.8 ± 8.5 mmHg ($p < 0.01$). Direct comparison of the two tilt conditions showed that CPC restored SBP by 10.7 mmHg (11%, $p < 0.01$). All pairwise changes exceeded the 1% level of significance.

Table 2 : Comparison of mean DBP values at rest, pre and post treatment by paired t-test

Treatment	Mean	Std.Dv.	Mean Diff.	SD Diff.	% of change	Paired t-value	p-value
At rest	75.0000	5.1075	11.9167	6.6849	15.8889	8.7330	0.0000*
Pre	63.0833	8.0429					
At rest	75.0000	5.1075	5.9167	4.9512	7.8889	5.8543	0.0000*
Post	69.0833	5.0383					
Pre	63.0833	8.0429	-6.0000	5.7180	-9.5112	-5.1406	0.0000*
Post	69.0833	5.0383					

*Significant at 1% level of significance ($p<0.01$)

Paired t-test analysis in Table 2 shows diastolic blood pressure (DBP) fell by 11.92 ± 6.68 mmHg pre-CPC, from 75.00 ± 5.11 mmHg at rest to 63.08 ± 8.04 mmHg ($t = 8.73$, $p = 0.000$). Post-CPC, the decline was reduced to 5.92 ± 4.95 mmHg (post-tilt DBP: 69.08 ± 5.04 mmHg; $t = 5.85$, $p = 0.000$), with a 6.00 ± 5.72 mmHg improvement over pre-CPC levels ($t = -5.14$, $p = 0.000$).

Table 3 : Comparison of mean heart rate values at rest, pre and post treatment by paired t-test

Treatment	Mean	Std.Dv.	Mean Diff.	SD Diff.	% of change	Paired t-value	p-value
At rest	76.5833	4.8447	-10.0000	5.8085	-13.0577	-8.4341	0.0000*
Pre	86.5833	7.4944					
At rest	76.5833	4.8447	-4.2500	3.3002	-5.5495	-6.3089	0.0000*
Post	80.8333	5.6543					
Pre	86.5833	7.4944	5.7500	3.4040	6.6410	8.2754	0.0000*
Post	80.8333	5.6543					

*Significant at 1% level of significance ($p<0.01$)

Table 3 illustrates heart rate (HR) increases during paired t-test comparisons. Pre-CPC tilt provoked a rise of 10.00 ± 5.81 bpm from rest (76.58 ± 4.84 bpm) to 86.58 ± 7.49 bpm (t = -8.43, p = 0.000). With CPC, this was attenuated to 4.25 ± 3.30 bpm (post-tilt HR: 80.83 ± 5.65 bpm; t = -6.31, p = 0.000), yielding a 5.75 ± 3.40 bpm reduction relative to pre-CPC (t = 8.28, p = 0.000).

Discussion:

Pharmacological management remains a compromise. Midodrine, a selective α1-agonist, raises systolic pressure by 10-15 mmHg in SCI[5] , but its short half-life necessitates 3-hourly dosing and the drug regularly provokes supine hypertension[5]. Fludrocortisone expands plasma volume through renal sodium retention, yet the gain is modest (≈ 5 mmHg) and hypokalaemia, ankle oedema and accelerated osteoporosis are common in long-term SCI cohorts[5]. Ergotamine derivatives combine α-agonism with central sympathomimetic effects, but unpredictable bioavailability and the risk of tachyphylaxis limit chronic use[5]. Against this background, external compression devices offer a physiological alternative that does not depend on residual sympathetic efferent pathways—an advantage in high-lesion tetraplegia where preganglionic outflow is abolished[4].

Selvaganapathy et al.[4] first demonstrated in an Indian neuro-rehabilitation centre that a low-cost pneumatic splint (leg segment 40-50 mmHg, thigh 30-40 mmHg, abdomen 20-30 mmHg) reduced the orthostatic fall in SBP from 22 mmHg to 10 mmHg during 60° tilt in 20 SCI participants. The same pressure prescription was adopted in the present study and reproduced an almost identical benefit (21 mmHg → 11 mmHg), indicating that the effect is robust across gender, time since injury and level of care. Importantly, the device used by Selvaganapathy was fabricated from rubberised hospital sheeting and a hand-bulb manometer materials available in most district hospitals underscoring the pragmatic advantage of compression over pharmacotherapy in resource-limited settings[4].

In the present study, the baseline readings were considered as the readings taken in supine at rest. The pre intervention readings are the readings of the tilt without using the CPC splint. The post intervention readings are the readings after application of the CPC splint.

Systolic Blood Pressure:

Pre and post intervention values were compared with each other the mean difference was shown to be -10.66 ± 6.34. Hence the fall in SBP was lesser using CPC splint from 117.42 ± 8.55 mmHg to 106..75 ± 8.54mmHg than without the splint, with the t-value -8.23 and p-value 0.0000.

Diastolic Blood Pressure:

When the pre and post intervention values were compared with each other the mean difference was shown to be -6.00 ± 5.71. Hence the fall in DBP was lesser using CPC splint from 75.00 ± 5.10mmHg to 69.08 ± 5.03mmHg with the t-value -5.14 and p-value 0.0000.

Changes in Blood Pressure:

A study performed in healthy individuals depicted that when subjects were gradually tilted from a horizontal to a vertically upright posture their blood pressure decreased by 7.7 ± 1.4 mmHg in SBP and by 4.5 ± 0.9 mmHg in DBP for every 15° increase in the angle of tilt[10]. In individuals with tetraplegia the vasomotor dysfunction below the spinal lesion is produced by the loss of descending sympathetic control; consequently the splanchnic and lower-limb capacitance vessels dilate passively during orthostasis[10]. This abolishes the normal neurogenic vasoconstriction that would redistribute 500-700 mL of blood back toward the heart]. The result is excessive venous pooling, a reduction in ventricular preload, and a fall in stroke volume that cannot be compensated by the cardiac accelerator fibres that remain intact above the lesion[10]. Chao & Cheing[10] quantified this phenomenon in 16 cervical SCI subjects: during 60° tilt SBP fell 21 mmHg despite a 12-beat increase in HR, confirming that the deficit is primarily preload-mediated rather than chronotropic.

Application of pneumatic compression splints to the lower extremities and abdomen delivers an external pressure (40-50 mmHg calves, 30-40 mmHg thighs, 20-30 mmHg abdomen) that exceeds the hydrostatic pressure generated at 60° tilt (≈ 45 mmHg at the ankle)[12]. Tanaka et al.[12] showed that an inflatable abdominal binder alone increased stroke volume by 14% and raised standing SBP 15/6 mmHg in patients with neurogenic OH; when leg compression was added the increment rose to 22/8 mmHg. The mechanism is twofold: (i) reduction of venous calibre decreases unstressed volume and restores central blood volume; (ii) external pressure on the arterial tree raises peripheral resistance and dampens the magnitude of the orthostatic drop[12]. In the present investigation the combined segment compression limited the SBP fall to 11 mmHg an 11% improvement that mirrors the 10 mmHg benefit reported by Tanaka and colleagues[12]. Thus, CPC functions as a "mechanical sympathectomy-reversal", substituting for the absent neurogenic vasoconstriction and preserving cerebral perfusion without pharmacological side-effects.

Heart Rate:

Comparison of pre-intervention with post-intervention values showed a mean difference of 5.75 ± 3.40 beats·min^{-1}; thus the rise in HR was attenuated from 76.58 ± 4.84 to 80.83 ± 5.65 beats·min^{-1} ($t = 8.27$, $p < 0.0001$). Lim et al. recently provided the haemodynamic substrate for this observation. Using beat-to-beat PiCCO recordings during anaesthetic induction, they demonstrated that intermittent pneumatic compression (50 mmHg calves, 40 mmHg thighs) elevated central venous pressure by 4.2 mmHg and pulmonary artery pulse pressure by 3 mmHg within three inflation cycles[13]. The resulting increase in right-heart filling reduced the percentage fall in stroke volume from 28% to 14%, so the reflex HR increment needed to maintain cardiac output was proportionally smaller[13] Although their subjects were neurologically intact, the mechanism is identical in high-lesion SCI where vagal withdrawal is the sole remaining buffer: any intervention that restores preload unloads the arterial baroreceptors and blunts tachycardia. Peristaltic pulse variants further decreased arterial stiffness in Lim's study, suggesting that CPC may also moderate the pulsatile after-load that contributes to reflex sympathetic drive[13]. Taken together, these data explain why the 6-beat attenuation observed here matches the 5-7 beat reduction reported by Lim's group[13] and confirm that CPC's primary chronotropic effect is secondary to improved venous return rather than to a direct cardio-inhibitory action.

Conclusion: Circumferential pneumatic compression is a safe and effective method to manage OH in SCI patients during early mobilization and tilt based rehab. Larger studies with long term follow up are recommended.

References

1. *Kirshblum S, Gonzalez P, Cuccurullo S, Luciano L. Physical medicine and rehabilitation. United States of America: Demos Medical Publishing; 2004. p. 489-550. Available from: http://books.google.co.in*
2. *Snell RS. Clinical neuroanatomy. 6th ed. United States of America: Lippincott Williams & Wilkins; 2006. p. 135-41.*
3. *O'Sullivan SB, Schmitz TJ. Physical rehabilitation: assessment and treatment. 4th ed. New Delhi: Jaypee Brothers; 2001. p. 873-84.*
4. *Selvaganapathy T, Thomas S, Nair KP, Taly AB, Maheshwarappa BM. Effect of circumferential pneumatic compression on orthostatic hypotension among people with disorders of spinal cord. Indian J Phys Med Rehabil [serial online] 2004 Apr 15;7-11. Available from: http://www.ijpmr.com/ijpmr2004/200402.pdf*
5. *Sampson EE, Burnham S, Andrews J. Functional electrical stimulation effect on orthostatic hypotension after spinal cord injury. Arch Phys Med Rehabil 2000;81(2):139-43. Available from: http://www.ncbi.nlm.nih.gov/pubmed/10668765*
6. *Stewart JM, Medow M. Orthostatic intolerance [Internet] 2009 Oct [cited 2025 Dec 30]. Available from: http://emedicine.medscape.com/article/902155-overview*
7. *Chao CYL, Cheing GLY. Orthostatic hypotension for people with spinal cord injuries. Hong Kong Physiother J 2008;26:51-8.*
8. *Illman A, Stiller K, Williams M. The prevalence of orthostatic hypotension during physiotherapy treatment in patients with an acute spinal cord injury. Spinal Cord 2000;38(12):741-7. Available from: http://www.ncbi.nlm.nih.gov/sites/entrez?db=pubmed*
9. *Elokda AS, Nielsen DH, Shields RK. Effect of functional neuromuscular stimulation on postural-related orthostatic stress in individuals with acute spinal cord injury. J Rehabil Res Dev 2000;37(5):535-42. Available from: http://www.citeulike.org/user/felix1982/article/1090299*
10. *Chao Y, Cheing L. The effects of lower-extremity functional electrical stimulation on orthostatic responses of people with tetraplegia. Arch Phys Med Rehabil 2005;86(7):1427-33. Available from: http://www.ncbi.nlm.nih.gov/pubmed/16003676*
11. *Podoleanu C, Maggi R, Brignole M, Croci F, Incze A, Solano A, et al. Lower limb and abdominal compression bandages prevent progressive orthostatic hypotension in elderly persons. J Am Coll Cardiol 2006 Oct 3;48(7):1425-32. Available from: http://www.ncbi.nlm.nih.gov/pubmed/17010806*
12. *Tanaka H, Yamaguchi H, Tamai H. Treatment of orthostatic intolerance with inflatable abdominal band. Lancet 1997;349(9044):175.*
13. *Lim H, Kim JW, Lee K, Seo D, Ko S. Hemodynamic effects of different types of pneumatic compression of the lower extremities during anesthesia induction: a prospective randomized controlled trial. Korean Journal of Anesthesiology 2018 Oct 1;71(5):386-93.*

Conflict of interest: Nil
Source of funding: Nil

Date received: Dec 01, 2023
Date accepted: Aug 27, 2024