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Effects of Patient-Controlled Abdominal Compression on Standing Systolic Blood Pressure in Adults With Orthostatic Hypotension

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Abstract

Objective—To assess the effects of patient-controlled abdominal compression on postural changes in systolic blood pressure (SBP) associated with orthostatic hypotension (OH). Secondary variables included subject assessments of their preferences and the ease-of-use.

Design—Randomized crossover trial.

Setting—Clinical research laboratory.

Participants—Adults with neurogenic OH (N=13).

Interventions—Four maneuvers were performed: moving from supine to standing without abdominal compression; moving from supine to standing with either a conventional or an adjustable abdominal binder in place; application of subject-determined maximal tolerable abdominal compression while standing; and while still erect, subsequent reduction of abdominal compression to a level the subject believed would be tolerable for a prolonged period.

Main Outcome Measures—The primary outcome variable included postural changes in SBP. Secondary outcome variables included subject assessments of their preferences and ease of use.

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Results—Baseline median SBP in the supine position was not affected by mild (10mmHg) abdominal compression prior to rising (without abdominal compression: 146mmHg; interquartile range, 124–164mmHg; with the conventional binder: 145mmHg; interquartile range, 129–167mmHg; with the adjustable binder: 153mmHg, interquartile range, 129–160mmHg; $P=.85$). Standing without a binder was associated with an -57 mmHg (interquartile range, -40 to -76 mmHg) SBP decrease. Levels of compression of 10mmHg applied prior to rising with the conventional and adjustable binders blunted these drops to -50 mmHg (interquartile range, -33 to -70 mmHg; $P=.03$) and -46 mmHg (interquartile range, -34 to -75 mmHg; $P=.01$), respectively. Increasing compression to subject-selected maximal tolerance while standing did not provide additional benefit and was associated with drops of -53 mmHg (interquartile range, -26 to -71 mmHg; $P=.64$) and -59 mmHg (interquartile range, -49 to -76 mmHg; $P=.52$) for the conventional and adjustable binders, respectively. Subsequent reduction of compression to more tolerable levels tended to worsen OH with both the conventional (-61 mmHg; interquartile range, -33 to -80 mmHg; $P=.64$) and adjustable (-67 mmHg; interquartile range, -61 to -84 mmHg; $P=.79$) binders. Subjects reported no differences in preferences between the binders in terms of preference or ease of use.

Conclusions—These results suggest that mild (10mmHg) abdominal compression prior to rising can ameliorate OH, but further compression once standing does not result in additional benefit.

Keywords

Orthostatic intolerance; Rehabilitation

Orthostatic hypotension (OH) is a disabling condition that is thought to affect as many as 5% to 30% of adults aged >65 years.¹ The range of its etiologies is wide and includes but is not limited to medication effects, cardiac impairment, neurogenic causes (eg, autonomic failure, spinal cord injury), hypovolemia, and deconditioning/prolonged bed rest. Symptoms may be as mild as transient lightheadedness or as severe and disabling as frank syncope. Although OH is typically defined as a >20 mmHg drop of systolic blood pressure (SBP) or a 10mmHg drop of diastolic blood pressure (DBP) within 3 minutes of standing,² its fundamental cause is the inability of the body to maintain a sufficient cardiac output during orthostatic stress. Treatment, whether with dietary changes, medication, or compression garments, often remains unsatisfactory.

Venous pooling has been identified as a major contributor to the pathophysiology of the condition.³ Two treatment approaches to improve venous return to the heart are in common use. The first uses dietary and pharmacologic manipulation to increase either the vascular system's blood volume (eg, sodium supplementation, fludrocortisone) or vasomotor tone (eg, midodrine). The second, however, focuses on mechanical compression of large capacitance venous vessels with a resultant improvement of venous return, cardiac filling, and cardiac output. In practice, the latter strategy is customarily performed through use of lower-extremity compression garments. Although compression of the lower extremities' veins is intuitively reasonable, it turns out that the splanchnic venous vascular bed forms the largest of the body's blood reservoirs (20%–30% total blood volume).⁴ At least in the laboratory setting, its compression with pneumatic antigravity suits appears more effective in lessening OH than similar compression applied to the lower extremities.^{5–7} Although the

efficacy of abdominal compression has been proven under strict experimental conditions, it remains to be clearly established whether more clinically relevant compressions with binders are effective or whether adjustments in binder compression once standing can provide additional benefits.

Given this, we undertook a study to investigate the usefulness of conventional elastic and a more easily adjustable pullstring abdominal binder in increasing standing blood pressure in adults with OH. Our hypothesis was that both binders would be beneficial but that the adjustable binder, given its potential for simpler adjustment, would be more effective.

Methods

This randomized crossover treatment trial was reviewed and approved by our institutional review board and carried out in the Research Autonomic Laboratory at the Mayo Clinic in Rochester, Minnesota, between July 2010 and June 2012. All subjects signed informed consent prior to participation. Trials took place during the morning in a room at ambient temperature of 22°C to 24°C. Subjects were told to maintain good hydration and fast for at least 4 hours before testing. They were also instructed to abstain from medications and substances known to affect the results of autonomic testing for at least 2 half-lives (eg, ethanol for 24h, nicotine for 4h, caffeine for 12h).

Participants

Thirteen adults with neurogenic OH were recruited using the Mayo Clinic Autonomic Laboratory Database (Rochester, MN). Subjects were included if they met clinical and laboratory criteria for moderately severe neurogenic OH as follows: supine to standing blood pressure drops of ≥ 30 mmHg SBP and ≥ 15 mmHg DBP; diagnosis of Parkinson disease, diabetic neuropathy, multiple system atrophy, or another neurologic condition associated with autonomic failure; and laboratory evidence of moderately severe adrenergic failure as measured by the compensatory hemodynamic responses to Valsalva-induced hypotension.⁸ We recruited only those who were ambulatory and able to stand for at least 3 minutes without developing presyncope, discomfort, or cognitive difficulties severe enough to impair ability to follow commands.

Exclusion criteria

Exclusion criteria included the following: positive pregnancy tests in premenopausal women or lactation, motor impairment affecting hand coordination, dementia, severe systemic illness, inability to tolerate withholding of anticholinergic-/alpha- and beta-adrenergic agonist therapies for at least 5 half-lives prior to the study, and inability to withhold midodrine the night before evaluation. Fludrocortisone doses of ≤ 0.2 mg/d were permitted.

Measurements

Hemodynamic variables—Continuous beat-to-beat arterial blood pressure was measured using plethysmography of the left index finger (Finapres model 5,^a 2300e Finapres NIBP monitor^a) held at the heart level in a manner that has been shown to accurately reflect changes in intra-arterial blood pressure both at rest⁹ and during orthostatic stress.¹⁰

Continuous heart rates were measured using a 3-lead electrocardiogram. Data from the Finapres and electrocardiogram devices were recorded and monitored on a computer console that displayed heart rate, SBP, DBP, and mean blood pressure continuously.

Orthostatic intolerance—The Orthostatic Symptom Scale^{6,11} grades the severity of orthostatic symptoms that occur during 5 minutes of standing. The subject reports on a visual analog scale (VAS) the severity of symptoms ranging from 0 (no symptoms) to 10 (syncope or presyncope), with intermediate scores of 2, 4, 6, and 8 defined as tiredness or momentary light-headedness, mental sluggishness or difficulty concentrating, dizziness or unsteadiness, and blurred vision or faintness, respectively. The Symptom Change Scale^{6,12} is a VAS that assesses whether subjects perceive themselves have changes in their ability to stay erect longer or the severity of their symptoms as a result of a study maneuver. Changes are marked on the scale as follows: -3 (much worse), -2 (moderately worse), -1 (mildly worse), 0 (no change), 1 (mildly improved), 2 (moderately improved), and 3 (much improved).

Binder preferences—Two VAS scales were used to assess binder preferences. The first, an ease of use scale, was created to grade the difficulty of adjusting the compression with the abdominal binder while standing and ranged from 0 (impossible to adjust) to 10 (extremely easy to adjust). A future use scale (where 0 is 0% or never, and 10 is 100% or for sure) was also created and used to assess the likelihood that the subject would use this type of binder in the future.

Abdominal compression—Two thigh-size sphygmomanometer cuffs connected with hook and loop fasteners to form a continuous large belt were wrapped around the subjects' lower abdomens between the pubis and umbilicus. The sphygmomanometer bladder used to measure the degree of abdominal compression was centered anteriorly above the pubic bone and was equal distance from the anterior iliac spines. The second sphygmomanometer cuff (which was used to maintain the positioning of the anterior bladder and was not inflated) was positioned between the low back and posterior pelvis. The anterior sphygmomanometer bladder was connected to a pressure transducer and inflated to 5mmHg prior to placement of a binder in a manner that has been used previously.⁷ Sphygmomanometric abdominal compression measurements were done with input to a computer console, which displayed abdominal compression measurement continuously. Similar methods with pressurized garments have yielded comparable standing blood pressure at comparable abdominal compression levels,⁷ suggesting that the validity of this measurement is relatively accurate.

Protocol

Evaluations took place during a single visit and were composed of 4 parts.

Part 1: Familiarization with binder use—Two types of abdominal binders were used (fig 1): a conventional elastic abdominal binder,^b adjustable by releasing and repositioning its anterior hook and loop fastener closure (width, 23 or 30cm; length, 76 or 114cm), and a pullstring binder (QuikDraw^c), adjustable by pulling drawstrings (width, 23 or 30cm; length,

76 or 114cm). Subjects were familiarized with the application, use, and adjustment of the binders prior to the onset of the study.

Part 2: Quantification of self-adjusted compression—The binder being assessed was wrapped around the lower abdomen overlying the previously wrapped sphygmomanometer cuffs (as described above). The tubing of the anterior bladder was connected to a pressure transducer to measure the degree of abdominal compression (mmHg). Subjects were then asked to stand erect and adjust each binder, first to a maximal tolerable level, which was defined as the compression that the subject judged would be acceptable for a short period (eg, 90s), and then to a comfortable level, which was defined as the compression that the subject judged would be comfortable for a more prolonged period (eg, 1h). Because the participants would not be able to self-adjust the binders during study standing maneuvers owing to the use of blood pressure monitoring devices on their hands and arms (Finapres^a and sphygmomanometer, respectively), study personnel used these measurements to recreate similar compressions during standing maneuvers.

Part 3: Standing without an abdominal binder—Subjects rose to a standing position after a 15-minute supine rest period and remained erect for 5 minutes (or less if presyncope developed) before lying back down for recovery. Data recording began 5 minutes prior to rising.

Part 4: Standing with a binder—A block randomization using a random number generator was used to assign the order of binder use. The first was supine abdominal compression. After lying supine for a 20-minute rest period to allow cardiovascular parameters to return to baseline and after 2 minutes of abdominal compression at 10mmHg to mimic the light fit that the subject might find comfortable while sitting or lying at home, the subject stood up and maintained an upright position for 5 minutes (or less if presyncope developed). The second was upright maximal abdominal compression. After completing 5 minutes of standing with the abdominal binder (or a shorter duration if pre-syncope developed), compression was further increased to the subject's maximal tolerable level (as previously described) for 1.5 minutes. If presyncope appeared, continued, or worsened during maximal abdominal compression, the subject resumed a supine position. Finally, there was upright comfortable abdominal decompression. After completing maximal tolerable compression, the binder was adjusted to the subject's previously selected comfortable level for 2 minutes (or less if the subject developed presyncope). The subject was allowed to lie down and reequilibrate for 20 minutes before part 4 was repeated for the second binder.

Outcome measures

The effect of abdominal binding (prior to rising, upright maximal compression, upright comfortable compression) on orthostatic blood pressure was measured as the change in SBP from supine to standing (Δ SBP) calculated as the difference between the pressures: [(SBP supine) – (SBP with standing maneuver)]. The effect of abdominal binding prior to rising on orthostatic symptoms was measured as the difference in Orthostatic Symptom Scale scores within 5 minutes of standing between use and nonuse of an abdominal binder. The effect of

further adjustment of abdominal compression while standing on orthostatic symptoms was measured as the difference in Symptom Change Scale scores of the last minute of adjusted compression and the 1 minute preceding the onset of maximal compression adjustment.

Statistical analysis

Continuous data were expressed as median (interquartile range [IQR]). Categorical data were expressed as n (%). Comparison between maneuvers was made using the Wilcoxon signed-rank test (given the small sample size of the data and the assumption of its non-Gaussian distribution) for matched data with a continuous outcome. The *P* values <.05 were considered statistically significant.

Results

The study group consisted of 13 adults (6 women, 7 men) with neurogenic OH who had been symptomatic for periods ranging from 1 to 18 years (table 1). The effects of the standing maneuvers and binder adjustments are subsequently summarized and presented in figure 2.

Binder characteristics

The degree of abdominal compression achieved by the subjects during upright adjustment was higher with a pullstring binder than an elastic binder both at the subjects' self-selected maximal tolerable level (33mmHg; IQR, 27–42mmHg vs 19mmHg; IQR, 16–22mmHg; *P*<.001) and at a comfortable level (28mmHg; IQR, 23–32mmHg vs 15mmHg; IQR, 14–18mmHg; *P*<.001). There were no differences in preferences between pullstring and elastic binders as measured by ease of adjustment to maximal compression (8 points; IQR, 6–9 points vs 6 points; IQR, 5–8 points, *P*=.12), ease of adjustment to a comfortable level (8 points; IQR, 7–10 points vs 8 points; IQR, 6–9 points; *P*=.24), or future use estimate scores (6 points; IQR, 5–7 points vs 5 points; IQR, 4–7 points; *P*=.45).

Baseline standing maneuvers

Supine SBP prior to standing without abdominal compression (no binder in place) (146mmHg; IQR, 124–164mmHg), with the 10mmHg conventional elastic binder (145mmHg; IQR, 129–167mmHg; *P*=.27), and with the 10mmHg pullstring binder (153mmHg; IQR, 129–160mmHg; *P*=.85) were comparable. Standing without abdominal compression resulted in a large orthostatic fall in blood pressure (SBP, –57mmHg; IQR, –40 to –76mmHg) and severe orthostatic intolerance (Orthostatic Symptom Scale, 5 points; IQR, 4–5 points). Compared with no abdominal binding, 10mmHg of abdominal compression while supine prior to rising was effective in attenuating OH with both the conventional (SBP, –50mmHg; IQR, –33 to –70mmHg; *P*=.03) and pullstring (SBP, –46mmHg; IQR, –34 to –75mmHg; *P*=.01) binders. Orthostatic symptoms were not affected by binder choice: conventional binder (Orthostatic Symptom Scale, –0.5 point; IQR, –1.3 to 0.3 point; *P*=.39) or pullstring binder (Orthostatic Symptom Scale, 0 point; IQR, –1.8 to 1.0 point; *P*=.41).

Adjustment of abdominal compression in the upright position

Once standing with an abdominal binder that had been set at the minimal 10mmHg prior to rising, further compression to the maximal tolerable level did not result in further attenuation of OH (conventional elastic SBP, -53mmHg; IQR, -26 to -71mmHg; $P=.64$; pullstring SBP, -59mmHg; IQR, -49 to -76mmHg; $P=.52$) and did not provide symptom improvement (conventional Symptom Change Scale score, 1 point; IQR, 0 to 1 point; pullstring Symptom Change Scale score, 0.5 point; IQR, 0 to 1 point). Decompression of binder pressure from the maximal tolerable level to the comfortable level tended to worsen OH (elastic SBP, -61mmHg; IQR, -33 to -80mmHg; $P=.64$; pullstring SBP, -67mmHg; IQR, 61 to 84mmHg; $P=.79$).

Discussion

The major findings of this study are that although mild (10mmHg) abdominal compression prior to rising provided modest improvement in standing blood pressure, once standing, further subject-controlled compression adjustments did not produce additional benefit. Contrary to our hypothesis, self-adjustment of abdominal bind pressures once a subject was standing proved to be an ineffective add-on maneuver. Subject assessments were similar in that they reported no preferences between the 2 binders as measured by ease of adjustment, symptomatology, or likelihood of future use.

The degrees of improvement in OH provided by the use of the abdominal binder found in this study were modest and below that that had been expected from previous work. The difference is likely because of the clinical nature of the study (subjects chose their compression forces) and because we used real-life binders rather than the more aggressive shock pant/antigravity implementation, which was featured in much of the earlier works. Another consideration is that the application of abdominal compression in the supine position has been shown to lead to a reduction of cardiac preloading and stroke volume,⁷ leaving the desirability of its application prior to standing questionable. However, the goal of this study was the development of a protocol that mimicked reality in that patients with OH are told to, and tend to, apply their compression garments prior to arising from bed. In any event, our findings are far more likely to reflect (as has been the experience of J.R.B.) the clinical situations where benefits from the use of binders and for that matter lower-extremity compression garments are far less than might be expected from the research literature.

The contrast between our findings and those of other studies that show that even small blood pressure increases may improve orthostatic tolerance considerably^{13,14} may be more apparent than real. More specifically, as previously noted, the studies assessing maneuvers to lessen orthostatic blood pressure drops differ significantly from ours not only in the nature of compressive garments (eg, shock pants) but also in the extent of compression (eg buttocks, lower extremities) and involvement of the musculoskeletal system (ie, leg crossing plus muscle tensing).¹⁵

It was surprising that the application of the subjects' self-determined maximal compression values during upright adjustment of their abdominal compressions did not improve their

standing SBP. This finding is in contrast with prior studies in which abdominal compression applied to hypotensive subjects after they had been tilted or had been standing briefly was associated with a significant pressor response.⁶ There are a number of possibilities for this discrepancy. First, these studies, Denq et al⁶ for example, tended to use specialized shock garments, which are likely to apply elevated pressures over larger areas than the clinically appropriate binders of this study. Second, our subjects selected their maximal pressures on the basis of what they felt was tolerable; other investigators may have been more forceful in their encouragement. Third, the subjects in this study had been standing for periods of up to several minutes prior to their maximal compression assessments, compared with the very brief periods of prior studies.^{16,17} This discrepancy, although it mimics 2 discrete but each clinically significant situations, may have permitted more time in our subjects for venous pooling, transcapillary filtration, and the transfer of blood from the thorax to lower regions.^{16–19}

The conventional elastic and pullstring binders did not differ in their blood pressure responses, ease of use, and preference scores. Different compression garments have similar pressor responses at comparable compressions.⁶ Therefore, it is reasonable to consider that abdominal binding at similar compressive levels will be comparably effective regardless of binder type and that subject garment preferences should not affect treatment response.

Study limitations

This study has its strengths and weaknesses. One of its strengths was the severity (>50mmHg) of the subjects' orthostatic drops with standing. Furthermore, although one might argue that the range of conditions assessed (eg, autonomic failure, multiple system atrophy, Parkinson disease) is heterogeneous, we would argue that they all have a common endpoint of impaired innervation and an inability to prevent pooling of blood in the lower portions of the body. The sample size is always an issue, but ours was relatively large with respect to similar studies of this nature. It seems large enough to suggest whether clinically meaningful results might be obtained in larger efforts. In addition, although the study size is an issue, the study was designed to minimize the influence of confounding variables and carryover effects by using a randomized crossover control design, careful physiological monitoring, prolonged recovery periods between maneuvers that were long enough to allow the subjects' physiological variables to return to baseline, and randomization of the order in which the binders were assessed.

It would be interesting to know more about the effects of abdominal compression over longer periods of time. To our knowledge, little formal testing has been done in this area. Our speculation is that effects would depend on the status of an individual's baroreflexes (especially their adrenergic component), plasma volume, and venomotor tone. People wearing binders with significant residual compensatory mechanisms might do well, whereas those with such mechanisms in abeyance would have progressive worsening of their blood pressure while standing.

Conclusions

The donning of an abdominal binder prior to rising is effective in ameliorating OH, but further upright adjustment of compression, at least under the conditions of this study, did not provide additional subjective or objective benefit. Subjects using abdominal binders should consider physical countermaneuvers¹⁵ or prompt postural changes (sitting or lying down) at the first warning of orthostatic deterioration rather than attempting to adjust their binders.

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List of abbreviations

DBP	diastolic blood pressure
IQR	interquartile range
OH	orthostatic hypotension
SBP	systolic blood pressure
VAS	visual analog scale

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Fig 1.
Elastic abdominal binder (A) and pullstring abdominal binder (B).

Fig 2.
Effect of adjusting abdominal compression on standing BP (vertical lines within bars represent IQRs). Abbreviation: BP, blood pressure.

Table 1

Subject characteristics

Characteristic	Value
Total	13
Female:male	6:7
Diagnosis	
Pure autonomic failure	5
Multiple system atrophy	4
Parkinson disease	2
Postradiation baroreflex failure	1
Autoimmune autonomic neuropathy	1
Age (y)	72 (62–79)
Duration of symptoms (y)	4 (1–18)
Orthostatic Symptom Scale score (range, 1–10)	5 (0*–9)

NOTE. Values are number of subjects or median (range).

* All subjects were symptomatic by history. The single 0 score was recorded by a subject who was asymptomatic on a single sit to stand maneuver but was symptomatic on all others.