Application of the Sit-Up Test for orthostatic hypotension in individuals with stroke

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Abstract

Orthostatic hypotension (OH) is an important consideration for individuals with stroke, given the shared occurrence of mobility limitations, fall risk and association with adverse cardiovascular outcomes. This study aimed to 1) establish the application of a simple bedside test of orthostatic challenge to identify OH after stroke, 2) examine differences in characteristics between those with and without OH and 3) determine cardiovascular correlates with hemodynamic responses. Forty-nine participants (n=29 men, mean±SD age 66±7 years, time post-stroke 4.5±3.1 years) performed an orthostatic challenge (Sit-Up Test). Eleven (22%) of the 49 participants presented with OH (n=7, of which 5 were asymptomatic) or symptoms of cerebral hypoperfusion with position change (n=4). Compared to participants without OH, those with OH had higher total:high-density lipoprotein cholesterol ratios (4.2 ± 0.9 vs. 3.3 ± 0.8 , $P=0.009$) and triglyceride levels (2.2 ± 0.8 vs. 1.4 ± 0.5 mmol/L, $P=0.001$). Multivariate linear regression revealed that high-density lipoprotein cholesterol and triglyceride levels explained 20% of the variance of the change in systolic blood pressure from the Sit-Up Test ($F(2,45)=5.68$, $P=0.006$). In conclusion, we used a simple bedside test of orthostatic tolerance to identify that over 20% of individuals with stroke presented with OH or symptoms of hypoperfusion. They also had more impaired cardiovascular risk profiles relative to those without OH. These individuals may be at even higher risk for mobility limitations and falls beyond that associated with stroke-related deficits alone.

Keywords

Stroke; orthostatic hypotension; risk factors

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Conflict of interest

None declared

Introduction

Orthostatic hypotension (OH) is defined by the American Autonomic Society and American Association of Neurology as a decrease in systolic or diastolic blood pressure (BP) of 20 or 10 mmHg within 3 minutes of standing (Freeman et al., 2011). OH is reported to be present in 6% (Freeman et al., 2011) to 30% (Luukinen et al., 1999) of community-dwelling older adults. It may be accompanied by symptoms of cerebral hypoperfusion (such as dizziness and lightheadedness) but may also be completely asymptomatic. Even among those who do not report hypotensive symptoms, there remains an elevated risk of syncope, falls, and mobility restrictions (Gupta and Lipsitz, 2007).

OH is also associated with adverse cardiovascular health outcomes in middle-age and older adults, including elevated risk for all-cause and cardiovascular mortality and cardiovascular disease (Rose et al., 2006, Verwoert et al., 2008, Masaki et al., 1998), myocardial infarction, transient ischemic attack (Fedorowski et al., 2010) and arterial stiffness (Mattace-Raso et al., 2006). It has been identified as a risk factor for ischemic stroke (Eigenbrodt et al., 2000). As a potential indicator of underlying autonomic dysfunction that occurs with diabetes, Alzheimer's disease and dementia (Novak and Hajjar, 2010), OH may be associated with impaired cognitive performance, but these findings are inconsistent (Viramo et al., 1999, Rose et al., 2010, Allcock et al., 2006) and the link remains unclear (Novak and Hajjar, 2010).

For people with stroke, OH has important clinical implications. The additional presence of OH after stroke may further compound the typical restrictions observed in mobility, exacerbate fall risk, and limit the ability to engage in daily activities. Further, among potential neurogenic or pharmacologic causes of OH, several are common among those with stroke, such as the presence of diabetes and peripheral neuropathy, or use of pharmacological agents such as anti-hypertensive medications (Maule et al., 2007, Mathias and Kimber, 1999). Autonomic function is also altered post-stroke, where impairments in baroreflex function and BP control may result in inadequate cerebral perfusion (Sykora et al., 2009, Kong and Chuo, 2003, Novak et al., 2010). Given these issues, as well as the prevalence of cardiovascular co-morbidities among individuals with stroke (Kopunek et al., 2007, Roth, 1993) and elevated risk for recurrent events (Mohan et al., 2011), it is important to identify people with stroke who also present with OH.

There have been several studies examining orthostatic responses early after stroke. Acutely after stroke, positional BP responses were comparable between individuals with ischemic stroke and non-stroke controls (Korpelainen et al., 1994, Panayiotou et al., 1999, Panayiotou et al., 2002), possibly attributed to sympathetic hyperactivity that occurs at this early stage (Panayiotou et al., 2002). Among 71 patients participating in inpatient stroke rehabilitation, over 50% of cases demonstrated OH on a tilt table test, and two-thirds of these were asymptomatic (Kong and Chuo, 2003). Despite the high rate of OH observed in this sub-acute phase of stroke, there were no negative effects on functional outcomes after rehabilitation intervention was completed, nor in length of rehabilitation stay (Kong and Chuo, 2003). Only one study focused on individuals in the later stages of stroke (> 1 year post-stroke), where OH was present with head-up tilt in 23% of the 43 participants with

middle cerebral artery territory infarct, and was associated with lower cerebral blood flow velocities in the stroke-affected hemisphere (Novak et al., 2010).

The presence of OH among individuals in the chronic stroke stage is an important clinical consideration. While this cohort has typically completed active rehabilitation interventions, they continue to be at risk for functional decline (Paolucci et al., 2001, van de Port et al., 2006), cardiovascular risk factors remain poorly managed (Kopunek et al., 2007) and falls that occur with OH-related syncope may increase the risk of injuries, such as hip fractures (Gill et al., 2009). Thus, it is important to routinely screen for the presence of autonomic dysfunction in this vulnerable population and prevent potential complications. Identifying individuals with orthostatic intolerance post-stroke may be challenging, due to limited access to specialized equipment, such as tilt testing facilities (Claydon and Krassioukov, 2006), and the limited ability for some individuals to attain and maintain a standing position. As such, we sought to establish the application of a simple bedside test of orthostatic tolerance to identify the presence of OH among individuals living in the community with stroke. We also aimed to compare the cardiovascular risk factor profiles between individuals with and without OH, and to identify correlates with the hemodynamic responses from the orthostatic challenge.

Materials and Methods

This study was part of a larger trial examining the effects of aerobic exercise on cardiovascular function among individuals with stroke. Study procedures were approved by local university and hospital research ethics boards. Informed written consent was obtained from all participants. While the main trial used a randomized controlled design, in this cross-sectional study, both study groups were collapsed into a single group for analysis of baseline (i.e. pre-training) data.

Participants

Participants were eligible for the study if they were at least 1 year post-stroke, living in the community and able to walk 5 meters independently. Individuals were excluded if they sustained stroke of non-cardiogenic origin (e.g. aneurysm, tumor, infection), were actively engaged in stroke rehabilitation services, had uncontrolled arrhythmias or a pacemaker, or presented with serious musculoskeletal (e.g. rheumatoid arthritis) or other neurological conditions (e.g. Parkinson's).

Assessments

Participant demographics were recorded, including age, sex, details of stroke (time post-stroke, type, location) and relevant medical history. To characterize the stroke severity and motor impairment of our sample, the National Institutes of Health Stroke Scale (Brott et al., 1989) was used where higher scores indicate greater severity (maximum score 42), along with the Chedoke-McMaster Stroke Assessment (Gowland et al., 1993) where higher scores indicate less motor impairment (maximum score 7). Functional mobility was quantified with self- and fast-paced gait speed measured over a 5-meter walkway, the 6-Minute Walk Test

(American Thoracic Society, 2002) for ambulatory capacity, and Berg Balance Scale (Berg et al., 1992) for functional balance. The use of gait aids for walking was also noted.

Orthostatic hypotension—An orthostatic challenge was performed using the Sit-Up Test (Claydon and Krassioukov, 2006). This simple, bedside test was chosen over the traditional tilt-table test as it is more feasible in the clinical setting and does not require extensive strapping, yet has been demonstrated to be effective in eliciting cardiovascular responses to evaluate orthostatic control in individuals with spinal cord injury (Claydon and Krassioukov, 2006). Further, while participants were all ambulatory to varying extents, many had impaired balance and poor activity tolerance, and some may not have tolerated 10 minutes of active standing. Thus, the Sit-Up Test was applicable to individuals with a broader range of functional abilities than one that required active standing.

In the Sit-Up Test, participants were requested to abstain from caffeine and alcohol 12 hours prior to the test and to eat a light meal no later than 2 hours prior. The test was performed with 2 trained assessors in a temperature-controlled laboratory. After 10 minutes of quiet, supine rest, BP (Dinamap, GE Healthcare, Buckinghamshire UK) was measured in the non-paretic arm at 1-minute intervals for 10 minutes. The participant was then moved passively from a supine to a sitting position, and instructed not to assist with the maneuver. BP was then measured every minute for an additional 10 minutes in the sitting position. The test was terminated early if severe symptoms of pre-syncope were demonstrated, and the participant returned to supine position (Claydon and Krassioukov, 2006). The maximum drop in systolic and diastolic BP within the first 3 minutes of changing to the upright position was the primary outcome of this test. Oxygen saturation and heart rate were monitored. Symptoms of cerebral hypoperfusion (dizziness, lightheadedness, shortness of breath or changes in vision) were noted.

Cardiovascular risk factor profile—The presence of cardiovascular risk factors was examined, including cardiovascular co-morbidities, measures of body composition, lipid panel, glucose control and aerobic capacity. Smoking status and presence of diabetes were noted and participants' body weight was measured (Mettler-Toledo, Columbus OH). Due to the known pharmacological effects of anti-hypertensive (beta-blockers, angiotensin-converting enzyme inhibitors, calcium-channel blockers) and diuretic (hydrochlorothiazide, furosemide) medications on postural hypotension, the use of these was also noted. Plasma levels of total, high- and low-density lipoprotein cholesterol, triglycerides, glucose and glycated hemoglobin were measured after 12-hour fast. Aerobic capacity was measured using a graded maximal leg exercise test with a ramp protocol (Pang et al., 2005) on a cycle ergometer (Excalibur, Lode Medical Technology, Groningen NL). Breath-by-breath gas exchange was continuously measured (ParvoMedics, Sandy UT). The American College of Sports Medicine guidelines for test termination were followed (American College of Sports Medicine, 2010). The protocol was adjusted to use 10- or 15-watt increments to maintain a test time between 8–10 minutes. VO_2peak was determined as the highest value achieved during the aerobic capacity test.

Analysis

Descriptive statistics were performed for participant characteristics and for variables in the cardiovascular risk factor profile. The proportion of participants who met the criteria for OH (Freeman et al., 2011) was determined, as well as those who demonstrated symptoms of cerebral hypoperfusion. Independent *t*-tests were performed to examine differences in participant characteristics and risk factor profiles between those who did and did not demonstrate OH. Correlation analyses were performed between variables in the cardiovascular risk factor profile and change in BP from the Sit-Up Test. Variables that were significantly associated were entered into a multivariate linear regression model. To ensure assumptions of the multivariate regression were met, scatterplots were visually inspected for outlier data and to confirm linearity of associations, and the correlation matrix, tolerance values and variance inflation factors were examined for multi-collinearity. Statistical Package for the Social Sciences (Version 17.0, Chicago IL) was used with a significance level of $P<0.05$.

Results

Characteristics for the 49 participants included in this study are presented in Table 1.

During the Sit-Up Test, systolic and diastolic BP dropped 5.8 ± 10.5 and 1.4 ± 5.9 mmHg, respectively. Seven of the 49 (14.2%) participants met the criteria for OH, where maximum drop in BP occurred within 3 minutes of attaining upright sitting position. There were no cases of initial or delayed orthostatic hypotension. Of these 7 participants with OH, 2 (29%) participants demonstrated symptoms of cerebral hypoperfusion, including 1 person who experienced near syncope. In all cases, symptoms resolved within 1 minute and the tests were completed. Five of these 7 (75%) participants did not demonstrate any hypotensive symptoms.

Differences between participants with and without OH are presented in Table 2. Relative to participants who did not demonstrate OH, those with OH had higher triglyceride levels and higher total:high-density lipoprotein cholesterol ratio. They also tended to have lower high-density lipoprotein levels and higher body weight. There were no differences in resting supine blood pressure between those with and without OH. Similarly, the proportions of individuals who currently smoke, had diabetes, or were taking anti-hypertensive (beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin-II receptor antagonists or calcium-channel blockers) or diuretic medications were similar between groups.

Of the 42 participants who did not meet the criteria for OH, there were 4 (10%) individuals who demonstrated symptoms of hypoperfusion with position change. In all 4 cases, symptoms resolved within 1 minute and the tests were completed.

Secondary analysis was performed that combined these 4 participants without OH but with hypotensive symptoms with the 7 who did meet the criteria for OH (OH/symptomatic group). Participants in the OH/symptomatic group ($n=11$) demonstrated a drop in BP of $15.9 \pm 12.5/5.1 \pm 7.9$ mmHg after attaining sitting position, whereas those in the rest of the sample ($n=38$) demonstrated drop in BP of $2.7 \pm 7.7/0.1 \pm 4.6$ mmHg ($P<0.0001$ and $P=0.01$).

for group differences in systolic and diastolic BP change, respectively). Consistent with the original comparison analysis, significant differences in triglyceride levels and total:high-density lipoprotein cholesterol ratio were maintained, where the OH/symptomatic group demonstrated poorer lipid profiles compared to the rest of the sample (1.4 ± 0.5 vs. 1.9 ± 0.8 mmol/L, $P=0.01$ and 3.3 ± 0.8 vs. 4.0 ± 0.9 mmol/L, $P=0.03$, respectively). Further, the initial trends towards greater body weight and lower high-density lipoprotein levels were significant in the secondary analysis (77.9 ± 16.3 mmol/L vs. 89.3 ± 15.8 , $P=0.04$ and 1.4 ± 0.4 vs. 1.2 ± 0.2 mmol/L, $P=0.04$, respectively). No other group differences were observed.

One case was identified as an outlier (decrease in BP greater than 2 SD from mean) and was thus removed from analysis to reduce leveraging effects on the correlation and regression models. The correlation matrix revealed that triglycerides ($R=0.33$, $P=0.02$), total:high-density lipoprotein cholesterol ratio ($R=0.45$, $P=0.001$) and high-density lipoprotein cholesterol ($R=-0.41$, $P=0.004$) were associated with change in systolic BP. To determine cardiovascular risk factor correlates with hemodynamic responses to the Sit-Up Test, multivariate linear regression was performed. Total:high-density lipoprotein cholesterol ratio was not entered into the regression model, as it was highly correlated with triglycerides ($R=0.65$, $P<0.001$) and high-density lipoprotein cholesterol ($R=-0.67$, $P<0.001$). The remaining two variables (triglycerides and high-density lipoprotein cholesterol) explained 20.2% of the variance of change in systolic BP from the Sit-Up Test ($F(2,45)=5.68$, $P=0.006$), and high-density lipoprotein cholesterol was a significant independent predictor (Table 3).

No variables were correlated with change in diastolic BP from the Sit-Up Test, thus multivariate regression was not performed.

Discussion

Using a simple bedside test of orthostatic tolerance, we demonstrated that OH affected approximately 15% of a cohort of community-dwelling people with stroke, and this proportion increased to 20% when individuals with symptomatic hypoperfusion were included. Participants with OH demonstrated greater dyslipidemia and tended to be heavier in body weight. Given that individuals with stroke present with compromised mobility and elevated recurrent stroke risk (Mohan et al., 2011), and that OH is associated with cardiovascular events, it is important to accurately identify those with OH after stroke and provide the appropriate interventions to minimize its occurrence.

Our cohort comprised of a representative sample of people living in the community with stroke. Study participants were ambulatory, and presented with mild impairment to upper and lower limb function, and moderate balance impairment. We were able to safely perform an orthostatic challenge without the use of specialized tilt table equipment to identify participants with OH. The Sit-Up Test is easy to administer in the clinical setting, provided that staff are appropriately trained to recognize and respond to symptoms. Guidelines recommend that resuscitation and cardiac life support procedures are in place prior to an orthostatic challenge test (Lahrmann et al., 2006). For safety, we performed this test with

two examiners, although it is possible for it to be done by one trained assessor, providing they are able to safely complete the passive maneuver from supine to sitting, while monitoring for hypotensive symptoms. Exercising clinical judgment is always paramount.

We observed a lower occurrence of OH compared to the 52% reported for individuals participating in inpatient stroke rehabilitation (Kong and Chuo, 2003), but is aligned with previously reported values of community-dwelling samples of individuals with middle-cerebral artery infarct (Novak et al., 2010) and older adults (Luukinen et al., 1999, Rutan et al., 1992). The disparity in occurrence rates may be attributed to differences inherent in rehabilitation versus community settings. Participants in the sub-acute stroke phase, particularly those in institutionalized environments, are less mobile and thus at greater risk for OH. Indeed, the occurrence of OH among institutionalized older adults is much higher relative to those living in the community (Freeman et al., 2011). It is not known whether the same cutpoints for blood pressure reduction that are recommended for standard standing tests of orthostatic intolerance (Freeman et al., 2011) may be applied to a sitting test, or whether lower values may be sufficient since full upright standing position is not attained. Nonetheless, the Sit-Up Test would thus generate a more conservative estimate of the occurrence of OH. That we were able to demonstrate that approximately 15% of our sample presented with OH suggests that this bedside test can be an important initial screen for individuals at risk for OH and is feasible for individuals who present with balance impairment or difficulty transferring to a standing position.

It has been suggested that clinically important OH is not common after stroke (Korpelainen et al., 1999) as it is with other populations, such as spinal cord injury (Claydon et al., 2006) or Parkinson's (Velseboer et al., 2011) where the clinical and health implications of OH is well established. However, with the combined occurrence of over 20% of participants demonstrating OH or hypotensive symptoms, we believe that orthostatic intolerance is prevalent and clinically important after stroke. It has the potential to compound stroke-related mobility limitations, balance impairment and contribute to fall risk in this already compromised group. Furthermore, given the association of OH with negative cardiovascular (Rose et al., 2006, Verwoert et al., 2008, Masaki et al., 1998) and cerebrovascular (Fedorowski et al., 2010, Eigenbrodt et al., 2000) outcomes, its occurrence after stroke may further elevate recurrent event risk in this at-risk population (Mohan et al., 2011). In the current study, participants with orthostatic intolerance demonstrated a more impaired cardiovascular risk profile relative to those who did not. Specifically, these individuals presented with greater dyslipidemia, and although OH is typically associated with lower body weight in older adults (Rutan et al., 1992), subjects in the current study were heavier, which is aligned with our findings of increased cardiovascular risk. Cardiovascular interventions, such as those aimed at managing dyslipidemia, are not only important as secondary preventative strategies, but have also been shown to slow the progression of autonomic neuropathy (Gaede et al., 2003).

That the majority of participants with OH did not demonstrate hypotensive signs or symptoms is noteworthy. Being asymptomatic makes this particular subgroup particularly vulnerable to negative health effects related to OH, as they are not able to perceive postural changes in BP. The mechanisms by which these individuals are able to tolerate transient

hypotension with positional changes are not known, given that cerebral autoregulation appears to be impaired after stroke (Novak et al., 2010). Nonetheless, this underscores the importance of objectively measuring BP during position changes to identify individuals with OH, rather than relying solely on subjective reports of hypotensive symptoms.

The cause of OH after stroke is likely due to a complex combination of factors (Kong and Chuo, 2003). Baroreceptor reflex dysfunction and altered BP regulation are associated with older age and with various cardiovascular conditions commonly observed in individuals with stroke, such as hypertension, coronary artery disease or carotid atherosclerosis (Sykora et al., 2009). Other potential contributors to postural hypotension are also common in individuals with stroke, including diabetes mellitus and peripheral vascular disease, and certain medications, such as BP and diuretic medications. In the current study, we did not observe differences in participants with and without OH and use of anti-hypertensive or diuretic medications, or presence of diabetes. Panayiotou and colleagues (Panayiotou et al., 2002) also did not find an association between OH and anti-hypertensive medication use in the acute phase of stroke, but this is likely due to sympathetic hyperactivity in the acute phase of stroke.

There are several limitations to this study. Some medications, in particular anti-hypertensive medications, may influence the results of the Sit-Up Test. In order to evaluate orthostatic tolerance within normal daily function, we instructed participants to continue taking all medications as prescribed. We did not, however, standardize the time between drug administration (for pharmacological half-life) and performance of the test. The site of neurological lesion (e.g. subcortical and brainstem) may be implicated in neurogenic OH (Gupta and Nair, 2008), but was not examined in the current study; however, Kong and Chuo (Kong and Chuo, 2003) did not observe any associations between type and location of stroke and OH in individuals with sub-acute stroke. Other potential contributors to OH, such as hypovolemia and hyponatremia, were also not examined in this study. Additionally, although participants were informed that the orthostatic tolerance test was to be performed completely passively and they were all instructed to refrain from assisting with the 'sit up' portion of the test, it is possible that some participants could have been provided some active assistance. Using a tilt table would have minimized this possibility, but would have detracted from the clinical feasibility of performing the orthostatic challenge. Future research is warranted to further validate the Sit-Up Test by comparing results to standard tilt table or standing tests, and to establish reproducibility of this measure. Lastly, the sample size was relatively small which reduces the power to detect small, but potentially important contributions from variables in the regression model. However, the assumptions for the linear regression model were satisfied.

Conclusions

We demonstrated that a simple bedside test of orthostatic tolerance may be used to identify individuals with stroke with OH. Over 20% of a sample of people living with stroke in the community were affected by OH or symptoms of hypoperfusion with positional change. Notably, the majority of participants with OH did not demonstrate hypotensive signs or symptoms. Since OH is associated with limited mobility, fall risk and adverse cardiovascular

outcomes, and individuals with stroke already present with mobility limitations and elevated risk for recurrent events, it is an important consideration for health professionals working with the stroke population. The Sit-Up Test is a simple bedside orthostatic challenge that may be conducted to identify those with OH.

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Table 1

Participant demographics

	n (%) or Mean $\pm$ SD (min-max)
Age, y	66.1 $\pm$ 7.0 (51–80)
Sex, men/women	29 (59)/20 (41)
Stroke type	
Lacunar / Ischemic / Hemorrhagic / Unknown	7 (14) / 19 (39) / 16 (33) / 7 (14)
Stroke location	
Cortical / Subcortical / Brainstem / Unknown	11 (22) / 22 (45) / 6 (12) / 10 (20)
Hemisphere affected	
Right / Left / Bilateral / Unknown	23 (47) / 22 (45) / 2 (4) / 2 (4)
Timepost-stroke, y	4.5 $\pm$ 3.1 (1.1–12)
Number of chronic conditions	4 $\pm$ 2.4 (1–14)
National Institutes of Health Stroke Scale	1.6 $\pm$ 2.2 (0–10)
Resting blood pressure, mmHg	122.3 $\pm$ 12.1 (90–144)/67.6 $\pm$ 7.2 (45–90)
Chedoke-McMaster Stroke Assessment	
Arm / Hand scores	5.8 $\pm$ 1.9 (1–7) / 5.6 $\pm$ 2.0 (1–7)
Leg / Foot scores	6.1 $\pm$ 1.0 (2–7) / 5.7 $\pm$ 1.8 (1–7)
Berg Balance Scale	49 $\pm$ 7.1 (20–56)
5-meter Walk speed	
Self-paced, m/s	0.92 $\pm$ 0.37 (0.10–1.69)
Fast-paced, m/s	1.28 $\pm$ 0.56 (0.11–2.65)
6-Minute Walk Test distance, m	310.1 $\pm$ 137.4 (27–600)
Gait aids	
None / Cane / Walker / Rollator	30 (61) / 15 (31) / 3 (6) / 1 (2)

Table 2

Comparison of participants with and without orthostatic hypotension

	Without orthostatic hypotension* n=42	With orthostatic hypotension n=7	P value
Age, years	65.9 ± 6.9 (51–80)	67.6 ± 8.0 (58–79)	0.56
Weight, kg	78.7 ± 16.3 (41–109)	91.1 ± 16.9 (73–113)	0.07
Lipid panel			
Total cholesterol, mmol/L	4.4 ± 0.9 (2.5–7.1)	4.6 ± 0.9 (3.7–6.4)	0.57
HDL cholesterol, mmol/L	1.4 ± 0.4 (0.8–2.4)	1.1 ± 0.2 (0.9–1.5)	0.054
LDL cholesterol, mmol/L	2.4 ± 0.7 (0.9–4.7)	2.5 ± 0.9 (1.1–4)	0.68
Total-HDL cholesterol ratio	3.3 ± 0.8 (1.8–5.5)	4.2 ± 0.9 (2.4–5.2)	0.009**
Triglycerides, mmol/L	1.4 ± 0.5 (0.4–3)	2.2 ± 0.8 (1.5–3.7)	0.001**
Glucose control			
Glucose, mmol/L	5.4 ± 1.4 (3.4–11.1)	5.1 ± 0.6 (4.4–6.3)	0.60
HbA1c, %	5.9 ± 1 (4.7–8.9)	5.7 ± 0.6 (5.2–6.8)	0.60
Aerobic capacity			
VO ₂ peak, ml/kg/min	16.7 ± 6.2 (6–35)	18.1 ± 8.3 (9–29)	0.60

Values are n (%) or Mean ± SD (min-max). Abbreviations: BP – blood pressure.

* OH defined as a decrease in systolic or diastolic blood pressure (BP) of 20 or 10 mmHg within 3 minutes of standing (Freeman et al., 2011)

**
P<0.05

Multivariate linear regression model of change in systolic blood pressure from the Sit-Up Test using cardiovascular risk factors

Table 3

Correlates	Unstandardized B (SE)	Standardized B	95% CI for B	R ²	P value
Model: R ² =0.20, F(2,45)=5.68, P=0.0006					
HDL	-8.6 (3.7)	-0.3	-16.1, -1.1	0.10	0.03*
Triglycerides	3.0 (2.2)	0.2	-1.4, 7.3	0.03	0.18

Abbreviation: HDL – high-density lipoprotein cholesterol

* P<0.05