Impaired Systolic Blood Pressure Recovery Directly After Standing Predicts Mortality in Older Falls Clinic Patients

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Background. Normally, standing up causes a blood pressure (BP) drop within 15 seconds, followed by recovery to baseline driven by BP control mechanisms. The prognostic value of this initial BP drop, but also of the recovery hereafter, is unknown. The aim of this study was to examine the prognostic value of these BP characteristics in response to standing.

Methods. In a retrospective cohort study of 238 consecutive patients visiting our falls outpatient clinic, we examined the relation between all-cause mortality and BP decline and recovery directly after active standing up with Cox proportional hazards analyses.

Results. Of 238 patients (mean age 78.4 ± 7.8 years), during a median follow-up of 21.0 months, 36 (15%) patients died. Neither absolute nor relative (%) initial BP drop after standing predicted mortality. In contrast, the magnitude of BP recovery 40–60 seconds after standing was associated with mortality, even after adjustment for age, comorbidity, and other baseline characteristics. When systolic BP had recovered to less than 80% of prestanding baseline after 60 seconds of standing, this was a powerful independent predictor of mortality (hazard ratio: 3.00; 95% confidence interval 1.17–7.68).

Conclusions. Failure to recover from BP decline in the first minute after active standing up is associated with excess mortality in falls clinic patients. A recovery of systolic BP to less than 80% of baseline after 60 seconds may be used as an easily available cardiovascular marker for increased mortality risk in older falls clinic patients.

Key Words: Cardiovascular—Multimorbidities—Physiology—Resilience—Outcomes.

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The blood pressure (BP) response after standing up has two main components. The first component is a BP decline (often preceded by a small transient initial increase in BP, possibly due to leg and abdominal muscle tensing) within the first 15 seconds due to a mismatch in blood volume entering and leaving the arterial vasculature. Upon standing, cardiac output increases due to the shift of blood to the thorax as a consequence of compression of leg and splanchnic venous vessels (1–3). However, despite this increase in cardiac output, BP decreases—after the transient small rise—because total peripheral resistance is reduced to a further degree than cardiac output is increased, due to instantaneous vasodilatation in the active leg muscles (1,4–8). After about 15 seconds, the second component follows, and this is the recovery of BP by counteracting regulatory mechanisms of the arterial baroreflex (3).

Regarding the first component, when the initial drop in BP is severe, this may pose a risk of serious complications, such as falls, fall-related injury, syncope, direct or indirect myocardial infarction, stroke, and even mortality (9–11). Pathophysiologically, these negative outcomes can be related to insufficient perfusion of the brain and the myocardium.

For the second component, an impaired recovery of BP after standing prolongs this state of insufficient perfusion of vital organs. In addition, impaired recovery of BP after standing may reflect compromised cardiovascular control and may be an indication of underlying disease. The prognostic meaning of these two components of the BP response after standing (ie, drop and recovery) are unknown. Recovery patterns of systolic BP (SBP) after standing have been studied but did not show an association with falls and frailty (12); however, the relation of these patterns with mortality has not been studied. We hypothesized that both the severity of BP decline after standing and the level of impairment of the BP recovery hereafter are associated with all-cause mortality in a geriatric population.
Methods

Study Population

This retrospective study included 313 consecutive patients who were referred to the geriatric outpatient falls clinic of the Radboud University Nijmegen Medical Centre, Nijmegen, The Netherlands, from May 2005 to June 2010. Patients were referred because of a fall, dizziness, or syncope. All patients were tested with an active standing test. This was part of a standardized diagnostic test protocol, which has been described in detail elsewhere (13). According to the Declaration of Helsinki, informed consent was asked verbally prior to the tests.

Baseline Assessment

Patients underwent a complete medical history and physical examination before testing. Baseline characteristics (age, gender, body mass index [BMI], SBP, diastolic BP [DBP], heart rate, medical history, and medication use) were documented. The presence and severity of comorbidity were recorded using the Cumulative Illness Rating Scale for Geriatrics (CIRS-G, ranging 0–56) (14).

We categorized medical history in the following disease groups: (a) dementia, (b) depression or anxiety disorder, (c) chronic obstructive pulmonary disease, (d) diabetes mellitus, (e) Parkinson’s disease or disorders with parkinsonism, (f) the cardiovascular disease group, including myocardial infarction, angina pectoris, heart failure, peripheral vascular disease, aortic aneurysm, stroke, and transient ischemic attack, (g) hypertension, and (h) malignancy.

For medication use, the following medication groups were used: (a) beta-blockers, (b) calcium channel blockers, (c) diuretics, (d) angiotensin-converting enzyme inhibitors or angiotensin II receptor antagonists, (e) nitrates, (f) platelet aggregation inhibitors, (g) cholesterol-lowering drugs, that is, 3-hydroxy-3-methylglutaryl coenzyme-A-reductase inhibitors (statins) or fibrates, (h) psychiatric medication, that is, antidepressants, antipsychotics, and anxiolytics, (i) medication for Parkinson’s disease or parkinsonism, and (j) alpha-blockers.

Testing and Data Acquisition

The patients fasted overnight, and medication was withheld from midnight the night before. During testing, BP and heart rate were constantly measured using a Finometer (Finapres) (15) and a three-lead electrocardiogram. BP was measured at the nondominant arm, which was held at heart level with a sling. The hydrostatic height correction system was used throughout the study to compensate for changing of the hand positions relative to heart level. The BP measurements were calibrated against brachial artery BP at baseline using the return to flow calibration system (16,17). The raw data of BP and heart rate were stored in a data file. Unfortunately from 71 patients, these raw data files could not be retrieved. This was a random selection, due to theft of the test laptop before backup. Four patients were excluded because the BP measurements were not adequately determined, leaving 238 patients for analysis in the final sample. Data were exported using Beatscope 1.1a software and analyzed with custom-written software in Matlab (version R2010b, The MathWorks Inc., Natick, MA). After a 10-minute resting period in the supine position, the patients were instructed to stand up as quickly as possible, sometimes with a helping hand, and remain standing for 10 minutes. Baseline values of BP and heart rate were defined as the average during the 20 seconds before standing up.

Hemodynamic Response After Active Standing

For the first component of the hemodynamic response after standing, we selected the decline of BP within the first 15 seconds. We determined the absolute maximum drop of SBP (∆SBP) and the lowest SBP value that was reached in these first 15 seconds. The lowest SBP value (SBPmin) was expressed as a percentage of baseline (prestanding) SBP: %SBPmin = (baseline SBP – ∆SBP/baseline SBP) × 100%

For example, if after standing up the SBP decreased from 120 to 90 mmHg this corresponds to a ∆SBP of 30 mmHg and a %SBPmin of 100 × (120 – 30)/120 = 75%. We did the same for DBP (∆DBP and %DBPmin).

The second component of the hemodynamic response after active standing is the recovery of BP after the first 15 seconds. Therefore, the time between 15 and 60 seconds after standing was divided in nine time intervals of 5 seconds. For each time interval, the average SBP and DBP was calculated as percentage of the baseline BP (%SBP15–20, %SBP20–25, … %SBP55–60 and %DBP15–20, %DBP20–25, … %DBP55–60). On the basis of the percentage SBP recovery at 55–60 seconds (%SBP55–60), we classified the patients into three groups with different recovery patterns in analogy to the earlier mentioned study of the association of SBP recovery patterns after standing with falls and frailty (12). We used this time interval because after 60 seconds the period of interest for “classical” orthostatic hypotension starts. We defined patients with a full-recovery pattern to have recovery to >95% of their baseline SBP. The patients with a partial-recovery pattern had 80%–95%, and patients with a no-recovery pattern had <80% recovery.

All-Cause Mortality

The follow-up period ended on August 2010. Data on vital status were ascertained through linkages with the Dutch municipal administration. When the municipal administration could not provide this information, the general practitioner or the patient’s family was contacted. For one patient, the exact date of death was not discovered, and another patient was lost to follow-up because of emigration. For these two patients, the year and month of death and emigration were known. For survival analysis, we
used the month as the unit of time. Therefore, follow-up was complete for all patients.

**Statistical Analysis**

Characteristics of the patients were compared with one-way analysis of variance or Kruskal–Wallis tests according to their recovery pattern. The results are presented as the means ± standard deviations or percentages unless otherwise stated.

Cox proportional hazards models were used to identify whether ∆SBP, ∆DBP, %SBP<sub>min</sub>, and %DBP<sub>min</sub> predicted mortality. The influence of the recovery of BP on mortality was studied with the same analyses on %SBP<sub>15–20</sub> to %SBP<sub>55–60</sub> and %DBP<sub>15–20</sub> to %DBP<sub>55–60</sub>. Finally, the same was done for the three recovery patterns. All analyses mentioned above were performed with two Cox proportional hazards models. The first model was unadjusted. The second model was adjusted for age, gender, BMI, comorbidity (CIRS-G score), and baseline SBP, DBP, and heart rate. The results were expressed as hazard ratios (HRs) with 95% confidence intervals (CIs). The results were considered to be significant for values of p < .05. All analyses were performed using SPSS software version 16 for windows (SPSS Inc., Chicago, IL).

**Results**

**Baseline Characteristics**

The mean age of the 238 patients was 78.4 ± 7.8 years, and 64% were women. Examples of the three different recovery patterns are shown in Supplementary Figure 1.

Table 1 describes the characteristics of the patients in the different recovery patterns; 95 patients (40%), 98 patients (41%), and 45 patients (19%) showed a full-, partial-, or no-recovery response, respectively. Patients with no recovery had a lower BMI than patients with a full or partial recovery (p < .05). Patients with partial recovery more often used calcium channel blockers than patients with full recovery (p < .05). Patients with no recovery more often used anti-Parkinson medication than patients with a full recovery (p < .05).

**Hemodynamic Response After Active Standing**

The mean overall change in SBP and DBP was 42 ± 25 mmHg and 23 ± 15 mmHg, respectively, for the whole group of 238 patients. Table 2 gives the results for ∆SBP, %SBP<sub>min</sub>, %SBP<sub>55–60</sub>, ∆DBP, %DBP<sub>min</sub>, and %DBP<sub>55–60</sub>, respectively. The absolute initial drop of SBP and DBP did not differ between the three recovery groups, however, as percentage of baseline %SBP<sub>min</sub> and %DBP<sub>min</sub> was slightly larger for the full-recovery response compared with the partial- and no-recovery response. Figure 1 shows the mean relative SBP and DBP responses of the patients with the three different recovery patterns. In the initial 15 seconds, these patterns are more or less the same; however, after 15 seconds, the patterns become substantially different from each other.

**All-Cause Mortality**

During the median follow-up period of 21.0 months (range: 1–61 months), 36 patients (15%) died. There were 8, 16, and 12 deaths in the 95, 98, and 45 patients with a full-, partial-, or no-recovery response, respectively. There was no association between ∆SBP, ∆DBP, %SBP<sub>min</sub>, and %DBP<sub>min</sub> and mortality in the unadjusted model (HR, respectively: 1.01 [95% CI 0.99–1.02]; 1.00 [95% CI 0.98–1.02]; 0.99 [95% CI 0.97–1.01]; and 1.00 [95% CI 0.98–1.01]).

During recovery of SBP after active standing, %SBP<sub>15–20</sub> %SBP<sub>30–35</sub> and %SBP<sub>40–45</sub>–%SBP<sub>55–60</sub> were significantly

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**Table 1. Comparison of the Baseline and Clinical Characteristics of the Patients With Different Blood Pressure Recovery Patterns After Change From Supine to Standing**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Full Recovery (n = 95)</th>
<th>Partial Recovery (n = 98)</th>
<th>No Recovery (n = 45)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>78.1 ± 8.7</td>
<td>78.0 ± 7.4</td>
<td>79.6 ± 6.5</td>
</tr>
<tr>
<td>BMI</td>
<td>27.0 ± 4.7</td>
<td>27.0 ± 4.6</td>
<td>24.7 ± 3.9*</td>
</tr>
<tr>
<td>Women, n(%)</td>
<td>55 (58%)</td>
<td>69 (70%)</td>
<td>29 (64%)</td>
</tr>
<tr>
<td>Baseline systolic BP</td>
<td>167 ± 25</td>
<td>167 ± 28</td>
<td>170 ± 33</td>
</tr>
<tr>
<td>Baseline diastolic BP</td>
<td>78 ± 12</td>
<td>83 ± 21</td>
<td>79 ± 10</td>
</tr>
<tr>
<td>Baseline heart rate</td>
<td>68 ± 12</td>
<td>69 ± 11</td>
<td>68 ± 13</td>
</tr>
<tr>
<td>%SBP&lt;sub&gt;min&lt;/sub&gt;</td>
<td>11.1 ± 4.6</td>
<td>11.1 ± 4.5</td>
<td>10.8 ± 4.4</td>
</tr>
<tr>
<td>Medical history</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dementia</td>
<td>7 (7%)</td>
<td>5 (5%)</td>
<td>4 (9%)</td>
</tr>
<tr>
<td>Depression or anxiety</td>
<td>17 (18%)</td>
<td>18 (18%)</td>
<td>9 (20%)</td>
</tr>
<tr>
<td>COPD</td>
<td>15 (16%)</td>
<td>24 (25%)</td>
<td>5 (11%)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>14 (15%)</td>
<td>18 (18%)</td>
<td>4 (9%)</td>
</tr>
<tr>
<td>Parkinsonism</td>
<td>2 (2%)</td>
<td>5 (5%)</td>
<td>4 (9%)</td>
</tr>
<tr>
<td>CVD</td>
<td>35 (37%)</td>
<td>40 (41%)</td>
<td>21 (47%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>43 (45%)</td>
<td>48 (49%)</td>
<td>23 (51%)</td>
</tr>
<tr>
<td>Malignancy</td>
<td>17 (18%)</td>
<td>17 (17%)</td>
<td>14 (31%)</td>
</tr>
<tr>
<td>Medication use</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>32 (36%)</td>
<td>46 (47%)</td>
<td>32 (34%)</td>
</tr>
<tr>
<td>CCB</td>
<td>9 (10%)</td>
<td>22 (22%)†</td>
<td>7 (16%)</td>
</tr>
<tr>
<td>Diuretics</td>
<td>36 (38%)</td>
<td>36 (37%)</td>
<td>16 (36%)</td>
</tr>
<tr>
<td>ACE/AT2</td>
<td>32 (34%)</td>
<td>38 (39%)</td>
<td>17 (38%)</td>
</tr>
<tr>
<td>Nitrates</td>
<td>14 (15%)</td>
<td>13 (13%)</td>
<td>4 (9%)</td>
</tr>
<tr>
<td>PPI</td>
<td>38 (40%)</td>
<td>35 (36%)</td>
<td>19 (42%)</td>
</tr>
<tr>
<td>Statins/fibrates</td>
<td>31 (33%)</td>
<td>25 (26%)</td>
<td>9 (20%)</td>
</tr>
<tr>
<td>Psychiatric drugs</td>
<td>41 (43%)</td>
<td>30 (31%)</td>
<td>20 (44%)</td>
</tr>
<tr>
<td>Anti-Parkinson drugs</td>
<td>1 (1%)</td>
<td>2 (2%)</td>
<td>4 (9%)*</td>
</tr>
<tr>
<td>Alpha-blockers</td>
<td>4 (4%)</td>
<td>3 (3%)</td>
<td>4 (9%)</td>
</tr>
</tbody>
</table>

Notes: Results are reported as means ± standard deviations or numbers (percentages). ACE/AT2 = angiotensin-converting enzyme inhibitors or angiotensin II receptor antagonists; BMI = body mass index (kg/m²); BP = blood pressure (mmHg); CCB = calcium channel blockers; CIRS-G = Cumulative Illness Rating Scale for Geriatrics; COPD = chronic obstructive pulmonary disease; CVD = cardiovascular disease; heart rate (beats per minute); PPI = platelet aggregation inhibitors.

*Significant differences (p < .05) between patients with full recovery and no recovery.

†Significant differences (p < .05) between patients with partial recovery and no recovery.

‡Significant differences (p < .05) between patients with full recovery and partial recovery.
associated with mortality, even after adjustment for age, gender, BMI, comorbidity (CIRS-G score), baseline SBP, baseline DBP, and baseline heart rate. The same was true for the recovery of DBP at %DBP\textsubscript{25–30} and %DBP\textsubscript{35–40}–%DBP\textsubscript{55–60}, although after adjustment this was no longer significant for %DBP\textsubscript{25–30} (Figure 2). The no-recovery group had a higher mortality risk than the full-recovery group (HR = 2.94; 95% CI 1.20–7.19). The partial-recovery group had an intermediate risk with an HR of 1.77, but this was not significant. The same pattern persisted after adjustment for baseline characteristics with HRs of 3.00 (95% CI 1.17–7.68) and 1.95 (95% CI 0.49–1.87), respectively, for the no- and partial-recovery groups (Table 2). Because calcium channel blocker and anti-Parkinson medication use differed between the different recovery patterns (Table 1), we additionally adjusted for these, but this did not alter the results (Table 3). Figure 3 shows the adjusted Cox proportional hazards cumulative survival curves with respect to the three different recovery patterns.

**Table 2. Comparison of the Results of the Patients With Different Blood Pressure Recovery Patterns After Change From Supine to Standing Position**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Full Recovery (n = 95)</th>
<th>Partial Recovery (n = 98)</th>
<th>No Recovery (n = 45)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ΔSBP (mmHg)</td>
<td>36 ± 24</td>
<td>44 ± 25</td>
<td>50 ± 23</td>
</tr>
<tr>
<td>%SBP\textsubscript{min} (%)</td>
<td>79 ± 13</td>
<td>74 ± 16*</td>
<td>70 ± 24*</td>
</tr>
<tr>
<td>%SBP\textsubscript{55–60} (%)</td>
<td>104 ± 7</td>
<td>88 ± 4*</td>
<td>69 ± 12*</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ΔDBP (mmHg)</td>
<td>19 ± 12</td>
<td>27 ± 19</td>
<td>25 ± 12</td>
</tr>
<tr>
<td>%DBP\textsubscript{min} (%)</td>
<td>76 ± 15</td>
<td>68 ± 16*</td>
<td>68 ± 18*</td>
</tr>
<tr>
<td>%DBP\textsubscript{55–60} (%)</td>
<td>102 ± 11</td>
<td>91 ± 12*</td>
<td>76 ± 14*</td>
</tr>
</tbody>
</table>

*Significant differences \((p < .05)\) between patients with full recovery and partial recovery.
†Significant differences \((p < .05)\) between patients with full recovery and no recovery.
‡Significant differences \((p < .05)\) between patients with partial recovery and no recovery.

**Discussion**

The gravitational effects of standing up on cardiovascular hemodynamics lead to an initial fall in BP that under normal conditions is restored to baseline within 1 minute. The main finding of this study is that impaired BP recovery in this first minute after standing predicts mortality, whereas the magnitude of BP decline within the first 15 seconds does not. The magnitude of the initial BP decline that follows standing is a reflection of hydraulic mechanical properties of the vasculature (leg muscle pumping, abdominal compression, and instantaneous vasodilatation) (3), whereas the

Figure 1. Mean blood pressure response in percentage from baseline upon active standing for the different systolic blood pressure recovery patterns. %SBP = percentage of the baseline systolic blood pressure; %DBP = percentage of the baseline diastolic blood pressure.
subsequent recovery of BP reflects active functioning of the arterial baroreflex, the major mechanism involved in short-term BP regulation (3).

The maximum SBP (42 ± 25 mmHg) and DBP (23 ± 15 mmHg) decline in our study was higher than in a previous study with 40 healthy participants older than 70 years of age (26 ± 23 mmHg and 12 ± 18 mmHg, respectively) (18). This could be the result of involuntary Valsalva straining during standing up in our study, for which we did not explicitly control (19,20). More likely, it is the result of studying different populations, that is, very healthy and active elderly participants versus elderly participants who visit a geriatric outpatient falls clinic. Our results were in line with a study in 442 community-dwelling elderly adults who reported a mean maximum SBP and DBP decline of 36 and 24 mmHg, respectively (12).

We did not measure the exact time it took to assume the upright position, but it is known that elderly participants need a longer time to stand up (3–9 seconds) than younger participants (2–3 seconds) (21). This likely explains why the curves in Figure 1 are slightly shifted rightward compared to younger participants (8,22). Still, the curves are in line with the three different BP response patterns that have been recognized by cluster analysis (12).

Our data add to the literature because for the first time to the authors’ knowledge the impact on mortality of the immediate response of BP in the first minute upon active standing has been studied. There were no differences in comorbidities and cardiovascular disease prevalence or hypertension between the three different recovery pattern groups. The observed higher mortality in the no-recovery group might, therefore, be a consequence of dysfunctional regulatory autonomic function although hypotensive syndromes in geriatric patients are not explained by autonomic dysfunction alone (23). The persistent BP decline and resulting hypoperfusion of organs might directly result in syncope, falls, and even mortality. However, seen as a causal risk factor, not the amount but the duration of the...
BP decline might be of more importance for mortality risk. This is in line with the observation that duration of SBP decline following active standing or sinus carotid massage is more important as a determinant of symptoms than SBP nadir or delta (24,25).

However, a noncausal indirect relation of an impaired BP recovery (as a risk marker) with mortality is more likely. Active standing is a perturbation test, which elicits a more or less similar response in the initial BP decline in all participants. However, the regulatory capacity to normalize this perturbation largely differs between participants. This failure to normalize BP is associated with excess mortality. In this context, BP recovery in the first minute after active standing can be seen as a physical sign that reflects a final common pathway of various forms of subclinically impaired physiology (26). Therefore, this BP recovery pattern fits in the search for easy, simple, and noninvasive indicators of mortality in elderly people as, for example, gait speed or the (modified) physiologic index (27–29).

For clinical application, the results of these two components of the BP response after standing (ie, drop and recovery) should be related to the well-known syndrome of orthostatic hypotension. Orthostatic hypotension is defined as a drop in SBP of at least 20 mmHg (30 mmHg in case of hypertension with resting SBP greater than 160 mmHg) and/or 10 mmHg in DBP often measured between 1 and 3 minutes after standing (30). The percentage of patients with orthostatic hypotension increased from 18% (17/94) in the full-recovery patients to 55% (53/97) and 100% (45/45) in the partial- and no-recovery patients, respectively. In our previous study, there was no relation between orthostatic hypotension and mortality after adjustment for confounders (13). Therefore, the recovery of SBP after 1 minute should be regarded as easily obtainable, additional information having better predictive value for mortality than the presence or absence of orthostatic hypotension.

Strengths and Limitations
This study has several strengths. First, the follow-up in our study was complete. Second, the BP measurements in our study were performed under standardized circumstances, with a beat-to-beat finometer, which is an accurate way to measure BP variability (31). Third, comorbidity was documented very thoroughly with the CIRS-G. The CIRS-G is frequently used in other studies, and good reliability and validity have been proven (14,32). Fourth, confounding effects of medication on the BP response were limited as much as possible. All medication was withheld from midnight, the night before the test. Moreover, there were no differences in medication use, except that patients with an impaired recovery (partial and
no-recovery pattern) used more anti-Parkinson medication or calcium channel blockers. However, post hoc correction for this medication use did not change the relation with mortality for these patients. Finally, we investigated a rather large group of 238 elderly patients on the relation of their response upon standing and mortality.

This study has also some limitations. First, this study was a retrospective analysis. The patients received a multifactorial falls intervention, which could have influenced mortality. However, a systematic review and meta-analysis showed no influence of multifactorial falls intervention on mortality among older people in community and emergency care settings (33). Thus, although unlikely, we cannot completely rule out that our results may have been confounded by these multifactorial interventions. Second, we analyzed all-cause mortality but not cause-specific mortality because it was impossible to reliably determine the cause of death. In older people, the cause of death is often unclear or related to multiple problems, and thus, it is hard to validly assess without postmortem data. Finally, this study investigated a selected patient population. All patients were referred because of syncpe, falls, and/or dizziness. This has the disadvantage of a specific selection, and therefore, our sample is not representative for the general population but only for populations referred to similar outpatient clinics. On the other hand, this is the geriatric population with the highest relevance of BP dysregulation.

CONCLUSIONS

In falls clinic patients, failure to recover from BP decline in the first minute after active standing is associated with excess mortality. Therefore, SBP recovery to less than 80% from baseline after 60 seconds may be used as an easily available cardiovascular sign for increased mortality risk in older falls clinic patients.

SUPPLEMENTARY MATERIAL

Supplementary material can be found at: http://biomedgerontology.oxfordjournals.org/.

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