Orthostatic hypotension: framework of the syndrome

Jochanan E Naschitz and Itzhak Rosner

doi:10.1136/pgmj.2007.058198

Updated information and services can be found at:
http://pmj.bmj.com/cgi/content/full/83/983/568

These include:

References
This article cites 85 articles, 28 of which can be accessed free at:
http://pmj.bmj.com/cgi/content/full/83/983/568#BIBL

Rapid responses
You can respond to this article at:
http://pmj.bmj.com/cgi/eletter-submit/83/983/568

Email alerting service
Receive free email alerts when new articles cite this article - sign up in the box at the top right corner of the article

Topic collections
Articles on similar topics can be found in the following collections

Other geriatric medicine (597 articles)
Other Cardiovascular Medicine (2102 articles)
Other Neurology (3667 articles)

Notes

To order reprints of this article go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to Postgraduate Medical Journal go to:
http://journals.bmj.com/subscriptions/
Orthostatic hypotension: framework of the syndrome

Jochanan E Naschitz, Itzhak Rosner

According to the 1996 consensus definition, orthostatic hypotension (OH) is diagnosed when a fall in systolic blood pressure of at least 20 mm Hg and/or diastolic blood pressure of at least 10 mm Hg within 3 min of standing is recorded. The elements of orthostatic blood pressure drop that are relevant to the definition of OH include magnitude of the drop, time to reach the blood pressure difference defined as OH, and reproducibility of the orthostatic blood pressure drop. In each of these elements, there exist issues that argue for modification of the presently accepted criteria of OH. Additional questions need to be addressed. Should one standard orthostatic test be applied to different patient populations or should tests be tailored to the patients’ clinical circumstances? Are different OH thresholds relevant to various clinical settings, aetiologies of OH and comorbidity? Which test has the best predictive power of morbidity and mortality?

Orthostatic hypotension (OH) has been formally defined by expert consensus as a fall in systolic blood pressure (SBP) of at least 20 mm Hg and/or diastolic blood pressure (DBP) of at least 10 mm Hg within 3 min of standing. When a normal individual stands, 10–15% of the blood is pooled in the legs, thereby secondarily reducing venous return, cardiac output and arterial pressure. This fall in blood pressure activates baroreceptors with a subsequent reflex increase in sympathetic outflow and parasympathetic inhibition, leading in turn to peripheral vasoconstriction and increased heart rate and contractility. There may be a slight fall in SBP, a slight rise in DBP and a mild increase in heart rate accompanying this chain of events. Orthostatic hypotension is an excessive fall in blood pressure occurring early on postural challenge when orthostatic stress overwhelms autonomic defences. Numerous factors may influence blood pressure homeostasis and impact on the occurrence of OH: autonomic nervous system function, intravascular volume, duration of erect posture, time of day, postprandial state and ambient temperature. Dizziness and syncope are the classical symptoms of postural stress and their connection to OH is readily established. The relationship of other symptoms to OH may not be obvious, unless one is aware of the wider spectrum of orthostatic disturbances. Thus, weakness, fatigue, visual blurring, vertigo, suboccipital and paracervical pain, chest pain on upright posture, headaches, palpitations, low back pain or dyspnoea may occur on standing and disappear when lying flat.

The diagnostic consensus criteria defining OH are necessarily arbitrary. There are questions open as to the magnitude of hypotension and rapidity of its development in the definition of OH. Patients may feel dizzy, weak or even faint upon standing while orthostatic blood pressure decrement is insufficient to meet the criteria for OH. And yet, if a patient has posturally induced symptoms and a decrease in BP but does not fit the strict definition for OH, it has been suggested that one may still consider OH as a possible diagnosis. Therefore, in limiting one’s self to the consensus criteria for the definition of OH one may lose useful clinical information.

IMPORTANCE OF DIAGNOSING ORTHOSTATIC HYPOTENSION

The burden of OH on public health is substantial, with a prevalence of 7% in the elderly, even when excluding patients with known risk factors to this condition. It increases to 13% and 55% in high risk groups. The prevalence of OH among patients presenting with syncope in the emergency room was 24% in one study and 64% among elderly patients hospitalised for acute conditions.

In the clinical setting, the diagnosis of OH has a threefold significance: OH may underlie symptoms of cerebral hypoperfusion as well as the above enumerated systemic symptoms; OH may be a risk factor for falls; it may be a marker of frailness and predictor of mortality.

Epidemiological studies have shown that individuals with OH have a propensity for falls. A prospective study of 844 elderly long-term nursing home residents were evaluated during a mean of 1.2 years. Among those with a fall in the previous 6 months, those with OH had an increased risk for recurrence (adjusted relative risk (RR) 2.1). Those who had OH demonstrated on two or more measurements had the greatest risk (RR 2.6). The association between OH and recurrent falls was independent of other measured demographic or clinical risk factors for falls. In disagreement with the latter are results of a meta-analysis, showing that OH was not an independent risk factor of falls in elderly community-dwelling persons. OH is predictive of ischaemic stroke. In 11 707 persons from the Atherosclerosis Risk in Communities cohort who were free of stroke and overt heart disease at baseline, the association between OH and incident ischaemic stroke over 7.9 years of follow-up was assessed. OH was independently predictive of ischaemic stroke, even after

Abbreviations: DBP, diastolic blood pressure; HUTT, head-up tilt test; OH, orthostatic hypotension; RR, relative risk; SBP, systolic blood pressure
Orthostatic hypotension

Search strategy and selection criteria

We searched PubMed using the key words “orthostatic hypotension” and “postural hypotension” with the relevant topics—“epidemiology,” “diagnosis,” “tilt test,” “symptoms,” “syncope,” “falls,” “morbidity” and “mortality.” Fields were restricted to publications in English. We also searched the citations from obtained papers. References were chosen based on the best evidence from clinical or laboratory studies. Review articles of orthostatic hypotension published in the past 10 years were also examined to ensure minimum overlap with this review article. Recent articles that provided comprehensive overviews were included where appropriate instead of multiple references of original work.

adjustment for numerous other stroke risk factors (hazard ratio 2.0). Also, OH was found to be a clinical correlate of pathological white matter findings on cranial magnetic resonance imaging in elderly people (p < 0.001).

OH is an independent predictor of all-cause mortality. In a prospective study of a cohort of 3522 elderly Japanese American men, OH, as measured in the supine position and after 3 min of standing, was assessed in relation to subsequent 4 year all-cause mortality. The overall prevalence of OH was 6.9% and it increased with age. Four year age-adjusted mortality rates in those with and without OH were 56.6 and 38.6 per 1000 person-years, respectively. With the use of Cox proportional hazards models, after adjustment for age, smoking, diabetes mellitus, body mass index, physical activity, seated SBP, antihypertensive medications, haematocrit, alcohol intake, and prevalent stroke, coronary heart disease and cancer, OH was a significant independent predictor of 4 year all-cause mortality (RR 1.64). There was a significant linear correlation between change in SBP from supine position to standing and 4 year mortality rates (p = 0.001), suggesting a “dose–response” relationship.

Another study evaluated the risks of non-vascular and vascular deaths, according to different definitions of OH, among 792 home-dwelling persons aged 70 years or older. The hazard ratio for vascular death associated with diastolic OH 3 min after standing up, adjusted for other significant factors associated with vascular death, was 2.04. The corresponding hazard ratio for systolic OH was 1.69. In another work the risk for vascular death associated with OH was especially high among diabetic patients; the hazard ratio of vascular death associated with diastolic OH was 3.69 and 2.7 with systolic OH.

METHODOLOGY

The diagnosis of OH depends on blood pressure measurement technique, positioning of the patient and timing of measurements.

Blood pressure measurement techniques

The mercury column sphygmomanometer is preferred by us and by others for routine clinical testing because of its reliability and simplicity. This is the standard method to which other non-invasive devices of blood pressure measurement are validated. Automatic arm-cuff devices, as they are programmed to repeat and confirm measurements when discrepant values are recorded, may be at a disadvantage in following the rapidly dropping blood pressure during OH. Auscultatory measurements of blood pressure should be done by a physician skilled in the technique recommended by the American Heart Association. To avoid an erroneous measurement, the cuffed arm should be positioned so that the brachial artery is held at the level of the heart. Potential confounding factors in detection of OH include: inadequate cuff inflation in those with an auscultatory gap; rapid cuff deflation in those with a slow or irregular pulse rate leading to underestimation of the SBP; and repeated measurements at intervals more frequently than 15 s causing venous stasis of the arm.

Beat-to-beat non-invasive blood pressure measurement with the Finapres monitor is widely used in research and tilt laboratories. The Finapres blood pressure monitor uses the method of Penaz to record the arterial waveform indirectly. Studies on its accuracy have suggested little systematic bias versus intra-arterial pressure but substantial variability. Inconsistency between studies, as relating to magnitude, direction and variance of bias, has been described in validation studies versus direct intra-arterial blood pressure. However, in combined data from 20 published studies, the average (SD) Finapres systolic bias was 2.2 (12.4) mm Hg and the Finapres diastolic bias was −0.3 (7.9) mm Hg. Another beat-to-beat non-invasive blood pressure measurement device has been developed to monitor radial artery pressure and its waveform based on arterial tonometry. Arterial tonometry provides near continuous and non-invasive arterial blood pressure monitoring and may be an alternative to direct intra-arterial measurement. It has been evaluated in adult and paediatric patients. Excellent agreement between arterial tonometric and intra-arterial blood pressure was documented.

Heart rate monitoring

Heart rate recording is integral to testing for OH and is indispensable to the differentiation between certain clinical syndromes. At the bedside, the heart rate is acquired intermittently together with blood pressure measurement when using standard automatic blood pressure measurement devices. The method routinely used by us, along with intermitted blood pressure measurements by mercury column sphygmomanometry, is continuous electrocardiographic heart rate monitoring. The option generally favoured is by beat-to-beat tracking of blood pressure and heart rate via Finapres. In a normal subject, the heart rate will increase by 5–12 beats/min when arising from recumbency. Failure of the heart rate to rise concurrent with a fall in blood pressure upon arising is indicative of sympathetic autonomic insufficiency—that is, neurogenic OH is typically associated with a blunted heart rate response on standing or passive tilt. There are exceptions to this rule: healthy older individuals may display a similarly blunted heart rate response due to downregulation of baroreceptors related to aging; when the heart rate may be slowed by medication; or there may be a paradoxical increase in heart rate in patients with autonomic failure, presumably due to parasympathetic withdrawal. On the other hand, an exaggerated increase in heart rate in the erect position may be indicative of a contracted intravascular volume.

Orthostatic stress tests

The ideal orthostatic test should be suitable for assessment of a variety of clinical conditions, should correspond to real life situations, be simple to perform, its instruments should be generally available, and should require minimal patient cooperation. Of the tests in common use, usually in assessment of dizziness and syncope, the lying-to-standing orthostatic test appears the closest fit, while the head-up tilt test (HUTT) is farthest from representation of the real-life situation. Few studies have compared results of different orthostatic tests directly. It has been suggested that the orthostatic fall in SBP may be greater on HUTT than on standing, but data on this issue are scarce. Many more studies of this nature are required for any definitive conclusions.

Three orthostatic challenges are widely applied in practice: the lying-to-standing test, lying-to-sitting test, and the head-up
The head-up tilt test

A more accurate evaluation of the haemodynamic response to postural challenge may be obtained with HUTT. This method utilises a controlled passive postural stress to challenge the cardiovascular response as measured in blood pressure and heart rate changes. HUTT comprises two phases: (1) supine pre-tilt phase; and (2) the passive head-up tilt. Various protocols are used in performing this test. According to recommendations of the European Society of Cardiology, the supine pre-tilt phase should last at least 5 min, when no venous cannulation is performed, and at least 20 min when venous blood sampling is part of the study; the tilt angle recommended is 60°–70°; the duration of passive tilt should be a minimum of 20 min and 15 min or more, [7, 28] or at “first morning standing up”. [29] However, there are individuals in whom onset of hypotension on standing may be delayed and these cases may be missed by the short bedside orthostatic test. [30, 31]

The lying-to-sitting orthostatic test

The methodology of the lying-to-sitting orthostatic test is not standardised. [32–35] One frequently used protocol involves a single blood pressure measurement after prolonged recumbence followed by blood pressure measurements after 1, 3 and 5 min of sitting. [36] The others differ in the number and timing of blood pressure measurements. Other technical details are similar to those of the above described supine-to-standing test.

 VALIDITY OF ORTHOSTATIC HYPOTENSION TESTING

Two important aspects relating to the validity of OH testing are accuracy and reproducibility of the measurements. Accuracy refers to the agreement between a measured value and its “true” value (or values that are as close to the true values as possible—that is, the gold standard). Reproducibility of the measurements is calculated by comparing several measurements taken in the same subject. Ideally, measurements utilised in OH testing should be both accurate and reproducible.

Reproducibility of cardiovascular responses on orthostatic challenge has been inconsistent. While some studies have observed gross reproducibility of OH, [37–39] others, on detailed examination, have shown uneven results on repeated challenges. [40–42] In one study utilising HUTT, asymptomatic orthostatic falls in SBP were reproducible 1 week after initial testing. [43] In another study in healthy volunteers, medium-term (1 week) and long-term (1 year) baroreflex responses proved to be reliable (r = 0.54–0.87). [44]

Variability of postural responses is well documented, with diurnal, day-to-day and seasonal patterns (see box 1). To evaluate the stability of OH during the day, orthostatic tests were performed three times during the day on 502 acute geriatric ward inpatients (mean age 81.6 years). Sixty-eight per

There is uncertainty as to the minimum duration of postural stress needed to detect OH. In a recent study, 88% of patients developed OH by 1 min of tilt, with an additional 11% developing OH by 2 min and the remaining 1% developing OH by 3 min. [45] Another study (Naschitz et al, personal communication, 2007) extracted data from 400 charts of consecutive patients evaluated for syncope or non-specific dizziness by HUTT. “Classical” OH was defined as reduction of SBP of ≥20 mm Hg and/or diastolic blood pressure of ≥10 mm Hg within 3 min of HUTT. A similar decrease in SBP occurring during the 4th or 5th min of HUTT, not associated with a decrease in heart rate by >10%, was referred to as “late” OH. In this study, 17.2% of patients developed OH by 1 min of tilt, another 9.5% by 2 min, and 4.7% more by 3 min. “Late” OH was diagnosed in an additional 7.2% of patients. Patients with “classical” and “late” OH had comparable clinical characteristics: systolic OH, diastolic OH and mixed systolic–diastolic OH occurred with similar frequency in “classical” and “late” OH; the “classical” and “late” OH were associated with identical heart rate changes on tilt, differing from vasovagal reactions; on continuation of tilt, after OH, blood pressure behaviour was similar in both groups. Thus, by all parameters examined, OH in the first 3 min and that occurring at the 4th and 5th min were comparable, different from the earlier described “delayed orthostatic intolerance”. [46]

Results of this study indicate that extending postural stress to 5 min may reveal a significant number of additional cases of OH.

Streten et al first drew attention to “delayed” OH, that occurred 13–30 min of standing. [47] In a recent study, “delayed” OH occurred as frequently as classical OH: out of 108 having OH, 46% developed in the first 3 min of tilt, 3% between 3–5 min, 12% between 5–10 min, and 39% after 10 min of tilt. Patterns of OH, describing the time course of the SBP during the initial 5 min of tilt, have been recently described: [48] stable OH; transient systolic OH with either early or late partial recovery of blood pressure; and progressive systolic OH with continuous reduction in blood pressure. Patients with progressive OH had significantly higher score on the adrenergic subscale of the Composite Autonomic Symptom Score and are assumed to have more severe adrenergic impairment. The progressive systolic OH pattern predicts increased risk of developing syncope on continuing postural challenge. [49] A recent study confirmed the correlation of progressive systolic OH with early tilt termination due to syncope. [50] Five minutes of orthostatic challenge also permit assessment of OH patterns, thereby providing additional information beyond the classical 3 min test.

www.postgradmedj.com
cent of patients manifested OH on one of the measurements, but only 35% on two or all three of the measurements. OH was most prevalent and severe in the morning, when subjects first arose. Several studies have shown that OH is underestimated when orthostatic testing is performed in the afternoon. In addition, the influence of meals on diurnal variation of OH has been observed. Postprandial hypotension, almost immediately apparent after a meal, can be found after any meal during the day. Blood pressure usually reaches a nadir within 30–60 min postprandially. The nutrient composition of meals seems to affect the magnitude of the decrease in postprandial blood pressure. Carbohydrates and, more specifically, glucose have been found to play a significant role.

A study evaluated nurses’ skills of supine and standing blood pressure measurements performed by nurses. One study evaluated nurses’ skills of supine and standing blood pressure measurement with absolute blood pressure levels, but also the correlation with the magnitude of the blood pressure drop on orthostasis is reflected by central blood pressure as measured non-invasively in the carotids. When carotid and brachial blood pressure were measured simultaneously using cuff-oscillometric and tonometric methods the orthostatic decline in blood pressure was more prominent in the carotid artery. Thus, in one study, while nine subjects were diagnosed with OH via the brachial blood pressure, 21 subjects were diagnosed with carotid blood pressure (p<0.001), results indicating that evaluation of OH by brachial blood pressure may underestimate OH.

### SYMPTOMATIC VERSUS ASYMPTOMATIC ORTHOSTATIC HYPOTENSION

The occurrence of orthostatic symptoms, largely representative of inadequate central nervous system perfusion with a fall in blood pressure, depends not only on the magnitude of the blood pressure decline upon assuming upright posture (fig 1), but also on a number of other factors. Cerebrovascular autoregulation acts as a safeguard to protect the brain against excessive oscillations of blood pressure. In the average individual under normal physiologic conditions, changes in mean arterial blood pressure between 60 and 160 mm Hg produce little or no change in cerebral blood flow. Thus, cerebral arterioles dilate as mean arterial blood pressure decreases and constrict as mean arterial blood pressure increases. Beyond these limits of autoregulation, cerebral blood flow is directly proportional to mean arterial blood pressure and is “pressure-passive”. A sudden decrease in cerebral blood flow occurs at the lower limit of autoregulation.

The limits of cerebral autoregulation, and hence susceptibility to orthostatic symptoms, can be affected by a number of conditions. For example, in chronically hypertensive adults, the autoregulatory curve is shifted to the right. Therefore, while in otherwise normal individuals orthostatic symptoms may appear with a drop in the mean blood pressure below 60 mm Hg, in hypertensives a less drastic drop in blood pressure will be accompanied by symptoms. In more acute conditions (for example, ischaemic stroke, subarachnoid haemorrhage, intracerebral haemorrhage and post-ictal), cerebral blood flow may become temporarily pressure passive, thus augmenting effects of OH on cerebral blood flow. In addition, hypercapnia (PaCO₂ >60 mm Hg) as well as hypocapnia (PaCO₂ <25 mm Hg) may consistently impair cerebral autoregulation and distort the correlation between symptoms and OH.

Not only do orthostatic symptoms not necessarily correlate with absolute blood pressure levels, but also the correlation with the magnitude of the blood pressure drop on orthostasis is
not constant. It has been proposed that symptoms induced during a decrease in blood pressure, not fully meeting the strict definition for OH, should still be considered expressions of possible OH. Recently, the Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure (the JNC 7 report) stated that a 10 mm Hg SBP drop should be regarded as clinically relevant when it is associated with symptoms.

Orthostatic symptoms may correlate with the nadir blood pressure on orthostasis, with the magnitude of blood pressure drop, and also with the rate of blood pressure change. Thus, in a study of 400 patients who had HUTT for evaluation of non-specific dizziness, the feeling of dizziness on tilt occurred uniformly only in patients in whom orthostatic criteria were met within 3 min, while only 25% of patients having a more gradual decrease in blood pressure complained of dizziness. Symptoms occurring with the onset of OH are routinely diagnosed as “orthostatic”. It should be noted, however, that similar symptoms may be induced by postural change, but without a drop in blood pressure, and be caused by a panic attack, occult hyperventilation, cerebrovascular disease or postural tachycardia syndrome.

The relationship between symptoms occurring during an orthostatic test, essentially representative of a laboratory phenomenon, and symptoms occurring in the patients’ natural ambience is not always clear. In one study, the finding of OH on HUTT and its connection to dizziness, which was the patients’ presenting complaint, was not straightforward. In another study there was also no statistically significant association between orthostatic symptoms and detection of OH. Thus, on the one hand, orthostatic symptoms may occur in the absence of measurable OH and, on the other hand, detected OH may be asymptomatic.

ARE PRESENT ORTHOSTATIC HYPOTENSION DIAGNOSTIC CRITERIA ADEQUATE?
The elements of orthostatic blood pressure drop that are relevant to the definition of OH include magnitude, time to reaching the blood pressure difference defined as OH, and reproducibility of orthostatic blood pressure drop.

Magnitude of orthostatic blood pressure decrease
It is unknown whether the above described lower OH threshold represents a general phenomenon or reflects varying susceptibilities of patient subsets to decreases in blood pressure in different clinical settings, aetiologies of OH and presence of comorbidity. For example, in chronic hypertension, acute traumatic brain injury, ischaemic cerebrovascular disease, subarachnoid haemorrhage, post-ictal state or hypercapnia, where cerebral autoregulation is impaired, a relatively smaller decrease in blood pressure on standing may result in a clinically important decrease in cerebral blood flow. Possibly, the magnitude of decrease in blood pressure required for diagnosing OH in the above conditions should be lower than presently recommended. In addition, a lower OH cut-off at 3 min of orthostatism may be warranted in patients exhibiting the “progressive systolic OH pattern” described by Gehring et al. This subset is associated with an increased risk of syncope later on continued postural challenge.

Furthermore, different OH cut-offs may be relevant in utilising the postural test as a diagnostic test in patients with presyncope or utilising the orthostatic test as a screening tool in evaluating the risk of falls, cardiovascular events and mortality. In the Honolulu Heart Program, there was a significant dose-response relationship between orthostatic change in blood pressure and mortality. The authors suggest that a better definition of OH in this cohort, in terms of its relation to subsequent mortality, would refer to a decrease in SBP of >10 mm Hg or a decrease in diastolic blood pressure of >5 mm Hg. The JNC 7 report recommend use of a 10 mm Hg blood pressure drop in blood pressure when associated with symptoms as clinically relevant.

Time threshold for orthostatic hypotension
As shown above, the optimal time duration of orthostatic challenge for the diagnosis of OH is probably longer than the 3 min set in the consensus definition. A 5 min test may permit the identification of systolic OH patterns, which, in turn, correlate with the severity of autonomic failure and predict the risk of syncope on continuing postural challenge. A 5 min test may also reveal a significant number of additional cases of OH (Naschitz et al, personal communication).

Repetitions of orthostatic tests required
A single orthostatic test demonstrating the presence of OH is sufficient for diagnosis. As OH is poorly reproducible, it has been shown that several measurements may be required, on several occasions, to detect OH. To improve sensitivity, readings should be timed for that time of the day when symptoms are reported to occur. In addition, assessing reproducibility of OH over several weeks may be helpful in differentiating acute OH (main causes: intercurrent illness, medications, transient hypovolaemia) from chronic OH (main cause: autonomic failure, baroreceptor desensitisation, hypoadrenalism).

A call for revision of the OH criteria has been recently expressed. The evidence cited above suggests there may be room for changes in the criteria with respect to the magnitude of OH drop, the time threshold for OH, and number of measurements required in order to establish the diagnosis of OH. Further questions may need to be addressed by the criteria. Should one standard orthostatic test be applied to different patient populations or should tests be tailored to the patients’ clinical circumstances? Are different OH thresholds relevant to various clinical settings, aetiologies of OH and comorbidity? Which test has the best predictive power of morbidity and mortality?

Key references
Orthostatic hypotension

mortality? There is at present no adequate response to the posed questions.

Standardising the assessment of OH is an intermediary goal, which could permit us to identify more accurately OH-related thresholds for falls, stroke and cardiovascular mortality. To this end, comparative studies of the variety of postural tests and instruments should be carried out. Multicentre studies are needed to compare prospectively the prognostic impact of OH as diagnosed according to present consensus criteria versus OH diagnosed by utilising a 10 mm Hg or 15 mm Hg SBP drop, a 5 mm Hg DBP drop and extending the duration of the postural challenge to 5 min. It is recognised that in reducing the blood pressure drop threshold and extending the duration of the test many more individuals may then be diagnosed with OH, with an unclear impact on medical care. On the other hand, a requirement for reproducibility of OH, which should be incorporated into the prospective studies, may again limit the number of those thus diagnosed.

MULTIPLE CHOICE QUESTIONS (TRUE (T)/FALSE (F); ANSWERS AFTER THE REFERENCES)

1. The following symptoms may be expressions of orthostatic hypotension:
   (A) Dizziness
   (B) Fatigue
   (C) Nocturia
   (D) Chest pain in upright posture

2. The following illustrate the relationship between orthostatic hypotension and orthostatic symptoms:
   (A) Orthostatic symptoms may occur in the absence of orthostatic hypotension
   (B) Orthostatic hypotension may occur in the absence of orthostatic symptoms
   (C) Population based studies showed no significant dose-response relationship between orthostatic change in blood pressure and mortality
   (D) A 10 mm Hg drop in systolic blood pressure when associated with symptoms is clinically relevant

3. The occurrence of orthostatic hypotension is affected by:
   (A) Time of the day
   (B) Season
   (C) Postprandial state
   (D) Febrile illness

4. In assessing orthostatic hypotension:
   (A) The golden standard is the head-up tilt test
   (B) Beat-to-beat blood pressure monitoring is essential for the diagnosis of orthostatic hypotension
   (C) Reproducibility of orthostatic hypotension has been definitely established
   (D) Monitoring heart during orthostatic tests is non-contributory

5. The burden of orthostatic hypotension on public health is reflected by the following:
   (A) According to different studies, the prevalence of orthostatic hypotension was 7% in elderly subjects and 55% in high risk groups
   (B) The prevalence of orthostatic hypotension among patients presenting with syncope in the emergency room was 24%
   (C) Orthostatic hypotension is independently predictive of ischaemic stroke
   (D) Orthostatic hypotension is independently predictive of cardiovascular mortality

Authors’ affiliations
Jochen E Naschitz, Department of Internal Medicine A, Bnai Zion Medical Center and Bruce Rappaport Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel
Itzhak Rosner, Department of Rheumatology, Bnai Zion Medical Center and Bruce Rappaport Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel

Competing interests: None.

REFERENCES


34 Naschitz, Rosner

35 table test


ANSWERS

1. (A) T (B) T (C) T (D) T

2. (A) T (B) T (C) F (D) T

3. (A) T (B) T (C) T (D) T

4. (A) B (F) C (F) D (F)

5. (A) T (B) T (C) T (D) T