Measurement and implications of postural changes in blood pressure

Postural changes in blood pressure are a commonly elicited vital sign. It is important that the measurements are reproducible in order to be clinically meaningful. This article makes recommendations about how and what to document as part of the technique of recording, on the basis of the literature.

The measurement of postural changes in blood pressure is considered to be an important vital sign in clinical practice. It is essential for the assessment of the elderly patient, especially with symptoms of potential postural origin, such as dizziness and syncope, and after an unexplained fall. It can also be used to evaluate possible significant concealed loss of circulating blood volume at an early stage, for example in the presence of occult gastrointestinal bleeding.

Postural or orthostatic hypotension has been defined as a reduction of systolic blood pressure of at least 20 mmHg or a reduction of diastolic blood pressure of 10 mmHg or more within 3 minutes of quiet and unsupported standing (Kaufmann, 1996).

The diagnosis of postural hypotension is dependent on appropriate measurement of supine and standing blood pressures under standardized conditions. In hospital practice, these readings are usually taken by nurses or by health-care assistants, but may also rarely be taken by medical staff. Inter-observer variation in the recording of vital signs in the emergency department has been recognized (Edmonds et al, 2002), and this may be compounded by variation in recording technique (Vloet et al, 2002). The causes of postural hypotension can be neurogenic or non-neurogenic. Neurogenic causes include:

- Primary autonomic failure
- Sensory neuropathies: diabetes mellitus, alcohol
- Multiple system atrophy
- Parkinson's disease with autonomic failure.

Non-neurogenic causes include:

- Volume depletion: haemorrhage, gastrointestinal fluid loss, diuretic therapy
- Increased age
- Medications: antidepressants; alpha blockers.

The authors became aware of variations in techniques used for measuring postural changes in blood pressure between health-care professionals in their practice, both in terms of the time intervals for recording and the preliminary preparation of the patient. They therefore explored the literature to evaluate the sources of guidance used regarding standards for postural blood pressure measurement.

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Methods

A literature search was performed using Medline and Embase databases from 1970 and 1974 respectively to the present, using the key words postural hypotension, orthostatic hypotension and blood pressure measurement. Potential sources of guidelines were explored, including the Cochrane Library, and the British Society for Hypertension and European Society of Cardiology guidelines. Standard textbooks on internal medicine, cardiology and neurology were consulted. The primary sources chosen were those which described methods of measuring postural blood pressure changes and those which attempted to correlate these changes with real-time symptoms. Any references selected had their reference lists were checked for other potential sources.

Discussion

On standing, gravitational pooling of around 500–800 ml of blood occurs in the distensible venous capacitance vessels in the lower limbs and in the splanchnic vasculature. This leads to reduced baroreceptor activation and a reflex increase in sympathetic activation of the peripheral circulation. This is associated with arteriolar and venous constriction, sympathetic-mediated positive inotropic and chronotropic effects on the myocardium, and activation of the renin–angiotensin–aldosterone system. The purpose of these changes is to maintain cerebral perfusion in the upright posture.

Normally, there is a postural increase in heart rate of around 30 beats per minute immediately after assumption of the standing position. There is a superimposed 10% reduction in plasma volume caused by filtration of plasma-free fluid into the interstitial spaces as a result of a high capillary transmural pressure gradient (Brignole et al, 2004).

Postural hypotension is an important clinical condition, being found in 10–30% of patients with ambula-

tory hypertension over the age of 60 years and in 50% of those in wards for the elderly (Weiss et al, 2004). In a generally healthy population of 4736 elderly participants enrolled in the Systolic Hypertension in the Elderly Program (SHEP), postural hypotension was found in 10.4% at 1 minute after rising from a seated position and in 12% at 3 minutes, with 5.3% having hypotension at both time intervals (Applegate et al, 1991). The only predisposing factor for postural hypotension in an unselected elderly population was coexisting hypertension (Räihä et al, 1995). Causative factors include arterial stiffness and baroreceptor insensitivity. Postural hypotension often coexists with supine hypertension and is also a well-recognized feature of syndromes of autonomic failure.

Postural hypotension can lead to cerebral hypoperfusion, with global cerebral dysfunction, on changing from the supine to the erect position. There is a variable relationship between CNS symptoms and the magnitude of postural blood pressure change. The symptoms themselves are often non-specific. Furthermore, the rapidity of rising from the supine to the standing position and diurnal variation (postural changes being accentuated in the morning) may affect readings.

The heart rate and blood pressure responses to the head-up posture have been placed in one of five categories (Khurana, 1995; Wieling and Karamaker, 1999). These categories vary in the magnitude and duration of systolic blood pressure and diastolic blood pressure reduction, and in the presence or absence of associated heart rate changes.

At least 20 minutes of prior recumbency has been recommended to allow for a stable level of blood pressure (Vargas and Lye, 1982). It must also be noted that while taking a sitting blood pressure may be convenient, such recordings can miss postural hypotension in up to two-thirds of patients (Carlson, 1999). Standing for less than 2 minutes might overstate the degree of hypotension, owing to age-related blunting of baroreceptor reflexes in otherwise normal individuals (Engstrom and Aminoff, 1997).

The concurrent measurement of heart rate is potentially important, as in neurogenic causes of orthostatic hypotension there is an absence of the usual compensatory increase in heart rate, which may exceed 15–30 beats per minute. Furthermore, the syndrome of postural orthostatic tachycardia syndrome, which is a subtype of postural syncope, may not be recognized. Postural orthostatic tachycardia syndrome is characterized by an increase in heart rate of at least 30 beats per minute after 10 minutes of standing, or a heart rate of 120 beats per minute or over following prolonged standing.

The authors did not find any specific allusion to same arm measurement. In manual measurement, the disappearance of sound (Korotkow phase V) is regarded as being closer to true diastolic blood pressure.

Areas of uncertainty

Some unclarified issues were identified in this literature search. It is interesting to note that as baseline blood pressure may fall slightly on repeated testing it has been suggested that blood pressure should be measured in a sequence of lying–standing–lying, comparing the second pair of readings (Donaghy, 2005). It should also be noted that readings taken within 2 hours of a meal may be unrepresentative (Lipsitz et al, 1983; Haigh et al, 1991). This should be considered and acted on in hospitalized patients, where measurements can be taken at controlled periods, as opposed to emergencies, where nothing can be done about in relation to time of presentation.

There appears to be no consistent correlation of acute blood loss with postural vital signs with losses under 500 ml. This is based on studies of phlebotomised adult volunteers and blood donors. Kosowsky et al (2002) demonstrated that change in heart rate with postural changes after a 500 ml phlebotomy was more discriminatory for the blood loss than blood pressure changes or the change in bioimpedance-based stroke index changes. In a meta-analysis of postural vital signs (McGee et al, 1999), a large postural pulse change (> 30 beats per minute) or severe postural dizziness preventing the completion of vital sign measurement supports hypovolaemia following acute blood loss.

Conclusions

The reviewed literature demonstrates that there is some lack of uniformity about the precise performance of the procedure for measuring postural hypotension. It is important, however, to have an agreed and reproducible measurement technique for recognizing postural hypotension, and to have guidance on interpretation of the findings elicited. This article demonstrates the need for a clear protocol for measuring postural blood pressure, which awaits clarification. The authors stress the importance of correlating postural blood pressure changes with changes in heart rate and in the provocation of symptoms. In the interim, documenting the precise technique of measurement is strongly recommended (*Table 1*). BJHM

Conflict of interest: none.

Table 1. Recommended documentation for postural hypotension measurement

State what positions were used: supine, sitting (with or without legs dangling) or standing
State the time interval(s) at which blood pressures were recorded after assumption of a particular posture
Document simultaneous postural changes in heart rate
Record any symptoms associated with postural change

Based on current available literature

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KEY POINTS

- When recording postural blood pressure measurements it is important to specify the recording technique.
- The presence of symptoms and heart rate changes should be documented.
- The role of sitting measurements must be emphasized in people who are unable to stand.