Mechanisms of initial blood pressure response to postural change


Department of Medicine and Department of Cardiology, Academic Medical Centre, University of Amsterdam, Amsterdam, and Department of Cardiology, University Hospital Utrecht, Utrecht, The Netherlands

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Summary

1. The influence of supine rest on the blood pressure response to standing and 70° head-up tilt was studied in detail for the first 30 s after the change of posture.

2. Following 20 min of supine rest, the active transition to standing was accompanied by an immediate increase in systolic pressure of 29±6 mmHg (mean ± SEM). This was followed by large fluctuations in systolic pressure: to -28 ± 2 mmHg below control after 7 s and to 22 ± 2 mmHg above control after 22 s (17 mmHg in excess of the systolic pressure level after head-up tilt).

3. Following 1 min of supine rest, there was no difference in the immediate increase in systolic pressure. However, the magnitude of the subsequent changes was significantly diminished.

4. With head-up tilt the immediate increase in blood pressure was absent and afterwards small changes were found that were also significantly influenced by the period of prior rest.

5. Taken in conjunction with earlier studies, the following mechanisms are suggested. The immediate blood pressure increase resulted from compression of arteries by the contracting postural muscles. The subsequent blood pressure fall was caused by at least two mechanisms: (a) the fall was predominantly of reflex origin, because the immediate pressure increase stimulated the systemic baroreceptors; (b) supine rest possibly augmented the translocation of blood from the thorax which contributed, approx. 5 s from standing, to the reflex fall of blood pressure.

Key words: arterial pressure, baroreceptor reflexes, catecholamines, head-up tilt, heart rate, posture, rest, standing, supine.

Introduction

When normal subjects stand there are large fluctuations in heart rate and blood pressure lasting for 20-30 s [1]. There is an immediate increase in heart rate which is mediated by withdrawal of vagal tone [2, 3]. This is attributed to a reflex originating in the contracting postural muscles and/or a ‘central command’ mechanism [4, 5]. There follows a further increase and a rapid decrease in heart rate accompanied by opposite changes in arterial pressure, suggesting that the heart rate changes are mediated by the arterial baroreceptor reflexes [1].

In the present study a detailed comparison is made of the changes in arterial pressure upon standing and head-up tilt in relation to the period of prior supine rest, and explanations are advanced to account for the temporal response patterns.

Methods

Subjects

Eight subjects participated in the study after giving informed consent. The investigative procedures were approved by the hospital ethical committee. Three subjects were healthy male volunteers, aged 25, 25 and 33 years. Five subjects, four male and one female, mean age 37 years (28-53 years), were investigated as part of a
larger study in which 24-h continuous intra-arterial ambulatory blood pressure monitoring was used to identify 'cuff-responders', i.e. subjects without any evidence of target organ damage but with a marked difference between casual readings taken in the office and intra-arterial pressure at rest at home. Untreated their office readings were $>160\text{ mmHg}$ systolic/$100\text{ mmHg}$ diastolic. At home, intra-arterial blood pressure at rest was $121\pm6\text{ mmHg}$ (±SD) systolic and $73\pm6\text{ mmHg}$ diastolic.

History, physical examination, routine laboratory tests and excretion urography or renography were all normal. They had no target organ damage (fundus, chest X-ray, serum creatinine, electrocardiogram and echocardiogram). Antihypertensive drug treatment was withdrawn, and 5 weeks afterwards 24-h continuous intra-arterial ambulatory blood pressure registration was performed with the Oxford-Medilog system, which is described fully elsewhere [6].

These subjects responded in a normal way to static and dynamic exercise, mental stress tests and the cold pressor test (G. A. Van Montfrans et al., unpublished work). The only abnormal finding was an exaggerated rise in blood pressure associated with taking a blood pressure reading using the cuff method. As a result, for the purpose of this study these subjects can be considered normal persons.

**Measurements**

In seven subjects the blood pressure measurements were obtained with the Oxford-Medilog system. The strain gauge was carried at heart level. The data from one volunteer were taken from the previous study [1] because supine control periods matched the present protocol.

Instantaneous heart rate was derived from the electrocardiogram and written on a pen recorder as described before [1, 3, 7]. The start of active and passive changes of posture was marked on the Oxford cassette tape and on the pen recorder with the same push-button activated event marker.

The experiments were performed around 11.00 hours, following the 24-h blood pressure monitoring period at home. The subjects abstained from coffee and smoking in the morning before the measurements [8].

**Comparison of standing and 70° head-up tilt**

The changes of posture were accomplished as described previously [1, 3] in about 3.5 s. We used a tilting table with foot support and anti-slip mattress. Subjects were instructed to avoid straining of muscles during the tilt. Although the subjects were not strapped to the tilting table, the tilting manoeuvre was considered a passive change of posture.

**Influence of supine rest**

We compared the circulatory transients evoked by standing after 1 and 20 min of supine rest. The initial responses to 70° head-up tilt after 1 and 10 min of rest were compared in the same manner. One minute supine rest was preceded by several active changes of posture [1]. In this paper we shall consider these conditions as 'no rest' and 'rest', respectively.

**Plasma noradrenaline**

Plasma noradrenaline was determined in four subjects at the end of each brief or long supine resting period according to the method of Endert [9], from venous samples obtained with an indwelling short brachial cannula that had been introduced in the right arm before the experiment. Average plasma concentrations are given for the rest and the no rest condition. In our laboratory, the intra-assay and inter-assay coefficients of variation are 6% and 10% respectively. The detection limit is 32 ng/l [9].

**Data analysis**

Tape recordings were replayed at 25 times the recording speed and the blood pressure signal was displayed on an Elema-Schonander ink-jet recorder running at a paper speed of 50 mm/s, with a sensitivity of 1 mm/5 mmHg (Fig. 1). We measured systolic pressure ($P_s$), diastolic pressure ($P_d$) and heart rate (HR) in chronological order at time $t = -10, -5, 0, t(\text{peak} P_s), 3, t(\text{minimum} P_s), t(\text{maximum} P_s), t(\text{minimum} \text{HR}), t(\text{maximum} \text{HR}),$ and 30 s (Fig. 2a, the minima and maxima are indicated by arrows). Time zero was defined as the start of the active or passive change of posture. The choice of sample points was prompted by the results of the previous study [1]. The times the various minima and maxima occurred were noted to 0.5 s. Both the magnitude and the timing of the various maxima and minima were compared in the rest and the no rest condition. When standing was compared with tilt, the magnitude of the maxima and minima in the response to standing were compared with the values found at the same time after the onset of head-up tilt.

Control values were averaged over 10 s before the change of posture and the results were
Blood pressure response to standing

Results

Systolic pressure ranged from 92 to 141 mmHg, diastolic pressure ranged from 65 to 85 mmHg, and HR ranged from 63 to 80 beats/min after 20 min of supine rest. Control values for standing and tilt were not significantly different. The forced respiratory sinus arrhythmia ranged from 11 to 36 (mean 23) beats/min, which indicates that vagal HR control was normal [10].

Fig. 1 shows an original record of the blood pressure changes at the start of standing. Fig. 2 shows an example of the influence of supine rest on the cardiovascular changes in a young volunteer. The pronounced respiratory sinus arrhythmia present at rest disappeared during the stand up. The results in eight normotensive subjects are summarized in Fig. 3.

Standing after 20 min of rest

On standing blood pressure increased abruptly in all subjects. The increase of systolic pressure reached a maximum of $29 \pm 6$ mmHg (vs control, $P < 0.01$) after 0.5-3 s (mean 1.4 s). After its peak, arterial pressure quickly returned to control. Between 2 and 4 s from standing it continued or resumed its fall and after 6 ± 1 s diastolic pressure reached a minimum (−15 mmHg, $P < 0.05$ vs tilt). The systolic pressure minimum (−28 mmHg) at $t = 7 \pm 1$ s contrasted with the 8 mmHg decrease observed at the same moment after tilt ($P < 0.01$).
FIG. 3. Mean changes (n = 8) ± SEM in heart rate (a), systolic (b) and diastolic arterial pressure (c) induced by active (left panel) and passive changes of posture (right panel), after a brief (○) and after a long period of supine rest (●). The immediate pressure increase upon standing is attributed mainly to mechanical compression of arterial vessels by the muscular effort of standing. The immediate diastolic pressure increase could not be determined reliably in some subjects and has been omitted.

When the HR increase reached its maximum of 33 ± 3 beats/min (vs tilt, \( P < 0.01 \)) at \( t = 11 ± 1 \) s diastolic pressure had recovered to the supine control level but systolic pressure was still below supine control (−6 mmHg). The HR increase reached a minimum after 22 ± 2 s (−6 beats/min) which undershot the response to tilt \( (P < 0.05) \). Systolic pressure showed an overshoot of 17 mmHg with respect to tilt \( (P < 0.01) \) after 22 s. After 30 s there was little or no difference between standing and tilt.

Comparison of 20 and 1 min of supine rest

Supine rest significantly modified the responses to postural change (Figs. 2 and 3).
Standing

There was no significant influence of supine rest on the immediate arterial pressure increase after 1 s, on the primary HR increase at 3 s, or on the minimum diastolic pressure. The fall of systolic pressure, however, was 50% larger after rest compared with no rest \((P < 0.01)\), and the systolic and diastolic pressure minima were slightly delayed. After rest the maximum HR increase exceeded the increase without rest by 24% \((P < 0.05)\) and was delayed by 2 s \((P < 0.05)\).

The recovery of systolic pressure after 7 s was accelerated after rest and was followed by a 17 mmHg overshoot with respect to tilt. No such overshoot was found on standing without rest \((P < 0.05)\). Diastolic pressure recovery, however, seemed to be independent of rest. After 0.5 min, no significant influence of rest was observed on the cardiovascular response to standing or tilt.

Head-up tilt

Without prior rest, systolic and diastolic pressure increased gradually upon head-up tilt. After rest, however, a temporary small arterial pressure decline \((\text{vs control}, P < 0.05)\) was observed, followed by recovery and rise of 5–10 mmHg above control.

Heart rate changes

In the initial 30 s of standing, HR and systolic pressure changed mainly in the opposite direction.

Plasma noradrenaline

At the end of the long supine resting period, plasma noradrenaline ranged from 204 to 514 ng/l. After arising several times, it was elevated by 93 ± 13% \((P < 0.01, \text{Student's } t\text{-test}, n = 4)\) when sampled at the end of the subsequent 1 min resting period. These values are within the normal range in our laboratory [7].

Discussion

The principal finding was that supine rest did not affect the initial blood pressure and HR changes during the stand up, whereas it significantly amplified the fluctuations in the next 30 s.

The present results confirm and extend a preliminary report on blood pressure responses to static muscle contractions and changes of posture [1].

Mechanism of immediate blood pressure increase

The immediate increase in arterial pressure upon standing (Figs. 1–3) may be attributed to several mechanisms: (1) a movement artefact, (2) the immediate HR increase, (3) a reflex effect of (static) exercise, (4) an increase in intrathoracic pressure, (5) compression of arterial vessels, (6) a cardiac output increase.

First, it is unlikely that it was a movement artefact, because it was absent at the beginning of head-up tilt and all measures designed to limit the arm and cannula movements failed to reduce it. Second, it cannot be caused by the HR increase because handgrip evoked the same abrupt HR increase without a concomitant blood pressure increase [1]. Third, the pressure increase cannot be of reflex origin [4, 5], because the 29 mmHg peak was found after about 1 s, whereas reflex vasoconstriction involves a 2–3 s delay and a long time constant [11, 12], as illustrated by the sluggish arterial pressure increase induced by handgrip [1]. Fourth, we have no experimental data on intrathoracic pressure, but when Valsalva straining was intentionally avoided during contraction of abdominal and leg muscles, a similar arterial pressure increase was observed [1]. That pressure increase has to be ascribed to, fifth, the mechanical increase of total peripheral resistance caused by compression of arterial vessels in the abdomen and the legs by contracting postural muscles. We attribute the arterial pressure increase during the stand up to the same cause. Sixth, it is to be expected that contraction of postural muscles compressed systemic capacitance vessels as well [13]. It is possible that the immediate blood pressure increase on standing was caused by an increase in cardiac output [13, 14] due to an increased right atrial pressure [13, 15]. However, we consider this mechanism less likely than the previous one because of a too rapid increase and decrease of the systemic pressure (Figs. 1 and 3). Loeppky et al. [14] reported a 1–2 s delay between the start of exercise and an increase in cardiac output measured by pulsed Doppler echocardiography.

Mechanism of blood pressure decrease

Two mechanisms probably contributed to the blood pressure decrease that began after about 3 s. (1) Passive translocation of blood from the thorax resulted in the temporary predominance of a cardiac output decrease over a reflex peripheral resistance increase [16, 17], as illustrated by the temporary fall of arterial pressure upon head-up tilt (Fig. 3). (2) Stimulation of systemic baro-
Influence of supine rest

Fig. 3 suggests that the period of prior rest affected the first but not the second component of the arterial pressure decrease. Fig. 3 indicates that, following rest, about one-third of the systolic pressure decrease after standing was related to the change in posture itself, and two-thirds to the reflex effect of the immediate pressure increase. When the change of posture was not preceded by rest, the first, passive component of the blood pressure decrease seems to have been largely absent, whereas the second, reflex component was unaffected. The latter observation is consistent with the finding that rest did not modify the immediate pressure increase.

The mechanism of the influence of rest on the passive component of the blood pressure decrease in unclear. It needs to be established whether the translocation of blood from the thorax was reduced in the no rest condition. If that can be demonstrated, the elevated plasma noradrenaline level found in that condition may have been related to reduced capacity of the systemic capacitance vessels [21].

Mechanism of blood pressure recovery

The recovery of diastolic pressure after 6 s is consistent with reflex vasoconstriction caused by (1) decreased stimulation of systemic baroreceptors by the arterial pressure fall in the preceding 3 s (Fig. 3) [12], and (2) decreased stimulation of cardiopulmonary receptors due to diminished right ventricular filling pressure once the upright posture has been attained [16–19]. The mechanism of the influence of rest on the recovery of systolic pressure remains to be determined.

Relation between heart and blood pressure

The present findings confirm and extend the results reported previously [1]. The timing and magnitude of the secondary [1] HR increase and the subsequent minimum corresponded through the arterial baroreceptor reflex largely to the time course of the opposite systolic blood pressure changes, because the latency of the vagally mediated reflex HR changes [2, 3] is about 0.5 s [12, 22]. The simultaneous increase of HR and blood pressure during the stand up was discussed earlier [1].

Clinical implications

In testing for autonomic neuropathy [3, 10, 23], the HR response to standing has limited diagnostic value in the individual patient because the mechanism of the response is complex [1, 3].

Supine rest and an abrupt stand up increase the likelihood of dizziness approx. 5 s from standing. This dizziness will last only a few seconds. The possible import of the present observations with respect to recently reported accidents in the elderly after arising suddenly [24] requires further investigation.

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