Circulatory adaptation to orthostatic stress in healthy 10–14-year-old children investigated in a general practice

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SUMMARY

1. The magnitude and time course of circulatory adaptation to active standing were investigated in healthy premenarchic girls and boys (n = 24; 10–14 years old) by non-invasive measurement of heart rate and continuous finger blood pressure (Finapres).

2. Four subjects (two girls, two boys) showed presyncopal symptoms after 4–9 min of free standing.

3. In the 20 non-fainting subjects, changes in blood pressure and heart rate upon standing did not differ between girls (n = 10) and boys (n = 10). In the initial phase of standing (first 30 s) systolic and diastolic blood pressures dropped by 22±14 (mean±sd) and 16±7 mmHg, respectively, at 8±2 s. Blood pressure subsequently recovered and showed an overshoot in all subjects. The transient drop in blood pressure was accompanied by an increase in heart rate of 40±7 beats/min. These characteristic transient changes were not observed with passive head-up tilt. During the early steady-state phase (2 min), systolic blood pressure was similar to the supine value and diastolic blood pressure rose by 11±5 mmHg. Heart rate increased by 25±11 beats/min. In six of the subjects (three girls, three boys) the increase in heart rate exceeded 30 beats/min (postural tachycardia). Little further changes were observed during prolonged (10 min) standing.

4. Typical findings in the four near-fainting subjects were higher supine heart rates, no blood pressure overshoot in the initial phase (in three out of four subjects), postural tachycardia in the early steady-state phase and progressive decreases in blood pressure and heart rate afterwards.

5. In conclusion, for investigation of orthostatic circulatory control between premenarchic girls and boys: orthostatic tachycardia and fainting appear to be common in both.

Key words: continuous blood pressure determination, heart rate, orthostatic hypotension, postural changes, 10–14-year-old children.

Abbreviations: BP, blood pressure; HR, heart rate; ΔHRmax. and ΔHRmin., initial peak heart rate increase and subsequent relative minimum expressed as differences from control values; I–E difference, inspiratory–expiratory difference.

INTRODUCTION

Complaints of orthostatic dizziness and impending syncope are not uncommon in teenagers and adolescents [1–5]. However, little is known about circulatory orthostatic adaptation in healthy children [3]. In a previous study on orthostatic circulatory control we found that 10–14-year-old boys have a striking increase in heart rate (HR) during the initial phase (first 30 s) of standing [6]. In that study blood pressure (BP) was measured conventionally in the upper arm, with the subjects supine, and after 1 min of standing. Consequently, observations on orthostatic BP behaviour were limited to a single reading in the early steady-state phase (1–2 min of standing) [7]. So far, for obvious reasons, the time course of the initial BP response (first 30 s) [7, 8] has not been studied in detail in this age group.

Recently, continuous non-invasive finger arterial blood pressure monitoring became available using Finapres [9]. Its accuracy during orthostatic stress testing [10], and its application in studies on orthostatic BP control in healthy adult and elderly subjects [11–13], have been described.

In the present study observations were made, by using Finapres, in healthy 10–14-year-old children on the adaptation of the circulation after a change of posture in
order to obtain insight into the time course and magnitude of BP and HR responses from the first second of standing until 10 min of standing. A comparison was made between the short-term effect of free standing and passive head-up tilt on the circulation.

METHODS

Subjects

The setting of the study was one general practice in a small town in the northern part of the Netherlands. Twenty-four healthy 10–14-year-old children (12 girls and 12 boys) were recruited consecutively when they consulted their family doctor (J.H.A.D.) for trivial ailments, such as epidermal warts, cuts and bruises. To avoid the influence of the menstrual cycle on the circulation [14], we admitted premenarchic girls only. Subjects had no evidence of cardiopulmonary disease by history and physical examination, were taking no medication and ate a normal diet without salt restriction. They had no history of postural complaints or frequent fainting and regularly took part in sporting events and physical exercise, such as cycling and swimming. None of the subjects smoked. Office blood pressures were normal for age [15]; glucosuria was excluded.

The study was approved by the Ethical Committee of the Academic Medical Centre, University of Amsterdam. Informed consent was obtained from the subjects themselves and their parents.

The experiments took place in the morning, at least 2 h after a normal breakfast. Coffee was not allowed from the previous evening onwards. The temperature of the examination room was maintained at 22–24°C.

Measurements

Continuous finger arterial pressure (BP) was recorded by means of a TNO model 5 Finapres. This is an updated version of the commercially available Ohmeda 2300, Finapres, NIBP Monitor. During laboratory tests Finapres follows intra-arterial pressure reliably [9, 10]. Subjects were instructed to keep the measuring finger cuff at heart level to avoid the interference of hydrostatic pressure. The investigators regularly checked the position of the cuffed finger and when movements were noted the manoeuvre was repeated. From each subject a single-lead ECG was recorded and input to a cardiotachometer to obtain instantaneous HR responses. BP, HR and an event marker was recorded on strip chart for direct inspection (7402-A, Hewlett-Packard, Los Angeles, CA, U.S.A.). For off-line analysis, the ECG, the BP signal and the event marker were recorded on tape using a cassette data recorder (TEAC R-61; TEAC Corp, Tokyo, Japan).

Manoeuvres

Before instrumentation of the subject, a 10–15 min period was used to practise the manoeuvres. The subjects were instructed in respect of the time needed to stand up, as well as the position of the hand during standing up and tilt. Experiments were performed in a fixed sequence: forced breathing, passive head-up tilt for 2 min and active standing for 10 min. Subjects were asked to report sensations of dizziness after assuming the upright position.

Forced breathing. Vagal HR control was assessed by measuring its variation during forced breathing. The subjects performed six consecutive maximal respirations at a rate of six breaths/min after 5 min of supine rest (forced respiratory sinus arrhythmia) [13].

Head-up tilt. To evaluate the differences in the BP and HR responses between active and passive changes of posture [7], the effects of 70° head-up tilt were examined. Passive head-up tilt was performed in about 3 s after 5 min of supine rest, using a tilt table with foot support. Subjects remained in the upright tilted position for 2 min.

Standing-up. Subjects were trained to stand up from the supine position in 2–3 s, starting on a verbal command. The standing-up manoeuvre was performed after 5 min of supine rest and subjects remained standing for 10 min [7, 13].

Analysis of data

Upon playback, a 30 s control period before and a period of up to 10 min after the onset of the postural changes were selected for further analysis. Finger arterial BP and the event-marker signals were analogue-to-digital converted at a sampling rate of 200 Hz and input to a PDP-11/44 computer system [13].

A signal analysis program was used to determine beat-to-beat systolic and diastolic BP and the instantaneous HR. Individual responses were averaged to obtain group responses. The onset of standing up and passive head-up tilt was defined as \( t = 0 \).

Standing up. Characteristic of the short-term circulatory adaptation to standing is an initial phase (first 30 s) with a transient marked drop in BP, which is followed by a rapid recovery and an overshoot. Stabilization of BP and HR is usually obtained within 1 min (Fig. 1). As in previous studies, we used BP and HR values at 1 and 2 min to define this early steady-state phase [7, 13]. The circulatory effects of prolonged standing were evaluated by measuring BP and HR after 5 and 10 min of standing [7, 13]. The BP and HR responses in the three time periods were quantified as described previously [7, 13].

1) Initial response (first 30 s, Fig. 1). Average BP and HR values of a 30 s period before standing up were taken as control (A). We identified the immediate systolic and diastolic BP rise upon standing (B), the systolic and diastolic BP drop (C) and subsequent overshoot (D), and the values at 30 s (E). The magnitude of the initial peak HR increase and the subsequent relative minimum were expressed as the differences from control values (\( \Delta HR_{max.} \) and \( \Delta HR_{min.} \)) [7].

2) Early steady-state response (1–2 min of standing). The average values for systolic and diastolic BP and HR during a 10 s period centred at 1 and 2 min of standing were calculated (Fig. 1) [13].
Circulatory response upon standing in 10–14-year-old children

Standing up (a)ABCD, M 2001 ' ' ' ' ' ' ' ' '

Fig. 1. Example of BP and HR responses upon prolonged standing in a 12-year-old boy (a) and in a 11-year-old boy with postural tachycardia (b). Instants A–E represent characteristic sample points: A, control; B, immediate BP rise; C, BP dip; D, BP overshoot; E, 30 s values.

3) Prolonged standing (5–10 min of standing). BP and HR values were averaged during a 10 s period centred at 5 and 10 min of standing [13].

Abnormal BP and HR responses in the early steady-state phase and during prolonged standing. Orthostatic hypotension in the early steady-state phase and during prolonged standing was defined as a drop in systolic BP of more than 20 mmHg and/or in diastolic BP of more than 5 mmHg [7]; postural tachycardia was defined as an increase in HR of > 30 beats/min (HR values above the 97.5th percentile for age) (Fig. 1) [7].

Passive head-up tilt. The changes in BP and HR in the initial and early steady-state phases were compared with the changes upon free standing.

Forced breathing. The so-called inspiratory–expiratory difference (I–E difference) in HR was computed as the mean difference between the maximal and minimal HR during each of six consecutive respiratory cycles of forced inspiration and expiration and was expressed in beats/min.

Statistics

Significant changes in BP and HR from control at instants B–E (Fig. 1) and at 1, 2, 5 and 10 min after the onset of standing were detected by means of a one-way analysis of variance. If significant differences were present they were identified by using the paired Student’s t-test. To adjust for multiple comparisons, a Bonferroni correction was performed [16, 16a].

Differences in the circulatory responses upon standing between girls and boys were identified by means of an unpaired Student’s t-test. Differences in circulatory responses between the 20 non-fainting and the four near-fainting children were assessed by means of the χ² test. Results are given as means ± SD. Actual P values are shown.

RESULTS

Four of the 24 subjects showed pre-syncopal signs during our observations; their data are discussed separately. Anthropometric data for all subjects are given in Table 1.

Non-fainting subjects (n = 20)

I–E differences were similar in girls and boys (Table 1). Supine BP did not differ between boys and girls. After 10 min of standing BP was lower in the girls (Fig. 2, Table 2). Supine HR was higher in the girls (P < 0.05). Standing evoked comparable changes in BP and HR in girls and boys (Fig. 2, Table 2); therefore the pooled results of the changes in BP and HR in the initial phase and the early steady-state phase are presented (Table 3).

Initial response (first 30 s). The drop in BP occurred at 8 ± 2 s and amounted on average to 22 mmHg in systolic BP and 16 mmHg in diastolic BP. A drop in systolic BP of 40 mmHg or more was present in one girl and one boy, and a drop in diastolic BP of more than 25 mmHg in one girl and one boy. The maximal BP over-
Shoot was reached at 17 ± 3 s. An overshoot of systolic BP was present in 18/20 subjects and an overshoot in diastolic BP was present in all subjects. At 30 s, systolic BP was at the control level and diastolic BP had risen beyond that level. The BP transients were accompanied by an instantaneous rise in HR, reaching a maximum at 11 ± 1 s (ΔHRmax. 40 ± 7 beats/min), followed by a rapid decrease (ΔHRmin. 12 ± 11 beats/min). At 30 s the average increase in HR was 18 ± 12 beats/min (Table 3). None of the subjects reported dizziness upon standing.

**Early steady-state response (1–2 min of standing).**

After 1 and 2 min of standing systolic BP did not differ from control, and diastolic BP remained elevated by about 10 mmHg (Table 3). A drop of more than 20 mmHg in systolic BP and/or 5 mmHg in diastolic BP was not observed. HR increased by 24 ± 14 beats/min after 1 min and by 25 ± 11 beats/min after 2 min. In 6/20 subjects (three girls, three boys) postural tachycardia (HR increase > 30 beats/min) was found; HR increased on average by 36 beats/min (range 32–44 beats/min). In the subjects with postural tachycardia systolic BP was

**Table 1. Clinical characteristics of the subjects participating in the study**

Values are means ± SD. Forced respiratory sinus arrhythmia is given as the I–E difference.

<table>
<thead>
<tr>
<th></th>
<th>Girls (n = 10)</th>
<th>Boys (n = 10)</th>
<th>Near-fainting subjects (n = 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>12 ± 1</td>
<td>12 ± 1</td>
<td>11 ± 1</td>
</tr>
<tr>
<td>Height</td>
<td>158 ± 8</td>
<td>160 ± 11</td>
<td>158 ± 14</td>
</tr>
<tr>
<td>Weight</td>
<td>43 ± 7</td>
<td>45 ± 9</td>
<td>44 ± 11</td>
</tr>
<tr>
<td>I–E difference (beats/min)</td>
<td>30 ± 9</td>
<td>29 ± 12</td>
<td>33 ± 11</td>
</tr>
</tbody>
</table>

**Table 2. Systolic BP, diastolic BP and HR during prolonged standing in 20 non-fainting subjects**

Values are means ± SD. Statistical significance: *P < 0.05, **P < 0.01 for boys compared with girls.

<table>
<thead>
<tr>
<th></th>
<th>Girls (n = 10)</th>
<th>Boys (n = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>114 ± 10</td>
<td>118 ± 6</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>57 ± 6</td>
<td>62 ± 5</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>68 ± 7</td>
<td>59 ± 9</td>
</tr>
<tr>
<td>5 min of standing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>117 ± 13</td>
<td>120 ± 5</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>69 ± 9</td>
<td>74 ± 4</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>90 ± 8</td>
<td>84 ± 16</td>
</tr>
<tr>
<td>10 min of standing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>110 ± 11</td>
<td>121 ± 5**</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>66 ± 6</td>
<td>74 ± 5**</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>94 ± 9</td>
<td>86 ± 17</td>
</tr>
</tbody>
</table>

Fig. 2. Average systolic BP, diastolic BP and HR responses upon standing (a) and passive head-up tilt (b) in the 20 non-fainting subjects. The thin traces represent group averages for 10 teenage girls, the bold traces those for 10 teenage boys.
Table 3. Systolic BP, diastolic BP and HR changes from baseline during the initial phase and early steady-state phase in 20 non-fainting subjects

The instants A-E represent characteristic points in the initial response. Values are means ± SD. Statistical significance (paired Student's t-test); *P < 0.05, **P < 0.01, ***P < 0.001 compared with baseline. According to the Bonferroni correction for six comparisons, a P value of < 0.01 is considered to represent significance.

<table>
<thead>
<tr>
<th>Initial response</th>
<th>Change from baseline</th>
<th>HR (beats/min)</th>
<th>HRmax. HR (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Systolic BP (mmHg)</td>
<td>116 ± 8</td>
<td>59 ± 6</td>
<td>63 ± 9</td>
</tr>
<tr>
<td>B Systolic BP (mmHg)</td>
<td>9 ± 9***</td>
<td>11 ± 6***</td>
<td>14 ± 3***</td>
</tr>
<tr>
<td>C Systolic BP (mmHg)</td>
<td>-22 ± 14***</td>
<td>-16 ± 3***</td>
<td>40 ± 7***</td>
</tr>
<tr>
<td>D Systolic BP (mmHg)</td>
<td>11 ± 3***</td>
<td>12 ± 6***</td>
<td>24 ± 8***</td>
</tr>
<tr>
<td>E Systolic BP (mmHg)</td>
<td>4 ± 11</td>
<td>9 ± 5***</td>
<td>18 ± 12***</td>
</tr>
<tr>
<td>Early steady-state response</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>60 s Diastolic BP (mmHg)</td>
<td>2 ± 7</td>
<td>10 ± 7***</td>
<td>24 ± 14***</td>
</tr>
<tr>
<td>120 s Diastolic BP (mmHg)</td>
<td>3 ± 7*</td>
<td>11 ± 5***</td>
<td>25 ± 11***</td>
</tr>
</tbody>
</table>

unchanged after 2 min of standing (-2 mmHg, range -12 to 8 mmHg) and diastolic BP had increased (9 mmHg, range 4-12 mmHg).

Prolonged standing (5-10 min of standing). In the boys BP remained at the level reached after 2 min throughout 10 min of standing, whereas in the girls BP decreased slightly, but not significantly, from 5 min onwards (Table 2). A drop of more than 20 mmHg in systolic BP and/or 5 mmHg in diastolic BP was not observed in any of the 20 non-fainting subjects. In the six subjects with postural tachycardia, after 10 min of standing HR had further increased by 44 beats/min on average (range 36-52 beats/min). Compared with control, systolic BP was still unchanged (-3 mmHg, range -19 to 8 mmHg) and diastolic BP remained increased (9 mmHg, range -3 to 17 mmHg). A typical example of pronounced postural tachycardia is shown in Fig. 1(b).

Comparison of circulatory responses between standing and passive head-up tilt. No differences were found between girls and boys (Fig. 2). The marked initial BP drop and large initial HR increase, as seen upon active standing, were not observed after passive head-up tilt. The circulatory changes upon head-up tilt and free standing in the early steady-state phase did not differ (Fig. 2); systolic BP was at the control level (-3 ±10 mmHg at 1 and 2 min tilt) and diastolic BP had increased by 6±6 mmHg after 1 min and by 8±7 mmHg after 2 min of tilt, HR increased by 21±10 beats/min after 1 min and by 24±11 beats/min after 2 min of tilt (P>0.05 versus standing).

None of the 20 non-fainting subjects reported symptoms of postural dizziness upon passive tilt.

Comparison of circulatory responses between non-fainting (n = 20) and near-fainting (n = 4) subjects

In two girls and two boys, free standing had to be discontinued after 4-9 min because of impending syncope. Subjects returned rapidly to the supine position to prevent actual fainting. The individual BP and HR responses of these four near-fainting subjects are shown in Fig. 3. In the supine position HR was high in the near-fainting subjects (mean 77 beats/min, range 67-82 beats/min). The I-E differences did not differ between non-fainting and near-fainting subjects (Table 1).

During the initial phase in the near-fainting subjects the dip in systolic BP was large (37 mmHg, range 12-56 mmHg) (Fig. 3). After the initial dip, BP increased, but an overshoot in systolic and diastolic BP (D in Fig. 1) was not observed in three out of four of the near-fainting subjects (presence of systolic BP overshoot: 1/4 near-fainting versus 18/20 non-fainting subjects, x² = 8.5, P < 0.01; presence of diastolic BP overshoot, 2/4 near-fainting versus 20/20 non-fainting subjects, x² = 10.9, P < 0.01; Fig. 3). The initial BP transients were not accompanied by symptoms of dizziness.

In three out of four of the near-fainting subjects a postural tachycardia was observed after 2 min of standing (average HR increase 28 beats/min, range 4-42 beats/min). After 2 min of standing systolic pressure had decreased by more than 20 mmHg in three of the four near-fainting subjects (x² = 17.1, P < 0.01). Diastolic BP had decreased by more than 5 mmHg in two of the four near-fainting subjects (x² = 10.9, P < 0.01). The impending syncope was preceded by a progressive decrease in diastolic BP in all subjects and a decrease in HR in two subjects. One of the four subjects reported feeling faint after 4 min of free standing (Fig. 3, upper panel) and returned to the supine position. Since we were not certain about the nature of his complaints the free standing manoeuvre was repeated after 5 min of supine rest. This time a vasovagal reaction was obvious (Fig. 4).

Passive head-up tilt in near-fainting subjects

The four subjects were pre-syncopal signs during prolonged standing showed different responses also during the 2 min of passive head-up tilt. At the end of the 2 min tilt, systolic BP had dropped on average by 24 mmHg (range -50 to -12 mmHg) and diastolic BP had dropped on average by 7 mmHg (range -30 to 5 mmHg). In three out of four subjects a postural tachycardia was observed after 2 min of head-up tilt; the average HR increase was 26 beats/min (range -10 to 50 beats/min).
DISCUSSION

When someone stands up, about 10% of the circulating blood shifts from the chest to the lower parts of the body. With different speeds various neurohumoral cardiovascular regulatory systems come into play in order to keep BP constant. In the following, the orthostatic circulatory adaptation in healthy 10-14-year-old children will be discussed paying separate attention to the initial response, the early steady-state response and the response to prolonged standing [7, 8, 13, 17].

Initial response (first 30 s)

The present study confirms and extends earlier data concerning the differences in responses between active standing and passive head-up tilt [6, 7]. Only active standing evokes a marked initial BP drop. The magnitude of the initial drop in BP upon standing of 22 mmHg in systolic BP and 16 mmHg in diastolic BP in this study (Fig. 2, Table 3) is comparable with what has been found earlier in adults [10, 12] and normotensive elderly subjects [13]. We conclude that upon standing an initial drop in systolic BP of as much as 40 mmHg and a drop in diastolic BP of as much as 25 mmHg appear to be normal findings independent of age [10, 12, 13, 18]. Therefore, the findings of the present study do not explain the reported high incidence of slight orthostatic dizziness in the initial phase of standing in young subjects [2, 18, 22].

Early steady-state response and effect of prolonged standing

Little or no change in systolic BP, a rise in diastolic BP of about 10 mmHg in combination with a large HR increase in the early steady-state phase (Fig. 2, Table 3) and minor further changes during prolonged standing (Table 2) are typical of orthostatic circulatory control in childhood and teenagers [3, 6, 19].

Castro et al. [3] found an average HR increase of 23 beats/min after 2 min of standing in 31 healthy children aged 10–12 years, and Palmer et al. [20] reported an average HR increase of 31 beats/min after 5 min of standing in seven subjects aged 10–20 years. Thus the postural HR increase of more than 30 beats/min that we found in six non-fainting subjects in the present study is not an unusual finding. Our previously calculated reference values for postural tachycardia in young subjects [7] should be redefined; HR increases of between 30 and 40 beats/min appear to be a normal finding in this age group [3, 20]. Postural tachycardia in childhood in
Standing up

![Graph of BP and HR responses upon prolonged standing performed after 5 min of supine rest after a near-faint.](image)

**Fig. 4.** BP and HR responses upon prolonged standing performed after 5 min of supine rest after a near-faint (the subject in the upper panel of Fig. 3).

Itself should not be considered a sign of orthostatic circulatory insufficiency, since, even after 10 min of standing, BP did not fall in the six non-fainting subjects with postural tachycardia. Nevertheless, postural tachycardia points to enhanced sympathetic cardiovascular activity as an indication of an increased load upon BP-regulating mechanisms [21, 22].

Clinical textbooks emphasize that girls have less orthostatic tolerance [1, 5]. In contrast, we found, in agreement with previous studies in healthy young adult female and male subjects [4, 8, 19, 23, 24], no significant difference in BP and HR responses induced by orthostatic stress between the sexes (Fig. 2); postural tachycardia and fainting were as frequent in girls as in boys. However, given the small sample sizes of these studies ([4, 19, 23, 24] and this study), in the community small differences in orthostatic tolerance between the sexes in this age group can not be excluded.

**Near-fainting subjects**

Vasovagal fainting has been attributed to two afferent mechanisms: (1) central inhibition of sympathetic outflow triggered by corticohypothalamic stimulation, i.e., simple emotional faint, and (2) reflex inhibition of sympathetic outflow caused by excessive stimulation of ventricular mechanoreceptors due to a powerful contraction around an almost empty heart chamber (a Bezold–Jarisch type reflex) [17]. It is impossible to discriminate between the central and peripheral types of fainting in our study. It is conceivable that interactions between the two types occurred: ventricular mechanoreceptors may have been stimulated by a fall in ventricular pressure due to pooling of blood in the dependent parts of the body after primary vasodilatation initiated by emotional stress.

The incidence of fainting in this study (four out of 24 subjects) is in contrast with that in elderly subjects, in whom we found that of 40 subjects none fainted using the same protocol [13]. The high incidence in the young subjects is in line with previous reports [23–25].

Although fainting typically manifests itself during prolonged orthostatic stress [7, 8, 25], we found evidence for a subtle difference already taking place in the initial phase of standing; the near-fainting subjects differed from the non-fainting subjects in that the initial systolic BP overshoot was not observed in three out of four subjects.

As in previous studies [26, 27], supine HR was about 10 beats higher in the near-fainting than in the non-fainting subjects. This might have been due to higher adrenaline levels, which could be instrumental in the cardiovascular response of near-fainting subjects [28]. The circulatory transients during the early steady-state phase (postural tachycardia and a decrease in systolic BP, no increase in diastolic BP) and during prolonged standing (marked drops in systolic BP, diastolic BP and HR) are classically observed before fainting (Figs. 3 and 4) [17, 29]. After 2 min of tilt and standing near-fainting subjects showed comparable responses.

The subject who was rechallenged by orthostatic stress after a questionable near-fainting response (Fig. 3, upper panel) showed a convincing vasovagal response (Fig. 4). This is in line with our previous experiences [17]: circulatory responses remain abnormal for hours after a vasovagal response.

We conclude that an initial transient marked drop in BP is a normal finding in young subjects. In healthy premenarchic girls and young boys no major differences appear to exist in orthostatic adaptation: orthostatic tachycardia and fainting after being upright for some minutes are common in both sexes. Young subjects who seek medical advice for postural dizziness should be evaluated against this background.

**ACKNOWLEDGMENTS**

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**REFERENCES**


