Heart rate control of blood pressure variability in children: a study in subjects with fixed ventricular pacemaker rhythm

Isabelle CONSTANT, Elizabeth VILLAIN*, Dominique LAUDE†, Arlette GIRARD†, Isabelle MURAT and Jean-Luc ELGHOZI†

ABSTRACT

1. To investigate the influence of heart rate variability on blood pressure variability, short-term variability in heart rate and blood pressure was studied in 10 children with fixed ventricular pacemaker rhythm (80 beats/min). Ten healthy children, in sinus rhythm, served as a reference population.

2. Arterial blood pressure and heart rate were measured continuously using a finger arterial device and an ECG respectively. Power spectra for heart rate and blood pressure (systolic and diastolic) were calculated in both supine and orthostatic positions. In addition, acute changes in blood pressure and heart rate during active standing were studied.

3. Healthy children exhibited considerable heart rate variability, which was slightly more pronounced in the supine position, while children with a fixed ventricular rate had no heart rate variability in either position.

4. Despite the differences in heart rate variability, mean systolic blood pressure and its variability profiles were poorly affected by the suppression of heart rate variability. The lack of autonomic control on the sinus node was associated with a reduction in magnitude of the changes in systolic blood pressure variability induced by orthostatic posture.

5. The suppression of heart rate fluctuations induced a noticeable decrease in diastolic blood pressure fluctuations, which was most conspicuous in the children with fixed cardiac rhythm when in the supine position. This may be explained by the lack of diastolic blood pressure fluctuations, physiologically due to heart rate fluctuations through the run-off effect: the longer the cardiac cycle, the greater the diastolic pressure decay. These results may challenge the classical theory of baroreflex-mediated diastolic blood pressure control described in adult patients.

6. During active standing, the early drop in systolic blood pressure was greater in subjects with fixed ventricular rhythm. A rise in heart rate of 36 beats/min was observed in the healthy subjects in response to active standing.

7. We conclude that in normal children, heart rate fluctuations increase the blood pressure variability rather than buffering it. However, during acute orthostatic stress, the abrupt baroreflex-mediated heart rate rise may partly compensate for the reduction in blood pressure.

Key words: autonomic nervous system, blood pressure, children, heart rate, pacemaker, respiratory sinus arrhythmia.
Abbreviations: BP, blood pressure; DBP, diastolic blood pressure; HF, high frequency; HR, heart rate; MF, mid frequency; PM, pacemaker; SBP, systolic blood pressure.
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INTRODUCTION

The regulation of blood pressure (BP) is complex. Mechanical factors such as respiration synchronous fluctuations in stroke volume, neurohormonal influences such as those of the renin-angiotensin system or sympathetic nervous system and acute stress such as sudden active standing, contribute to beat-to-beat variability of BP in humans. Since heart rate (HR) is a determinant of stroke volume and of cardiac output, it plays a major role in BP regulation. The modulatory influence of HR, driven by the autonomic nervous system, is in turn regulated by the arterial baroreflex [1].

Spectral analysis of continuous measurements of BP and HR provides information about the relationship between HR and BP variabilities. Heart rate variability may interact with BP variability in two ways: on one hand it may have a feedforward effect on BP variability, and the loss of HR variability may lead to a decrease in BP variability. On the other hand, HR variability may have an anti-oscillatory role in BP regulation (feedback effect) through the conventional baroreflex mechanism, and the loss of HR variability may increase BP variability. In addition, it has been suggested that the influence of HR fluctuations on BP variability depends in part on the body’s position, as demonstrated for respiratory sinus arrhythmia and respiratory systolic blood pressure (SBP) oscillations [2].

The most obvious way to elucidate the relationship between BP and HR is to study BP variability profiles when HR does not fluctuate. Withdrawal of HR oscillations can be obtained by pharmacological blockade but this procedure may modify the physiological relationship between HR and BP. This relation may also be analysed in patients with fixed ventricular pacemaker rhythm, but most of these patients suffer from cardiovascular disease that may impair the normal interaction between HR and BP [3]. To avoid this bias, we studied young subjects with normal cardiac performance, in whom a pacemaker was implanted for congenital impairment of atrioventricular conduction. These paced subjects with fixed ventricular rhythm were compared with age-matched healthy subjects, in whom the physiological HR variability is usually very pronounced, in order to evaluate the results of the withdrawal of HR fluctuations on short-term BP regulation.

Systolic and diastolic BP were measured using a non-invasive finger BP device. Heart rate was derived from the R-wave of the ECG. These parameters were subjected to spectral analysis as described previously [4]. The study was performed in both the supine and orthostatic positions to characterize the sympathetic and vagal contributions to the relationship between BP and HR. In addition, changes in BP and HR during standing were studied in order to evaluate the results of the lack of baroreflex-mediated HR response during acute physiological stress.

METHODS

Study population

Results from 10 children with an implanted bipolar DDD pacemaker (age 8.7 ± 2.2 (6–13) years, weight 27.6 ± 10.8 kg) and 10 healthy children with sinus rhythm (age 8.6 ± 2.5 (6–13.5) years, weight 28.8 ± 6.0 kg) form the basis of this report. The indication for pacemaker implantation was isolated congenital complete heart block. All paced children had normal cardiac anatomy and left ventricular function as assessed by echocardiography. None of the patients was taking medication. One hour before recordings, the paced children (PM group) were programmed to the VVI mode at a pacing rate of 80 beats/min.

Study protocol

Children were studied in the morning in a quiet room at 22°C. Electrocardiograph measurements were taken with disposable electrodes attached to the thorax, placed to provide clear R-waves, and connected to a Datex cardicap II monitor (Instrumentation Corp., Helsinski, Finland). Finger arterial pressure was monitored non-invasively by a Finapres device (model 2300; Ohmeda, Trappes, France). In this study, all children had a finger circumference of between 42 and 60 mm. To ensure an optimal Finapres BP measurement we used appropriate cuff sizes (S or M) according to the manufacturer’s instructions. The cuff was fitted to the third finger of the right hand, which was passively maintained at heart level during the manoeuvres. Three recordings were obtained: the first during 5 min in the supine position after a 10-min rest, the second during the acute change from the supine to standing position, and the third, of 5 min duration, when the subjects were in orthostatic posture, 3 min after active standing.

Breathing was quantified by respiratory inductance plethysmography with a Respitrace device (Ardley, New York, U.S.A.). During recordings obtained in the supine and standing positions, children breathed spontaneously during the first minute and were then asked to control their breathing frequency using an auditory signal from an electronic metronome. The controlled respiratory rate was determined from the spontaneous breathing rate of each child during the first minute of recording. All HR and BP variability studies were performed during controlled breathing.

The investigation conformed to the principles outlined in the Declaration of Helsinki. The nature of the research was explained to the parents and informed consent was obtained.
Data processing and spectral analysis

The details of data sampling and analysis have already been described [4]. The analogue output of the Datex monitor and of the Finapres device was connected to an analog-to-digital converter to permit data acquisition, storage and analysis using a microcomputer. The BP and ECG signals were digitized (500 Hz) and processed by an algorithm based on feature extraction to detect and measure the characteristics of a BP cycle and an R-wave (Anapres 3.0, Notocord Systems, Croissy-Seine, France). Systolic and diastolic blood pressure (DBP) were extracted from the BP signal, and HR was calculated as 60000 divided by the heart period in milliseconds, measured from the corresponding R-wave and the preceding one. A sampling rate of 10 Hz was chosen without interpolation, i.e. SBP, DBP and HR values were replicated every 0.1 s until a new BP cycle or R-wave occurred within a 0.1 s window.

The evenly spaced sampling allowed direct spectral analysis using a fast-Fourier transformed (FFT) algorithm on a 2048-point stationary time series. This corresponded to a period of 3 min 25 s at our sampling rate. Thus, each spectral band corresponded to a period of 3 min 25 s at our sampling rate. This time period was used for spectral analysis.

The power of the HR or BP (power) was used for calculations and statistical analysis. The evenly spaced sampling allowed direct spectral analysis using a fast-Fourier transformed (FFT) algorithm on a 2048-point stationary time series. This corresponded to a period of 3 min 25 s at our sampling rate. Thus, each spectral band corresponded to a period of 3 min 25 s at our sampling rate.

Statistical analysis
Data are expressed as means ± S.E.M. Significant differences between patient groups and postures were determined by two-way analysis of variance for repeated measures. A logarithmic transformation of the data was performed before the comparisons when the variance ratios of the different groups were significant. When the F-value of the analysis of variance was significant, differences between groups and postures were tested using the modified t-statistics based on the Bonferroni method. Differences were considered to be statistically significant when the P value was less than 0.05.

RESULTS

Effect of suppression of HR variability in physiological stationary conditions (supine and standing)

Respiration
Breathing frequency was effectively controlled in all subjects, with a similar spontaneous respiratory rate in

**Table I** Average SBP, DBP and HR values in the supine and standing positions obtained in paced children (group PM) and healthy controls (group C)

This table includes the standard deviations of SBP, DBP and HR time series. Data are means ± S.E.M. Statistical significance: *P < 0.05, **P < 0.01, ***P < 0.001, group PM versus group C; †P < 0.05, ††P < 0.01, †††P < 0.001, standing versus supine.

<table>
<thead>
<tr>
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<th>SBP (mmHg)</th>
<th>S.D. SBP (mmHg)</th>
<th>DBP (mmHg)</th>
<th>S.D. DBP (mmHg)</th>
<th>HR (beats/min)</th>
<th>S.D. HR (beats/min)</th>
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<td>Supine</td>
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<tr>
<td>Group PM</td>
<td>84.3 ± 3.2</td>
<td>4.6 ± 0.4</td>
<td>43.9 ± 3.3</td>
<td>2.2 ± 0.2</td>
<td>80.2 ± 0.0</td>
<td>0.1 ± 0.0***</td>
</tr>
<tr>
<td>Group C</td>
<td>76.2 ± 4.5</td>
<td>4.5 ± 0.4</td>
<td>38.8 ± 3.1</td>
<td>3.0 ± 0.2</td>
<td>84.5 ± 4.2</td>
<td>7.4 ± 0.5</td>
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<tr>
<td>Standing</td>
<td></td>
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<tr>
<td>Group PM</td>
<td>94.8 ± 2.9</td>
<td>4.6 ± 0.4</td>
<td>54.5 ± 2.7</td>
<td>2.7 ± 0.1</td>
<td>80.3 ± 0.1***</td>
<td>0.3 ± 0.1***</td>
</tr>
<tr>
<td>Group C</td>
<td>99.8 ± 6.2††</td>
<td>5.7 ± 0.6††</td>
<td>62.6 ± 4.3††</td>
<td>3.0 ± 0.2</td>
<td>102.9 ± 5.9††</td>
<td>6.4 ± 0.4††</td>
</tr>
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</table>
the two groups. Mean values, in supine and standing position respectively, were 21.5 ± 0.9 and 21.7 ± 1.4 breaths/min in the PM group and 19.2 ± 1 and 19.4 ± 2 breaths/min in the healthy children.

Heart rate
In the supine position, the fixed HR of the PM group was similar to that observed in the control group. Standing position was associated with an increase in the average HR in the control group (18.4 ± 2.1 beats/min). In the paced individuals HR was unresponsive to changes in posture (Table 1).

Healthy children exhibited a high degree of HR variability, as estimated by the S.D. of the distribution, ranging between 4 and 10 beats/min. Frequency domain analysis of this variability revealed classical components, mainly assessed by the MF peak and the respiratory (HF) peak of the power spectra (Figure 1 and Table 2). Overall variability and HF peak were decreased in the upright position.

As expected, in children with cardiac pacing, beat-to-beat variability was markedly reduced and power spectra were dramatically flat (Figure 1, Table 2).

Blood pressure
The fixed HR did not affect average SBP and DBP values (Table 1). In all children, standing position was associated with higher levels of SBP and DBP compared with the supine position. This postural effect was less pronounced in the PM group for both SBP (+10.5 ± 3.9 mmHg versus +23.6 ± 2.9 mmHg, P < 0.01) and DBP (+10.6 ± 4.5 mmHg versus +23.8 ± 1.6 mmHg, P < 0.01). Despite the lack of HR fluctuations in children with a fixed ventricular pacemaker rhythm, overall variability and power spectra of SBP were similar in the two groups (Table 2 and Figure 1) in the supine position. Standing position was associated with an increase in MF and HF components in the control group, while in the PM group this postural effect was not significant.

In contrast, the DBP of the paced children displayed a reduction in overall variability in the supine position. This was associated with a decrease in the MF and HF components of the power spectra when compared with that of the control group. In response to upright posture only the HF component was reduced (Figure 1 and Table 2).

Effect of suppression of HR variability during acute physiological stress (active standing)
These results are summarized in Table 3, and in Figure 2.

Heart rate
The programmed HR of the PM group and the average resting HR of the control group were comparable. No change in HR was detected in children with fixed ventricular rhythm during active standing up. The characteristic HR acceleration upon standing was observed in control subjects with an average maximum rise of 36.0 ± 3.2 beats/min, followed by a deceleration leaving the HR 15 beats/min above the baseline 1 min after active standing up. The time interval calculated from the initial SBP rise to the maximal HR increase in control subjects was 12.8 ± 0.9 s.

Blood pressure
Resting supine BP was similar in the two groups examined. Both SBP and DBP increased in all children during the first few seconds after the transition from the supine to the standing position. The mean initial increase in BP did not differ between groups (14.2 ± 2.1/10.0 ± 2.4 mmHg in the PM group and 14.0 ± 4.2/10.0 ± 3.1 mmHg in the control group). The subsequent
reduction in SBP was significantly greater in the PM group (22.9 ± 4.0 mmHg) than in the control group (9.5 ± 3.1 mmHg). There was a tendency (not statistically significant) for DBP to behave in a similar manner. The overshoot of SBP and DBP was present in all children and did not differ between the two groups. The early steady-state level of BP after 1 and 3 min in the standing position was also similar in the two groups.

DISCUSSION

Young subjects, previously implanted with a cardiac pacemaker for the alleviation of a congenital impairment of atrioventricular conduction but with no other clinical indications, provide a clinical model whereby the relationship between BP and HR can be assessed. In this study we provided evidence documenting: (i) the absence
of a pronounced effect of the lack of HR fluctuations on SBP variability in steady posture, except for a reduction in the magnitude of changes in SBP variability induced by standing posture; (ii) a noticeable reduction of DBP variability in cardiac-paced subjects, indicating that in healthy children changes in HR were the predominant modulator of DBP; and (iii) a larger drop in SBP during standing up in children deprived of baroreflex-mediated HR response.

The original feature of our population of healthy children lies in the considerable HR variability, which is spontaneously observed and more pronounced than that observed in adults. It should be underlined that the respiratory contribution to HR variability in healthy children was comparable to the MF contribution in supine and standing position, in contrast to the findings described in adults [7]. These data reflect a high vagal modulation of HR in children, whatever the posture.

Reliability of continuous BP measurements
For continuous BP measurement we used the non-invasive Finapres 2300 device. In adults, the accuracy of the Finapres has been widely evaluated in steady-state conditions and during active standing up using intraarterial pressure as the reference [6,8,9]. Triedman and Saul [10] compared intra-arterial and continuous non-invasive BP measurements in paediatric patients in intensive care. We recently compared finger and intraarterial BP measurements in children during anaesthesia and during the recovery period [11]. Our results, in agreement with those obtained by Triedman and Saul [10], demonstrated reliable estimation of BP variability profiles using the Finapres device, with a low inpatient standard deviation of bias.

Effect of lack of HR variability in supine and standing positions
The main components of SBP variability obtained in physiological stationary conditions were similar in children with fixed ventricular rhythm and in healthy children, despite the suppression of short-term HR fluctuations in the former group. However, withdrawal of the autonomic control on the sinus node was associated with a blunted response to orthostatic posture.

The extent to which BP variability is linked to HR variability is debatable. Heart rate oscillations mediated by the arterial baroreflex may buffer BP variations [1,12] whereas HR changes may induce variations in cardiac output and BP. Cardiac pacing is associated with a reduction in respiratory BP fluctuations in both dogs [13] and in healthy human subjects [2], whereas in exercising subjects atropine produces an increase in SBP variability [1]. In contrast, after pharmacological intervention in humans, Triedman and Saul [14] demonstrated that changes in SBP were insensitive to autonomic cardiac blockade and were therefore probably mediated by the mechanical effects of intrathoracic pressure. Our results are in accordance with the latter study.

The MF SBP oscillations are dependent on an intact autonomic nervous system and may stem from a central oscillator of sympathetic tone. High-frequency changes in SBP proceed from changes in cardiac output, probably via respiratory-induced changes in HR and ventricular filling [15,16]. In the present study, the healthy children showed the classical increase in MF and HF components of SBP power spectra when shifting from a supine to a standing position [17,18]. In contrast, the paced children presented a dampened increase in the MF and HF SBP components during the same change in posture. This blunted orthostatic response has previously been observed after surgical cardiac denervation in cardiac transplant patients [19], and after pharmacological blockade in healthy subjects in whom SBP variance was decreased in the standing position [16]. Furthermore, these findings agree with the blunted reflex increase in the MF component of BP variability observed during nitroglycerine infusion in dogs with bilateral stellectomy [18,20]. Together these results suggest that fluctuations of HR may increase fluctuations of SBP rather than reduce them. Our results confirm a noticeable effect of posture on interactions between HR, respiration and BP [2,15].

<table>
<thead>
<tr>
<th>SBP (mmHg)</th>
<th>Control</th>
<th>Minimum SBP</th>
<th>HR rise</th>
<th>Overshoot</th>
<th>Steady state, 1 min</th>
<th>Steady state, 3 min</th>
</tr>
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<tbody>
<tr>
<td>Group PM</td>
<td>82.9 ± 1.9</td>
<td>60.0 ± 3.2</td>
<td>77.9 ± 3.3</td>
<td>96.7 ± 3.9</td>
<td>95.1 ± 4.4</td>
<td>94.7 ± 5.1</td>
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<tr>
<td>Group C</td>
<td>81.8 ± 4.4</td>
<td>72.3 ± 4.8</td>
<td>88.8 ± 4.8</td>
<td>102.7 ± 3.2</td>
<td>97.3 ± 4.2</td>
<td>99.6 ± 4.8</td>
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<td>HR (beats/min)</td>
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<tr>
<td>Group PM</td>
<td>80.2 ± 0.0</td>
<td>80.2 ± 0.0</td>
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<tr>
<td>Group C</td>
<td>83.9 ± 4.4</td>
<td>109.9 ± 4.2</td>
<td>120.6 ± 4.8</td>
<td>97.9 ± 3.9</td>
<td>98 ± 5.4</td>
<td>102.2 ± 6</td>
</tr>
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</table>

Table 3 Effects of active standing on SBP and HR in paced children (group PM) and in healthy controls (group C)
Overshoot of SBP was taken 20 s after the initial rise of SBP. Data are means ± S.E.M. Statistical significance: *P < 0.05, ***P < 0.001, group PM versus group C.
<table>
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<tr>
<th>Systolic and diastolic arterial pressure (mmHg)</th>
<th>Heart rate (bpm)</th>
<th>Respiration (arbitrary units)</th>
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<tr>
<td>100</td>
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<td>240</td>
<td>220</td>
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<tr>
<td>260</td>
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Figure 3 Experimental records from one supine control subject (left) and one supine paced subject (right)

The healthy 7-year-old child exhibited DBP fluctuations in which each decrease occurred synchronously with the HR deceleration. In the paced 7-year-old child, the lack of fluctuations of HR was associated with markedly reduced DBP changes. Furthermore, the periodic SBP decreases occur in both cases at the end of inspiration.

The orthostatic accentuation of SBP fluctuations observed in control subjects might be due to marked HR variations resulting in cardiac output and subsequent BP variations. These BP fluctuations may in turn initiate baroreflex-mediated vasomotor oscillations. Moreover, pressure fluctuations may be increased by the baroreflex closed loop pressure-pressure. Changes in ventilation pattern contribute to changes in the magnitude of respiratory SBP fluctuations [21]. In our study, breathing was monitored by respiratory inductance plethysmography, but the signal was not calibrated, thereby preventing us from converting the amplitude of the respiratory curve into volume, as required for calculation of tidal volume. Although the respiratory rate was comparable between the two groups, and within each group in the two positions, we cannot fully eliminate changes in respiratory volume as a mechanism contributing to the differences observed between the two groups. A congenital heart block with normal cardiac function is an uncommon pathology, a relatively low number of subjects was included in our study. Hence the possibility of the existence of a type II error cannot be discounted.

Our results revealed that suppression of HR fluctuations through pacing unexpectedly reduced DBP variability. This interesting finding may be explained by the lack of DBP fluctuations via the run-off effect (i.e. the longer the cardiac cycle, the greater the diastolic pressure decay) when the R–R interval is fixed. Cardiac cycle length directly influences the amount of diastolic decay and thus DBP. Suppression of HR variability would therefore be expected to stabilize DBP. This mechanism is illustrated in Figure 3 by the simultaneous fluctuations of DBP and HR as occur in a healthy child, compared with those in a paced child where the DBP fluctuations were markedly reduced because the R–R interval and diastolic decay remained constant. These results are in accordance with the decrease in magnitude of respiratory modulation of DBP after cardiac autonomic blockade in healthy adults with atropine and propranolol [14]. Our findings contrast with the beat-to-beat model of the cardiovascular system published by de Boer et al. [12]. They postulated that the HR is passively modulated by BP fluctuations, over the baroreflex arc. An increase in SBP due to respiratory effects induces a lengthening of the R–R interval, and hence the diastolic run-off period; thus the diastolic respiratory fluctuations are much less than the systolic respiratory fluctuations. Following this hypothesis, suppression of the R–R interval’s ability to vary should result in an increase in DBP variability. The discrepancy between this model of adult cardiac function and our present findings may result from the ample vagally mediated respiratory sinus arrhythmia observed in children. Indeed, our results suggest that respiratory-induced HR fluctuations may overcome the fast baroreflex-mediated control of R–R interval. This effect could be more evident in the supine position where parasympathetic tone is enhanced. Metronome controlled breathing has been demonstrated to enhance respiratory sinus arrhythmia [18]. However, in our study, the metronome respiratory rate was chosen so that breathing would be close to each subject’s spontaneous respiratory rate and would not therefore influence the magnitude of respiratory sinus arrhythmia to any significant degree [22].

As DBP variability determines to a large extent the sympathetic outflow via the arterial baroreflex mechanism [23], the dampening of DBP fluctuations may affect SBP variability, especially in the upright position where baroreflex mechanisms are activated. The dampening of SBP changes relating to orthostatic posture in the
PM group compared with the healthy group is consistent with this hypothesis.

**Effect of lack of HR variability in acute physiological stress**

During active standing we demonstrated that the fall in SBP was greater in children with a fixed ventricular rate than in healthy children.

The initial circulatory responses upon rising have been well described [24,25]. The mechanism underlying the characteristic initial reduction in BP upon standing is thought to be as a result of muscular vasoconstriction insufficiently matched by an increase in cardiac output [26,27]. The resultant unloading of the carotid and cardiopulmonary mechanoreceptors decreases the inhibitory input to the vasomotor centres with a consequent decrease in vagal tone and increase in sympathetic outflow, resulting in an increase in cardiac chronotropic and inotropic states and vasomotor tone. The initial HR response to standing in young subjects is usually considered as bimodal with an immediate rise resulting from abrupt inhibition of vagal activity followed by a more gradual increase in HR resulting from an increase in sympathetic outflow to the heart combined with vagal inhibition. The HR response seems to be age-related, and young subjects adjust to orthostatic stress mainly by a marked increase in HR [27]. In accordance with observations in paced adults [28] and in children after cardiac transplantation [4], our results demonstrated a greater reduction in SBP in children with a fixed ventricular rate. The lack of a cardiac component of the rapid baroreflex response to standing up may explain this transient hypotension. In our study, the magnitude of the SBP drop observed in the control group was lower than previously described in adults and in children over 10 years of age [29]. This discrepancy may be due to the young age of our subjects given that only one was 10 years old. Similarly, Yamagushi et al. [30] demonstrated a less important BP fall in prepubescent children compared with that of older counterparts during active standing. Few data are available on the influence of age on dilator responsiveness of vascular smooth muscle; in this context, it should be noted that a low sympathetic basal tone was evident in young animals [31] and children [32].

The rapid BP increase after the initial reduction is due to an increase in total peripheral resistance. The incremental rise in BP and the steady-state level of SBP were similar in the two groups. These results demonstrate that the regulation of peripheral vascular resistance can compensate very quickly after the acute initial stress, even in the absence of a HR response.

In conclusion, our study demonstrated that in stationary children with no functional autonomic control of their sinus node, SBP variability was no different to that observed in healthy control subjects. On the other hand, our data revealed that the suppression of HR fluctuations markedly reduced DBP fluctuations, especially in the respiratory frequency range. This result suggests that, in children, the vagally mediated HR fluctuations tend to increase BP variability. However, during acute orthostatic stress, the rapid HR response induced by the baroreflex system may contribute to the attenuation of the BP reduction in healthy children.

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