Multiresolution wavelet analysis of time-dependent physiological responses in syncopal youths

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Nowak JA, Ocon A, Taneja I, Medow MS, Stewart JM. Multiresolution wavelet analysis of time-dependent physiological responses in syncopal youths. Am J Physiol Heart Circ Physiol 296: H171–H179, 2009. First published November 7, 2008; doi:10.1152/ajpheart.00963.2008.—Our prior studies indicated that postural fainting relates to thoracic hypovolemia. A supranormal increase in initial vascular resistance was sustained by increased peripheral resistance until late during head-up tilt (HUT), whereas splanchic resistance, cardiac output, and blood pressure (BP) decreased throughout HUT. Our aim in the present study was to investigate the alterations of baroreflex activity that occur in synchrony with the beat-to-beat time-dependent changes in heart rate (HR), BP, and total peripheral resistance (TPR). We proposed that changes of low-frequency Mayer waves reflect sympathetic baroreflex. We used DWT multiresolution analyses to measure their time dependence. We studied 22 patients, 13 to 21 yr old, 14 who fainted within 10 min of upright tilt (fainters) and 8 healthy control subjects. Multiresolution analysis was obtained of continuous BP, HR, and respirations as a function of time during 70° upright tilt at different scales corresponding to frequency bands. Wavelet power was concentrated in scales corresponding to 0.125 and 0.25 Hz. A major difference from control subjects was observed in fainters at the 0.125 Hz AP scale, which progressively decreased from early HUT. The alpha index at 0.125 Hz was increased in fainters. RR interval 0.25 Hz power decreased in fainters and controls but was markedly increased in fainters with syncope and thereafter corresponding to increased vagal tone compared with control subjects at those times only. The data imply a rapid reduction in time-dependent sympathetic baroreflex activity in fainters but not control subjects during HUT.

baroreflex; syncope; variance

SYNCOPE (FAINTING) IS DEFINED by a sudden transient loss of consciousness and postural tone due to cerebral hypoperfusion (8). Simple postural faint is identified with upright vasovagal syncpe in which vasodilation-induced hypotension and bradycardia are relieved by recumbency (18). Upright orthostatic stress testing and specifically upright tilt table testing have been used to provoke postural fainting episodes. We recently reported on the time course of physiological changes during upright tilt using a system of fiducial points. Fiducial points allowed for the comparison of relevant physiological events across patients with simple postural fainting. The fiducial points were baseline, 1-min after head-up tilt (HUT), the onset of gradual blood pressure (BP) decline, mid decline, late decline just prior to the actual faint, and the faint itself, which was the final fiducial point comprising the rapid fall in BP and heart rate (HR) (Fig. 1). These events were consistently present in all young syncopal patients.

During early tilt we demonstrated a time-dependent increase in HR coinciding with an initially stable arterial pressure (AP) and followed by a slow decrease in AP. The slow decline corresponded to a decrease in cardiac output (CO) and in central hypovolemia caused by failure of splanchnic vasoconstriction and enhanced splanchic blood pooling (34, 36). However, total peripheral resistance (TPR) was initially increased compared with control. Since AP = CO/TPR, the increase in TPR incompletely compensated for the fall in CO, resulting in a slow fall in AP during the mid stages of upright tilt. We infer that increased TPR and HR primarily result from baroreceptor unloading with increased baroreflex-mediated sympathetic activity and decreased reflex baroreflex-mediated cardiovascular activity (vagal withdrawal). During the actual faint these reflex responses are reversed: bradycardia occurs and is attributed to increased vagotonia (37) originating in the preganglionic parasympathetic nerve centers, whereas peripheral vasodilation occurs and is attributed to withdrawal of vascular sympathetic nerve activity from preganglionic baroreflex neurons in the rostral ventral lateral medulla (24).

Our aim in the present study was to investigate the alterations of baroreflex activity that occur in synchrony with the beat-to-beat time-dependent changes in HR, BP, and TPR. For this purpose we wished to employ techniques of HR and BP variability. Often such investigations use Fourier analyses, which are suitable for examining stationary, time-invariant signals. Rhythms in BP and in the RR interval that pertain to sympathetic and parasympathetic control are evident in such analyses. Specifically, low-frequency (LF) oscillations (<0.15 Hz) in BP, first described by Mayer (22), are highly correlated with sympathetic nerve activity in humans (2, 11, 13) and likely reflect sympathetic baroreflex closed-loop negative feedback (11). High-frequency (HF) oscillations (>0.20 Hz), most evident in HR or RR interval time series, are synchronized with the respiratory sinus arrhythmia and represent an index of parasympathetic (vagal) activity (6, 7, 31). Although Fourier techniques are completely adequate for stationary time-invariant signals, they are inherently unsuited for the large time-dependent changes observed during fainting. Time-dependent representations of RR interval and AP variation are therefore required, and discrete wavelet transform (DWT) techniques satisfy this requirement.

Discrete wavelets incorporate the idea of the frequency content of a signal by partitioning it into a finite number of different scales that can be related to a particular frequency (28). Thus, instead of the infinite time scale of a sinusoidal signal, there are confined time and frequency responses of the signal, there are confined time and frequency responses of the
analyzed signal, constrained only by the uncertainty principal. By use of DWT, a discrete sampled biological signal is optimally and completely decomposed into a small group of time-dependent signals. Wavelet coefficients uniquely and completely represent that digital signal yet contain specific scales with particular physiological significance that can be easily examined. Particular contributions of LF and HF power to the energy contained within any signal can therefore be conveniently measured as a function of time.

We measured time-dependent sympathetic activity by studying the AP wavelet coefficients at a scale corresponding to the LF Mayer wave. We examined the time-dependent RR interval wavelet coefficients at a scale corresponding to the respiratory arrhythmia. We used the ratio of LF RR interval power to LF AP power [the alpha index (27, 30)] to describe changes in the cardiovagal baroreflex sensitivity.

A priori, we hypothesized that fainters would have a more marked increase in wavelet LF power compared with control subjects with the onset of upright tilt corresponding to increased peripheral resistance. We proposed that there would be a steady decrease in LF activity during late stages of tilt, culminating in complete absence of LF activity preceding the faint itself. These findings, if present, would reflect an initial increase in sympathetic activity followed by subsequent sympathetic withdrawal (23). We further hypothesized that fainters would have excessive vagal withdrawal initially, signaled by marked reduction in HF RR interval wavelet power after tilt. Thereafter, we expected the HF wavelet power to be reduced compared with controls, since fainters are likely to have increased vagal withdrawal due to increased baroreflex unloading, that is, until fainting supervenes and vagotonia ensues.

METHODS

To test these hypotheses, data were extracted from a well-characterized group of fainters. The study was approved by the Institutional Review Board of the New York Medical College. Informed consent was obtained from all subjects.

Subjects

Fainting subjects comprised 14 patients ages 13–21 (median age 16 yr, 6 male, 8 female) who were referred for three or more episodes of postural fainting and had a vasovagal response within 10 min of HUT at 70°. Control subjects comprised 8 healthy volunteers (median age 17 yr, 4 male, 4 female) who had no history of orthostatic intolerance and did not have a vasovagal response during 10 min of HUT. HUT time was limited to 10 min because prolonged upright tilt can produce fainting in healthy teenagers. All subjects were free of systemic illness, were non-smokers, and were not taking any medication. There were no trained athletes or bedridden subjects. All subjects had a normal electrocardiogram (ECG), echocardiogram, and physical examination.

Protocol

Testing began at 10 AM. Subjects fasted for 4 h prior to testing and refrained from xanthine and caffeine for at least 72 h before testing. No subject smoked. Subjects were familiarized with procedures and then instrumented for ECG, respiratory plethysmography, impedance plethysmography, continuous BP recording, and capnography. For purposes of the present study only data concerning HR (or equivalently RR interval), AP, peripheral resistance, and respirations are reported. After a 30-min supine acclimatization period we assessed HR, BP, and respirations for a baseline period of 5 min.

Subjects were then tilted upright to 70° for a maximum of 10 min. Continuous HR, BP, and respirations were recorded. Subjects were tilted back to supine if fainting ensued. Postural vasovagal fainting was defined as a tilt-induced decrease in mean arterial pressure (MAP) to <60 mmHg or a decrease in systolic BP (SBP) <80 mmHg associated with symptoms of impending loss of consciousness, severe lightheadedness, nausea, or diaphoresis. All of the fainters developed a classic vasovagal faint with bradycardia as well as hypotension while upright. A representative faint is shown in Fig. 1.

Fiducial event markers. In accordance with previous work (36), we chose to examine and display results at fiducial time markers rather than at specific time points. Fiducial markers related to times at which physiological events occurred. Although the actual time between physiological events may vary, fiducial time markers allow for the study of events with corresponding physiology specific for each fainter. For the present study, eight defining events were determined for fainters from the AP trace of each subject. The first fiducial point

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Fig. 1. Arterial pressure (AP; top), RR interval (RR; middle), and total peripheral resistance (TPR; bottom) of a representative fainting patient during head-up tilt (HUT) to 70°. The fiducial points “baseline,” “early” after HUT, “mid” decline, “late” decline just prior to the actual faint, and “faint” are indicated. A time-dependent increase in heart rate (decrease in RR) is coupled with an initial increase in TPR, allowing for an initially stable AP. These events are followed by a decline in TPR and AP until faint.
was baseline pretilt. The second fiducial point was chosen to be 1 min after HUT. Immediately after a tilt-up younger subjects are particularly likely to develop a transient and sometimes large decrease in BP and a reciprocal increase in HR (33). BP and HR recover quickly and within 1 min in healthy volunteers, thus defining the second fiducial point. The third point was defined in fainters by a gradual decline in BP relative to the second point. Subsequently, BP falls off abruptly which defined the fifth point, just prior to faint. Objective criteria for fiducial points 3 at the early BP fall and 5 at the late BP fall were determined by using scale 6 smooth of the multiresolution analysis. Lower frequency oscillations are thereby removed from the computation. The extrema of the second derivative define “corner” or “edge” points that correspond visually to fiducial points 3 and 5. The fourth point was defined as a point midway between points 3 and 5. The sixth point occurred at the end of tilt (at faint for fainters, at 10 min for control subjects). We included seventh and eighth fiducial points representing immediate and later recovery of HR and BP from upright tilt. Early recovery started 5 s after tilt-down. Late recovery occurred when BP had returned to baseline. Thus the eight fiducial event markers are 1) “baseline” (before tilt), 2) “1 min” (1 min after tilt), 3) “early decline” (early fall in BP), 4) “mid decline”, 5) “late decline,” 6) “faint,” 7) “early recovery,” and 8) “late recovery” and are shown for a representative subject in Fig. 1. Healthy control subjects do not have a fall in BP and do not faint. Therefore, we defined equivalent fiducial time points for healthy control subjects by averaging the actual time of fiducial markers 3, 4, and 5 in the fainters. Point 6 was defined by end of HUT. By this means, control subjects and fainting subjects were compared at equivalent average times to allow for a uniform evaluation of cardiorespiratory parameters across groups.

HR and BP monitoring. HR was monitored by a single-lead electrocardiogram. Upper extremity arterial BP was continuously monitored with a Finometer placed on the right index or middle finger which measured finger AP using height correction (Finometer, FMS, Amsterdam). Finometer data were calibrated to a brachial artery oscillographic pressure.

Respiratory volumes. Relative respiratory volumes were obtained by using a respiratory inductance plethysmograph (Respiract, NIMS Scientific) normalizing the respiratory volume on startup to an internal scale. After normalization we calibrated normalized supine Respiract volumes against a pneumotachograph (model RSS100-HR, Hans Rudolph, Kansas City, MO), which also yielded tidal volumes. This enabled the use of the Respiract as a comparative measure of relative changes in tidal volume throughout testing.

Upright tilt-table testing. An electrically driven tilt table (Collin Medical, San Antonio, TX) with footboard was used. After supine measurements the table was tilted up in 6 s to 70° and remained at that angle of tilt for 10 min or until syncope ensued. Syncope was operationally defined as a decrease in SBP to <60 mmHg or a decrease in SBP to <80 mmHg associated with symptoms of impending loss of consciousness, severe light-headedness, nausea, or diaphoresis.

Signal processing and wavelet analysis. HR and RR interval were derived from ECG. ECG, Respiract, and Finometer pressure data were interfaced to a personal computer through an analog-to-digital converter (DI-720 DataQ Ind, Milwaukew, WI). BP, RR interval, and respiratory signals were low-pass filtered below 1.0 Hz. Filtered data were resampled at 2 Hz. For the present analysis we used Mallat’s dyadic pyramid algorithm for computing the fast DWT (20, 21) to obtain a multiresolution analysis (also known as the multiscale approximation).

We used the Daubechies 4 mother (generating) wavelet function (4). The mother wavelet is used to generate an orthonormal basis, where each term represents a doubling of scale (dyadic dilation). This provides a unique decomposition of each biological signal into sub-bands of wavelet coefficients, each representing the contribution of the scaled and translated wavelet function at the given scale. We employed an extended version of the DWT as described by Percival and Walden (28) to produce a “maximal overlap discrete wavelet transformation (MODWT).” The MODWT fills all time points at each scale, allows precise alignment of the signal and its wavelets, allows for any sample size (not just a power of 2), has zero phase-shifted details (representing each scale’s contributions to the original signal as a function of time; a detail is the inverse DWT of the Wavelet coefficients at given scale), and zero phase-shifted smooths (representing averages over all scales larger than the scales of interest as a function of time). Wavelet coefficients can be used to perform an analysis of variance. Averages over wavelet coefficients at any scale are zero. The square of the wavelet coefficient at each scale in the time sequence corresponds to the power or variance contained at that scale as a function of time. By Parseval’s theorem, the sum of all power over all scales equals the total variance as a function of time. By this approach, the first (finest) wavelet scale corresponds to 1 Hz, the second wavelet scale corresponds to 0.5 Hz, the third scale corresponds to 0.25 Hz, the fourth to 0.125 Hz, the fifth to 0.0625 Hz, the sixth to 0.03125 Hz, and larger scales (lower frequencies) are combined into a smooth. The first and second scales correspond to the HR and are simply removed from the analysis. The fifth and sixth scales corresponding to 0.0625 and 0.03125 Hz contain considerable power only at end points and little power during the tilt. Multiresolution analysis for the systolic AP from a representative healthy subject is shown in Fig. 2. The original signal, the pertinent details, corresponding power, and smooth are shown. We focused attention on the third and fourth scales corresponding to 0.25 and 0.125, which contain the wavelet power classically related to the respiratory rhythm (0.25 Hz) and to sympathetic baroreflex activity (~0.1 Hz). Multiresolution analyses were performed for SBP, diastolic BP, MAP, RR interval, and respiration.

Data Analysis and Statistics

Data were digitized and stored in a personal computer and were analyzed offline with custom software. Wavelet power at the LF band (4th dyadic scale ~0.125 Hz) and HF band (3rd dyadic scale ~0.25 Hz) for RR interval, BP, diastolic BP, MAP, and respiration were determined at each fiducial point and averaged over subjects. Data are expressed as the logarithm base 10 of averages over 15-s intervals centered at these time markers to accommodate the wide dispersion of values. The alpha index, a measure of cardiovascular baroreflex sensitivity, was computed as the square root of the ratio between LF RR interval power and LF systolic AP power.

There were two groups of subjects, fainters and control subjects, whose data are represented over time. We used repeated-measures ANOVA to compare group and time-dependent differences at fiducial points. This defined “time effects” or significant differences within a group and “group effects” or significant differences between the groups. If significant, a Bonferroni test was used as a post hoc test for multiple comparisons. To compare data between fainters and control subjects, results were calculated by using SPSS (Statistical Package for the Social Sciences) software version 14.0 and graphed by using GraphPad Prism (San Diego, CA) software version 4. Graphic results are reported as means ± SE.

RESULTS

Results are depicted in Table 1 and Figs. 3–7. Power (variance) is expressed in arbitrary units (au).

Table 1 shows age and resting vital signs in fainters and control subjects. There are no significant differences in age; gender distribution; systolic, diastolic, and mean BPs; or respiratory rate.

Arterial Pressure

Figure 3 shows a multiresolution analysis of systolic AP from a typical fainter in contrast to a similar analysis shown for a control subject in Fig. 2. The LF (0.125 Hz) and HF (0.25 Hz) power is contained within a box. There is a decrease in LF and
HF power from the middle of upright tilt until fainting occurs. Such a decrease is not observed in control subjects.

Figure 4, top compares the systolic and diastolic pressures for fainters with control subjects. The figure shows a small ($P < 0.05$) increase in systolic pressure during upright tilt. There was an abrupt decrease in systolic and diastolic pressure at the time of the faint.

Averaged LF (0.125 Hz) variance (power). Figure 4, bottom shows systolic, mean, and diastolic wavelet power for fainters and control subjects. Systolic, diastolic, and MAP LF variances (Mayer waves) were similarly increased during HUT compared with baseline ($P < 0.01$) for both fainters and control subjects. Baseline variance of MAP and diastolic pressure were significantly decreased for fainters compared with control subjects. Although systolic, diastolic, and MAP variances were not different between fainters and control subjects at 1 min post-HUT, a group effect reduction in systolic, diastolic, and MAP variances was present in fainters compared with control sub-
Table 1. Demographic and resting hemodynamics in fainters and healthy control subjects

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Fainters</th>
<th>Controls</th>
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</thead>
<tbody>
<tr>
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<td>8</td>
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<tr>
<td>Age, yr</td>
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<tr>
<td>Sex, %female</td>
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<td>58%</td>
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<td>Resting SBP, mmHg</td>
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<tr>
<td>Resting DBP, mmHg</td>
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<td>67±3</td>
</tr>
<tr>
<td>Resting MAP, mmHg</td>
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<td>85±3</td>
</tr>
<tr>
<td>Resting HR, beats/min</td>
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<td>65±4</td>
</tr>
<tr>
<td>Resting RR, min⁻¹</td>
<td>18±1</td>
<td>19±1</td>
</tr>
</tbody>
</table>

Age and resting vital signs in fainters and control subjects. There are no significant differences in age; gender distribution; systolic (SBP), diastolic (DBP), and mean (MAP) blood pressures; or respiratory rate (RR). HR, heart rate.

Overall respiratory variance was significantly increased from baseline by HUT in both fainters and control subjects. Respiratory variance was significantly greater than controls (P < 0.01) leading up to the time of fainting.

**Alpha Coefficient**

Data are depicted in Fig. 7.

The alpha coefficient decreased significantly in both fainters and control subjects as a function of time compared with baseline (P < 0.05). During HUT the alpha coefficient was significantly higher in fainters than controls (P < 0.001). Although this difference was greatest during recovery, it was present throughout HUT.

**DISCUSSION**

Our data show that RR interval, arterial BP, and respirations can be readily analyzed at dyadic scales by using the DWT to produce time-dependent multiresolution analyses of these biological signals (Fig. 2 and 3). These produce analytic products capable of distinguishing physiological changes in young patients with simple vasovagal faint. The typical and often reported findings in fainters of increased HR (decreased RR interval) and initially increased SBP compared with control subjects are present, as is the gradual decrease in SBP and TPR (Fig. 1 and Ref. 36) until fainting ensues and HR and BP fall precipitously.

**RR Interval Power**

The decrease in LF RR power shown in Fig. 5 was commensurate in fainters and control subjects. LF RR power is often attributed to sympathetic activity. Sympathetic activity is known to increase during early tilt (3, 15). However, investigators have demonstrated that the transduction of BP variation (Mayer waves) into HR variation is primarily vagal and, thus, parasympathetic (7, 10). Figure 4 shows that LF BP power is reduced in fainters compared with control subjects. This demonstrates that cardiogal gain, represented by the alpha index, shows a similar decrease from baseline and thereafter for fainters and control subjects during HUT. However, the alpha index is larger in fainters at all times. Increased gain/sensitivity implies increased transfer gain. In fainters gain is increased such that significantly reduced AP power is transduced to an unreduced RR interval power.

HF RR interval power increases to a significantly greater degree in fainters compared with control subjects only around the time of faint, implying an increase in vagal tone that has its onset at, but not prior to, the time of faint. Furlan and colleagues (9) noted a similar increase in HF RR interval in a study of young healthy volunteers who experienced fainting for the first time during a 90° head-up tilt. Differences in overall HR variability were confined to faint and the postfaint period.

**Arterial Pressure Power**

Although controversial, we propose that LF AP variance, representing the well-known Mayer wave phenomena (13, 22), is an adequate and sufficient time-dependent index of sympathetic baroreflex sensitivity/gain. It increases in both control subjects and fainters with upright tilt (5) corresponding to the

**Respiration**

Data are depicted in Fig. 6.

Respiratory variance (power) was assessed at the scale corresponding to 0.25 Hz, which approximates the respiratory frequency. Respiratory frequency is not disturbed by HUT (36). There was no difference in baseline respiratory variance between fainters and control subjects.

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increase in sympathetic nerve activity (9) and is (appropriately) directionally opposite to changes in cardiovagal gain as observed in muscle sympathetic nerve activity (MSNA)-tilt experiments (26). The progressive decrement observed in vasovagal fainters from the first minute of tilt resembles the falloff in LF oscillations of sympathetic nerve activity (14). LF diastolic pressure is the most markedly affected, which is comparable to the relationship between diastolic BP and MSNA during peroneal microneurography (35). LF AP power in fainters is always less than or equal to control, consistent with observations of sympathetic baroreflex dysfunction in patients with vasovagal syncope (1). The time course of fall in...
LF wavelet AP power precedes and predicts a fall in TPR at a later fiducial point as is shown in prior work (36). We interpret these findings to signify that a consistent and progressive central process produced by a progressively reduced sympathetic baroreflex is detected by changes in Mayer wave variance and accounts for the time course of faint.

Respiratory Power

It is now well known that hyperpnea and hypocapnia precede simple faint in a majority of cases (16). Our data and those of Bernardi’s group (29) demonstrate increased respiratory variance in fainters compared with control subjects, which may adversely affect cerebral blood flow and potentiate syncope.

Results in the Young

Of significance is the age group of our subjects. In the Prevention of Syncope Trial (POST), Sheldon and colleagues (32) reported a mode age of 13 yr and a median age of 18 yr as the most frequent ages of onset of vasovagal syncope. Since simple faint most commonly begins during adolescence, it may be important to study children’s autonomic responses to tilt since they are physiologically different from adults. A variety of time-frequency methods have been employed in adults and have yielded different results. Thus Novak and coworkers (18, 25) used Wigner-distribution based time-frequency analysis and found no differences between fainters and control subjects at Mayer wave frequencies. In contrast to our data, those investigators suggested that very-low-frequency waves (0.01–0.05 Hz) could represent sympathetic activity and showed that an increase in the amplitude of LF systolic and diastolic oscillations during the first 5 min of tilt followed by a decrease and eventual disappearance of the very-low-frequency (0.01–0.05 Hz) oscillations in fainters. Age differences or different analytic methods may account for these discrepancies.

Other researchers have found no decrease in BP variability at all. For example, Lipsitz et al. (20) found that systolic and diastolic BP variabilities were similar comparing fainter and nonfainters. They found that although BP decreased during HUT there was no significant change in high or LF BP during the 3 min before syncope.

Limitations

A coherence-transfer function analysis is not a natural part of the discrete wavelet approach. Cross correlation-covariance analyses can provide somewhat similar information but have not been well developed for discrete wavelets applied to nonstationary signals. Continuous wavelets can be used to produce a gain-phase approach but cannot supply a finite orthonormal basis for signal approximation.

The use of fiducial time markers allowed us to compare subjects at similar physiological time points but removed the absolute time dependence of the observed phenomena. We did not directly measure sympathetic or parasympathetic activity nor did we seek to manipulate them. However, the preponderance of evidence suggests that Mayer waves take their origin in sympathetic baroreflex feedback loops whereas HF HR variability and the alpha index can serve as adequate surrogate indexes of cardiac parasympathetic activity.
We studied the subjects during 10 min of HUT, which was considered an appropriate time on the basis of prior studies (12). Increasing the time of HUT may give different information; however, healthy control subjects may faint during prolonged standing.

Our study focused on syncope in the young. The peak incidence of syncope occurs during adolescence. Therefore our findings may not completely translate to adults. For example, a gradual decrease in BP followed by a rapid increase is characteristic of young patients whereas older subjects may have only an abrupt fall in BP, a gradual fall in BP, or both.

The study describes the pattern of changes in oscillations of BP and RR interval that are in some ways similar to those reported by Kamiya et al. (15), who also documented accompanying MSNA changes. Dr. Kamiya focused on MSNA but also RR interval and AP comparing data from healthy male subjects who fainted during a 60° upright tilt to healthy male subjects who did not faint. Such healthy fainters would be regarded as “false positives” and could not be assumed to be equivalent to the episodic fainters that we studied. Currently, the American Heart Association uses clinical criteria to determine the diagnosis of reflex neurocardiogenic syncope (simple faint). One reason for this is the plethora of “false positive” results especially in the young. Had Dr. Kamiya studied female false positives as well, he might have noted a diversity of responses including vaso-depressor responses and sinus tachycardia that would have fallen outside the range of an investigation of postural syncope. In addition he studied a somewhat different age group using the technique of complex frequency demodulation, which differs from multiresolution wavelet analysis used here. The demodulation technique employs classic Fourier-based filtering techniques to low-pass filter a frequency-shifted signal. Although it is similar in some ways to wavelets, which can be interpreted as band-pass filters with interesting scaling properties, there remain differences. Nevertheless, the general trends in Dr. Kamiya’s results and our own are similar. This may imply that false positive fainters are similar to episodic fainters.

Fig. 5. RR interval (RRI) as a function of time is shown at top left. LF wavelet power as a function of time at top right, HF wavelet power (variance) as a function of time at bottom right, and LF-to-HF ratio at bottom left. ■, Fainters; □, healthy controls. RR interval was smaller (heart rate was higher) during tilt in fainters until fainting supervened, after which RR interval was larger in fainters than in control subjects. LF power changed similarly with time for fainters and controls alike. HF power decreased significantly and similarly with time in fainters and controls until fainting supervened, after which HF power was larger in fainters than in control subjects. LF-to-HF ratio tended to be increased in early HUT in fainters and was significantly decreased at the time of faint and during early recovery. *P_{time*group}, probability of group and time differences.

Fig. 6. Changes in respiratory power (variance) during HUT in fainters (closed boxes) and control subjects (open boxes). Power started from a similar baseline and increased with HUT for fainters and control subjects. Power was greater in fainters throughout HUT (P < 0.01).

Fig. 7. Alpha index computed as the square root of the ratio between LF RR interval power and LF systolic AP. Alpha index was used as an index of cardiovagal baroreflex sensitivity. ■, Fainters; □, healthy controls. The index decreased significantly with HUT in fainters and control subjects. The index was larger in fainters compared with control subjects during HUT, at the faint itself and during recovery.
To conclude, fainting in young patients is associated with an initial rise and then a steady falloff of lower frequency AP wavelet power (Mayer wave power) as a function of time during upright tilt. This occurred in every fainter and the initial decrease in power was absent in control subjects and predictive of fainting. There are reasons to believe that LF power reflects sympathetic baroreflex activity. The diminished baroreflex activity is best related to diastolic BP. Differences in cardiovagal sympathetic baroreflex activity are limited to the acute faint and recovery. Disturbed respirations occur. However, sympathetic baroreflex activity is typically enhanced by hypocapnia, creating a question of causality.

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