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Windowed FFT – a time-variant spectral analysis: applicability during the head-up tilt test

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The spectral assessment of heart rate variability (HRV) and blood pressure variability (BPV) is a well-established method for the identification of rhythmic fluctuations during stationary conditions, but there is no generally accepted method of describing dynamic changes in such spectral patterns. Our goal was to introduce an alternative means of assessing the dynamics of spectral HRV.

Continuous ECG and non-invasive BP recordings on 29 subjects during head-up tilt testing were subjected to analysis. The total spectral power and the power over the low (LF: 0.04–0.15 Hz) and the high-frequency (HF: 0.15–0.4 Hz) spectral bands were recalculated in an overlapping series with constant time shifting of the initial data-point.

The time course of LFHRV augmentation, with an early peak and subsequent levelling within the first 2 of tilting, was also documented, with parallel changes in LFBPV (LFHRV ms²/Hz: 290 ± 96 supine, 2707 ± 1557 maximum after tilt, $p < 0.05$ LFBPV mmHg²/Hz: 10 ± 4 supine, 58 ± 26 maximum after tilt, $p < 0.05$). In a subgroup of 7 patients who exhibited syncope upon tilting, a statistically significant early increase HFHRV was also detected, followed by significant decline by the second minute of tilting (HFHRV ms²/Hz: 143 ± 80 supine, 1054 ± 902 maximum after tilt, 125 ± 84 minimum after tilt). This early HFHRV peak was absent in the group of tilt-negative subjects. Another characteristic feature of the tilt positive group was a second phase of LFBPV and LFHRV elevation preceding the syncopal episode.

Windowed fast Fourier transformation is a suitable method for assessment of dynamic HR and BP spectral changes during upright tilt testing. The method is well applicable for the analysis of tilting-induced autonomic responses. *J Clin Basic Cardiol 1999; 2: 241–4.*

Key words: spectral analysis, heart rate variability head up tilt

Modulation of cardiac interbeat intervals has been subjected to extensive studies. A certain degree of consensus regarding the spectral assessment of the heart rate variability (HRV) has already been reached [1, 2]. When fast Fourier transformation (FFT) is used to assess HRV, a minimum period of 2–3 mins is required [1, 2]. This is somewhat of a paradox: during 2 mins of a manoeuvre in which the autonomic tone can change, the significant changes in autonomic tone are completed. Thus, the spectral approach focuses on discrete sections of the events and there is no appropriate tool to characterize the dynamics of the spectral fluctuations. A new time-dependent analysis, based on the autoregressive method, was recently reported [3]. Our present goal was to introduce an alternative means of assessing the dynamics of the spectral HRV and blood pressure variability (BPV). As head-up tilt testing (HUT) results in characteristic changes in the cardiovascular autonomic tone, we tested the applicability of our suggested method, windowed FFT, by analysing the records on 29 subjects during HUT. Since the literature data relating to the modification of HRV and BPV during HUT are controversial, a further goal was to describe the HRV and BPV changes during HUT with this novel method, focusing on the dynamics of the changes.

Methods

Patient population

Records on 29 patients during HUT were selected from our data base. All subjects had been referred to the arrhythmia service because of unexplained syncope. During the initial assessment, including history, physical examination, ECG and echocardiography, a cardiac cause of the syncope was definitely excluded in each case. Only virtually noise-free records were considered for further analysis. Records with ectopic beats were also excluded from the study.

Head-up tilt test

The tilt-table test was performed in a quiet room with low light and always between 10 and 12 a.m. After 30 min in a supine resting position, patients were tilted to 70° for 40 min. A table with a footboard support was utilised, and the tilt position was reached in 30 s. Baseline ECG and BP recordings were made for 10 min prior to tilting, and were continued throughout the study. Syncope was defined as a transient loss of consciousness, accompanied by a loss of postural tone. Warning symptoms of syncope include nausea, vomiting, impaired vision, the hearing of distant sounds, a slow response to verbal commands, and a partial loss of postural tone. Dizziness accompanied by one or more of the above symptoms was defined as presyncope.

Data acquisition and analysis

BP was monitored with a photoplethysmograph (Finapres 2300, Ohmeda). ECG signals were recorded by a 5-lead system. The BP and ECG signals were transmitted through an amplifier, filter and analogue-digital converter into an IBM-AT-compatible computer. Data were stored and analysed off-line by means of a program developed in our laboratory. With our system the precision of RR interval detection is 2 ms, and that of BP detection is 1 mmHg. Power spectrum analysis was performed on 2-min segments of the ECG and BP records by using FFT over two frequency bands. The low-frequency band (LF) of the HRV and systolic BPV was defined at 0.04–0.15 Hz, and the high-frequency component (HF) at 0.15–0.4 Hz [4].

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Windowed FFT

Windowed FFT is a narrow time Fourier transformation. Briefly, the method is based on recalculation of the FFT with a variable shifting of the initial complexes in time, allowing the generation of 3D representations of spectral changes. The principles of the mathematical assessment were formulated by Gábor in 1946 [5]; the corresponding equation is:

$$X(f,\tau) = \int_{-\infty}^{\infty} w(t,\tau)x(t)e^{-i2\pi ft}dt$$

where x(t) is the signal to be transformed, w(t,τ) is the window function, τ is the time variable for the time-dependent spectrum X(f,τ).

For w(t), the Hamming window is used with 2-min durations. FFT was performed to calculate the power spectrum for all frequencies for this time range. Shifting the window in time yields the time-dependent power spectrum. In this study, the method was applied for both the RR interval and BP records. The time course of HR and systolic BP spectral changes was analysed in each case, and power spectrum values of certain characteristic tilt stages were determined. Values calculated from the last sequence prior to tilting were defined as baseline. Post-tilt maximum and minimum values in the LF and HF power bands for both HR and systolic BP were determined and compared with the baseline. For those exhibiting syncope, the corresponding values were also determined at the time of fainting.

Statistical analysis

Changes were compared with the baseline by using ANOVA for repeated measures. For parameters exhibiting a non-Gaussian distribution, the Friedman one-way repeated measures ANOVA on ranks was used. The level of statistical significance was set at $p < 0.05$.

Results

Twenty-two subjects exhibited no abnormal reactions during the tilt test (tilt-negative cases). Syncope developed in 7 subjects (tilt-positive cases), 7 to 30 min after tilting. The tilt-positive subjects displayed a "mixed response", characterised by bradycardia and a variable degree of hypotension. Demographic data on the tilt-positive subjects are shown in Table 1. The mean ages of the tilt-positive and negative subjects were similar (22.1 ± 4.4 and 23.5 ± 3.7 years, respectively; p = NS).

Upon tilting, there were immediate fluctuations in HR and systolic BP power spectral components (Figures 1 and 2). A statistically significant increase in the total-frequency systolic BPV (TFBPV) within 2 min from the beginning of the manoeuvre was mainly due to an LFBPV in both groups (Tables 2 and 3). Subsequently, TFBPV and LFBPV decreased in both groups and levelled at lower-than-maximum values for several minutes. Nevertheless, the minimum TFBPV and LFBPV values after tilting in this phase remained significantly higher than the baseline in the tilt-negative group (Table 2). The same tendency was seen in the tilt-positive group, but the difference in minimum TFBPV and LFBPV did not reach statistical significance as compared with the baseline (Table 3). TFHRV and LFHRV increased significantly in response to tilting, and then declined quickly to post-tilt minimum values, but these were still significantly higher than the baseline in both groups. Peaking occurred within the first minute after the completion of tilting. A different response was seen when HFHRV was assessed. In the tilt-negative group, HFHRV decreased immediately on tilting and underwent no subsequent changes, whereas a significant increase in this parameter was seen in the tilt-positive group. This increase reached its peak within 60 s after tilting, and HFHRV then quickly returned to a lower-than-baseline level (Table 3). The syncopal episode itself in the tilt-positive group was characterised by a diminution of all HRV spectral bands, though this decline reached statistical significance for only the TFHRV and LFHRV bands as compared with the baseline. There was a similarly marked reduction in the TFBPV and LFBPV components in association with syncope. In contrast, the HFBPV component displayed a small and statistically non-significant increase (Table 3).

Table 1. Demographic and tilt-table test data on patients with positive tilt-table test

Pt. no.	Age (yr)	Gender	Time of syncope from tilting (min)	dSBP (mmHg)	dHR (l/min)
1	22	F	8	135	85
2	30	F	7	50	59
3	18	M	10	72	39
4	22	M	8	95	41
5	24	F	10	47	43
6	23	F	6	62	41
7	16	F	30	68	52

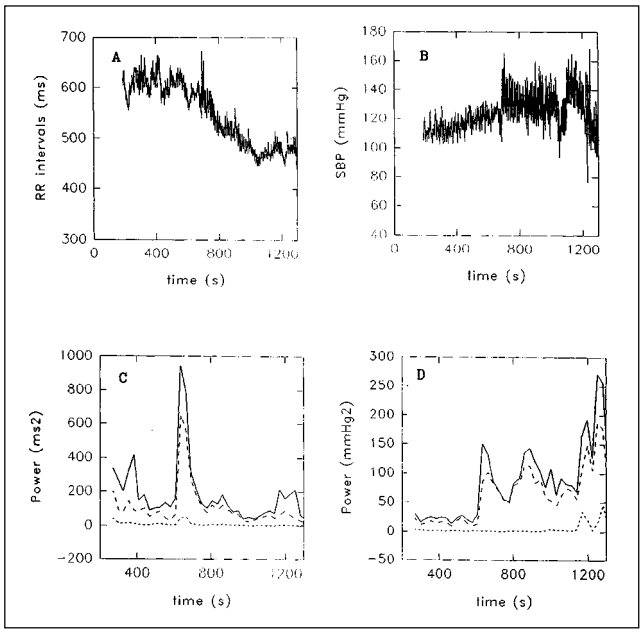

Figure 1. Upright tilt-induced responses of a patient from the tilt-negative group. *Panel A.* Normal heart rate response to upright tilt. The postural change at 600 s of the recording is accompanied by a certain RR interval shortening. *Panel B.* Although the fluctuations in systolic BP are augmented following the upright tilt, no clinically significant hypotension is recorded. *Panels C and D.* Variations in HR (C) and systolic BP (D) powers with time, as assessed by the windowed FFT method. Solid lines indicate total power, dashed lines indicate LF power, and dotted lines indicate HF power. The upright tilt manoeuvre elicits transient elevations in the LFHRV and TFHRV power. The BP power reveal immediate tilt-related increases in the same spectral bands, followed by subsequent fluctuations of lesser magnitude. The terminal increases in the BPV components are related to the motion artefacts at the time of termination of tilting.

Discussion

The assessment of HRV by spectral analysis is gaining increasing use in clinical cardiology [1, 2]. However, the method of spectral analysis dissects the series of events into discrete sequences, allowing no overlap in the assessment. Only a few publications related to time-dependent spectral assessment [3, 6], but the proposed methods are not yet widely accepted. Thus, spectral analysis currently remains the best possibility for the description of stationary states. Dynamic changes are usually expressed as differences between two stable conditions, without any analysis of the process of transition between the two states. The effect of upright tilting on HRV is typically characterised by comparing the supine resting condition with that in a given post-tilt period. This post-tilt transient is defined either in a given time interval after the beginning of or prior to termination of the tilt manoeuvre [7–9], or at a fixed time preceding the syncopal event [10]. Our results indicate complex fluctuations in the HRV and BPV spectral parameters upon tilting. The major finding in this study is that LFHRV and LFBPV increase markedly and promptly after the tilt position is reached. If FFT is used this effect can not be observed, because it occurs in the first 30 s of the post-tilt period. At the time of presyncope, when the sympathetic tone is declining, these components are greatly decreased. Stationary analysis would yield a quite different spectrum in an early assessment starting with the tilt procedure, as compared with a delayed analysis performed even only 2 min post-tilting. Our observations suggest that certain discrepancies in the literature tilt test results might have been due to the different timings of the assessment. Theodorakis et al. generated a series of adjoining spectral segments to characterise the tilt-induced responses [11], and presented first documented time-dependent spectral fluctuations. Pagani et al. selected "stationary sections of data both at rest and during tilting" for assessment; the exact timing of their post-tilt data acquisition, however, was not reported [12]. Montano et al. reported on a study in which they applied different extents of angle tilting for periods of 10 min. From the continuous recordings, stationary segments devoid of arrhythmia [200 to 500 RR intervals] were analysed, but the timing of data acquisition in relation to the onset of tilting was not stated [13]. Boulos et al. determined spectral changes by comparing the baseline values with those for the last 256 consecutive beats of a 15-min tilt test [9]. Bootsma et al. reported on the effects of different angle tilting, but they excluded from their analysis the first minute of recordings in each position: min 2 to 5 were used for computation of HRV spectra [7]. Mizumaki et al. established tilt-induced spectral responses by comparing supine resting values with those recorded during the last 200 beats until 1 min before the end of the tilt [10]. Bloomfield et al. performed comparisons between data acquired in a supine position and those recorded during the last 5 min of a 13-min tilting [8]. In general, the investigators tended to select artefact-free stationary segments of the recordings, thereby precluding assessment of the phase of the postural change itself. Most of the cited reports indicated a

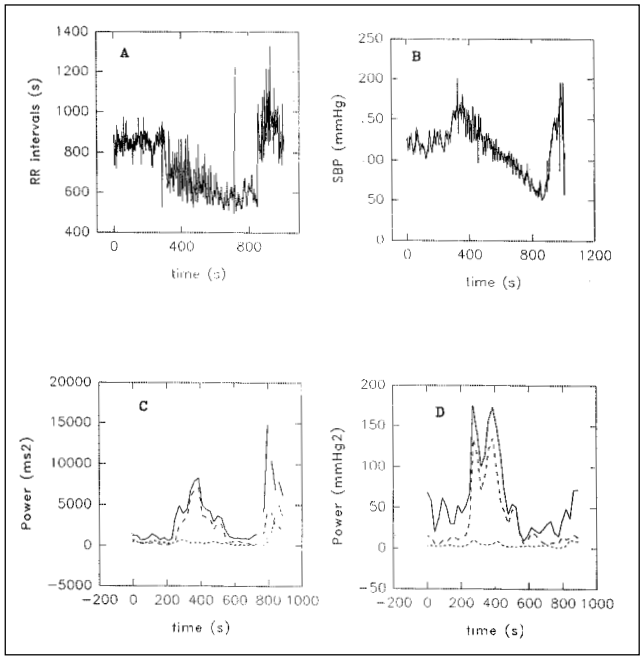

Figure 2. Typical tilt-induced responses of a tilt-positive patient. *Panel A.* The RR interval tachogram. The tilting, indicated by a vertical line is reflected by an immediate HR acceleration. The sudden-onset bradycardia after 800 s of the recording coincides with the syncopal episode. *Panel B.* The systolic BP trend curve reveals transient stabilisation post-tilting, followed by a marked hypotension, eventually leading syncope. *Panels C and D.* Variations in HR (C) and systolic BP (D) with time, as assessed by windowed FFT in the same arrangement as in Figure 1. The upright tilt manoeuvre is paralleled by elevations in the LFHRV and TFHRV powers. The duration of the elevations in these frequency ranges is prolonged, however, relative to the normal, and they are interrupted by further peakings. The subsequent decreases in the spectral components precede the syncopal episode.

Table 2. Changes in spectral components during head-up tilt test in the tilt-negative group (n = 22)

	Supine position	Maximum after tilt	Minimum after tilt
TFHRV (ms^2/Hz)	1255 ± 392	3835 ± 1892*	1618 ± 415*
LFHRV (ms^2/Hz)	514 ± 163	2300 ± 977*	888 ± 223*
HFHRV (ms^2/Hz)	557 ± 234	289 ± 94NS	247 ± 97NS
TFBPV ($mmHg^2$/Hz)	12 ± 3	110 ± 28*	38 ± 10*
LFBPV ($mmHg^2$/Hz)	5 ± 1	54 ± 11*	22 ± 5*
HFBPV ($mmHg^2$/Hz)	1 ± 0	4 ± 1NS	2 ± 0NS

TFHRV: total-frequency heart rate variability, LFHRV: low-frequency heart rate variability, HFHRV: high-frequency heart rate variability, TFBPV: total-frequency blood pressure variability, LFBPV: low-frequency blood pressure variability, HFBPV: high-frequency blood pressure variability; * $p < 0.05$ vs. supine position, NS non-significant

Table 3. Changes in spectral components during head-up tilt test in the tilt-positive group (n = 7)

	Supine position	Maximum after tilt	Minimum after tilt	During syncope
TFHRV (ms^2/Hz)	673±202	4379±2064*	1072±670*	243±105*
LFHRV (ms^2/Hz)	290±96	2707±1557*	663±394*	92±31*
HFHRV (ms^2/HZ)	143±80	1054±902*	125±84NS	38±28NS
TFBPV ($mmHg^2$/Hz)	24±9	86±33*	37±21NS	17±6*
LFBPV ($mmHg^2$/Hz)	10±4	58±26*	23±14NS	2±0*
HFBPV ($mmHg^2$/Hz)	24±0	2±0NS	3±1NS	4±2NS

TFHRV: total-frequency heart rate variability, LFHRV: low-frequency heart rate variability, HFHRV: high-frequency heart rate variability, TFBPV: total-frequency blood pressure variability, LFBPV: low-frequency blood pressure variability, HFBPV: high-frequency blood pressure variability; * $p < 0.005$ vs. supine position, NS non significant

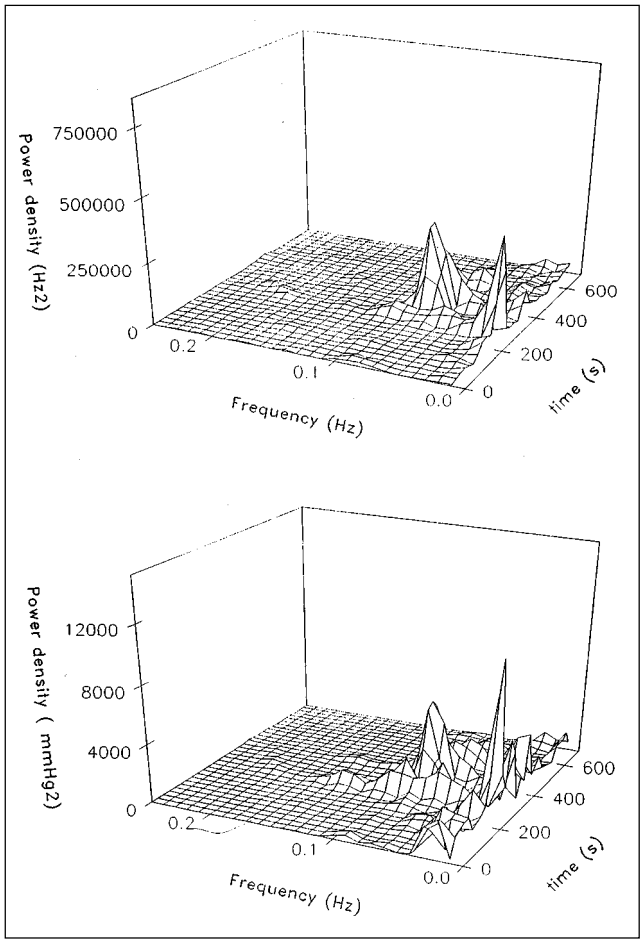

Figure 3. 3D projection of the HRV *(upper panel)* and BPV *(lower panel)* spectral changes following the upright tilt as assessed by windowed FFT in the same syncopal patients as in Fig. 2. The spatial projection allows a thorough assessment of the temporal changes and interrelationship of all spectral components. Thus, it is apparent that the double elevation in the LFBPV response depicted in Fig. 2 is related to changes in different segments of the LF band. Even the two minor elevations in the HFBPV band are quite discernible.

tilt-induced increase in LFHRV, with an increased ratio LFHRV/HFHRV [8–10]. Our findings are in general agreement with those of previous studies, but we found that these changes are most pronounced within the first 2 min post-tilting. Nevertheless, the subsequent equilibrium, which is basically maintained until the end of tilting in tilt-negative subjects, is still characterised by a predominance of LFHRV markers.

The continuous assessment allowed us to detect a short-lasting increase in HFHRV among tilt-positive subjects very early after tilting. This temporary increment in HFHRV among syncope subjects is strikingly different from the immediate HFHRV diminution detected in the group of tilt-negative patients, and may indicate parallel changes in the vagal regulation. Some of the previous studies demonstrated an increased HFHRV just prior to an episode of syncope [9, 11], but no early post-tilt peak of HFHRV has been reported previously. The significance of this finding is not clear and demands further studies. During the phase of equilibrium, ie, after the first 2 min. of tilting, no apparent differences in spectral trends were seen between the two groups. The spectral constellation among negative subjects remained unchanged until the end of the study (Figure 1). In contrast, a second phase of LFHRV and HFHRV elevation was seen among tilt-positive subjects prior to syncope (Figures 2 and 3). However, the magnitude of these peaks did not attain the level of the initial increments. Since our program predicted determinations of only one minimum and maximum value for each subject, these presyncopal peaks in the tilt-positive group remained numerically uncharacterized. Nevertheless, similar presyncopal increments in UHRV have been repeatedly reported [11, 14]. The LFHRV component presumably represents sympathetic activation; indeed, studies on presyncopal catecholamine levels [15] and muscle sympathetic nerve activity recordings documented transient pre-syncopal elevations [16, 17]. The mechanism of presyncopal elevation in HFHRV is not clear. The increase may represent the intense vagal stimulation preceding the onset of syncope [9]. However, we have observed a presyncopal increase in HFBPV as well (Figures 2 and 3), a phenomenon difficult to explain in terms of any vagal mechanism. We hypothesize that this HFBPV peak is a consequence of the presyncopal decrease in venous return, which results in central hypovolaemia and in increased breathing-related fluctuations in the stroke volume. The increased HFBPV may in turn contribute to the genesis of the HFHRV peak via baroreflex mechanisms. One limitation of our study is that the magnitude and timing of the presyncopal spectral peaks remained undefined. It is well known that the duration of presyncope varies widely in these patients. Thus, a stationary analysis in a fixed time interval prior to the full-blown syncope may be inadequate for a representation of the presyncopal constellations. Individual characterization of the presyncopal peaks would be desirable.

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