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

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

T. Szili-Török1, Z. Gingl2, A. Kardos3, L. Halmai1, L. Rudas1

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 min of tilting, was also documented, with parallel changes in LFBPV (LFBPV 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 syncope episode.


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

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Head-up tilt test

The tilt-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.

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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From the 1Medical Intensive Care Unit, Albert Szent-Györgyi Medical University, 2Department of Experimental Physics, Attila József University, Szeged, and the 3Second Department of Internal Medicine, Albert Szent-Györgyi Medical University, Szeged, Hungary.

Correspondence to: Tamás Szili-Török, MD, Medical Intensive Care Unit, Albert Szent-Györgyi Medical University, Szeged, Korányi fásor 7, H-6725, Hungary; E-mail: torok@comser.szote.u-szeged.hu
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, $w(t, \tau)$ is the window function, $\tau$ is the time variable for the time-dependent spectrum $X(t, \tau)$.

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 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 HFBPV was assessed. In the tilt-negative group, HFBPV 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 HFBPV 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).

![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.](image-url)
Discussion

The assessment of HRV by spectral analysis is gaining increasing use in clinical cardiology [1, 2]. However, the method of spectral analysis dissect 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 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.

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

<table>
<thead>
<tr>
<th></th>
<th>Supine position</th>
<th>Maximum after tilt</th>
<th>Minimum after tilt</th>
</tr>
</thead>
<tbody>
<tr>
<td>TFFHRV (ms²/Hz)</td>
<td>1255 ± 392</td>
<td>3835 ± 1892²</td>
<td>1618 ± 415²</td>
</tr>
<tr>
<td>LFHRV (ms²/Hz)</td>
<td>514 ± 163</td>
<td>2300 ± 977²</td>
<td>888 ± 223²</td>
</tr>
<tr>
<td>HFFHRV (ms²/Hz)</td>
<td>557 ± 234</td>
<td>289 ± 94¹ NS</td>
<td>247 ± 97² NS</td>
</tr>
<tr>
<td>TFBPV (mmHg²/Hz)</td>
<td>12 ± 3</td>
<td>110 ± 28</td>
<td>38 ± 10¹</td>
</tr>
<tr>
<td>LFBPV (mmHg²/Hz)</td>
<td>5 ± 1</td>
<td>54 ± 11¹</td>
<td>22 ± 5¹</td>
</tr>
<tr>
<td>HFPPV (mmHg²/Hz)</td>
<td>1 ± 0</td>
<td>4 ± 1 NS</td>
<td>2 ± 0 NS</td>
</tr>
<tr>
<td>TFFHRV: total-frequency heart rate variability, LFHRV: low-frequency heart rate variability, HFFHRV: high-frequency heart rate variability, TFBPV: total-frequency blood pressure variability, LFBPV: low-frequency blood pressure variability, HFPPV: high-frequency blood pressure variability. * p &lt; 0.05 vs. supine position, NS non-significant</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th></th>
<th>Supine position</th>
<th>Maximum after tilt</th>
<th>Minimum after tilt</th>
<th>During syncope</th>
</tr>
</thead>
<tbody>
<tr>
<td>TFFHRV (ms²/Hz)</td>
<td>673 ± 202</td>
<td>4379 ± 2064²</td>
<td>1072 ± 670²</td>
<td>243 ± 105¹</td>
</tr>
<tr>
<td>LFHRV (ms²/Hz)</td>
<td>290 ± 96</td>
<td>2707 ± 1557²</td>
<td>663 ± 394¹</td>
<td>92 ± 31¹</td>
</tr>
<tr>
<td>HFFHRV (ms²/Hz)</td>
<td>143 ± 80</td>
<td>1054 ± 902²</td>
<td>125 ± 84¹ NS</td>
<td>38 ± 28¹ NS</td>
</tr>
<tr>
<td>TFBPV (mmHg²/Hz)</td>
<td>24 ± 9</td>
<td>86 ± 33¹</td>
<td>37 ± 21¹ NS</td>
<td>17 ± 6¹</td>
</tr>
<tr>
<td>LFBPV (mmHg²/Hz)</td>
<td>10 ± 4</td>
<td>58 ± 26³</td>
<td>23 ± 14¹ NS</td>
<td>2 ± 0¹</td>
</tr>
<tr>
<td>HFPPV (mmHg²/Hz)</td>
<td>24 ± 0</td>
<td>2 ± 0 NS</td>
<td>3 ± 1 NS</td>
<td>4 ± 2 NS</td>
</tr>
</tbody>
</table>

TFFHRV: total-frequency heart rate variability, LFHRV: low-frequency heart rate variability, HFFHRV: high-frequency heart rate variability, TFBPV: total-frequency blood pressure variability, LFBPV: low-frequency blood pressure variability, HFPPV: high-frequency blood pressure variability. * p < 0.005 vs. supine position, NS non significant
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 sum and maximum value for each single increment, 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 HFRHRV 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 hypovolemia and in increased breathing-related fluctuations in the stroke volume. The increased HFBPV may in turn contribute to the genesis of the HFRHRV 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 presyncopal varies widely in these patients. Thus, a stationary analysis in a fixed time interval prior to the full-blown syncpe may be inadequate for a representation of the presyncopal constellations. Individual characterization of the presyncopal peaks would be desirable.

References
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