Respiration Differentially Modulates HRV Obtained from Arterial Pressure Wave and Electrocardiogram

Salvador Carrasco-Sosa and Alejandra Guillén-Mandujano*

Departamento Ciencias de la Salud, Laboratorio de Fisiología Médica, Universidad Autónoma Metropolitana, Mexico City, Mexico

The equivalence between the heart rate variability (HRV) derived from RR and systolic pressure to systolic pressure intervals (SPSP) remains controversial. To address this issue, we assessed the variability of the series resulting from the subtraction of the RR intervals from the SPSP intervals (SPSP-RR) and examined how it is affected by two maneuvers that modify the cardiac autonomic status. From ECG, noninvasive arterial pressure and respiratory movements recorded in 20 healthy subjects the RR, SPSP, SPSP-RR and respiratory series were computed, with a resolution of 0.5 ms, in three 5-min conditions: supine (S), postural change (P) and 100 W exercise (E). High (HF) and low (LF) frequency components were computed from the spectral analysis of the series, performed using the Welch periodogram method, and coherence function was obtained by cross-spectral analysis. While the HF component of SPSP intervals was larger than that of RR intervals in all conditions ($p<0.001$), their LF components were not different ($p>0.05$). In the three conditions power spectra of the SPSP-RR series showed a single component located in the HF band, highly coherent with the respiration spectra as indicated by a coherence of 0.88 ± 0.06. Averages of the HF component of SPSP-RR series were 6 ± 3 ms2, 9 ± 5 ms2 and 8 ± 6 ms2 in S, P y E respectively and were not different between conditions ($p>0.05$). The SPSP-RR HF to RR HF ratio was about 4% in S and P but of 420% during E. Our results indicate that the HRV obtained from RR and SPSP intervals are not equivalent because the respiratory influence is greater in the latter. This effect is clearly evidenced by the SPSP-RR series power spectra, especially during exercise. Moreover, they suggest that the respiratory modulation is produced by a non-neural mechanism, probably exerted mechanically onto the arteries.