BACKGROUND: The cardiovascular signals expressed as time series of R-R interval and systolic blood pressure fluctuate at the frequency of 0.1 Hz in healthy subjects (Mayer waves). The shift of R-R interval oscillation pattern to the higher frequencies (>0.1 Hz) is an independent predictor of mortality among cardiac patients (1, 2). However, the physiological background of altered cardiovascular fluctuation pattern is not known. The purpose of this study was to assess the effects of muscle metaboreflex mediated sympathetic activation on R-R interval and blood pressure fluctuations.

METHODS: R-R intervals and beat-to-beat blood pressure were recorded for nine healthy subjects at rest and during isometric handgrip exercise followed by muscle metaboreflex activation induced by post-exercise forearm circulatory occlusion and during a recovery phase after releasing the occlusion (5 min each phase). The prevalent oscillation frequency of cardiovascular signals (PLF) were measured as a frequency at peak power from the spectral band of 0.04-0.15 Hz separately for R-R intervals and systolic blood pressure (SBP) by the autoregressive power spectra method after subtracting the very-low frequency spectral components.

RESULTS: Blood pressure increased from the baseline level of 80±5/139±10 to 95±6/155±11 mmHg (p<0.001, for both) during handgrip exercise and remain significantly elevated during post-exercise circulatory occlusion (92±7/150±13 mmHg, p<0.001 for both) as an evidence of muscle metaboreflex activation. Heart rate increased significantly from the baseline level of 62±8 to 68±6 bpm (p=0.006) during handgrip exercise but returned back to the baseline level during post-exercise circulatory occlusion phase (63±7 bpm, p=ns). PLF(R-R) increased significantly from the baseline level of 0.081±0.011 to 0.089±0.009 Hz (p=0.020) during handgrip exercise and shifted even higher frequencies during post-exercise circulatory occlusion (0.094±0.008 Hz, p=0.017, compared with baseline). Similarly, PLF(SBP) shifted from the baseline level of 0.063±0.014 to 0.081±0.013 Hz (p=0.001) during handgrip exercise and remained significantly elevated during post-exercise circulatory occlusion (0.081±0.015 Hz, p=0.005). All variables returned back to the baseline level when the occlusion was released.

CONCLUSION: The low-frequency fluctuation pattern of both cardiovascular signals, R-R interval time series and continuous blood pressure, are shifted to the higher frequencies during increased sympathetic outflow caused by muscle metaboreflex activation. The altered fluctuation pattern of R-R intervals is sensitive method to detect muscle metaboreflex mediated sympathetic activation.

REFERENCES
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