

Model-Based Analysis of Heart Rate and Blood Pressure Variability

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SUMMARY

Introduction. The autonomous control of heart rate (HR) and blood pressure (BP) involves several feed-forward and feedback mechanisms. In addition, respiration affects HR and BP through neural and mechanical mechanisms. To analyze dynamic interactions between HR and BP variability, there is a need for closed-loop modeling methods, which can control the respiration-related influences. Because of neural mechanisms, the onset of HR change is reported to precede the onset of instantaneous lung volume (ILV) change. This cannot be properly controlled by the purely causal model structures.

Objectives: 1. To develop a closed-loop model, which includes an anticausal transfer mechanism to handle the phase lead of ILV over HR 2. To apply the model on real physiological data to assure its validity.

Methods. The model may be written as

$$\begin{aligned}
 HR(t) &= -\sum_{i=1}^{M_A} a_{11}(i)HR(t-i) - \sum_{i=1}^{M_A} a_{12}(i)SBP(t-i) \\
 &\quad + \sum_{i=-d}^{M_B} b_{11}(i)ILV(t-i) + e_1(t) \\
 SBP(t) &= -\sum_{i=1}^{M_A} a_{22}(i)SBP(t-i) - \sum_{i=0}^{M_A} a_{21}(i)HR(t-i) \\
 &\quad + \sum_{i=0}^{M_B+d} b_{21}(i)ILV(t-i) + e_2(t) \\
 ILV(t) &= -\sum_{i=1}^{M_F} f(i)ILV(t-i) + e_{ul},
 \end{aligned}$$

where *SBP* denotes systolic BP, *a*, *b*, and *f* are the model coefficients, M_A , M_B , and M_F are the model orders, and e_j represent model noise sources (unknown disturbances). Note that present value of HR is affected by future values of ILV (negative delay *d*). This anticausal structure allows modeling the phase lead of HR over ILV.

Healthy young males (n=14) were studied in supine and standing positions during random interval breathing. After

control condition, either atropine (0.03 mg/kg, n=7) or propranolol (0.2 mg/kg, n=7) was injected to the subjects and the tests were repeated. Finally, the test was repeated with double blockade (n=14).

ECG, intra-arterial BP and ILV were registered and transformed into 3 Hz re-sampled time series of HR, SBP and ILV. Baseline drifts were removed and model was fitted to data by least squares method ($M_A = 30$, $M_B = 20$, $M_F = 20$, $d = 5$). Transfer function gains, noise spectra, and impulse responses were computed from the model.

In the analysis, supine vs. standing, sympathetic vs. parasympathetic (standing-atropine vs. supine-propranolol) and control vs. double blockade states were compared.

Results. The model was able identify the different physiological states. The results are physiologically feasible and in good agreement with the previous studies.

Conclusion. The model is valid and provides physiologically feasible information.

RELATED PUBLICATIONS

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