

Monika Petelczyc

FACULTY OF PHYSICS, WARSAW UNIVERSITY OF TECHNOLOGY, WARSAW, POLAND
e-mail: petelczyc_m@if.pw.edu.pl

Jan Jacek Żebrowski

FACULTY OF PHYSICS, WARSAW UNIVERSITY OF TECHNOLOGY, WARSAW, POLAND
e-mail: zebra@if.pw.edu.pl

Rafał Baranowski

NATIONAL INSTITUTE OF CARDIOLOGY, WARSAW, POLAND
e-mail: rbaranowski@ikard.pl

Correlation in human heart rate variability from a stochastic model

The extraction of Kramers-Moyal coefficients [1] from measurement data was applied to human heart rate variability. The expansion truncated at the second element is known as the Fokker-Planck equation. The Langevin equation is equivalent to a model of the system dynamics consisting of two parts: a deterministic one and a stochastic term. The necessary assumption is that the noise term be due to δ -correlated noise [2,3]. For heart rate variability, we found that such a description is valid only for daytime recordings of heart rate variability. Nighttime heart rate variability is characterised by non-negligible higher order Kramers-Moyal coefficients [4]. This effect can be explained by the correlation properties of heart rate variability. Correlations may be related to both deterministic and stochastic components of the heart rate. Using Kramers-Moyal expansion the drift (deterministic) and diffusion (stochastic) terms are calculated. Deterministic term corresponds to regulatory processes in the cardiorespiratory coupling. The stochastic one is a measure of the noise amplitude.

We will present the analysis of shortterm correlations. Especially a particular, asymmetric form of the dependence of the diffusion coefficient on the heart rate will be discussed. This is a measure of the ability of the system to lengthen and shorten the RR intervals [5]. Moreover, for different recordings we obtained a different ranges and shapes of the slow-varying diffusion term as a function of the heart rate close to its minimum. This property can be related to arrhythmic RR intervals. To illustrate this, several recordings from patients with hypertrophic cardiomyopathy will be compared with time series from healthy men.

We will also focus on the occurrence of higher order Kramers-Moyal coefficients and their meaning in terms of correlations [4]. We will discuss the variability of heart rate (mechanisms of increasing and of decreasing of the heart rate) including the effect of recorded pathology on the obtained Kramers-Moyal expansion.

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