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How random is your heart beat?

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Abstract

We measure the content of random uncorrelated noise in heart rate variability using a general method of noise level estimation using a coarse-grained entropy. We show that usually, except for atrial fibrillation, the level of such noise is within 5–15% of the variance of the data and that the variability due to the linearly correlated processes is dominant in all cases analyzed but atrial fibrillation. The nonlinear deterministic content of heart rate variability remains significant and may not be ignored.

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Keywords: Noise estimation; Heart rate variability

1. Introduction

The standard ECG trace that our physician examines looks regular. The pattern visible in the printout of the electrocardiogram seems to repeat itself while in fact the time intervals between heart beats usually change in a complex and irregular way. This phenomenon called heart rate variability is observable when the proper time resolution is used (tens to hundreds of milliseconds of change from heart beat to heart beat). A variety of physiological factors affect human heart rate. It is now well known that the properties of heart rate variability may be an important factor in the assessment of serious cardiac conditions especially of the risk of sudden cardiac death [1]. An open question is the source of heart rate variability.

The normal heart cycle begins with the electrical activity of a specialized group of cells in the right atrium of the heart, the sino-atrial node (SA), which acts as the principal pacemaker of the heart. The action potential then propagates along the atria reaching the second node of the heart, the atrio-ventricular node (AV). Reacting to the potential of the AV node, the His-Purkinje system of fibers inside the ventricles delivers stimuli

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at different locations allowing the ventricles to contract in a concerted way. Both branches of the autonomous nervous system act on the SA and the AV node, constantly moderating the heart rate. The activity of the nervous system is a function of a number of feedback loops, of which the one controlling blood pressure (the baroreceptor system) and the one keeping the level of carbon dioxide in the blood at bay (the chemoreceptor system) play a decisive role [2]. All together, the interplay of at least five nonlinear oscillatory processes affect the human blood distribution system [3,4] and so the heart rate.

Heart rate variability is measured as a time series of the time intervals between successive contractions of the ventricles of the heart (i.e., the RR intervals of the ECG recording). If the sinus node is the pacemaker responsible for the heart rhythm, then such a rhythm is called sinus rhythm. It is the most common and natural rhythm.

An important aspect of the heart rate variability generation process is the interplay between sinus rhythm and the propagation of the action potentials in the form of waves originating in the SA and AV nodes within the atria and the ventricles. In certain circumstances, parts of the heart tissue may become self-oscillatory (a property called automatism) so that various forms of arrhythmia in the atria and in the ventricles occur, disrupting the normal (sinus) rhythm [2]. In particular, during atrial fibrillation, when a breakup of the waves occurs within the atria, the heart rate variability is so large and complex that the rhythm is accepted to be random [5].

However, even without arrhythmia, the variability of sinus rhythm in a healthy individual is very complex (Fig. 1). It is now accepted that, in general, disease as well as age may result in a decrease of heart rate variability. Denervation of the heart due to cardiac infarction or heart transplant reduces heart rate variability severely. In clinical practice, standards exist for the measurement of the properties of the variability of heart rate [1], a means of assessing the state of the heart rate control system, mainly that of the autonomous nervous system. In this context, both time domain (e.g. standard deviation of the heart rate) and frequency domain methods (power spectral analysis) are used for diagnostic purposes and the assessment of the risk of sudden cardiac death, in particular. In many cases these methods are ineffectual: the standard deviation of the heart rate for both healthy individuals and for the high-risk patients may be indistinguishable (Fig. 2) while a large number of arrhythmia in the heart beat sequence renders frequency analysis of the sinus rhythm useless [1].

For this reason, a search for better diagnostic tools for heart rate variability analysis is under way and the sources of the variability itself are being researched. Both goals are, of course, closely related. Considerable effort has gone into methods based on the assumption that, in view of the complexity of the activity of the autonomous nervous system, at least a major part of the variability of the heart rate may be treated as a

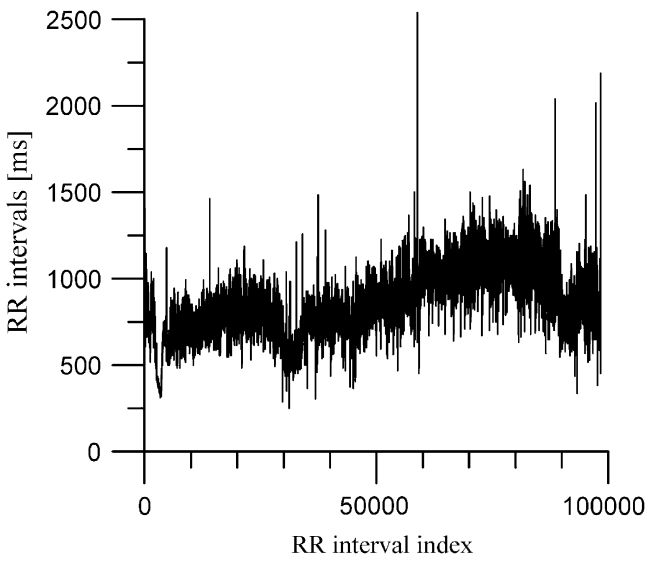

Fig. 1. 24-h time series of the time intervals between heart beats measured as the RR intervals of the ECG recording for the normal CHM. The heart rate variability decreased at around index 500 due to a 10 min exercise stress test. The pauses exceeding 1.5 s are normal.