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Modeling heart rate variability by stochastic feedback

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Abstract

We consider the question of how the cardiac rhythm spontaneously self-regulates and propose a new mechanism as a possible answer. We model the neuroautonomic regulation of the heart rate as a stochastic feedback system and find that the model successfully accounts for key characteristics of cardiac variability, including the $1/f$ power spectrum, the functional form and scaling of the distribution of variations of the interbeat intervals, and the correlations in the Fourier phases which indicate nonlinear dynamics.

The principle of homeostasis asserts that biological systems seek to maintain a constant output after perturbation [1]. Recent evidence, however, indicates that healthy systems display highly irregular dynamics with complex fluctuations [2]. A particularly striking example is heart rate variability [3–6]. Contrary to what we would naively expect [7], the human heart rate is not a stable quantity. Even at rest, the human heart rate shows high variability (cf. Fig. 1(a)). We study this variability in the framework of fluctuations in critical phenomena [8] in order to obtain some insight into the mechanisms regulating the heart rate.

Here, we present a physiologically-motivated model that introduces the concept of stochastic feedback to the study of physiological systems [9]. The model assumes that the heart rate is set by the competing inputs of different neuroautonomic centers. These centers bias the heart rate towards specific rates which are set by random environmental stimuli. The model yields several interesting features not fully explained by other models [10]: (1) $1/f$ power spectrum, (2) stable scaling form for the distribution $P(A)$ of amplitudes A of the variations in the interbeat intervals and (3) Fourier phase correlations.

First, we review some basic physiological aspects of the neuroautonomic control of the heart rate. The healthy human heart rate is mainly determined by three major inputs: the sinoatrial (0) node; and the parasympathetic (+) and sympathetic (−) branches of the autonomous nervous system.

- The sinoatrial node or pacemaker is responsible for the initiation of each heart beat; in the absence of other external stimuli, it is able to maintain an essentially constant interbeat interval [1]. Experiments in which parasympathetic and sympathetic inputs are blocked reveal that the interbeat intervals are very regular and average 0.6 s [11].
- The parasympathetic fibers conduct impulses that increase (+) the interbeat intervals. Suppression of sympathetic stimuli, while under parasympathetic regulation, can result in the increase of the interbeat interval to as much as 1.5 s [11]. The activity of the parasympathetic system changes with external stim-

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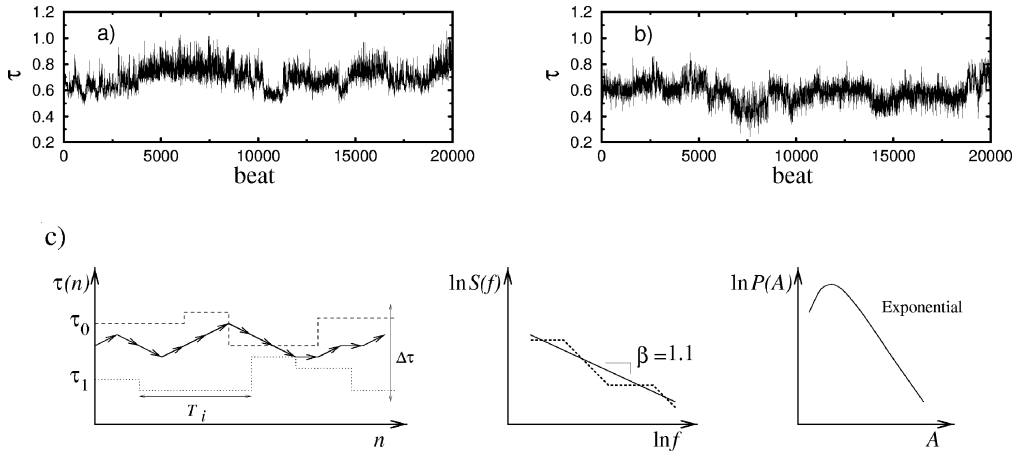

Fig. 1. (a) Sequence of interbeat intervals τ for a healthy individual. (b) Sequence of interbeat intervals for the model (the values of the parameters are given in [9]). (c) Schematic representation of the dynamics of the model: Random walk with two stochastic feedback controls. The levels of attraction τ_0 and τ_1 change values in time. Each level persists for a time interval T_i drawn from a distribution with an average value T_{lock}. Perturbed by changing external stimuli, the system nevertheless remains within the bounds defined by $\Delta\tau$ even after many steps. We find that this dynamical mechanism, based on a single characteristic time scale T_{lock}, generates a $1/f$ power spectrum over several decades. Moreover, $P(A)$ decays exponentially [12], which we attribute to the nonlinear character of the dynamics.

uli and with internal cycles such as the sleep/wake cycle.

- The sympathetic fibers conduct impulses that decrease $(-)$ the interbeat intervals. Abolition of parasympathetic influences when the sympathetic system remains active can decrease the interbeat intervals to less than 0.3 s [11]. There are several centers of sympathetic activity which are highly sensitive to environmental influences [11].

Thus, we can assume as a starting point that the changes in the interbeat interval τ are described by:

$$\tau(n+1) - \tau(n) = I_0(n, \tau_0) + I_+(n, \tau_+) + \sum_{j=1}^{N} I_-^j\left(n, \tau_-^j\right). \tag{1}$$

Clearly, Eq. (1) cannot fully reflect the complexity of the human cardiac system. However, it provides a general framework that can easily be extended to include other physiological controls (such as breathing, baroreflex, etc.). On the other hand, many of the inputs not considered in (1) do not contribute to the frequency regime where scale-free behavior is reported [3–6]. Thus, we can expect that Eq. (1) captures the essential ingredients responsible for a number of important scaling properties of the healthy heart rate.

For each of the inputs in (1), we assume the following mathematical form:

$$I_k(n) = \begin{cases} w_k(1+\eta), & \text{if } \tau(n) < \tau_k, \\ -w_k(1+\eta), & \text{if } \tau(n) \geqslant \tau_k. \end{cases} \tag{2}$$

Here, the weight w_k is the strength of the feedback input biasing the interbeat interval τ to return to its preferred level τ_k, and η is an uncorrelated noise term. From a biological point of view, it is clear that the preferred levels τ_k of the inputs I_k cannot remain constant in time, for otherwise the system would not be able to respond to varying external stimuli. Hence, we assume that each preferred interval τ_k is a random function of time, with values correlated over a time scale T_{lock}^k. We next coarse grain the system and choose (a) $\tau_k(n)$ to be a random step-like function drawn from an uniform distribution and constrained to have values within a certain interval and (b) the length of the steps from a normal distribution with an average value T_{lock} (Fig. 1(b)–(c)).

A notable feature of the present model is that in addition to the power spectra, it accounts for the functional form and scaling properties of $P(A)$, which are independent from the power spectra [12]. No similar tests for nonlinear dynamics have been reported for other models [10].

We study the model for changes in the parameters and find an extended "zone" in parameter space where scaling behavior holds [9]. This robustness of the model to changes in the parameters is consistent with the observation that all healthy individuals obey approximately the same scaling properties in spite of the many differences among them.

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