Dual Antagonistic Autonomic Control Necessary for 1/f Scaling in Heart Rate

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Summary. Although the phenomenon of 1/f noise in heart rate has been known for more than two decades, ours has been the first systematic study showing the importance of antagonistic dynamics between the two branches of the autonomic nervous system \([1]\). We now confirm a previously posed but unproven conjecture that 1/f scaling in heart rate is caused by the intricate balance between antagonistic activity of the sympathetic (SNS) and the parasympathetic (PNS) nervous system. Further, we elaborate on the requirement for dual antagonistic control and present systematic evidence for the corresponding emergence and breakdown of 1/f scaling in human heart rate. We demonstrate that modifying the relative importance of either of the two branches of the autonomic nervous system leads to a substantial decrease in 1/f scaling. In particular, the relative PNS suppression, both by congestive heart failure (CHF) and by the parasympathetic blocker atropine, results in a substantial increase in the Hurst exponent \(H\) and a shift of the multifractal spectrum \(f(a)\) from 1/f towards random walk scaling 1/f. Surprisingly, we observe a similar breakdown in the case of relative and neurogenic SNS suppression by primary autonomic failure (PAF). The observation is further confirmed, not only by group comparison, but also by precise matching of subjects.

1 Introduction

Although the phenomenon of 1/f noise in heart rate has been known for more than two decades \([2-5]\) and has recently also been attributed multifractal scaling properties \([6]\), there has been no successful verification of the importance of antagonistic dynamics between the two branches of the autonomic nervous system.

One conjecture previously posed is that 1/f (global) scaling and local multifractal scaling in heart rate is caused by the interaction between the activity of sympathetic (SNS) and parasympathetic (PNS) nervous systems \([3]\), leading respectively to an increase and a decrease in heart rate. However, the evidence for this is scarce.

A recent attempt to provide such evidence \([7]\) through a drug-induced suppression study has not been fully successful and is rather difficult to interpret because the suppression of only one branch of the autonomic regulatory system at the effector level (i.e. the heart) would lead to compensatory dynamics through the other intact branch.

In \([1]\) we presented the first systematic evidence for the requirement of the dual antagonistic control for the emergence of 1/f scaling in human heart rate. Here, we further elaborate on the duality requirement of the control system arrangement for the
origins of $1/f$ scaling and multifractality in human heart rate. We demonstrate that modifying the relative importance of either of the two branches of the autonomic nervous system leads to a substantial decrease in $1/f$ scaling, showing that $1/f$ scaling in healthy heart rate requires the existence and the intricate balance between antagonistic activity of PNS and SNS.

This supports the view, recently established in [8], of the cardiac neuroregulation as a system in a critical state [9], and permanently out of equilibrium, in which concerted interplay of the SNS and PNS is required for preserving momentary 'balance'. This view of cardiac neuroregulation is consistent with a broad class of models of phenomena which, to a large extent, has been established using the implicit or explicit concept of the balance of competing agents or scenarios.

Further, we observe an intriguing interaction between the multifractality of the heart rate and the absolute variability. While it is generally believed that lower absolute variability results in monofractal behaviour, as has been demonstrated in relative PNS suppression both by congestive heart failure (CHF) [6] and by the parasympathetic blocker atropine [7], we observe conservation of multifractal properties in relative and neurogenic SNS suppression by primary autonomic failure (PAF) at substantially reduced absolute variability to levels closer to CHF. This suggests the relevance of the intrinsic PNS dynamics for multifractality.

We believe these findings to be important in putting forward the dual antagonistic scenario for complex (multi-)fractal dynamics that has now been observed in a wide variety of real-world signals [10], and also in helping diagnose a range of patients with abnormality in their autonomic regulatory system.

2 Methods

We analyse four groups of subjects, of whom long-term heart rate data were measured as sequential heart inter-beat intervals. The first group consists of 115 healthy subjects (26 women and 89 men; ages 16 - 84 yrs) without any known disease affecting the autonomic controls of heart rate, who underwent ambulatory monitoring during normal daily life [Figure 1(a)]. The total number of whole-day data sets is 181, as some of the subjects were examined for two consecutive days, with each data set containing on average 105 heartbeats. Details of the recruitment of the subjects, screening for medical problems, protocols and the data collection are described in Ref. [11]. We analysed both whole-day data, containing periods asleep and awake, and daytime only data, with essentially identical results. In this paper, we therefore present daytime results only.

The second group is of healthy, young males (21 - 26 yrs), who underwent laboratory testing during the administration of the parasympathetic blocker atropine, which reduces parasympathetic control by blocking the action of a parasympathetic neurotransmitter at the heart. Each data set contains > 6,000 heartbeats. Details of the subjects, protocols and data collection are described in Amaral et al. [7].

The third group of subjects are 12 patients with CHF, of whom whole-day ambulatory data [Figure 1(c)] are available from Physionet [14]. This severe heart failure is known to be associated with both increased SNS [15, 16] and decreased PNS [15, 17] activity. Thus, this data set contains information on how heart rate is (multi-) scaled during relative PNS suppression.