Power Laws, Transients, Attractors, and Entropy: Possible Implications for Cardiovascular Dynamics

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Abstract. Considerable work has been done to demonstrate that the dynamics of the cardiovascular system is nonlinear. What has not been well recognized is that the system is essentially governed by transients. Conversely, then, if a system component's dynamics become dominated by basins of attraction, pathology may ensue. It is heuristically demonstrated that a critical feature of possible pathology is the relation of the system entropy to component entropy: if a component's (sub)basin of attraction is accompanied by a decrease in entropy, it may become informationally isolated. A method, originally suggested by Eckmann, et al., is proposed which may be able to detect such changes without the need to reconstruct a global attractor.

1 Introduction

The heart has long been the subject of investigation from a nonlinear dynamics perspective, and a considerable theory of its function has been developed [1]. Experimentally, much interest has been focused on the demonstration of pathologic dynamics with suitable change of critical control parameters.

An often unstated supposition in such experimental time series is that they are obtained from autonomous systems with characteristic times shorter than that of the series itself. Clearly, such assumptions cannot often be substantiated, and a given explanation for a system may yield variable results depending on which "modes" are captured for a given calculation. As a remedy to this situation, Ruelle has proposed the existence of adiabatically fluctuating parameters such that [2]

$$dx/dl = F(x, \lambda(l)) \quad (continuous \quad time), \tag{1}$$

$$x_{n+1} = f(x, \lambda(n)) \quad (discrete \quad time). \tag{2}$$

The suggestion is that instead of a fixed attractor A, there is a family of attractors, A_λ, depending on $\lambda = \lambda(t)$ or $\lambda(n)$. This, however, is dependent upon a faithful representation of the dynamics in $\Re^n$.

In the case of cardiovascular dynamics, it has been demonstrated for the case of inter-beat intervals that there are a number of degrees of freedom acting often in nonlinear ways on time scales much slower than the mean heart rate, and with differing feed-back delays. This is to

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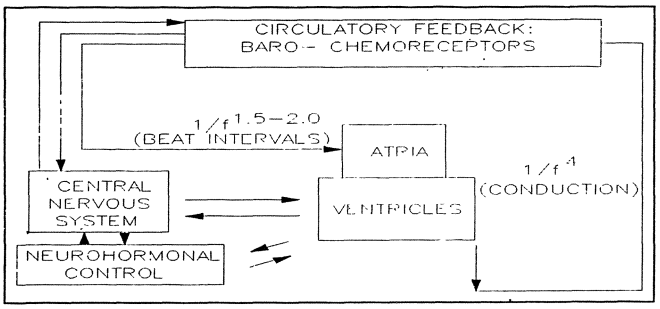

Fig. 1. Schematic representation of major cardiovascular controls.

say that the cardiovascular system is a spatio-temporal system of many degrees of freedom, whose inputs typically exhibit periodicities. With relatively long relaxations relative to the heart's beating, their effects are transients and noise, which proscribe any reasonable global modeling of its dynamics. And certainly this is not unremarkable in that the controls of the heart must constantly be readjusted in order to accommodate the requirements of the larger organism to which it is strongly coupled. (Fig.1)

Although such a description would suggest pessimism relative to an actual rigorous model of the cardiovascular system, the opposite may be true. By suggesting that the normal "stability" of the system is characterized by transients, pathology may be characterized by the absence of the ability to respond to such transients. It is argued that attractors and their (sub)basins of attraction are nonetheless relevant to the dynamics of the cardiovascular system as markers for pathologic states. This is to say that if the organism were allowed to fully relax, the heart dynamics would settle down to an attractor (chaotic or otherwise). But this is not the case, and a resultant approach to an attractor suggests a limitation in phase space in terms of being able to respond to a multidimensional dynamics by virtue of changes in its entropy.

In Section 2, possible models for transient generation in the context of power laws are presented. Section 3 briefly reviews some experimental evidence for the transient nature of cardiovascular controls. Section 4 proposes a heuristically derived theoretical dynamical framework; and Section 5 presents a method which might evaluate these dynamics.

2 Power Laws and Transients

In recent years, it has been emphasized that, at least on the phenomenological level, many cardiac processes exhibit a power law scaling so typical in many natural processes. Considerable attention has been generated by propositions that heart beat and electrocardiogram (ECG) spectra exhibit an inverse power law. The hypothesis is that the normal, healthy heart is characterized by scaling relationships, whereas pathology is characterized by a loss of "spectral reserve," i.e., by increased regularity [3]. Along with these observations, there has been the speculation that this represents deterministic chaos. This derives from the fact that 1/f scaling can be a signature for chaotic dynamics.

Although such a hypothesis is intriguing, attention to the actual values and form of the power spectrum questions the specific formulation of such a hypothesis. Specifically, it is noted that the exponent of scaling is typically > 1 in normal subjects, and depending upon the actual number of beats as well as realizations (scales of seconds to