Available online at www.sciencedirect.com
SCIENCE DIRECT®
Physica A 355 (2005) 439–460
PHYSICA A
www.elsevier.com/locate/physa

Model for the respiratory modulation of the heart beat-to-beat time interval series

Alberto Capurro[a,*], Luis Diambra[b], C.P. Malta[a]

[a]*Instituto de Física, Universidade de São Paulo, Rua do Matão, Travessa R 187, CEP 05508-900 São Paulo, SP, Brazil*
[b]*Instituto de Ciências Biomédicas, Universidade de São Paulo, Av. Lineu Prestes 1524, CEP 05508-900 São Paulo, SP, Brazil*

Received 16 January 2004
Available online 18 April 2005

Abstract

In this study we present a model for the respiratory modulation of the heart beat-to-beat interval series. The model consists of a set of differential equations used to simulate the membrane potential of a single rabbit sinoatrial node cell, excited with a periodic input signal with added correlated noise. This signal, which simulates the input from the autonomous nervous system to the sinoatrial node, was included in the pacemaker equations as a modulation of the i_{NaK} current pump and the potassium current i_K. We focus at modeling the heart beat-to-beat time interval series from normal subjects during meditation of the Kundalini Yoga and Chi techniques. The analysis of the experimental data indicates that while the embedding of pre-meditation and control cases have a roughly circular shape, it acquires a polygonal shape during meditation, triangular for the Kundalini Yoga data and quadrangular in the case of Chi data. The model was used to assess the waveshape of the respiratory signals needed to reproduce the trajectory of the experimental data in the phase space. The embedding of the Chi data could be reproduced using a periodic signal obtained by smoothing a square wave. In the case of Kundalini Yoga data, the embedding was reproduced with a periodic signal obtained by smoothing a triangular wave having a rising branch of longer duration than

*Corresponding author. Tel.: +55 11 3091 6976; fax: +55 11 3091 6833.
E-mail address: alberto@fma.if.usp.br (A. Capurro).

doi:10.1016/j.physa.2004.04.135

the decreasing branch. Our study provides an estimation of the respiratory signal using only the heart beat-to-beat time interval series.

Keywords: Respiratory modulation; Pacemaker; Heart rate variability; Rabbit SA node; Meditation

1. Introduction

Cells within the sinoatrial (SA) node comprise the primary pacemaker site within the heart. These cells are characterized as having no true resting potential, but instead generate regular spontaneous action potentials. The membrane slowly depolarizes during the diastole of the cardiac contraction cycle leading to the onset of an action potential. The repolarization, that occurs after the spike, activates the so called slow diastolic depolarization again and initiates a new cycle. For a comprehensive review of the ionic currents underlying this membrane voltage oscillation see [1].

The intrinsic automaticity (spontaneous pacemaker activity) of the SA node determines a spiking rate of 100–110 action potentials per minute. This intrinsic rhythm is modulated by autonomic nerves, with parasympathetic (vagal) influence being dominant over sympathetic influence at rest. The "vagal tone" brings the resting heart rate down to 60–80 beats/min. Sympathetic activation increases the pacemaker rate with concomitant inhibition of vagal tone [2].

The acetylcholine (ACh) released by the vagal nerve binds to the muscarinic receptors at the post-synaptic membrane, and induces three main actions [1,3,4]: (i) through a reduction in the cyclic adenosine monophosphate (AMPc) production it inhibits the cationic current i_{NaK}, thus decreasing the slope of the slow diastolic depolarization and slowing down the pacemaker rate without changing the membrane potential, (ii) it opens a G-protein coupled potassium channel that hyperpolarizes the cell and slows down the pacemaker rate, and (iii) it inhibits the long lasting component of the calcium current i_{Ca}. The first mechanism is opposite to the β adrenergic action of the sympathetic system that increases the AMPc production [5] and the slope of the slow diastolic depolarization. The sympathetic action also activates the i_{Ca}, but this current has little or no effect on the heart rate. Experimental evidence indicates that the main process that mediates the effect of low-concentration ACh actions on the heart rate is the inhibition of the cationic current i_{NaK} [6].

The normal respiratory cycle is accompanied by changes in autonomic tone that modulates the heart rate. The activity of the vagal nerve endings increases during exhalation, and the activity of sympathetic fibers increases during inhalation, causing the "respiratory modulation" (RM) or "sinus arrhythmia", i.e. during inhalation the heartbeat intervals shorten and during exhalation they stretch. The oscillation in vagal action is responsible for most of the RM, because it is faster than the sympathetic action [2]. At typical respiratory frequencies (greater than