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J. Math. Anal. Appl. 307 (2005) 606–627

Journal of
MATHEMATICAL
ANALYSIS AND
APPLICATIONS

www.elsevier.com/locate/jmaa

Numerical treatment of a mathematical model arising from a model of neuronal variability

M.K. Kadalbajoo *, K.K. Sharma

Department of Mathematics, IIT Kanpur, India

Received 23 June 2003

Available online 1 April 2005

Submitted by R.E. O'Malley, Jr.

Abstract

In this paper, we describe a numerical approach based on finite difference method to solve a mathematical model arising from a model of neuronal variability. The mathematical modelling of the determination of the expected time for generation of action potentials in nerve cells by random synaptic inputs in dendrites includes a general boundary-value problem for singularly perturbed differential–difference equation with small shifts. In the numerical treatment for such type of boundary-value problems, first we use Taylor approximation to tackle the terms containing small shifts which converts it to a boundary-value problem for singularly perturbed differential equation. A rigorous analysis is carried out to obtain priori estimates on the solution of the problem and its derivatives up to third order. Then a parameter uniform difference scheme is constructed to solve the boundary-value problem so obtained. A parameter uniform error estimate for the numerical scheme so constructed is established. Though the convergence of the difference scheme is almost linear but its beauty is that it converges independently of the singular perturbation parameter, i.e., the numerical scheme converges for each value of the singular perturbation parameter (however small it may be but remains positive). Several test examples are solved to demonstrate the efficiency of the numerical scheme presented in the paper and to show the effect of the small shift on the solution behavior.

Keywords: Singular perturbation; Action potential; Fitted mesh; Differential–difference equation; Positive shift; Negative shift; Boundary layer

* Corresponding author.
E-mail addresses: kapils1@lycos.com (M.K. Kadalbajoo), s-kapil@lyscos.com (K.K. Sharma).

0022-247X/$ – see front matter
doi:10.1016/j.jmaa.2005.02.014

1. Introduction

A human brain consists of approximately 10^{11} computing elements called neurons. A typical neuron has three major regions: soma, axon and the dendrites. Dendrites form a dendritic tree, which is very fine bush of thin fibers around the neuron's body. Dendrites receive information from neurons through axons long fibers that serve as transmission lines. An axon is a long cylindrical connection that carries impulses from the neuron. The end part of an axon splits into a fine arborization. Each branch of it terminates in a small end-bulb almost touching the dendrites of neighboring neurons. The axon-dendrite contact organ is called a synapse. The synapse is where the neuron introduces its signal to the neighboring neuron. The neurons communicate through a connection network of axons and synapses having a density of approximately 10^4 synapses per neuron. The hypothesis regarding the modeling of the natural nervous system is that neurons communicate with each other by means of electrical impulses. The neurons operate in chemical environment that is even more important in terms of actual brain behavior. The input to the network is provided by sensory receptors. Receptors deliver stimuli both from within the body, as well as from sense organs when the stimuli originate in the external world. The stimuli are in the form of electrical impulses that convey the information into the network of neurons. As a result of information processing in the central nervous systems, the effectors are controlled and give human responses in the form of diverse actions. We thus have a three stage system, consisting of receptors, neural network, and effectors.

On the theoretical side there have been many advanced model of nerve membrane potential in the presence of random synaptic input. Reviews can be found in J.P. Segundo et al. [4], S.E. Fienberg [5], Holden [6]. Due to the analytic difficulties in solving any realistic model, computer simulation has played an important role as a first step. Stein have given a differential–difference equation model incorporating stochastic effects due to neuronal variability and approximate the solution using Monte Carlo techniques [1]. Stein's model contains the following assumptions:

- (i) Excitatory impulses arrive according to a Poisson process $\pi(f_e, t)$, each event of which leads to an instantaneous increase in the membrane depolarization $V(t)$ by a_e, whereas inhibitory current impulses arrive at event times in a second Poisson process $\pi(f_i, t)$, which is independent of $\pi(f_e, t)$ and causes $V(t)$ to decreases by a_i.
- (ii) If depolarization reaches a threshold of r units, the neuron fires an impulse.
- (iii) After each neuronal firing there is a refractory period of duration, t_0, during which the impulses have no effect and the membrane depolarization, $V(t)$, is reset to zero.
- (iv) At times $t > t_0$, each impulse produces unit depolarization.
- (v) For sub-threshold levels, the depolarization decays exponentially among impulses with time constant μ.

In 1967, Stein generalized this model to deal with a distribution of postsynaptic potential amplitudes [7]. Johannesma [8] and Tuckwell [9] included the reversal potentials into account. Various other models for neuronal activity have been proposed and many are discussed in Holden's book [6].