Gen. Math. Notes, Vol. 11, No. 2, August 2012, pp. 35-46
ISSN 2219-7184; Copyright © ICSRS Publication, 2012
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Mathematical Catastrophe with Applications

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(Received: 27-7-12/Accepted: 16-8-12)

Abstract

In this paper, we have studied the nerve impulse (action potential impulse), and amplitude of the nerve impulse, and then we attempt to fit a catastrophic model for the differential equation which represents the Nerve cell behavior specially excitation of the nerve cell and its catastrophic phenomena by methods of catastrophe theory. The main aim of this paper is to find a catastrophe model to represent the catastrophic behavior of nerve cells, and we have shown that there is a catastrophic behavior of the nerve cell and that there is a mathematical model to represent a Nerve Cell behavior. Furthermore, Nerve behavior is of Cusp type Catastrophe.

Keywords: *Nerve Cell behavior, Mathematical Catastrophe, Catastrophic Model, Cell Membrane.*

1 Introduction

In this paper, we have illustrated the synaptic and local potentials on the folding part of the cusp surface where x is the Nerve impulse parameter that control the frequency depending on the parameter "α" which appears in the differential

equation and control jumps of the excitation of Nerve cell when the parameter crosses the bifurcation set (BS). We divided this paper into seven sections the first section is the introductory, in section 2 we studied catastrophe theory-elementary catastrophes, In section 3 we studied application of catastrophic model to represent a nerve cell and we studied the Nerve cell and its behavior. Also we studied the nonlinear differential equation which has a relationship with Nerve cell behavior. In Catastrophe Theory, manifolds are used to explain sudden changes in the course of an event due to shifts in environmental factors. In catastrophe theory: There are seven elementary types of catastrophes the first four catastrophe geometries [7] are: Fold, Cusp, Swallowtail, and Butterfly catastrophe. Without going into the mathematics of their geometry, we need only to observe that the Cusp manifold has one cusp point, which is the point of coming together of two folds in a sharp spike like intersection. The Swallowtail manifold has two cusp points and the Butterfly manifold has three. Catastrophe theory, in mathematics, a set of methods used to study and classify the ways in which a system can undergo sudden large changes in behavior as one or more of the variables that control it, are changed continuously. Catastrophe theory is generally considered as a branch of geometry because the variables and resultant behaviors are usefully depicted as curves or surfaces, and the formal development of the theory is credited chiefly to the French topologist René Thom.
Catastrophe theor**y** is a branch of bifurcation theory in the study of dynamical systems.
Bifurcation theory studies phenomena characterized by sudden jumps in behavior arising from small changes in parameters, analyzing how the qualitative nature of equation solutions depends on the parameters that appear in the equation.
Catastrophe theory, which originated with the work of the French mathematician René Thom in the 1960s, and became very popular due to the efforts of Christopher Zeeman in the 1970s, considers the special case where the long-run stable equilibrium can be identified with the minimum of a smooth, well-defined potential function.
Small changes in certain parameters of a nonlinear system can cause equilibriums to appear or disappear, or to change from attracting to repelling and vice versa [10], leading to large and sudden changes of the behavior of the systems.

2 Elementary Catastrophes

Catastrophe theory analyses *degenerate critical points* of the potential function i.e. points where not just the first derivative, but one or more higher derivatives of the potential function are also zero. These points are called germs.
If the potential function depends on two or fewer active variables, and four or fewer active parameters, then there are only seven generic structures for these bifurcation geometries, with corresponding standard forms into which the Taylor series around the catastrophe germs can be transformed by diffeomorphism (a smooth transformation whose inverse is also smooth). There are seven fundamental types, with the names that system will make a transition to a new case, very different behavior[7].

2.1 The Potential Function of Cusp Type Catastrophe

The potential function of Cusp type catastrophe is of the form

$f(x)=x^4+ax^2+bx$

The parameters a and b are called splitting and normal factors (respectively)

Let $\Delta = 8a^3 + 27b^2 = 0$

The bifurcation set is equal to the set[10]
$\{(a.b) : 8a^3 + 27b^2 = 0\}$

The diagram of cusp type catastrophe is shown below.
Thom gave them. We will study only cusp types
If the factor a is slowly increased, the system can follow the stable minimum point. But at a=0, the stable and unstable extrema meet. This is the bifurcation point. At a>0 there is no stable solution. If a physical system is followed through a fold bifurcation, one therefore finds that as a reaches 0 the stability of the a<0 solution is suddenly lost
The potential function of the fold type catastrophe is of the form:
$V(x)=x^3+ ax$. So $\frac{dv}{dx} = 3x^2 + a$ .The equilibrium surface

is $\frac{dV}{dx} = 0, i.e.\ 3x^2 + a = 0$. Stable and unstable pair of extrema disappears at a fold bifurcation

3 Applications

Scientists often describe events by constructing a mathematical model. Indeed, when such a model is particularly successful, it is said not only to describe the events but also to explain them, if the model can be reduced to a simple equation. It may even be called a law of nature.
Many phenomena of human behavior involve sudden changes, bimodality, hysteresis, and divergence. Catastrophe theory suggests several models for such behavior. A description of catastrophe theory is presented that includes points of special interest to psychologists and a section on mathematical considerations. If we attempt to find results in science we will to fit a mathematical model to it and then we project them to science.

Now we study a catastrophic model to represent a nerve cell as follows:

3.1 Catastrophic Model to Represent a Nerve Cell

3.1.1 Nerve Cell

The Main Parts of the Nerve Cell [9]

The nerve cell may be divided on the basis of its structure and function into three main parts:
(1) the cell *body*, also called the soma;
(2) numerous short processes of the *soma*, called the *dendrites*; and,
(3) the single long nerve fiber, the *axon*.

These are described in Figure 1.

3.1.2 Nervous System

Nervous system, network of specialized tissue that controls actions and reactions of the body and its adjustment to the environment. Virtually all members of the animal kingdom have at least a rudimentary nervous system. Invertebrate animals show vertebrate varying degree of complexity in their nervous systems, but it is in the vertebrate animals [phylum chordate, subphylum vertebrata] that the system reaches its greatest complexity. The nervous system is built up of nerve cells, called neurons, which are supported and protected by other cells. Of the 200 billion or so neurons making up the human nervous system, approximately half are found in the brain. From the cell body of a typical neuron extend one or more outgrowths (dendrites), threadlike structures that divide and subdivide into ever-smaller branches. The nervous system is divided into two parts: Central Nervous System (CNS) and Peripheral Nervous System. Unit bolding of nervous system is a neuron and the nervous system of human consists of two main types of cells: Glia cells and Neurons. Neuron consists of cell body and axon. cell body consists Nucleons and has dendrites which have relationship for transition or reception the impulse and the cell body receives the electrical impulse from other neurons by their dendrites .The body of a nerve cell (see also (Schadé and Ford, 1973)) is similar to that of all other cells. The cell body generally includes the nucleus, mitochondria, endoplasmic reticule, ribosome, and other organelles. Nerve cells are about 70 - 80% water; the dry material is about 80% protein and 20% lipid. The cell volume varies between 600 and 70,000 μm^3. (Schadé and Ford, 1973) The short processes of the cell body, the dendrites, receive impulses from other cells and transfer them to the cell body. The effect of these impulses may be ***excitatory*** or ***inhibitory*** *(see Fig 2).* A ***cortical neuron*** may receive impulses from tens or even hundreds of thousands
of neurons (Nunez, 1981). The long nerve fiber, the ***axon***, transfers the signal from the cell body to another nerve or to a muscle cell. Mammalian axons are usually about 1 - 20 µm in diameter. Some axons in larger animals may be several meters in length. The axon may be covered with an insulating layer called the ***myelin sheath*** *[Fig 1*] illustrates the construction of myelin sheath) which is

formed by ***Schwann cells***[(1)] The myelin sheath is not continuous but divided into sections, separated at regular intervals by the ***nodes of Ranvier***[(2)]

(1) named for the German physiologist Theodor Schwann, 1810-1882, who first observed the myelin sheath in 1838).

(2) named for the French anatomist Louis Antoine Ranvier, 1834-1922, who observed them in 1878.

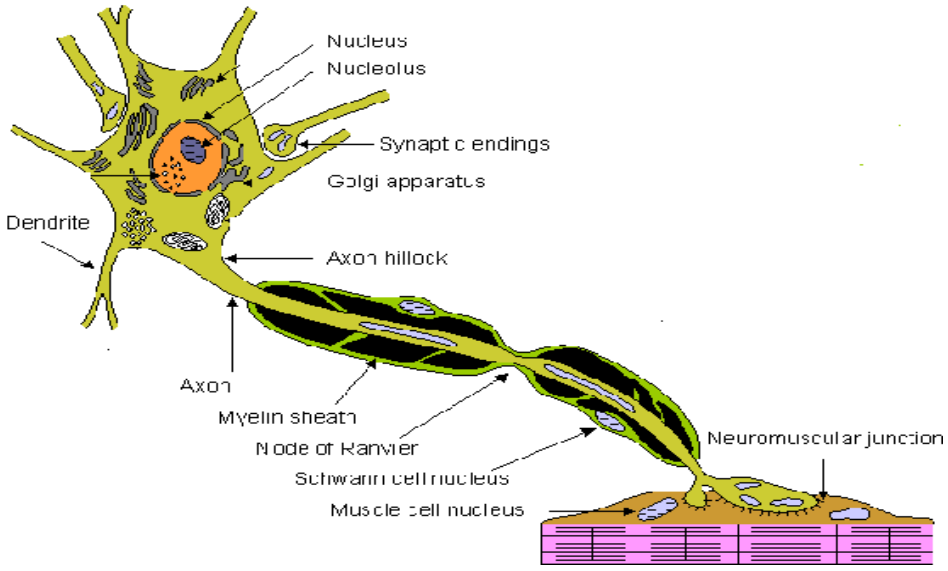
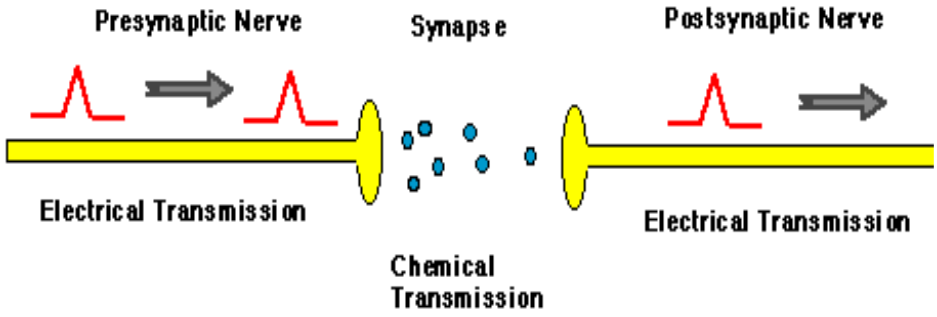


Fig. 1

Nervous system, the system of cells , tissues , and organs that regulates the body's responses to Nervous system internal and external stimuli vertebrates it consists of the brain, spinal cord, nerves, ganglia, and parts of the receptor and affect or organs. Axon expands from cell body and transfers the electrical impulse from Neuron. The axon surrounded by Myelin sheaths which a nonconductor material and nessasur for transferring electrical impulse .The collection of axons with each other make the nerves, and nerves are divided into two types: Pre-Ganglion Nerves and Post-Ganglion Nerves.[9]

Axons expend at their ends into synaptic terminals which make contact with nerves or other types of cells. if the nerves contacts a muscle cell the junction is called a nervous cular junction . Each nerve cell makes contacts with thousands of other nerves. Usually at the dendrites we note that the chemical transmitters carry the signal across synopses. At the synaptic gap the action potential ends. In most cases further transmission of the signals requires chemical transmitter. there are a few examples of electrical synapses known, but most are chemical, synapses delay the signal : chemical transmission is slower than electrical transmission,

chemical transmitters are made and stored in the presynaptic terminal .The nerve carrying the impulse into the synapse is called the presynaptic nerve
The nerve leaving the synapse is called the postsynaptic nerve (the Fig below illustrates the Electrical and Chemical Transmission)



* For transmission to occur the chemical transmitter must be made and stored at the presynaptic side.

*$_1$ Stored in membrane bound vesicles
*$_2$ Transmitter is ready to be released whenever an action potential arrive

Excitatory postsynaptic potential: An electrical change in the membrane of a postsynaptic neuron caused by the binding of an excitatory neuron transmitter from a presynaptic receptor, makes it move likely for a postsynaptic neuron to generate an action potential because the transmitter is only on one side the impulse can go in only one direction [6].

3.1.3. Nerve and Muscle Cells:

An important physical property [1] of the membrane is the change in sodium conductance due to activation, the higher the maximum value achieved by the sodium conductance, the higher value of the sodium ion current and the higher the rate of change in the membrane voltage .the result is a higher gradient of voltage, increased local currents, faster excitation, and increased conduction velocity. The decrease in the threshold potential facilitates the triggering of the activation process.
The capacitance of the membrane per unit length determines the amount of change required to achieve a certain potential and therefore affects the time needed to reach the threshold. Large capacitance values, with other parameters remaining the same, mean a slower conduction velocity. The velocity also depends on the resistivity of the medium inside and outside the membrane since these also affect the depolarization time constant. The temperature greatly affects the time constant of the sodium conductance; a decrease in temperature decreases the conduction velocity [1].

The above effects are reflected in an expression derived by Muler and Markin (1978) using an idealized nonlinear ionic current function for the velocity of the propagating nerve impulse in unmyelinated axon, they obtained

$$v = \sqrt{\frac{i_{Na\ max}}{r_i c_m^2 V_{th}}}$$

Where **v**= velocity of the nerve impulse (m/s) .
$i_{Na\ max}$ = maximum sodium current per length (A/m) .
$\mathbf{V}_{th}$ = threshold voltage (V) .
$\mathbf{r_i}$ = axial resistance per unit length(/m) .
$\mathbf{c_m}$ = membrane capacitance per unit length (F/m) .

A myelinated axon can produce a nerve impulse only at the nodes of rangier. In these axons the nerve impulse propagates from one node to another.
The membrane capacitance per unit length of a myelinated axon is much smaller than in an myelinated axon. Therefore, the myelin sheath [Fig 5] increases the conduction velocity. The resistance of the axoplansm per unit length is inversely proportional to the cross-sectional area of the axon and thus to the square of the diameter. the membrane capacitance per unit length is directly proportional to the diameter . Because the time constant formed from the product controls the nodal trans- membrane potential, it is reasonable to suppose that the velocity would be inversely proportional to the time constant. On this basis the conduction velocity of the myelinated axon should be directly proportional to the diameter of the axon.

3.1.4 Bioelectric Function of the Nerve Cell

The ***membrane voltage*** (V_m) of an excitable cell is defined as the potential at the inner surface (Φ_i) relative to that at the outer (Φ_o) surface of the membrane, i.e. $V_m = (\Phi_i) - (\Phi_o)$. This definition is independent of the cause of the potential, and whether the membrane voltage is constant, periodic, or no periodic in behavior. Fluctuations in the membrane potential may be classified according to their character in many different ways. The classification for nerve cells developed by Theodore Holmes Bullock (1959)[9]. According to Bullock, these transmembrane potentials may be resolved into a resting potential and potential change due to activity. The latter may be classified into three different types [9]:

1. ***Pacemaker potentials***: the intrinsic activity of the cell which occurs without external excitation.
2. ***Transducer potentials*** across the membrane, due to external events. These include ***generator potentials*** caused by **receptors** or ***synaptic potential*** changes arising at synapses. Both subtypes can be inhibitory or excitatory.
3. As a consequence of transducer potentials, further response will arise. If the magnitude does not exceed the threshold, the response will be ***no propagating***. If

the response is great enough, a ***Nerve impulse*** [9] will be produced which obeys the all-or-nothing law and proceeds unattenuated along the axon or fiber.

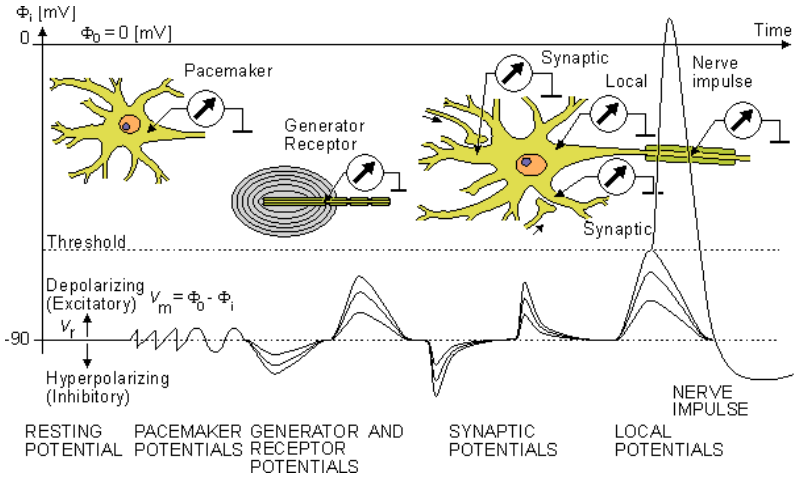


Fig. 2

3.1.5 The Cup Catastrophe and its Properties [9]

The general form of the cusp catastrophe is[7]:

$$V: R^3 \rightarrow R$$

Such that

$$V(x,a,b) = x^4 + ax^2 + bx$$

And a, b are parameters, depending on which the excitation value increases or decreases as the values of a and b varying.
The set {(a, b) ϵ R^2} is called **Control space** (see Fig 3). The catastrophic surface (see Fig 3) is represented by the expression'[7]:

$$\frac{\partial V}{\partial x} = 0 \ ,$$

That is,

$$4x^3 + 2a\,x + b = 0.$$

We are considering (V) and (x) also to be functions of the control variables a, b. Note that a, b are called splitting factor, normal factor respectively and x is the state. The curve (the boundaries of excitation catastrophe) of folding part represented by the expressions [7]:

$$\frac{\partial V}{\partial x} = 0 \quad and \quad \frac{\partial V^2}{\partial x^2} = 0$$

When we eliminate the variable x in these two equations we obtain the bifurcation set {(a, b) :$8a^3+27b^2=0$} this curve is the boundaries of cell excitation catastrophe.

- The input (control) space is two-dimensional; the two control parameters are named *a* and *b.*
- The output space is one-dimensional (the nerve impulse).

In a three-dimensional space data are put together on a surface which seems split. Above some parts of the control space, there are two sheets of the data surface (see Fig 3). When the representative point of the system

- goes on the rip, it jumps from one sheet to the other one.
- The fig. 3 describes the cusp surface. There are jumps but there is also continuous pathway from green to blue.
- The green color is meant the maximum value of the excitation of nerve cell the blue color is meant the minimum value of the excitation, which jumps from one sheet to the other one.
- One can fit a cup catastrophic model in the Brain.
- Now, we have to interpret this split surface.

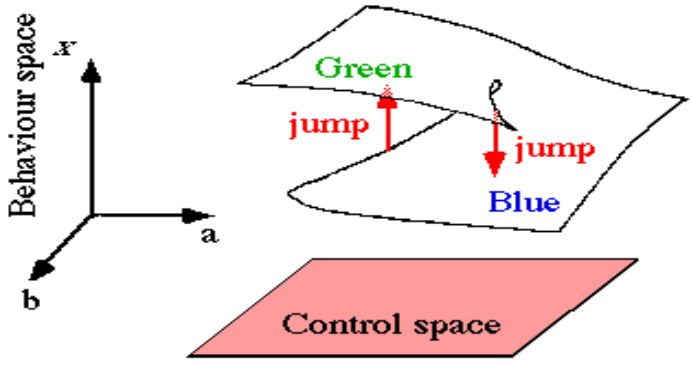


Fig. 3 The cusp as a model for the nerve impulse behavior

The excitation potential V is a function of x and controlled by a and b, what we write $V_{a,b}(x)$. The system may only choose x. We know that the system have **two** possible behavior for some inputs; so we are searching for excitation potential $V_{a,b}(x)$ which may have two minima (see Fig 6).

3.1.6 Nonlinear Differential Equation and Nerve System:

The general form of the nonlinear differential equation of the nerve impulse considered here is written as follows:-

$$\ddot{y} + \varepsilon\alpha\dot{y} + y + \varepsilon\alpha y^3 = \varepsilon(1-\alpha)\cos\omega t \quad . \qquad (\bullet \equiv d/dt) \qquad (6.1)$$

Where ε is very small parameter.

If ε=0 then we have the linear form in which case we are note interested because catastrophic behavior of the nerve cell appear only in the nonlinear differential equation.
For ε≠0 , we proceed to obtain the approximate solution of equation (6.1) as follows

$$\text{Let } \dot{y} = v \qquad (6.2)$$

And, from equations 6.1 and 6.2, we have

$$\dot{v} = -\varepsilon\alpha\dot{y} - y - \varepsilon\alpha y^3 + \varepsilon(1-\alpha)\cos\omega t \qquad (6.3)$$

To satisfy equations 6.2 and 6.3, we further assume that

$$y(t) = A\cos(\omega t + ¢) \qquad (6.4)_a$$
$$v(t) = -A\sin(\omega t + ¢) \qquad (6.4)_b$$

where A is considered as a nerve impulse amplitude.

Substitute eq.s $(6.4)_a$ and $(6.4)_b$ into (6.2) and (6.3) we can find two simultaneous equations ,solving them we can find the non-autonomous systems $\dot{A}$ and $\dot{\phi}$ and integrating w.r.t the time t from 0 to 2π/ω.

We get the following response equation[8]:

$$A^2(¾\alpha A^2 - 2\omega)^2 = (1-\alpha)^2 - \alpha^2 A^2 \qquad (6.5)$$

let $x = A^2$ then after some calculation we get

$$3/4\alpha x^3 - 3\alpha\omega x^2 + (4\omega^2 + \alpha^2)x - (1-\alpha)^2 = 0 \qquad (6,6)$$

By some change of coordinate we can eliminate the term which contains x^2 then (6.6) becomes

$$x^3 - 16/3[\omega^2 - (\omega^2/\alpha) - 1/4\alpha]\,x + 16/9(\omega^2 + \omega\alpha) = 0 \qquad (6.7)$$

Here we note that the change in α cause the change in frequency value.

Or $x^3 + ax + b = 0$ (6.8)

Where

$a = -16/3[\omega^2 - (\omega^2/\alpha) - 1/4\alpha]$ and $b = 16/9(\omega^2 + \omega\alpha)$

The cubic equation (6,8) can have one or three real roots (synaptic or Local potentials as shown on Fig 4) and the condition for the existence of three real roots is[8]

$$4a^3 + 27b^2 < 0$$

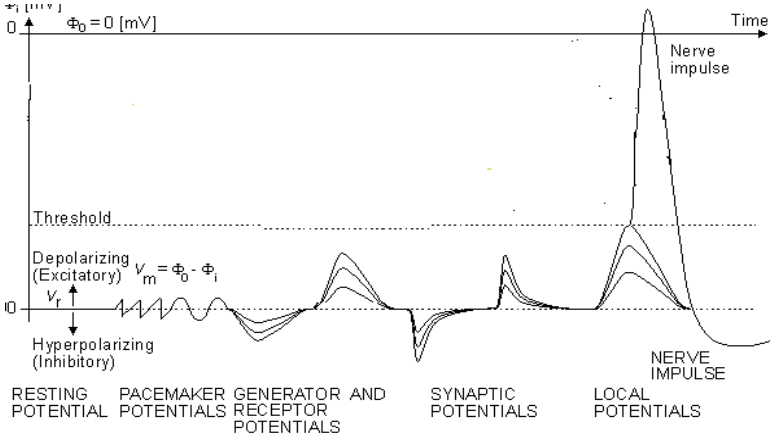


Fig. 4

The surface represented by equation (6.8) can be plotted as shown on figure 5

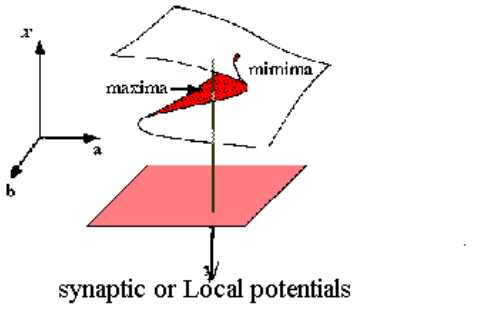


Fig 5 Illustrates the Nerve impulse (triple curve) as shown on Fig 4

Now, after integration of equation (6.8) with respect to x , the excitation potential function is obtained as follows:

$V(x,a,b) = 1/4x^4 + 1/2ax^2 + bx$ (6.9)

x is the amplitude of the Nerve impulse

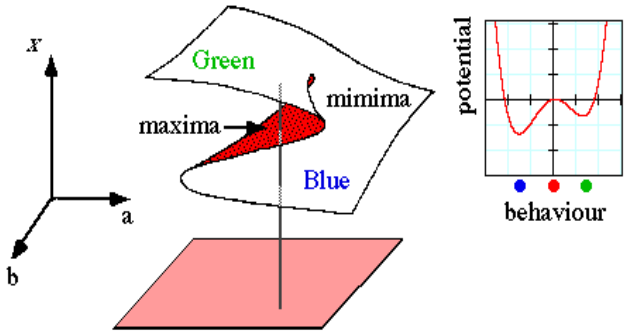


Fig 6 Discuses the catastrophic surface and behavior of potential function (6.9)

4 Conclusion

There are the main results of the paper

Proposition 1 *There is a catastrophic behavior of the nerve cell*
Proposition 2 *There is a mathematical model to represent a Nerve cell behavior*
Proposition 3 *Nerve behavior is of Cusp type Catastrophe*

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