FUZZY PERCOLATION MODEL FOR LOSS OF CONSCIOUSNESS UNDER ACCELERATION STRESS

E.Luzuriaga, B. Onaral, and J.P. Cammarota

School of Biomedical Engineering, Science and Health Systems
Drexel University, Philadelphia, PA 19104, USA

ABSTRACT

In science and engineering, models of real systems are constructed to identify underlying mechanisms and properties, to make predictions or to control their behavior. In the majority of cases, the system to be modeled is based on abstract concepts, on parameters difficult or impossible to precisely quantize, and on experiments limited by technology and the system itself. Due to the unavoidable presence of uncertainty and the usual need to simplify the system and reduce its computational complexity prior to modeling, engineers need to make reasonable assumptions based on their knowledge of the system dynamics. The fuzzy percolation model presented here is an extension to the percolation model (crisp model) introduced in [3]. Both models were built using the tools and concepts of complexity theory by mapping the reticular activating system (RAS) in the brain into a percolation network whose nodes represent a non-specific area of the central nervous system (CNS). To construct the percolation model, data from neuroscience, medical science and aviation medicine were used to determine the local node parameters. Both models utilize the same structure, parameters and variables to represent the cardiovascular and nervous systems; the crisp percolation model makes reasonable assumptions about not well known parameters without taking into account the inherent uncertainty and unavoidable imprecision in their representation. In this article, we show how this uncertainty can be modeled using fuzzy systems. We then test the model and compare its performance to the crisp model using human data. Since both crisp and fuzzy models were able to duplicate human studies with a high degree of fidelity, we conclude that a precise description of the parameters is not necessary when modeling a complex system, provided the parameters remain within physiologically reasonable bounds

1. INTRODUCTION

During airplane maneuvers and centrifuge experiments, pilots are exposed to high G forces that can overcome the human cardiovascular system, decreasing the ability of the heart to supply blood to the brain. Inadequate supply of blood may cause the pilot to experience neurologic and psychologic symptoms such as loss of vision, loss of hearing, disorientation and acceleration (+Gz) induced Loss of Consciousness (G-LOC). Modeling such a complex system as the human response to high G-forces is a challenge. A G-LOC model of the central nervous system (CNS) that captures the global dynamics leading to loss of consciousness under acceleration stress was presented in [3]. This model was built using the tools and concepts of complexity theory along with conventional mathematics. Successful development of the model required careful combination of basic knowledge of the p system both at the local (microscopic) a (macroscopic) levels with experimental data appropriate mathematical tools [4]. The local pa model were derived from a wide range of human a physiologic data. Although reasonable assumpti need to precisely under acceleration stress were quantize the uncertainty inherent in the represen of the parameters were not taken into account w decisions about the state of the local element. following question: to what precision should the local dynamics be known? In this paper, this addressed by introducing imprecision and uncerta crisp G-LOC model using the concepts and tools theory. In the next section, we provide the back for the model development. Section III gives an the previous crisp G-LOC model. Section IV des construction of the fuzzy model. We then co predictions of the two models with respect to hu indicate the future extensions of this work.

2. FOUNDATION TOPICS

2.1 Complexity

In complex systems, spatial and temporal glob is produced through the rich interactions among of local subsystems. Modeling such complex syste challenge. Complexity theories draw their powe recognition that the behavior of a complex dyn does not, in general, depend on the physical pa local elements but rather on how they interact globally observable behavior that emerges from th of the local dynamics [4]. This emergent global appears without the need of a central controller top-down influence on the local elements [6].

The G-LOC model presented here views the nervous system (CNS) as a complex system whos behavior is the transition from consciou unconsciousness. This behavior can be describe (phase) transition which is defined as an abrupt the physical and/or dynamics states of a syste familiar examples of phase transitions are t fundamental states of matter: gas, liquid, pla other examples of phase transitions are chan crystalline structure of matter, in the degree of the dynamics of the system. Determining the stat not always straightforward. Sometimes the appa matter changes when the scale of observation (versus microscopic) is changed. This change of different scales is due to the spatial renor

averaging that takes place over a specified range when multiple elements are replaced with a single element [11].

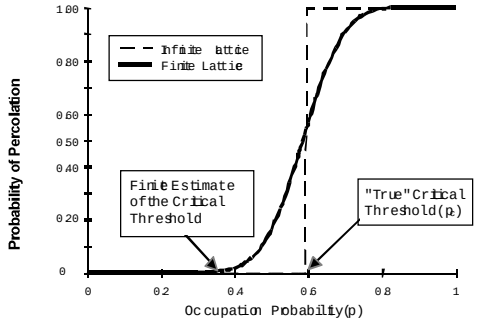

Figure 1 Probability of Percolation (Infinite & Finite)

2.2 Percolation

Percolation networks are often used to model phase transitions [14]. A percolation model is created by using a simple regular geometric framework and by establishing simple interaction rules among the elements on the grid. Percolation theory is based on infinite networks. In site percolation, each node in the grid has only two states, occupied or vacant (on or off). The nodes in the lattice are populated based on a uniform probability distribution, independent of the state of any other node. Nodes that are neighbors on the grid link together to form clusters. Clusters represent connections between nodes in the lattice. Anything associated with the cluster can then flow to any node that belongs to the cluster. The probability at which the first spanning cluster appears is called critical threshold. Computer simulations of large but finite two dimensional square networks have estimated that the critical threshold for an infinite network is 0.593 (fig. 1). The G-LOC model was modeled using a 20x20 percolation network to model the connectivity in the CNS and the transitions between consciousness and unconsciousness.

2.3 Fuzzy Theory

Fuzzy theory attempts to describe realistic problems by allowing elements to belong, and to not belong to a group or set at the same time [16]. The two most important concepts of fuzzy theory are the fuzzy set and the membership function. These two concepts are similar and are used interchangeably. A fuzzy set is a collection of elements without a crisp or a clearly defined boundary distinguishing members and nonmembers. The membership function, on the other hand, is that function which indicates the degree of membership of an element to a set, the fuzzy set. The shape of the membership functions are arbitrary ranging from the simple straight line to nonlinear membership functions.

Fuzzy numbers are fuzzy sets defined over the set of real numbers [9]. A fuzzy system uses a collection of fuzzy numbers and rules to reason about data and make decisions. A fuzzy system consists of four parts [12]:

- Fuzzy Rule Base: This part contains the fuzzy rules of the system. Each rule has an antecedent (If part of the rule) and consequent part (then part of the rule).
- Fuzzification: In this part, the value for each input variable is applied to the set of rules. The antecedent of each rule is computed to obtain a single number called support of the rule. When a rule has a degr zero, this rule does not apply. If the degre say that the rule has fired, i.e. the rule is
- Inference: Consists of two parts: implic aggregation. On implication, the value from th degree of support is applied to the conseq results in assigning for each rule a fuzzy aggregation part, the fuzzy sets obtained fr are combined to obtain a single fuzzy set fo variable.
- Defuzzification: In this part of the fuzzy solution for each output variable is defuzzi converted back to a classical number.

3. PREVIOUS MODEL OVERVIEW

$+G_z$ - Induced Loss of Consciousness (G-LOC) i as the condition that results when humans are ex levels of acceleration$_z$ stress ($+G_z$) during airpla maneuvers and centrifuge experiments. After establishing a link between the loss of bloo unconsciousness, G-LOC was considered a conseque failure of the cardiovascular system to maintain flow to the central nervous system (CNS). The ne was not introduced until 1989 [15] suggested that unconsciousness is an active neurol mechanism triggered in response to a metabolic t simply a passive response to ischemia. The fir took into consideration the cardiovascular an mechanisms behind G-LOC was introduced in [2]. T created using percolation theory, was able to model dynamics of the induction of unconsciousne to cerebral ischemia. The model mapped th activating system (RAS) into a percolation net nodes represent a non-specific area of the CNS. T neurologic system responsible for the all-or-not consciousness. The model is based on several hyp some reasonable assumptions had to be made a behavior of neural tissue under acceleration reasonable assumptions were associated with b sensitivity to ischemia, and neural functional th The model views acceleration stress as the responsible for cerebral ischemia. When the bo acceleration stress, the cardiovascular resp overcome the downward pressure to maintain the b the brain. As acceleration increases, the abili supply adequate blow flow decreases. When blood hence the supply in oxygen and other substances system (neural cell) is halted, the oxygen rem tissues decreases at a rate that depends on the needs. If the oxygen concentration in the tiss certain level (neuronal functional threshold (representing an area of the brain), is assume participate in the RAS and is removed from the network. If the stress is severe enough, the n removed increases. This will break the comm pathway between the top and bottom part of th resulting in the induction of unconsciousness as [2]. The bottom row corresponds to brainstem formation and, the top row, to higher cortical ar

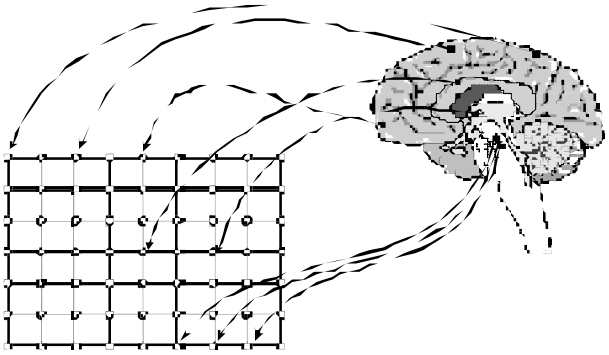

Figure 2. Mapping the RAS onto a percolation network

4. FUZZY MODEL CONSTRUCTION

Several changes were made to the crisp model in order to account for the uncertainty present in its parameters. These modifications are associated directly with the blood flow, or equivalently, the oxygen delivery, the neuronal functional threshold and the node state and, indirectly, with the metabolic rate or the oxygen use.

Oxygen Delivery: In the crisp model, the extent of ischemia for each of the nodes is determined by calculating the blood flow, first, by determining the eye level blood pressure in response to acceleration stress. This is accomplished by using an existing model developed by Gillingham [7]. Subsequently, the calculated pressure is distributed throughout the percolation network representing the cortex. Each node is assigned a local hydrostatic barrier chosen from a series of uniform distributions that span the brain stem to the higher cortex. In the crisp model, the mean blood pressure for the individual is compared with the local blood pressure at the node. The blood flow at each node is then individually determined to be either adequate (lower than the baseline, positive difference) or ischemic (higher than the baseline, negative difference). In the crisp case a drastic change occurs in blood flow from 100% (adequate) to 0% (ischemic) at the moment the local hydrostatic height exceeds the mean blood pressure at heart level (fig. 4).

In the fuzzy model, the assumption of constant blood flow is modified to account for the pulsatile blood flow that exists for pressure values between the dyastolic and systolic pressures. For hydrostatic barriers less than the dyastolic pressure, the blood flow is normal; for barriers above the systolic pressure, blood flow is zero, and for barriers between the two, blood flow is pulsatile. In order to characterize the blood flow between these two extreme cases, the area above the blood pressure pulse at each pressure is calculated and compared to the mean area of the pulse (fig. 3). There is a smooth transition from adequate to ischemic blood flow.

Oxygen Use: As the neural tissue becomes ischemic, the cells begin to use the available nutrients (oxygen) at a rate determined by the local metabolic needs. Each node in the network is assigned a local metabolic rate from a Gaussian distribution whose mean represents a global metabolic rate. The oxygen use curve is S-shaped and was determined based on in-vivo measurements of ischemic retinal t Ischemic nodes with higher metabolic rates w available oxygen at a faster rate than those repr the neural tissue with relatively low metabolic d

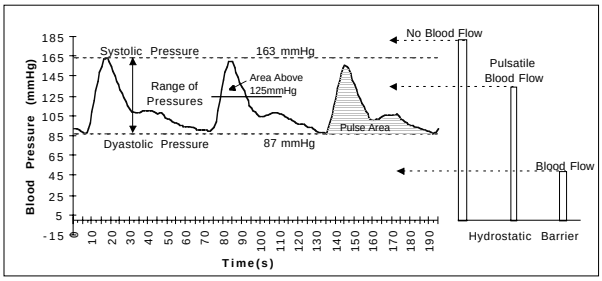

Figure 3 Blood Flow Types

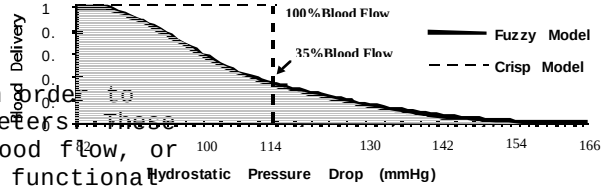

Figure 4. Blood flow (Oxygen Delivery) vs. press

Neuronal Functional Suppression Threshold: that the CNS is not homogeneously sensitive to h node is assigned a critical threshold from distribution. The value and distribution of suppression threshold is extracted from experimen malfunction of neurons under ischemic condition measured: electrical function is affected at app of normal cerebral blood flow, complete neurona failure occurs around 30% and release of potass death at approximately 10% [1]. In the crisp mo oxygen level in the node falls below the assi threshold (e.g. 30%), the node, representing an tissue, is suppressed (fig. 5). This area is ass participate in the RAS and the node is remove percolation network. This threshold, called lo suppression threshold (LFT), indicates the end normal neuronal function for the particular are i.e. the node no longer participates in the perc The precise value of this threshold is not know the uncertainty in determining the exact val thresholds and the fact that each node in the ne not only one neuron, but a nonspecific area of instantaneous change from proper function to no exactly one instant in time is not justified.

Fuzzy Model Development: In order to accoun uncertainty in the threshold, a fuzzy inferen created. The fuzzy node is proposed as the relationship of this fuzzy inference system. The final result determine the node state for the corresponding ar The intermediate states of the fuzzy node ac uncertainty in determining the functional state brain, where some neurons or structures are fu others are not. A fuzzy node indicates a node w state is continuous and not limited to the biva crisp node. In fact, for both models, the node

renormalization process for the state of an area of the brain. Each node is viewed as a macroscopic representation of an area which itself represents a group of smaller areas, which are also representing a group of smaller areas in cascade down to the neuronal level where, theoretically, each node represents the state of a single neuron (fig. 2, 6). Obviously, in practice it is not possible to represent each neuron or to know which neuron is operational and contributes to the RAS.

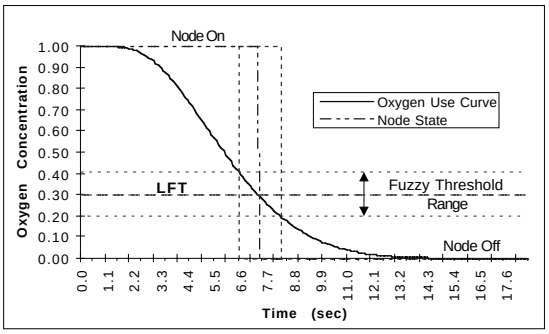

Figure 5 Fuzzy Threshold Range and Node State

The fuzzy node therefore represents the state of areas at lower scales through a cascade of percolation networks whose final occupation probability value is given by the value of the fuzzy node. A value of '1' means all the areas are operational, a '0' indicates that none are functional, while intermediary values indicate that some parts are operational. The value of the fuzzy node is not static; it changes as the oxygen concentration approaches the functional suppression threshold and when this threshold is exceeded. The range of oxygen concentration values over which the fuzzy node varies is referred to as the fuzzy threshold region (fig. 5).

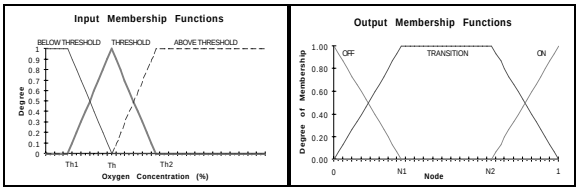

Figure 6 Membership Functions

The fuzzy node, i.e. the input-output relationship of the fuzzy system, depends on five parameters describing the triangular and trapezoidal input (N1, N2) and output (Th1, Th, Th2) membership functions (fig. 6). The value of these parameters are not known a priori and must be determined. To do so, the model was exercised with fuzzy nodes to study the effects of the fuzzy system parameters (Table 1). The use of fuzzy nodes results in a fuzzy percolation network. In contrast with the crisp percolation network, where the binary nature of the nodes clearly defines the clusters, in the fuzzy percolation network, such definition does not exist. In fact, there is no theory dealing with a fuzzy percolation network. It is however possible to converge to a decision using a rule which maps the fuzzy percolation network onto a classic percolation network.

In conventional percolation theory, the probability of percolation can be determined if the node occupation probability is known. For an infinite network, if the occupation probability is above the critical threshold, the probability of percolation is 1, if it is below the criticality probability is 0 (fig. 1). This is also the ru which relates the percolation network onto the classic networ shows that for an infinite network is 0.593 [13]. We propose to map the fuzzy percolation network to a classic network in order to determine if a cluster exists, i.e. if the network is percolati the individual is conscious. Otherwise, loss of

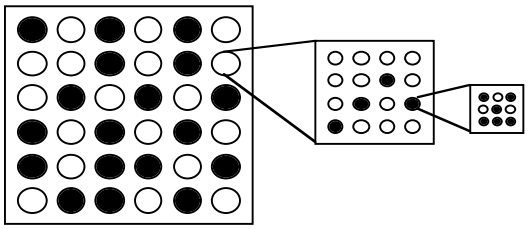

Figure 7. Cascade of Percolation Networks

At each time increment, the fuzzy percolation mapped to a classic network in order to determine cluster exists, i.e. if the network is percolati the individual is conscious. Otherwise, loss of results otherwise.

The effect of the fuzzy system parameters on where studied when blood flow to the node is ab [13]. This condition was chosen to avoid contri other factors on the time to loss consciousnes differential blood perfusion to the brain. Th simulation showed that the time for a fuzzy node from the percolation network was almost the sam crisp case. The maximum time difference between t and crisp case was 0.6 seconds. In modern centr the recognition of unconsciousness by a central been shown to have a variability of +/- 2 s Therefore, the 0.6 seconds variation is well measurement resolution for human experiment Montecarlo simulation uncovered the relationship fuzzy system parameters, the fuzzy node, and the of a fuzzy node from the percolation network. T did not however determine the fuzzy threshold ran nor the values for the output parameters of the f

In order to determine the fuzzy threshold ran neuronal ischemia study was used as reference tests were performed for which the input thresho of the fuzzy system were fixed to the Astrup ran Th2=40%) for all the nodes or were normally di Th, Th2) each node as determined by the local function (LFT-0.2, LFT+0.1). After several tests of th so, the network the final configuration was determined (

Table 1. Final Configuration for the Fuzzy N

OUTPUT FUZZY PARAMETERS	
N1 and N2	Uniform Distribution [0,1]
INPUT FUZZY PARAMETERS	
Threshold 1 = Th1	LFT –0.2
Threshold = Th = LFT	Normal Distribution (0.3, 0.05)
Threshold 2 = Th2	LFT +0.1

Testing of the Model. The model was tested using same protocols as three of the historical human e in the centrifuge ('Acute Arrest' [13], 'Pensa Beckman [2] studies). The 'Acute Arrest' study

time to loss consciousness in humans when cerebral circulation was abruptly halted. In the 'Pensacola' study, presumably were exposed to the following acceleration stress profiles under identical experimental conditions [5]: each run started at a peak G_z held for 10 sec. If there were no symptoms reported during the run, the peak G level for the next exposure was increased by 0.5 G_z. If G-level or blackout did occur, the peak G_z of successive runs was increased by increments of 0.3 G_z until unconsciousness occurred. Both the squared test showed that for the 'Acute Arrest' and studies, the probabilities that the differences between the models' and the human experiment distributions are due to random fluctuations are respectively, 90%, 80% (fuzzy) and 80%, 85% (crisp) (fig. 8, 9).

In the Beckman study [2], a set of profiles were developed to analyze the loss of consciousness induction times associated with very high onset rates to high levels of G_z. The experimental protocol starts with a rapid onset to peak G with the duration at peak G set to zero seconds. The duration was increased in 0.3 seconds steps until a loss of consciousness occurred. For the Beckman study, both models show a good correlation to the human data, with R^2 = 0.961 (fuzzy) and 0.954 (crisp). These results show that both models were able to duplicate human studies with a high degree of fidelity.

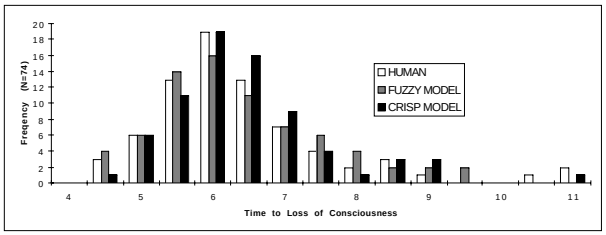

Figure 8 Model Results from Acute Arrest Test

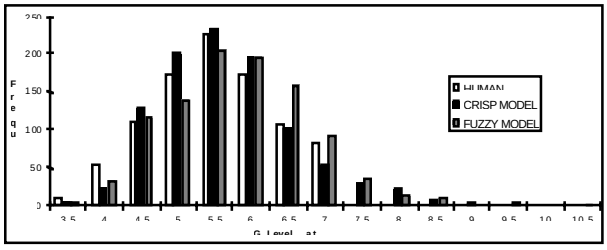

Figure 9 Models Result Pensacola Study

5. CONCLUSION

This article describes a fuzzy extension of the percolation model, or crisp model, of the reticular activating system under acceleration stress [3]. The percolation network was used to represent the connectivity of the reticular activating system (RAS) where each of the nodes represents a non-specific area of the cephalic nervous system parameterized by blood flow, sensitivity to ischemia and neuronal functional threshold. The bivalent characteristic of the oxygen delivery (blood flow) and the node state functions in the previous crisp model were changed to a continuous multivalent characteristic. These modifications improved the previous model design by introducing a more realistic blood flow dependence on the local perfusion pressure, and by accounting for the presence of the local functional suppression threshold. However, the addition of the fuzzy node did not significant difference in the global response (T for the models. We also postulate that the interac the local elements in the percolation network ca small variations existing between the crisp and fuzzy models replicated the human studies with a h Additionally, based on these observations, we propos knowledge of the physiological parameters are not nec modeling a complex system, since slight variation dynamics do not significantly alter the glob is essential, however, to model the interplay a elements [10].

6. REFERENCES

[1] Astrup J., Symon L, Branston NM and Lassen NA. Viable Potential and Extracellular K^+ and H^+ at Criti Brain Ischemia. Stroke 8(1):51,1977 [2] Beckman EL, Duane TD, Ziegler JE and Hunter H. Some Observations on Human To Acceleration Stress. Aviat. Space Environ. Med. 25:561-66, 1954. [3] Camarota J.P., A Dynamic Percolation Model of the cer System Under Acceleration Stress (+Gz) Induced Ische Stress Ph.D. Thesis, Drexel University, Philadelphi Cammarota J.P. and Onaral B.,``State Transitions in Systems: A Complexity Model for Loss of Consciousne IEEE Transactions on Biomedical Engineering, 45(8):1017-1023,1998. Cochran LB, Gard PW and Norsworthy ME. Variations in Tolerance to Positive Acceleration. USNSAM-001 059.02.10:1-13, 1954. [6] Forrest S. Emergent Computation: Self-Or Collective, and Cooperative Phenomena in Natural and Computing Networks. Physica 42 D 1990. [7] Gillingham K Freeman J.J. and McNee R.C. Transfer Functions for Eye Pressure During +Gz Stress. Aviat. Space Enviorn. Med. 48(11):1026, 1977 [8] Houghton J.O., McBride D.K. and Hannah K., `` and Physiological Effects of Acceleration-Induced (+ Consciousness. Aviat. Space Environ. 56:956, 1985. [9] Klir G.J. and Yuan B., Fuzzy Sets and Fuzzy Logic, Theory and Appl New Jersey: Prentice-Hall, 1995. [10] Luzuriaga E., Fuzzy Percolation Model for Loss of Consciousness Under A Stress Master Thesis, Drexel University, Philadelphia Onaral B. and Cammarota J.P.,``Complexity, Scaling, an Biomedical Signals.''in: CRC Handbook of Biomedical E J.Bronzino, Ed. Boca Raton, Fl: CRC, 933-944, 1995. [12] and Gulley N., Fuzzy Logic Toolbox. Massachusetts: The MathWorks Inc., 1995. [13] Rosen R, Kabat R and Anderson JP. Acute A the Cerebral Circulation. Arch. Neurol. Psychiat. 50:510-528, 1943 [14] Stauffer D., Introduction to Percolation Theo Taylor \& Francis Ltd, 1985. [15] Stoner J.E. Observations on Neurophysiologic Theory of Acceleration (+Gz) Induce Consciousness. Aviat Space Environ. Med. 59:589-598, 1988. [16] Zadeh L.A. A Fuzzy Set Interpretation of Linguistic He Cybernetics 2(3):4, 1972. [17] Zuckerman R. Effects of Br Mixtures on the Spatial and Temporal Distribution of O of Retinal Tissues During Hypoxia/Ischemia. NAWCADWAR 0215:1, 1992.

Acknowledgements: Valuable contributions of Dr. Hrebi H.H. Sun are gratefully acknowledged. This work has be in part by the ONR-NAWC