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MENTATION PAGE

Form Approved
OMB No. 0704-0188

1a. **AD-A201 434** C	1b. RESTRICTIVE MARKINGS OTIC FILE COPY
2a.	3. DISTRIBUTION / AVAILABILITY OF REPORT
2b. DECLASSIFICATION / DOWNGRADING SCHEDULE DEC 15 1988	

4. PERFORMING ORGANIZATION REPORT NUMBER(S)	5. MONITORING ORGANIZATION REPORT NUMBER(S) AFOSR·TR· 88-1295

6a. NAME OF PERFORMING ORGANIZATION Marine Biological Labs	6b. OFFICE SYMBOL (If applicable)	7a. NAME OF MONITORING ORGANIZATION AFOSR/NL
6c. ADDRESS (City, State, and ZIP Code) Woods Hole, MA 02543		7b. ADDRESS (City, State, and ZIP Code) Bldg. 410 Bolling AFB, DC 20332-6448

8a. NAME OF FUNDING / SPONSORING ORGANIZATION AFOSR	8b. OFFICE SYMBOL (If applicable) NL	9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER AFOSR-88-0293

8c. ADDRESS (City, State, and ZIP Code) Bldg. 410 Bolling AFB, DC 20332-6448	10. SOURCE OF FUNDING NUMBERS			

	PROGRAM ELEMENT NO.	PROJECT NO.	TASK NO	WORK UNIT ACCESSION NO.
	61102F	2313	A5	

11. TITLE (Include Security Classification)

Methods in Computational Neuroscience Summer 1988 Marine Biological Laboratory

12. PERSONAL AUTHOR(S)

James M. Bower, Christof Koch

13a. TYPE OF REPORT Final	13b. TIME COVERED FROM 1 Aug 88 TO 31 Jul 89	14. DATE OF REPORT (Year, Month, Day) 11/88	15. PAGE COUNT 13

16. SUPPLEMENTARY NOTATION

17. COSATI CODES			18. SUBJECT TERMS (Continue on reverse if necessary and identify by block number)
FIELD	GROUP	SUB-GROUP	

19. ABSTRACT (Continue on reverse if necessary and identify by block number)

This three week course was offered for the first time at the Marine Biological Laboratory in Woods Hole, MA from August 14 to September 3. Twenty students were selected from an applicant pool of 105. Students who were prepared in both computer science and neuroscience were selected because thye could benefit immediately from the high level of discussion without training in either area. One half of the students in the course were not residents of the U.S.A. The course had two lectures per day plus a laboratory wherein students worked with GENESIS, the simulation software developed at Caltech. The last week of the course was a concurrent workshop on "Computational Neuroscience" run by Terry Sejnowksi

20. DISTRIBUTION / AVAILABILITY OF ABSTRACT ☐ UNCLASSIFIED/UNLIMITED ☐ SAME AS RPT. ☐ DTIC USERS	21. ABSTRACT SECURITY CLASSIFICATION
22a. NAME OF RESPONSIBLE INDIVIDUAL John F. Tangney	22b. TELEPHONE (Include Area Code) (202) 767-5021	22c. OFFICE SYMBOL NL

DD Form 1473, JUN 86   Previous editions are obsolete.   SECURITY CLASSIFICATION OF THIS PAGE

88   10   032   UNCLASSIFIED

METHODS IN COMPUTATIONAL NEUROSCIENCE

August 6 - September 2, 1989

This course is for advanced graduate students and postdoctoral fellows in neurobiology, physics, electrical engineering, computer science and psychology with an interest in "Computational neuroscience". A background in programming (preferably in C or PASCAL) is highly desirable. The course is limited to 20 students.

This four-week course presents the basic techniques necessary to study single cells and neural networks from a computational point of view, emphasizing their possible function in information processing. The aim is to enable participants to simulate the functional properties of their particular system of study and to understand the advantages and pitfalls of this approach to understanding the nervous system.

The first section will focus on simulating the electrical properties of single neurons (compartmental models, active currents, interactions between synapses, calcium dynamics). The second part of the course will deal with the numerical and graphical techniques necessary for modeling neuronal networks. Examples of such simulations will be drawn from the invertebrate and vertebrate literature (visual system of the fly, learning in Hermissenda, mammalian olfactory and visual cortex). In the final section, connectionist neural networks relevant to perception and learning in the mammalian cortex, as well as learning algorithms (e.g. back-propagation) will be analyzed and discussed from a neurobiological point of view.

The course includes two daily lectures and labs. The lab section is organized around GENESIS, the Neuronal Network simulator developed at Caltech, running on 20 state-of-the-art, single-user, graphic color workstations provided by SUN Microsystems. Students are expected to work on a simulation project of their own choosing.

Co-Directors: James M. Bower and Christof Koch, Computation and Neural System Program, California Institute of Technology.

Visiting Faculty: Paul Adams, Stony Brooks; Dan Alkon, NIH; Rudolfo Llinas, NYU. John Hildebrand, Arizona; John Hopfield, Caltech; John Rinzel, NIH; David Rumelhart, Stanford; Idan Segev, Jerusalem; Terry Sejnowski, Johns Hopkins; David van Essen, Caltech; and others.

METHODS IN COMPUTATIONAL NEUROSCIENCE

SUMMER 1988

MARINE BIOLOGICAL LABORATORY

James M. Bower and Christof Koch, Co-Directors
Computation and Neural Systems Program
California Institute of Technology
Pasadena, California 91125

This three week course was offered for the first time at the Marine Biological Laboratory in Woods Hole, MA from August 14 to September 3.

Twenty students were selected from an applicant pool of 105. Students who were prepared in both computer science and neuroscience were selected because they could benefit immediately from the high level of discussion without training in either area. One half of the students in the course were not residents of the U.S.A.

The course had two lectures per day plus a laboratory wherein students worked with GENESIS, the simulation software developed at Caltech. The last week of the course was a concurrent workshop on "Computational Neuroscience" run by Terry Sejnowksi.

Faculty and students were very enthusiastic about the course, about the quality of the lectures, the computers and software, and especially with the three course assistants from Caltech and their devotion to the course. The merits and drawbacks of the simulation software will not be reviewed here. Even with this endorsement there is room for improvement and fine-tuning.

Based on suggestions offered by the twenty students, we will make some changes.

The course will be expanded to four weeks in 1989 in order to accommodate the large amount of reading, lectures and laboratory work, and to provide time for more informal discussions and longer student simulation projects. Two lectures will be held daily, each 90 minutes long.

Emphasis in several areas of the curriculum will be changed: More information will be given on the mathematical aspect of modeling (partial differential equations, volterra equations, numerical methods) by increasing the number of small, informal group seminars on these technical matters. We plan to expand the number of talks in the area of high-level connectionist neural networks. We also plan to redefine the overlap between our course and Sejnowski's workshop.

SCIENTIFIC RESEARCH (AFSC)
TO DTIC
been reviewed and is
IAW AFR 190-12.

Division

On the hardware side, we expect to expand the number of SUN Workstations to 20 single-user, graphic color machines.

The problem of handouts with equations and bibliography will be solved by the December 1988 publication of our <u>Methods in Neuronal Modeling</u>, MIT Press. A substantial number of faculty have contributed chapters toward this book (Segev, Koch, Adams, Bower, Nelson, Sejnowski, Getting). Students from 1988 will be notified that the book is available, and students in subsequent years will all have a copy when they enroll.

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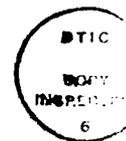
STUDENT PROJECTS

METHODS IN COMPUTATIONAL NEUROSCIENCE

MARINE BIOLOGICAL LABORATORY

AUGUST 14 TO SEPTEMBER 3, 1988

Werner Backhaus
Free University of Berlin
West Germany

Some aspects of motion detection in the visual system of the blowfly Calliphora including receptor cells, simple version of the correlation-model, and a small compartmental circuit for the H1-neuron.

Jim Cummings
Neuroscience Graduate Group
University of Pennsylvania, Philadelphia

The superior colliculus (SC) has been implicated in the generation of saccadic eye movements, which are the quick eye movements with which we fixate our center of gaze. For my course project I choose to implement Van Gisbergen et al. (1986) model of population coding of saccades in the SC. In the following two weeks I was able to implement the model and get it to generate accurate saccades. I was not able to test the model in any detail. I plan to further refine this model and use it as a teaching example of the simulator and of how neuronal information could be coded within a population of neurons.

Allan Dobbins
Computer Vision and Robotics Laboratory
Dept. of Electrical Engineering
McGill University
Montreal, Canada

A single compartment model containing several different voltage and time dependent conductances was examined. The goal was to study the contribution of the smaller, "silent" conductances and their effect on the pattern of action potential firing.

Ed Kairiss
Dept. of Psychology
Yale University
Box 11A Yale Station
New Haven, CT

As a first step towards developing biophysically realistic network models of hippocampal subsystems, a model of single CA3 and CA1 pyramidal neurons was constructed with GENESIS. Biophysical and electrophysiological data were used to develop an equivalent compartmental representation, and implemented a 5-compartment model. Dendritic synaptic input was provided from simplified granule cells. A hebbian synaptic function was written in C and added to the simulation, and the response of the neuron to patterned input was investigated. The model provides the basis for examining the adaptive behavior of hippocampal networks. GENESIS provides a powerful means for investigating the effects of changes in membrane- and synaptic- level parameters on network behavior.

A. B. Kirillov
Research Computer Centre
U.S.S.R. Academy of Sciences
Pushchino, Moscow Region

Project: study the effect of excitatory lateral interactions on orientation selectivity of neurons in visual cortex.

I had to expand the existing Visual Cortex (VC) simulator adding one more network with excitatory connections, then explore the properties of the expanded model. Unfortunately only the first part (programming) was finished. I reprogrammed most of the computational part of the VC. The reason is that current version of VC requires a lot of memory, sometimes resulting in the memory allocation failure. And expanding it more requires even more memory.

The source code of the VC program is only briefly commented and not easy to understand. That is why I spent quite a time trying to find how to rewrite it. I used simple linear lists to connect cells and replaced the synaptic alpha function by difference of exponents. With this type of alpha function there is no need to store all the previous spikes. The program now is more compact, runs a bit faster and it is easier to implement new features in it.

It is sad that I only began to play with the program. However, it was very useful for me to understand in detail C. Koch's model of visual cortex, and the principles of deriving model from experimental data. It was also interesting to see how the model is programmed and do some programming for this type of models.

Guy Major
Dept. of Physiology
University of Oxford
United Kingdom

Modelling dendritic trees of cortical pyramidal cells: active and passive compartmental models.

I only got as far as simulating various ionic conductaries (which will be useful later on in detailed "active" compartmental models) and stringing some of them together in linear cable structures, extending a pre-existing Hodgkin-Huxley demo. In order to simulate Ca^{2+}-sensitive conductances I needed to write additional segment evaluation functions for GENESIS and some new structures.

a) To introduce Ca^{2+}-dependency into the rate constants of the Hodgkin-Huxley-like eqns.

b) To simulate (first order) Ca^{2+} clearance from the "cell.

I also introduced a more complicated voltage-dependent conductance type (c) and a simpler one (d) - it would be nice to use the simplest form that describes experimental data. I "succeeded" in implementing "intrinsic bursting" - using Traub's (1982) parameters. I also implemented anomalous rectification using Schwindt et al (1985) and the M-current and A-current (Bullfrog values) which could be used to produce spike-frequency adaptation, together with a persistent gNa (again Stafstrom et al, wild guesses for parameters) and a slow calcium activated potassium conductance.

Yair Manor
Hebrew University
Israel

The subject I worked on for the course project is simulation of action potential
propagation in highly branching myelinated and de-myelinated axons. I modeled
the membrane conductance using Hodgkin and Huxley's sodium and potassium
channels. The questions I wish to ask and answer with this program where:
1. Is there a correspondance between optimal dimensions of nodes and internodes
for conduction velocity in the simulated model and in the observed values in
nature. 2. How is myelin affecting delays at bifurcation points, for different
geometrical ratios? 3. Will action potentials fail at the junction of a
myelinated section and a demyelinated axon, as expected theoretically?

Hiroyoshi Miyakawa
NY Medical College
Using GENESIS I've built a three compartmental model of a single neuron,
one for soma and two for dendrites. I put several types of ion channels into
membranes of this model. In addition to fast Na and delayed rectifier K
channel, I put 2 types of T type voltage dependent Ca channel, and Ca^{2+} and V
dependent K channel. Descriptions of the 2 Ca channel were fitted to whole cell
V-clamp data given by experimental values from the literature. Paul Adams'
lecture and the conductance building party were big helps for me to do so.
Although my description seems to fit well to experimental V-clamp data there are
other problems to reconstruct proper shape of Ca spike. Which Ca channel use,
which channel to use, what ratio of G_{cu}/G_K to use, E_{ca}, Eleak, Capacitance, etc.
etc.
So far I've tried delayed rectifier K channel as a repolarizing current.
In this case, fast Ca channel seems to have too fast activation kinetics and
Hayiwara type seems to have too slow activation kinetics. Because my model also
takes Ca^{2+} buffering into account I hope someday I will be able to test the
validity of the model by comparing Ca concentration output with experimental Ca
transient data which I've been working on by using optical dyes.

Barbara Moore
Artificial Intelligence Laboratory
MIT, Cambridge

Project: modelling the marine nucli branch Aplysia's habituation,
sensitization, and classical conditioning with presynaptic facilitation.
The first-order explanations for presynaptic facilitation involve Ca^{++}
concentration; phenomena I'll try to model include the factors affecting
intracellular calcium concentration.

The questions I'll be exploring involve a characterization of interactions
between factors affecting $[Ca^{++}]$.

Laura Reece
Dept. of Physiology and Biophysics
University of Washington, Seattle

Most of my work with the simulator GENESIS involved several evolving tutorials, two-cell and multi-layer during the first two weeks and the pyriform cortex model when it appeared during the last few days. Using scripts from those tutorials in order to gain familiarity with use of the system, I began building a basic circuit intrinsic to the hippocampus. The circuit included a CA1 pyramidal cell and two inhibitory interneurons, one feed-forward, one feedback. Using known parameters for those cells from electro-physiology studies, I attempted to build up a family of groups of these cells in order to examine output from the pyramidal cell when all the cells were included in a network. This work was facilitated, especially in the use of the connection specifications, when the pyriform cortex model appeared. The simulation (or various versions of it) on which I was working never worked entirely, but it will provide a beginning for more work when I get home.

Clay Reid
Rockefeller University, New York

I spent most of my time learning how to use GENESIS rather than implementing a model of directional selectivity in cat striate cortex. By the end, I had modified the piriform cortex in a few ways to approximate the geniculo-cortical pathway. The geniculate system was modelled simply as units whose instantaneous firing frequency was a specifiable function of time, since their output can be quite well modelled using the "black-box" approach. The cortex consisted of two populations of cells, excitatory spiny stellate cells and inhibitory smooth stellate cells, both of which are found in the input layer 4. Directional selectivity was modelled to first approximation simply by having these two populations spatially offset in terms of retinotopic coordinates.

John Rose
Dept. of Computer Science,
SUNY at Stony Brook

My project involved changing the formalism used to model the calcium dependency of I_C, the noninactivating voltage and calcium dependent potassium conductance, in the bullfrog sympathetic ganglion cell simulator. The formalism employed by Yamada, Koch and Adams, while exhibiting a calcium dependency, is unrealistically simple. The basic scheme is:

where C indicates the closed state of the channel and O indicates the open state. In this formalism, the forward rate constant f is voltage and calcium dependent while the reverse rote constant b is only voltage dependent.

The formalism I implemented (Moczydlowski & Latorre, J. Gen Physiol $\underline{82}$, 511 (1983))

is a more plausible formalism and specifies forward and reverse rates constants which are dependent on both calcium concentration and voltage.

Comparisons of voltage clamp simulations with no external Na and raised external K of both formalism with experimental data indicate that the rise of the total current at the time of depolarization is more accurate with the complex formalism than with the simple formalism. Analysis of tail currents, unfortunately, indicates that the rate constant of I_C falls somewhere between the two formalisms. Long current clamp simulations (1 sec) in which I_{AHP} and I_M are poisoned in all possible permutations do not reveal any significant difference in action potential firing rate between simulations employing the two formalisms, although simulations employing the second formalism do show a somewhat higher firing rate when I_M or I_M and I_{AHP} are poisoned.

Franklin Schuling
University of Groningen
Biophysics Department
The Netherlands

A structural model of the directional-selective motion-sensitive circuitry in the fly visual system.

In a first attempt to model both behavioral and electrophysiological results obtained from detailed experiments for this visual system, the neural network simulator GENESIS has been used during the Computational methods in Neuroscience course to design a structural network which should incorporate the gross anatomical and physiological functions and characteristics available. The network design consists of four layers. Each layer is modeled as a two-dimensional layer of 9 x 9 identical neural elements. A two-dimensional stimulus pattern (for instance a moving bar) is presented in a stepwise manner to the input receptor layer of the network. The photoreceptor properties are modeled according to reported electrophysiological data. Time- constants and receptor membrane properties are taken from literature. These receptors are connected via inhibitory synapses in a retinotopical manner to the layer of the Large Monopolar Cells. The model behavior of these LMC's are in good agreement with experimental data. Next, a biophysical implementation of the correlation scheme, originally suggested by Reichardt and Hassenstein (1956), is adapted from the scheme proposed by Torre, and Poggio (1978). These neurons are located in the third layer, where each cell is supposed to represent a small patch of the dendritic tree of the motion-sensitive H1-neuron. Finally, the H1 neuron integrates the activity of all individual direction-selective elements and generates an actionpotential whenever a certain threshold value is exceeded. The general idea is that in response to an ongoing movement in the preferred direction the neuron fires with an increased firing rate (that is, compared with the spontaneous rate) and that it ceases to fire for a stimulus moving in the opposite direction. So far, the parameter settings are sufficient to induce stable responses to intensity flashes. The directionality is a matter of future modeling effort.

Jeff Segall
Max Planck Institute for Biochemistry
Martinsried, West Germany

My project was to begin modelling amoeboid movement, using the GENESIS system. My approach was to describe a cell as a 2 dimensional object bounded by a membrane. The membrane was divided into 100 compartments, each described as a spring with a resting length, in parallel with damping. Both damping and an intracellular pressure were necessary to avoid instabilities. Even though only a few ranges of spring constants (which vary with time to simulate movement) have been tried, the modelling suggests that the simple ideas I had regarding the generation of motion in amoeba are not sufficient. Unfortunately, there was not enough time to experiment with more complex models. This work will continue back at my lab.

Bill Skaggs
University of Colorado, Boulder

I decided to try to set up a neural circuit that could use inhibitory feedback to switch excitation between different pools of cells; the circuit was intended to help me explore some ideas about how the basal ganglia operate.

The system I set up consisted of four groups of units, which I called, for heuristic reasons, "cortex", "striatum", "globus pallidus", and "substantia nigra". The units were "integrate and fire" neurons, with no time delays, each spike giving rise to an alpha-function conductance transient in all post-synaptic cells. The "cortex" contained fifty cells, divided into five pools of ten cells each. Every cortical cell was connected to all other members of its pool, but to no other cortical cells, by excitatory synapses. The "striatum" also contained fifty cells, each one receiving excitatory input from exactly one cortical cell. The "globus pallidus" contained ten cells, each one receiving inhibitory input from every striatal cell, and projecting excitatory input to every cortical cell. The "substantia nigra" contained five cells, each one sending an inhibitory projection to every striatal cell. This connection scheme reflects the anatomical arrangement except that the path from the globus pallidus to the cortical actually goes through several intermediate areas.

With the network set up, I tried to arrange the parameters (of which there were essentially nine: five connection weights and four background stimulus intensities) so that only one cortical pool at any time could be active, and so that giving a sufficient stimulus to a different pool could reduce the pallidal excitation enough to switch off the currently active pool and allow the stimulated pool to take over. After a substantial amount of fiddling I succeeded in making it work, though the parameters had to be tuned quite precisely to give good results.

My original intent in setting up this simulation was to make "lesions" in the model corresponding to the pathologies of Parkinson's disease and Huntington's disease, and then see how the model's behavior changed. The changes I see do indeed correspond, at least in a somewhat metaphorical sense, to the deficits seen in the two diseases, but I am not sure how much this means. The simulation has considerably weakened my faith in the theorized mechanism of basal ganglia-switching on which it was based: this sort of switching via dis-excitatory feedback seems just too slow and unreliable to be effective.

Brian H. Smith
University of Arizona

Locust Jump

The locust jump reflex, elicited during escape behavior and the initiation of flight, is typical of the jump of many invertebrates. The jump is initiated by a period during which the flexor and extensor muscles co-contract, thereby deforming the rigid leg cuticle in which the power for the jump is stored. Initial contraction is facilitated by descending visual, auditory, and tactile stimuli. Deformation of the cuticle exerts propioceptive input to the flexor and extensor motor neurons, causing more intensive contraction. The same sensory inputs excites an interneuron that inhibits the flexer muscle. This interneuron has a high voltage threshold, and a single spike wil_ cause the flexor to relax and release the jump. During the three week course I was able to assemble a circuit constructing the basic elements in the jump central pattern generator (CPG), and, through manipulation of several parameters, the behavior of this model approximates that of the biological system. In the near future I expect to elaborate this basic model to investigate the switch between walking, when the muscles contract antagonistically, and jumping, as well as how the jump CPG is linked to the flight CPG, which has also been extensively documented.

Xias-Jing Wang
Department of Physiology-Anatomy
University of California at Berkeley

Project: Modeling corticothalanic relay cell in view to understand the generation of oscillatory rhythms(s) in terms of dynamics of different ionic channels (Na, Ca, K).

Donald Weir
University Laboratory of Physiology
Parks Road, Oxford, OX1 3PT
England

My project involved modelling some of the mechanisms that may underlie the vestibulo-ocular reflex. This reflex maintains a stable image on the retina in the face of head and body movements, thus allowing high visual acuity. The long term aim is to const_ain the model both by the nature of the problems solved by the reflex (e.g. 3D co-ordinate transform, high speed accurate computation, adaptation) and by experimental observations from the biological system. Using the library facilities at MBL the representation of information at the input (semi-circular canals) and output (oculomotor neurons) was obtained. An attempt was then made to produce a network model with the simplest rchitecture that was consistent with the known, gross anatomy. A gradient descent learning algorithm was used to ascertain whether a network with this architecture could make the transformation between the input and output representations. It was shown that the simple architecture could only carry out some of the computations required by the reflex. In addition, some time was spent assessing whether a structural model of the reflex could be produced easily with GENESIS. It was concluded that this could be done. I intend to carry out this further modelling exercise, once I have completed my analysis of what components are necessary for the network model to mimic the observed behavior.

Haggith Zabrodsky
Comp. Science Dept.
Hebrew University
Jerusalem, Israel

Modeling nonlinear membrane in a branching dendrite.

Studying the variance in attenuation of voltage-current injected at a terminal site, as "recorded" in the soma when membrane resistance is changed.

Membrane resistance as constant value and membrane resistance as a function of the local voltage value.

If time allows, also displaying the electrical length changes due to the resistant changes.

LECTURE SCHEDULE

METHODS IN COMPUTATIONAL NEUROSCIENCE

Course Directors: James Bower and Christof Koch, Divisions of Biology and
Engineering and Applied Science, California Institute of Technology

Lectures will be held in the Whitman Auditorium, except on August 22, 23
and 24 lectures will be held in Loeb 32

Monday, August 15
9:15 a.m.   JAMES BOWER
                             Aims of the course; methods, requirements
                             CHRISTOF KOCH
                             Introduction to Computational Neuroscience

11:00 a.m.   IDAN SEGEV
                             Introduction into cable theory, Rall's model of
                             neurons, d-3/2 law

Tuesday, August 16
9:15 a.m.   CHRISTOF KOCH
                             Synaptic input; nonlinear interaction between
                             synaptic input; Shunting inhibition and AND-NOT
                             gating

11:00 a.m.   IDAN SEGEV
                             Compartmental model of neurons
                             The use of electrical circuit simulators; SPICE;
                             Simulating alpha-motoneurons

Wednesday, August 17
9:15 a.m.   PAUL ADAMS
                             Voltage-dependent channels, Hodgkins-Huxley model

11:00 a.m.   IDAN SEGEV
                             Dendritic spines: anatomy, passive models; spines
                             and plasticity; spines and active currents;
                             calcium dynamics in spines

Thursday, August 18
9:15 a.m.   CHRISTOF KOCH
                             Solving nonlinear parabolic differential equations
                             (cable equation); Numerical methods

11:00 a.m.   RUDOLFO LLINAS
                             Bursting and oscillating cells: Neurons in the
                             mammalian thalamus, inferior olive and cerebellum

Friday, August 19
9:15 a.m.   PAUL ADAMS
                             Calcium dynamics,. and calcium dependent currents
                             A typical vertebrate neuron: bullfrog sympathetic
                             ganglion cells

11:00 a.m.   MARK NELSON
                             Modeling neural networks on parallel computers

2:30 p.m.   JOHN HILDEBRAND
                             Introduction to invertebrate nervous systems

Lectures will be held in the Whitman Auditorium, except on August 22, 23 and 24 the lectures will be in Loeb 32

Monday, August 22
9:15 a.m.   EVE MARDER
                   Modulation of pacemaker neurons

11:00 a.m.   JOHN MILDEBRAND
                   The insect olfactory system

2:30 p.m.   JOHN RINZEL
                   Phase space analysis of Hodgkin-Huxley like
                   systems; Bonhoffer and van der Pol; theory of
                   dynamical systems

Tuesday, August 23
9:15 a.m.   DAN ALKON
                   Neural networks which mediate associative learning

11:00 a.m.   JOHN RINZEL
                   Phase space dynamics of bursting neurons

2:30 p.m.   EVE MARDER
                   Modulation of oscillatory networks

Wednesday, August 24
9:15 a.m.   JAMES BOWER
                   The mammalian olfactory system

11:00 a.m.   CHRISTOF KOCH
                   The motion system in insects: correlation model

2:30 p.m.   CHRISTOF KOCH
                   Computational vision: detecting edges; Marr &
                   Hildredth; scale space; psychophysics and
                   physiology

Thursday, August 25
9:15 a.m.   JAMES BOWER
                   Large scale simulations of mammalian cortex; how
                   can such models be experimentally verified;
                   multielectrode recordings

11:00 a.m.   JOHN HOPFIELD
                   Small nervous systems and analytical approaches;
                   Lateral inhibition in Limulus

Friday, August 26
9:15 a.m.   RUDOLFO LLINAS
                   From single cells to network properties:
                   inferior olive and the cerebellum

11:00 a.m.   CHRISTOF KOCH
                   Large scale simulations of cat visual system;
                   Orientation selectivity

Lectures will be held in the Whitman Auditorium, except August 22, 23 and 24 lectures will be held in Loeb 32.

Monday, August 29

9:00 a.m.   JAMES BOWER
                             Integrative systems; the cerebellum

10:45 a.m.   ROGER TRAUB
                             Modeling the hippocampus; epileptic behavior

4:00 p.m.   RICHARD ANDERSEN
                             Introduction to Eye Movements

Tuesday, August 30

9:00 a.m.   STEPHEN LISBERGER
                             The oculo-motor system

10:45 a.m.   THOMAS ANASTASIO
                             Wiring the oculo-motor system using back-
                             propagation

4:00 p.m.   DAVID VAN ESSEN
                             Introduction to the Mammalian Visual System

Wednesday, August 31

9:00 a.m.   THOMAS ALBRIGHT
                             Motion processing in area MT

10:45 a.m.   CHRISTOF KOCH
                             Modelling motion in primates: gradient model

4:00 p.m.   CHRISTOF KOCH
                             Detecting figure-ground in the fly; experiments
                             and model

Thursday, September 1

9:00 a.m.   V.S. RAMACHANDRAN
                             Visual perception and psychophysics

10:45 a.m.   NICOS LOGOTHETIS
                             Color perception in the primate's visual system

4:00 p.m.   JOHN HOPFIELD
                             Studying neuronal dynamics; Associative memory;
                             minimizing variational functionals in networks

Friday, September 2

9:00 a.m.   DAVID VAN ESSEN
                             Shifter circuits for motion and stereo

10:45 a.m.   TERRENCE SEJNOWSKI
                             Learning algorithms; Backpropagation; investi-
                             gating the mammalian cortex via network
                             algorithms; learning shape-from-shapding

4:00 p.m.   SANDY PENTLAND
                             Shape representation: algorithms and computer
                             vision

Saturday, September 3

9:30 a.m.   JAMES BOWER
                             Conclusion