INTERACTING NEURAL NETWORKS ON A CORTICAL SURFACE

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1. INTRODUCTION

The availability of CSMP, a Continuous Systems Modeling Program, has assisted both the consideration of physiological problems in terms of alternative models and the comparison of model simulations with experimental data. Among the advantages of CSMP, an application-oriented language developed first by IBM, are that it is becoming fairly widely used for the simulation of dynamic physiological processes, and that it has been developed to rapidly handle most functions commonly encountered in physiology. The possibility of interjecting Fortran IV subroutines into CSMP programs gives a special flexibility, allowing construction of functions or subroutines to describe, for example, non-linear network properties.

Using CSMP, it has been possible to simulate interacting neural populations in the mammalian hippocampal cortex (1,2). These studies have led to a model for the hippocampus which involves pyramidal cells in a forward branch and interneurons in a feedback branch. This model is correlated with physiological studies and anatomical arrangements of neurons (3-7). In studies on cats, Dichter and Spencer (8-9) have proposed that a strongly inhibited ring of cortex surrounding an epileptogenic central area can limit the spread of seizure activity. The interactions of a central and a surrounding area of cells on a cortical surface thus have interest, both in terms of understanding basic neural mechanisms and in terms of elucidating experimentally induced epilepsy.

A primary intent of this study was to simulate the interaction of central and surrounding cortical neural populations in the hippocampus of cats and rabbits. The effect of a surround can be shown by first studying the central area of the cortex isolated from its surround, and then by studying the rejoined central and surround areas. The study illustrates the use of CSMP for modeling a dynamic and complex neurophysiological system.

2. PHYSIOLOGICAL AND ANATOMICAL BACKGROUND

The hippocampus is a relatively simple single-layered anatomical structure within the brain of mammals. A major group of cells found in this cortical layer are the pyramidal cells; and they appear to be responsible for much of the electrical activity recorded from the layer (4,5). These pyramidal cells in the hippocampus are connected to themselves and to other cells in an orderly fashion, channeling signals over particular pathways. The hippocampus is similar to another area of the brain, the prepyriform cortex, also a relatively simple cortical structure. Both prepyriform and hippocampal cortex are considered in this report, with emphasis placed on hippocampal activity.

Of the many properties of neurons, only a few key aspects are chosen here for simulation. Each of the thousands of cells of the cortex receives, processes, and transmits signals. Several presently relevant but much-simplified aspects of these functions are as follows: A neuron has areas reserved for reception of incoming signals from other neurons (e.g., synapses on its dendrites and its cell body). Incoming synaptic signals tend to either excite or inhibit the neuron. If excitatory input overrides inhibitory input, the neuron generates output action potentials. The action potential is propagated on one or several axonal processes and arrives at the terminal synaptic end(s) of the axon. (Many neurons, including those considered here, have branching axons so that the output signal is split into several signals identical to the parent signal.) When an action potential arrives at the synapse at the axons terminal end, a neurotransmitter diffuses across the synapse to either excite or inhibit the receptor area of another neuron (dependent on the nature of the transmitter). A single neuron may process several incoming signals; if excitatation overrides inhibition, the neuron usually generates a discrete signal which, in turn, can excite or inhibit other nerve cells.

Translating these basic neural features into a model requires their formulation as functional elements. Thus, the model neurons should have receptor elements whose response to excitatory and inhibitory signals resemble experimentally observed responses. They should have threshold or level detectors to determine when excitation has overriden inhibition. They must generate signals and propagate them over pathways corresponding to axonal processes, with appropriate delays and temporal dispersions. Connections between neurons and their excitatory or inhibitory character must be specified.

The present model of hippocampal neurons divides a section of the hippocampal cortex into two regions, an inner central region and an outer surrounding region. We can arbitrarily set parameters so that the two regions are either separate or connected. Each region contains many neurons, each neuron receiving and transmitting signals. In each region we model a simple neural circuit for which there is considerable experimental support (3-6).

The circuit takes the form shown in Fig. 1, and contains two types of cells, pyramidal cells and basket cells. As shown, a pyramidal cell (open circle) excites a basket cell (filled circle) which, in turn, inhibits the pyramidal cell. In addition, the pyramidal cell excites other pyramidal cells (a connection not shown in Fig. 1). That is, if a second pyramidal cell was included in the network, a feedback branch would be included showing that pyramidal cells excite pyramidal cells. Thus two recurrent pathways are present, an inhibitory pathway and an excitatory pathway. The pyramidal cell can be excited by a shock delivered at site S. An output can be measured at the site O.

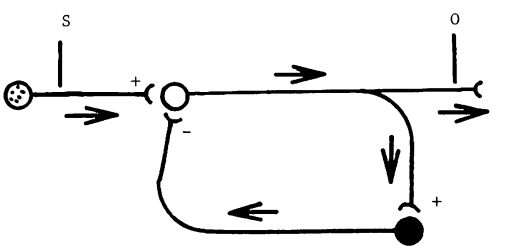

Figure 1. Basic neural network for the central and also surround regions. The symbol + denotes an excitatory connection and - denotes an inhibitory connection. A stimulus at S serves to send a signal to the right which excites the pyramidal cell (open circle). The pyramidal cell sends a signal as shown which can be recorded at O and also a signal over a branch to excite the interneuron (filled circle). The interneuron makes an inhibitory connection back to the pyramidal cell. The arrows indicate the direction of propagation of signals.

This neural circuit and its two cells are representative of thousands of such circuits connecting the two populations of cells. Thus, the modeled activity of this circuit corresponds to the mean activity of signal levels at corresponding points in the modeled populations. The normal baseline activity of the network is considered to be non-zero; that is, the pyramidal population output is not silent (1,2). Since the simulated waveforms presented in this report are averages, they can be related to experimentally recorded potential waveforms generated by the hippocampal cell populations (1,2).

Figure 2 shows such a record of the averaged post-stimulus activity recorded from the prepyriform cortex of a cat. (When a shock is delivered to the cortex, the neural network responds, generally transiently; summation and averaging of many such responses to shocks allows the response signal to be extracted from the ongoing EEG activity.) The averaged responses of a cortex (the central and surround regions joined together) is a transient waveform that often shows significant oscillations, indicating that cells can respond in a repeatable pattern to an electrical shock. This response pattern reflects aspects of a neural network of which the responding cells are a part.

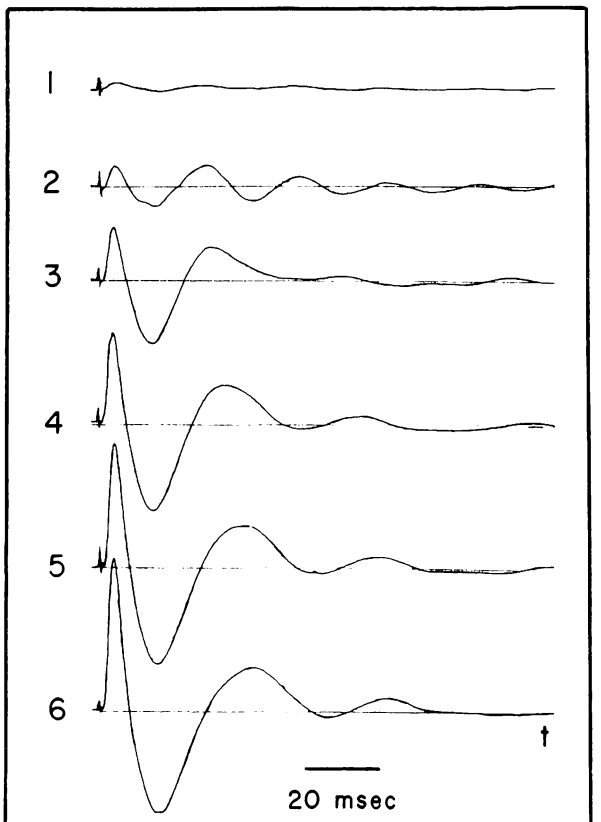

Figure 2. Averaged evoked responses recorded from cell populations in the prepyriform cortex of the cat following electrical stimulation. Traces 1 - 6 show responses as stimulus intensity increases from threshold values (Trace 1). Waveforms are from cat anesthetized with Sodium Pentobarbital.

Figure 3 shows a record of electrical activity in the unrestrained, unanesthetized rabbit. This record demonstrates that even in the normally functioning animal there can be synchronous oscillatory activity in cell populations. The oscillations indicate that whole subpopulations of cells are firing in unison. The 4-7 Hz waveform in Fig. 3 (denoted the theta rhythm) is a waveform wich is easily recorded and has attracted the attention of both psychologists and physiologists because it appears to be correlated with complex behavioral states such as alerting, motor activity, and memory processes (10,11). Thus experimental data suggests that it is appropriate to study the coordinated behavior of cell populations as well as the individual behaviors of single cells.

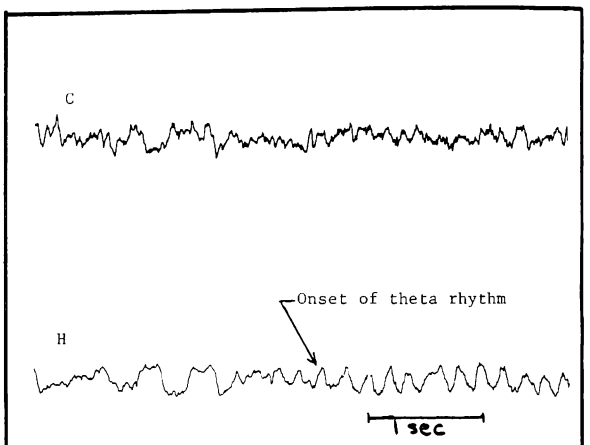

Figure 3. EEG waveforms from an unanesthetized rabbit. The upper trace, C, is from an electrode placed on the neocortex and the lower trace, H, from the hippocampus. H shows the synchronous activity of hundreds of pyramidal cells.

Since it would be impossible to draw the connections of hundreds of neurons contributing to oscillatory waveforms, a simpler network of connected populations was modeled as described above. We have modeled a network substantiated by electrophysiological analysis of single cells (3-6) and anatomical descriptions of the cells and their connections (7), recognizing that this circuit is only one of many within the hippocampus.

3. INTERACTING NEURAL NETWORK MODEL AND CALCULATED WAVEFORMS

3.1 The model.

The model for interacting neural networks takes the form of a CSMP program (listed on the following page) for a central region (1) and a surrounding region (2) on a cortical surface. The output variables of interest (SPIKE 1, SPIKE2) are signals which would be recorded by an electrode summing the activity of hundreds of pyramidal cells of either the central or surrounding areas. The input to the model is an electrical shock delivered to the pyramidal cells (see STIMULUS GENERATION section of program). The input is related to the output by the symbolic network shown in Fig. 1.

```
* CSMP SOURCE FROM TIMESHARE, VERSION 27.000.000
*************THE CENTER-SURROUND MODEL*********************
**********FOR HIPPOCAMPAL PYRAMIDAL CELLS*****************
*EMBEDDED IN EXCITATORY AND INHIBITORY FEEDBACK LOOPS****
*
*
************THIS MACRO CREATES A GENERALIZED PSP***************
MACRO  PSP=SYNAPS(A,B,C,K,M,SCALE,INPUT)
       M2DOT=M1*M2*INPUT
       P2DOT=K*P2DOT/M
       PDOT=INTGRL(B-P2DOT)
       M1=C*PDOT
       PSHAPE=INTGRL(0.,PDOT)
       M2=K*PSHAPE
       PSP=SCALE*PSHAPE
ENDMAC
*
*
*************************PARAMETER INITIALIZATION*********************
*
INITIAL
*
PARAM GINPT1=200., GLOPY1=100., GLOPZ1=TS
PARAM GINPT2=300., GLOPY2=100., GLOPZ2=TS
 PARAM GLOY11=TS, GLOY12=TS, GLOZ21=TS, GLOZ12=TS
PARAM LAYST1=0.6, DLOPY1=0.040, DLOPZ1=0.010
PARAM LAYST2=0.6, DLOPY2=0.040, DLOPZ2=0.010
*
PARAM IDLY12=60, IDLZ12=15
PARAM IDLY21=60, IDLZ21=15
*
PARAM FIRE1=0.0, STRAT1=1.5, PWIDE1=0.01
PARAM FIRE2=3.0, STRAT2=1.5, PWIDE2=0.01
*
PARAM IND1=20, THRES1=0.0
PARAM IND2=20, THRES2=0.0
*
PARAM XA1=0., XB1=0., XC1=-700., XK1=-100000., XSCAL1=100.
PARAM XA2=0., XB2=0., XC2=-700., XK2=-100000., XSCAL2=100.
PARAM YA1=0., YB1=0., YC1=-600., YK1=-10000., YSCAL1=-100.
PARAM YA2=0., YB2=0., YC2=-900., YK2=-10000., YSCAL2=-100.
PARAM ZA1=0., ZB1=0., ZC1=-600., ZK1=-10000., ZSCAL1=100.
PARAM ZA2=0., ZB2=0., ZC2=-600., ZK2=-80000., ZSCAL2=100.
*
PARAM YA12=0., YB12=0., YC12=-200., YK12=-10000., YSCL12=-100.
PARAM YA21=0., YB21=0., YC21=-200., YK21=-10000., YSCL21=-100.
PARAM ZA12=0., ZB12=0., ZC12=-600., ZK12=-80000., ZSCL12=100.
PARAM ZA21=0., ZB21=0., ZC21=-600., ZK21=-80000., ZSCL21=100.
*
PARAM XM1=1., YM1=1., ZM1=1., YM12=1., ZM12=1.
PARAM XM2=1., YM2=1., ZM2=1., YM21=1., ZM21=1.
*
PARAM MAX1=1.0, MAX2=1.0
PARAM BPSP1=0.10, BPSP2=0.10
*
*
BONDY1=DLOPY1-(IND1/2000)
BONDY2=DLOPY2-(IND2/2000)
*
*
*****************RECTANGULAR DISPERSION ARRAYS*****************
FIXED I,J,M
/DIMENSION BUF1(500), BUF2(500), DISPR1(500), DISPR2(500)
/DIMENSION ADLY12(500), ADLY21(500)
/DIMENSION ADLZ12(500), ADLZ21(500)
*
NOSORT
       DO 10 J=1,IDLY12
    10 ADLY12(J)=0.
       DO 20 J=1,IDLY21
    20 ADLY21(J)=0.
       DO 30 J=1,IDLZ12
    30 ADLZ12(J)=0.
       DO 40 J=1,IDLZ21
    40 ADLZ21(J)=0.
       TOP1=1.0/IND1
       DO 50 J=1,IND1
       BUF1(J)=0.
    50 DISPR1(J)=TOP1
       TOP2=1.0/IND2
       DO 60 J=1,IND2
       BUF2(J)=0.
    60 DISPR2(J)=TOP2
SORT
*****************DYNAMIC PORTION OF PROGRAM*****************
*
DYNAMIC
*
********SMOOTH LOOPING OF BACKGROUND ACTIVITY***************
FRAC=(1+SIN(3.14*(TIME-.250)/0.5))/2
EMIT=TIME
PROCED FRPSP1,FRPSP2=SINUS(FRAC,PPSP1,BPSP2,EMIT)
       FRPSP1=PPSP1
       FRPSP2=BPSP2
       IF(EMIT.LT.0.50) FRPSP1=FRAC*BPSP1
       IF(EMIT.LT.0.50) FRPSP2=FRAC*BPSP2
ENDPRO
*
*
*******ORTHO- AND ANTIDROMIC STIMULATION EPSPS***************
XPSP1=SYNAPS(XA1,XB1,XC1,XK1,XM1,XSCAL1,XINPT1)
XPSP2=SYNAPS(XA2,XB2,XC2,XK2,XM2,XSCAL2,XINPT2)
*
**********INHIBITORY FEEDBACK IPSPS ARE GENERATED************
YPSP1=SYNAPS(YA1,YB1,YC1,YK1,YM1,YSCAL1,YINPT1)
YPSP2=SYNAPS(YA2,YB2,YC2,YK2,YM2,YSCAL2,YINPT2)
YPSP12=SYNAPS(YA12,YB12,ZC12,YK12,YM12,YSCL12,YUPT12)
YPSP21=SYNAPS(YA21,YB21,YC21,YK21,YM21,YSCL21,YUPT21)
*
**********EXCITATORY FEEDBACK EPSPS ARE GENERATED**************
ZPSP1=SYNAPS(ZA1,ZB1,ZC1,ZK1,ZM1,ZSCAL1,ZINPT1)
ZPSP2=SYNAPS(ZA2,ZB2,ZC2,ZK2,ZM2,ZSCAL2,ZINPT2)
ZPSP12=SYNAPS(ZA12,ZB12,ZC12,ZK12,ZM12,ZSCL12,ZNPT12)
ZPSP21=SYNAPS(ZA21,ZB21,ZC21,ZK21,ZM21,ZSCL21,ZNPT21)
*
*
*************DENDRITIC RESPONSES TO INPUTS*****************
DNRIT1=XPSP1+YPSP1+YPSP21+ZPSP1+BPSP1
DNRIT2=XPSP2+YPSP12+YUPT2+ZPSP2+BPSP2+FRPSP2
*
*
**************SPIKE GENERATION AT THE AXON HILLOCKS***********
PROCED SPIKE1,SPIKE2=FORREST(DNRIT1,DNRIT2)
       SPIKE1=0.
       SPIKE2=0.
       IF(DNRIT1.GT.THRES1) SPIKE1=DNRIT1-THRES1
       IF(DNRIT2.GT.THRES2) SPIKE2=DNRIT2-THRES2
       IF(SPIKE1.GT.MAX1) SPIKE1=MAX1
       IF(SPIKE2.GT.MAX2) SPIKE2=MAX2
ENDPRO
*
*
***************SPIKE DISPERSAL BY CONVOLUTION*****************
PROCED DISPK1,DISPK2=SMEAR(SPIKE1,SPIKE2)
       DISPIK=0.
       I=IND1
       M=IND1-1
       DO 70 J=1,M
       BUF1(J)=BUF1(J+1)
       DISPIK=DISPIK+BUF1(J)*DISPR1(I)
       I=I-1
    70 CONTINUE
       BUF1(IND1)=SPIKE1
       DISPK1=DISPIK+BUF1(IND1)*DISPR1(1)
*
       DISPIK=0.
       I=IND2
       M=IND2-1
       DO 80 J=1,M
       BUF2(J)=BUF2(J+1)
       DISPIK=DISPIK+BUF2(J)*DISPR2(I)
       I=I-1
    80 CONTINUE
       BUF2(IND2)=SPIKE2
       DISPK2=DISPIK+BUF2(IND2)*DISPR2(1)
ENDPRO
*
*
*****************DELAY FACTORS IN LOOPS*******************
PROCED SPKY12,SPKY21=ILAG(DISPK1,DISPK2)
       SPKY12=ADLY12(IDLY12)
       DO 100 J=2,IDLY12
   100 ADLY12(J)=ADLY12(J-1)
       ADLY12(1)=DISPK1
       SPKY21=ADLY21(IDLY21)
       DO 110 J=2,IDLY21
   110 ADLY21(J)=ADLY21(J-1)
       ADLY21(1)=DISPK2
ENDPRO
*
PROCED  SPKZ12,SPKZ21=ILAG(SPIKE1,SPIKE2)
       SPKZ12=ADLZ12(IDLZ12)
       DO 120 J=2,IDLZ12
   120 ADLZ12(J)=ADLZ12(J-1)
       ADLZ12(1)=SPIKE1
       SPKZ21=ADLZ21(IDLZ21)
       DO 130 J=2,IDLZ21
   130 ADLZ21(J)=ADLZ21(J-1)
       ADLZ21(1)=SPIKE2
ENDPRO
*
SPIKY1=DELAY(800,.001,DISPK1)
SPIKY2=DELAY(800,.001*BONDY2,DISPK2)
SPIKZ1=DELAY(800,.001,SPIKE1)
SPIKZ2=DELAY(800,.001*BONDZ,SPIKE2)
*
*
*****************INPUTS TO SYNAPTIC BUTTONS*****************
YINPT1=GLOBY1*SPIKY1
YUPT21=GLOY21*SPKY21
YINPT2=GLOBY2*SPIKY2
YUPT12=GLOY12*SPKY12
ZINPT1=GLOBZ1*SPIKZ1
ZNPT21=GLOZ21*SPKZ21
ZINPT2=GLOBZ2*SPIKZ2
ZNPT12=GLOZ12*SPKZ12
*
*
*****************STIMULUS GENERATION**********************
SHOCK1=IMPULS(FIRE1,STRAT1)
SHOCK2=IMPULS(FIRE2,STRAT2)
FILE1=SQUARE(PWIDE1,SHOCK1)
FILE2=SQUARE(PWIDE2,SHOCK2)
SYNAP1=DELAY(800,LAYST1,FIBER1)
SYNAP2=DELAY(800,LAYST2,FIBER2)
XINPT1=GINPT1*SYNAP1
XINPT2=GINPT2*SYNAP2
*
*
*****************TIMING AND PRINTING PARAMETERS***************
TIMER DELT=0.001, FINTIM=2.0, OUTDEL=0.010
PRTPLT SPIKE1, SPIKE2
*
*
STOP
ENDJOB
```

Turning to the CSMP listing, we can indicate the overall signal flow in the program:

DENDRITIC RESPONSES TO INPUTS (halfway into the listing) show the several input signals which are summed by the pyramidal cells of the two cortical regions. These sums are compared to a threshold and determine SPIKE GENERATION AT THE AXON HILLOCKS in the following program section. The variables, SPIKE1 and SPIKE2, are evaluated here and are the variables plotted in Fig. 4.

These pyramidal output signals are temporally dispersed (SPIKE DISPERSAL BY CONVOLUTION), delayed (DELAY FACTORS IN LOOPS) and returned as INPUTS TO SYNAPTIC BOUTONS to the pyramidal cell receptor areas.

Post-synaptic potentials (PSPs) are created at these receptor areas. Modeled to resemble data, they take the form of solutions to overdamped second order linear differential equations (described in detail in IBM's users manual H20-0367-1 for CSMP). Thus, a spike input yields an overdamped transient PSP. Since there is a PSP for every synaptic type in the model (i.e., shock-stimulated excitatory synapses, XPSPs; inhibitory feedback synapses, YPSPs; excitatory feedback synapses, ZPSPs), a MACRO to generate the individual PSP programs was written (see top of program listing). Thus, using particular parameters for particular PSP shapes, and using appropriate input signals, ORTHO AND ANTIDROMIC STIMULATION EPSPS, INHIBITORY FEEDBACK IPSPS and EXCITATORY FEEDBACK EPSPS (just following the DYNAMIC title in the program), are generated and summed (DENDRITIC RESPONSES TO INPUTS). Thus, we have come full circle from summed pyramidal input, to output, around the feedback loops and back through the synapses.

Two of the variables not yet mentioned, but used in DENDRITIC RESPONSES TO INPUTS, are FBPSP1 and FBPSP2. They represent constant excitatory drive to the two cortical pyramidal cell populations. At the start of a simulation run, these drives are slowly increased from zero up to a constant level by the SMOOTH LOADING OF BACKGROUND ACTIVITY (immediately following start of the DYNAMIC section).

Simulation proceeds, following PARAMETER INITIALIZATION and buffer clearing and defining of the dispersing function (array) for the feedback signals (RECTANGULAR DISPERSION ARRAYS), by stepping of a time variable, in increments of DELT, and evaluation of all time-dependant variables in the DYNAMIC section at each point in time until FINTIM is reached. Printout is given in time increments of OUTDEL for the variables, SPIKE1 and SPIKE2.

3.2 Calculated waveforms.

Operation of the program simulates two variably coupled neural networks, one generating electrical activity in a central cortical region, and a similar one generating activity of the surrounding cortex. The networks are based on properties and connections of individual neurons. Dichter and Spencer (9) proposed that the surround could confine and stop the spread of unstable activity from an epileptogenic central area. Epileptogenesis was hypothesized to arise from enhanced excitatory feedback (9). Using the neural model, waveforms were calculated to determine if the model could show these properties.

Figure 4A shows the responses of the central area and the surround area when the areas are not connected and the excitatory feedback level is normal in both. A shock was delivered to the central region alone. The waveforms show transient damped oscillations in response to the shock (see also Fig. 1). Since the surrounding cortex is not coupled to the central area, there is no evoked activity from it.

Figure 4B shows the responses of the two areas, again unconnected, for a single shock delivered to the central area. In this case, the excitatory feedback in the central network was greatly increased, as hypothesized for epileptogenesis (9). As a result, there is a sustained oscillatory waveform generated by this area. Again, there is no activity evoked from the surround.

Figure 4C shows the responses of the two areas when they are connected, again for a single shock delivered to the central area. The parameters of each of the networks are the same as in Fig. 4B. Note that the effect of a normal surround is to dampen the oscillatory activity of the central area, thus confining the spread of instability.

Finally, in Fig. 4D the responses of the two connected areas for a single shock delivered to the central area are again shown. For this case, however, the excitatory feedback of the surround was raised to the excessive level already present in the center. In this situation, unstable non-decrementing oscillations spread out to the surrounding cortex from the central region.

4. DISCUSSION.

The configuration of neural connections within the hippocampus is complex; and the particular network simulated in this study is embedded in a larger network. A major consideration, therefore, was the selection of a limited number of properties and connections in a CSMP model of basic features of the selected network. A critical test of the model was to determine if, with the limited number of properties selected, the calculated responses of coupled populations corresponded to experimentally measured waveforms.

The choice of a model network was constrained by anatomical and physiological descriptions of the hippocampus. Anatomical studies show that one cell type, the pyramidal cell, can be connected with a second cell type, the basket cell, which, in turn, relays signals back to pyramidal cells (7). Physiological studies show that pyramidal cells excite interneurons, probably basket cells, which inhibit the pyramidal cells (3-5). Pyramidal cells also send branching fibers to excite pyramidal cells (9,12). The model of this report has been constructed on the basis of these particular data for this particular area of the brain and selects a circuit which is represented a vast multiplicity of times in the hippocampal pyramidal cell layer. Within a central area, there are many pyramidal cell - basket cell and pyramidal cell - pyramidal cell connections; and in the surrounding area, there are also many more such connections.

Even though only one basic circuit is considered, it has proven sufficient to simulate the interaction of center and surround effects as proposed by Dichter and Spencer (9). That is, when the center is not joined with the surround, a single shock to it can, under conditions of high positive feedback as suggested for epileptiform cortex (9), can lead to sustained oscillations of the central region (Fig. 4B). When the center is joined with a normal surround (with normal positive feedback), and when a single shock is again delivered to the central region, there is a suppression of the oscillatory activity and stability is achieved (Fig. 4C). Thus, a model originally designed for simulation of experimentally observed oscillatory waveforms in the prepyriform cortex and hippocampus (Figs. 1 and 2) can also be extended to give simulations consistent with experimental data concerning epileptogenesis (9). This neural model clearly differs from models of a few representative neurons as emphasis can be placed on overall population responses and such factors as background excitatory levels are easily simulated.

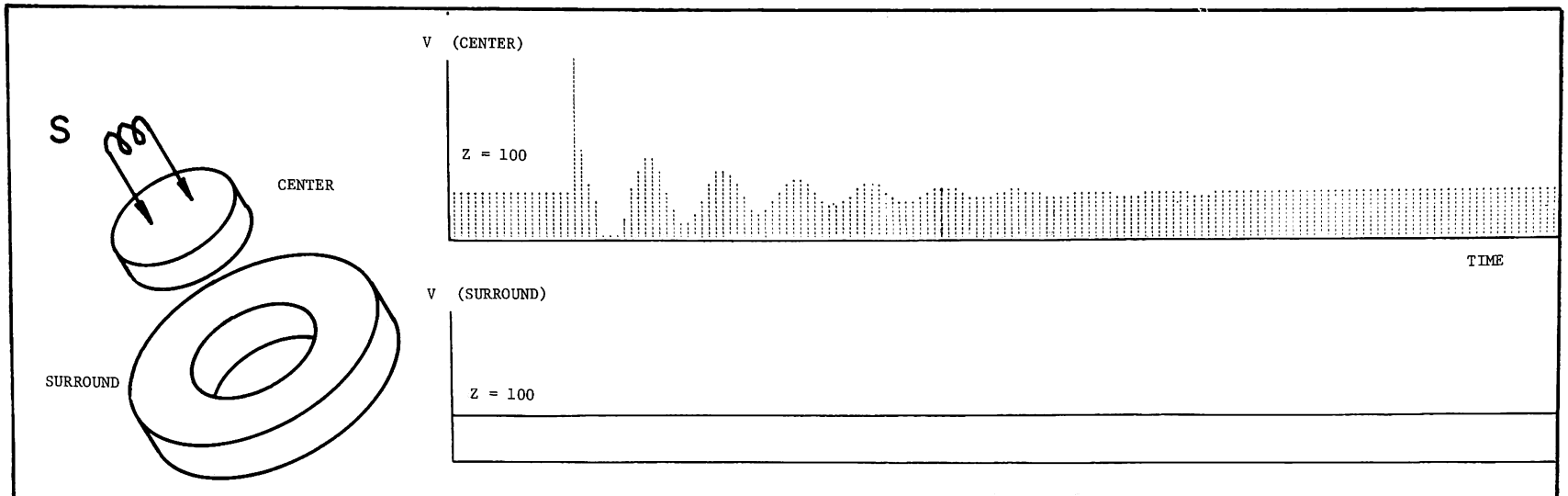

Figure 4A. Simulated pyramidal output from center and surround cortex following shock stimulation, S, of the center. Center and surround are uncoupled. All corresponding parameters of center and surround networks were held identical, including excitatory loop gain, Z. Stimulation causes a transient oscillatory response of the center network.

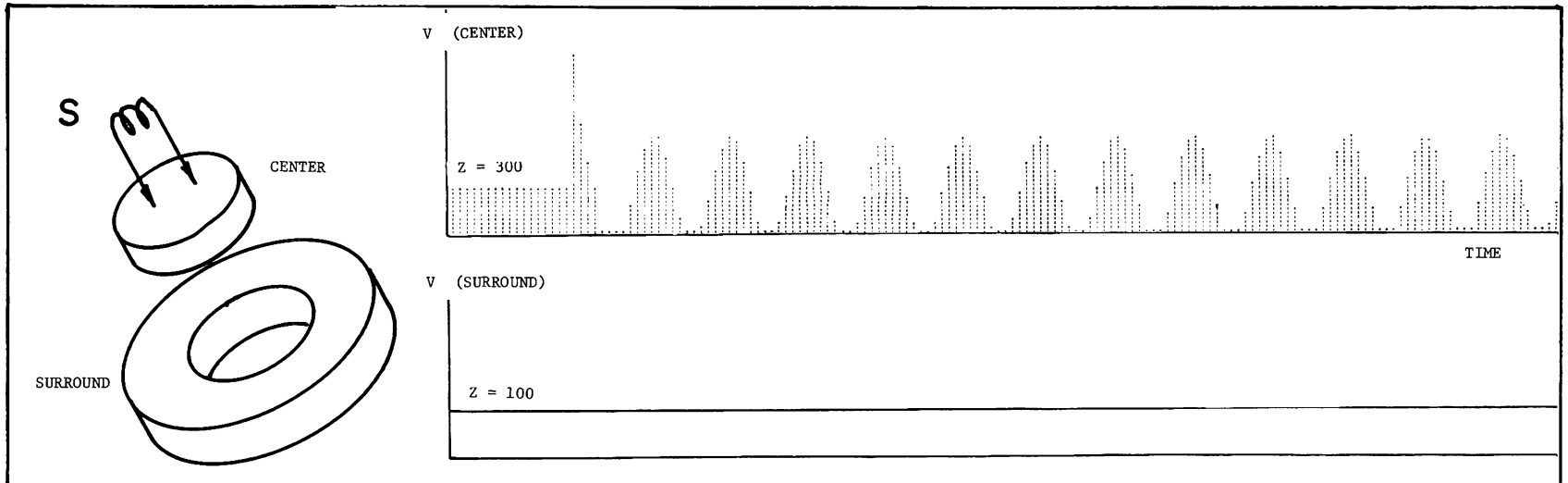

Figure 4B. Simulated pyramidal output from center and surround cortex following shock stimulation, S, of the center. Corresponding parameters of center and surround networks are identical except excitatory loop gain, Z. The center network shows instability with this high positive feedback.

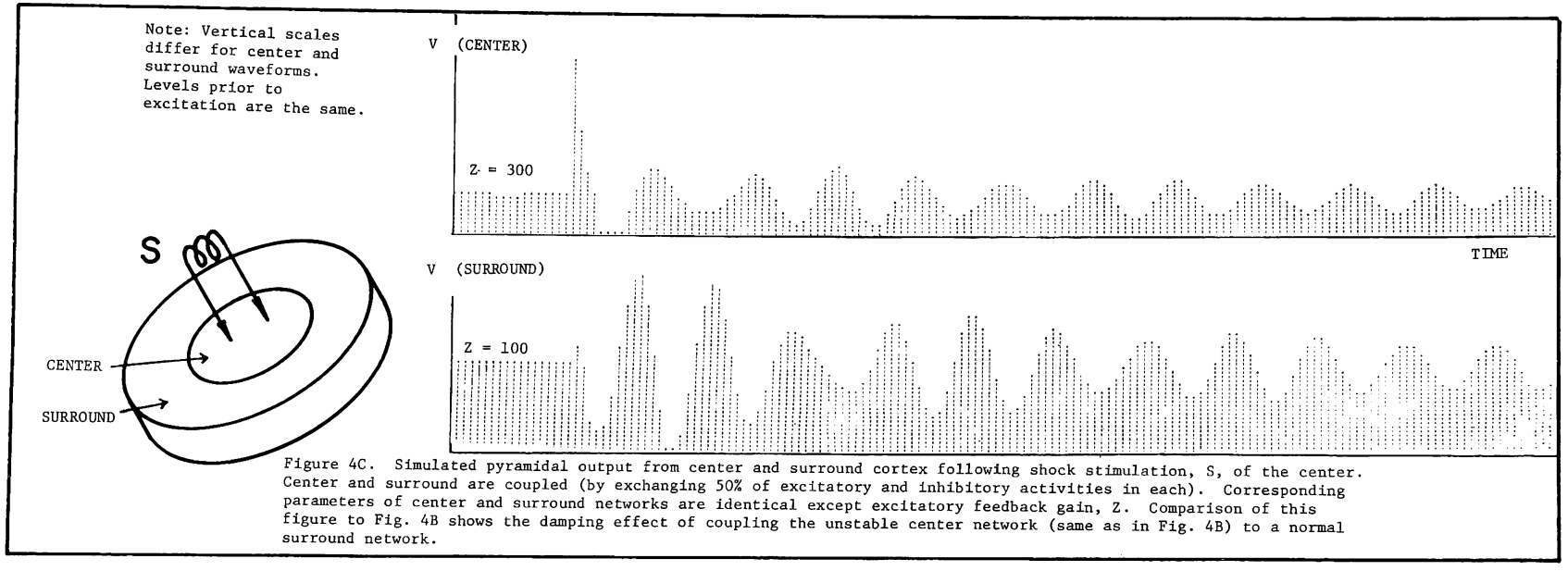

Figure 4C. Simulated pyramidal output from center and surround cortex following shock stimulation, S, of the center. Center and surround are coupled (by exchanging 50% of excitatory and inhibitory activities in each). Corresponding parameters of center and surround networks are identical except excitatory feedback gain, Z. Comparison of this figure to Fig. 4B shows the damping effect of coupling the unstable center network (same as in Fig. 4B) to a normal surround network.

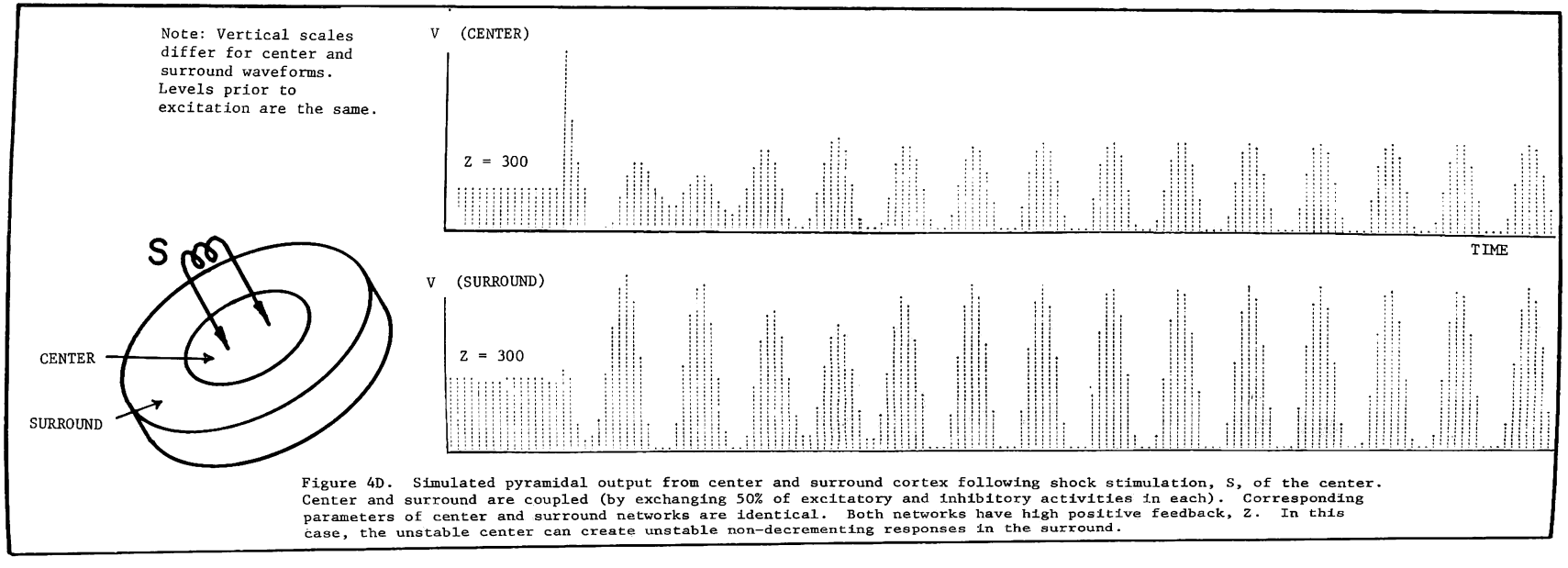

Figure 4D. Simulated pyramidal output from center and surround cortex following shock stimulation, S, of the center. Center and surround are coupled (by exchanging 50% of excitatory and inhibitory activities in each). Corresponding parameters of center and surround networks are identical. Both networks have high positive feedback, Z. In this case, the unstable center can create unstable non-decrementing responses in the surround.

5. REFERENCES

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