TESTS ON A CELL ASSEMBLY THEORY OF THE ACTION OF THE BRAIN, USING A LARGE DIGITAL COMPUTER

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Abstract

Theories by D.O. Hebb and P.M. Milner on how the brain works were tested by simulating neuron nets on the IBM Type 704 Electronic Calculator. The formation of cell assemblies from an unorganized net of neurons was demonstrated, as well as a plausible mechanism for short-term memory and the phenomena of growth and fractionation of cell assemblies. The cell assemblies do not yet act just as the theory requires, but changes in the theory and the simulation offer promise for further experimentation.

Introduction

The problem of how the brain works can be approached by investigating the elementary components, the neurons, and then seeing how larger and larger assemblies of these operate. Or it can be approached by observing the behavior of the entire organism and working back to determine what the components must be. The former activity is called neurophysiology and the latter is called psychology. Before we can say that the problem is well in hand, these two approaches must meet in the middle so that we have a single consistent picture that firmly connects psychology and neurophysiology.

As the neurophysiologist considers more and more complicated structures of neurons he gets into problems that are less and less related to his normal way of thinking. Curiously, however, some of these problems do not begin to resemble parts of psychology. What is happening is that the neurophysiologist is beginning to think about information handling machines that are too complex to be understood without the specialized knowledge of other disciplines. These other disciplines are information theory, computer theory, and mathematics. People in these other fields need to augment the work of the neurophysiologists and psychologists before the brain can be properly understood.

In the experimental study of the brain it is not yet possible to observe well the electrical interconnections among neurons. No one has yet been able to simultaneously record input and output signals of a single neuron in the brain. For this reason it has not yet been possible to test certain theories about how the brain works by experimentation on animals.

It is possible to measure the electrical characteristics of an isolated neuron in some circumstances.[1,2] One can imagine an elaborate network of such neurons and conjecture on the behavior of the network. The analytical treatment of these networks has proved that one can construct any desired kind of logical machine from elements that are probably much less powerful than neurons.[3,4]

The analytical approach has not been very effective in actually describing the behavior of complicated networks of neurons. However, it has proved effective to simulate such networks and to draw conclusions from the behavior of the simulated network of neurons.

Two sets of simulation experiments were made and another is in progress. In the first of these it was possible to simulate a network of up to 99 neurons and a test was made of part of the theory advanced by D.O. Hebb in his monograph, The Organization of Behavior.[5] The second set tested an unpublished revision of P.M. Milner of part of Hebb's theory with a network of 512 neurons. The third set is to test a further revision. In each case the original neurophysiological theory had to be interpreted in order to get something definite enough to simulate, and these interpretations were done by the present authors.

THE 69-NEURON DISCRETE PULSE SIMULATION

In this paper the term "neuron" will generally be used as an abbreviation for the term "simulated neuron". Likewise the term "synapse" will be used to stand for the term "simulated synapse", in other words for the simulation of the coupling mechanism that enables one neuron to send signals to another. Where ambiguity could arise, qualifying adjectives will be used.

The basic idea of the simulation can be seen by reference to Fig. 1. The large rectangle in Fig. 1 stands for all of the 2048-word high speed electrostatic memory of the Type 701 calculator. The memory was divided into 70 parts, one for each neuron and one for the program. In the area reserved for each simulated neuron were some numbers that might theoretically be measured on a corresponding living neuron. These numbers gave all of the information that was needed about each neuron. Specifically, the things that were known about each neuron, either from its location in memory or from the numbers stored there, were:

1. It's number (name)
2. How long since it had fired
3. How tired it was from having been fired excessively
4. For each of 10 output (efferent) synapses:
   - 4.1 The number of the (efferent) neuron that it simulated
   - 4.2 The magnitude of the signal that it sent to that (efferent) neuron when this (afferent) neuron fired.

Under control of the program, the calculator repeatedly scanned the 69 neurons and, by making calculations, caused these numbers to change as they would have changed if the network had actually been constructed. Therefore, after each pass over the data in memory, the data represented, in great detail, the state of each neuron and synapse in the network at the next instant of time.

In this model, time was quantized into time steps. A neuron could fire at any time step, but not between. A time step corresponded approximately to the interval between the firing of one neuron in a chain to the firing of the next. In the simulation, the average length of time required for a single time step was about 5.3 seconds and this corresponded to perhaps 0.7 milliseconds in the brain. Therefore, the simulation was slower by a factor of 7600.

At any given time step a neuron was either fired or in some state of recovery from being fired. Various recovery curves were used and the one shown in Fig. 2 was typical.

During any given run on the calculator, the neurons were interconnected in a particular net. Each neuron was connected so as to stimulate 10 other neurons. Usually the net was designed by the calculator. It would make a random choice of the neuron to be stimulated by each of the 10 output synapses of each neuron. It would record these choices on punched cards and retain them for the rest of the run.

If a neuron fired at time step (n-1) it would stimulate 10 neurons so as to tend to cause them to fire at time step n. The size of the signals sent to the 10 neurons would depend only upon the fact that the original neuron fired and upon the magnitudes of the interconnecting synapses. To say this another way, the input signals to a neuron, together with its threshold, would determine whether or not the neuron would fire, but if it did fire, the strength of firing would not depend on the input signals.

The input situation of a typical neuron is shown in Fig. 3 with some possible values of synapse magnitudes. The behavior of neuron x is shown in the following table.

Neurons that fired on step (n-1)	Mag	Threshold of x	Would x fire on step n ?
AB	295	256	Yes
BCE	252	256	No
BCDE	336	256	Yes
ABCDEFG	839	839	Yes
ABCDEFG	839	938	No
ACF	408	376	Yes
EFG	376	376	Yes

It can be seen that the input circuits to such a neuron can provide quite sophisticated switching.

Not all of the properties of the simulated neurons have been described. However, to make the exposition easier to follow, it is convenient to skip ahead and show some observations on the behavior of networks of neurons. Except for some minor difficulties, this behavior would be obtained with neurons like those already described. The discussion of these minor difficulties will be clearer after showing these results.

Fig. 4 shows an example of what will be called diffuse reverberation. Each row in this figure indicates with a 1 those neurons that fired and with an 0 those neurons that did not fire in a particular time step. Each column, of the 64 columns at the right, shows the history of a single neuron. The right hand 64 columns of Fig. 4 show, therefore, the complete firing history of 64 neurons for 50 time steps.

Fig. 5 shows, as a function of time, the number of neurons that were simultaneously fired. The time covered here is a little larger

than in Fig. 4 and shows the complete history beginning with a quiescent net and continuing until the activity died out.

We propose this diffuse reverberation as a plausible mechanism for short term memory, the kind of memory that is involved in remembering the intermediate results in mental arithmetic. We will discuss later some conjectures as to how the brain can make use of such a memory mechanism.

Now another property of the neurons will be described. When neuron A participated in firing neuron B, the synapse that enabled A to stimulate B was increased in magnitude unless it already had reached the limit of 938, in which case it remained constant. This characteristic was our version of Hebb's basic neurophysiological postulate. Hebb postulated that, "When an axon of cell A is near enough to excite cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased."[6]

This property of simulated neurons is somewhat curious. No process of just this sort has been observed in living tissue. However, it has not been possible to demonstrate, by measurement, that the Hebb postulate is false. Nothing else has been observed that could account for learning and memory in a plausible way. The Hebb postulate suggests a plausible machine that does not contradict experiment.

The purpose of the assumption about the growth of synapses is to get a mechanism for the retention of long term memory. When an animal experiences some event there will be activity in its brain. This activity will consist of a spacial and temporal pattern of firing of neurons. During the experience, the synapses involved will be strengthened, according to Hebb's postulate. Therefore, the same, or a similar, sequence of neural events is more likely to take place later than it would have been if the animal had not had that experience. A repetition of some part of the neural events that were associated with an experience is assumed to be the act of recalling the experience. It is evident that the mechanism that Hebb postulated would tend to cause recollections. The question of whether or not the postulate is sufficient is, in a sense, the main topic of this paper.

If no additional rule were made, the Hebb postulate would cause synapse values to rise without bound. Therefore, an additional rule was established: The sum of the synapse values should remain constant. This meant that, if a synapse was used by one neuron to help cause another to fire, the synapse would grow. On the other hand, if a synapse was not used effectively, it would degenerate and become even less effective, because active synapses would grow and then, to obey the rule about a constant sum of magnitudes, all synapses would be reduced slightly, so the inactive synapses would decrease.

Before discussing network action further, another property of the neurons will be mentioned. A neuron fired at too high a frequency becomes less sensitive, so that more stimulation is required to fire it. The effect of this is shown in Fig. 6, which shows the threshold as a function of time when the neuron is fired repeatedly with a constant level of stimulation. As with a living neuron, this simulated neuron fires rapidly at first and then settles down to a lower rate of firing.

This process is called fatigue because of the obvious analogy to living neurons. A significant aspect of fatigue is that it is a form of memory and, as such, may plan an important part in the operation of the brain

The concept of cell assembly occupies a key position in Hebb's theory. A cell assembly is a group of neurons that are interconnected in a very complex fashion and within which diffuse reverberation can take place. Fig. 4 shows just such a situation.

Parts of the cortex are imagined to consist of a large number of cell assemblies, each of which contains a large number of neurons. Only a small fraction of the cell assemblies are aroused at any one time. In other words signals are reverberating in only a few cell assemblies at once. Just which cell assemblies would be aroused at any one time would depend in large part upon what cell assemblies had been aroused at a previous instant of time, and in small part upon signals from elsewhere.

In the language of information theory, this part of the brain can be considered to be a finite state transducer, in which the internal state is determined by noting which cell assemblies are aroused and which are quiescent. In other words, the brain should exhibit a kaleidoscopic sequence of patterns of cell assembly arousal. It is outside the scope of this paper to expound Hebb's theory, so it will be assumed henceforth that the reader either understands the significance of a finite state transducer or has read Hebb's book.

In passing, it is worthwhile to point out how appropriate the finite state transducer description is for Craik's "hypothesis on the nature of thought."[8]

Hebb's theory required that it be possible for a neuron to belong to several different cell assemblies and that not all of these assemblies be aroused at once. Hebb's theory also required that it be possible for a neuron to change its affiliation from one cell assembly to another. It may be possible to devise a theory that has only the second requirement, but no further consideration of this possibility will take place in this paper.

The problem of how cell assemblies can arise and how they become modified, is vital to this theory. It will be shown that Hebb's scheme is unlikely to work with neurons of the type described so far. It will also be shown that, by suitably improving the neurons and by making the network more complex, cell assemblies can be made to form spontaneously. It will further be shown that these cell assemblies are not entirely satisfactory but that there is a plausible course for further investigation.

Suppose that there is initially some activity in a network of neurons and that input signals are impinging on the network. Suppose also that from time to time a particular input signal, S, arrives. When S first arrives, it will impinge on some internal state, I_j. In other words it will impinge upon some particular configuration of states of individual neurons. The particular sequence of internal states, I_{j+1}, I_{j+2}, I_{j+3}, ..., that follows will strengthen certain synapses in such a way that the sequence I_j, I_{j+1}, I_{j+2}, I_{j+3}, ... is more likely to occur again. It was conjectured that the next time S occurred, some part of I_j would be in existence and that some part of the sequence I_j, I_{j+1}, I_{j+2}, I_{j+3}, ... would be reinforced. As S appeared repeatedly some characteristic response to S gradually would become sufficiently reinforced as to be identifiable. As the characteristic sequence was arising there would appear, in it, points where diffuse reverberation could occur. In other words there would be some internal state I_{j+k} which would repeat some part of an earlier state in the sequence. As soon as this happened the rate of reinforcement of the connections would increase, because each time the stimulus S occurred the sequence of states would be such as to give several reinforcements to some of the connections instead of just one reinforcement. It was conjectured that cell assemblies corresponding to some common stimuli would arise in the brain in this way.

In order to test this conjecture about the manner in which cell assemblies form, a program was written to generate an appropriate environment for the neuron network, and an arrangement was set up for the network to receive signals from the environment. To receive the signals, six neurons were chosen to act as receptors. It was arranged that no neurons would stimulate these receptors. Instead, they could be fired only by an external program to enable the calculator to reach in and modify the one bit on each of the six neurons that indicated whether or not it had just received enough stimulation to fire. The synapses from the receptors spread out diffusely through the network.

The neuron net was stimulated once every ten time steps with a 6-bit signal that could define the state of each of the six receptors. The signals were chosen by a program whose action is illustrated in Fig. 7. It is a Markov process in which there is some probability that the input will be random but mixed in with the random signals are frequent occurrences of certain sequences. The network then had the opportunity to develop a characteristic response to each of the three sequences.

The network did not develop any characteristic responses and there was no sign of development of cell assemblies. A number of variations on this experiment were tried, all with the same result. Then the reason for the difficulty was realized and a simulation experiment was run to verify the explanation.

In such a neuron network, the idea that a detailed temporal-spacial pattern of firing can be effectively reinforced by a partial repetition is false. The reason can be seen from the following experiment. A simulation experiment was run to a convenient point where diffuse reverberation was taking place. Then all the data was punched on tabulating cards. These cards contained all relevant information so that, if they were read by the calculator, the simulation would go on from where it left off. Before the cards were read by the calculator, however, they were reproduced to give four identical decks of cards. Then three of these decks were slightly modified, each in a different way. In each case the modification was to choose some neuron that was about to be fired and manually change the number that specified its state of recovery so that it wouldn't fire quite so soon. Then four simulation experiments were run, one with each deck.

The four sets of results were compared and it was found that the detailed patterns of firing diverged rapidly. In just ten time steps, in each case, over 30 per cent of the neurons firing were different. This result is shown in Fig. 8. This shows that even slight differences rapidly grow to be large differences so there is little chance that a detailed pattern of firing can be effectively reinforced.

It was concluded from this work that some additional structure was needed within a network to allow all assemblies to form. A plausible model of a short term memory had been demonstrated but rather convincing evidence had been found to show that Hebb's postulate was not enough to make cell assemblies form.

Some other experiments were run which coincided in time with the work of Farley and Clark[9] and which reached essentially identical results. However, these did not seem to throw any light on the central problem of how the brain works, so this line of investigation was dropped.

512-Neuron F. M. Simulation

At this point we conferred with D. O. Hebb and one of his people, P. M. Milner. Milner had been working on a revision of part of Hebb's theory to introduce more recent neurophysiological data. The essence of Milner's idea was that inhibitory synapses, as well as excitatory synapses, are needed and that within a cell assembly most synapses are excitatory, while between cell assemblies most synapses are inhibitory. This idea sounded to us like a plausible cure for the troubles in the first model. It made engineering sense.

The significance of the idea can be seen by considering two cell assemblies. These will act like an Eccles-Jordan Flip Flop circuit. Suppose one is aroused. It keeps itself going by its internal excitatory connections and keeps the other quiescent by the inhibitory interconnections. Finally it begins to fatigue. As it begins to falter, it inhibits the other less strongly, so sporadic residual activity in the other begins to increase. This in turn inhibits the aroused cell assembly, causing it to falter more. This feedback condition causes an abrupt switching so that the aroused one becomes quiescent and the quiescent one becomes aroused. A more detailed discussion of this can be found in Appendix 1.

It seemed certain that the switching action would take place, but it was not clear whether the possibility of having inhibitory synapses would be enough to allow cell assemblies to arise or whether some cell assembly structure would have to be built in at the start.

Experiments with the discrete pulse model indicated that diffuse reverberation was a fairly reliable sort of thing in a net of 63 neurons, but quite erratic in a net with 21 neurons. Therefore it was felt that in a new experiment there should be a larger number of neurons in a net. A major obstacle to this was that the calculator was not fast enough to manage a very much larger net, even though this was to be done on the Type 704 which is faster than the 701. Something had to be sacrificed.

It was decided to sacrifice the knowledge of exactly when an individual neuron fired. All that the machine or the experimenter could know was the frequency at which a neuron was firing, and not the exact instants of time at which it did fire. The frequency would vary from time to time, so this was called the FM model.

One particular version of the FM model will be described here. There were 512 neurons, each with 6 input (afferent) synapses and a number of output (efferent) synapses that varied from one neuron to another. The synapse magnitude lay between -1 and +1 and changed as long term learning took place. The frequency of a neuron varied from 0 to 15. Equations are given in Appendix 2 to specify precisely how these quantities varied from time to time, and a qualitative description is given below in the text.

The magnitude of a synapse was much like a correlation coefficient between the two neurons that it connected. If the frequencies of the two neurons usually went up and down together, the synapse magnitude would grow toward +1. If, on the other hand, one neuron was usually inactive while the other was active, the synapse magnitude would approach -1. This is the FM version of Hebb's basic neurophysiological postulate.[6]

The frequency of a neuron was obtained essentially by calculating, for each synapse, the product of the synapse magnitude and the frequency of the stimulating (afferent) neuron, adding these products, and normalizing. It was further bounded by not being allowed to go negative. Therefore a neuron could have a high frequency only if it was stimulated through positive synapses by neurons with large frequencies and not simultaneously stimulated through negative synapses by neurons with large frequencies .

The fatigue increased if the frequency was high; stayed constant if the frequency was intermediate; and decreased if the frequency was low. Furthermore, it was not allowed beyond the bounds of 0 and 7. A fatigue of 7 could nearly stop a neuron while a fatigue of 3 did little to it.

An important change was made in the nature of the connections in the net. A distance bias was introduced so that two nearby neurons were more likely than two remote neurons to be connected together through a synapse. In the experiment described in this paper, the neurons were visualized as being arranged in a cylinder, as shown in Fig. 9. The cylinder was 16 neurons high and 32 neurons around. If two neurons were within eight of each other, they were as likely to be connected by a synapse as any other two neurons that were within eight of each other. However, no neurons that were farther apart were connected by synapses.

Four blocks of four neurons each were selected to act as receptors. These four blocks are shown in Fig. 9. The procedure that was used most of the time was that receptor areas 1 and 4 were controlled to have maximum activity for three successive time steps and then the net was allowed to operate with no external stimulation for three time steps. Then areas 2 and 3 were controlled to have maximum activity for three time steps and then the net was again left alone for three time steps. This cycle was repeated many times. The cycle was considered to be the equivalent of about 0.2 seconds in an animal and took about 160 seconds on the calculator.

Cell assemblies did actually build up around each of the receptor areas. Within a cell assembly the interconnections were largely excitatory and between cell assemblies they were largely inhibitory.

The activity of each neuron at each time step for one complete cycle is given in Table 1.

Table 1

Activity During One Complete Cycle of Stimulation

1.

```
00400000000006000000140314100300
00400003100100000077010400000000
00000020700000000077010000120 00
00000000000050010000500030000000
00100053011004000006104000200000
00000101000400000005001100000000
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00000010000000000001100010000000
00000400003000030000000000000001
00000001000000410500000000400110
00000000000000600000004100000000
00000000000006000000000000000000
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00000000050010100377000000010000
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00000000050100000100000010000010
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2.

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00040000010001000000000001000000
00000010100000000000000000000000
00000000001000010007000000000000
00000001100000100200000400000000
00000000010000410041000000000000
00000000000003100130000020000000
00000230000000000000000000000000
00000010000310000047700000000000
00010200000000000477000000200000
00000100000000000000000000000000
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3.

```
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00202003310000020077010020000000
00000000000010300077507000000000
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00000000000000000000000000000000
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00030000000001000000000000000000
00000010000000000000000000000000
00000000000000001307000000000000
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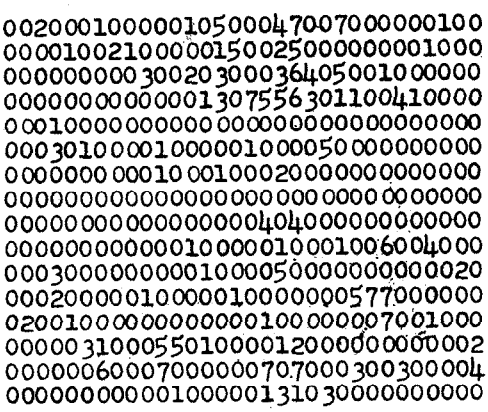
4.



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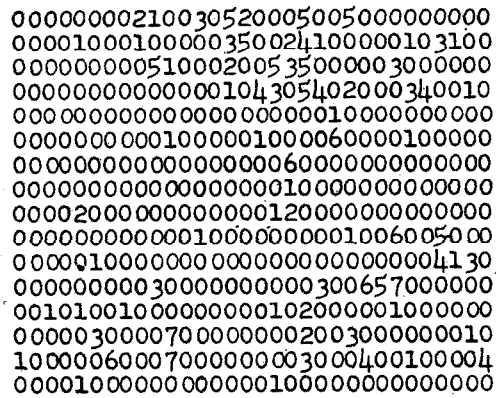
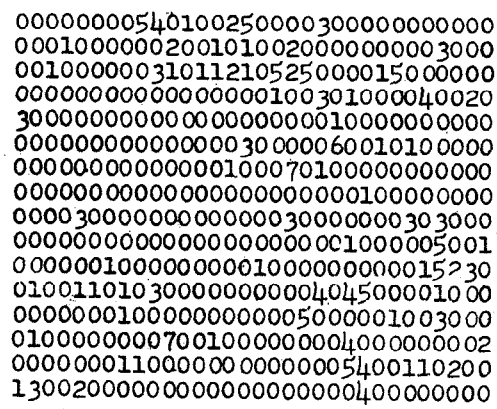
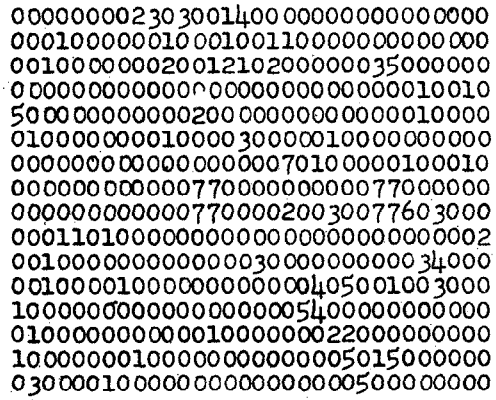


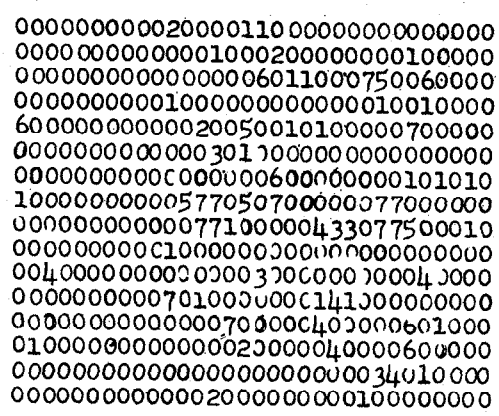
6.



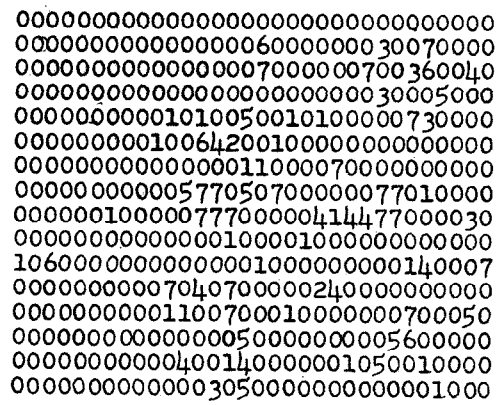
7.



8.



9.



10.

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0000000000000040000600000000100
1000000000000000100070000000007
0000000010535070000000000000000
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0000100000040047000020003002OOOO
0000000003300405000000000013000
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11.

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0000000013037140001300300000000O
5000000000000053002070030020000
.0000000000011601020010004000100O
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0000000007400206000000100003OOOO
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12.

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0010000000000000100005403000000
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0000000000000031100010000001000O
0000000005210300000000100003000l
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In Table 1 the numbers are just half of the frequencies of the neurons. This was done to reduce the cost of printing. Since it is difficult to see what is going on without practice and effort, a small section of the sixth and ninth steps have been reproduced side by side in Fig. 10. These times were chosen to contrast the arousal of 1 and 4 which suppress 2 and 3, with the arousal of 2 and 3 which suppress 1 and 4. Nearly every neuron has chosen allegiance to one cell assembly or another. Only 3 of the 224 neurons shown are active at both times.

Examination of the synapses also showed that cell assemblies had formed. A dividing line was found between area 1 and area 2. Synapses that crossed this line were predominately negative while synapses that failed to cross it were predominantly positive.

There is no doubt that cell assemblies did form. A very detailed statistical study of the allegiance of neurons to cell assemblies was not made because, as will soon be evident, the model still needs improvements, and the statistics of the improved model would be different.

A further significant characteristic needed by the Hebb-Milner theory was evident. Over sequences of 100 or so time steps (perhaps the equivalent of 17 minutes) neurons were observed to change allegiance from one cell assembly to another. In other words, "fractionation" and "recruiting" were observed.

Some evidence was found to indicate that one cell assembly tended to arouse another. However, the tendency was weak. It can be seen that the only possible excitatory synapses between cell assemblies would be those involved with neurons of dubious allegiance. Apparently this was insufficient to allow spontaneous activity in the net. The only activity was caused by the input signals and the arousal of cell assemblies was completely determined by the input. The theory requires that the preceding central activity (set) be much more influential than the input stimuli, so clearly some changes are needed.

Plans for the Future

After studying the detailed results of this experiment, we arrived at a conjecture as to what should be done next. This conjecture was based on our intuition gained from experience in designing computing machines. We felt that the inhibitory synapses should be separated from the excitatory synapses and should follow different rules. Appendix 1 describes some detailed con-

siderations of the transmission of activity from cell assembly to cell assembly.

We then consulted again with P. M. Milner and learned that he had just produced a further revision of the theory that had just this property of synapses with differeing characteristics. His new model appears also to have the characteristic that the cell assemblies would be much more diffuse than in the FM Model described here. This would correspond better to what is expected in the brain and would make a better machine because one cell assembly could directly affect a larger number of others. It is not within the scope of this paper to discuss this new scheme because we have not yet reduced it to our terminology and tested it. However, the work is proceeding.

Summary

The first set of experiments, designed to test parts of the theory advanced in The Organization of Behavior, by D. O. Hebb, simulated a network of 69 neurons with a "Discrete Pulse Model." This set of experiments clearly illustrated the diffuse reverberation that is advanced as an explanation of short term memory. There was, however, no tendency for neurons to group into cell assemblies.

The second set of experiments were designed to test P. M. Milner's revision of Hebb's theory with an "F. M. Model" which kept track of the frequency of firing of 512 neurons but ignored the precise timing of individual firings. Cell assemblies formed and exhibited the "fractionation" and "recruiting" required by the theory. The cell assemblies, however, were not able to arouse one another, so this model was too heavily dominated by environment.

A third set of experiments is in progress. It is hoped that this set will get around the next major obstacle in producing a model that will do what the neurophysiological theory requires.

This kind of investigation cannot prove how the brain works. It can, however, show that some models are unworkable and provide clues as to how to revise the models to make them work. Brain theory has progressed to the point where it is not an elementary problem to determine whether a model is workable. Then, when a workable model has been achieved, it may be that a definitive experiment can be devised to test whether or not the workable model corresponds to a detail of the brain.

Appendix 1.

The Interaction of Cell Assemblies

Suppose that all synapses within a cell assembly are excitatory and that both excitatory and inhibitory synapses go between all assemblies. Suppose also that the effect of stimulation at a synapse rises suddenly when the preceding (afferent) neuron fires, and then dies out more slowly. For example, a model of this could be a chemical transmitter that was discharged on the stimulated (efferent) neuron and that was destroyed at an exponential rate. Suppose also that the effect of an excitatory synapse fades more slowly than the effect of an inhibitory synapse. In terms of the chemical transmitter, this could mean that two different chemicals were used for inhibition and excitation, and that these were destroyed at different rates. Finally, suppose that the total inhibitory stimulation of an aroused cell assembly on a quiescent cell assembly dominates the total excitatory stimulation.

While a cell assembly is firing actively it will suppress its neighbors. However, when its neurons tire and it begins to falter, the inhibition will drop more rapidly than the excitation. When the level of inhibition drops below the level of excitation, switching will take place.

This sort of interaction between neurons is being built into the third set of experiments.

Appendix 2
Equations Describing the FM Model

The structure of the net is given by

$$j = g(h, i)$$

where i is the number of the efferent neuron, j is the number of the afferent neuron, and h is the number of the afferent synapse for the ith neuron. g (h, i) is determined at the beginning of an experiment, and remains constant.

The following quantities for all i, j determine the state of the model at any time t.

Symbol	Number of Bits	Description
x (i, t)	4	frequency of neuron i at time t
x (i, t)	4	average frequency of neuron i at time t
d (i, t)	3	fatigue of neuron i at time t
r (i, j, t)	8	magnitude of the synapse at time t coupling stimulation from neuron i to neuron j
R (i, t)	8	a function of x(i, t), x(i, t-1), ...

Initial conditions for the net are given by the values $x(i,0)$, $\bar{x}(i,0)$, $d(i,0)$, $r(i,j,0)$, and $R(i,0)$.

The quantities $S(i,j,t)$ and $x'(i,t)$ are intermediate results in the calculation. A single time step consists of the successive evaluation of the following formulas:

1) $$S(i,j,t) = r(i,j,t)\sqrt{R(i,t)\,R(j,t)}$$

2) $$R(i,t+1) = \left(1 - \frac{1}{m}\right) R(i,t) + \left(x(i,t) - \bar{x}(i,t)\right)^2$$

$$R(j,t+1) = \left(1 - \frac{1}{m}\right) R(j,t) + \left(x(j,t) - \bar{x}(j,t)\right)^2$$

$m = 32$

3) $$S(i,j,t+1) = \left(1 - \frac{1}{m}\right) S(i,j,t) + \left(x(i,t) - \bar{x}(i,t)\right) \cdot \left(x(j,t) - \bar{x}(j,t)\right)$$

$m = 32$

4) $$r(i,j,t+1) = S(i,j,t+1) \Big/ \sqrt{R(i,t+1)\;R(j,t+1)}$$

5) We define $p(i,t) = j$ such that $r(i,j,t) \geqslant 0$, and $q(i,t) = j$ such that $r(i,j,t) < 0$, including only values of j such that the synapse (i,j) exists.

Then

$$x'(i,t+1) = k_o \left[\frac{\sum_{p(i,t+1)} \left[r(i,j,t+1) + k_1\right] x(j,t)}{\sum_{p(i,t+1)} r(i,j,t+1) + k_1} - \frac{\sum_{q(i,t+1)} \left[r(i,j,t+1) - k_1\right] x(j,t)}{\sum_{q(i,t+1)} r(i,j,t+1) - k_1} \right]$$

$k_o = 1.25$, $k_1 = 1/512$

6) $d(i,t+1) = f\left(d(i,t),\ x'(i,t+1)\right)$

$d(i,t)$ \ $x'(i,t+1)$	0-4	5-9	10-15
0	0	0	2
1	0	1	3
2	1	2	4
3	2	3	5
4	3	4	6
5	4	5	7
6	5	6	7
7	6	7	7

7) $$x[i,t+1) = \begin{cases} \text{an externally controlled value, when the neuron } i \text{ is a stimulated receptor} \\ X(x'(i,t+1), d(i,t+1)) \text{ when neuron } i \text{ is not a stimulated receptor} \end{cases}$$

Table of $x(i,t) = X(x'(i,t), d(i,t))$

x' \ d	0	1	2	3	4	5	6	7
0	0	0	0	0	0	0	0	0
1	1	1	1	1	1	1	1	0
2	2	2	2	2	1	1	1	0
3	3	3	3	2	1	1	1	0
4	4	4	4	3	3	2	1	1
5	5	5	4	4	4	3	2	1
6	7	6	6	4	4	3	2	1
7	7	7	7	6	5	4	2	1
8	8	8	8	7	5	5	2	1
9	9	9	9	7	5	5	2	1
10	10	10	9	8	6	5	3	1
11	11	11	11	8	6	5	3	1
12	12	12	12	9	6	6	3	2
13	13	13	12	9	7	6	3	2
14	14	13	13	10	7	6	4	2
15	15	15	13	10	7	6	4	2

8) $$\bar{x}(i,t+1) = \left(1 - \frac{1}{m}\right) \bar{x}(i,t) + \frac{x}{m}(i,t+1)$$

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1. Brink, F. Jr. "Excitation and Conduction in the Neuron" and "Synaptic Mechanisms". pp. 50-120 in Handbook of Experimental Psychology, Ed. by S. S. Stevens, John Wiley and Sons, Inc., New York; 1951.

2. Eccles, J. C., The Neurophysiological Basis of the Mind, Oxford: The Clarenden Press, 1953.

3. McCulloch, W. S., and Pitts, W., "A Logical Calculus of the Ideas Immanent in Nervous Activities", Bull. Math. Biophysics, vol. 5, pp. 115-133, 1943.

4. Kleene, S. C., "Representation of Events in Nerve Nets in Finite Automata", in Automata Studies, Annals of Mathematics Studies, No. 34. Ed. by C. E. Shannon and J. McCarthy. Princeton: Princeton University Press, 1956.

5. Hebb, D. O., The Organization of Behavior, New York: John Wiley and Sons, Inc., 1949.

6. Ref. (5), p. 62.

7. Ref. 2, and notice that Hebb's postulate (Ref. 6) is not necessarily related closely to Eccles "post-tetanic potentiation". On p. 196 Eccles shows the effect of a million volleys (Fig. 6A, 36 minute curve) and this is much more severe than is relevant for the present discussion.

8. Craik, K. J. W., The Nature of Explanation, Cambridge: The University Press, 1952.

9. Farley, D. G., and Clark, W. A., Proceedings of the Western Joint Computer Conference, 1955.

0	1	2	3	4	5	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31
32	33	34	35	36	37	38	39
40	41	42	43	44	45	46	47
43	49	50	51	52	53	54	55
56	57	58	59	60	61	62	63
64	65	66	67	68			
PROGRAM							

Fig. 1 - Allocation of memory.

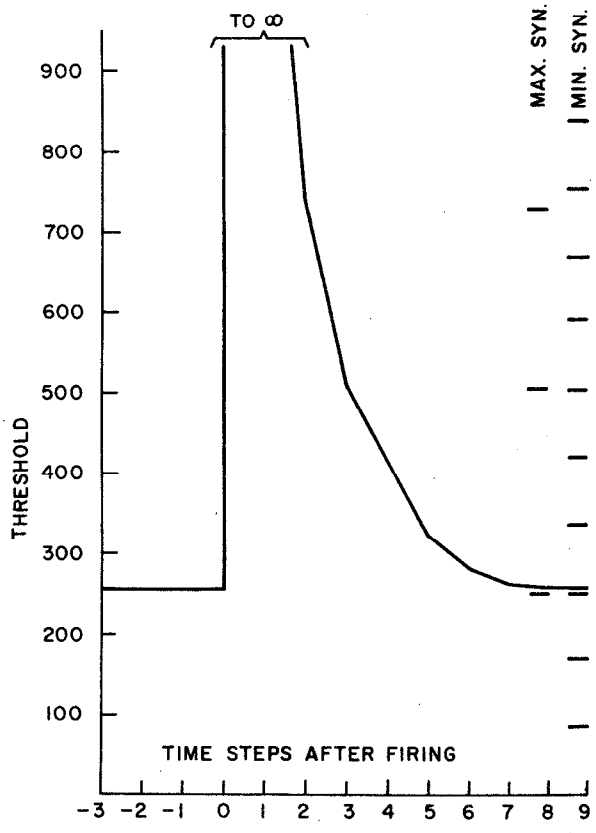

Fig. 2 - Threshold curve.

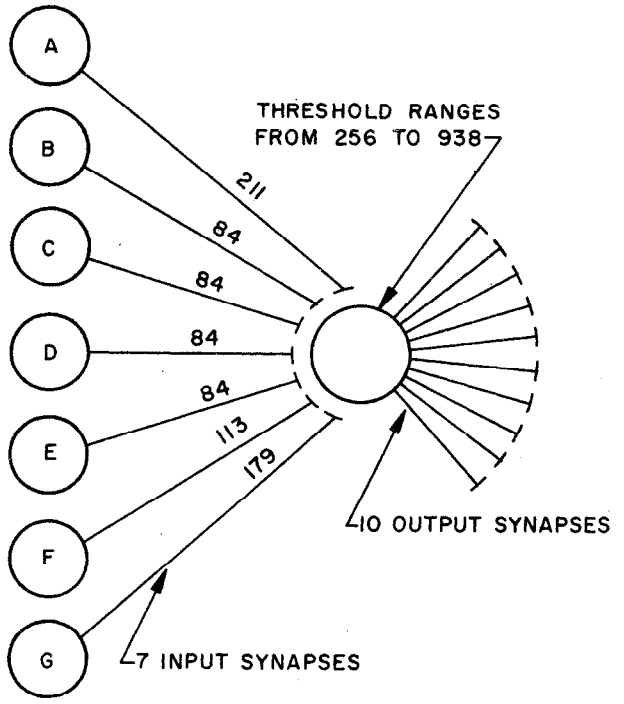

Fig. 3 - Example of a simulated neuron and its connections.

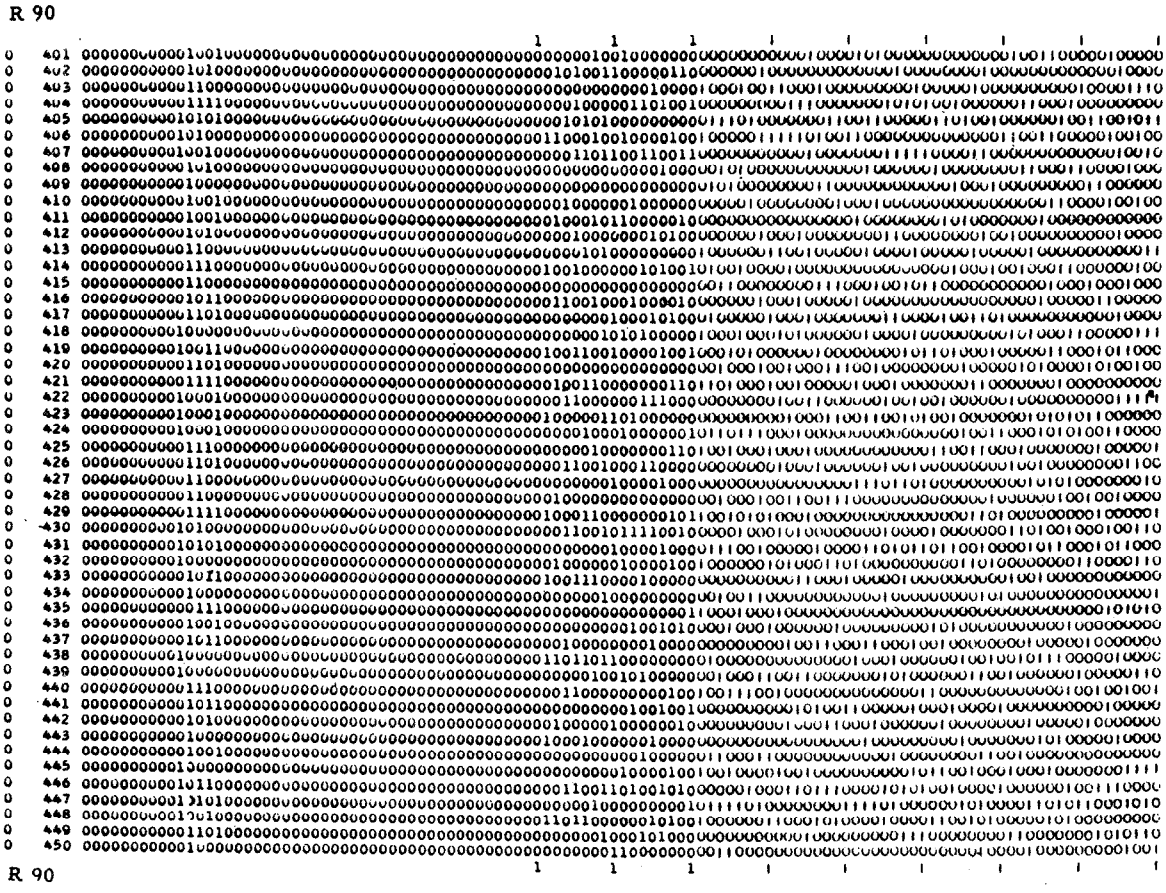

Fig. 4 - Firing pattern of 64 neuron for 50 time steps showing diffuse reverberation.

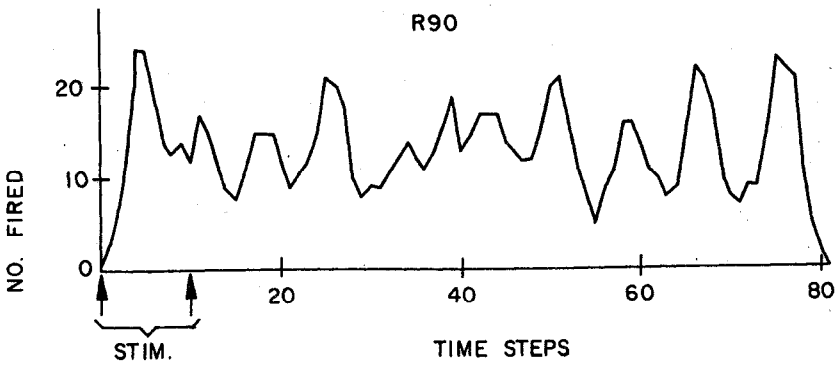

Fig. 5 - Number of neurons firing at each time step showing diffuse reverberation.

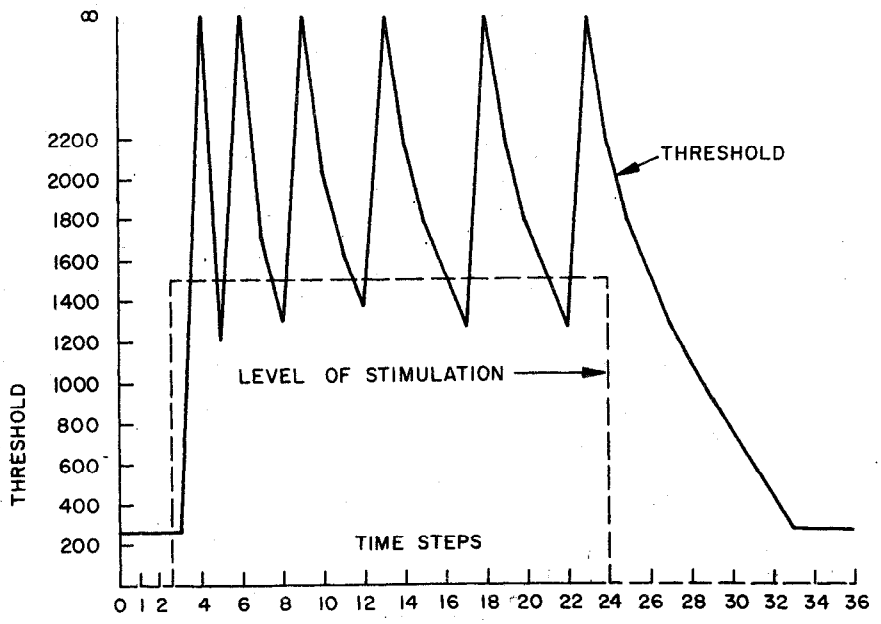

Fig. 6 - Threshold as a function of time.

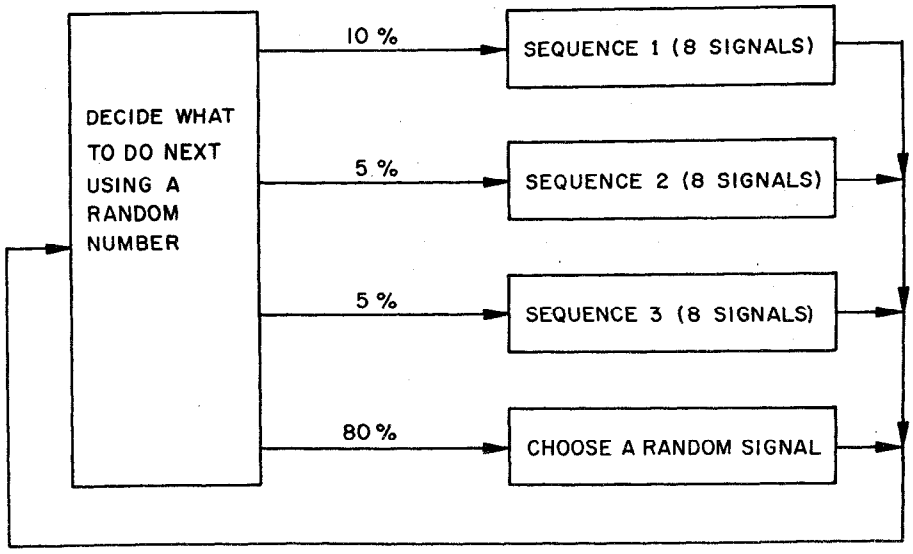

Fig. 7 - Environment.

Time Step	Total Neurons Firing C Cntl. Run	A N40 Sup.	B N61 Sup.	D N70 Sup.	Different Neurons Firing A·C	B·C	D·C
151	31	30	30	30	1	1	1
152	30	30	28	28	0	2	2
153	28	29	27	27	1	3	5
154	30	31	29	31	1	9	5
155	31	31	28	30	2	19	9
156	33	34	26	30	3	27	17
157	30	29	31	31	5	37	19
158	31	26	32	29	9	37	16
159	32	30	32	34	14	32	22
160	34	32	33	38	20	35	22

Three separate runs are represented in this chart in addition to the control run, C. In run A, neuron 40 was suppressed; in B, N61 was suppressed, and in D, N70 was suppressed.

Fig. 8 - Divergence after suppressing one firing of one neuron.

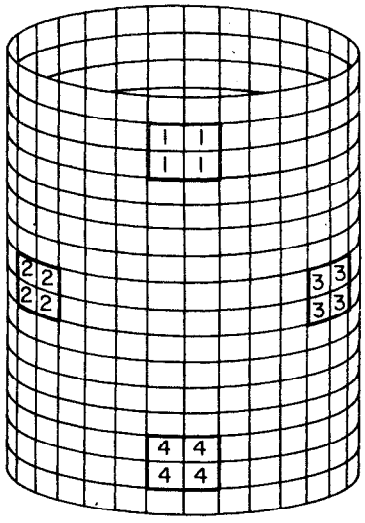

Fig. 9 - Arrangement of neuron in fm model.

Fig. 10 - Illustration of cell assemblies.

00	00	20	50	00	00	00	00	30	00	00	00	00	00
00	10	00	10	06	00	20	00	00	00	00	00	03	00
10	10	20	10	07	50	20	50	00	00	00	07	10	50
00	00	00	00	00	10	00	00	30	00	10	00	03	00
01	00	00	05	00	00	01	00	01	10	00	00	00	00
06	04	02	30	00	01	00	00	00	60	00	00	10	00
00	00	10	01	01	00	70	00	10	07	00	00	00	00
07	07	00	05	00	07	00	00	00	00	00	10	07	07
07	07	07	00	00	00	30	00	04	01	04	04	07	07
00	00	01	00	00	00	00	01	00	00	10	00	00	00
00	00	00	00	11	00	00	00	00	00	00	00	00	00
04	00	07	00	00	00	00	00	42	04	40	50	00	00
00	00	07	00	00	00	01	50	00	00	00	00	00	10
00	10	00	05	00	00	00	00	00	00	40	00	00	05
00	00	01	04	00	00	00	00	00	00	51	40	05	00
00	03	00	05	00	00	00	00	00	00	00	40	00	00