ELECTRONIC SIMULATION OF AN ASSOCIATIVE NEURON

by

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I. INTRODUCTION

The neuron is generally considered to be the basic functioning element of the nervous system. It transmits the information "bits" from point to point in the nervous system and performs certain logic functions with them. Recent research has made it clear that there is a much higher degree of specialization among neurons than has previously been believed. They vary in shape, size, transfer function, and even in some of the more common features described below. The problem of simulation is considerably complicated by this wide variation. It becomes necessary to select those features which have the most widespread significance. Attention will be focused on the logic or associative neurons in this thesis which are the principal neurons in the brain. The sensory (input) neurons and the motor (output) neurons will not be simulated.

There are two basic approaches which may be followed: to create an electrically useful device, or to simulate as closely as possible the physical behavior of a neuron. The former finds application in the construction of bionic computers, the latter in the study of physical systems. This thesis will try to produce a closer simulation to the functional characteristics of a neuron than has been achieved previously in a relatively small unit.

The purpose of biological simulation is to study a complex system where there are many unknowns with a simpler system where the

functional relationships are known. As the model achieves a greater degree of accuracy, the study using it will have more meaning. One should note that the model will be significant only in those aspects for which it has been functionally designed. A new type of stimulus or a variation of a known one will not necessarily evoke a valid response. The advantage of the model is that its behavior can be closely monitored, whereas such monitoring is not possible in the biological system. Thus clues as to how the biological system functions may be obtained by this method.

II. NEURON CHARACTERISTICS

Certain characteristics are common to most neurons and will be discussed here (1). The neuron consists of essentially three parts: a soma (cell body), an axon, and the dendrites (Figure 1). Dendrites serve as the principal receptors of signals called stimuli. A single neuron may have one dendrite or a large number of them. These may branch profusely until their surface area may be several hundred times that of the cell body. Dentrites range from a few microns to a few millimeters in length. They taper away from the soma, particularly as they branch. In composition they resemble the soma. Unlike the axon, they are never covered with sheaths.

The neuron cell body has much in common with other types of cells. It has a nucleus, metabolizes food for energy and repair, and discharges waste products. Because Ribonucleic Acid (RNA) content of nerve cells is among the highest of all cells in the body, they synthesize proteins at a very high rate, much of which is in the form of enzymes to support electrical activity (2). The size of the cell body seems to depend upon the extensiveness of the dendritic and axon system it has to supply.

Although each cell may have many dendritic inputs, it has only one output: the axon. However, this axon may branch numerous times to conduct the signal from the cell to many points. It may vary from a few microns to several feet in length, is essentially

constant in diameter, differs in composition from the cell body, and is often covered with sheaths.

The entire neuron is encased in a very thin membrane across which a potential exists. Membrane thickness is about 0.01 micron and the resting potential varies between 50 and 100 millivolts (mv) giving a potential gradient across the membrane on the order of 10^8 volts per centimeter. It is this potential which gives a neuron the ability to "fire". Two mechanisms are responsible for maintaining the resting potential, an ion pump, and the selective permeability of the membrane. The ion pump brings potassium ions into the cell and forces sodium ions out in equal numbers. Since potassium ions will pass through the membrane more easily than sodium, more of them will leak back out than sodium ions back in. Chloride ions try to follow the potassium ions out, but the membrane will not pass them as readily. A negative charge results on the inside of the membrane with respect to the outside. Complete depolarization, which causes an "action potential", occurs when this resting potential drops below a threshold value of about 50 mv (Figure 2). The membrane looses its resistance to sodium ions and they rush through causing the interior to swing about 30 mv positive with respect to the exterior (3). As one area on the membrane looses its ionic balance, it disturbs the adjacent areas on the membrane. Ions will naturally tend to flow from regions of higher to lower charge density. Thus the depolarization spreads throughout the membrane much as a fuse would burn. The depolarization, or action

potential, lasts from about 0.6 ms to over 1 ms after which the ion pump mechanism restores the polarized state. A period of hyperpolarization during which the interior of the membrane is slightly more negative than in the resting state concludes the firing cycle. This hyperpolarization, called the relative refractory period, is not observed in myelinated fibers to be discussed in the following paragraph. During the recovery period, which lasts about one millisecond, the membrane is refractory, i.e., incapable of further response.

Action potential magnitude is dependent upon the state of the membrane at the region where the stimulus is received and travels with a speed which is independent of the stimulus (4). The speed of transmission is directly related to fiber diameter, except in myelinated fibers. In this case, the fiber surface is insulated by a myelin sheath with periodic gaps called nodes. Since the intervening membrane is insulated, depolarization progresses from node to node and thus the speed is greater. Action potential in the axon is of a all-or-nothing nature. It delivers all the response of which it is capable at the moment without being decremented. The action potential frequently originates at the "axon hillock", a region near the junction of the axon and soma which has a low threshold. Such an action potential spreads back throughout the entire neuron once it occurs.

Dendritic conduction contrasts with the all-or-nothing principle in that depolarization is of a decremental nature there.

A depolarization originating toward the end of a dendrite may be so attenuated by the time it reaches the cell body that all it does there is to create a slight depolarization which effectively lowers the amount of stimulus required to fire the neuron. This has led to the theory that peripheral stimuli are used chiefly to modulate the excitability of a neuron while stimuli nearer to the cell body are used to trigger an action potential quickly (5).

The point where the axon of one neuron makes "contact" with a dendrite or the cell body of another is called a synapse (Figure 3). These synapses are the single most important part of the nervous system. In fact, the brain has been described as a"system of synapses connected by neurons" (6 - original not seen). Most neural properties are determined at or near the synapse. Whereas the theory of synaptic activity presented here has not been conclusively proved, there has not been any serious challenge to its validity by either theory or experimental evidence. Recent findings have tended to strengthen it considerably.

A single axon, or presynaptic fiber, may branch to synapse with several postsynaptic neurons, and branch even farther to make several contacts with each one. These synapses range in size from one micron in diameter to much larger proportions. As much as 40% of the cell body and dendritic surfaces may be covered by these terminals, which seem to be insulated by a mosaic of glia end feet (protrusions from the surrounding glia cells to be discussed later). The presynaptic and postsynaptic membranes are approximately 0.005

micron thick and are separated by a synaptic cleft of about 0.02 micron in width. Small spherules called neurovesicles are found only on the presynaptic side of the synaptic cleft. Their presence indicates a probable mechanism for pulse transmission across the cleft. Neurovesicles are believed to be composed of a transmitter substance which passes into the cleft when an action potential reaches the synapse. Since they exist only on the presynaptic side of the synapse, the property of unidirection transmission can be explained.

Potential changes in particular the action potential, are believed to cause the secretion of these transmitters into the cleft. Here they modify the electrical characteristics of the postsynaptic membrane, causing it to be more or less inclined toward discharge (2). Excitatory transmitters tend to decrease the polarization of the postsynaptic membrane, an excitatory postsynaptic potential (EPSP), increasing the probability and ease of firing. By one of two mechanisms to be discussed later, inhibitory transmitters reduce the ability of a neuron to fire. This is "inhibitory postsynaptic potential" (IPSP). Excitatory and inhibitory stimuli are generated by different neurons and arrive at the postsynaptic membrane in different fibers at different locations (1,2,7,8). An excitatory neuron cannot produce an inhibitory stimulus nor can an inhibitory neuron produce an excitatory stimulus. The difference in function is determined by the type of transmitter substance they emit. Preliminary research indicates that the preferred location for inhibitory synapses is on or near the cell body while the excitatory

synapses may be on either the dendrites or cell body (8,3,7).

Transmitter chemicals do not remain in the cleft indefinitely. They either diffuse away or are neutralized by some unknown process causing the EPSP's to decay with a time constant on the order of 4 ms. A single incident pulse seldom being sufficient to cause an action potential in the postsynaptic membrane, several pulses generally are required. Thus the synapse performs a temporial summation process. Summation may also be spatial in nature, inputs from more than one presynaptic neuron being required to reduce the membrane potential to the threshold. Small subthreshold disturbances of a graded nature also cause a secretion of transmitter chemicals proportional to their magnitude. These small EPSP's are cumulative and may sum to the degree necessary to discharge the postsynaptic membrane. When no action potential results, there is no refractory period and the net effect is that of "priming the pump". Only a brief period of hyperpolarization follows a subthreshold stimulus (2). Summation is spatial only to a limited degree. Neither EPSP's or IPSP's propagate very far from their point of origin. Therefore, the neuron has a patchwork of activities, any set of which may sum to the threshold independent of the others (2,4).

Recently a great deal has been learned about the characteristics of inhibition. Two, and possibly three, types must be considered: presynaptic inhibition, postsynaptic inhibition and, perhaps, a lowering of the background which stops the priming action (9).

Presynaptic inhibition is the depolarization of the terminal

portion of the presynaptic fiber (7,10,11,12). This causes a lowering of the synaptic excitatory action of these fibers. An inhibitory neuron causes this depolarization, which spreads from the postsynaptic junction in a decremental manner. The time course of presynaptic inhibition is a brief latent period followed by a quick rise to the maximum depolarization summit and a considerably longer decay (Figure 4). Areas close to the synaptic knob show a rapid onset of depolarization. A typical range of values would be a latent period of 2 to 12 ms, a rise time of 5 to 20 ms, and a decay from the summit of about 100 ms.

Postsynaptic inhibition, frequently called hyperpolarization (7,10), effectively raises the threshold of the neuron by increasing the potential across the membrane. As before, an inhibitory interneuron is the mediator. In the spinal chord its time course is usually brief, 10 to 20 ms. However, in the thalamus, hippocampus, cerebellum, and neocortex, hyperpolarization is at least 100 ms in duration. The same brief rise to summit of maximum hyperpolarization is observed as for presynaptic inhibition, the maximum occuring in about 20 ms, with perhaps a latent period of 8 to 10 ms.

Memory is a problem concerning which there has been comparatively little experimental evidence to date. There are several theories concerning it, but the one generally considered most plausible is that of synaptic adaptation. The supporters of this theory hypothesize that once a synapse has passed a pulse, it will be more likely to pass the next one, and conversely. Although no mechanism has been

demonstrated, possibilities are that the presynaptic membrane becomes more permeable to the transmitter chemicals or that the postsynaptic membrane may be depolarized more easily.

Recently, other researchers have speculated that memory may be a complex chemical reaction. One possibility along this line is linked with the Ribonucleic acid configuration (2). The brain stores about 10^{15} bits of information during a lifetime. RNA complex structure could encode with ease 10^{15} information bits or more. Protein is synthesized on the RNA molecules and could possibly dissociate more rapidly with a familiar pulse pattern, facilitating the emission of transmitter substances.

Glial cells surround each neuron. These cells, which outnumber neurons 10 to 1, act as a medium for all blood-neuron transfers, since the neurons are not themselves in contact with the blood vessels. Experimental evidence has shown that the glial cells and neurons are energetically linked together in a complimentary fashion, the biochemical changes in adjoining glial and neuron cells being opposite. This has led to the conclusion that glia cells may control the sensitivity and firing thresholds of the neurons.

Since no single transfer function holds for all synapses, coding in the nervous system also poses a problem. In the past it was felt that a larger stimulus would produce a higher frequency of discharge. For many areas of the nervous system this holds true, but the more centrally the neuron is located, the less this rule applies (4). Elliot (13) states that once fired, a neuron will fire

several more times spontaneously. Except for this feature, a pulse train would soon die out because one pulse is generally insufficient to fire a postsynaptic neuron. Many neurophysiologists have concluded that messages are differentiated by their distribution in space, as well as in time (4); thus the pattern of excitation and inhibition is as much a factor as any coding of inputs.

III. CHARACTERISTICS DESIRABLE FOR SIMULATION

There are many features which may be included in a simulation. Economics, size, and circuit complexity tend to limit what can be done practically. An experimenter must compromise by choosing those features he considers most important and omitting the others. The following are considered to be the desirable characteristics for this simulation.

A. The neuron should be capable of receiving stimuli from a large number of other neurons with input characteristics which are independent of the number of neurons which may stimulate it. These stimuli may arrive in coincidence, near coincidence, or widely separated in time. There should be little cross-talk between inputs. An input should be adjustable to give a variation in the effect, or "weight", which it may have.

B. The neuron should be capable of receiving inputs of three kinds: an excitatory input which stimulates the neuron toward firing and decays with a time constant of about four milliseconds; an inhibitory input which increases the amount of stimulation required to fire the neuron; and an inhibitory input which lowers the amplitude of the output pulse. The time course of the latter two is discussed in the section on neuron characteristics.

C. A summation with respect to time should be made of the input stimuli with those of nearer time proximity having greater effect. This summation is compared to a fixed threshold value to determine whether the neuron will produce an output pulse. The condition for an output pulse may be expressed as

$$Q - \sum_{i=1}^{n} E_i + \sum_{j=1}^{m} I_j \leq T \qquad (1)$$

where Q = Quiescent point of the summation circuit,

E_i = Instantaneous magnitude of excitation delivered through input "i",

I_j = Instantaneous magnitude of inhibitory stimulation delivered through input "j",

T = Threshold value.

D. An output pulse is generated whenever the summation circuit voltage is below the threshold value. The duration of the pulse should be fixed in the range of 0.6 ms. to over 1.0 ms. It is followed by a refractory period of at least one millisecond, during which the neuron cannot fire. A pulse is followed by a period of slightly decreased sensitivity which decays out in about 100 ms. When the neuron recovers from the refractory period, it will fire again if the summation circuit is still below threshold. This will continue until the summation circuit voltage has decayed to a point higher than threshold. If a stimulus does not have sufficient magnitude to fire the neuron, it will be

decremented and passed on to the output as a small pulse. This is called subthreshold response.

Such features as a variable delay in the pulse transmission, fatigue, and automatically variable weighing to achieve synaptic adaptation have been omitted in order to reduce the circuit complexity. The major addition of the simulation to be presented here is in the inhibition inputs. Previous simulations have not allowed for a presynaptic inhibitory input and have let the postsynaptic inhibition have the same time characteristics as an excitatory input (14,15,16,17,18,19).

IV. SIMULATION

A. Summary of Operation

A block diagram of the circuit is shown in Figure 5. The input pulse is in the negative direction for all inputs, excitatory or inhibitory. Synaptic weighting is accomplished by the use of input resistors which supply the base current of a common collector stage. This stage prevents the inputs from influencing the characteristics of the summation circuit. The summation circuit is at a quiescent voltage level which is lowered by the input pulse. It then decays back up toward its quiescent value. If the voltage is lowered sufficiently to turn on the threshold detector, it in turn starts the pulse generator, which will continue to produce pulses as long as the summation circuit voltage remains below the threshold value. These pulses are fed to the output emitter follower and passed on to the next neuron(s). Subthreshold response may be passed to the output if desired.

The hyperpolarization, or postsynaptic inhibition, input raises the resting voltage of the summation circuit, increasing the stimulation required to fire the neuron. A pulse applied to this input invokes a response in the summation circuit of the form

$$E_s = A\ e^{-\frac{t}{\tau_1}} (1-e^{-\frac{t}{\tau_2}}) \qquad (2)$$

where E_s is the voltage at the summation circuit (with no excitatory

inputs) τ_1 is the time constant determined by the inhibition circuit (about 50 ms.), τ_2 is the normal decay of the summation circuit (4.7 ms.), and "A" is a constant determined by the peak voltage which would be obtained if the summing capacitor were disconnected (Figure 9).

The depolarization, or presynaptic inhibition, circuit decreases the amplitude of the output pulse by an equation of the form

$$E_o = E_m \left[1-Be^{-\frac{t}{\tau_3}} \left(1-e^{-\frac{t}{\tau_4}}\right)\right] \qquad (3)$$

where E_o is the output pulse amplitude, E_m is the normal pulse amplitude, "B" is a constant similar to "A" in equation (2), τ_3 is a time constant determined by the circuit (about 40 ms.), and τ_4 is a shorter time constant of about 6 ms (Figure 12). This response is triggered by a pulse at the depolarization input.

A fraction of the output pulse may be fed back into the hyperpolarization input in order to achieve a relative refractory period.

An emitter follower provides a low impedance output to drive successive stages. A complete schematic diagram of the actual circuit is given in Figure 6. This circuit is broken down into its major sections in Figures 7, 10, 11, and 13.

B. Circuit Description

1. The input, summation, and hyperpolarization circuits are shown in Figure 7. In the quiescent state T2 controls the current

through R1 to hold point "A" at 5.4 volts. Resistors R3 and R4 provide the biasing network for T2. Isolation is provided by T1, which is in a common collector configuration. This provides for a high input impedance to the model which will not load the input signal source excessively. T1 is normally cut off or very slightly conducting. An input signal which has a negative swing sufficient to forward bias the emitter-base junction will turn T1 on and lower the voltage at point "A". This voltage will then decay back toward its quiescent value with a time constant R_1C_1 . The extent to which the voltage at "A" will be depressed by an input signal of a given amplitude is determined by the synaptic weighting resistor R_e. For example, if R_{e1} is 220 K-ohm, an input signal which drops from above 5.4 volts to zero (created by a pulse generator) will lower the voltage at "A" to 4.2 volts (Figure 8), after which it will rise toward 5.4 volts with a time constant of 4.7 ms. If a shorter or longer decay time is desired, it may be accomplished by altering C1.

C1 is called the summing capacitor because, if a second pulse arrives before the effects of the first have decayed, the depression the second pulse makes in the voltage at "A" is added to what is already present. From this comes temporal summation.

Hyperpolarization is accomplished by decreasing the current through T2. A negative-going pulse fed into the hyperpolarization input will discharge C2 through the appropriate weighting resistor, R_h (Figure 6). The drop in voltage across C2 is transmitted through R5 to the base of T2 causing it to conduct less. As a result, the

voltage at "A" trys to rise sharply. Capacitor C1 prevents this from happening, since its voltage cannot change instantly. The curve of equation (2) results (Figure 9). R5 is used to adjust the time constant of the longer decay as desired. With an imput pulse which drops to zero volts from above 1.8 volts (obtained with a pulse generator and fed in through a diode) and an R_h of zero ohms, the voltage of point "A" will rise to 6.9 volts in 15 ms. and decay out in about 150 ms. (Figure 9). If a greater effect is desired, a bypass capacitor may be connected across R2. The quiescent voltage across C2 is 1.8 volts, so a negative-going pulse delivered to the hyperpolarization input which drops below that value is required to actuate the circuit. This resting voltage may be raised by adjustment of R2, R3, and R4 if this is desirable. C2 also acts as a summing capacitor for inhibitory inputs.

2. The threshold and pulse generation circuits are shown in Figure 10. A zener diode holds the emitter of T3 at 4.2 volts. Therefore T3 is normally cut off, since the voltage at "A" to which the base resistor R6 joins is above this value. As the voltage at "A" drops, T3 turns on, raising the voltage at point "B", which in turn allows the relaxation oscillator to start operation. Because variations in Beta of T3 may allow some difference in the voltage change at "B" resulting from a given change at "A"-or if the threshold needs to be changed - R6 may be made variable in order to set accurately the voltage at "A" which will start the oscillator.

In the resting state the voltage at "B" (2.6 volts) is determined

by the current flowing in R7 plus that flowing in D2. As T3 turns on, the rise in voltage at "B" lowers the current flowing through R10, thus raising the voltage at the emitter of unijunction transistor T4. Resistor R11 in series with T4 determines the voltage at which the emitter of T4 will conduct and so may also be used to vary the threshold voltage (voltage at "A" required to start the relaxation oscillator). Using a value of 33 KΩ for R6, the voltage at "A" must fall to 3.2 volts before the relaxation oscillator will turn on.

When the voltage at the omitter of T4 rises sufficiently for the emitter to conduct, the resistance from the emitter to base one of the unijunction suddenly drops, lowering the voltage at point "C" and on the emitter. This back biases D3, and the discharge of C3 through R9 back biases D2. Capacitor C3 will continue to discharge through R9 until its voltage is low enough for D3 to again conduct. Then C3 will start to charge through R10, and the emitter of T4 will stop conduction. As C3 continues to charge, D2 will become forward biased, and the voltage across C3 will charge toward its equilibrium value. If T3 is still conducting sufficiently, the voltage at the emitter of T4 will rise enough to conduct again and the cycle repeats.

Unijunction transistor T4 has only two states: emitter not conducting, with point "C" at 5.8 volts; or emitter conducting, with point "C" at 4.8 volts. The duration of the negative pulse is determined by the time required for C3 to discharge to the conducting emitter voltage, and the spacing between pulses is determined by the time required for C3 to charge back up to the firing voltage.

When point "A" is just below the threshold voltage, the duration of the negative pulse is 0.7 ms. and the refractory period between pulses is 1.7 ms. As the voltage at "B" continues to rise because of a decrease at "A", the charging time for C3 will decrease, shortening the refractory period to 1.0 ms. The neuron normally operates with the shorter refractory period, since the transition is made over a small change in voltage at "A".

A square wave of one volt amplitude is the resulting wave at point "C". Its pulse duration corresponds to the action potential duration of the biological neuron, and the spacing between pulses corresponds to the refractory period of the biological neuron. Since a stronger stimulus will overcome the hyperpolarization following a pulse more quickly than a weaker one, the variation in refractory period also corresponds to biological behavior. The pulse output will continue as long as the voltage at "A" is below threshold.

3. Figure 11 shows the depolarization and amplification circuit. The depolarization circuit cuts the pulse amplitude transmitted to the output by a clipping technique. Resistor R12 couples the negative square wave from the oscillator (point C) to the base of T5, which amplifies and inverts the pulse. If no clipping is present, the voltage swing at "D" rises from a quiescent value of 6.8 volts up to 10 volts. Bypass capacitor C4 prevents negative feedback, holding the emitter essentially constant by its long time constant. The emitter of T5 is at about 4.2 volts so that it will swing almost to cutoff.

Clipping is accomplished by lowering the voltage at point "E" so that diode D4 conducts and the upper portion of the pulse is cut off by C5. The response at point "D" due to a pulse input to the depolarization circuit is shown in Figure 12 (Summation circuit set so that the neuron fires continuously).

A pulse delivered to the depolarization input decreases the current flowing in T7 and causes a sharp rise in the voltage at its collector. This rise is coupled to the base of T6 by R17 causing it to conduct more and to lower the voltage at point "E". Capacitor C5 prevents a sudden drop in the voltage at "E", providing the shorter time constant in equation (3). Resistor R22 in conjunction with C8 is used to obtain the longer time constant. Bypass capacitor C6 and C7 allow a greater voltage swing at the collector of T6 and T7.

A latent period before the onset of depolarization may be achieved by maintaining the voltage at "E" somewhat higher than the maximum pulse voltage at "D". Therefore it takes a brief period for the voltage at "E" to drop sufficiently for D4 to conduct. The degree of clipping and the equilibrium point at "E" may be adjusted by R15, R17, R20 and R21.

4. The pulse inversion, amplification, and output stage is shown in Figure 13. Resistor R23 couples the positive pulse at "D" to the base of T8 where it is inverted and amplified slightly to provide a sufficient swing at point "F". A resting level at "F" of 6.4 volts determines the output voltage of a little over 5 volts. When a negative pulse appears at "F" the tandem connected emitter

follower transmits it to the output terminal which will drop to zero volts. If clipping has occurred at "D", the output swing will fall short of zero by an amount dependent on the degree of clipping. Diode D5 prevents interaction of the input of a neuron with the outputs of others when more than one presynaptic neuron is connected to the next neuron in a system. It also allows only the portion of the output pulse which falls below the voltage of the next input to be transmitted.

5. Two optional characteristics are provided for in the simulation: subthreshold response, and a slight hyperpolarization effect after a pulse output. Subthreshold response is achieved by connecting point "A" to point "D" with a resistor of one meg-Ω and a capacitor of 470 μμf in series (Figure 6). This gives an output for subthreshold response as shown in Figure 14-a. No delay is available for the subthreshold response, so it proceeds an output pulse when the stimulus is sufficient to fire the neuron (Figure 14-b).

Some fraction of the output pulse may be fed back to the hyperpolarization input by means of a diode and a resistor of around 47 K-Ω as indicated in Figure 6. This will give the hyperpolarization response following a suprathreshold stimulus (sufficient to fire the neuron) as shown in Figure 16. The wave shown is observed at point "A" and slightly increases the stimulation to fire the neuron until it decays out.

C. Discussion of Circuit Response

The synaptic weighting resistor of any particular input will determine the voltage depression produced at point "A". If the weighting resistor has the value of 220 K-Ω, one pulse is not sufficient to fire the neuron. Using input pulses which swing from above the voltage at "A" down to zero (maximum amplitude allowed at the output of the model) and a pulse duration of 0.7 ms., a pulse repetition rate of 294 pulses per second (pps) is required to fire the neuron. Figure 16 shows the relationship of the input pulse to the response at "A" and to the output pulse. The minimum point on the "A" voltage curve is the threshold value of the neuron. Under these conditions, the output pulse is delayed by the width of the input pulse. As the pulse repetition rate is increased at the input, this delay is shortened until the neuron is firing at its maximum rate, 590 pps. At this point delay ceases to have meaning. The biological neuron has a fixed delay for any one input, but it did not seem worthwhile to simulate this feature.

If the synaptic weighting resistor is lowered to 120 K-Ω, a different feature may be observed: one input pulse would trigger the neuron. The input could be set to yield only one output pulse. An increase in the input pulse magnitude yields a second output pulse, and a further increase yields three output pulses. Using this circuit, three output pulses is the maximum obtainable from a single input pulse. This behavior is shown in Figure 17. If feedback into the hyperpolarization circuit is used, the number of input pulses required to maintain a regular output pulse is increased.

The effect at the output of a single maximum amplitude pulse delivered to the hyperpolarization input is shown in Figure 18. A maximum amplitude pulse with a repetition rate sufficient to fire the neuron at its maximum output rate was fed into the input of the neuron through a 220 K-Ω resistor. The pulse into the inhibition input was at the origin (time zero) of Figure 18. Firing ceased for about 20 ms. before slowly returning to the maximum rate.

When a maximum amplitude pulse was delivered to the depolarization input, a variation as shown in Figure 19 resulted. The neuron was stimulated to fire continuously, and the inhibition pulse was delivered at the origin of Figure 19. The output pulse amplitude was decreased from 5 volts to 2.6 volts, and then it rose back to 5 volts. If it is desired, the circuit may be adjusted to reduce the output pulse amplitude all the way to zero. This may be accomplished as described in the depolarization circuit description.

V. REMARKS

The circuit presented here duplicates most of the essential characteristics of an associative neuron. It has as advantages that it includes the inhibition responses, that its responses may be adjusted over a wide range, and that it requires only one power supply of 12 volts. The author used an entirely new circuit design which was dictated by the demands of adding the two inhibition effects.

It is suggested that the inhibition effects may be much more simply achieved by the use of field effect transistors when they become available at reasonable prices.

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Suggested Further Reading

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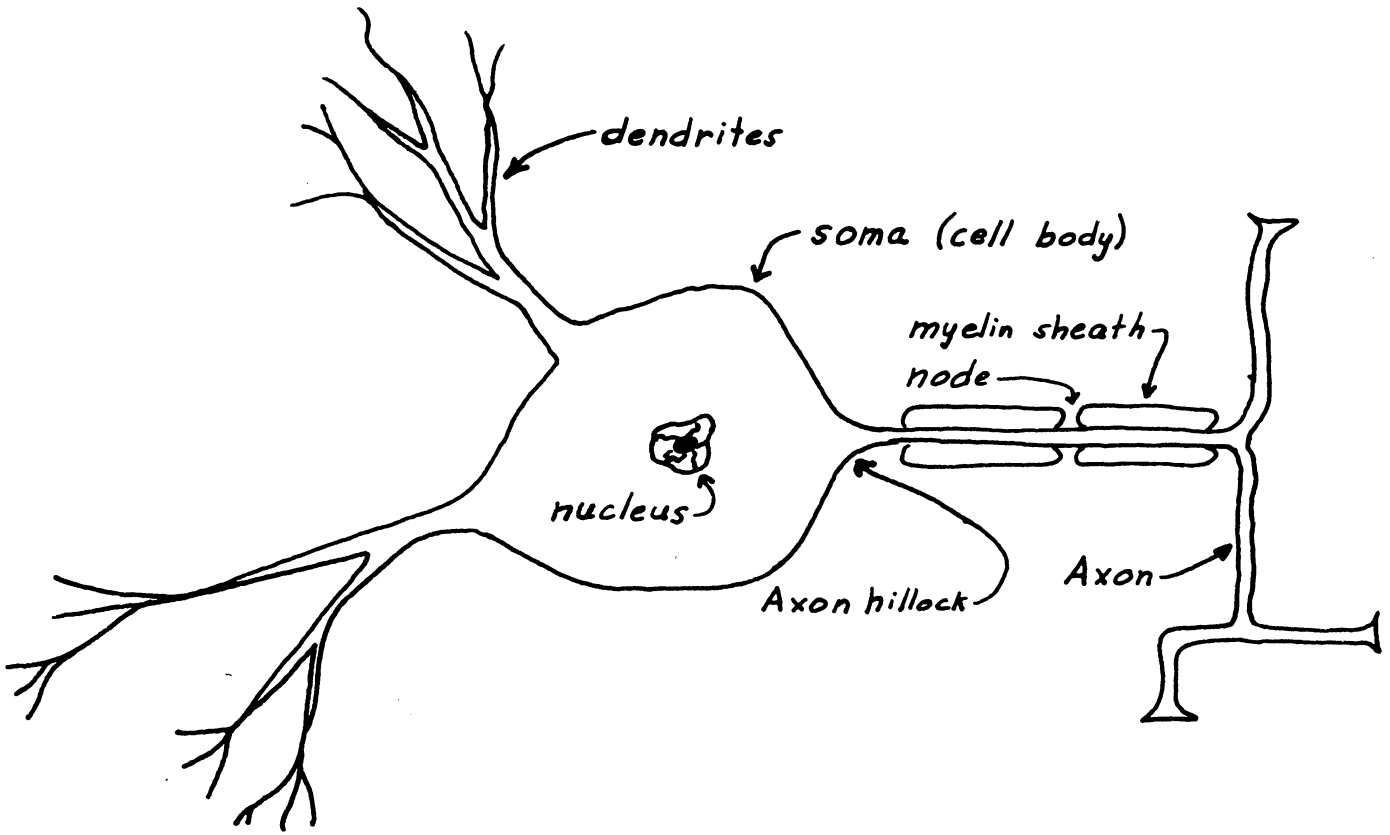


Figure 1.

Neuron

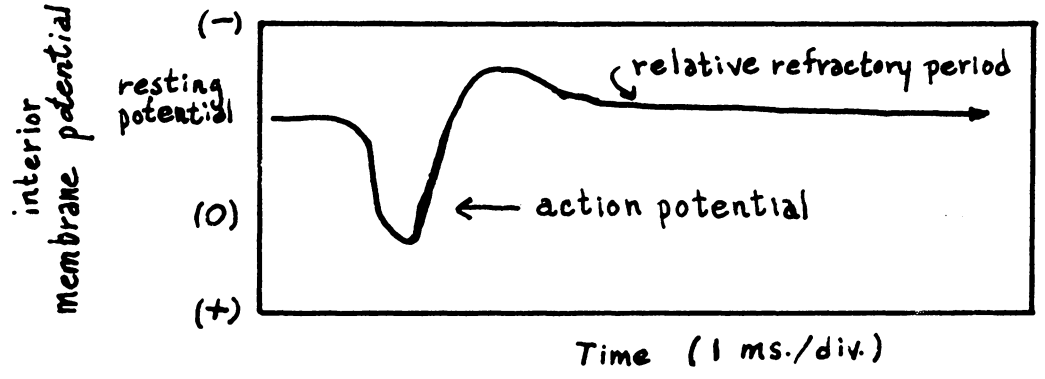


Figure 2
Firing Cycle

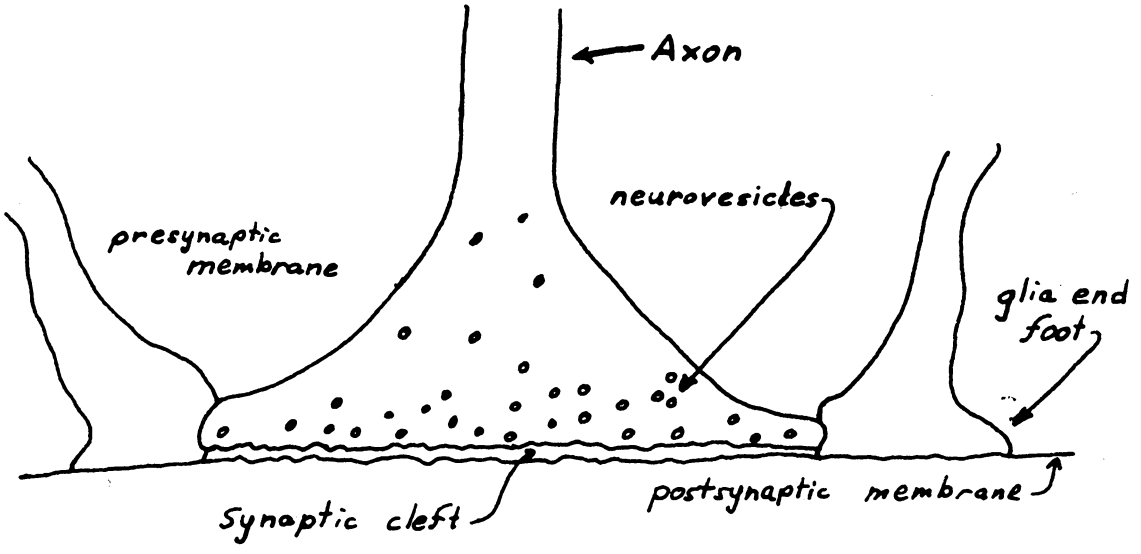


Figure 3
Synapse

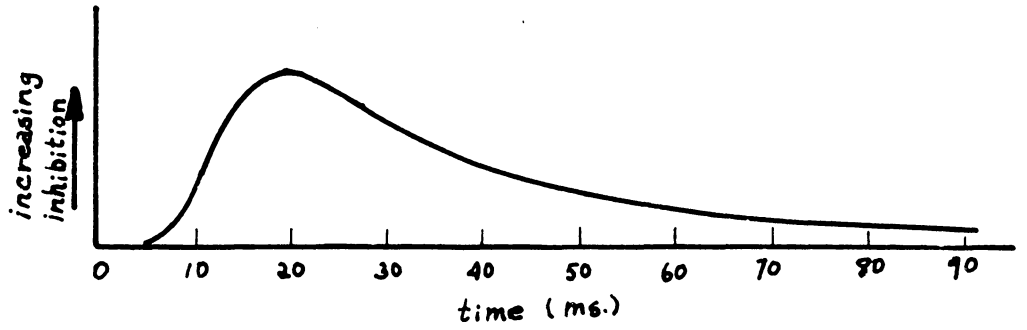


Figure 4. Inhibition Time Course (approximate)

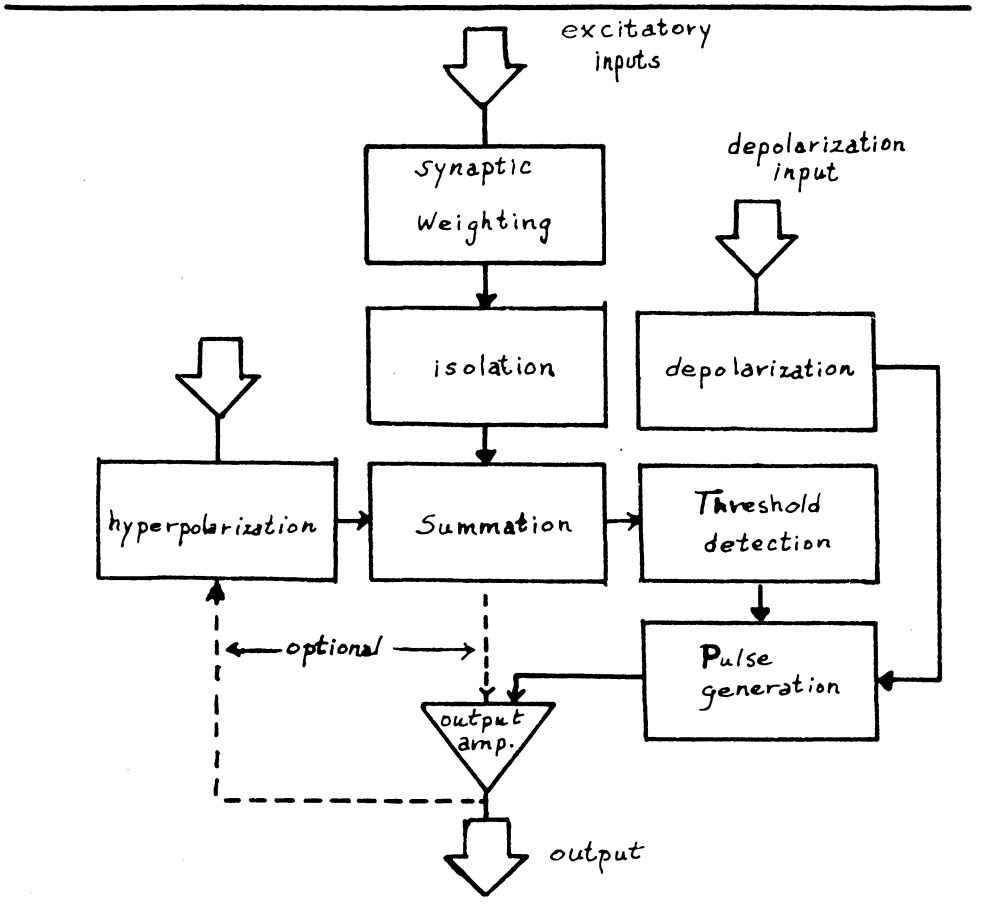


Figure 5. Block Diagram of Simulation.

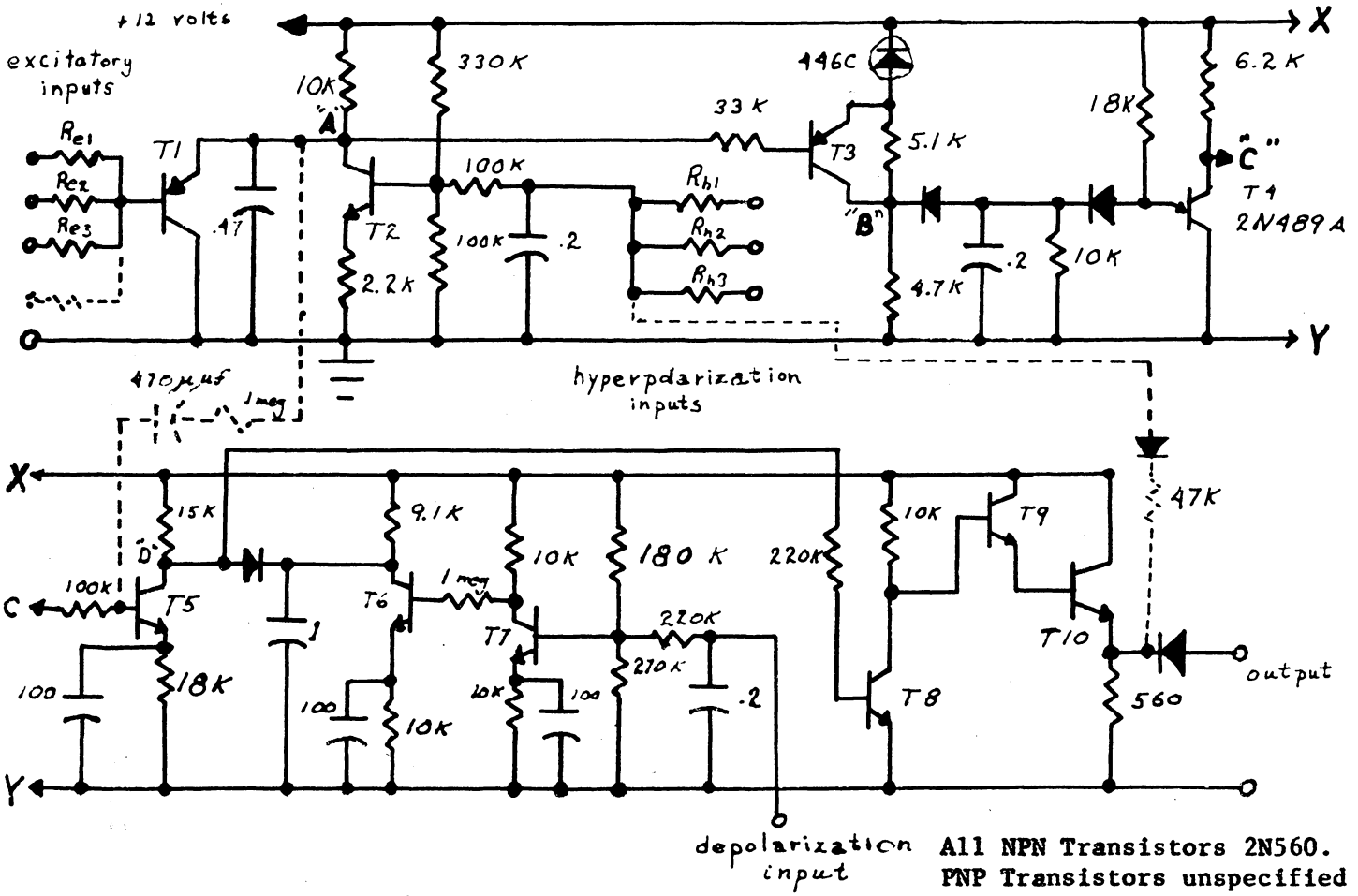


Figure 6. Complete Circuit.

All NPN Transistors 2N560.
PNP Transistors unspecified (Beta = 30)
All diodes are general purpose, silicon.
All capacitors in microfarads.

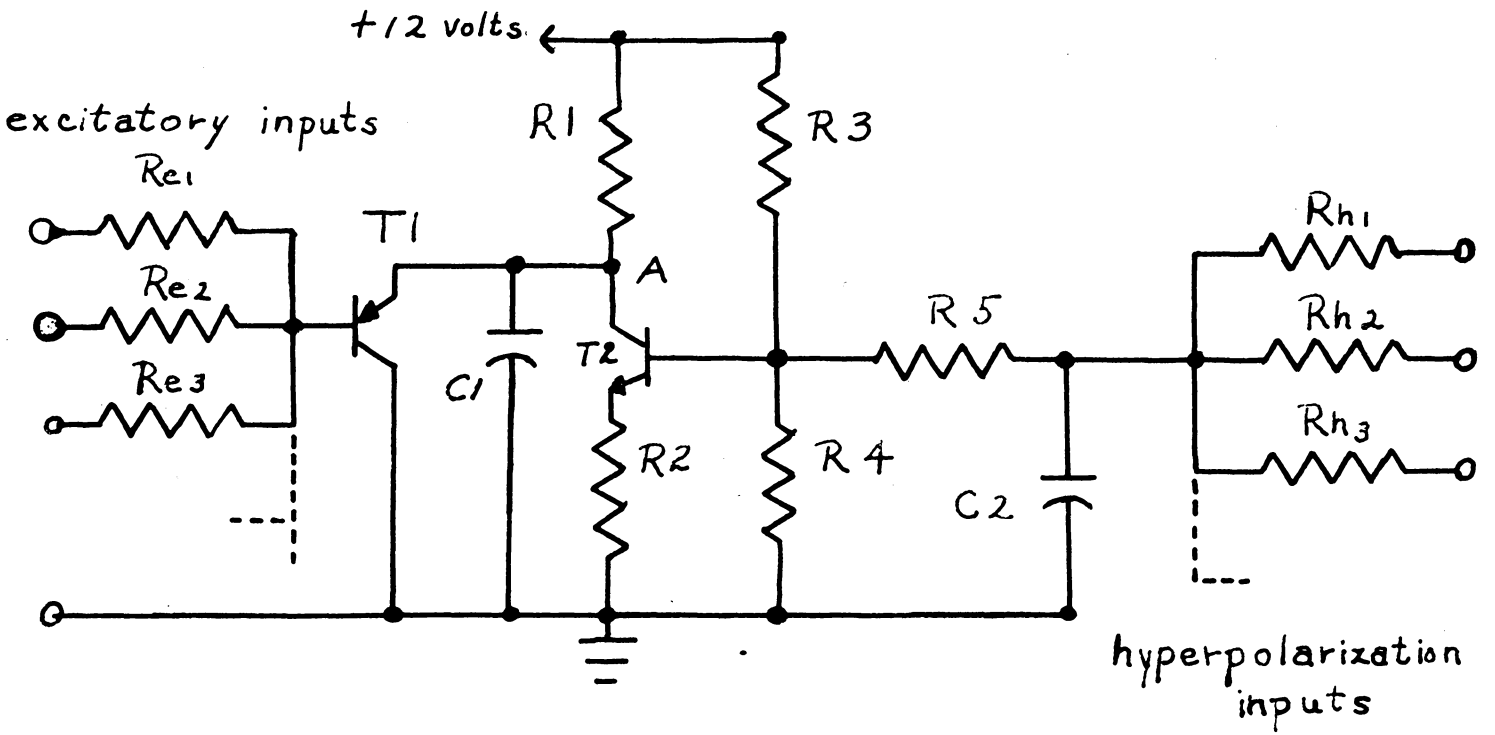


Figure 7. Input, Summation, and Hyperpolarization Circuit.

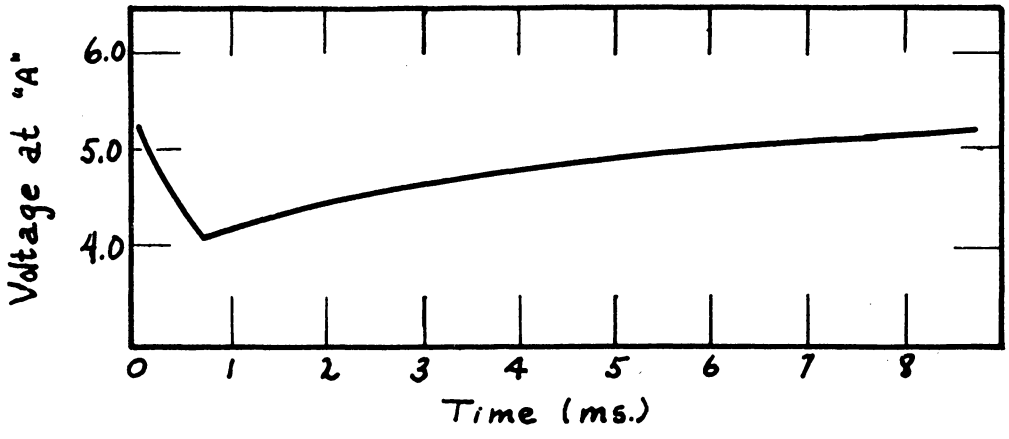


Figure 8. Summation Circuit Response to Stimulation, Point "A".

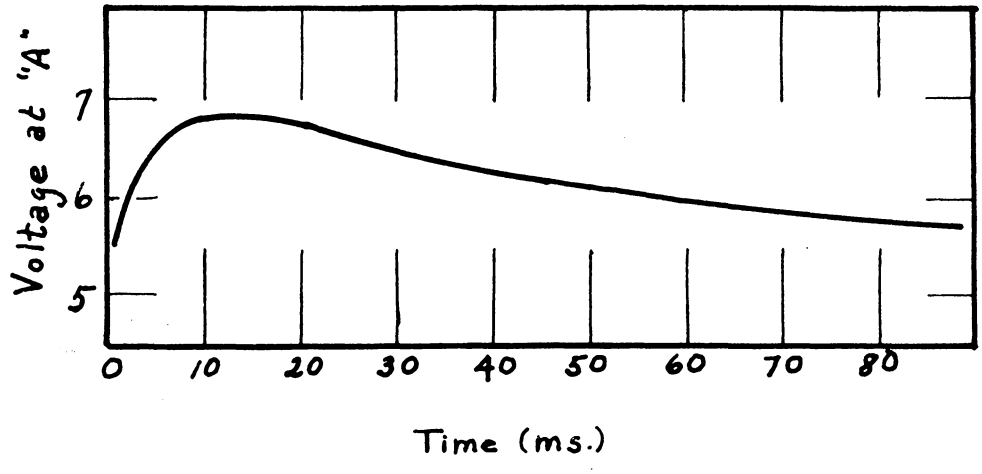


Figure 9. Hyperpolarization Response, Point "A".

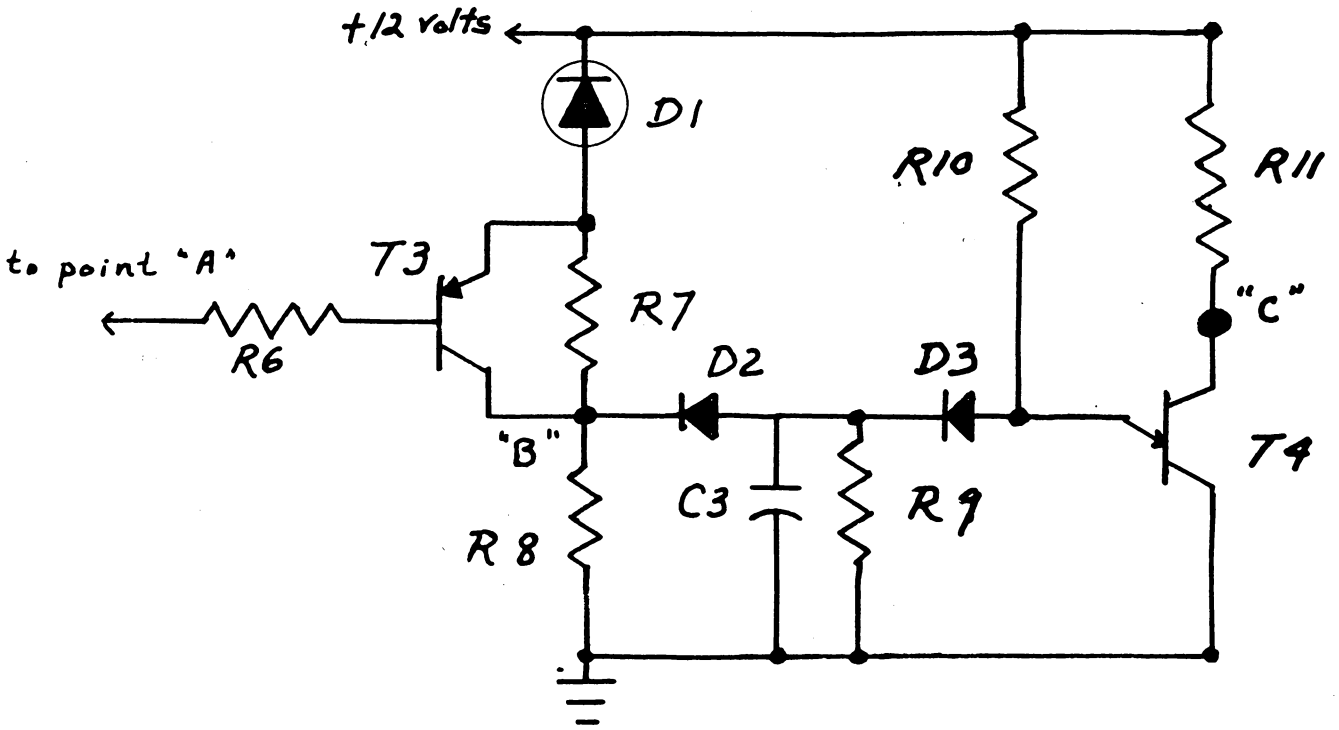


Figure 10. Threshold and Pulse Generation Circuit.

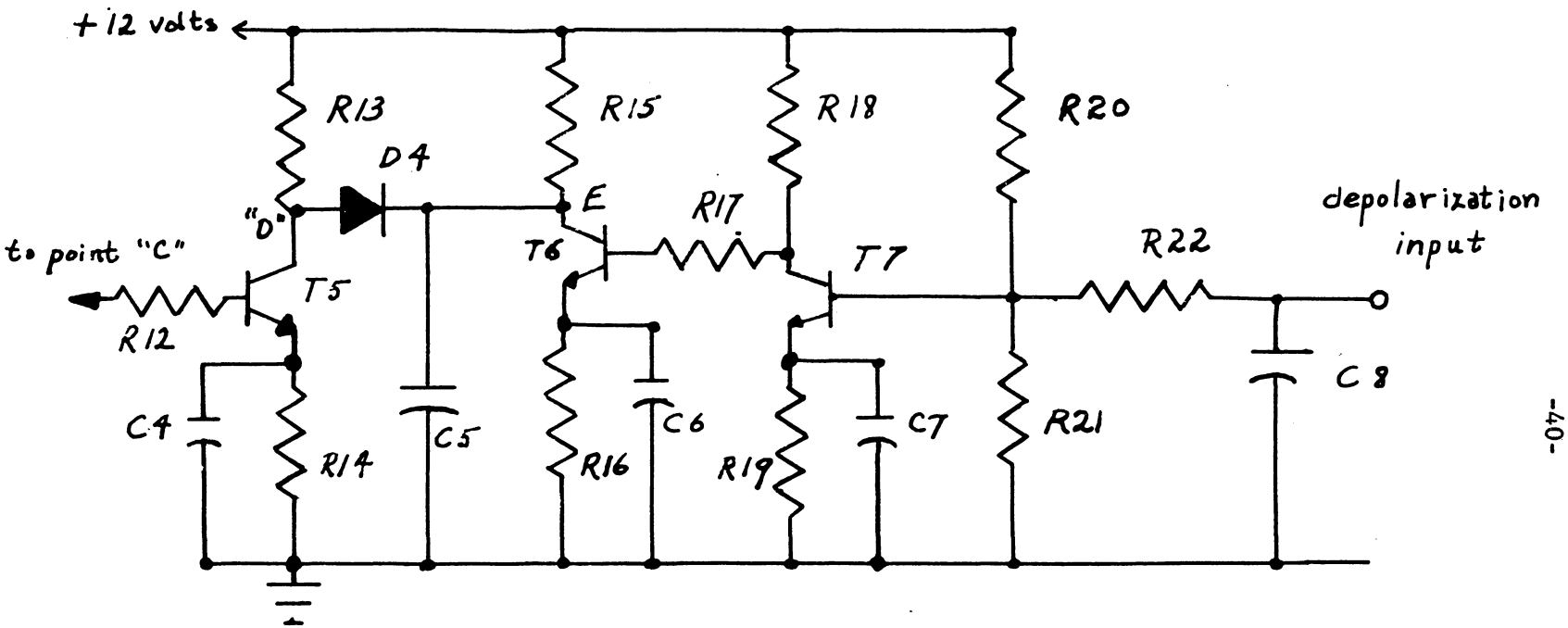
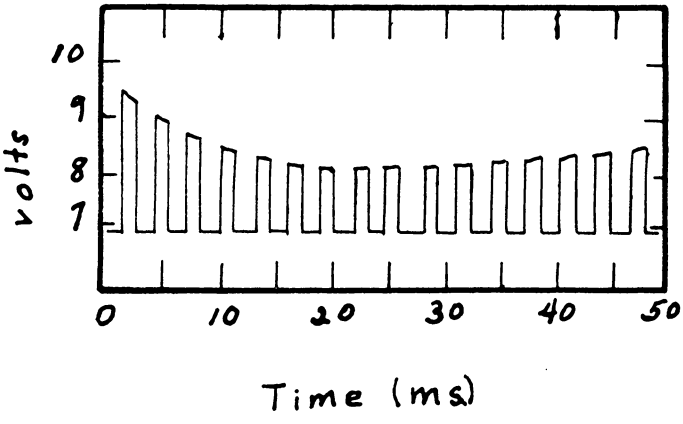


Figure 11. Amplification and Depolarization Circuit.

Figure 12. Pulse Clipping at Point "D".



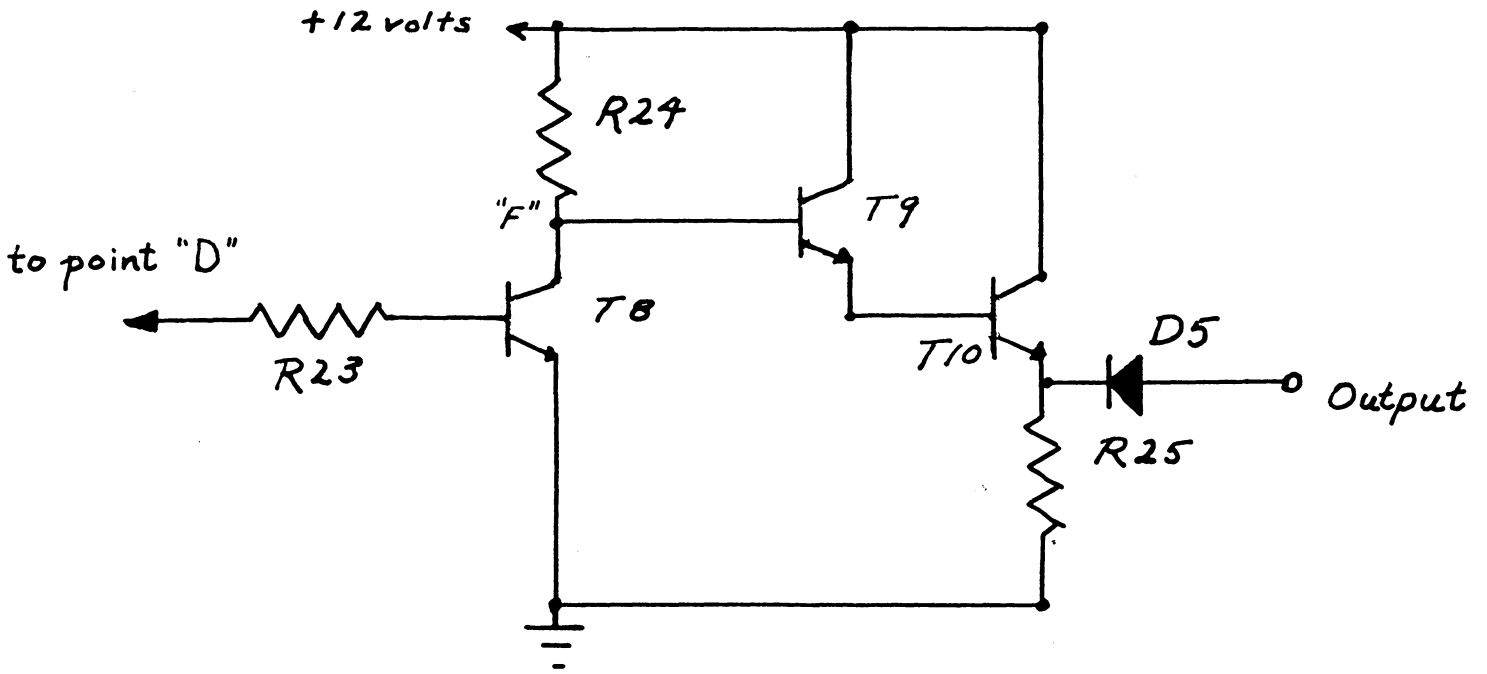


Figure 13. Pulse Inversion, Amplification, and Output Circuit.

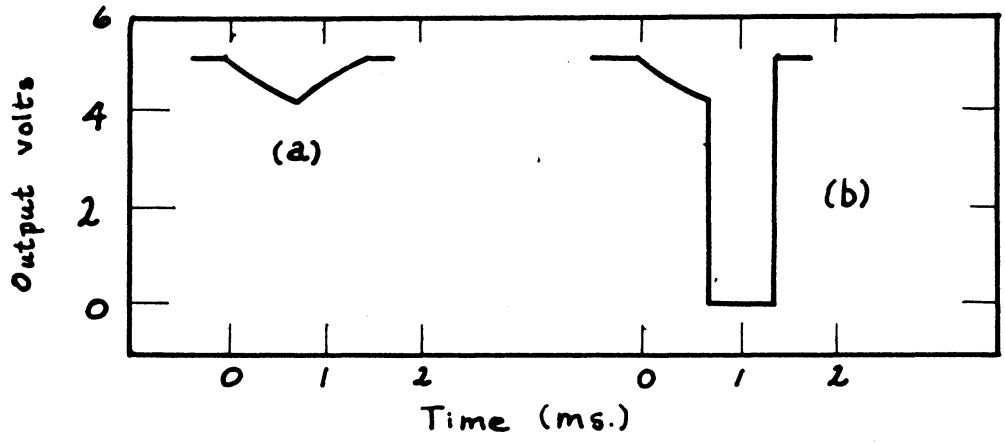


Figure 14. Subthreshold Response.
(a) No output pulse.
(b) Followed by output pulse.

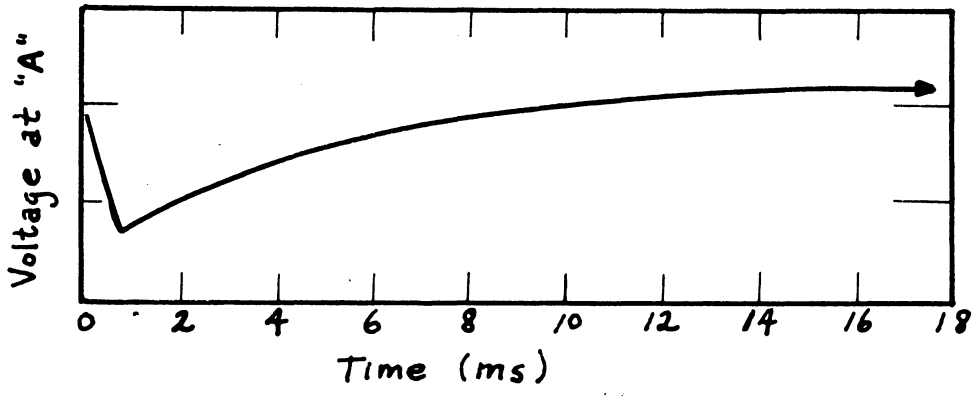


Figure 15. Hyperpolarization Following Single Pulse Output.

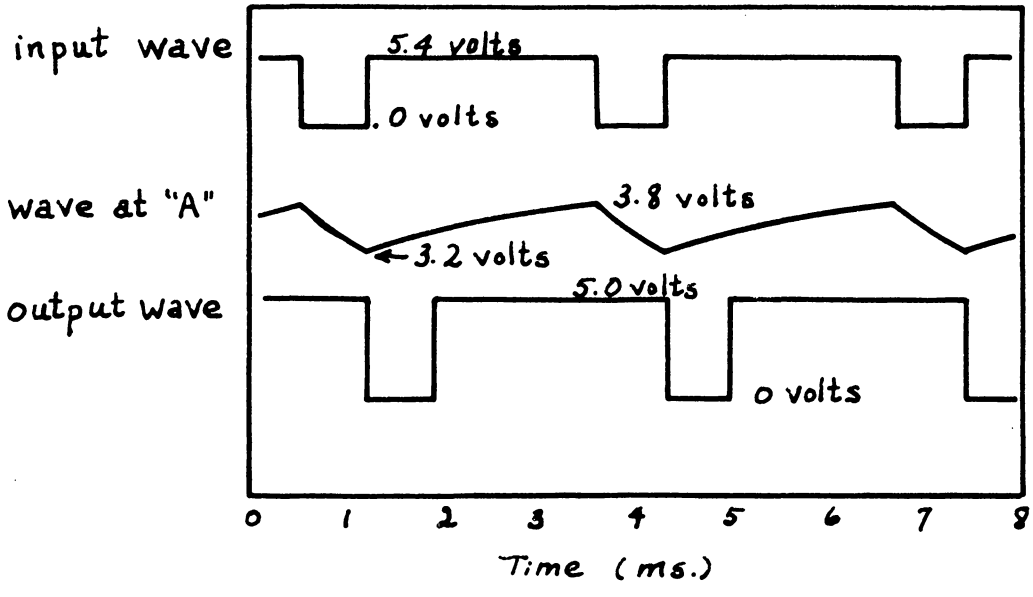


Figure 16. Relative Waveforms.

Input rate minimum required to fire neuron,

R_e = 220 KΩ.

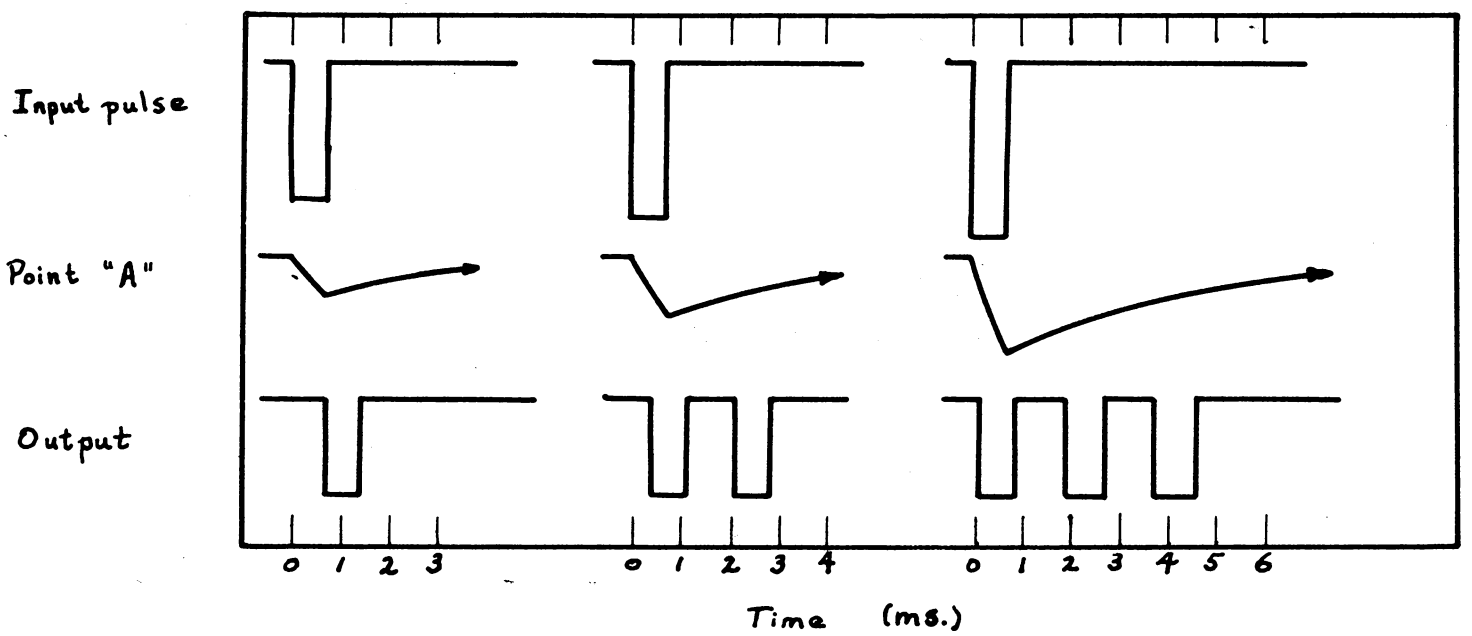


Figure 17. Influence of Pulse Magnitude

(R_e = 120 KΩ)

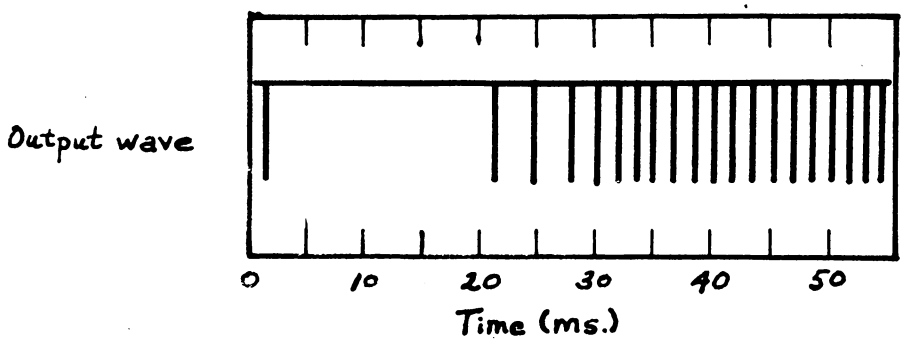


Figure 18. Effect of Hyperpolarization.
(continuous pulse train at input, inhibition input stimulated at time zero).

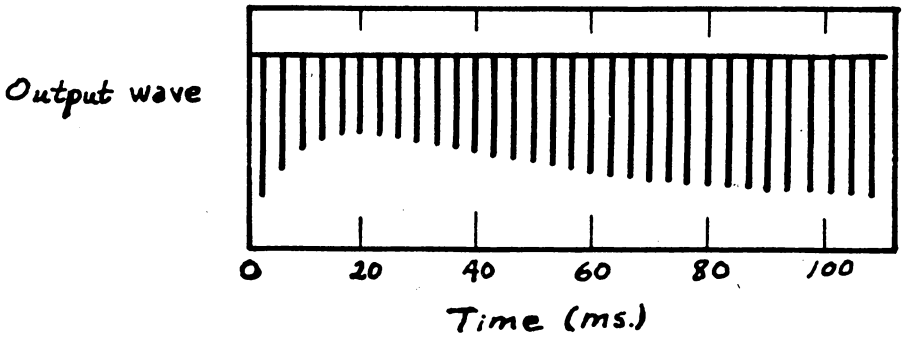


Figure 19. Effect of Depolarization.
(neuron firing continuously, inhibition input stimulated at time zero).

ABSTRACT

In this thesis the various characteristics of a neuron are discussed at some length. Particular attention is given to the more important features which determine the input and output characteristics and the responses of a neuron. Included are both of the major inhibition characteristics: presynaptic inhibition (depolarization), and postsynaptic inhibition (hyperpolarization).

The simulation presented uses a single 12 volt power supply, operates on pulses which swing in the negative direction, has a high input impedance and a low output impedance, may be easily modified to adjust the response characteristics to individual system needs, and uses only 132 milliwatts power. Input pulses are weighted and transmitted to the summation circuit by a common collector stage which provides isolation of the inputs from the summation circuitry. A hyperpolarization input raises the resting voltage at the summation circuit, requiring more stimulation to lower the summation voltage to the threshold value. This inhibition follows a predetermined time course. The simulation will produce output pulses as long as the voltage at the summation circuit is below the threshold value. These pulses have a duration of 0.7 ms. and are followed by a refractory period of about one millisecond. A depolarization input lowers the amplitude of the output pulse by a predetermined time course.