CHAPTER 4

NEURONS

The basic anatomical unit in the nervous system is a specialized cell called the neuron. An artist's view of a typical neuron is shown in Figure 4.1. Many extensions of the single cell are long and filamentary, these structures are called processes. Every neuron plays several functional roles in a neural system:

1. Metabolic machinery within the cell provides a power source for information-processing functions. In addition, the cell enforces a certain unity for biochemical mechanisms throughout its extent.

2. A tree of processes called dendrites is covered with special structures called synapses, where junctions are formed with other neurons. These synaptic contacts are the primary information-processing elements in neural systems.

3. Processes act as wires, conveying information over a finite spatial extent. The resistance of fine dendrites allows the potential at their tips to be compared with only partial coupling to other computations in the tree.

4. Temporal integration of signals occurs over the short term through charge storage on the capacitance of the cell membrane, and over the longer term by means of internal second messengers and complex biochemical mechanisms.
5. Certain neurons are equipped with a long, specialized process called an axon. The axon is used for “digitizing” data for local transmission, and for transmitting data over long distances.

We shall first review the classical doctrine of how a neuron operates. We then shall be in a position to integrate several more recent findings, out of which a considerably richer and more complex picture emerges. The classical neuron is equipped with a tree of filamentary dendrites that aggregate synaptic signals from other neurons. The input currents are integrated by the capacitance of the cell until a critical threshold potential is reached, at which point an output is generated in the form of a nerve pulse—the action potential. This output pulse propagates down the axon, which ends in a tree of synaptic contacts to the dendrites of other neurons.

The resistance of a nerve’s cytoplasm is sufficiently high that signals cannot be transmitted more than about 1 millimeter before they are hopelessly spread out in time, and their information largely lost. For this reason, axons are equipped with an active amplification mechanism that restores the nerve pulse as it propagates. In lower animals, such as the squid, this restoration is done continuously along the length of the axon. In higher animals, many axons are wrapped with a special insulating material called myelin, which reduces the capacitance between the cytoplasm and the extracellular fluid, and thereby increases the velocity at which signals propagate. The sheaths of these myelinated axons have gaps called nodes of Ranvier few millimeters. These nodes act as repeater sites, where the signal is periodically restored. A single myelinated fiber can carry signals over a distance of 1 meter or more.

Even the most casual exploration of nervous tissue with an electrode reveals a host of signals encoded as trains of action potentials. For this reason, the mechanism of initiation of the nerve pulse, and of its restoration as it propagates down the axon, became the center of early physiological investigations. The first quantitative work was carried out by Hodgkin, Huxley, and Katz (Hodgkin et al., 1952a, Hodgkin et al., 1952b, Hodgkin et al., 1952c, Hodgkin et al., 1952d). On the giant axon of the squid an unmyelinated structure nearly 1 millimeter in diameter. This classic investigation revealed the following fascinating story:

1. The cytoplasm in the cell’s interior is normally polarized—charged with a potential of approximately ±80 millivolts with respect to the extracellular fluid.

2. This potential difference is supported across a cell membrane so thin that it can be resolved only by an electron microscope.

3. If sufficient current is injected into the cytoplasm in the direction to depolarize the membrane to a threshold potential of approximately ±40 millivolts, a nerve pulse is initiated.

4. The pulse travels in both directions from the initiation point, and its shape rapidly becomes independent of the mechanism through which the initiation took place.

What are the mechanisms by which the axon initiates and propagates the action potential? That question motivated a sustained investigation by many workers over more than 5 decades. In any great scientific detective story, the resolution of a mystery at one level sharpens the focus of the researchers, and creates pressing questions at the next level. In the following sections, we will trace the story of the axon clue by clue. In the process, we will see how the nerve membrane is constructed, and how the electrical mechanism embedded in that membrane is responsible for the active transmission of nerve pulses. Only after we understand the axon will we be in a position to investigate how information processing is done in the dendritic tree of the neuron. That story is still unfolding.

NERVE MEMBRANE

All electrical activity in a neuron takes place in the thin membrane that electrically separates the neuron’s interior from the extracellular fluid. The nerve membrane is formed from phospholipid molecules arranged in a bilayer about...
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curved into the aqueous solutions on either side, to which they are attracted by these electrostatic forces. Each head-group is an electric dipole, a positive and a negative charge separated by some distance. The energy of these charges in water is much lower than in the hydrocarbon phase, and results in the stability of the bilayer structure. The entire structure is a wonderful example of self-assembly. A tiny drop of liquid on the surface of a dish filled with water will spread out into a monolayer—the molecules immediately bury their head-groups in the water, leaving their hydrocarbon tails sticking straight up into the air. If we place a fine wire parallel to the surface and immerse it slowly through the monolayer into the water, the wire will carry the two halves of the monolayer with it. The exposed monolayer hydrocarbon surfaces of the two halves join quickly to avoid confronting the hostile water molecules. We now have two monolayers back to back, forming the membrane structure shown in Figure 4.2. This monolayer technique is commonly used to create artificial bilayers in the laboratory.

Nature has developed a grand trick—by the use of the electrical polarrability of water to attract charged solutes and molecules. This technique is the ultimate basis of the three-dimensional conformation of most biologically important entities. It is the primary reason that the plan for holding a three-dimensional organism can be embedded in a one-dimensional genetic code. Biological molecules fold into their stable conformations in concert with the same hydrophobic and hydrophilic forces that organize the lipid bilayer. If there were to be a secret of life as we know it, this would be it.

In quantitative terms, we can estimate the energy of a single change in water or hydrocarbon in the following way. The possibility ϵ is a measure of the polarrability of a medium. The permittivity of free space is

\[ \epsilon_0 = 8.85 \times 10^{-12} \text{farads per meter}^2 \]

The permittivity of water is about 80 times that of free space, whereas that of the hydrocarbon phase in the interior of the membrane is only about two times that of free space. The energy of a charged ion in either phase can be calculated by integrating the energy required to assemble the charge q in this position from a large number of individual charges at infinity. The potential for a given accumulated charge can be calculated by integration of Equations 2.1 (p. 13) with respect to r, from infinity to theionic medium r. The result of this integration is

\[ V = \frac{q}{r} \]

The potential computed in this way is also the energy per unit charge required to bring and individual charge dq to the total charge. The total energy W is given by

\[ W = \int_0^r \frac{dV}{dr} dr = \frac{q^2}{2\epsilon_0} \]

The energy of an ionic such as sodium, which has a 1.0 Å radius in
**PART I  BASICS**

water, is approximately 0.1 electron volts. In the interior of the bilayer, the energy is approximately 2.4 electron volts, which is approximately 100 kT. The energy barrier in difference between the two energies is an **energy barrier** that prevents ions in the aquaporin phase from entering the membrane. The energy barrier is strictly the aquaporin phase difference between the polarity of water and that of the hydrocarbon phase.

**ELECTRICAL OPERATION**

The energy barrier formed by the nerve membrane is so high that, at room temperature, vanishingly few ions are able to surmount it. For this reason, it is possible to treat the membrane as a perfect insulator. Any current flow through it is negligible. The ions will have to be mediated by a second agent other than the bare ions in the aquaporin solution on either side. It is the manipulation of these agents that living systems achieve the gain in signal energy required for information processing. What kind of agents operate in the nerve membrane, and what operations do they perform?

**Power Supply**

Before there is gain, there must be a power supply. The most basic charge-transfer agents in all nerve membranes are the metabolically driven pumps that actively expel sodium ions from the cytoplasm and concurrently import potassium ions from the extracellular fluid. As a result of this pumping process, the sodium concentration is enriched in the potassium and depleted of sodium, whereas the cytoplasmic is true of the extracellular. The concentrations of relevant ions inside and outside a nerve cell are shown in Table 4.1 (Katz, 1966, p. 44). A concentration gradient of any charged particles can be used to power a neuron as a function of the moment, that the membrane is permeable to the ion involved. As a result of this pumping process, the sodium concentration is enriched in the potassium and depleted of sodium, whereas the cytoplasmic is true of the extracellular. The concentrations of relevant ions inside and outside a nerve cell are shown in Table 4.1.

**Equivalent Circuit**

A schematic diagram that summarizes the contribution of the three ions-

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**TABLE 4.1** Typical concentrations of ions inside neural processes and in the extracellular fluid. SOURCE: Adopted from Katz (1966).

<table>
<thead>
<tr>
<th>Ion</th>
<th>Inside (mM)</th>
<th>Outside (mM)</th>
<th>Reversal potential (mV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>K⁺</td>
<td>49</td>
<td>10</td>
<td>-92</td>
</tr>
<tr>
<td>Na⁺</td>
<td>50</td>
<td>460</td>
<td>25</td>
</tr>
<tr>
<td>Cl⁻</td>
<td>40</td>
<td>540</td>
<td>-60</td>
</tr>
</tbody>
</table>

* K⁺: potassium; Na⁺: sodium; Cl⁻: chloride.
gradients to the membrane current is shown in Figure 4.3. From it, we can visualize the operation of the membrane over a wide range of conditions. In this diagram, the $V_s$ are the reversal potentials of the ions, and the $G_s$ are the conductances of the membrane for the flow of these ions. Using Figure 4.3, we can compute the membrane current $I$ for any given cytoplasmic potential $V$, and for any values of ionic conductances:

$$I = (V_K - V)G_K + (V_Na - V)G_Na + (V_Cl - V)G_Cl$$

Any set current will change or discharge the capacitance of the membrane until the current is reduced to zero. Hodgkin et al. [Hodgkin et al., 1962b] found that, under normal conditions, the chloride current can be neglected. Making this assumption, we can solve Equation 4.3 for the voltage $V_C$ at which the current is zero:

$$V_C = \frac{V_KG_K + V_NaG_Na}{G_K + G_Na}$$

$V_C$ is called the resting potential of the cytoplasm, because it is the potential at which the cell will come to rest if left electrically undisturbed. In a typical neuron, $G_K$ is approximately 30 times $G_Na$. Using that value, and the concentrations given in Table 4.1, $V_C$ is -85 mV. The resting potential can vary considerably from one set of experimental conditions to another.

We have come to a solution of the first riddle in the axon story: A neuron at rest is polarized to a negative potential because its membrane is selectively permeable to potassium. A nerve impulse, or a transient excursion of the cytoplasmic potential in a positive direction, is an example of an excitatory signal, because it depolarizes the membrane. If the membrane is charged more negatively than its resting voltage, it is said to be hyperpolarized, in which case the signal is inhibitory.

We achieve electrical activity in a patch of nerve membrane by making one or more of the ionic conductances depend on some control quantity. That quantity can be the voltage (as in the axon), the concentration of a chemical substance (as in chemical synapses), the intensity of light (as in photoreceptors), or the degree of mechanical deformation (as in the hair cells in the ear). We will first see how these conductances are responsible for the generation and propagation of the action potential in an axon. In later sections, we will consider other ways that the potential inside the neuron is manipulated by the nervous system to accomplish information-processing tasks.

**THE ACTION POTENTIAL**

We have seen that the membrane potential can be manipulated by any agent that selectively increases the permeability of the membrane to one ionic species. How can agents of this kind be employed to initiate and propagate an action potential?

**Initiation of the Action Potential**

If we inject small pulses of current into the cytoplasm, the potential will respond as shown in Figure 4.4. For current levels that depolarize the axon membrane less than approximately 20 millivolts from its resting state, the potential shows a somewhat sluggish response that saturates after a few milliseconds. This type of response is characteristic of any circuit consisting of a capacitance in parallel with a conductance, as we will discuss in Chapter 8. In the case of the axon, the capacitance is that of the membrane, and the conductance is due to the potassium permeability. At higher current levels, we observe an exponential increase in the cell potential, culminating in the explosive generation of an action potential. If we terminate the current pulse before the potential has reached approximately -40 millivolts, the membrane recovers, and no pulse is generated. Once the potential is more positive than approximately -40 millivolts, however, a pulse is generated even if the driving current is terminated. That potential is therefore a threshold, beyond which a self-reinforcing reaction is underway, and no recovery is possible.

All the information we have presented so far was known by 1950. The key question was, what mechanism was responsible for the self-reinforcing reaction? That question was unravelled by the detective work for which Hodgkin and Huxley received the Nobel Prize in 1963. The giant axon of the squid is large enough.
In response to a depolarization of the membrane, there is a transient increase in the sodium conductance, followed by a delayed but prolonged increase in the potassium conductance. The currents through these conductances, setting on the capacitance of the membrane, create the setpoint potential. Although this picture explains the qualitative shape of the action potential once the pulse is triggered, it tells us nothing about the mechanism that leads to the overshoot, and to the all-or-nothing response. To understand that behavior, we must know how the conductances depend on the membrane potential.

**Voltage Dependence of the Conductances**

By carrying out similar experiments at several depolarizing potentials, Hodgkin and Huxley gathered data on the time course of the two currents as a function of the membrane potential. From these data, they reconstructed the time dependence of the sodium and potassium conductances for different membrane potentials. Plots of the peak sodium conductance and the sustained potassium conductance as functions of the membrane potential are plotted in Figure 4.6. At low current levels, both conductances are exponential functions of the membrane potential, increasing by a factor of e for approximately 4-millivolt increase in voltage. At higher current levels, both curves saturate—allegorical reminiscence of the dependence of transducer current on gate voltage shown in Figure 3.7 (p. 38). The quantitative difference between the two curves indicates that the latter has an exponential characteristic that is six times as large as that of the sodium current.
time steeper than that of the former! It is this exponentially increasing current that gives the axon membrane the gain required to produce the self-reinforcing reaction leading to the all-or-nothing response.

Although Hodgkin and Huxley did not address the mechanism by which this remarkable exponential dependence comes about, their findings did allow them to reconstruct the initiation and propagation of the action potential. The summary in their 1952 paper is still the best short description of the phenomenon extant:

When the membrane potential is suddenly reduced (depolarisation), the initial pulse of current through the capacity of the membrane is followed by large currents carried by ions (chiefly sodium and potassium), moving down their own electromotive gradients. The current carried by sodium ions rises rapidly to a peak and then decays to a low value; that carried by potassium ions rises much more slowly along an S-shaped curve, reaching a plateau which is maintained with little change until the membrane potential is restored to its resting value.

These two components of the membrane current are enough to account qualitatively for the propagation of an action potential, the sequence of events at each point on the nerve fibre being as follows: (1) Current from a neighbouring active region depolarizes the membrane by spread along the cable structure of the fibre ('local circuits'). (2) As a result of this depolarisation, sodium current is allowed to flow. Since the external sodium concentration is several times greater than the internal, this current is directed towards and depolarizes the membrane still further, until the membrane potential reverses its sign and approaches the value at which sodium ions are in equilibrium. (3) As a delayed result of the depolarisation, the potassium current increases and the ability of the membrane to pass sodium current decreases. Since the internal potassium concentration is greater than the external, the potassium re-enters the cell. When it ceases the sodium current, it repolarizes the membrane, raising the membrane potential to the neighbourhood of the resting potential, at which potassium ions inside and sodium ions outside are in equilibrium. (Hodgkin et al., 1952a, p. 470)

IONIC CHANNELS

The question of electrical activity in the axon has been sharpened still further—what is the mechanism by which the membrane conductance achieves its remarkable exponential dependence? To find the answer, we need to take a close look at the sodium current.

Channel Conductance

A close look at the sodium current, finally captured in 1986 by Keller (Keller et al., 1986) and his coworkers, is shown in Figure 4.7. The ion-specific conductance changes in discrete steps; the height of each step is approximately linear in the membrane potential relative to the reverse potential of the ion. At low currents, the number of steps and the width of each step are both exponential functions of the membrane potential. At any given voltage, the steps are all the same height. This remarkable finding suggests that each step is the result of an atomic action on the part of a single molecular entity. The molecular entities responsible for selective permeability of nerve membranes to specific ions are aggregates called channels. The channels responsible for propagating the nerve impulse in an axon are voltage-controlled.

Because the detailed structure has not been worked out for either the sodium or the potassium channels, we will exercise a bit of artistic license to visualize how one of these channel types might operate. The result of this creative endeavor is shown in Figure 4.8.

Imagine a molecule about 50 angstroms long, with two positive charges on one end of a long hydrocarbon backbone and two negative charges on the other. The backbone is sprinkled with occasional polar groups along its length. We suppose that, in their normal stable configuration (shown in the highly schematized
Voltage-Dependent Conductance

When a channel is open, the conductance is determined by the prevalent open state, and by the surface configuration of the channel. The channel conductance is highly modulated, and in detail it is certainly not constant. In general, the conductance of the channel is expressed in terms of the known properties of channels. The distribution of the conductance across the membrane is given by the Boltzmann distribution:

\[ N = N_0 e^{-\frac{\Delta V}{kT}} \]

where \( N \) is the relative conductance at voltage \( V \), \( N_0 \) is the conductance at zero membrane voltage, \( \Delta V \) is the transmembrane voltage, \( k \) is the Boltzmann constant, and \( T \) is the temperature.

The equilibrium distribution of the conductance across the membrane is given by the Boltzmann distribution:

\[ \frac{N}{N_0} = e^{-\frac{\Delta V}{kT}} \]

where \( N \) is the relative conductance at voltage \( V \), \( N_0 \) is the conductance at zero membrane voltage, \( \Delta V \) is the transmembrane voltage, \( k \) is the Boltzmann constant, and \( T \) is the temperature.

By using Equation (4) in terms of the function \( e^{-\frac{\Delta V}{kT}} \), we can solve for the conductance:

\[ N = N_0 e^{-\frac{\Delta V}{kT}} \]

For small values of \( \Delta V \), the conductance will be negligible, and the number of channels that are open will be very small. For large values of \( \Delta V \), the conductance will be high, and the number of channels that are open will be high.

If we consider the case of a single channel, we can write the conductance as a function of the transmembrane voltage \( V \):

\[ G = G_0 e^{-\frac{\Delta V}{kT}} \]

where \( G \) is the conductance at voltage \( V \), \( G_0 \) is the conductance at zero membrane voltage, \( \Delta V \) is the transmembrane voltage, \( k \) is the Boltzmann constant, and \( T \) is the temperature.

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By using Equation (4) in terms of the function \( e^{-\frac{\Delta V}{kT}} \), we can solve for the conductance:

\[ G = G_0 e^{-\frac{\Delta V}{kT}} \]
a number of gating charges is involved in the reaction that opens the channel. For Figure 4.6, taken from the giant axon of the squid, there are approximately six gating charges involved. Recent work on the node of Ranvier of the frog suggests that two molecules form a channel in that system [Dubois et al., 1985].

The voltage dependence of Figure 4.7 gives approximately four gating charges. It thus seems that vertebrates employ a voltage-gated channel of two molecules, each carrying two charges through the membrane. If the charges in the reaction do not go all the way through the membrane, the energy they contribute is only a fraction of eV, and hence measurable quantities often are observed.

The exponential dependence of conductance on membrane potential is a result of the behavior of the population of channels, rather than of the conduction properties of any given channel. A close look at Figure 4.7 reveals that a discrete increase in current as each channel opens, and an equal decrease as each channel closes. The size of an individual step is roughly linear in the difference between the membrane voltage and the reversal potential for the selected ion, indicating that an individual channel is ohmic (i.e., current proportional to voltage). The rate at which channels open increases, and the rate at which they close decreases, with voltage. Both dependencies are exponential, and can be seen clearly in Figure 4.7. The number of channels open at any time is a result of the balance of these two processes.

The exponential current-voltage relation in the squid is a result of the same physical laws responsible for the exponential transistor characteristic. There is an energy barrier between a state in which current can flow and one in which current cannot flow. The height of that barrier is dependent on a control voltage. The Boltzmann distribution determines the fraction of the total population that is in the conducting state. In the transistor, the electron in the channel form the population in question, and the same electrons carry the current. Is the nerve membrane, the channels form the population in question, and ions in the channels carry the current. In both cases, the number of individual charges in transit is exponential in the control voltage, and the transport of these charges results in a current that varies exponentially with the control voltage.

COMPUTATION

We have traced the ideas elucidated in one of the great endeavors in intellectual history; in the process, we have learned a great deal about the electrical machinery in the neuron. At one time, people might have supposed that understanding the action potential would be the key that would unlock a full understanding of neural information processing in the brain. It is now clear that, although we have a good understanding of how the nervous system transmits information over long distances, this knowledge does not shed much light on how the information is computed. We have yet to test a single unifying principle in neural computation that shines with the same clarity as the axon story does; that work is ahead of us. The balance of this book describes one approach (of many) to a deeper understanding of the basic principles underlying neural computation. This quest will require the work of specialists in many fields over many years.

A few comments may serve to render our task a bit less daunting. Computation in neural systems uses the same kind of machinery that we have already encountered in the axon story. Once we can control the logic conductances, we can manipulate the resting potential of the membrane. An increase in sodium conductance depolarizes the membrane, and is the action responsible for initiation of a nerve pulse. An increase in potassium conductance can hyperpolarize the membrane, and hence acts as an inhibitory influence.

If the chloride reversal potential is near the resting potential, as is often the case, an increase in chloride conductance will not have much effect on the potential of the membrane, but can decrease the effect of either sodium or potassium conductance by requiring more current for a given excursion in potential. This reduction in the sensitivity of the membrane by increasing its conductance to the resting potential is called shunting inhibition.

These and other methods of manipulating the membrane potential essentially in the rigidly branched tree of dendrites to produce the complex interconnection of electrical and chemical signals that is neural computation. Interactions is the dendritic tree can all work in a continuous analog fashion, as indicated by Equation 4.3. They neither require nerve pulses for their operation, nor necessarily result in the generation of a nerve pulse. In fact, the vast majority of computation in the nervous system is done with slowly varying analog signals in the dendritic tree of neurons. These signals come about through the actions of synaptic contacts with other neurons. The result of the computation way or may not ever be converted into an action potential to be transmitted to the far reaches of the brain.

The synapses provide an entirely new class of function in dendritic computations. We can say that the synapses is to e−γ̅ν̅ what the voltage-controlled channel is to communication. The story of the synapse is still being worked out, and the following section gives only the briefest account of this fascinating and rapidly evolving field.

SYNAPSES

We have seen how the potential across a nerve membrane can cause an exponential change in current through the same membrane. This section is appropriate for the propagation of an action potential, but is not sufficient for general compu-
tation. What we need is the ability to control the conductance through a second membrane. The ability to control the current into or out of one electrical node by the potential on another node is the key ingredient that makes all information processing possible. This capability results in a natural direction in the flow of information, in neural systems, it is provided by synapses. These specialized structures are the central information-processing devices in neural systems.

![Figure 4.9 Simplified sketch of a synapse](Image)

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A drawing depicting a typical synaptic arrangement is shown in Figure 4.9. The function of a synapse is to control the conductance of the membrane separating the interior of the postsynaptic cell from the extracellular fluid. That conductance is controlled by the potential across the presynaptic membrane.

The detailed mechanism by which synapses operate is extremely interesting, and will no doubt be the subject of study for many years. We will present here only the essential principles of synaptic operation. There are several excellent reviews of the subject at various levels of detail [Shepherd, 1979].

Inside the presynaptic membrane is a high concentration of specific neurotransmitter molecules. When the presynaptic membrane is depolarized, calcium channels allow calcium ions (Ca^{2+}) to flow into the presynaptic cell from the synaptic cleft. The calcium ions activate the subcellular machinery that causes release of neurotransmitter molecules into the synaptic cleft, where these molecules diffuse to the postsynaptic membrane and initiate a chain of events that results in the opening of ion-specific channels. The quantity of neurotransmitter released, and therefore the change in conductance of the postsynaptic membrane, is an exponential function of the presynaptic potential, as shown in Figure 4.10.

![Figure 4.11 Examples of synaptic microcircuits by which neural processes interact](Image)
If the channels opened by the neurotransmitter are specific for sodium, for example, a depolarization of the presynaptic membrane will result in a depolarization of the postsynaptic membrane, and the synapse is said to be excitatory.

If the neurotransmitter leads to the opening of potassium-specific channels, the postsynaptic membrane will be hyperpolarized, and the synapse is said to be inhibitory. As we have noted previously, chloride channels can act to increase the conductance of the membrane without appreciable change in potential—synapses leading to this behavior are called asymmetrical. Within these broad categories, many variations are possible. In addition, synapses are known that open channels for molecules other than sodium, potassium, or chloride. We shall not discuss any of these complications here.

A single synapse is the neural counterpart of a transistor. The tip of every neural process ends in a synapse, and there are many synaptic contacts along the branches of the dendritic trees as well. As in electronic computational machinery, synapses occur not in isolation, but rather in circuit arrangements. Dendrites form a wide variety of synaptic connections with dendrites and axons of other neurons. The specialization of function of the many areas in the nervous system is largely a result of these synaptic circuit arrangements. Cross-sections through several representative synaptic structures are shown in Figure 4.11. Specific circuits for many parts of the brain are discussed in Gordon Shepherd’s excellent book The Synaptic Organization of the Brain [Shepherd, 1979]. In addition, a lucid popular account has appeared in Scientific American [Shepherd, 1978]. Many of these arrangements have (not altogether by accident) parallels in circuits we will discuss in subsequent chapters.

REFERENCES


