Chapter 2
Neuronal Properties

The structure and dynamic characteristics that account for the competence of any articulated physical system are critically dependent on the properties of its primitive components. In the development of a neuronal model, we must first specify the minimal relevant properties of the nerve cells that are to compose it. Then the theoretical problem is to formulate a competent computational structure that is biologically plausible with respect to the postulated characteristics of the individual neurons and to their patterns of connectivity.

Any living neuron is an enormously complex biophysical system. For the purposes of what is to be a system-theoretical account of the human cognitive brain, I need to explicate only those properties and assumptions that are essential for the operational integrity of the proposed models. We will take for granted all of the biochemical, electrical, and structural processes and characteristics that contribute to the singular viability of a functioning nerve cell (Cotman and McGaugh 1980; Horridge 1968; Kandel 1976; Kuffler, Nicholls, and Martin 1984; Shepherd 1983) and focus initially on those aspects specifically required in the mechanisms and networks to follow.

Basic Properties of Neurons

In terms of shape (figure 2.1), mammalian neurons exhibit great diversity. The cell body of a neuron can range from 5 to 100 microns on its longest axis. Projection neurons such as pyramidal cells may have dendrites that are several millimeters in length and axons that extend many centimeters. Local circuit neurons may have no axon and only very short dendritic processes that extend no more than a few microns. Modern estimates of the number of neurons that occupy the cortical mantle of the human brain range from 10 billion to somewhat more than 16 billion (Blinkov and Glezer 1968). Despite their striking variability in size and shape, neurons are all alike in one respect: their principal function is to receive and integrate electrochemical pulses from sensory transducers or other neurons and, con-

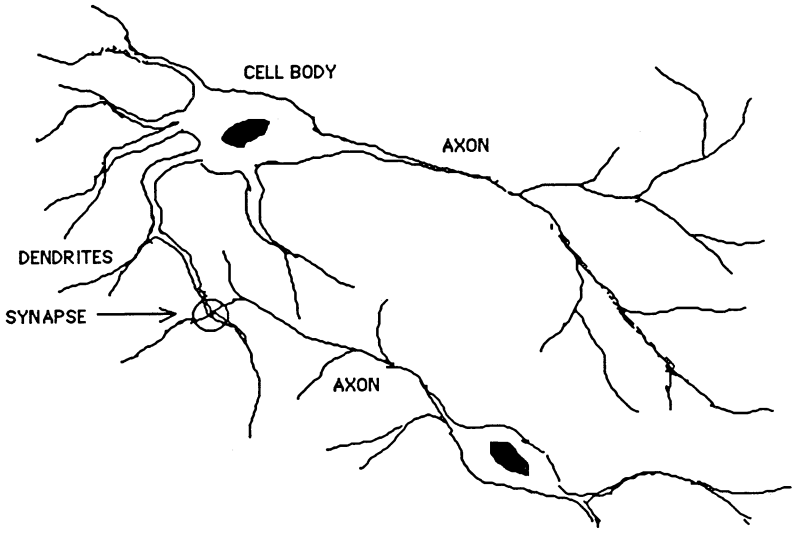

Figure 2.1
Two neurons with a synaptic junction between the axon of one and a dendrite of the other.

tingent on the characteristics of the received pulses, output their own characteristic pulses to other neurons or muscle fibers.

The fact that there are contingencies between the input activity on a neuron and the output it sends to its targets means that it is not simply a biological device for relaying signals. It performs a computation on its input. The combined computations of many neurons in specialized connective architectures account for cognition.

Figure 2.2 illustrates some fundamental properties of neurons. At the top of the figure is a schematic drawing of a generalized nerve cell. Stimulation to the cell is provided at discrete junctions (synapses) by quanta of neurotransmitters released into the gap (synaptic cleft) between an active axon terminal (S_i) of a presynaptic cell and a specialized receptor patch on the postsynaptic cell membrane. The neurotransmitters released by the donor cell may either excite or inhibit a target cell. An excitatory input induces an inward flow of sodium and other cations that drives the resting membrane potential of the target cell (typically, near -70 millivolts at equilibrium) in a positive direction (depolarization) toward discharge threshold. A change in membrane potential of this kind is called an excitatory postsynaptic potential (EPSP) (see the bottom illustration in figure 2.2). If discharge threshold is reached, an action potential (a positive-

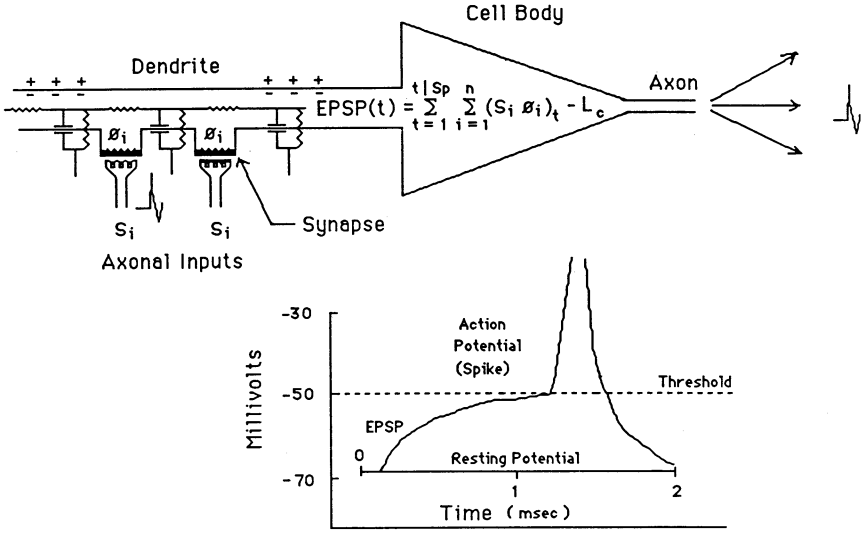

Figure 2.2
Top: Schematic showing the bioelectrical properties of a typical neuron. A single dendritic shaft represents the available surface of all the dendritic branches of the cell. *Bottom:* Dynamic characteristics of a typical neuron showing relationship of resting potential, EPSP, discharge threshold, and action potential.

going spike of voltage that is measured inside the cell against an outside reference) is initiated near the junction of the axon and the cell body. This voltage spike is the result of an abrupt regenerative local increase in the membrane's permeability to sodium cations. The action potential propagates down the length of the axon and its branches, where it induces the release of neurotransmitter at each of the cell's synaptic terminals. After each action potential, the neuron will exhibit a brief period of absolute and then relative refractoriness in its response to stimulation. In contrast, an inhibitory input increases the membrane's permeability to potassium cations and/or chloride anions and drives the membrane potential in the direction of greater negativity (hyperpolarization), away from the threshold potential required for spike discharge and toward the lower equilibrium potential for these ion species (increased within-cell negativity). A change in membrane potential of this kind is called an inhibitory postsynaptic potential (IPSP). The specialized postsynaptic receptors at the dendritic membrane (the darkened areas beneath the synaptic cleft in the top schematic) can be characterized in terms of their efficacy in mediating changes in EPSP and IPSP in response to presynap-

tic neurotransmitter release. This property is termed the synaptic transfer weight and is designated by the symbol ϕ (Trehub 1975a).

The dendritic membrane in figure 2.2 presents a parallel circuit of capacitance and resistance to any potential gradient across the membrane, causing the neuron to behave as a leaky spatial-temporal integrator of its synaptic inputs (see the summation expression within the cell body shown at the top of figure 2.2). The expression

$$MP(t) = RP + \left(\sum_{t=1}^{t/S_p} \sum_{i=1}^{n} (S_i \phi_i)_t - L_c \right) \tag{2.1}$$

gives a rough linear approximation for the relative value of the membrane potential (*MP*) in a cell at time t after the initiation of synaptic input. In this formula, *RP* represents the resting potential of the neuron, S_i represents the activity level of the ith axonal input terminal where each excitatory input has a positive sign and each inhibitory input a negative sign; ϕ_i is a coefficient representing the synaptic transfer weight at the ith axon-dendrite junction; $(S_i\phi_i)_t$ is the product of S_i and ϕ_i at time t; t/S_p is the time following stimulus onset at which threshold is reached and a spike discharge is generated; and L_c represents the reduction of EPSP by leakage of the membrane charge. As we shall see later, the simple fact that neurons are time-sensitive leaky integrators is a useful property in the behavior of certain kinds of neuronal mechanisms.

Equation 2.1 implies that with transfer weights (ϕ_i) held constant, the slope of time-integrated EPSP will increase as the net excitatory input to a cell is increased, either by having a greater number of active excitatory inputs, an increase in the activity level in one or more of a fixed set of excitatory inputs, or a decrease in the total inhibitory input (IPSP) to the cell. A rise in the rate of EPSP integration means that the latency to discharge threshold will decrease during the stimulation epoch. If the stimulus is sustained, the neuron will exhibit a frequency of discharge that is monotonically related to the slope of EPSP integration. Thus

$$F \propto \frac{d(EPSP)}{d(t)} \tag{2.2}$$

where F represents the frequency of cell discharge. The relationship between the rate of EPSP integration and the frequency of spike output is illustrated in figure 2.3. The higher the frequency of discharge is in any given cell, the stronger is its stimulation to its target cells and, if the output is excitatory, the shorter is the latency to discharge

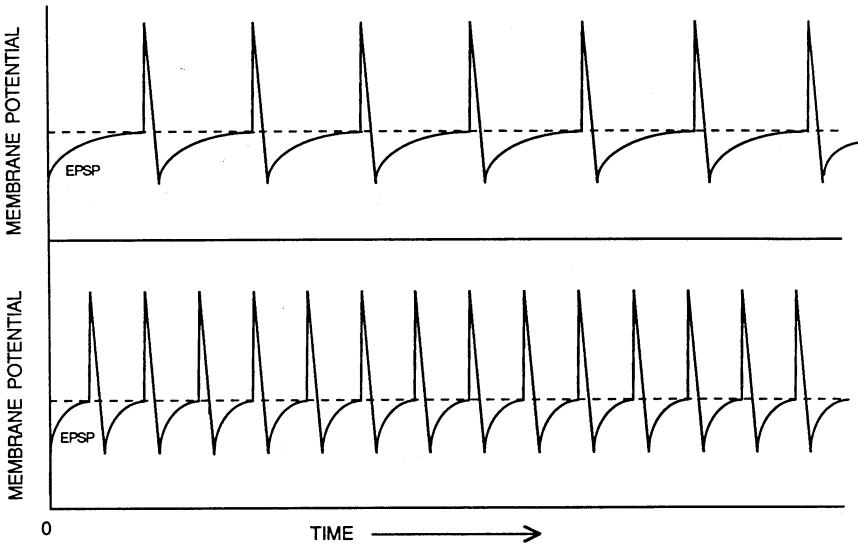

Figure 2.3
Relationship between rate of EPSP integration and frequency of spike discharge.

in the target cells. Discharge frequencies as high as 800 spikes per second have been recorded in large myelinated neurons.

Suppose that there are two general sources of input to a cell: a source of excitatory information to which the cell must respond and a subthreshold source of biasing input that shifts the membrane potential closer to or farther away from its discharge potential. The closer to threshold that a neuron is biased, the fewer the number of stimulus spikes that will be required to fire the cell, the shorter its response latency will be, and the higher its output frequency will be in response to a given sustained excitatory input. Conversely, the farther from threshold a neuron is biased (by inhibitory input), the longer its response latency will be and the lower will be its output frequency (figure 2.4). In a neuronal system of many cells that receive parallel input from a common stimulus source but control a variety of competing response options, the selective biasing of the cells can profoundly shape the stimulus-response behavior of the system.

So far, I have mentioned only synaptic junctions between axons and dendrites (axodendritic synapse), but there are a variety of other kinds of synaptic contacts among neurons: contacts between an axon and a cell body (axosomatic), between two axons (axoaxonic), and between dendrites (dendrodendritic). Presynaptic activity at any of

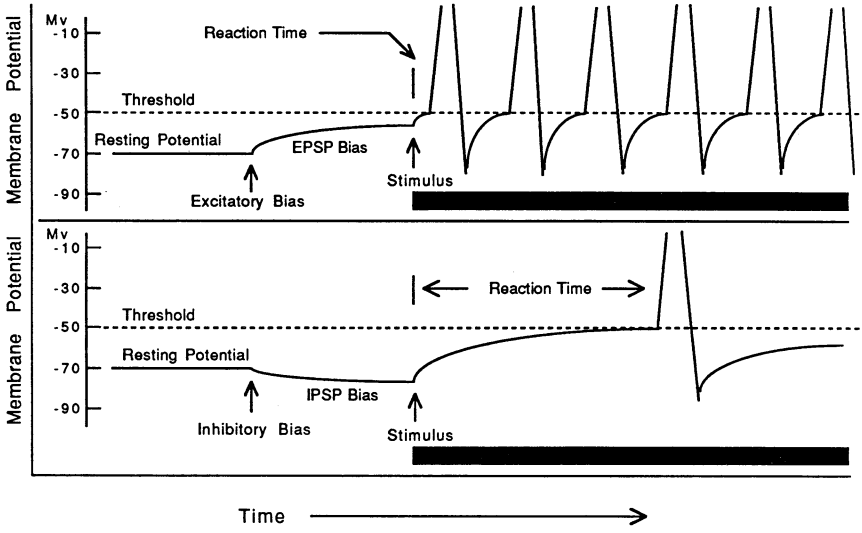

Figure 2.4
Relationship of cell bias, reaction time (latency), and discharge frequency given a standard stimulus. Dark bar represents onset and continuation of stimulus.

the various kinds of synaptic arrangements will affect the nature and degree of ionic flows across the membrane of the target cell and, thus, the membrane potential. In the case of dendrodendritic synapses, cell-to-cell influences are typically mediated by graded ionic processes without the requirement of a spike discharge at the presynaptic terminal. As we shall see, the dendrodendritic structure is well suited for establishing gradients of membrane bias over large neuronal arrays.

Autaptic Neurons

Autaptic neurons, which are characterized by having one or more of their axon collaterals in feedback synapse with their own dendrites or cell body (Shepherd 1979, van der Loos and Glaser 1972), have a simple structural property that allows them to serve as short-term memory processors in cognitive mechanisms (top, figure 2.5). In cells of this kind, it is assumed that if there is sufficient sustained positive bias (for example, by subthreshold excitatory input from the reticular activating system), a transient stimulus will cause the autaptic cell to continue firing even after input from the initiating stimulus has stopped. If the subthreshold bias is removed, excitation from its own recurrent axon collateral is insufficient to sustain spike discharge, and the autaptic neuron stops firing (figure 2.5, bottom).

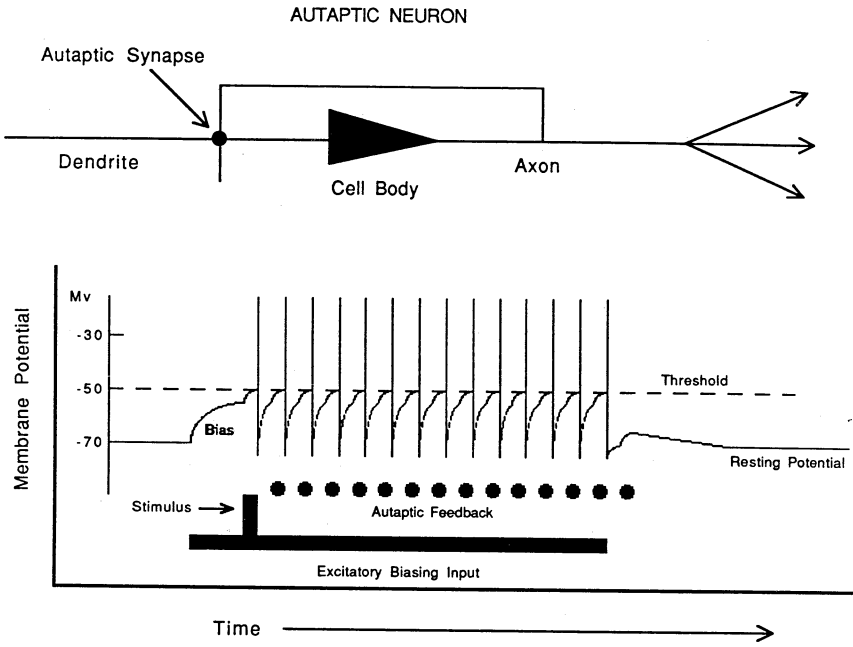

Figure 2.5
Top: Schematic of autaptic neuron. *Bottom:* Dynamic characteristics of an autaptic neuron. Cell continues to discharge after a single suprathreshold stimulus pulse as long as subthreshold excitatory bias is sustained.

Autaptic cells can serve as bistable elements in more complex neuronal circuits and mechanisms. For example, large arrays of autaptic cells can "capture" a transient afferent pattern and hold it as a short-term memory representation for any additional processing required, or a fleeting categorical event on a single axonal line can be represented by continuing activity in an autaptic target so that it can be effectively related to other brain events that may require more time to evolve. Since autaptic output can also be used as a subthreshold biasing (priming) input to other processing modules, autaptic cells can play a useful role as gating or strobing mechanisms in networks that perform delicate timing operations.

Adaptive Neurons

How can the human brain establish new internal representations and input-output mappings that are adaptive to the changing physical and social environment and the motivational needs unique to each

individual? When we raise this question, we confront the challenge of explaining how complex learning can be effectively accomplished within the biological constraints of a neuronal system. For the reasons given in chapter 1, I believe that a comb-filter model is the preferred approach to cognitive representation and mapping. In models of this kind (and in PDP systems as well), the pattern of activation-transfer weights among similar processing units determines the content of the system's memory, its concepts, and its input-output mappings. Selective modification of synaptic transfer weights (ϕ) is generally assumed to be the principal physical process underlying learning and memory in the neuronal networks of the brain. But what specific processes might provide a reasonable biological basis for these adaptive synaptic changes?

Before I describe the proposed mechanism for selective synaptic modification, it will be helpful to consider a number of basic problems that must be solved in a biological system that is to learn useful representations and stimulus-response mappings. At the level of a single adaptive neuron, we should ask the following questions:

1. Under what conditions will an adaptive synapse change its transfer weight (ϕ)?
2. How are transfer weights (ϕ) on the adaptive cell normalized to prevent significant system errors that can occur because the complete segment of unit activity representing one stimulus may be included within the pattern of a different stimulus, because of variation in the sheer number of active input units associated with particular stimuli, or because some stimuli are experienced more frequently than others?
3. How is a particular learned pattern of ϕ on a cell protected from "overwriting" by subsequent inputs?
4. How quickly do appropriate changes in ϕ take place, and how long are particular changes in ϕ maintained?

Initiating Change in Transfer Weights

Each of us builds and elaborates an expanding base of world knowledge by establishing internal representations (memories) of our cumulative sensory experiences. Because we cannot maintain representations of all sensory input, even over relatively short periods of time, we are obliged to specify those principles by which a constant stream of sensory input is to be sampled and registered by discrete and enduring physical changes in the brain. In terms of adaptive cells within a neuronal model, this means that we must propose biologically plausible mechanisms for initiating change in their synap-

tic transfer weights on principles that can account for an appropriate sampling of afferent activity.

Normalization of Transfer Weights

Recall that a fundamental computational problem in processing information from complex external and internal environments is that of pattern categorization. In a comb filter system, we want a physically indexed cell that has been tuned to a particular pattern of activation over the array of input lines to signal reliably that pattern's presence by producing an output greater than that of any other cell, which may be tuned to a different input pattern. To illustrate the kind of difficulty that can arise in relation to the settings of transfer weights, let us imagine a simple comb filter consisting of only two adaptive cells (table 2.1). Suppose that when a stimulus vector V1 was presented, a learning mechanism simply changed the values of ϕ on cell A from 0 to 1 at each synaptic junction corresponding to the axonal contact from an active cell in the input array and that the values of ϕ on cell B were changed in a similar fashion when stimulus V2 was presented. Now, suppose that V1 corresponds to the stimulus vector 000111000 and V2 to the vector 110111011, where 1 represents an active unit and 0 an inactive unit in the input array. In this case, if V2 were presented later, cell B would correctly respond with a spike output frequency greater than cell A because its sum of the products between active axonal inputs and their corresponding activation transfer weights would be greater than that for cell A. If V1 were presented, however, the sum of products for cells A and B would be equal, and there would be no difference in the spike output frequencies of the cells. Given these circumstances, the system is unable to classify the input properly.

Suppose, on the other hand, the weights given by this procedure were strictly normalized by dividing each by the number of active units in the input vector at the time of learning. In this case, if V1 were later presented, cell A would respond appropriately, but if V2 were presented, the sum of products for cells A and B would be equal, and again the system would be unable to classify the input. What seems to be required is a mechanism that combines the respective advantages of unnormalized and normalized synaptic weightings. This problem in categorization, related to the inclusion of a given pattern within another, is one that must be solved in a biologically credible fashion.

Another consideration in pattern recognition concerns the relative effects of noise on performance in response to stimuli that evoke a

Table 2.1
Sum of products for each cell of a two-cell filter

Input (S_i)	ϕ_i Cell A[a]	ϕ_i Cell B[a]
0	0 (0)	1 (.14)
0	0 (0)	1 (.14)
0	0 (0)	0 (0)
1	1 (.33)	1 (.14)
1	1 (.33)	1 (.14)
1	1 (.33)	1 (.14)
0	0 (0)	0 (0)
0	0 (0)	1 (.14)
0	0 (0)	1 (.14)
	3 (.99)	3 (.42)

a. Nonnormalized weights are in the left part of the columns; normalized weights appear in parentheses.

small number of active input units compared to those evoking a large number of active units. We would expect small stimuli to be more vulnerable to noise than large stimuli, but since small stimuli can be just as important in an ecological sense as large stimuli, it would be advantageous for adaptive neurons to compensate, at least partially, for such differences in vulnerability to noise. Problems of this kind, related to differences in stimulus size, can be reduced to the extent that synaptic transfer weights approach true normalization over a wide range in the number of coactive input units.

An analogous difficulty must be addressed with respect to the number of times a particular stimulus might be experienced. Since an appropriate response to a stimulus that is infrequently encountered can be more critical in some circumstances than a response to a more commonly experienced stimulus, the selective tuning of adaptive cells should occur after as few stimulus repetitions as possible.

Overwriting Weight Distributions

After the distribution of synaptic transfer weights on an adaptive cell has been changed so that the cell is tuned to respond maximally to a particular stimulus, how is its established ϕ pattern protected from further change or overwriting by subsequent stimuli? If such overwriting could occur fortuitously, then memory would be volatile and short-lived. Thus, the mechanism for adaptive weight change must be able to ensure the stability of desired ϕ distributions.

Duration of Established Weights

Related to the problem of overwriting is the question of the length of time a particular memory is to be maintained. Certainly forgetting is a significant phenomenon in cognitive behavior. Must the ϕ changes that represent each learned stimulus remain intact throughout the life of the neuron that undergoes such changes? If so, then forgetting must occur because of some failure to process the output of adapted cells properly. Alternatively, synaptic weights, once established after learning, might decay to their original base values. Might there be several classes of adaptive cells so that some exhibit little, if any, decay while others decay at varying rates?

The implications of all of these issues should be taken into account in the formulation of hypothesized mechanisms for adaptive neurons and in the design of the processing modules in which they serve as component parts.

A Model for Synaptic Plasticity

Figure 2.6 represents the synaptic junctions of several axons on the dendrite of an adaptive neuron. The following assumptions govern the dynamics of plastic changes in such synapses and constitute a provisional physiological model for learning and memory that can satisfy the basic computational requirements of an adaptive array-to-line filter.

1. Two kinds of biochemical species are essential for the long-term modification (increase) of synaptic transfer weight (ϕ): axon transfer factor (ATF) and dendrite transfer factor (DTF).
2. A long-term increase in ϕ can occur if and only if ATF and DTF are locally coactive within the postsynaptic dendritic matter.
3. Effective interaction of ATF and DTF can occur if and only if the presynaptic cell(s) discharges when activity in the postsynaptic target cell(s) is above some threshold level (θ).
4. During ATF-DTF coactivity, ϕ elevation occurs only at active synapses and is mediated by specific macromolecular changes induced by ATF-DTF interaction.
5. The magnitude of ϕ at each synapse is limited by saturation, determined by the maximum amount of DTF that can be utilized in the macromolecular change at the local receptor region (figure 2.7).
6. In all ATF-DTF interactions, ATF makes a fixed contribution to the transfer weight (ϕ) of its local synapse, whereas DTF is

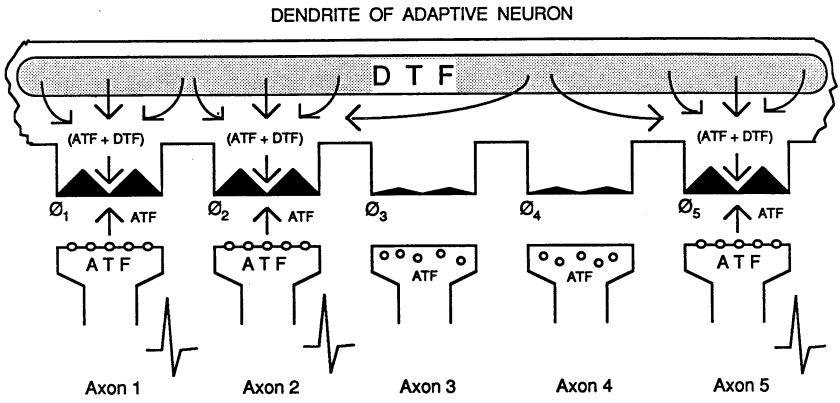

Figure 2.6
Schematic of synaptic junctions among the axons of five cells and the dendrite of an adaptive neuron. Input cells 1, 2, and 5 have fired, resulting in an ATF-DTF reaction at receptors in the corresponding synapses of the postsynaptic membrane. Volume of darkened area represents magnitude of transfer weight following synaptic modification. No change has occurred at the synapses of inactive axons 3 and 4.

distributed over all active synapses and makes a ϕ contribution to each that is, up to the local saturation limit, inversely proportional to the number of concurrently active axonal inputs on the postsynaptic cell (figure 2.7).

7. ATF is constantly generated and rapidly renewed and available for release in axon terminals.
8. DTF is renewed relatively slowly and at different rates in different populations of adaptive cells.
9. The threshold (θ) for ATF-DTF interaction is an inverse function of the concentration of free DTF in the postsynaptic dendrite (figure 2.8).
10. Synaptic transfer weights (ϕ) decay at different rates in different populations of adaptive cells.
11. There is a positive correlation between the rate of ϕ decay and the rate of DTF renewal in any given adaptive cell.

Expression 2.3 gives the general formula for determining the transfer weight of any plastic synapse that has been modified in the course of learning.

$$\underset{b\rightarrow Lim}{\phi_{im}} = b + S_{im}(c + kN^{-1}) \tag{2.3}$$

where $\underset{b\rightarrow Lim}{\phi_{im}}$ is the transfer weight of ϕ_{im}, from the basal value (b) to the saturation limit (Lim), on an adaptive cell m; b is the initial transfer

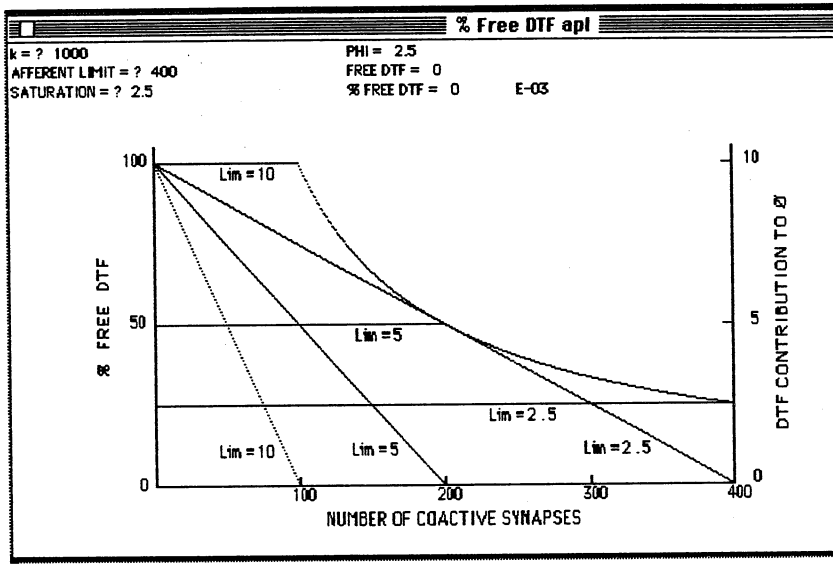

Figure 2.7
Graph showing the change in percentage free DTF and DTF contribution to the transfer weight (ϕ) as a function of the number of coactive synapses. Examples are given for three different saturation limits (*Lim* = 10, 5, 2.5) and an arbitrary initial store of DTF (k = 1000). Linear, negatively sloping plots at left represent change in percentage free DTF. Plots starting with flat horizontal segments represent change in DTF contribution to ϕ.

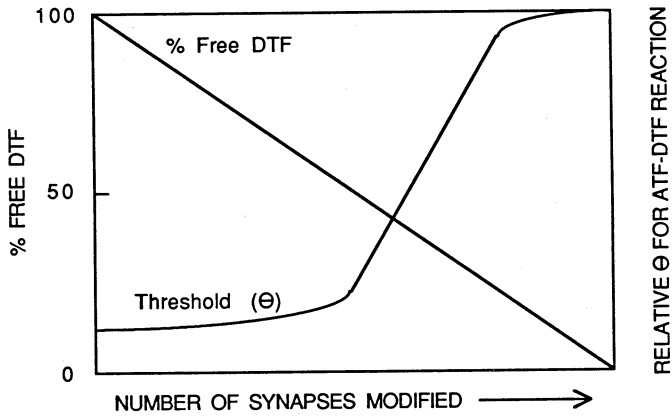

Figure 2.8
Threshold (θ) for ATF-DTF interaction as a function of the concentration of free DTF.

weight of the unmodified synapse; c is the ATF contribution from the active axonal contact; kN^{-1} is the proportional contribution of DTF in the postsynaptic cell, taking account of N coactive axons on the cell m and the total store of free DTF in the postsynaptic cell, which is represented by the coefficient k; and S_{im} is the activity level of axonal input at ϕ_{im}. It is assumed that $b < c \ll k$. These formulations constitute the basic learning rule for the systems described later.

There is no direct physiological confirmation of the specific details of this model for synaptic plasticity; however, the model is broadly consistent with a number of relatively recent empirical findings at the neurophysiological level. The discovery of a long-term increase in the efficacy of synaptic transmission in the hippocampus following brief high-frequency stimulation of the perforant pathway (Bliss and Gardner-Medwin 1973, Bliss and Lømo 1973) provided direct evidence of synaptic plasticity in vertebrates and has prompted investigators to search vigorously for the underlying mechanisms for this effect. Since the initial observations by Bliss and his colleagues of long-term potentiation (LTP) of the hippocampal response, other studies have demonstrated LTP in a number of different brain structures at the cortical and subcortical levels (Gerren and Weinberger 1983; Ito 1983; Komatsu et al. 1981; Lee 1982; Racine, Milgram, and Hafner 1983; Racine et al. 1986; Voronin 1985).

There appears to be general agreement about some of the events in the cellular processes involved in LTP. Figure 2.9 summarizes the details that have been revealed by various neurochemical probes and together seem to represent a broadly accepted basic description of neuronal processes underlying LTP (Akers et al. 1986; Bank, LoTurco, and Alkon 1987; Brown et al. 1988; Browning et al. 1979; Larson and Lynch 1987; Linden, Sheu, and Routtenberg 1987; LoTurco et al. 1987; Lynch and Baudry 1984; Murakami, Whitely, and Routtenberg 1987; Routtenberg 1984). Brief high-frequency stimulation (typically 100 pulses per second for 1 second) results in a sharp influx of calcium cations and translocation of protein kinase C (PKC) from the cytosol of the postsynaptic neuron to its membrane, where it induces a stable phosphorylation of membrane-bound protein. This phosphorylation is thought to be associated with an increase in the activity of a particular subclass of postsynaptic receptors, which occurs only beyond some high threshold of stimulation. These specialized postsynaptic receptors are called NMDA receptors because they are specifically responsive to the compound N-methyl-D-aspartate, a synthetic analog of aspartate that is a putative neurotransmitter. Significantly, low-frequency stimulation (1 pulse per second for 100 seconds) does not result in long-term potentiation. Moreover, specific chemical

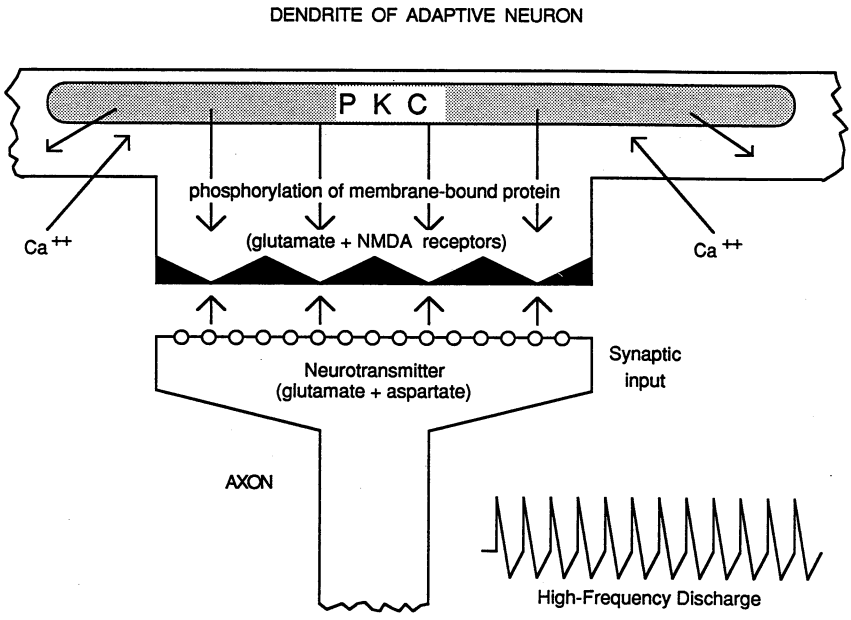

Figure 2.9
Neuronal processes underlying long-term potentiation.

blockade of NMDA receptors prevents the initiation of LTP without eliminating normal synaptic transmission. If LTP has already been induced, then blockade of NMDA receptors does not reduce the established increase in synaptic efficacy (Larson and Lynch 1987; Linden, Sheu, and Routtenberg 1987).

These recent empirical findings are consistent with the main features of the earlier proposed model for synaptic plasticity (Trehub 1975a):

1. Synaptic modification can occur on a single brief input if the axonal spike frequency is high enough to reach the threshold for protein phosphorylation.
2. The increase in synaptic weight (potentiation), once established, is relatively stable over time.
3. Aspartate (or an analog of it) provides a plausible candidate for the substance represented by ATF (the axon transfer factor in the earlier model).
4. PKC provides a plausible candidate for the substance represented by DTF (the dendrite transfer factor in the earlier model).
5. When LTP is induced, PKC translocates from the cytosol of

the dendrite to bind at the dendritic membrane. This corresponds to the reduction in free DTF after synaptic modification, as I hypothesized in my model.

The assumption of an inverse relationship between the number of coactive axonal inputs and the magnitude of the DTF contribution to each active synapse expressed in the basic learning formula (equation 2.3) is consistent with the experimental findings of Hillman and Chen (1979), who demonstrated in the cerebellum that when the number of parallel fiber inputs is experimentally reduced (fewer coactive axons), the area of postsynaptic density (transfer weight) at the lesser number of synapses on the target Purkinje cells increases proportionally. Thus, the inverse relationship between the contribution of DTF at synaptic sites and the number of coactive axons predicted in equation 2.3 conforms with a physiological process actually found to occur in a part of the brain that is accessible to the appropriate experimental procedures. The inverse relationship between transfer weight (ϕ) caused by local DTF effects and the number of coactive axons on an adaptive cell provides an intrinsic mechanism for quasi-normalization of synaptic transfer weights that is critical in pattern learning.

Summary of Neuronal Properties

The following key properties that I assume to be true characteristics of real neurons are relevant to the models in the following chapters:

- There are two major classes of neurons: excitatory and inhibitory. Spike discharge along the axon of an excitatory neuron causes an EPSP in its contiguous target neuron(s); similar discharge of an inhibitory neuron causes an inhibitory postsynaptic potential in its contiguous target neuron(s).
- When EPSPs and IPSPs occur concurrently on a common neuron, total EPSP is reduced as some monotonic function of total IPSP.
- Spatially and temporally distributed postsynaptic potentials (PSPs) are integrated in leaky fashion within the neuron.
- Whenever the integrated EPSP reaches a threshold level, the neuron discharges a spike output.
- The membrane potential of a neuron that has shifted in a positive direction, reflecting EPSP integration, can be reset to its initial resting level by sufficiently strong IPSP input to the cell.
- Some neurons (autaptic cells) receive excitatory synaptic input from recurrent collaterals of their own axons and will continue

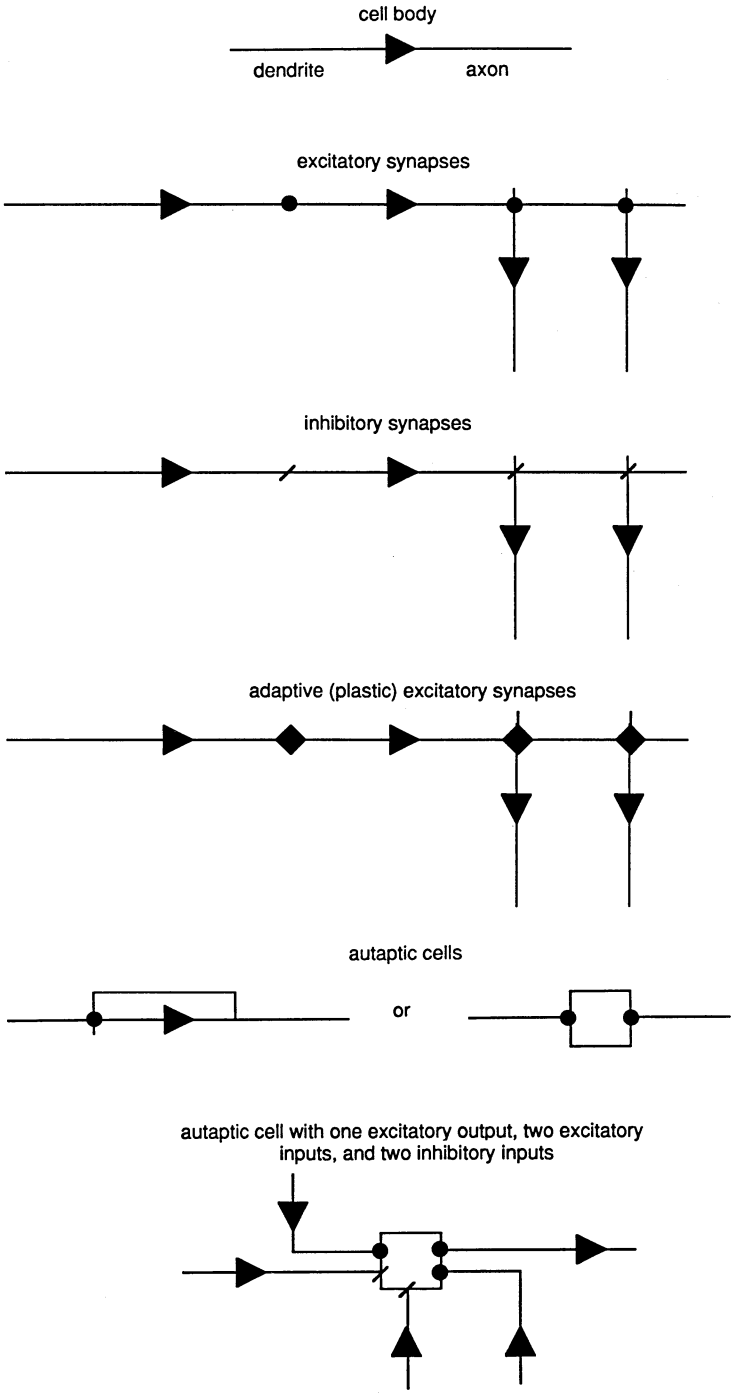

Figure 2.10
Standard symbols used for the neurons and synapses illustrated in this book.

discharging after a transient suprathreshold stimulus if there is sufficient subthreshold priming input from another source.

- Some neurons have excitatory synapses that can be modified in a graded and relatively stable fashion with respect to the magnitude of their contribution to the integrated EPSP of the associated cells (synaptic plasticity) given standard presynaptic input.
- The chemical factor within the axonal terminal of the presynaptic cell (ATF) that contributes to a change in synaptic efficacy during learning is constantly regenerated and available for use at its local synapse.
- The chemical factor within the dendrite of the postsynaptic cell (DTF) that contributes to a change in synaptic efficacy during learning is limited in quantity, is distributed over all coactive synaptic junctions up to the limit of utilization at each synapse, and is regenerated relatively slowly.
- The threshold for a reaction between ATF and DTF that is required for an increase in synaptic efficacy (transfer weight) to occur is inversely related to the proportion of free DTF in the target dendrite.

Symbols Used for Circuit Diagrams

Many of the model mechanisms and networks I set forth will be illustrated by circuit diagrams. The standard symbols I will use in these drawings are shown in figure 2.10.