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MACHINE INTELLIGENCE

PART 2: BIOLOGICAL FOUNDATIONS

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This report describes the material aspects of how perceptions, existing as codons within the neocortex, are formed through synaptogenesis, synaptic potentiation, depotentiation, and shedding. A simulation of this process on an array of transputers is also discussed.

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INTRODUCTION

Our approach to intelligent machine design is to reverse engineer the mammalian brain. A necessary foundation is a working knowledge of the pertinent neuroanatomy and neurophysiology. Constructing a thinking machine is not easy, and the circuitry of the brain is only partially known; but we will make do with what we have. Since the reality of neural evolution is our guide, we will never speculate as to the purpose of a brain structure, but only about how it contributes to survival. Neural scientists have amassed vast amounts of data on the interconnections between the brain centers, on the symptoms of human brain pathology, and on the behavior of laboratory animals with lesioned brains. (In the words of P. S. Churchland, "It threatens to become a clutter.") We cannot necessarily infer the function of a brain center from the dysfunction in its absence. However, we can use this data to guide us in the overall structure of our design.

Our basic premise is that the brain can be viewed as a passive electronic device, that the activity of the brain can be looked at as the interplay of varying potentials and currents, and furthermore, that this activity is independent of the substratum (biological neurons) and can be transferred to a different substratum (silicon) without loss of function. A second, and independent, premise is that the mind can be taken to be the electromagnetic field supported by these currents, and thus is completely independent of the substratum.

This report describes the biological foundations upon which an electronic brain can be designed. We present a condensed anatomical and physiological description of the brain to support the hypothesis of the brain as passive circuitry. Flow diagrams for the functional units of the circuitry are labelled using the jargon of the neuroscientists.

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We are not neuroanatomists. This information was gathered from many sources, but primarily from the medical texts of Heimer and of Nauta and Feirtag (refs 1 and 2).

BACKGROUND

The Neurons

We take the fundamental unit of the nervous system to be the neuron. A generalized neuron (Figure 1) consists of a soma, an axon, and a bushy tree structure of dendrites. The soma contains the cell nucleus and manufactures the proteins vital to the life of the cell. The axon protrudes from the soma at the axon hillock, and provides the principal mechanism of communication with other cells. The axon terminates in boutons at specialized points of contact with other neurons called synapses. The synapse generally occurs at the soma or one of the many dendritic branches of another neuron. The bouton contains vesicles of chemical neurotransmitters that move to the membrane and are released into the synaptic cleft when stimulated by electrical activity on the axon. When released, these molecules traverse the synaptic junction to the postsynaptic membrane on the dendrite or soma. They initiate the opening of ion channels, resulting in ion flow that chemically induces a change in the electrical resting potential of the cell (approximately -70 mV). Depending on the type of neurotransmitter released, hyperpolarizing or depolarizing potentials are pro-duced. "Dale's" hypothesis is that a given neuron synthesizes and releases only one type of neurotransmitter (ref 3), however this does not mean that the action must be exclusively excitatory or inhibitory at the synapse.

We like the working hypothesis that the postsynaptic neuron spatially and temporally integrates the potentials of the soma and dendrites. If the

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momentary depolarization at the axon hillock is sufficient, a pulse is generated. The pulse produced at the axon hillock is transmitted, without attenuation, as an action potential along the axon. These potentials are all of the same magnitude and duration (rising from -70 to +55 mV for approximately 1 ms) (ref 4). The neuron cannot fire again until after a brief refractory period, resulting in a minimum of about 6.5 ms between rising edges. It is important to note that this is a general model of neural signal transmission. In addition to axodendritic and axosomatic transmission, there exist axoaxonic (cerebellum), dendrodendritic (olfactory and retina), and dendroaxonic (substantia gelatinosa) synapses.

There are also electrical (ephaptic) synapses in which ions move directly (through opposed channels) from one neuron to another. Also, the mere proximity of a neuron to an active neuron can initiate a response. This last synapse may be a neural synchronizing mechanism.

ANATOMY OF THE BRAIN

It is estimated that the brain consists of 10^{12} neurons with as many as 10^4 connections per neuron. The interconnections between these neurons are highly ordered by simple rules. (Topology tends to be preserved as one brain center is connected to another.) The target of a tract, and the chemistry which maintains topology as the axons grow, is genetically determined; but the fine detail of connection is determined postnatally by signal energy from the environment. There is not enough genome for it to be otherwise.

There are three major anatomical subdivisions of the brain: the prosencephalon (forebrain), mesencephalon (midbrain), and rhombencephalon (hindbrain) (Figure 2). In a very real sense we have two brains, but they are so synchronized in their activity that they act as one. The dual nature of our nervous

system is ignored in the following discussion. If duality should ever prove

essential, the suggested circuitry can be replicated.

The Forebrain

The forebrain is subdivided into paired cerebral hemispheres, and the dien-

cephalon. The surface of the cerebral hemispheres is approximately 2 mm of gray

matter, predominately neural soma, called the cerebral cortex. The cortex is a

laminar mosaic of columnar volumes of similar neurons extending through as many

as six layers. The cortex folds upon itself as the cortical layers gradually

reduce to a single layer forming the hippocampus. The hippocampus plays a

crucial role in the formation of memories (engrams). Memories are not stored

there, but they are not stored anywhere (or at least they are not accessible) if

both hippocampi are dysfunctional. The white matter directly below the cortex

is comprised of tracts of myelinated axons connecting many parts of the brain.

Included in the white matter is the corpus callosum connecting the two

hemispheres.

The amygdala is a subcortical body of several nuclei which is coupled with

the cortical hippocampus and gyrus fornicatus to form the limbic system. The

limbic system is fundamental to the motivation of the mammal. It is here that

we feel loneliness, familiarity, and strangeness. It is here that we distin-

guish between dangerous threat and preposterous bluff. The structures of the

limbic system are related in that they all interact directly with the hypothal-

amus, part of the diencephalon. The hypothalamus (below the thalamus) regulates

the endocrine glands and governs basic behavior, including hunger, thirst, rage,

and predatory attack. The limbic system and hypothalamus project and receive

cortical connections from the frontal lobe.

The basal ganglia are subcortical gray matter comprising the corpus striatum, pallidum, and septum. They are responsible for complex coordinated motor control. The thalamus is a key neural structure in the diencephalon (Figure 3). It is the gateway to the neocortex for all sensory input except (possibly) olfactory. Neurons in the thalamus have a rhythmic activity and have been grouped into many nuclei (ref 5). Visual, auditory, and somatic sensory signals project to specific thalamic nuclei. The lateral geniculate nucleus (LGN) accepts massive neural input from the retina over the optic tract and relays the signals to the primary visual cortex (striate cortex, Brodmann area 17). The medial geniculate body gates auditory signals via the lateral lemniscus to the primary auditory cortex (area 41). Somatosensory input projects by way of the spinothalamic tract and medial lemniscus to the ventral posterior nucleus which relays the signals to the somatosensory cortex (areas 1, 2, and 3). The thalamic projections to the cortex are coupled with reciprocal connections from the cortex back to the thalamus.

The great conceptual division of the brain occurs between ascending and descending systems, between sensory input and motor output, between percept and program. The ansa lenticularis and brachium conjunctivum carry motor signals from the basal ganglia and cerebellum respectively to the ventral anterior nucleus and ventral lateral nucleus (VA - VL complex). The thalamus relays these signals to the premotor (areas 6 and 8) and motor (area 4) cortex.

The reticular nucleus is a structure that surrounds the thalamus and is traversed by all fibers connecting the cortex to the thalamus. The reticular nucleus has inhibitory projections on the thalamus that are capable of inhibiting the relay action of thalamic nuclei (ref 6).

The Midbrain

The midbrain is a short section of the mammalian brain comprised of the tectum and cerebral peduncles. Two pairs of protrusions on the surface of the tectum are called the superior colliculi and inferior colliculi. The superior colliculus receives massive projections from the retina and is associated with involuntary eye movements. The inferior colliculus serves as an integrating relay station for auditory signals en route to the medial geniculate body and primary auditory cortex.

The cerebral peduncle is further subdivided into tegmentum and basis pedunculus. The tegmentum contains the red nucleus and reticular formation. The red nucleus is associated with motor control and the reticular formation (reticular activating system - RAS) is fundamental to arousal and awareness. The RAS monitors sensory input in general, and arouses the brain if there is any decided change. The basis pedunculus is comprised primarily of the substantia nigra and pes pedunculus. The substantia nigra is involved with reflex motion and the pes pedunculus is a nerve tract carrying motor signals from the cerebral cortex to the spinal cord. The locus ceruleus, which inhibits motor activity during sleep, is also located in the midbrain.

The Hindbrain

The hindbrain is subdivided into the metencephalon and myelencephalon. The metencephalon is comprised of two primary anatomical structures; the cerebellum and the pons. The cerebellum is a prominent protrusion on the dorsal surface with a basic structure similar to the cerebral hemispheres. It is comprised of a laminated cerebellar cortex, and subcortical white matter and gray cerebellar nuclei. The principal neuron in the cerebellum is the Purkinje cell. By definition (ref 5), principal neurons are neurons whose axons project outside

the local brain region. The Purkinje cells occupy a single layer in the cerebellum and project entirely to the deep cerebellar nuclei.

In general, intrinsic neurons have only local efferents. Intrinsic neurons called granule cells occupy a layer beneath the Purkinje cells. Three other intrinsic neurons have been identified in the cerebellar cortex including basket, Golgi, and stellate cells. The cerebellum receives inputs from virtually all parts of the neocortex as relayed through the pons. It also receives massive sensory projections and is primarily involved with coordinating body movements.

The principal anatomical structure of the myelencephalon is the medulla oblongata, which is part of the brainstem and spinal cord. The medulla regulates visceral functions including heartbeat and respiration. The medulla oblongata along with the pons and mesencephalon are grouped together to form the brainstem.

The Spinal Cord

The spinal cord contains ascending nerve tracts carrying sensory information to the brain and descending nerve tracts carrying motor commands from the brain. The pyramidal tract is the primary descending tract from the motor cortex to the motor neurons. The lemniscal system and spinothalamic tract are two distinct tracts that project somatosensory information to the thalamus en route to specific areas on the neocortex.

The spinal cord also contains the connections involved in reflex actions where the brain is not involved.

The Neocortex

It is widely believed that the evolutionary expansion of the neocortex portion of the cerebral hemispheres into the dominant brain structure makes man

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unique. The neocortex is actually only an extension of the thalamus and man is
in no way unique (ref 7). Virtually all parts of the neocortex project to the
thalamus, striatum, pons, and much of it to the superior colliculus. The
neocortex is a laminated structure of functionally similar columns of neurons
with massive local inhibition between them. As few as 2 and as many as 60 dif-
ferent neurons have been identified in the neocortex. The two primary types of
cells are the pyramidal cell and stellate cell. The pyramidal cell is a prin-
cipal neuron with an axon that can project out of the cortex. The stellate cell
is an intrinsic neuron with only regional axonal projections. Six cortical
layers have been identified including the molecular, external granular, external
pyramidal, internal granular, internal pyramidal, and multiform. Cortical
thickness and cell density have been used to divide the cortex into approxi-
mately 50 different sections called Brodmann areas. The cortex is also parcelled
into sensory, motor, and association areas. All cortical areas receive projec-
tions from specific thalamic nuclei as well as other cortical areas. Thalamic
projections usually synapse at cortical layer 4 which is a high concentration of
receptor stellate cells. The reciprocal projections to the thalamus generally
originate at layer 6 pyramidal cells. Afferents from other cortical areas can
arrive at any layer, and pyramidal efferents to other cortical regions may
likewise project from any layer, although most originate in layers 2 and 3 (ref
8). Primary sensory areas are dominated by input from thalamic sensory relay
nuclei. These unimodal areas project to cortical areas where the signals are
integrated with inputs from other unimodal areas to form multimodal or asso-
ciation areas. These, in turn, project to other association areas creating a
hierarchical structure in the cortex culminating in the frontal lobe. Associa-
tion cortex occupies the greatest area, probably because a cortical region whose
function is not well understood is called an association area.

As an example, the optic nerve, a great tract, runs from the retina to the LGN, where it is relayed to the primary visual cortex (area 17) at the caudal end of the cortex. Cortical connections project from there to area 18, and then to area 19, eventually integrating with auditory and somatic information in tertiary associating areas and finally proceeding to the frontal lobe. The frontal cortex associates these signals with input from the limbic system and many believe this to be the highest level of association before motor response is nominated in the basal ganglia.

THE EVOLVING BRAIN

The evolution of the brain started with the first neuron, the first cell that had, as its specialty, the ability to communicate with other cells. We should not be too precise here. The sponge is probably the simplest multi-cellular animal that exists, yet its cells communicate with each other. Touching the sponge at one point, causes waves of contraction to spread out from the point that was touched. Still none of the cells of the sponge are neurons (or else they are all neurons); they are not specialized for communication, yet they communicate.

There are sensory cells. The environment (exterior and interior) interfaces with sensory neurons through sensory cells. The distinction is artificial, but it proves useful.

In order to simplify the discussion, we will lump all the sensory systems into one and call it the "sensor." For the same reason, we will lump all of the brain's output together and call the recipient of this output, the "motor." This is much the case in practical robotry, where one tends to see interfaces erected between the sensors and the processing unit on the one hand, and between the central processor and the robotic mechanisms on the other. We skip over the

simplest organisms, and start out with the neurons already specialized into sensory and motor types.

<center>sensor -> motor</center>

The reactions of this organism, at first glance, are wholly determined. Touch it at a certain spot, and it moves in a certain way. However, a phenomenon known as habituation has appeared. If the organism is repeatedly touched at the same spot, the amplitude of its reaction steadily decreases. It is as if the organism is learning that something new has appeared in its universe, and that something new is harmless. Even with this simplest of neural systems, there is plasticity, there is change, and that change cannot be distinguished from "learning." This arrangement is found in certain jellyfish.

In still other jellyfish, a new neuron is found, specializing in neither sensation nor initiating movement, but solely in communication; it is the interneuron. The interneuron transmits signals from the sensory neuron to the motor neuron, and to other interneurons. The internet has appeared. From now on there will not necessarily be a simple relationship between sensory input and motor output. The internet is the brain. After this, the evolution of the nervous system is only a complication, there is nothing new.

<center>approach
sensor ->   -> motor
flee</center>
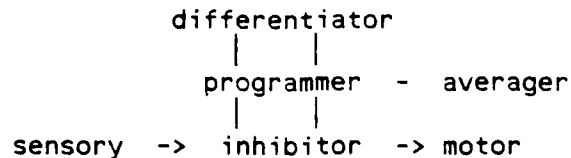
This simple organism lives by one rule: eat, or be eaten. Go after the food, but run from the predator. Any big, moving thing is a potential predator. It should be noted that passive avoidance does not require any special mechanism. If neither approach nor flee is activated, then there is no way of distinguishing the resulting behavior from "passive avoidance."

<center>10</center>

With the filter feeder, any change in the environment signals a predator. However, does a change in the environment signal prey or a larger predator?

```
              the differentiator
                   |    |
                approach
     sensor ->              -> motor
                  flee
```
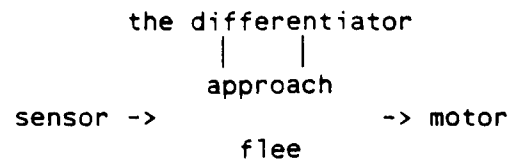
A new function appears. It was there, in potential, as soon as there were more than two interneurons. Now there is the capability for distinguishing between dangerous big things and harmless big things. The survival increment is that feeding is more efficient if it is not interrupted by every big thing. Whether this learning is genetic or epigenetic, is not all that important.

```
              differentiator
                 |    |
             programmer  -  averager
                 |    |
       sensory -> inhibitor -> motor
```

Now we see an entire brain. The inhibitor is capable of holding up the incoming signal energy, and refusing to release any indicated motor programs, until it is satisfied. And what indicates satisfaction? The inhibitor is satisfied when a motor program is suggested that has no bad consequences, given the present environment. One notes that there is no mention of decision. Afferents that excite the inhibitor are "bad"; those that disinhibit the inhibitor are "good." As long as the suggested motor program, coupled with the present signal energy filtered through the discriminator, excites the inhibitor, there will be no motor output.

Sensory stands for all the sensory neurons, except (possibly) olfactory. The inhibitor is the thalamus. The discriminator is the neocortex. The programmer is composed of the basal ganglia. The averager is the cerebellum.

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The motor is the musculature of the body. Here we have the whole functional brain.

The signal energy flows through the thalamus to the neocortex, and from the neocortex to the basal ganglia. As the energy flows through the basal ganglia, a motor program is nominated. The energy flows back from the basal ganglia to the neocortex, and forward through the thalamus. The motor program is blocked by the thalamic reticular nucleus until it is inhibited by outflow from the neocortex. We say that as long as the neocortex output is exciting the reticular nucleus of the thalamus, we are undecided. As soon as the reticular nucleus is inhibited, we say that a decision has been made.

The role of the cerebellum is to keep a running average of executed motor programs.

LEARNING

Epigenesis

The brain of the human infant contains more neurons, yet weighs only one fifth as much as an adult brain. Development continues several years after birth through the growth of axons, dendrites, and the formation of new synapses. It is clear that genetic material alone cannot establish all of the estimated 10^{16} synaptic connections in the cerebral hemispheres. Genes determine reproducible patterns of cerebral organization by defining a neural "growth cone." Neuronal growth is guided by this genetic envelope with the distribution of final synapses resulting from epigenetic experience (ref 8). As an example, genes determine the regular geometric mapping from the retina to the LGN. Topology is preserved as the connections are relayed to the striate cortex. Yet

it has been shown that connectivity within the striate cortex can be altered by changing the environment during postnatal development (ref 9).

Transient Redundancy

There is profuse axonal growth in early years of postnatal development called transient redundancy (ref 8). Many connections are established that are not utilized and are eventually lost later in life. Simple rules govern the behavior of the expanding network. As the cerebral hemispheres develop, sensory input is channeled through the thalamus to specific regions on the cortex. This signal energy, coupled with inputs from other brain centers (hypothalamus, visceral), determines the final distribution of synapses within the constraints of the genetic envelope. Cortical connections are established reflecting the nature of the input and the consequences of that input. The signal energy is epigenetic experience. Connections are established which tend to maximize the divergence among epigenetic events. Highly active neurons have a larger propensity for growth and it is the environment that determines which cells are active. It is hypothesized that inactive cells secrete a trophic material that attracts the growing axons of the growth cone. Consequently, inactive neurons are prone to new excitatory innervation. This pairing of hyperactive presynaptic cells with hypoactive target cells results in the environment evoking the maximum neural response from the postnatal brain (ref 10). We see the function of growth as the environment fine-tuning the brain, rather than the genome matching the environment. Synaptogenesis is discussed in more detail in Reference 11.

Selective Stabilization

Redundant connections are shed through selective stabilization of synapses as afferents compete for available postsynaptic sites. Selective stabilization is a combination of associative learning and synaptic shedding (ref 8). A popular position is that in the case of the 85 percent of synapses which are excitatory, the efficiency of the synapse increases when both the presynaptic and postsynaptic cells are hyperactive. This is Hebb's law. Associative learning is Hebbian learning. Postsynaptic hyperactivity coupled with presynaptic inactivity results in a decrease in the efficiency of any common synapses. Therefore, those cells contributing the most to the hyperactivity of the postsynaptic cell acquire the most postsynaptic area. The afferents with minimal influence on postsynaptic cell activity are "pushed out." These synapses are the redundant connections that are eventually shed.

THINKING

Of all the categories of mental activity, this is the easiest to explain. It is an epiphenomenon. Carnivores routinely display foresight in their hunting strategies. They proceed with intention. Human thought is merely foresight carried beyond what is useful.

It is a simple, straightforward process. Any complication must be in the multiplicity of units, for the available genetic material can only establish primitive patterns and overall designs for proliferation. Secondly, it must be an inherent operation, not forced from the outside. Both of these constraints are satisfied if we think of association as the natural activity of the cortex when it is not receiving sensory information, and as the activation of neurons in patterns which are related to the patterns excited by sensory input.

If we look at a tree, a pattern of neurons is excited. Changeux (ref 8) defines this as a perception. If we are thinking of a tree, some of the same neurons are excited. Changeux defines this as a mental object. We define both as a codon. It is the signal energy from the environment, internal and external, flowing through the cortex that causes the molecular rearrangements within the brain called codons. A codon is the result or record of an experience. It has the static aspect of existing as potentiated synapses and a dynamic aspect of channeling signal energy. At each "moment" there is an active codon in the neocortex. These moments come at about ten to the second (10/sec) as marked off by the rhythmic activity of neurons in the reticular nucleus of the thalamus. The thalamus provides the channels through which the signal energy flows on its way to the motor. It furnishes the channel complexity which allows one set of signals to activate one motor pattern, and a slightly different set of signals to activate another motor pattern. The complexity is in the neocortex which, functionally, is an extension of the thalamus. The incoming signals are forwarded to the neocortex, and the energy flows back to either excite or inhibit the thalamic reticular nucleus (Figure 4). An active thalamic reticular nucleus inhibits the forwarding of a program to the motor. The energy continues to flow back from the neocortex until the thalamic reticular nucleus is inhibited which allows the motor program to complete. Codons represent the activity of a large number of neurons in the various cortical regions. Some of these codons return signals which are routed to the motor cortex and result in a response to the stimulus. The massive reciprocal connections from the cortex to the thalamus may result in a codon evoking a thalamic response that alters the gating cycle. If a codon is excited which has excitatory synapses on the thalamic reticular nucleus, then the relay of signals to the motor cortex is inhibited and the

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organism hesitates while thinking continues. When a codon is excited that does not result in excitation of the "bad" center, the inhibitor is inhibited, the motor signals pass through to the motor cortex, and the sensory signals pass through to the primary cortex. The organism moves and starts thinking about its next step. This inhibiting of the inhibitor is the material side of "making a decision." Hesitation is its obvious outside indication. If there is a delay between stimulus and response, something is going on, and that something is thinking. When sensory input is blocked for an extended period, the codon dies down as its store of molecules is depleted. Dynamic instability in the neocortex insures that as one assemblage of neurons dies down, an associated set of neurons is excited.

This fading in and out of associated sets of cortical neurons (free association) is the mind thinking. Thinking did not suddenly appear with Cro-Magnon man, it evolved gradually from the first protochordate. It is quite possible, that as we go down the phylum we will arrive at an organism, of which we will say, "It does not think." But that organism must be a long way down, way too far down for us to say "Man is the only thinking animal." It would seem much easier to take the position that mice think, than to take the alternative position that chimpanzees do not think. Brain systems are important.

SUMMARY

The combination of synaptogenesis and selective stabilization is learning. It is a network with growth, pruning, potentiation, and depotentiation. Neural axons grow and aggressively seek out places to form new synapses. Synapses which are not effective are pushed out. In this way the environment tunes the brain.

It is the mind that thinks; the brain associates. Association is the material aspect of the fading in and out of codons in our awareness when sensory input is blocked by the thalamus. We are speaking of the philosopher's intention. This fading in and out of codons is the normal activity of the neocortex whenever it is not receiving signal energy. In our awareness, this results in fragmentary visions of past experience, flowing freely and forming new combinations which have never been experienced together. This is the philosopher's chimera, and the process is his rational thought.

When this process results in a mountain goat adjusting his leap so that he will land with the proper attitude to make the next leap possible, it has an obvious survival increment. When it results in a man writing a sonnet, the survival increment is not nearly as obvious. In fact, the very definition of the intelligence quotient suggests that an IQ of 100 confers the greatest survival increment upon a member of the human race.

REFERENCES

1. Heimer, L., The Human Brain and Spinal Cord, Springer-Verlag, New York, 1983.

2. Nauta, W.J.H. and Feirtag, M., Fundamental Neuroanatomy, W.H. Freeman and Company, New York, 1986.

3. Dale, H.H., "Pharmacology and Nerve Endings," in: Proc. Roy. Soc. Med., Vol. 28, 1935, pp. 319-332.

4. Kuffler, S.W., Nichols, J.G., and Martin, A.R., From Neuron to Brain: A Cellular Approach to the Function of the Nervous System, 2nd ed., Sinaur, Sunderland, Massachusetts, 1984.

5. Shepard, G.M., The Synaptic Organization of the Brain, 2nd ed., New Oxford University Press, New York, 1979.

6. Waszak, M. and Schlag, J. "Responses of Cells in Thalamic Reticular Nucleus to Thalamic and Cortical Stimulation," Federation Proc., Vol. 30, 1971, p. 489.

7. Penfield, Wilder, The Mystery of the Mind, The Princeton University Press, Princeton, New Jersey, 1975.

8. Changeux, J.-P., Neuronal Man, Pantheon Books, New York, 1985.

9. Hubel, D.H., Wiesel, T.N., LeVay, S. "Plasticity of Ocular Dominance Columns in Monkey Striate Cortex," Philos. Trans. R. Soc. Lond. [Biol.], Vol. 278, No. 961, April 26, 1977, pp. 377-409.

10. Levy, W.B. and Desmond, N.L., "The Rules of Elemental Plasticity," in: Synaptic Modification, Neuron Selectivity, and Nervous System Organization, (Levy, W.B., Anderson, J.A., Lehmkuhle, S., eds.), Lawrence Erlbaum Associates, Hillsdale, New Jersey, 1985.

11. Scanlon, R. and Johnson, M., "Synaptogenesis," U.S. ARDEC Technical Report, Benet Laboratories, Watervliet, N.Y., to be published.

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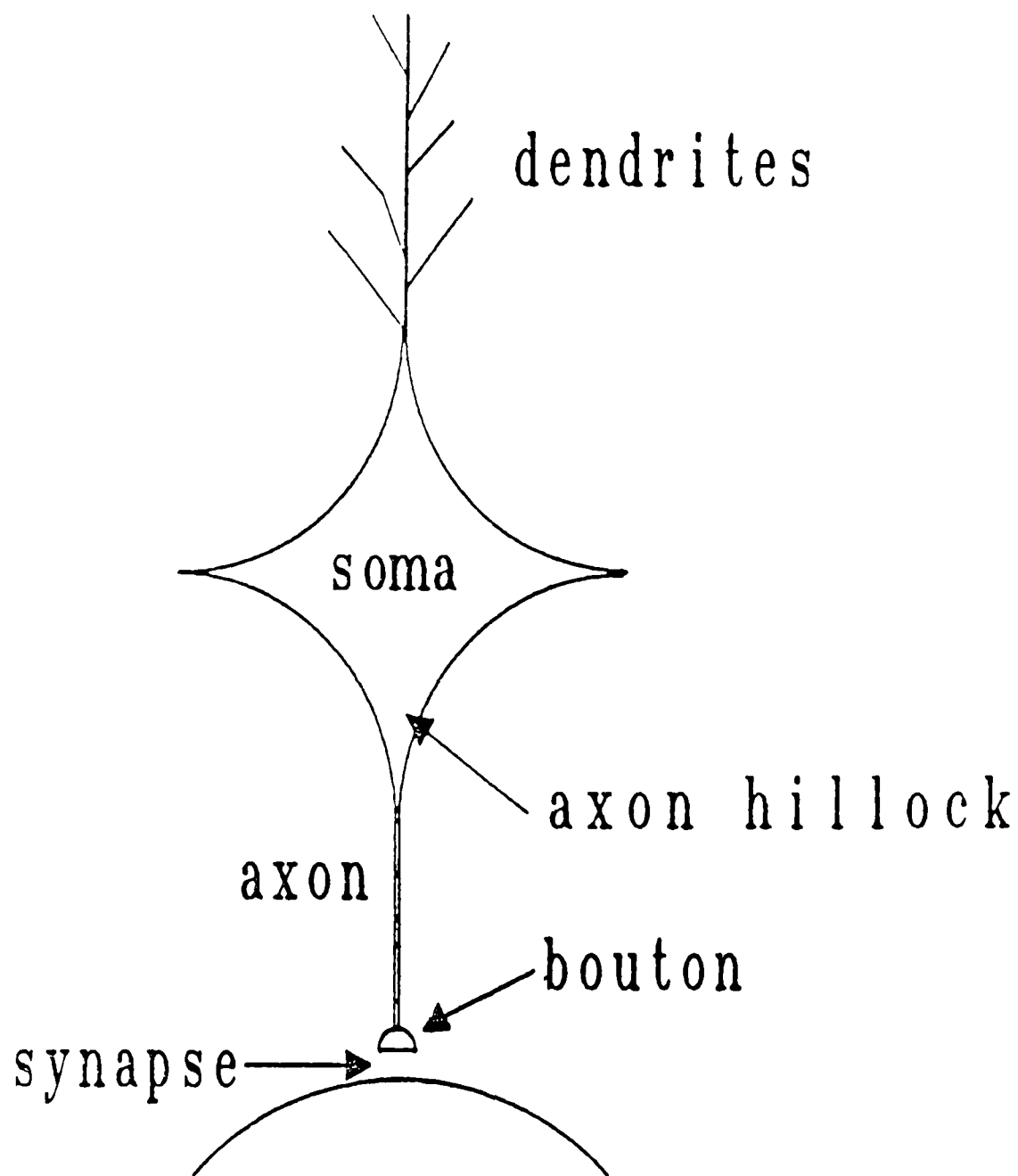
Figure 1. Generalized neuron.

SPINAL CORD

ascending — spino-thalamic tract, lemniscal system

descending — pyramidal system

HINDBRAIN

metencephalon — cerebellum, pons

myelencephalon — medulla oblongata

MIDBRAIN

mesencephalon — tectum — superior colliculus, inferior colliculus

cerebral peduncles

locus ceruleus

FOREBRAIN

telencephalon — cortex (hippocampus)

basal ganglia — corpus striatum, globus pallidum, septum

limbic system — amygdala, hippocampus, gyrus fornicatus

diencephalon — thalamus hypothalamus

Figure 2. Major subdivisions of the brain.

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superior colliculus   retina

optic tract

lateral   inferior
lemniscus   colliculus

spinothalamic tract,   somatic
medial lemniscus   sensory

ansa lenticularis   basal ganglia

brachium conjunctivum   cerebellum

lateral geniculate nucleus

medial geniculate body

ventral nucleus

ventral posterior nucleus

ventral anterior nucleus
ventral lateral nucleus
(VA–VL complex)

area 17 striate cortex

area 41 primary auditory cortex

areas 1,2,3 somatic sensory cortex

areas 6,8,7 premotor & motor cortex

Figure 3. The thalamus.

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Figure 4. Cerebral thinking architecture.

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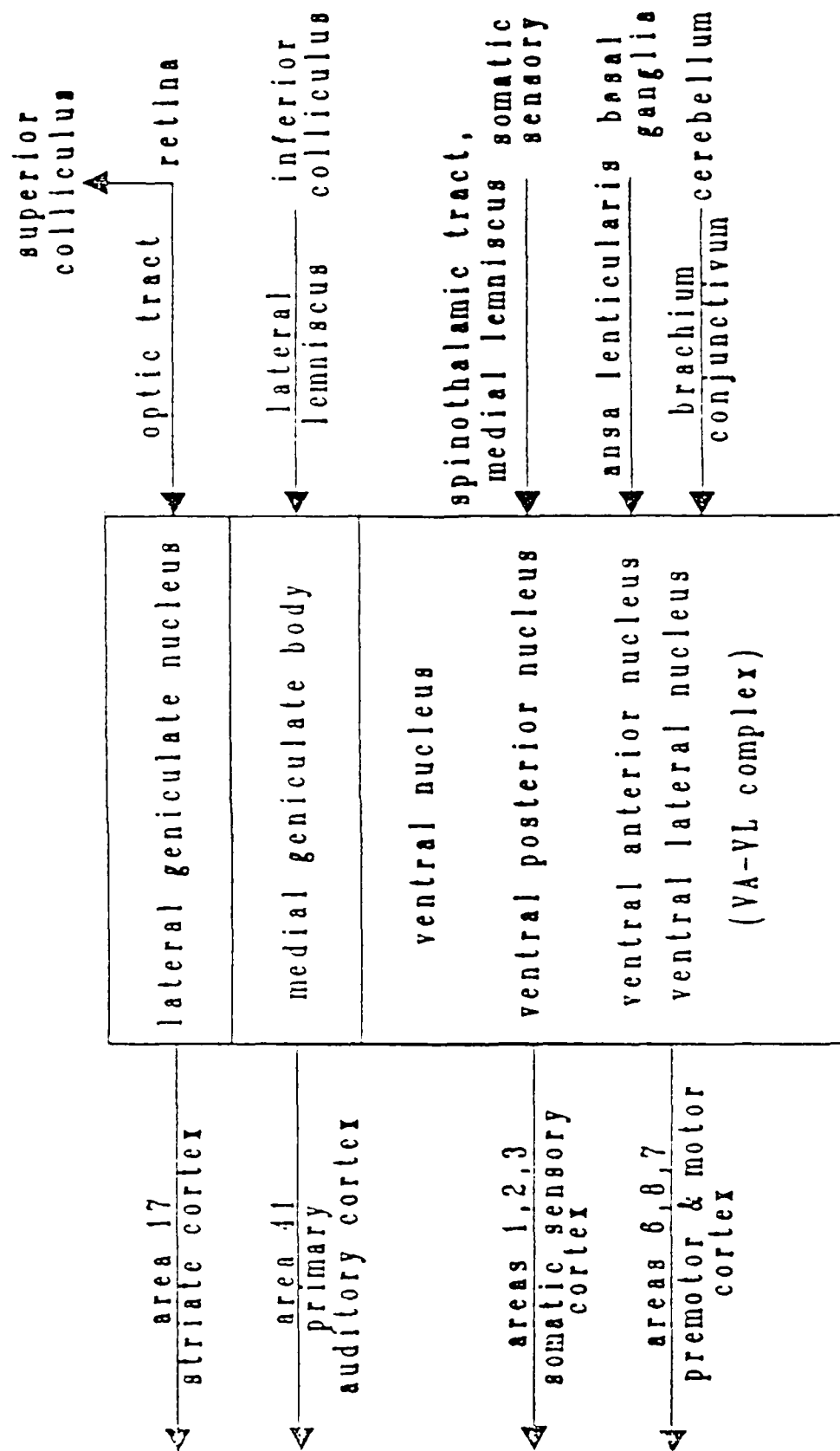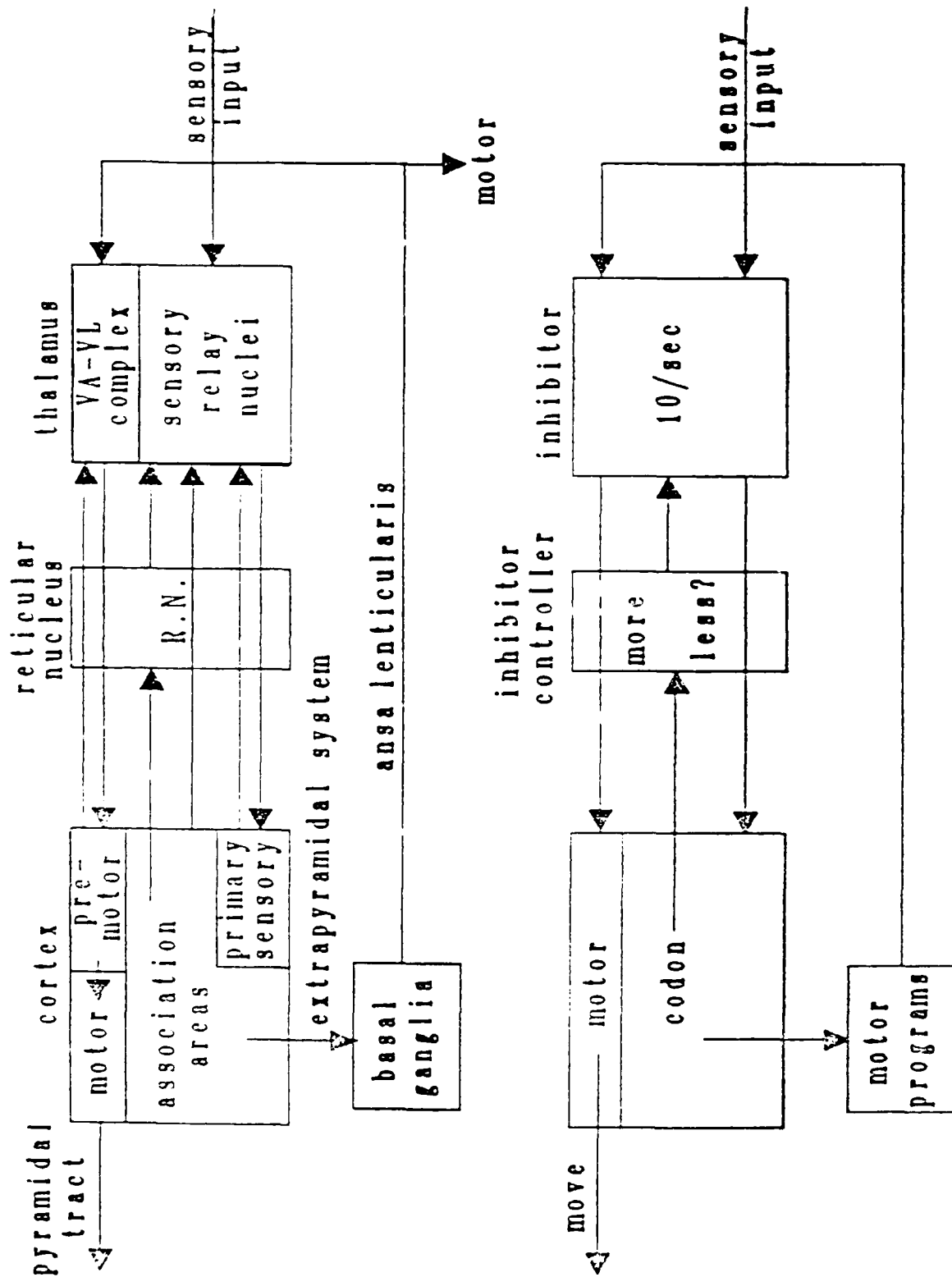   -DA   1
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