Clinical Electroencephalography

Fourth Edition

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Preface

The great and growing importance of event related potentials has evoked a new chapter from the enthusiastic pen of Adrian Upton, one time colleague in Newcastle upon Tyne, now Professor of Neurology at McMaster University. Although 'specific' responses are, it is currently realized, rather less specific than was once thought, there is no doubt that these techniques have achieved a lasting place in neurophysiological investigation.

On the other hand, the clinical exploitation of overnight sleep recording has not developed to the same extent. The former chapter on special techniques has therefore been dismembered and the parts distributed to the appropriate places in this edition. Long-term monitoring by radio or cable telemetry, or by means of a portable cassette recording system, has already had some impact on the investigation of attack disorders and is discussed in the final chapter.

The great majority of the illustrations are the same as in previous editions in keeping with the emphasis on basic techniques and applications which change little over the years. The 10–20 system of electrode placement is more widely accepted and used than at the time of the previous edition and there is now probably a more general acceptance of bipolar derivation as *sine qua non* for routine work. This must *not* be taken to mean that it is the *only* necessary method of recording: common and average reference techniques—not to mention the latest technique of source derivation—all have a place in certain circumstances and are occasionally essential for a proper display of the scalp EEG.

We are indebted to the staff of the EEG Departments within the Divisions of Psychiatry and Neurology in the hospitals of the Newcastle Area Health Authority (Teaching) and of the Division of Neurology, McMaster University, and to those who have allowed us to use material from other sources. The Department of Photography at Newcastle and the Audio-Visual Department at McMaster University are to be thanked for preparation of the illustrations. We are grateful also for the help of our secretarial assistants: Janet Barton, Linda Glazier and Judith Oxford.

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Historical introduction

The distinction of making the first observations of the electrical activity of the brain goes to Caton who, in 1875, reported that he had detected currents from electrodes placed on the skull or exposed brain in rabbits and monkeys, but it was not until half a century later that Hans Berger recorded the first human electroencephalogram (EEG) from electrodes on the scalp. Over the ensuing decade he recorded from a wide variety of patients, including many with psychiatric disorders. His findings were published between 1929–38, principally as a series of papers in the *Archiv für Psychiatrie und Nervenkrankheiten*; these are available in an English translation by Gloor (1969). Although Berger took many precautions to avoid contamination of his records by artefacts, his original publications were received with scepticism. It was not until some five years after they first began to appear that Adrian and Matthews confirmed his discovery of the 'alpha rhythm' and by a demonstration to the Physiological Society, ensured recognition of his findings.

The initial reluctance of physiologists to accept Berger's work was in part because of legitimate doubt about the reliability of his technique, which initially consisted of connecting a galvanometer directly between a pair of electrodes either inserted into or placed on the scalp, and in part because of the unfamiliar form of the brain potentials themselves. A galvanometer that is sufficiently sensitive to give an indication of heart potentials of the order of a millivolt is not a satisfactory instrument for the examination of brain potentials which may be ten or even a hundred times smaller. No electrical amplification was used and the optical system whereby Berger first recorded the oscillations of a mirror galvanometer on moving film might well have been susceptible to mechanical vibration. Thus some of his early photographic records showed only the merest ripple of allegedly cerebral activity. The electrocardiogram and the action potentials of muscle and peripheral nerves were well enough known at the time, but the fluctuating rhythmicity and relatively smooth contour of brain potentials bore little resemblance to them. Furthermore, the electrical activity of muscle and nerve is directly related to their physiological activity, whereas the rhythm from the brain appeared to be greatest during mental relaxation. These considerations contributed to the general view that Berger's records were artefactual.

The work of Adrian and Matthews (1934 a & b) carried out with the aid of a valve amplifier and pen recorder, left no doubt about the authenticity of Berger's findings which, by the end of 1934, had been repeated by Davis and Jasper in the United States of America. The potentialities of the new technique were then appreciated and workers in several centres began to apply it to the investigation of cerebral disorders. Berger's observation that epileptic seizures were accompanied by major electrical disturbances in the brain was confirmed and Walter (1936) was the first to demonstrate an association between the presence of focal slow waves in the EEG and a cerebral tumour.

Grass and Gibbs (1938) were the first to attempt a spectral analysis of EEG data, but it was Walter (1943) who designed and built the first practical frequency analyser. Further crucial advances were made by Dawson when he used photographic superimposition for the delineation of evoked responses (1947) and subsequently designed the first averager (1951) for signal enhancement. The development and application of these methods of analysis have opened new fields in clinical neurophysiology, some of which are described in Chapters 10 and 11.

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Contents

1 Anatomy and physiology of the cerebral cortex

In order to understand the origin of the electrical activity that can be recorded from the surface of the head it is necessary to study the structure and function of those cells in the cerebral cortex which are generating the activity. Therefore, succeeding parts of this chapter deal with the gross anatomy of the brain, the morphology of typical cortical nerve cells and the different kinds of electrical activity developed by cortical cells. The architecture of the cerebral cortex is described and is followed by accounts of the dynamic interrelationships of cells and of the functional properties of large areas of cortex. Finally, consideration is given to two regions of the brain which are of particular relevance to EEG phenomena—the thalamus and the reticular formation. The various aspects of cortical structure and function are integrated in Chapter 2, which describes the neural mechanisms underlying the EEG.

Gross anatomy of the brain

The brain consists of two cerebral hemispheres, the cerebellum and the brainstem; the latter is subdivided into the midbrain, pons and medulla (*Figure 1.1a*). Occupying a large central region throughout the length of the brainstem is a diffuse complex of nerve cells and fibres termed the *reticular formation*. In a

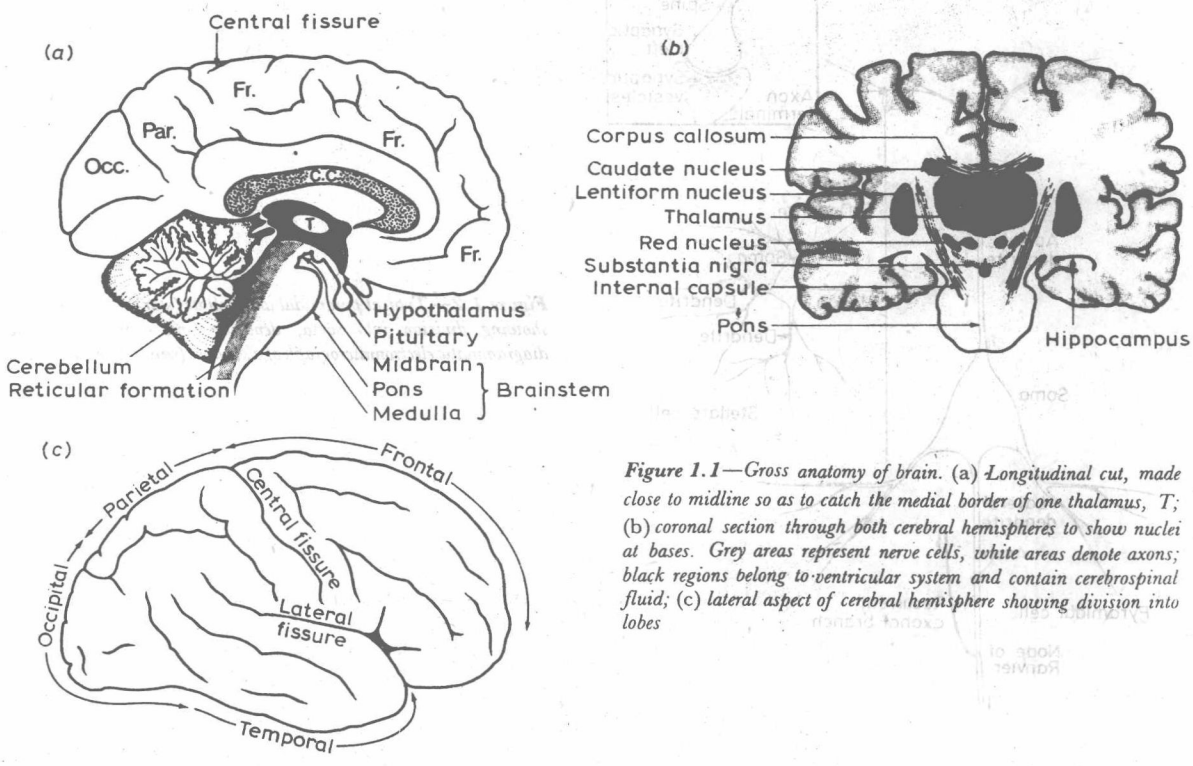

Figure 1.1—Gross anatomy of brain. (a) *Longitudinal cut, made close to midline so as to catch the medial border of one thalamus, T;* (b) *coronal section through both cerebral hemispheres to show nuclei at bases. Grey areas represent nerve cells, white areas denote axons; black regions belong to ventricular system and contain cerebrospinal fluid;* (c) *lateral aspect of cerebral hemisphere showing division into lobes*

rostral direction the reticular formation merges with the *thalamus*, which is one of several large clusters of nerve cells (*nuclei*) lying at the base of each cerebral hemisphere (*Figure 1.1b*). The large inner part of each hemisphere contains only the fibres of nerve cells and therefore appears white; the bodies of these cells are restricted to the outermost 1–4 mm where they form the cerebral cortex. The surfaces of the two hemispheres are indented by a number of fissures (*sulci*) of which the largest are the central (Rolandic) and lateral (Sylvian); the rounded areas of cortex lying between the fissures are termed *gyri*. The fissures have been used to subdivide the cerebral cortex on each side into four lobes—frontal, parietal, occipital and temporal (*Figure 1.1c*). Running between and inter-connecting the two cerebral hemispheres is a large band of nerve fibres termed the *corpus callosum* (CC in *figure 1.1a*).

Morphology of neurones

The central nervous system is made up of two types of cell—the nerve cell proper, or *neurone*, and the *glial* cell. The glial cells far outnumber the neurones and provide a structural framework within which the latter are held; they also form myelin coverings for the axons of some neurones. In addition, the close proximity of glial cells to neurones raises the possibility of important metabolic interrelationships. Although glial cells might conceivably contribute to very slow changes in cortical potentials, they do not discharge impulses or show any other evidence of excitability. In the next chapter it will be seen that the EEG can be adequately accounted for in terms of the known behaviour of neurones.

It has been estimated that there are in the region of 2.6×10^9 neurones in the human brain (Pakkenberg, 1966) and that a slab of cortex with a surface area of 1 mm^2 contains approximately 50 000 (Thompson, 1899). The shapes and sizes of neurones vary enormously but all possess the same anatomical subdivisions (*Figure 1.2*).

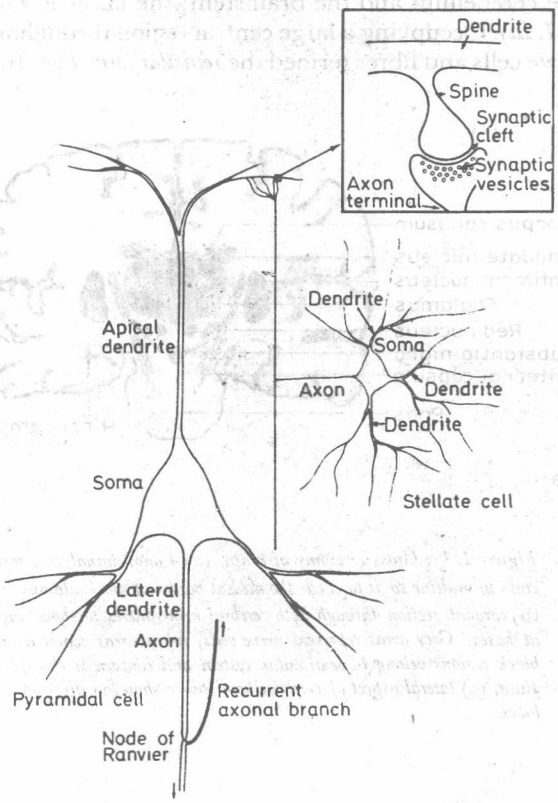

***Figure 1.2**—Typical pyramidal and stellate (star) cells, in each case showing division into soma, dendrites and axon; inset shows diagrammatic electronmicrograph of a synapse* (see *text for dimensions*)

1. The *soma*, or cell body, contains the nucleus, Golgi apparatus and Nissl substance (RNA). Apart from directing the metabolic activities of the whole neurone, the soma also receives information from other neurones through synapses on its surface.
2. The *dendrites* are tapering structures arising from the soma which branch repeatedly; they are specialized for receiving inputs from other nerve cells through numerous synaptic connections.
3. The *axon* is a process for sending impulses to other nerve cells. In some neurones the impulses are initiated at the junction of the axon and the soma—the axon hillock—and are then transmitted along the axon to other nerve cells. Some axons are surrounded by myelin sheaths derived from glial cells and this substantially increases the velocity of impulse propagation. When the axon nears its target cells it divides into many fine branches which form synapses on the soma, dendrites or axons of these cells.
4. The *synapse* (*Figure 1.2*) is a specialized interface between two nerve cells where the respective membranes are separated by a narrow cleft, typically 200 Å wide. The rounded swelling of the axon terminal contains various organelles including synaptic vesicles; these are spherical or ovoid structures some 500 Å in diameter which are thought to contain the appropriate chemical transmitter substance.

Electrical activity of neurones

The advent of microelectrode recording has made it possible to study the electrical behaviour of individual cortical neurones *in situ*; the most detailed information has been obtained by impaling the soma membrane with a microelectrode and measuring the transmembrane potential with respect to a gross extracellular electrode. It has been found that all neurones possess an appreciable membrane potential at rest and that this can be modulated by excitatory or inhibitory synaptic inputs from other cells. The ionic basis of the membrane potential may now be considered.

Resting membrane potential

When a neurone is relatively quiescent, receiving minimal synaptic bombardment from other nerve cells, a potential difference exists across its membrane such that the inside of the cell is some 80 mV negative with respect to the outside. This 'resting potential' is a necessary consequence of the differences between concentrations of ions inside and outside the cell. These concentration differences result from two factors.

1. The negatively charged ions (anions) inside the cell are mainly structural proteins and are not free to diffuse.
2. The resting membrane is freely permeable to potassium ions (K^+) and chloride ions (Cl^-) but not to sodium ions (Na^+).

The internal anions exert an electrical force which attracts cations into the cell; however, only K^+ can diffuse freely across the membrane and hence their internal concentration is raised. Nevertheless the internal K^+ concentration is not quite sufficient to equalize the internal anions because K^+ tends to leave the cell by diffusing passively down its concentration gradient. The surplus of anions inside the cell is responsible for the internal negativity of the resting potential. Under equilibrium conditions the inward (electrically driven) flow of K^+ balances the outward (chemically driven) one; the potential across the membrane (E_m) is then the potassium 'equilibrium potential' (E_k) and its relationship to the potassium concentrations inside and outside the cell is given by the Nernst equation:

$$E_k = -\frac{RT}{F}\log_e \frac{(K_i^+)}{(K_o^+)}\,\text{mV}$$

Where R is the universal gas constant, T is the absolute temperature and F is the Faraday constant; (K_i) and (K_o) are the internal and external concentrations of potassium respectively. At a body temperature of 37°C the equation can be simplified to:

$$E_k = -61 \log_{10} \frac{(K_i^+)}{(K_o^+)} \text{mV}$$

If it is assumed, for convenience, that the intracellular K^+ concentrations are 150 and 5 mM respectively, then:

$$\begin{aligned} E_k &= -61 \log_{10} 30 \text{ mV} \\ &= -61 \times 1.48 \text{ mV} \\ &= -90.1 \text{ mV}, \end{aligned}$$

a value close to observed measurements of resting potential in mammalian nerve and muscle cells. Like K^+, Cl^- passes freely across the cell membrane and is distributed unequally on the two sides. This inequality is due to the fact that, whereas organic anions provide most of the negative charges inside the cell Cl^-, being an abundant anion, is required to balance the external cations (mostly Na^+). The Cl^- equilibrium potential, calculated from the Nernst equation, also approximates to the actual resting potential.

Synaptic potentials

In the central nervous system the nerve cell membrane is never completely at rest since it is continually influenced by activity in other neurones, with which it has synaptic connections. These influences may be excitatory or inhibitory but, although the final effects on the cell are different, the preliminary stages in synaptic transmission are similar. The most likely sequence of events may be summarized in the following way.

1. The arriving nerve impulses depolarize the axon terminal.
2. This depolarization allows calcium ions (Ca^{++}) to enter the axon terminal.
3. The calcium ions cause many, possibly several hundred, synaptic vesicles to empty their transmitter substance into the synaptic cleft. Each vesicle probably contains several thousand molecules of transmitter substance.
4. The transmitter molecules rapidly diffuse across the cleft and combine with receptor molecules on the 'postsynaptic' membrane of the dendrite or soma.
   The subsequent stages depend on the nature of the transmitter.
5. If the transmitter is *inhibitory*, it increases the permeability of the membrane for Cl^- and 'stabilizes' the membrane. If the membrane is already partially depolarized the inhibitory transmitter action temporarily restores it to the resting level; this induced change in membrane potential is termed an *inhibitory postsynaptic potential* (IPSP; *Figure 1.3*).
   If the transmitter is *excitatory*, it causes the sodium permeability of the postsynaptic membrane to increase and the membrane potential falls (depolarizes). If the excitatory synapse is on the soma or is situated close to the soma on a dendrite, this depolarization is termed the *excitatory postsynaptic potential* (EPSP; *Figure 1.3*).
6. If the excitatory synapse is on a dendrite the depolarization may set up an impulse in the dendrite which propagates relatively slowly, for example, 0.3–0.5 m/second, towards the soma (Cragg and Hamlyn, 1955). The continued passage of the impulse will depend on the presence or absence of impulses in other parts of the dendritic tree. As the dendritic impulse approaches the soma it causes an increasingly large current flow across the membrane of the soma and axon hillock. It has been suggested (Diamond, Gray and Yasargil, 1969) that excitatory synaptic transmission will be particularly effective at synapses located on dendritic spines.

7. If the depolarization of the soma or dendrite is sufficiently large, it causes an action potential to be set up in the axon hillock or soma of the neurone.
8. The transmitter is hydrolysed by enzymes situated on the postsynaptic membrane and the reaction products enter the axon terminal for recombination.

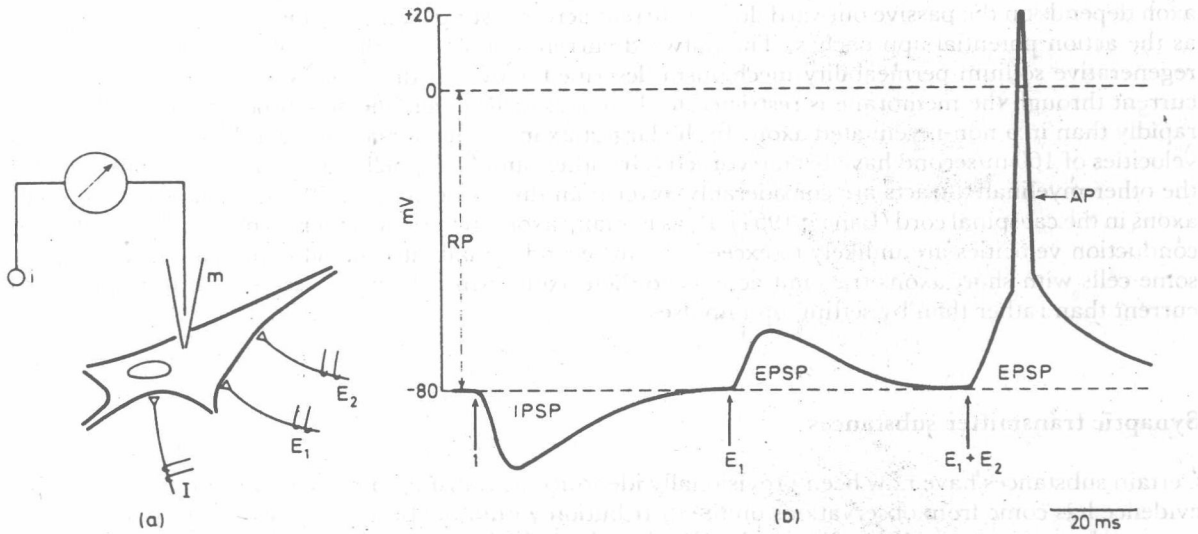

Figure 1.3—(a) *Technique for recording membrane potential from cortical neurone with intracellular microelectrode* (m) *and extracellular indifferent electrode* (i). E_1 *and* E_2 *represent two groups of excitatory axons;* I *is an inhibitory group.* (b) *membrane potential of neurone at rest and following stimulation of inhibitory axons* (I), *one group of excitatory axons* (E_1), *and both groups of excitatory axons* ($E_1 + E_2$). RP, *resting potential;* AP, *action potential;* IPSP, *inhibitory postsynaptic potential;* EPSP, *excitatory postsynaptic potential* (see *text*)

The account of synaptic transmission given above probably applies to the majority of neurones in the CNS. However, recent experiments have raised the possibility that there may be some situations where synaptic transmission is not mediated by chemicals. Instead, sufficient 'electronic' current flows from the axon through the postsynaptic membrane to depolarize the latter. Finally, mention should be made of a second type of inhibition which, although widely present elsewhere in the brain and spinal cord, does not occur in the cerebral cortex and therefore need not be considered in detail. This inhibition takes place at synapses formed between two axon terminals and is termed *presynaptic*.

Initiation of action potentials in the axon hillock

Although the somatic or dendritic membranes are depolarized, the membrane of the axon hillock possesses no synapses and therefore has a membrane potential which is initially at the resting level. The difference in potential between the various parts of the neurone causes current to flow outwards through the membrane of the axon hillock. This membrane has a certain resistance which is inversely related to the ease with which ions cross it (that is, to the membrane permeability). The effect of an outward flow of current across this membrane resistance is to reduce the transmembrane voltage, that is, to depolarize the axon hillock. This depolarization induces an increase in the sodium permeability of the membrane. The increased sodium permeability in turn causes further depolarization, thereby making the system regenerative. If the depolarization is sufficiently large, the sodium permeability increases until the inside of the cell becomes positive with respect to the outside. This reversal of membrane polarity constitutes the action potential (*Figure 1.3*); it lasts only a millisecond or so since the permeability of the membrane alters yet again, the sodium permeability declining and the potassium permeability increasing.

During the action potential a small change in the ionic composition of the neurone takes place. While the membrane sodium permeability is high, Na^+ will enter the cell under the influence of the $[Na^+]$ gradient and the internal negativity. Correspondingly, during the ensuing phase of high potassium permeability, K^+ will leave the cell down its concentration gradient. The cell now pumps out the Na^+ in

exchange for K^+; energy, derived from the hydrolysis of ATP (adenosine triphosphate), must be expended since work is done against the $[Na^+]$ and $[K^+]$ gradients.

Once the action potential has been initiated in the axon hillock it flows backwards into the soma and, at the same time, starts to travel along the axon. In some cortical cells the action potential may be set up in the soma directly rather than in the axon hillock. The centrifugal conduction of the action potential in the axon depends on the passive outward flow of current across resting axonal membrane which takes place as the action potential approaches. The outward current depolarizes the membrane and induces the regenerative sodium permeability mechanism described above. If the axon is myelinated the flow of current through the membrane is restricted to the nodes of Ranvier; the action potential travels more rapidly than in a non-myelinated axon. In the largest axons of the dorsal spinocerebellar tract impulse velocities of 160 m/second have been recorded (Grundfest and Campbell, 1942); the maximal values in the other myelinated tracts are considerably lower than this, for example, 70 m/second for pyramidal axons in the cat spinal cord (Lance, 1954). If, as in many axons in the CNS, there is no myelin sheath, the conduction velocities are unlikely to exceed 1–2 m/second. Finally it is possible that, as in the retina, some cells with short axons transmit activity to their axon terminals by a passive flow of electrotonic current than rather than by setting up impulses.

Synaptic transmitter substances

Certain substances have now been provisionally identified as transmitters at synapses in the CNS. The evidence has come from observations on the distribution within the brain and spinal cord of either the suspected transmitter itself or of its synthesizing and hydrolyzing enzymes. In addition, the activities of single nerve cells have been studied following the application of the transmitter through a micropipette. Another approach has been to look for changes in spontaneous or evoked neuronal discharges after pharmacological blockade with the antagonist of the suspected transmitter. Lastly, in some situations it has been possible to collect the substance released from stimulated neurones. The following compounds are currently regarded as likely transmitters.

Excitatory transmitters

Acetylcholine (ACh). This substance has been conclusively identified as the transmitter released at synapses between motoneurones and Renshaw cells in the ventral horn of the spinal cord. It is probably involved in transmission through certain 'specific' thalamic nuclei (for example, VPM, VPL, VL, LG in *Figure 1.14*) and in the caudate nucleus, cerebellar cortex and hippocampus. In the cortex ACh excites cells in layer V, particularly in the sensory areas and motor cortex. ACh is also released during EEG 'activation' induced by stimulation of the reticular formation (Kanai and Szerb, 1965).

L-glutamic and L-aspartic acid. These amino acids are distributed widely in the brain and spinal cord; they excite cells by a depolarizing action.

Inhibitory transmitters

There is good evidence that *γ-aminobutyric acid* (GABA) is an inhibitory transmitter in the nervous systems of invertebrates and it now appears that it has a similar role in the mammalian brain and spinal cord. The substance occurs in high concentrations throughout the central nervous system and, when applied iontophoretically, depresses neural activity rapidly and reversibly. Recent intracellular recordings from cortical cells have shown that this inhibition is associated with hyperpolarization of the membrane, as in naturally occurring IPSPs. In the spinal cord and brainstem evidence is accumulating that *glycine* acts as an inhibitory transmitter substance. Acetylcholine may act as an inhibitory transmitter in some parts of the nervous system, as for example, in the superficial layers of the cerebral cortex.

Other transmitters

Three monoamines are secreted by cells in the medullary reticular formation and are also thought to act as transmitters; these are *dopamine, noradrenaline* and *5-hydroxytryptamine (Figure 1.4)*. While dopamine has mainly inhibitory actions the other two substances have both excitatory and inhibitory effects on nerve cells. Among other possible locations, dopamine has been found in the substantia nigra, noradrenaline in the medullary reticular formation and 5-hydroxytryptamine in the raphe nuclei. Other possible neurotransmitters include *histamine, ATP* and *substance P*. To this list should now be added the recently discovered class of compounds known as *endorphins*.

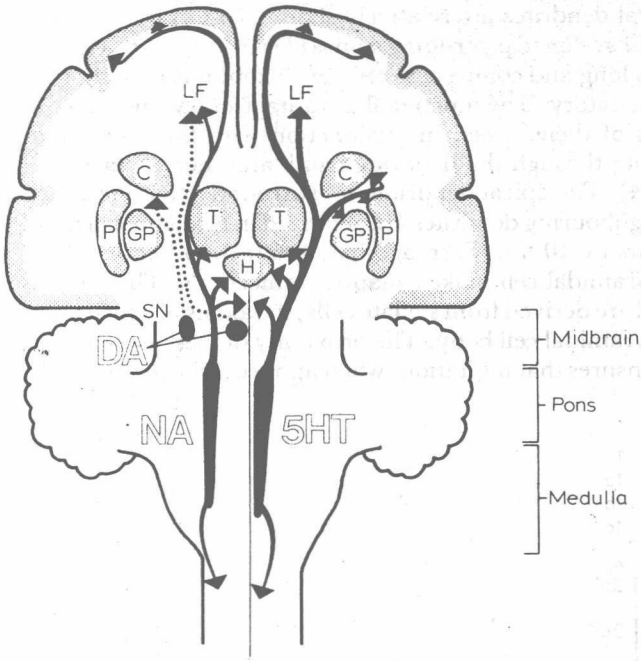

***Figure 1.4**—Coronal section through the brain to show the main projections of the monoamine-releasing neurones in the brainstem; dopaminergic* (DA) *and noradrenergic* (NA) *systems shown at left and 5-hydroxytryptamine* (5HT) *cells at right.* C, *caudate nucleus;* GP, *globus pallidus;* H, *hypothalamus;* LF, *limbic forebrain;* P, *putamen;* SN, *substantia nigra;* T, *thalamus.*
(Based on findings of Andén *et al.*, 1966).

D.C. potentials

Under normal circumstances there is a steady (or d.c.) potential across the thickness of the cerebral cortex such that the surface is some 5–20 mV positive with respect to electrically indifferent areas (Bures, 1957). It is probable that this potential results mainly from a steady potential difference between the apical dendrites and somas of the pyramidal cells. Thus intracellular recordings from pyramidal cells, for example, during experimentally induced seizures, reveal 'slow changes' (that is, lasting several seconds) in transmembrane potential which are in phase with alterations in the transcortical d.c. potential (Sugaya, Goldring and O'Leary, 1964). It is possible that the glial cells also contribute to shifts in d.c. potential since they have been shown to undergo slow changes in membrane potential; these result from the extracellular accumulation of potassium ions released by discharging neurones (Orkand, Nicholls and Kuffler, 1966). Although individual glial cells are too small to act as effective dipoles, a series of glial cells orientated radially to the cortical surface might do so. Differing from the conditions in neurones, the areas of contact between glial cells are characterized by low electrical resistances which would allow appreciable current to flow from one cell to another. As well as in epilepsy, the d.c. cortical potential is reduced during anoxia, anaesthesia and the 'spreading depression' of Leão (1944). In this last condition, which results from any form of severe cortical damage, it has now been shown that there is a marked

depolarization of neurones as the depression advances over the cortex (Collewijn and van Harreveld, 1966).

Architecture of the cerebral cortex

In the cerebral cortex the cell bodies are either conical, star or spindle shaped; accordingly the cells are classified as pyramidal, stellate and spindle respectively.

Each *pyramidal cell* (*Figures 1.2* and *1.7*) possesses a long apical dendrite which usually reaches almost to the surface of the cortex; in comparison the lateral dendrites are relatively short. Most of the synaptic connections are made on to the trunk of the dendrites or on to protruding spines (*Figure 1.2, inset*). Each of the several hundred spines is approximately 2 μm long and connects to only one axon; it is thought that most, if not all, of the axospinous synapses are excitatory. The functional advantage of having spines is not clear; possibly the high internal resistances of their necks, in conjunction with the membrane capacitance, prolong the synaptic currents flowing through the dendritic trunk and thereby facilitate summation between asynchronous EPSPs (page 4). The apical dendrites are surrounded by fine axons which run alongside so as to form 'cartridges'; neighbouring dendrites are arranged in hexagonal arrays, each dendrite being separated from its neighbours by 10 μm (Szentagothai, 1969). In addition to the spines, the dendritic trunks and soma of each pyramidal cell make synaptic connections. The 50–100 synapses on each soma appear to be inhibitory and are derived from stellate cells, the axons of which form a pericellular 'basket' arrangement around the pyramidal cell body. The proximity of these synapses to the site of impulse generation in the axon hillock ensures that inhibition, when applied, will be extremely

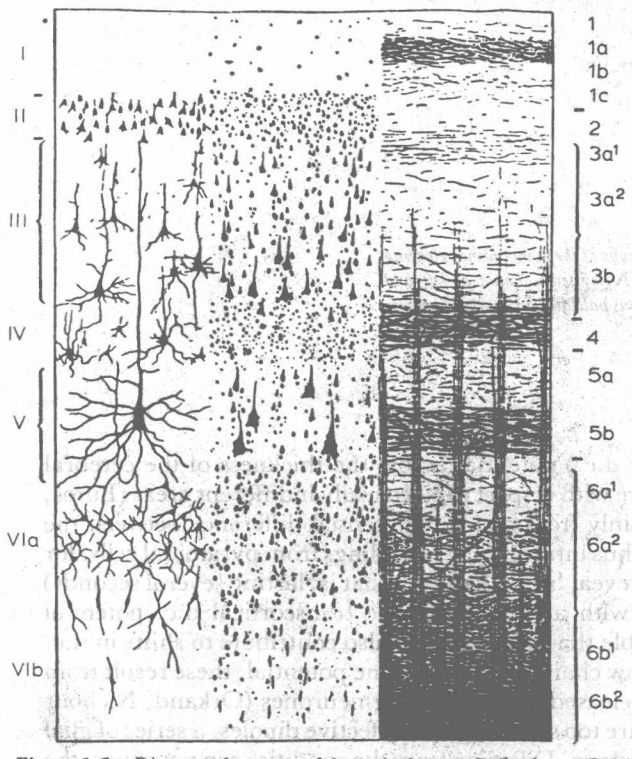

Figure 1.5—Diagram of structure of the cerebral cortex. Left: *from a Golgi preparation;* centre: *from a Nissl preparation;* right: *from a myelin sheath preparation.* I, *plexiform layer;* II, *external granular layer;* III, *pyramidal layer;* IV, *internal granular layer;* V, *ganglionic layer;* VI, *multiform layer* (From Brodal (1969) by courtesy of the author and Oxford University Press, after Brodmann and Vogt)