B.Sc. PSYCHOLOGY - I YEAR

DJP1B : BIOLOGICAL PSYCHOLOGY

SYLLABUS

UNIT I: INTRODUCTION

Basic reasoning in biology of behaviour – Historical background – Approaches that relate brain and behaviour - Levels of Analysis.

UNIT II: EVOLUTION AND DEVELOPMENT OF THE BRAIN

Evolution of Brain and Behaviour - Need to study other species - Lifespan Development of the Brain and Behaviour: Processes of Growth and Development of the Brain – stages of the development of the Nervous system – Glial cells – interaction of intrinsic and extrinsic factors influence of Experience on Brain Development - Maldevelopment of Brain – Changes in Brain due to aging – Time scales for describing Brain Development.

UNIT II: THE NERVOUS SYSTEM

Composition of the Nervous System – Divisions of the Nervous System – Functional descriptions of Brain Structures – Neurotransmitters - Blood supply to the Brain – Monitoring Neural activity – Species typical behaviour

UNIT IV: NEUROPHYSIOLOGY

Neurophysiology of arousal, attention, consciousness and sleep – Psychophysiology of emotions – Physiological and chemical correlates of learning and memory – Neurological bases of Aphasia

Unit – V : COGNITIVE NEUROSCIENCE

Language and Cognition: Development of Evolution of Speech and Language – Causes of Language disorders – organization of Language in the Brain-Functional Neuroimaging functions of Left and the Right Brain frontal Lobes and the higher order cognitive and emotional Funcitons – Deficits in Spatial perception and Brain injury – Recovery of Brain Functions.

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BIOLOGICAL PSYCHOLOGY

UNIT I: INTRODUCTION

Meaning of Biological Psychology

Biological psychology, in common with all branches of psychology, is enmeshed in a complex philosophical and scientific fabric. Biological psychologists study the "animal roots" of behavior, relating actions and experiences to genetics and physiology.

Biological psychology is the study of the physiological, evolutionary, and developmental mechanisms of behavior and experience. It is approximately synonymous with the terms biopsychology, psychobiology, physiological psychology, and behavioral neuroscience. The term *biological psychology* emphasizes that the goal is to relate biology to issues of psychology. *Neuroscience* includes much that is relevant to behavior but also includes more detail about anatomy and chemistry.

Biological psychology is not only a field of study. It is also a point of view. It holds that the proper way to understand behavior is in terms of how it evolved and how the functioning of the brain and other organs controls behavior. We think and act as we do because we have certain brain mechanisms, and we evolved those brain mechanisms because ancient animals with these mechanisms survived and reproduced better than animals with other mechanisms.

Approaches that relate brain and behaviour

Software/mind and hardware/brain distinction: One useful distinction in mind–brain relations is that between software and hardware.It has been tempting throughout the history of psychology to liken psychological processes to communication technology. Back in the 1940s the brain was likened to a telephone exchange. Today, the brain/mind is likened to a digital computer, which makes a sharp distinction between hardware and software. The brain may be seen as hardware (i.e., the processing units comparable to the central processing unit (CPU) of a computer), and the mind as software. Language is a good example. Following Noam Chomsky's seminal work in the 1950s, it is now assumed that we

are born with an innate *capacity* to learn a language (a universal grammatical structure which underlies all languages). However, different communities speak different languages; that is, they acquire different software (words, pronunciation, etc.) via exposure to language environments (i.e., learning).

Processes and output: Much of the activity of the brain is not accessible to the conscious mind. For example, when we utter a sentence we are aware of the final sentence – often only after we have spoken it – but not the processes that led to its construction. This example highlights something fundamental about the brain–mind; that is, brain processes do not correspond directly to the psychological contents that dominate conscious awareness. It is believed that we are largely unaware of the *process* of cognition, and aware only of its *outputs* expressed. in a 'high-level language' which differs from the more basic neural code used in the computation (processing) of the brain–mind. The distinction between high-level and low-level processes may be understood by reference to how digital computers use these different level processes.

Cause and effect

In common with other sciences, biological psychology focuses on the fundamental importance of separating *cause* and *effect*. This focus stands in contrast to some areas of psychology, and the social sciences in general, which are more often concerned with accounting for *associations* between different types of data. For example, much of social psychology is concerned with explaining observational data; and differential psychology is largely concerned with describing the covariation observed in personality and intelligence variables. Often there is no interest in causal relations – indeed, in some areas of psychology, there is a belief that psychological phenomena *cannot* be reduced to biological constructs and processes. In other areas of psychology (e.g., cognitive psychology), there is not a concern with the underlying biological reality, but rather with developing theories at a given level of explanation (e.g., computational theories). Biological psychology attempts to reduce data to the simplest level, formalized in cause–effect relations and couched in reductionist terms.

Phylogenetic continuity: Biological psychology makes use of observations and experiments on many different animals; in contrast, almost all other branches of

psychology study only one animal: *Homo sapiens*. This choice of species results from a number of factors. First, we may simply be interested in human beings (e.g., in social interaction research); or we might want to study a feature of this species (e.g., language). Second, although less clearly articulated, there is a widespread belief in psychology that research on non-human animals cannot yield useful information about our species. This belief is almost certainly false and is perhaps an intellectual leftover of *dualism* – i.e., the belief that the mind and body are separate. Coupled with this belief is the idea that human beings are *qualitatively* different from other species. Darwinian evolution and genetics place human beings on a *phylogenetic continuum* with other animals; they do not set us apart any more than they set a bird and a whale apart. In fact, what Darwinian theory does is to rob us of our species arrogance – in the grand scheme of the natural world, we are no better and no worse than any other species.

Social vs. biological perspectives: It is somewhat unfortunate that social and biological perspectives are often seen as separate – indeed, they are often seen as opposing, mutually incompatible approaches. However, biological research has much to say about issues that have until recently been thought of as essentially social in nature; and social research has important implications for the manner in which the brain and behaviour are related – there is now an emerging 'neuroscience of social behaviour'. Most of the important effects of the brain on behaviour depend upon environmental input; and this input is important for how the mind structures the social environment. For example, although there may be genetic influences on aggression, the types of stimuli that activate aggressive responses are environmental, and meaning and interpretation attached to these stimuli can be restructured (learned), thus altering actual behaviour. Therefore, brain-based aggression is only a *potential* (the necessary condition); the social environment, and our cognitive interpretation of it, is the *potentiator* (the sufficient condition). This line of reasoning has proved remarkably successful in cognitive behaviour therapy, which restructures the thinking of patients suffering from clinical conditions (e.g., depression). Often, the more we learn about genetics and neurophysiology, the more importance needs to attach to environmental and social factors

Branches of Biological Psychology of Viewpoints to explore Biology of Behavior

Specialization	Description
Research Fields	**Research positions ordinarily require a PhD. Researchers are employed by universities, hospitals, pharmaceutical firms, and research institutes.**
Neuroscientist	Studies the anatomy, biochemistry, or physiology of the nervous system. (This broad term includes any of the next five, as well as other specialties not listed.)
Behavioral neuroscientist (almost synonyms: psychobiologist, biopsychologist, or physiological psychologist).	Investigates how functioning of the brain and other organs influences behavior.
Cognitive neuroscientist	Uses brain research, such as scans of brain anatomy or activity, to analyze and explore people's knowledge, thinking, and problem solving.
Neuropsychologist	Conducts behavioral tests to determine the abilities and disabilities of people with various kinds of brain damage and changes in their condition over time. Most neuropsychologists have a mixture of psychological and medical training; they work in hospitals and clinics.
Psychophysiologist	Measures heart rate, breathing rate, brain waves, and other body processes and how they vary from one person to another or one situation to another.
Neurochemist	Investigates the chemical reactions in the brain.
Comparative psychologist (almost synonyms: ethologist, animal behaviorist)	Compares the behaviors of different species and tries to relate them to their habitats and ways of life.
Evolutionary psychologist (almost synonym: sociobiologist)	Relates behaviors, especially social behaviors, including those of humans, to the functions they have served and, therefore, the presumed selective pressures that caused them to evolve.
Practitioner Fields of Psychology	**In most cases, their work is not directly related to neuroscience. However, practitioners often need to understand it enough to communicate with a client's physician.**
Clinical psychologist	Requires PhD or PsyD. Employed by hospital, clinic, private practice, or college. Helps people with emotional problems.
Counseling psychologist	Requires PhD or PsyD. Employed by hospital, clinic, private practice, or college. Helps people make educational, vocational, and other decisions.
School psychologist	Requires master's degree or PhD. Most are employed by a school system. Identifies educational needs of schoolchildren, devises a plan to meet the needs, and then helps teachers implement it.
Medical Fields	**Practicing medicine requires an MD plus about 4 years of additional study and practice in a specialization. Physicians are employed by hospitals, clinics, medical schools and in private practice. Some conduct research in addition to seeing patients.**
Neurologist	Treats people with brain damage or diseases of the brain.
Neurosurgeon	Performs brain surgery.
Psychiatrist	Helps people with emotional distress or troublesome behaviors, sometimes using drugs or other medical procedures.
Allied Medical Field	**These fields ordinarily require a master's degree or more. Practitioners are employed by hospitals, clinics, private practice, and medical schools.**
Physical therapist	Provides exercise and other treatments to help people with muscle or nerve problems, pain, or anything else that impairs movement.
Occupational therapist	Helps people improve their ability to perform functions of daily life, for example, after a stroke.
Social worker	Helps people deal with personal and family problems. The activities of a clinical social worker overlap those of a clinical psychologist.

Levels of Analysis:

Biological explanations of behavior fall into four categories: physiological, ontogenetic, evolutionary, and functional.

A **physiological explanation** relates a behavior to the activity of the brain and other organs. It deals with the machinery of the body—for example, the chemical reactions that enable hormones to influence brain activity and the routes by which brain activity controls muscle contractions.

An **ontogenetic explanation** describes how a structure or behavior develops, including the influences of genes, nutrition, experiences, and their interactions. For example, the ability to inhibit impulses develops gradually from infancy through the teenage years, reflecting gradual maturation of the frontal parts of the brain. The term *ontogenetic* comes from Greek roots meaning the origin (or genesis) of being.

An **evolutionary explanation** reconstructs the evolutionary history of a structure or behavior. For example, frightened people get "goose bumps"—erections of the hairs, especially on their arms and shoulders. Goose bumps are useless to humans because our shoulder and arm hairs are so short. In most other mammals, however, hair erection makes a frightened animal look larger and more intimidating. An evolutionary explanation of human goose bumps is that the behavior evolved in our remote ancestors and we inherited the mechanism.

A **functional explanation** describes *why* a structure or behavior evolved as it did. Within a small, isolated population, a gene can spread by accident through a process called *genetic drift.* For example, a dominant male with many off -spring spreads all his genes, including neutral and harmful ones. However, a gene that is prevalent in a large population presumably provided some advantage—at least in the past, though not necessarily today. A functional explanation identifies that advantage. For example, many species have an appearance that matches their background. A functional explanation is that camouflaged

appearance makes the animal inconspicuous to predators. Some species use their behavior as part of the camouflage.

UNIT II: EVOLUTION AND DEVELOPMENT OF THE BRAIN

Evolution of Brain and Behaviour

Evolution is a change over generations in the frequencies of various genes in a population. Evolution includes *any* change in gene frequencies, regardless of whether it helps or harms the species in the long run. We distinguish two questions about evolution: How *did* some species evolve, and how *do* species evolve? To ask how a species did evolve is to ask what evolved from what, basing our answers on inferences from fossils and comparisons of living species. For example, biologists find that humans are more similar to chimpanzees than to other species, and they infer a common ancestor.

Biologists have constructed "evolutionary trees" that show the relationships among various species. As new evidence becomes available, biologists change their opinions of how closely any two species are related. The question of how species *do* evolve is a question of how the process works, and that process is, in its basic outlines, a logical necessity. That is, given what we know about reproduction, evolution *must* occur. The reasoning goes as follows:

- Off spring generally resemble their parents for genetic reasons.
- Mutations of genes occasionally introduce new heritable variations that help or harm an individual's chance of surviving and reproducing.
- Certain individuals successfully reproduce more than others do, thus passing on their genes to the next generation. Any gene that is consistently associated with reproductive success will become more prevalent in later generations. That is, the current generation of any species resembles the individuals that successfully reproduced in the past.

Because plant and animal breeders have long known this principle, they choose individuals with a desired trait and make them the parents of the next generation. This process is called **artificial selection,** and over many generations, breeders have produced exceptional racehorses, hundreds of kinds of dogs, chickens that lay huge numbers of eggs, and so forth. Charles Darwin's (1859) insight was that nature also selects. If certain individuals are more successful than others in finding food, escaping enemies, attracting mates, or protecting their off spring, then their genes will become more prevalent in later generations.

Evolutionary Psychology

Evolutionary psychology deals with how behaviors have evolved, especially social behaviors. The emphasis is on *evolutionary* and *functional* explanations, as defined earlier—that is, the presumed genes of our ancestors and why natural selection might have favored genes that promote certain behaviors. The assumption is that any behavior characteristic of a species must have arisen through natural selection and must have provided some advantage. Although exceptions to this assumption are possible, it is at least a helpful guide to research.

- Some animal species have better color vision than others, and some have better peripheral vision. Presumably, species evolve the kind of vision they need for their way of life.
- Mammals and birds devote more energy to maintaining body temperature than to all other activities combined. We would not have evolved such an expensive mechanism unless it gave us major advantages.
- Bears eat all the food they can find, and small birds eat only enough to satisfy their immediate needs. Eating habits presumably relate to different needs by different species.

On the other hand, some characteristics of a species have a more debatable relationship to natural selection. Consider two examples:

- People grow old and die, with an average survival time of about 70 to 80 years under favorable circumstances. Do we deteriorate because of genes that cause us to get out of the way and stop competing with our children and grandchildren?
- More men than women enjoy the prospect of casual sex with multiple partners. Theorists have related this tendency to the fact that a man can spread his genes by impregnating many women, whereas a woman cannot multiply her children by having more sexual partners. Are men and women prewired to have different sexual behaviors?

Development of the Brain

Understanding the structure and function of the brain is one of the important challenges facing science today. This scientific challenge has fundamental implications (a) for our understanding of the nature of the psychological processes that go to make up the *self*, as well as (b) for our understanding of a range of pathological conditions, for example neurodegenerative diseases (e.g., Parkinson's disease, dementia and stroke). Much of the excitement in neuroscience is driven by the desire to develop effective interventions to prolong and enrich human life. For example, stem-cell research promises to provide a means by which damaged nerves in the brain can regenerate.

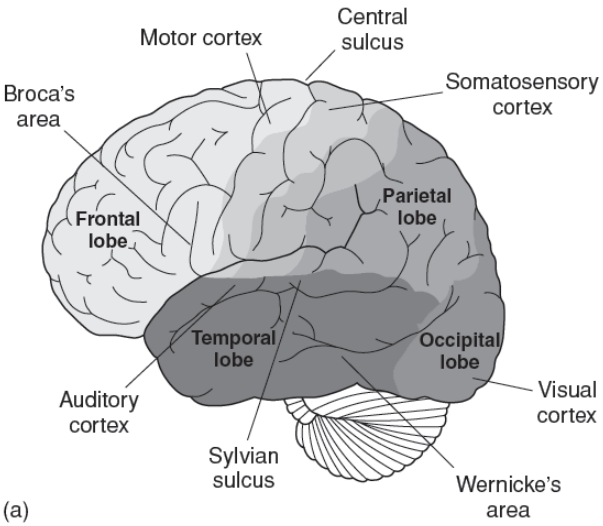
It is known that the brain has a well-defined anatomical structure, arranged as a hierarchy of different neural circuits and centres that are responsible for specific psychological functions. It is also known that the brain is in constant communication with the rest of the body, via the spinal cord (together comprising the *central nervous systems*, CNS); and that there are neural pathways distributed throughout the body that carry out the orders of the CNS, as well as being responsible for vital functions such as respiration and digestion (i.e., the *peripheral nervous system*, PNS). It is also known that the brain can send instructions to other organs in the body by the release of chemical messengers into the bloodstream called hormones. Lastly, it is known that there are specialized organs for receiving information from the environment as well as specialized organs for executing the demands of the CNS. These systems work in a similar way to an orchestra, with the brain being the overall conductor and the peripheral systems being the dedicated musicians:

when they work harmoniously together, the overall effect is effortless, smooth coordination; when they fail, disease results.

Quite apart from the marvellous psychological achievements of the nervous system, the neurophysiology of this system has its own charm. This is nowhere more apparent than in the basic processing unit in the nervous system: the nerve cell, called the *neuron* (or *neurone*). It is the interplay of billions of these neural processes that allows us to think, feel, love, hate, hope and despair.

Brain

The brain is where higher psychological functions are found. There are several different ways of looking at the brain. The human brain weighs between 1,300 and 1,400 grams (approx. 3 lbs). This size compares with a rhesus monkey brain of 420 g, a cat brain of 30 g and a rat brain of 2 g; however, the brain of the elephant is more than four times bigger (6,000 g). Clearly size is not everything! Although the human brain is only 2 per cent of total body weight, it consumes 20 per cent of available oxygen and energy in the blood – this is 10 times as much for its size as any other organ. The brain needs a regular supply of energy as it has no storage capacity and must run continuously all day and all year around.



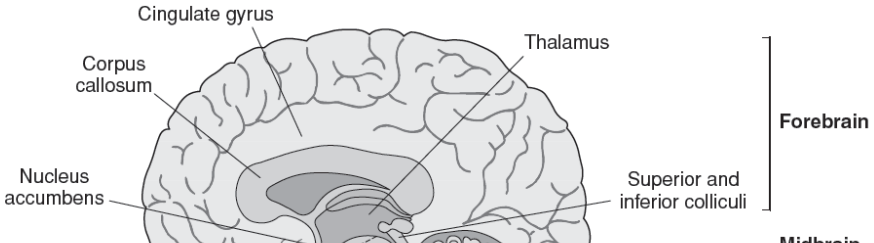


Figure: The major regions of the brain are shown in (a), along with some of the important structures within these regions corresponding to hindbrain, midbrain and forebrain (b).

From a horizontal plane, the brain is bilaterally symmetrical (the two halves of the brain look much the same); from this view the *cerebrum* dominates the appearance of the brain. The cerebrum consists of clefts (called *sulci*; singular: *sulcus*; also known as *fissures*); and the ridges formed along the sulci are *gyri* (singular: *gyrus*). These two halves of the brain are connected together by the *corpus callosum* (as well as by other structures).

In the centre of the brain are the *ventricles*; these spaces are filled with cerebrospinal fluid (CSF).

The brain may be divided into three sections: (a) the hindbrain, (b) the midbrain, and (c) the forebrain. These divisions are characterized by (a) their evolutionary development (phylogeny), and (b) their functions.

Hindbrain (rhombencephalon)

The hindbrain is essentially a continuation of the spinal cord. There are a number of important structures here involved in basic physiological functions. (a) The *medulla oblongata* is that part of the brain stem closest to the spinal cord; it controls such vital functions as the heartbeat, circulation and respiration and acts as a relay station for afferent and efferent impulses. Its destruction results in immediate death. (b) The *pons* acts as a relay station carrying impulses from various parts of the cerebral cortex to the cerebellum; it is also involved in the reflexes controlling breathing. (c) The *reticular*

formation runs through the middle of the hindbrain, and enters the midbrain; it receives sensory impulses (concerning sound) from higher brain centres and then passes these back up to the thalamus (this formation is vital for arousal and sleep). (d) The *locus ceruleus* contains relatively few neurons (approximately 30,000), but it sends out fibres extensively to other parts of the brain (this system is involved in states of vigilance). (e) The *cerebellum* consists of two deeply convoluted and separate hemispheres that control bodily balance and muscular coordination, including smooth eye movements. It also seems involved in the integration of movements (e.g., speech). This structure contains some 30 billion neurons that integrate information from the muscles, joints and tendons, leading to skilled automatic movements.

Midbrain (mesencephalon)

Above the hindbrain is the midbrain, which is a relatively small section of the brain. It contains neural centres that control motor reactions and auditory and visual functions. (a) The roof of the midbrain is the tectum, and the structures on either side are the *superior colliculus* and *inferior colliculus.* Under the tectum is (b) the *tegmentum*, with parts of the reticular formation and the nuclei of some of the cranial nerves. (c) The *substantia nigra* is involved in smooth bodily movements; damage to this structure leads to Parkinson's disease. (d) The *ventral tegmental area* (VTA) is important in incentive motivation (i.e., motivation to an appetitive stimulus, e.g., food, sex).

Forebrain (prosencephalon)

The forebrain is the highest level of organization, containing those structures responsible for higher-order psychological functions: (a) *thalamus*, (b) *hypothalamus*, (c) *pituitary gland*, (d) *basal ganglia*, and (e) *limbic* system, and (f) two *cerebral hemispheres* (also called the *telencephalon*).

The thalamus and hypothalamus form the *diencephalon.* The *thalamus* is a collection of various centres (nuclei) that serve as reception centres for the cerebral hemispheres. Neurons from the eyes, ears, skin and motor centres pass to the thalamus; after processing,

it then sends information to the cerebral hemispheres. One of these nuclei is the *lateral geniculate nucleus*, which receives visual signals from the eye, via the optic nerve.

The *hypothalamus* is composed of a number of distinct nuclei. It is situated below the anterior thalamus (*hypo* = 'below'). It has widespread connections with the rest of the forebrain and midbrain. It is involved in basic physiological processes, for example, eating, drinking, maintenance of temperature and sexual activity. It also exerts some of its effects via the neuroendocrine system: it stimulates the release of hormones from the *pituitary gland* (the source of eight hormones). One area of the hypothalamus, the *suprachiasmatic nuclei*, is responsible for the biological clock, working on a 24-hour cycle.

The *basal ganglia* comprise a group of subcortical structures (the *caudate nucleus*, the *putamen* and the *globus pallidus*). The basal ganglia have many subdivisions, each of which exchanges information with a different part of the cortex.

The *limbic system*, which comprises a collection of structures located near the centre of the cerebral hemispheres, is involved in emotional and motivational processes, as well as learning and memory. This system comprises the *hippocampus*, which is involved in memory, goal processes and emotion; the *fornix* (which is a major axon tract linking the two lobes of the cortex); and the *amygdala* (Latin: 'almond'), involved in the generation of emotional arousal, especially but not exclusively fear.

The two *cerebral hemispheres* (also called *lobes*) are both divided into four major divisions: (a) *frontal*, (b) *parietal*, (c) *occipital* and (d) *temporal*. The thin outer layer of the brain is the *cerebral cortex* (in Latin, *cortex* means bark, as the bark on a tree).

Cerebral cortex

The *cerebral cortex* is approximately 3 mm thick (if it were stretched out), and is composed of a densely packed network of nerve fibres capable of highly complex interconnections. Although this structure is thin, it comprises a large proportion of the entire human brain because it is deeply folded and convoluted (like screwing up a large sheet of paper to fit it into a smaller space). This newest part of the brain is either absent or underdeveloped in lower animals, but quite well developed in primates. It is thought that this structure is responsible for our cognitive processing, allowing planning, mental simulations, imagination and intelligence.

The cerebral cortex is composed of two separate hemispheres, each of which has *relatively* specialized sensory and motor functions relating (mostly) to the contralateral (opposite) side of the body. At the cerebral cortex is seen a layer of grey matter (cell bodies), and their axons extend inward from the cortex – this forms the white matter of the cerebral hemispheres. The two hemispheres communicate via the *corpus callosum* and the smaller *anterior commissure*.

Cerebral lobes

Let us take a closer look at the four cerebral lobes. The convolutions observed here increase the amount of cortex that can be fitted into a limited space – it is thus an adaptive solution to the problem of increasing processing capacity without first waiting for the total size of the skull to increase. The large furrows seen are known as *fissures* – these divide the lobes. (The central fissure and the lateral fissures serve as useful landmarks to demarcate the lobes. The smaller furrows are known as *sulci*, and the ridges between fissures and sulci are called *gyri.*) The midline fissure that separates the two hemispheres is called the *longitudinal fissure*.

Most (approximately 90 per cent) of the cerebral cortex is new (i.e., it is *neocortex*), that is, of relatively recent evolution. In these layers there are two very different types of neurons: (a) *pyramidal* (cell bodies are pyramid-shaped) neurons have a large dendritic tress, which extends from the top of the pyramid to the cortex surface, and a long axon; and (b) *stellate* (star-shaped) neurons, which are interneurons, with short axons. The six layers of the cortex contain different combinations of these two types of neuron. These columns of neurons form circuits that perform a specific function.

The *frontal lobe* comprises a large part of the cerebral cortex, and is involved in higherorder cognitive processes (e.g., planning and inhibition of prepotent responses). This lobe extends from the central sulcus to the anterior surface of the brain; it contains the motor cortex, in the precentral gyrus, which is responsible for coordinating motor movements, and the prefrontal cortex, which is responsible for the coordination of information from all sensory systems – it does not itself receive sensory input directly.

The *parietal lobe* is found between the central sulcus and the occipital lobe. This area is charged with somatosensory processing (e.g., touch). The *postcentral gyrus* contains

the primary somatosensory cortex, where skin sensation is processed. There are four bands of cells running parallel to the central sulcus, and each band relates to different parts of the body. This region processes different representations of the sensory states of different parts of the body.

The *occipital lobe* is located at the back of the head (i.e., the posterior). It receives fibres from the thalamus that convey visual information. The *primary visual cortex* (or *striate cortex*) is found at the most posterior part of this lobe, and damage to this region causes cortical blindness. The *temporal lobe* is found near the temples; it is the primary area for the processing of auditory information (superior temporal gyrus), and it is vital for the comprehension and production of language. It also subserves other functions, including complex visual processing (e.g., face recognition). Temporal lobe epilepsy is sometimes associated with auditory and visual hallucinations.

Projection areas

The cerebral lobes contain a number of structures that are responsible for receiving sensory information; they also serve the function of dispatch centres for motor commands: (a) *sensory projection areas* receive information from the various senses; and (b) *motor projection areas* send commands that ultimately get executed at muscles. Sensory and motor areas of the body are mapped (i.e., projected) onto specific areas of the cortex. The *somatosensory cortex* is found in the parietal lobe next to the central fissure, the *motor cortex* in the frontal lobe next to the central fissure. Similar projection areas are found for vision and hearing, located in the occipital and temporal lobes, respectively.

The projection areas of the brain comprise about one-quarter of the total brain volume; the remaining areas of the cerebral cortex are *association areas*. These areas are involved in higher complex functions such as planning, thinking and speaking. The frontal lobes are especially important in this regard. They are involved in planning and complex cognitive processes. Damage to the frontal lobes impairs the ability to plan ahead, leading to behavioural impulsiveness.

Ventricles

The brain is bathed in fluid – cerebrospinal fluid. This fluid protects the brain from shock and it also serves to suspend the brain so the pressure on the base of the brain is lessened.

The brain contains a number of empty spaces – interconnected chambers called *ventricles*. The anatomy of these ventricles need not concern us. Cerebrospinal fluid is extracted from the blood, under continuous production, and the half-life is a mere 3 hours. *Hydrocephalus* ('water on the brain') may result from an obstruction in the flow of CSF; this build-up in pressure can lead to damage to surrounding tissue.

Neurodevelopment

The formation of the body, including the nervous systems, is the result of *maturation*: given an adequate environment, these structures and processes develop according to the genome – of course, the environment is important, but there would be no environmental effects without this blue-print.

After only a few hours the fertilized ovum (egg) starts dividing: it has now started its journey of development, which will take many years to complete. The resulting divisions upon divisions (exponential growth) produce the immense complexity in the structure and function of the mature nervous system. At two weeks of gestation the first signs of the development of the nervous system are evident: the embryo starts to thicken, finally forming the *neural tube* around day 23. This neural tube is made up of stem cells (these undifferentiated cells have the potential to make any tissue in the body); in the case of the nervous system, these all-purpose cells make neurons and supporting (glial) cells. The length of the neural tube forms the rostal–causal axis of the spinal cord and brain. The inside of the tube will eventually become the fluid-filled ventricles in the brain, and the stem cells on the inner surface of the tube will eventually comprise the different regions of the brain and spinal cord. At this time three distinct regions of swelling in the neural tube can be seen – these are the three interconnected chambers: these chambers are destined to become the hindbrain, midbrain and forebrain. Later in the developmental process the distinctive gyri and sulci that segregate brain areas may be seen.

It is still largely a mystery how cell stems develop into specific neural processes and areas; however, there is some understanding of how this remarkable feat is achieved. By the time of birth the brain weighs around 350 grams; but more growth is seen in the first

year with brain weight increasing to 1,000 grams – not far off the weight of the adult brain (approx. 1,400 grams).

Neural development is not just about growth but also about loss and organization: at the age of three years, synaptic density is at its highest; but from this age synaptic links are pruned to form effective systems of neurotransmission. A number of processes are entailed in the maturation of the adult nervous system.

1. *Neurogenesis*, or *proliferation.* This process consists of cell division among the stem cells in the neural tube; this produces neurons and glial cells – unlike glial cells, neurons are thought not to divide and reproduce themselves (there are some exceptions to this rule, and now neurons can be encouraged to grow from undifferentiated stem cells). However, compared with glial cells, diseased neurons are very difficult to replace – as with lost innocence, once gone neurons are difficult to replace.

2. *Migration*. Neurons must move from their place of origin in the neural tube to their target location in the nervous systems. This is a major problem for the developing nervous system, especially given the long distances nerve fibres (axons) have to travel. For example, axons need to travel from the cerebral cortex to the spinal cord, and from the spinal cord to muscles all over the body. One of the major scientific prob lems facing developmental embryologists is to work out the process underlying cell migration: how do cells 'know' where to go to form synaptic connections with neurons that are often some distance away? It seems that some supporting cells (specifically *radial glial cells*, a type of glial cell) act as guide-wires for migrating ells (specifically the axons that travel from the cell nucleus to the next cell). In addition, cells may chemically attract other cells – acting as a chemical allure for their co-existence.

3. *Differentiation*. In the beginning all cells look alike, but neurons need to be differentiated to take on their distinctive characteristics; the result is different types of neurons in different locations in the brain (i.e., there are a variety of types of axons, dendrites, terminal buttons, etc.). Then some axons myelineate, adding a fatty coat around the axon fibre (myelin is important for neural conduction). The major source of influence here is gene expression: genes must know when to turn on and off their effects, otherwise chaos would ensue – chaos does sometimes ensue, resulting in brain abnormalities.

4. *Synaptogenesis*. Neurons are functional to the extent that they communicate with other neurons, and the way this is achieved is to form synaptic connections. The formation of synapses is, of course, fundamental to effective neurotransmission.

5. *Cell death/murder*. There is an excess of neuronal material in the developing brain, and useless neurons (i.e., neurons that are not functionally effective in forming meaningful connections with other neurons) either die away or are actively killed. Many neurons do die; weak neurons go to the wall; fitter neurons survive and make effective connections. *Neurotrophic factors* exist which are secreted by target cells (i.e., postsynaptic cells) and taken up by travelling cells, which allow the cell to prosper. It is as if the target cell is talking to the presynaptic cell, in the form of a chemical message, which says, 'I'll be your friend. Don't kill yourself '. A nerve growth factor has already been identified. Less successful cells are subject to *programmed cell death* – this suicide, known as *apoptosis*, results in the pruning of useless neurons in the form of housekeeping to maintain an effective nervous system. It is thought that specific genes activate a process of cell suicide.

There are other forms of neurotrophin in the brain (*trophin* is ancient Greek for nourishment); they serve important functions: they encourage axons to survive during early development rather than commit suicide (apoptosis); different neurotrophins affect different neurons, leading to differentiation of structure and function; in later development, neurotrophins are activated by a neuron or hormone, resulting in the axonal branching; and neurotrophins help to repair damage to connected neurons by promoting the re-establishment of functional neuronal links.

6. *Synaptic reorganization*. In reaction to the environment and experience, neural plasticity ensures that effective connections are made between neurons that serve functional roles. The extent to which wiring together is coded within the genome or is the result of exposure to regularities in the environment (learning) is still unclear: it is likely that genetic disposition and experience are *both* necessary for effective organization (the neuronal organization of the visual cortex is known to be dependent upon sensory input – people who have gained sight after blindness from birth find the visual environment very strange and discomfiting, even to the extent that some would have preferred to remain blind).

All too often assaults and injury to the developing brain lead to permanent and irreversible pattern abnormalities. Given the enormous complexity of brain development, entailing the interplay of genetic, chemical and environmental factors, it is truly aweinspiring to realize that most of us develop a reasonably normal brain. However, noteveryone is so lucky.

Most of us assault our brains with a variety of everyday chemicals (e.g., alcohol, nicotine, caffeine); and some of us even play a neural form of Russian roulette by using potent psychoactive drugs (e.g., cocaine). In all such cases we may develop an addiction, and even develop severe brain conditions (e.g., Korsakoff 's syndrome in the case of alcoholism); in the latter case, we may trigger off a genetic disposition to psychosis or neurotoxicity, resulting in depression and perhaps even dementia (this is especially suspected in the case of Ecstasy, which is known to be neurotoxic). However, in the main, our brains are remarkably robust to our everyday chemical assaults.

A particularly marked neurodevelopmental abnormality is *fetal alcohol syndrome*. This condition is caused by excessive alcohol intake in the pregnant mother. The condition is characterized by decreased alertness, hyperactivity, mental retardation, motor problems, and various physical abnormalities, such as a defective heart and irregularities in facial features. In these children, the dendrites that form connections with other cells tend to be short and few in number. Other children may be similarly affected, but may not show irregularities in facial features. How much alcohol is safe during pregnancy? The answer depends on the stage of neural development: it may be the case that *any* intake of alcohol is potentially dangerous. There does seem to be a correlation between amount of alcohol intake during pregnancy and behavioural problems (e.g., hyperactivity and impaired school performance) – a finding that should encourage all pregnant women to avoid alcohol for the duration of pregnancy.

Glia

Glia (or neuroglia), the other major components of the nervous system, do not transmit information over long distances as neurons do, although they do exchange chemicals with adjacent neurons. In some cases, that exchange causes neurons to oscillate in their activity. The term *glia,* derived from a Greek word meaning "glue," reflects early investigators' idea

that glia were like glue that held the neurons together. Although that concept is obsolete, the term remains. Glia are smaller but also more numerous than neurons. Overall, they occupy about the same volume.

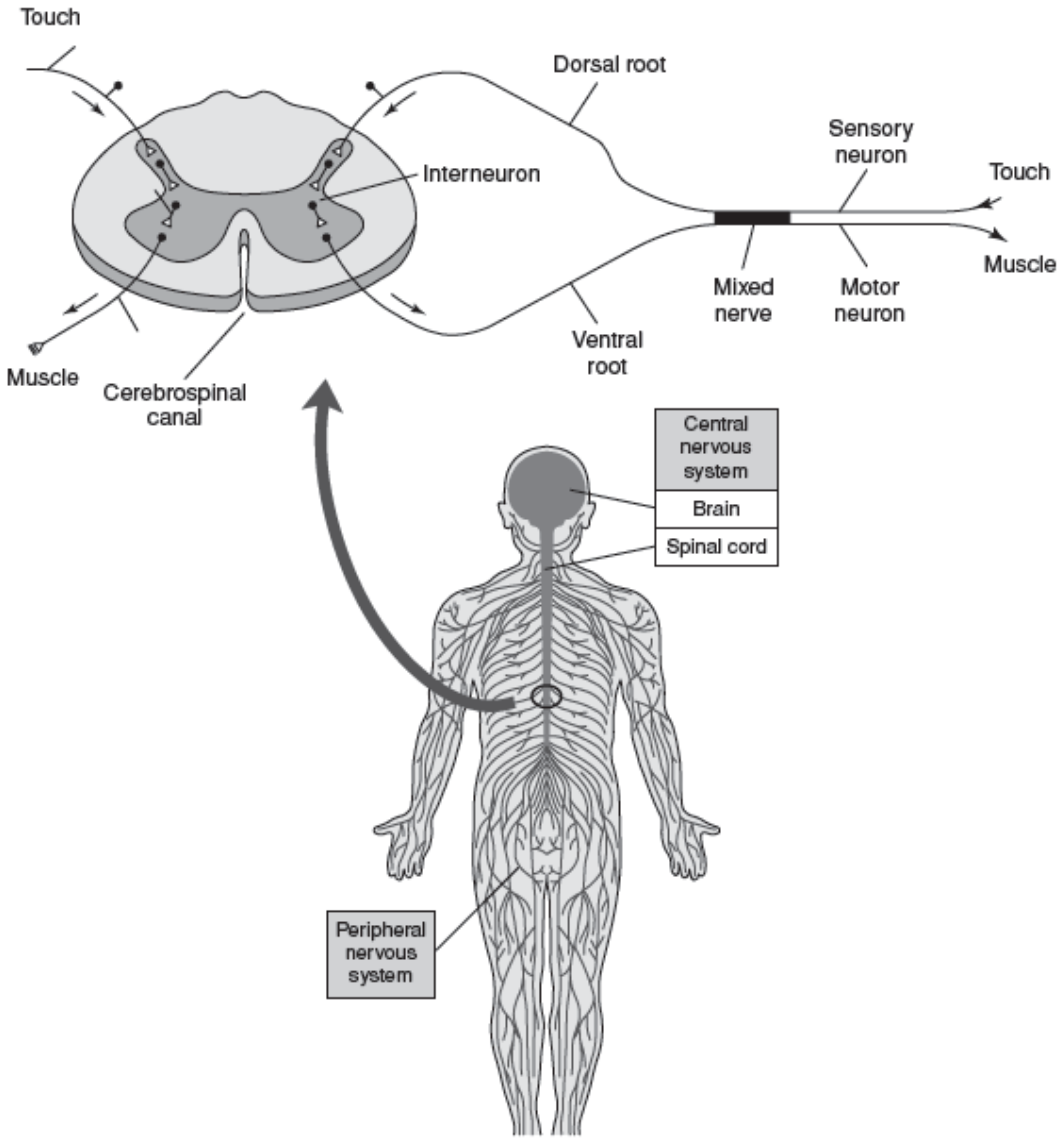
The brain has several types of glia with different functions. The star-shaped **astrocytes** wrap around the presynaptic terminals of a group of functionally related axons, by taking up chemicals released by axons and then releasing them back to axons, an astrocyte helps synchronize the activity of the axons, enabling them to send messages in waves. Astrocytes also remove waste material created when neurons die and control the amount of blood flow to each brain area. An additional function is that during periods of heightened activity in some brain area, astrocytes dilate the blood vessels to bring more nutrients into that area. Furthermore, astrocytes release chemicals that modify the activity of neighboring neurons. Clearly, astrocytes do more than just support neurons. They are an important contributor to information processing.

Microglia, very small cells, also remove waste material as well as viruses, fungi, and other microorganisms. In effect, they function like part of the immune system. **Oligodendrocytes** (OL-i-go-DEN-druh-sites) in the brain and spinal cord and **Schwann cells** in the periphery are specialized types of glia that build the myelin sheaths that surround and insulate certain vertebrate axons. **Radial glia** guide the migration of neurons and their axons and dendrites during embryonic development. When embryological development finishes, most radial glia differentiate into neurons, and a smaller number differentiate into astrocytes and oligodendrocytes.

UNIT III: THE NERVOUS SYSTEM

The nervous system is composed of two major parts: (a) the *central nervous system* (CNS; the brain and spinal cord), which is contained in bone (skull and spinal column); and (b) the *peripheral nervous system* (PNS; all nervous structures outside the CNS), found outside these bony structures. Figure below shows the whole nervous system, and its division into the CNS and the PNS. It is customary to speak of *afferent* and *efferent* nerves and systems. The bundles of nerve fibres that conduct excitation to the CNS are known as

afferent nerves (from the Latin, *affere*, 'to bring to'); the bundles of fibres that travel to the effector systems are called *efferent nerves* (from the Latin, *effere*, 'to bring forth').



Types of receptor neurons

Receptor cells are sensitive to external energy (e.g., pressure, light, sound); and *transduce* physical energy into electrical patterns of activity that are recognized by other neurons in the nervous system. The cell bodies of some receptors (e.g., tactile receptors) are found next to the spinal cord, located in clusters called *ganglia*. In some systems (e.g., skin pressure), the same cell undertakes the job of both *transduction* and *transmission*. However, more often transduction and transmission are jobs carried out by different cells.

Interneurons (also called *association neurons*) are intermediate neurons, in the brain or spinal cord, between incoming (*afferent*) and outgoing (*efferent*) neurons that organize and control afferent nerve impulses to create efferent effects (some sensorimotor systems are very simple and do not contain interneurons). *Motor neurons* receive efferent impulses from other neurons (often *interneurons*) and activate the skeletal musculature and glands. Upon firing, a motor neuron releases a chemical substance (*neurotransmitter*), which causes the muscle fibres to contract.

It is estimated that there are some 100 billion neurons in the CNS, each with as many as 1,000 to 10,000 connections with other neurons. The dense interconnections of these neurons are truly astonishing. However, they compose only some 10 per cent of cells in the brain, the others being support cells of various types. Raise your hand and touch your face now: you have just activated the whole sequence of neuronal and chemical events in one effortless motion, and activated millions of neurons in the process. The complexity of these processes is not fully appreciated until they malfunction.

The largest structure in the brain is the cerebral cortex, which is contained within a thin layer only a few millimetres thick. In order for this expanded space to fit within the brain, the cortex folds in on itself, resulting in a series of ridges and groves that create the characteristic wrinkled appearance of the brain.

Anatomy of Neurons

The nervous system consists of two kinds of cells: *neurons* and *glia*. Neurons receive information and transmit it to other cells. Glia serve many other functions that are difficult to summarise. According to one estimate, the adult human brain contains approximately 100 billion neurons

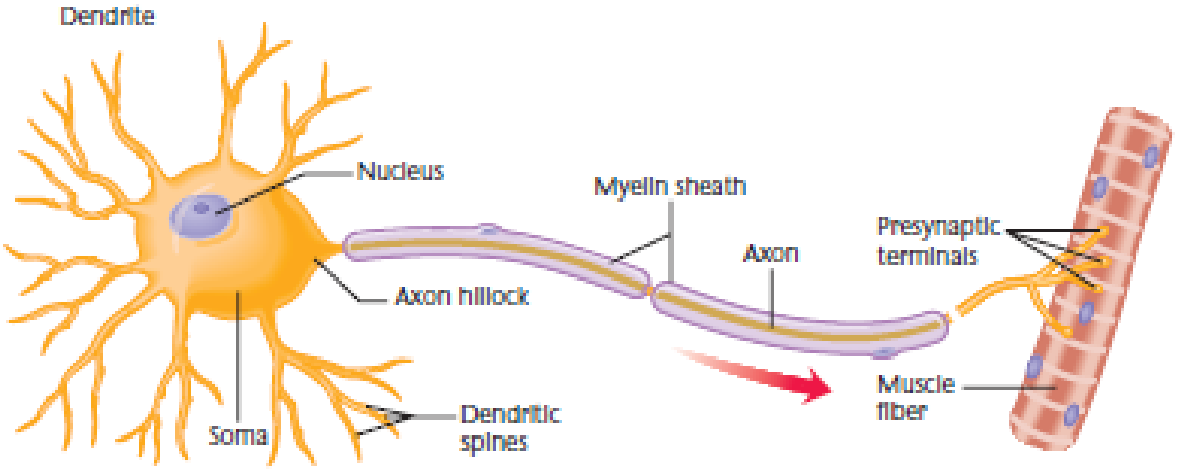
The Structure of a Neuron

Neurons are distinguished from other cells by their shape. The larger neurons have these components: dendrites, a soma (cell body), an axon, and presynaptic terminals. (The tiniest neurons lack axons, and some lack well-defined dendrites).

A **motor neuron** has its soma in the spinal cord. It receives excitation from other neurons through its dendrites and conducts impulses along its axon to a muscle. A **sensory**

neuron is specialized at one end to be highly sensitive to a particular type of stimulation, such as light, sound, or touch. The sensory neuron conducts touch information from the skin to the spinal cord. Tiny branches lead directly from the receptors into the axon, and the cell's soma is located on a little stalk off the main trunk.

Dendrites are branching fibers that get narrower near their ends. (The term *dendrite* comes from a Greek root word meaning "tree"; a dendrite is shaped like a tree.) The dendrite's surface is lined with specialized *synaptic receptors,* at which the dendrite receives information from other neurons. The greater the surface area of a dendrite, the more information it can receive. Some dendrites branch widely and therefore have a large surface area. Some also contain **dendritic spines,** the short outgrowths that increase the surface area available for synapses. The shape of dendrites varies enormously from one neuron to another and can even vary from one time to another for a given neuron. The shape of the dendrite has much to do with how the dendrite combines different kinds of input



The **cell body,** or **soma** (Greek for "body"; pl.: somata), contains the nucleus, ribosomes, mitochondria, and other structures found in most cells. Much of the metabolic work of the neuron occurs here. Cell bodies of neurons range in diameter from 0.005 mm to 0.1 mm in mammals and up to a full millimeter in certain invertebrates. Like the dendrites, the cell body is covered with synapses on its surface in many neurons.

The **axon** is a thin fiber of constant diameter, in most cases longer than the dendrites. (The term *axon* comes from a Greek word meaning "axis.") The axon is the information sender of the neuron, conveying an impulse toward other neurons or an organ or muscle. Many vertebrate axons are covered with an insulating material called a **myelin sheath** with interruptions known as **nodes of Ranvier** (RAHN-vee-ay). Invertebrate axons do not have myelin sheaths. An axon has many branches, each of which swells at its tip, forming a **presynaptic terminal,** also known as an *end bulb* or *bouton* (French for "button"). This is the point from which the axon releases chemicals that cross through the junction between one neuron and the next.

A neuron can have any number of dendrites, but no more than one axon, which may have branches. Axons can range to a meter or more in length, as in the case of axons from your spinal cord to your feet. In most cases, branches of the axon depart from its trunk far from the cell body, near the terminals.

If a cell's dendrites and axon are entirely contained within a single structure, the cell is an **interneuron** or **intrinsic neuron** of that structure. For example, an intrinsic neuron of the thalamus has its axon and all its dendrites within the thalamus.

The Blood-Brain Barrier

Although the brain, like any other organ, needs to receive nutrients from the blood, many chemicals cannot cross from the blood to the brain. The mechanism that keeps most chemicals out of the vertebrate brain is known as the **blood-brain barrier.**

Why We Need a Blood-Brain Barrier: When a virus invades a cell, mechanisms within the cell extrude virus particles through the membrane so that the immune system can find them. When the immune system cells identify a virus, they kill it and the cell that contains it. In effect, a cell exposing a virus through its membrane says, "Look, immune system, I'm infected with this virus. Kill me and save the others."

This plan works fi ne if the virus-infected cell is, say, a skin cell or a blood cell, which the body replaces easily. However, with few exceptions, the vertebrate brain does not replace damaged neurons. To minimize the risk of irreparable brain damage, the body

builds a wall along the sides of the brain's blood vessels. This wall keeps out most viruses, bacteria, and harmful chemicals, but also most nutrients.

"What happens if a virus does enter the nervous system?" Certain viruses, such as the rabies virus, evade the blood-brain barrier, infect the brain, and lead to death. For several other viruses that enter the nervous system, microglia and other mechanisms attack the viruses or slow their reproduction without killing the neurons they occupy. However, a virus that enters the nervous system probably remains with the person for life. For example, the virus responsible for chicken pox and shingles enters spinal cord cells. No matter how effectively the immune system attacks that virus outside the nervous system, virus particles remain in the spinal cord, from which they can emerge decades later. The same is true for the virus that causes genital herpes.

How the Blood-Brain Barrier Works: The blood-brain barrier depends on the arrangement of endothelial cells that form the walls of the capillaries. Outside the brain, such cells are separated by small gaps, but in the brain, they are joined so tightly that virtually nothing passes between them.

The brain has several mechanisms to allow certain chemicals to cross through the endothelial cells. First, *small uncharged molecules,* including oxygen and carbon dioxide, cross freely. Water crosses through special protein channels in the wall of the endothelial cells. Second, *molecules that dissolve in the fats of the membrane* also cross passively. Examples include vitamins A and D.

For certain other essential chemicals, the brain uses **active transport,** a protein-mediated process that expends energy to pump chemicals from the blood into the brain. Chemicals that are actively transported into the brain include glucose (the brain's main fuel), amino acids (the building blocks of proteins), purines, choline, a few vitamins, iron, and certain hormones.

The blood-brain barrier is essential to health. In people with Alzheimer's disease or similar conditions, the endothelial cells lining the brain's blood vessels shrink, and harmful chemicals enter the brain.

However, the barrier also poses a difficulty in medicine because it keeps out many medications. For example, brain cancers are difficult to treat because nearly all the drugs used for chemotherapy fail to cross the blood-brain barrier.

The All-or-None Law

Action potentials occur only in axons and cell bodies. When the voltage across an axon membrane reaches a certain level of depolarization (the threshold), voltage-gated sodium channels open wide to let sodium enter rapidly, and the incoming sodium depolarizes the membrane still further. Dendrites can depolarize also, but they don't have voltage-gated sodium channels, so opening the channels a little, letting in a little sodium, doesn't cause them to open even more and let in still more sodium. Thus, dendrites don't have action potentials. If the dendrites depolarize the cell enough, its axon produces an action potential.

For a given neuron, all action potentials are approximately equal in amplitude (intensity) and velocity under normal circumstances. This is the **all-or-none law:** The amplitude and velocity of an action potential are independent of the intensity of the stimulus that initiated it.

Although the amplitude, velocity, and shape of action potentials are consistent over time for a given axon, they vary from one neuron to another. The earliest studies dealt with squid axons because squid have very thick axons that are easy to study. More recent studies of mammalian axons have found much variation in the types of protein channels and therefore in the dimensions of the action potentials.

The all-or-none law puts constraints on how an axon can end a message. To signal the difference between a weak stimulus and a strong stimulus, the axon can't send bigger or faster action potentials. All it can change is the timing.

Neurotransmitters

At a synapse, one neuron releases chemicals that affect a second neuron. Those chemicals are known as **neurotransmitters.** Research has gradually identified a hundred or more chemicals believed or suspected to be neurotransmitters. Some major categories are:

- **amino acids** acids containing an amine group (NH2)

- **neuropeptides** chains of amino acids
- **acetylcholine** (a one-member "family") a chemical similar to an amino acid, except that the NH2 group has been replaced by an N(CH3)3 group
- **monoamines** neurotransmitters containing one amine group (NH2), formed by a metabolic change in certain amino acids
- **purines** a category of chemicals including adenosine and several of its derivatives
- **gases** nitric oxide and possibly others

All but a few of the neurotransmitters are amino acids, derivatives of amino acids, or chains of amino acids. The most surprising exception is **nitric oxide** (chemical formula NO), a gas released by many small local neurons. Nitric oxide is poisonous in large quantities and difficult to make in a laboratory. Yet, many neurons contain an enzyme that enables them to make it efficiently. One special function of nitric oxide relates to blood flow: When a brain area becomes highly active, blood flow to that area increases.

Hormones. A **hormone** is a chemical that is secreted, in most cases by a gland but also by other kinds of cells, and conveyed by the blood to other organs, whose activity it influences. A neurotransmitter is like a signal on a telephone line: It conveys a message directly and exclusively from the sender to the receiver. Hormones function more like a radio station: They convey a message to any receiver that happens to be tuned in to the right station. Figure below presents the major **endocrine** (hormone-producing) **glands.**

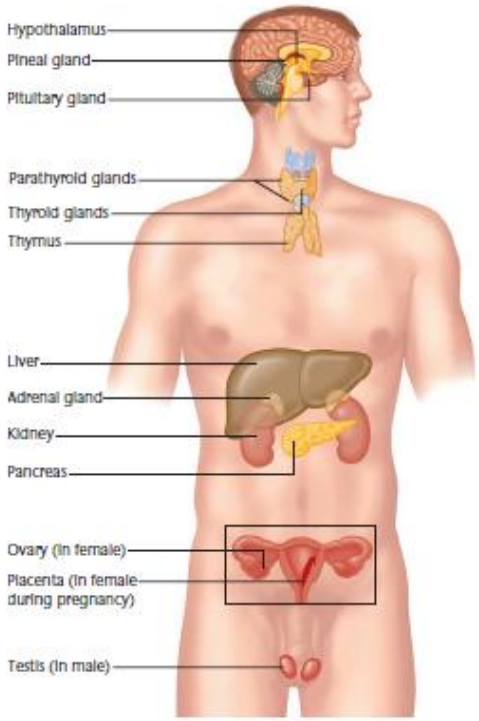


Table below lists some important hormones and their principal effects. Hormones are particularly useful for coordinating longlasting changes in multiple parts of the body. For example, birds that are preparing to migrate secrete hormones that change their eating and digestion to store extra energy for a long journey. Among the various types of hormones are **protein hormones** and **peptide hormones,** composed of chains of amino acids. (Proteins are longer chains and peptides are shorter). Protein and peptide hormones attach to membrane receptors, where they activate a second messenger within the cell—exactly the same process as at a metabotropic synapse. In fact, many chemicals—including epinephrine, norepinephrine, insulin, and oxytocin—serve as both neurotransmitters and hormones.

Organ	Hormone	Hormone Functions
Hypothalamus	Various releasing hormones	Promote or inhibit release of various hormones by pituitary
Anterior pituitary	Thyroid-stimulating hormone (TSH)	Stimulates thyroid gland
	Luteinizing hormone (LH)	Increases production of progesterone (female), testosterone (male); stimulates ovulation
	Follicle-stimulating hormone (FSH)	Increases production of estrogen and maturation of ovum (female) and sperm production (male)
	ACTH	Increases secretion of steroid hormones by adrenal gland
	Prolactin	Increases milk production
	Growth hormone (GH), also known as somatotropin	Increases body growth, including the growth spurt during puberty
Posterior pituitary	Oxytocin	Controls uterine contractions, milk release, certain aspects of parental behavior, and sexual pleasure
	Vasopressin (also known as antidiuretic hormone)	Constricts blood vessels and raises blood pressure, decreases urine volume
Pineal	Melatonin	Increases sleepiness, influences sleep–wake cycle, also has role in onset of puberty
Thyroid	Thyroxine Triiodothyronine	Increase metabolic rate, growth, and maturation
Parathyroid	Parathyroid hormone	Increases blood calcium and decreases potassium
Adrenal cortex	Aldosterone	Reduces secretion of salts by the kidneys
	Cortisol, corticosterone	Stimulate liver to elevate blood sugar, increase metabolism of proteins and fats
Adrenal medulla	Epinephrine, norepinephrine	Similar to effects of sympathetic nervous system
Pancreas	Insulin	Increases entry of glucose to cells and increases storage as fats
	Glucagon	Increases conversion of stored fats to blood glucose
Ovary	Estrogens	Promote female sexual characteristics
	Progesterone	Maintains pregnancy
Testis	Androgens	Promote sperm production, growth of pubic hair, and male sexual characteristics
Liver	Somatomedins	Stimulate growth
Kidney	Renin	Converts a blood protein into angiotensin, which regulates blood pressure and contributes to hypovolemic thirst
Thymus	Thymosin (and others)	Support immune responses
Fat cells	Leptin	Decreases appetite, increases activity, necessary for onset of puberty

Just as circulating hormones modify brain activity, hormones secreted by the brain control the secretion of many other hormones. The **pituitary gland,** attached to the hypothalamus, consists of two distinct glands, the **anterior pituitary** and the **posterior pituitary,** which release different sets of hormones. The posterior pituitary, composed of neural tissue, can be considered an extension of the hypothalamus. Neurons in the hypothalamus synthesize the hormones **oxytocin** and **vasopressin** (also known as antidiuretic hormone), which migrate down axons to the posterior pituitary. Later, the posterior pituitary releases these hormones into the blood.

The anterior pituitary, composed of glandular tissue, synthesizes six hormones, although the hypothalamus controls their release. The hypothalamus secretes **releasing hormones,** which flow through the blood to the anterior pituitary. There they stimulate or inhibit the release of the following hormones:

Adrenocorticotropic hormone (ACTH) - Controls secretions of the adrenal cortex
Thyroid-stimulating hormone (TSH) - Controls secretions of the thyroid gland
Prolactin - Controls secretions of mammary glands
Somatotropin, also known as
growth hormone (GH) - Promotes growth throughout the body
Gonadotropins
Follicle-stimulating hormone (FSH) -
Luteinizing hormone (LH) - Control secretions of the gonads

The hypothalamus maintains fairly constant circulating levels of certain hormones through a negative feedback system. For example, when the level of thyroid hormone is low, the hypothalamus releases *TSH-releasing hormone,* which stimulates the anterior pituitary to release TSH, which in turn causes the thyroid gland to secrete more thyroid hormones

The peripheral nervous system (PNS)

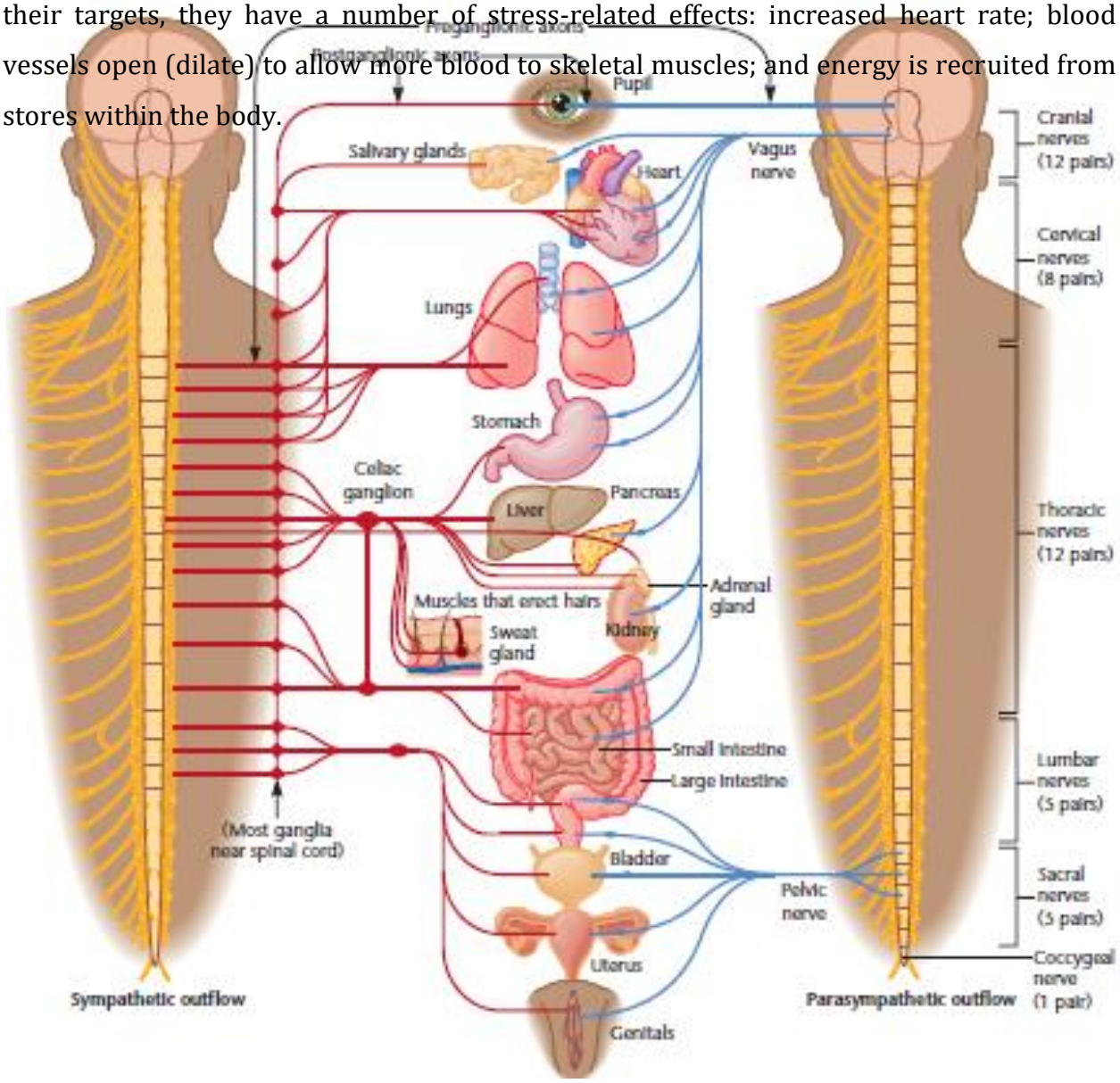
The CNS would be of little use if it could not communicate with the organs and glands in the rest of the body. As well as conveying information to the CNS, the PNS carries out the commands of the CNS, as well as regulating some vital bodily functions in its own right. The PNS has two divisions: (a) the *somatic nervous system* (SNS); and (b) the *autonomic nervous system* (ANS*)*. The SNS is responsible for interacting with the external environment; that is, information from the sensory receptors (in skin, muscles and joints) to the CNS, and for sending motor signals from the CNS to muscles and glands. The ANS is responsible for interacting with the internal environment, regulating basic processes of the body (e.g., such as the heart, blood vessels, digestive system and genital organs). Figure below shows some of the major jobs carried out by the PNS, and from where in the spinal cord these instructions are issued.

Some sensory nerves do not go via the spinal cord: above the neck, information transfer between the body and the brain is via 12 pairs of *cranial nerves* (e.g., the optic nerves, which convey visual information from the eye to the brain; and the oculomotor nerves, which control eye movements).

The ANS is divided into *sympathetic* (fight–flight) and *parasympathetic* (rest–digest) branches, which exert opposite effects (by the release of different types of neurotransmitter). In times of emergency, the sympathetic branch dominates and inhibits the parasympathetic branch. This results in increased heart rate and increased blood flow to the muscles (to ready them for fight or flight). In times of quiet, the parasympathetic system dominates, resulting in lower heart rate and blood being diverted from the skeletal muscles to the gut to assist digestion.

The CNS may activate the ANS directly (e.g., when threat is detected). Under these aversive conditions, the brain will also activate the neuroendocrine system, which will be responsible for releasing hormones into the bloodstream. For example, the brain response to stress triggers the release of corticosteroids from the adrenal glands. When they reach

their targets, they have a number of stress-related effects: increased heart rate; blood vessels open (dilate) to allow more blood to skeletal muscles; and energy is recruited from stores within the body.

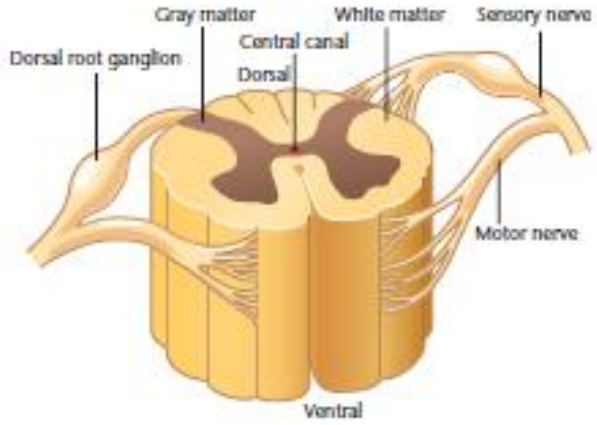


Central nervous system (CNS)

The CNS is composed of the spinal cord and the brain, as follows.

Spinal cord

The spinal cord communicates with all the sense organs and muscles except those of the head. It is a segmented structure, and each segment has on each side a sensory nerve and a motor nerve. The spinal cord contains 31 pairs of spinal nerves; these are 'mixed' nerves because each contains both sensory and motor axons. Outside the spinal cord these nerves separate: all sensory axons pass into the *dorsal root ganglion*, where their cell bodies are located, and then into the spinal cord itself; and all motor axons pass into the *ventral roots* after they leave the spinal cord, before uniting with the sensory axons to form the mixed nerves.



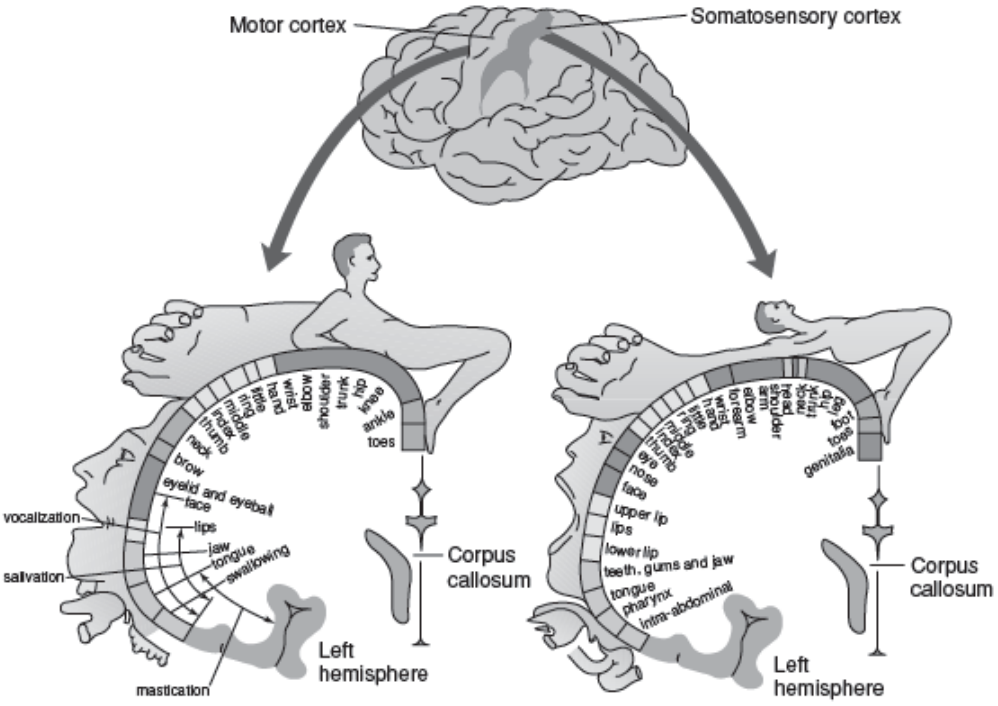
The H-shaped **gray matter** in the center of the cord is densely packed with cell bodies and dendrites. Many neurons of the spinal cord send axons from the gray matter to the brain or other parts of the spinal cord through the **white matter,** which consists mostly of myelinated axons. Each segment of the spinal cord sends sensory information to the brain and receives motor commands from the brain. All that information passes through tracts of axons in the spinal cord. If the spinal cord is cut at a given segment, the brain loses sensation from that segment and below. The brain also loses motor control over all parts of the body served by that segment and the lower ones.

Structure–Function Relations

The billions of neurons in the brain are organized into assemblies of specialized functional systems (e.g., visual system, motor system, olfactory system, motivational system). Often, especially in the case of complex psychological processes, a particular neural system in the

brain may be involved in very different psychological functions. For example, the hippocampus (an important structure in the limbic system) seems to be involved in a range of psychological functions, including memory, learning, anxiety and resolution of goal conflict.

There is a mapping of sensory and motor functions in the cerebral hemispheres. As is shown in figure below, those parts of the body that require greater sensory processing (e.g., fingers) get more cortical space than other areas that are grosser in their operation (e.g., knees). These are the sensory and motor homunculi (Latin: 'little men'). The somatosensory cortex (also known as the *postcentral gyrus*) of the parietal lobe processes information from the skin, joints and muscles. Neural transmission via the PNS enters the spinal cord and then the thalamus of the brain before being sent to the somatosensory cortex for processing. The primary motor cortex (also known as the *precentral gyrus*) sends information from the cortex to the motor systems in the body.



UNIT IV: NEUROPHYSIOLOGY

The term *neurophysiology* is used to refer to the experimental (direct) study of physiological processes in the brain, as opposed to merely correlating activity in the brain with function. Neurophysiology is distinct from neuropsychology, psychophysiology and neuroimaging in that it entails the *experimental* manipulation of brain processes, which is necessarily invasive and often destructive.

Attention & Consciousness:

Consciousness is difficult to define, but for practical purposes, researchers use this operational definition: If a cooperative person reports the presence of one stimulus and cannot report the presence of a second stimulus, then he or she was **conscious** of the first and not of the second. This definition does not apply (one way or the other) to individuals who cannot speak—such as infants, people with Broca's aphasia, or nonhuman animals. We might draw inferences about their consciousness based on other criteria, but we won't use them for research on consciousness.

By this definition, consciousness is almost synonymous with attention. At any moment, a huge number of stimuli reach your brain, but you are conscious of (i.e., able to report) only those to which you direct your attention. Various stimuli compete for your conscious attention. A stimulus can grab your attention by its size, brightness, or movement, but you can also voluntarily direct your attention to one stimulus or another in what is called a "top-down" process— that is, one governed by other cortical areas, principally the prefrontal and parietal cortex.

Endogenous Cycles

An animal that produced its behavior entirely in response to current stimuli would be at a serious disadvantage. Animals often need to prepare for changes in sunlight and temperature before they occur. For example, migratory birds start flying toward their winter homes before their summer territory becomes too cold. A bird that waited for the first frost would be in serious trouble. Similarly, squirrels begin storing nuts and putting on extra layers of fat in preparation for winter long before food becomes scarce.

Animals' readiness for a change in seasons comes partly from internal mechanisms. For example, several cues tell a migratory bird when to fl y south for the winter, but after it reaches the tropics, what tells it when to fl y back north? In the tropics, the temperature and amount of daylight are nearly the same throughout the year. Nevertheless, a migratory bird flies north at the right time. Even if it is kept in a cage with no clues to the season, it becomes restless in the spring, and if it is released, it flies north. Evidently, the bird generates a rhythm that prepares it for seasonal changes. We refer to that rhythm as an **endogenous circannual rhythm.** (*Endogenous* means "generated from within." *Circannual* comes from the Latin words *circum*, for "about," and *annum*, for "year.")

Similarly, animals produce **endogenous circadian rhythms** that last about a day. (*Circadian* comes from *circum*, for "about," and *dies*, for "day.") If you go without sleep all night—as most college students do, sooner or later—you feel sleepier and sleepier as the night goes on, but as morning arrives, you feel less sleepy. For one reason, the light from the sun helps you feel less sleepy. Furthermore, your urge to sleep depends partly on the time of day, not just how many hours you have been awake.

Humans also generate wake–sleep rhythms. Mammals, including humans, have circadian rhythms in their waking and sleeping, eating and drinking, urination, secretion of hormones, sensitivity to drugs, and other variables. For example, although we ordinarily think of human body temperature as 37°C, normal temperature fluctuates over the course of a day from a low near 36.7°C during the night to almost 37.2°C in late afternoon. Circadian rhythms differ among individuals. Some people ("morning people," or "larks") awaken early, quickly become productive, and become less alert as the day progresses.

Not everyone falls neatly into one extreme or the other, of course. A convenient way to compare people is to ask, "On holidays and vacations when you have no obligations, what time is the middle of your sleep?" For example, if you slept from 1 a.m. until 9 a.m. on those days, your middle would be 5 a.m. People differ by age. As a child, you almost certainly went to bed early and woke up early. As you entered adolescence, you started staying up later and waking up later, when you had the opportunity. The mean preferred time of going to sleep gets later and later until about age 20 and then starts a gradual reversal.

Setting and Resetting the Biological Clock

Our circadian rhythms generate a period close to 24 hours, but they are not perfect. We readjust our internal workings daily to stay in phase with the outside world. Sometimes, we misadjust them. On weekends, when most of us are freer to set our own schedules, we expose ourselves to lights, noises, and activity at night and then awaken late the next morning.

By Monday morning, when the clock indicates 7 a.m., the biological clock within us says about 5 a.m., and we stagger off to work or school without much pep. Although circadian rhythms persist without light, light is critical for resetting them. I used to have a windup wristwatch that lost about 2 minutes per day, which would accumulate to an hour per month if I didn't reset it. It had a **free-running rhythm** of 24 hours and 2 minutes—that is, a rhythm that occurs when no stimuli reset or alter it. The circadian rhythm of the body is similar. Without something to reset it, it would drift further and further. The stimulus that resets the circadian rhythm is referred to by the German term **zeitgeber**, meaning "time-giver." Light is the dominant zeitgeber for land animals. (The tides are important for many marine animals.) In addition to light, other zeitgebers include exercise, noise, meals, and the temperature of the environment. However, these additional zeitgebers merely supplement or alter the effects of light. On their own, their effects are generally weak. For example, people who are working in Antarctica during the Antarctic winter, with no sunlight, try to maintain a 24-hour rhythm, but different people generate different free-running rhythms, until they find it more and more difficult to work together.

Even when we try to set our wake–sleep cycles by the clock, the sun has its influence. Consider what happens when we shift to daylight savings time in spring. You set your clock to an hour later, and when it shows your usual bedtime, you dutifully go to bed, even though it seems an hour too early. The next morning, when the clock says it is 7 a.m. and time to get ready for work, your brain still registers 6 a.m. Most people are inefficient and ill-rested for days after the shift to daylight savings time. The adjustment is especially difficult for evening people and those who were already sleep-deprived, including most college students.

Particularly impressive evidence for the importance of sunlight comes from a study in Germany. The "sun" time at the eastern end of Germany differs by about half an hour from that at the western edge, even though everyone is on the same "clock" time. Researchers asked adults for their preferred times of awakening and going to sleep and determined for each person the midpoint of those values. (For example, if on weekends and holidays you prefer to go to bed at 12:30 a.m. and awaken at 8:30 a.m., your sleep midpoint is 4:30 a.m., or 4.5 hours.) Figure 9.4 shows the results. People at the eastern edge have a sleep midpoint about 30 minutes earlier than those at the west, corresponding to the fact that the sun rises earlier at the eastern edge.

The data shown here apply to people in towns and cities with populations under 300,000. People in larger cities show a less consistent trend, presumably because they spend more time indoors and have less exposure to the sun. What about blind people, who need to set their circadian rhythms by zeitgebers other than light? The results vary. Some do set their circadian rhythms by noise, temperature, meals, and activity. However, others who are not sufficiently sensitive to these secondary zeitgebers produce free-running circadian rhythms that are a little longer than 24 hours. When their cycles are in phase with the clock, all is well, but when they drift out of phase, they experience insomnia at night and sleepiness during the day.

Jet Lag

A disruption of circadian rhythms due to crossing time zones is known as **jet lag.** Travelers complain of sleepiness during the day, sleeplessness at night, depression, and impaired

concentration. All these problems stem from the mismatch between internal circadian clock and external time. Most of us find it easier to adjust to crossing time zones going west than east. Going west, we stay awake later at night and then awaken late the next morning, already partly adjusted to the new schedule. We *phase-delay* our circadian rhythms. Going east, we *phase-advance* to sleep earlier and awaken earlier. Most people find it difficult to go to sleep before their body's usual time.

Adjusting to jet lag is more stressful for some people than for others. Stress elevates blood levels of the adrenal hormone *cortisol,* and many studies have shown that prolonged elevations of cortisol damage neurons in the hippocampus, a brain area important for memory. One study examined flight attendants who had spent the previous 5 years making flights across seven or more time zones—such as Chicago to Italy—with mostly short breaks (fewer than 6 days) between trips. On the average, they showed smaller than average volumes of the hippocampus and surrounding structures, and they showed some memory impairments. These results suggest a danger from repeated adjustments of the circadian rhythm, although the problem here could be just air travel itself.

Shift Work

People who sleep irregularly—such as pilots, medical interns, and shift workers in factories—find that their duration of sleep depends on when they go to sleep. When they have to sleep in the morning or early afternoon, they sleep only briefly, even if they have been awake for many hours. People who work on a night shift, such as midnight to 8 a.m., sleep during the day. At least they try to. Even after months or years on such a schedule, many workers adjust incompletely. They continue to feel groggy on the job, they do continues to peak when they are trying to sleep in the day instead of while they are working at night. In general, night-shift workers have more accidents than day-shift workers. Working at night does not reliably change the circadian rhythm because most buildings use artificial lighting in the range of 150–180 lux, which is only moderately effective in resetting the rhythm. People adjust best to night work if they sleep in a very dark room during the day and work under very bright lights at night, comparable to the noonday sun.

Mechanisms of the Biological Clock

How does the body generate a circadian rhythm? Curt Richter (1967) introduced the concept that the brain generates its own rhythms—a biological clock—and he reported that the biological clock is insensitive to most forms of interference. Blind or deaf animals generate circadian rhythms, although they slowly drift out of phase with the external world. The circadian rhythm is surprisingly steady despite food or water deprivation, x-rays, tranquilizers, alcohol, anesthesia, lack of oxygen, most kinds of brain damage, or the removal of hormonal organs. Even an hour or more of induced hibernation often fails to reset the biological clock. Evidently, the biological clock is a hardy, robust mechanism.

The Suprachiasmatic Nucleus (SCN)

The biological clock depends on part of the hypothalamus, called the **suprachiasmatic** (soo-pruh-kie-as-MAT-ik) **nucleus,** or **SCN.** It gets its name from its location just above ("supra") the optic chiasm. The SCN provides the main control of the circadian rhythms for sleep and body temperature, although several other brain areas generate local rhythms. After damage to the SCN, the body's rhythms are less consistent and no longer synchronized to environmental patterns of light and dark. The SCN generates circadian rhythms itself in a genetically controlled, unlearned manner. If SCN neurons are disconnected from the rest of the brain or removed from the body and maintained in tissue culture, they continue to produce a circadian rhythm of action potentials. Even a single isolated SCN cell can maintain a circadian rhythm, although interactions among cells sharpen the accuracy of the rhythm.

How Light Resets the SCN

The SCN is located just above the optic chiasm. The relationship is similar in other mammals.) A small branch of the optic nerve, known as the *retinohypothalamic path,* extends directly from the retina to the SCN. Axons of that path alter the SCN's settings.

Most of the input to that path, however, does not come from normal retinal receptors. Mice with genetic defects that destroy nearly all their rods and cones nevertheless reset their biological clocks in synchrony with the light.

For all mammals, the retinohypothalamic path to the SCN comes from a special population of retinal ganglion cells that have their own photopigment, called *melanopsin,* unlike the ones found in rods and cones. These special ganglion cells respond directly to light even if they do not receive any input from rods or cones. They do, nevertheless, receive some input from the rods and cones, which supplements their own direct response to light. The special ganglion cells are located mainly near the nose, not evenly throughout the retina. These cells respond to light slowly and turn off slowly when the light ceases. Therefore, they respond to the overall average amount of light, not to instantaneous changes in light. The average intensity over a period of minutes or hours is, of course, exactly the information the SCN needs to gauge the time of day. Because they do not contribute to vision, the cells do not need to respond to momentary changes in light.

The Biochemistry of the Circadian Rhythm

Research on the mechanism of circadian rhythms began with insects, where the genetic basis is easier to explore, because they reproduce in weeks instead of months or years. Studies on the fruit fly *Drosophila* discovered genes that generate a circadian rhythm. Two genes, known as *period* (abbreviated *per*) and *timeless* (*tim*), produce the proteins Per and Tim. Those proteins start in small amounts early in the morning and increase during the day. By evening, they reach a high level that makes the fl y sleepy. That high level also feeds back to the genes to shut them down. During the night, while the genes no longer produce Per or Tim, their concentration declines until the next morning, when the cycle begins anew. When the Per and Tim levels are high, they interact with a protein called Clock to induce sleepiness. When they are low, the result is wakefulness. Furthermore, a pulse of light during the night inactivates the Tim protein, so extra light during the evening decreases sleepiness and resets the biological clock.

In mammals, light alters the production of the Per and Tim proteins, which increase the activity of certain neurons in the SCN. Understanding these mechanisms helps make

sense of some unusual sleep disorders. Mice with damage to their *clock* gene, which interacts with the *per* and *tim* genes, sleep less than normal, and presumably, some cases of decreased sleep in humans might have the same cause. Various genes modify the activity of the *clock* and *period* genes, and mice with a mutation in one of the modifier genes, known as *overtime*, produce circadian rhythms lasting 26 hours instead of 24. Any people with a similar mutation would have extreme difficulty waking up at the normal time. They would feel as if they were moving two time zones east every day. One mutation of the *period* gene has been found in humans. People with this mutation have a circadian rhythm that runs faster than 24 hours, as if they were moving one or two time zones west every day. They consistently get sleepy early in the evening and awaken early in the morning. Most people look forward to days when they can stay up late and then sleep late the next morning. People with the altered *period* gene look forward to days when they have the opportunity to go to bed even earlier than usual and waken especially early the next day. Most people with this sleep abnormality suffer from depression. Sleep difficulties and depression are closely linked.

Melatonin

The SCN regulates waking and sleeping by controlling activity levels in other brain areas, including the **pineal gland**, an endocrine gland located just posterior to the thalamus. The pineal gland releases the hormone **melatonin,** which influences both circadian and circannual rhythms. The human pineal gland secretes melatonin mostly at night, making us sleepy at that time. When people shift to a new time zone and start following a new schedule, they continue to feel sleepy at their old times until the melatonin rhythm shifts.

People who have pineal gland tumors sometimes stay awake for days at a time. Melatonin secretion starts to increase about 2 or 3 hours before bedtime. Taking a melatonin pill in the evening has little effect on sleepiness because the pineal gland produces melatonin at that time anyway. However, people who take melatonin at other times become sleepy within 2 hours. Melatonin pills are sometimes helpful when people travel across time zones or for any other reason need to sleep at an unaccustomed time.

Melatonin also feeds back to reset the biological clock through its effects on receptors in the SCN. A moderate dose of melatonin (0.5 mg) in the afternoon phase-advances the clock. That is, it makes the person get sleepy earlier in the evening and wake up earlier the next morning. A single dose of melatonin in the morning has little effect, although repeated morning doses can phase-delay the clock, causing the person to get sleepy later than usual at night and awaken later the next morning.

Taking melatonin has become something of a fad. Melatonin is an antioxidant, so it has some health benefits. However, in laboratory animals, it has been shown to impair learning, presumably as a result of increasing drowsiness. Also, long-term use impairs animals' reproductive fertility and, if taken during pregnancy, harms the development of the fetus.

Emotions, Autonomic Arousal, and the James-Lange Theory

Emotional situations arouse the autonomic nervous system, which has two branches—the sympathetic and the parasympathetic. The sympathetic nervous system prepares the body for brief, vigorous "fight-or-flight" responses. The parasympathetic nervous system increases digestion and other processes that save energy and prepare for later events. However, each situation evokes its own special mixture of sympathetic and parasympathetic arousal. For example, nausea is associated with sympathetic stimulation of the stomach (decreasing its contractions and secretions) and parasympathetic stimulation of the intestines and salivary glands.

According to the **James-Lange theory**, the autonomic arousal and skeletal actions come first. What we experience as an emotion is the label we give to our responses: I am afraid *because* I run away; I am angry *because* I attack. An emotion has three components: cognitions, actions, and feelings. The cognitive aspect comes first. You appraise something as good, bad, frightening, or whatever. Ordinarily, you make that appraisal within a split second. Your appraisal of the situation leads to an appropriate action, such as running away, attacking, or sitting motionless with your heart racing. When William James had said that arousal and actions lead to emotions, what he meant was the *feeling* aspect of an emotion. The James-Lange theory leads to two predictions: People with weak autonomic or

skeletal responses should feel less emotion, and causing or increasing someone's responses should enhance an emotion.

The Functions of Emotions

If we evolved the capacity to experience and express emotions, emotions must have been adaptive to our ancestors, and they probably are to us as well. What good do emotions do?

For certain emotions, the answer is clear. Fear alerts us to escape from danger. Anger directs us to attack an intruder. Disgust tells us to avoid something that might cause illness. The adaptive value of happiness, sadness, embarrassment, and other emotions is less obvious, although researchers have suggested some plausible possibilities.

Also, emotions provide a useful guide when we need to make a quick decision. Sometimes, your "gut feeling" is useful. In one study, college students viewed a series of slides of snakes and spiders, each presented for just 10 ms, followed by a masking stimulus—a random array of unrecognizable patterns. Under these conditions, people cannot identify whether they saw a snake or a spider. For each participant, one kind of stimulus—either the snakes or the spiders—was always followed by a mild shock 5.6 seconds later. Most of those shocked after spider pictures developed a bigger heart rate increase after spider pictures, and people shocked after snake pictures learned an increased heart rate after snake pictures, even though neither group could consciously identify the pictures. On certain trials, participants were asked to report any perceived changes in their heart rate, which were compared to measurements of their actual heart rate. On other trials, after the stimulus, they guessed whether a shock was forthcoming. In general, those who were most accurate at reporting their heart rate increases were the most accurate at predicting whether they were about to get a shock. The interpretation is that people who are good at detecting their autonomic responses may have valid gut feelings about dangers that they cannot identify consciously.

Physiological and chemical correlates of Learning and Memory:

Localized Representations of Memory

What happens in the brain during learning and memory? One early idea was that a connection grew between two brain areas. The Russian physiologist Ivan Pavlov pioneered the investigation of what we now call **classical conditioning**, in which, pairing two stimuli changes the response to one of them. The experimenter starts by presenting a **conditioned stimulus (CS),** which initially elicits no response of note, and then presents the **unconditioned stimulus (UCS),** which automatically elicits the **unconditioned response (UCR).** After some pairings of the CS and the UCS (perhaps just one or two, perhaps many), the individual begins making a new, learned response to the CS, called a **conditioned response (CR).** In his original experiments, Pavlov presented a dog with a sound (CS) followed by meat (UCS), which stimulated the dog to salivate (UCR). After many such pairings, the sound alone (CS) stimulated the dog to salivate (CR). In that case and many others, the CR resembles the UCR, but in some cases, it does not. For example, if a rat experiences a CS paired with shock, the shock elicits screaming and jumping, but the CS elicits a freezing response.

In **operant conditioning,** an individual's response leads to a reinforcer or punishment. A **reinforcer** is any event that increases the future probability of the response. A **punishment** is an event that suppresses the frequency of the response. For example, when a rat enters one arm of a maze and finds Froot Loops cereal (a potent reinforcer for a rat), its probability of entering that arm again increases. If it receives a shock instead, the probability decreases. The primary difference between classical and operant conditioning is that in operant conditioning the individual's response determines the outcome (reinforcer or punishment), whereas in classical conditioning the CS and UCS occur at certain times regardless of the individual's behavior. (The behavior is useful, however, in anticipating the effects of the UCS.)

Some cases of learning are difficult to label as classical or operant. For example, after a male songbird hears the song of his own species during his first few months, he imitates it the following year. The song that he heard was not paired with any other stimulus, as in classical conditioning. He learned the song without reinforcers or punishments, so we can't call it operant conditioning either. That is, animals have specialized methods of learning other than classical and operant conditioning. Also, the

way animals (including people) learn varies from one situation to another. For example, in most situations, learning occurs only if the CS and UCS, or response and reinforcer, occur close together in time. But if you eat something, especially something unfamiliar, and get sick later, you learn a strong aversion to the taste of that food, even if taste and illness are separated by hours.

Types of Memory

Psychologists distinguish between learning and memory. If nothing else, learning researchers and memory researchers use different methods. Most learning researchers focus on classical or operant conditioning, using laboratory animals.

Memory researchers ask people to describe events in words. In reality, the distinction is arbitrary, as you can't learn something without remembering it, and you can't remember something without learning it. Nevertheless, regardless of whether we use the term *learning* or *memory,* we need to draw distinctions among various types. Decades ago, psychologists expected to find laws of learning or laws of memory that would apply to all situations. Gradually, they became aware of important differences among different types of learning and memory. Researchers continue to explore exactly what are the best distinctions to draw, and studies of brain damage make an important contribution to this pursuit.

Short-Term and Long-Term Memory

We form memories quickly, and some memories last a lifetime. These can be distinguished as **short-term memory** of events that have just occurred and **long-term memory** of events from further back. Several types of evidence supported this distinction:

- Short-term memory and long-term memory differ in their capacity. If you hear a series of numbers or letters, such as DZLAUV, you can probably repeat no more than about seven of them, and with other kinds of material, your maximum is even less. Long-term memory has a vast, difficult-to-estimate capacity.

- Short-term memories fade quickly unless you rehearse them. For example, if you read the letter sequence DZLAUV and then something distracts you, your chance of repeating the letters declines rapidly over about 20 seconds. You can recall long term memories that you haven't thought about in years.
- With short-term memory, once you have forgotten something, it is lost. With long-term memory, a hint might help you reconstruct something you thought you had forgotten. For example, try naming all your high school teachers. After you have named all you can, you can name still more if someone shows you photos and tells you the teachers' initials. Based on these distinctions, researchers proposed that all information initially entered a short-term storage, where it stayed until the brain had time to **consolidate** it into longterm memory. If anything interrupted the rehearsal before consolidation took place, the information was simply lost.

Working Memory

Later studies weakened the distinction between short-term and long-term memory. For example, most of the research demonstrating rapid loss of unrehearsed short-term memories dealt with meaningless materials, such as a series of letters or numbers. You hold onto many memories for hours or days without constant rehearsal—such as where you plan to meet someone for lunch, where you parked your car, or when is your next dentist's appointment. Furthermore, the time needed for consolidation varies enormously. You know this from your own experience. If someone tells you something you consider interesting, about a topic you already know well, you learn it quickly and remember it well. If you hear something about a topic unfamiliar to you, remembering it is much more difficult. The same is true for laboratory animals: If they have had much training of a particular type, remembering new material of the same type is easy and requires little practice.

As an alternative to the concept of short-term memory, A. D. Baddeley and G. J. Hitch introduced the term **working memory** to emphasize that temporary storage is not a station on the route to long-term memory but the way we store information while we are working with it. A common test of working memory is the **delayed response task,** which

requires responding to something that you saw or heard a short while ago. For example, imagine that a light shines above one of several doors. The light goes off, you wait a few seconds, and now you have to go to the door where you saw the light. The delay can be increased or decreased to test your limits. This task can be modified for use with nonhumans as well as humans. During the delay, the learner has to store a representation of the stimulus, and much research points to the prefrontal cortex as the primary location for this storage. Initially, researchers assumed that the cells stored the information by repetitive action potentials. However, action potentials consume much energy. The brain may use some more economical way of representing temporary information, such as elevated levels of calcium, which would potentiate later responses, when the time comes.

Many older people have impairments of working memory, probably because of changes in the prefrontal cortex. Studies on aged monkeys find decreases in the number of neurons and the amount of input in certain parts of the prefrontal cortex. Older humans who show declining memory show declining activity in the prefrontal cortex, whereas those with intact memory show *greater* activity than young adults. Presumably, the increased activity means that the prefrontal cortex is working harder in these older adults to compensate for impairments elsewhere in the brain. Furthermore, stimulant drugs that enhance activity in the prefrontal cortex produce a long-lasting improvement in the memory of aged monkeys.

The Hippocampus and Amnesia

Amnesia is memory loss. One patient ate lunch and, 20 minutes later, ate a second lunch, apparently having forgotten the first meal. Another 20 minutes later, he started on a third lunch and ate most of it. A few minutes later, he said he would like to "go for a walk and get a good meal". However, even in severe cases like this, no one loses all kinds of memory equally. This patient still remembered how to eat with a knife and fork, for example, even though he could not remember what he had eaten or when. Studies on amnesia help clarify the distinctions among different kinds of memory and enable us to explore the mechanisms of memory.

People With Hippocampal Damage

In 1953, a man known as H. M. suffered about 10 minor epileptic seizures per day and a major seizure about once a week, despite trying every available antiepileptic drug. Eventually, he and his neurosurgeon considered a desperate measure. Because of evidence suggesting that epilepsy sometimes originates in the hippocampus, the neurosurgeon removed it from both hemispheres, as well as much of the amygdala and other nearby structures in the temporal cortex. Researchers knew almost nothing about the hippocampus at the time, and no one knew what to expect after the surgery. We now know that various parts of the hippocampus are active during both the formation of memories and later recall. Although the operation reduced H. M.'s epilepsy to no more than two major seizures per year, he almost certainly would have preferred to remain epileptic.

Anterograde and Retrograde Amnesia.

After the surgery, H. M.'s intellect and language abilities remained intact, and his personality remained the same except for emotional placidity, probably related to the amygdala damage. For example, he rarely complained (even about pain) or requested anything (even food). However, he suffered massive **anterograde amnesia** (inability to form memories for events that happened after brain damage). He also suffered a **retrograde amnesia** (loss of memory for events that occurred before the brain damage). Initially, researchers said his retrograde amnesia was confined to 1 to 3 years before the surgery. Later, they found it was more extensive. H. M. is representative of many people who have suffered amnesia after damage to the hippocampus and surrounding structures, which together constitute the medial temporal lobe. All show both anterograde and retrograde amnesia, with the retrograde amnesia being most severe for the last few years before the damage. For example, amnesic patients can usually tell where they lived as a child and where they lived as a teenager but might not be able to say where they lived 3 years ago.

UNIT V: COGNITIVE NEUROSCIENCE

Cognitive Neuroscience is concerned with the relationship between physiological structures and processes and psychological functions. This is a broad definition and encompasses most areas of biological psychology involving structure–function relations,

including those in non-human animals. Traditionally, the study of brain structure and psychological function has relied upon congenital deficits, accidents, strokes, etc. This type of study has been successful, although problematic, leading among other things to the discovery of the speech areas of the brain. It was not until Wilder Penfield's pioneering electrical stimulation of the cortex in awake patients that a more direct approach could be adapted to mapping functions in the brain.

How Did Humans Evolve Language?

How did we evolve the ability to learn language so much more easily than other species? Most theories fall into two categories: (a) we evolved language as a by-product of overall brain development or (b) we evolved it as a brain specialization.

Language as a Product of Overall Intelligence

The simplest view is that humans evolved big brains and therefore great intelligence, and language developed as an accidental by-product of intelligence. In its simplest form, this hypothesis faces serious problems.

Brain Damage and Language

Because almost every healthy child develops language, we infer that the human brain is specialized to facilitate language learning. Much of our knowledge about the brain mechanisms of language has come from studies of people with brain damage.

Broca's Aphasia (Nonfluent Aphasia)

In 1861, the French surgeon Paul Broca treated the gangrene of a patient who had been mute for 30 years. When the man died 5 days later, Broca did an autopsy and found a lesion in the left frontal cortex. Over the next few years, Broca examined the brains of additional patients with **aphasia** (language impairment). In nearly all cases, he found damage that included this same area, which is now known as **Broca's area**. The usual cause was a stroke (an interruption of blood flow to part of the brain). Broca published his results in 1865, slightly later than papers by other French physicians, Marc and Gustave Dax, who also pointed to the left hemisphere as the seat of language abilities.

Broca is given the credit, however, because his description was more detailed and more convincing. This discovery, the first demonstration of a particular function for a particular brain area, paved the way for modern neurology. We now know that speaking activates much of the brain, mostly in the left hemisphere, and not just Broca's area. Damage limited to Broca's area produces only minor or brief language impairment. Serious deficits result from extensive damage that extends into other areas as well. The symptoms vary and are not completely predictable from the location of the damage.

When people with brain damage suffer impaired language production, we call it **Broca's aphasia,** or **nonfluent aphasia**, regardless of the exact location of the damage. People with Broca's aphasia also have comprehension deficits when the meaning of a sentence depends on prepositions, word endings, or unusual word order—in short, when the sentence structure is complicated.

People with Broca's aphasia are slow and awkward with all forms of expression, including speaking, writing, and gesturing. The frontal cortex is also important for the sign language of the deaf, although the right hemisphere makes more contributions than it does for spoken language. So Broca's aphasia relates to language, not just the vocal muscles.

When people with Broca's aphasia speak, they omit most pronouns, prepositions, conjunctions, auxiliary (helping) verbs, quantifiers, and tense and number endings.

People with Broca's aphasia have trouble understanding the same kinds of words that they omit when speaking, such as prepositions and conjunctions. They often misunderstand sentences with complex grammar, such as "The girl that the boy is chasing is tall". However, most English sentences follow the subject-verb-object order, and their meaning is clear even without the prepositions and conjunctions. Still, people with Broca's aphasia have not totally lost their knowledge of grammar.

Wernicke's Aphasia (Fluent Aphasia)

In 1874, Carl Wernicke (usually pronounced WER-nih-kee by English speakers, although the German pronunciation is VAYR-nih-keh), a 26-year-old junior assistant in a German hospital, discovered that damage in part of the left temporal cortex produced a diff erent kind of language impairment. Although patients could speak and write, their language

comprehension was poor. Damage in and around **Wernicke's area**, located near the auditory cortex, produces **Wernicke's aphasia,** characterized by poor language comprehension and impaired ability to remember the names of objects. It is also known as **fluent aphasia** because the person can still speak smoothly. As with Broca's aphasia, the symptoms and brain damage vary. We use the term Wernicke's aphasia, or fluent aphasia, to describe a certain pattern of behavior, independent of the location of damage.

The typical characteristics of Wernicke's aphasia are as follows:

1. *Articulate speech*. In contrast to people with Broca's aphasia, those with Wernicke's aphasia speak fluently, except for pauses to try to think of the name of something.

2. *Difficulty finding the right word*. People with Wernicke's aphasia have **anomia** (ay-NOME-ee-uh), difficulty recalling the names of objects. They make up names (e.g., "thingamajig"), substitute one name for another, and use roundabout expressions such as "the thing that we used to do with the thing that was like the other one." When they do manage to find some of the right words, they arrange them improperly, such as, "The Astros listened to the radio tonight" (instead of "I listened to the Astros on the radio tonight")

3. *Poor language comprehension*. People with Wernicke's aphasia have trouble understanding spoken and written speech and—in the case of deaf people—sign language. Although many sentences are clear enough without prepositions, word endings, and grammar (which confuse Broca's aphasics), few sentences make sense without nouns and verbs (which trouble Wernicke's patients).

Studies with various kinds of brain scans confirm the importance of Wernicke's area and Broca's area for language. When you listen to speech, especially difficult or confusing speech, your brain responds first in the temporal lobe, including Wernicke's area, and then in the frontal lobe, including Broca's area.

Although Wernicke's area and surrounding areas are important, language comprehension also depends on the connections to other brain areas. For example, reading the word *lick* activates not only Wernicke's area but also the part of the motor cortex responsible for tongue movements. Reading *kick* activates the part of the motor cortex controlling foot movements.

Type	Pronunciation	Content of Speech	Comprehension
Broca's aphasia	Poor	Mostly nouns and verbs; omits prepositions and other grammatical connectives	Impaired if the meaning depends on complex grammar
Wernicke's aphasia	Unimpaired	Grammatical but often nonsensical; has trouble finding the right word, especially names of objects	Seriously impaired

The Left and Right Hemispheres

The left hemisphere of the cerebral cortex is connected to skin receptors and muscles mainly on the right side of the body. The right hemisphere is connected to skin receptors and muscles mainly on the left side. As an exception to this rule, both hemispheres control the trunk muscles and facial muscles. The left hemisphere sees only the right half of the world. The right hemisphere sees only the left half of the world. Each hemisphere gets auditory information from both ears but slightly stronger information from the contralateral ear. Taste and smell, however, are uncrossed. Each hemisphere gets taste information from its own side of the tongue and smell information from the nostril on its own side.

Why all vertebrates (and many invertebrates) evolved so that each hemisphere controls the contralateral (opposite) side of the body, no one knows. At any rate, the left and right hemispheres of the cerebral cortex exchange information through a set of axons called the **corpus callosum** and through the anterior commissure, the hippocampal commissure, and a couple of other small commissures. Information that initially enters one hemisphere crosses quickly so that both hemispheres have access to the information.

The two hemispheres are not mirror images of each other. In most humans, the left hemisphere is specialized for language. Such division of labor between the two hemispheres is known as **lateralization.** If you had no corpus callosum, your left hemisphere could react only to information from the right side of your body, and your right hemisphere could react only to information from the left. Because of the corpus callosum, however, each hemisphere receives information from both sides. Only after damage to the corpus callosum (or to one hemisphere) do we see clear evidence of lateralization.

The Right Hemisphere

The right hemisphere is dominant for recognizing emotions in others, including both pleasant and unpleasant emotions. In a split brain person, the right hemisphere does better than the left at recognizing whether two photographs show the same or different emotions. Moreover, studies of brain-intact people show that when the left and right hemispheres perceive different emotions in someone's face, the response of the right hemisphere dominates. The right hemisphere appears more adept than the left at comprehending spatial relationships.

Brain Injury

Historically, neuropsychology has had to rely upon brain damage to reveal patterns of cognitive deficits that would give clues to the association of brain regions and psychological performance. Such brain damage results from a variety of sources:

1. Traumatic head injury, resulting from road traffic accidents, falls, accidents or military missiles/gunshot;
2. Cerebral vascular accident (stroke);
3. Viral infection of the brain (e.g., encephalitis);
4. Hypoxic (oxygen-deficit) brain damage following myocardial infarction (heart attack), carbon monoxide poisoning (e.g., suicide attempts) or an anaesthetic accident;
5. Wernicke–Korsakoff syndrome as a result of alcoholism and inadequate nutrition;
6. Brain tumour (including unavoidable damage during surgery for tumour removal);
7. Degenerative disease (e.g., Alzheimer's disease and Huntington's disease);
8. Surgery that deliberately lesions part of the brain in order to control such neuropathologies as epilepsy (e.g., cutting the corpus callosum to prevent electrical discharges travelling from one hemisphere to the other.

Accidental brain damage

Each year many thousands of people suffer brain damage due to a variety of accidents, ranging from falling over and hitting the head on hard objects and road traffic accidents, to ingesting toxic substances (and non-accidents such as physical assault).

A peculiar fame: Phineas Gage

One of the most famous cases of accidental brain damage concerns a 25-year-old construction foreman working on the railway in Vermont, USA. In September 1848, Phineas Gage was loading explosives with a tamping rod (3 feet, 7 inches long, and 1 1/4 inches in diameter1 at one end, tapering to 1/4 inch at the other end, weighing about 13 lbs) in order to blast rock: the rod he was using to push in the explosives set them off, and the explosion sent the rod through his left cheek, passing through his left frontal lobe, and exiting at the top of his head, finally landing some 25 yards away. In fact, although he was knocked over by the blast, after a short while he was able to walk and talk (apparently asking the doctor whether he could return to work!). Remarkably, he survived this accident. As noted by the *Boston Post* (14 September 1848) the following day, 'The most singular circumstance connected with this melancholy affair is, that he was alive at two o'clock this afternoon, and in full possession of his reason, and free from pain.' Ten weeks after the accident, Phineas was at home recovering.

In the middle of 1849, Phineas returned to work. However, family, friends and colleagues noticed a change in his personality: before the trauma, he was sensitive, intelligent and respectful, being the mining contractor's most efficient foreman; now he was rude, profane, impatient, unable to settle on any plans for the future, and showing little regard for his fellow workers – because of his behaviour, he was not re-employed by the contractors. He lived until 1860; and in 1867 his body was exhumed and his skull removed. This was one of the early cases showing that frontal lobe damage can lead to disinhibited, impulsive and reckless behaviour; the damage seems to be located at the ventromedial region of the frontal lobes on both sides – speech and motor areas were spared.

There are several noteworthy features of this case. First, it was the first case which pointed to the relationship between brain damage to the frontal lobes and personality, and, in general, one of the best examples of the relationship between brain and behaviour. Second, it also had an influence on the development of brain surgery: Gage's injury

suggested that psychological functions are localized (the first brain surgery for a tumour was in 1885). In 1894, the first *psychosurgery* was performed (i.e., surgical disconnection or

Phineas Gage's skull and bar. The path of the tamping bar as it entered Gage's cheekbone and exited the ventromedial region of the frontal lobes.

removal of brain tissue to alleviate psychological/psychiatric symptoms): this patient reported becoming dull in thinking, generally lazy and slow in mental activity, and unable to express his ideas clearly. The surgeons made explicit reference to the 'American crowbar case' (i.e., Gage), which provided the theoretical grounds for the collection of further cases showing symptoms related to damage in specific brain areas.

For many years psychological symptoms were used as a diagnostic sign for brain tumours. Surgeons often removed the whole lobe, as the tumour was not sufficiently differentiated from the surrounding tissue. This procedure led to the further discovery that there were often few effects on patients' behaviour. Such observations encouraged the view that frontal lesions could be used to alleviate psychological/ psychiatric symptoms.

Phineas Gage is remarkable because his trauma was anatomically extensive, but his cognitive deficits were relatively minor (his emotional life, though, was greatly changed). However, in most cases of head injury there are notable cognitive consequences.

Cognitive Deficits

Traditional clinical neuropsychology has identified a number of specific deficits that show the high specificity of cognitive impairments.

1. *Aphasia*: brain damage that results in a deficit in communication, ranging from the inability to construct a sentence (Broca's aphasia) to the inability to comprehend speech (Wernicke's aphasia).
2. *Visual neglect*: this type of damage results in information from one hemisphere being ignored (such patients may not be aware of one-half of their visual environment).
3. *Agnosia*: this deficit consists in the patient's inability to recognize familiar objects.
4. *Amnesia*: this memory deficit can affect memories of events before the brain damage (*retrograde amnesia*) or after the damage (*anterograde amnesia*).

Hemisphere Specialization

One way to study structure–function relations of the two hemispheres is experimentally to split their processing. Split hemispheric processing can be assessed by presenting a stimulus to one hemisphere very briefly and measuring the reaction time (RT) of each hemisphere in turn: RT differences are then used as an index of the processing efficiency of each hemisphere. For example, the experimental participant may be required to name, by making a response (e.g., pressing button A for correctly spelled words and button B for non-words); this elicits a faster RT from the left hemisphere (i.e. right visual field).

Underlying such experimental studies of hemispheric processing is the assumption that each hemisphere is specialized and that this functional specialization results in faster RTs, as compared with the alternate hemisphere – of course, if enough time were allowed for the response, then hemispheric differences would disappear due to hemispheric transfer of information (via corpus callosum).

Split-brain patients: two brains, two minds

Let us imagine that it were possible to prevent the hemispheric transfer of information: this would truly separate processing in the two hemispheres. This is exactly what is done in split-brain patients. The first split-brain operations were performed in the 1950s, initially on cats and later monkeys; finally, the procedure was sufficiently perfected to be used on human beings (in 1961) for the alleviation of symptoms of severe epilepsy (to prevent the seizure travelling to the other hemisphere). The operation, known as commissurotomy, involves the cutting of the fibres (the corpus callosum) that connect the two hemispheres. Some of the most important – certainly the most spectacular – findings relating to structure–brain functions come from observations on split-brain patients.

Roger Sperry is now famous for his initial split-brain investigations; Michael Gazzaniga (1970; Gazzaniga & LeDoux, 1978), who was Sperry's doctoral student, has extended these split-brain investigations. Such patients provide a unique opportunity to study cortical specialization because information can be presented to one hemisphere only. The left brain is relatively easy to investigate because it can speak; the non-speaking right hemisphere is less easy to study and must rely upon performance measures.

Perhaps the most surprising thing about split-brain patients is how normally they behave: unless you knew, you would probably not guess from their behaviour that their brains were split in two. However, patients sometimes report a number of peculiar things which suggest that not only is their brain split, but so too is their mind. After the operation, patients sometimes report that their left hand (i.e., right hemisphere) has a 'mind of its own'. For example, patients may find themselves putting down a book held in their left hand, even if they had been reading it with great interest. This conflict occurs because the right hemisphere cannot read, therefore it finds holding the book boring, so it switches to a more interesting motor programme. A patient may go to make a cup of tea, only to find the left hand (right hemisphere) reaching for the coffee jar: in extreme instances the assistance of someone else is required to remove the object from the left hand. At other times, the left hand may make obscene gestures, embarrassing the left hemisphere. This is the

phenomenon of 'alien hand'. It is tempting to extend this observation to the everyday indecisions we all face.

In the intact brain there must be inhibitory mechanisms that suppress the wishes of one hemisphere in order to focus attention and behaviour on to a single goal: this achieves hemispheric integration. Even in split-brain patients there is integration of behaviour, but this comes from two sources: (a) *subcortical integration*; and (b) *cross-cueing*.

Subcortical integration

Only the fibres at the level of the cortex are severed; other smaller commissures and subcortical routes remain intact. These small connections allow, among other things, interhemispheric *negative priming*: slowed reaction times to a target previously ignored in the contralateral visual field. Negative priming is thought to reflect an inhibitory attentional mechanism: if a stimulus is ignored then it is more difficult – that is, reaction times are slower – to name the stimulus when it becomes a salient target in the next phase of the experiment.

Cross-cueing

The two separate brains, minds and consciousnesses work together because they share information by behavioural cues. For example, in experimental settings, the left hand may stroke the teeth of a comb so that it could be heard by the left hemisphere and interpreted as a 'comb'. Or the left hemisphere may shake the head when the right hemisphere is making a mistake when the stimulus (e.g., colour) is projected only to the right hemisphere. In everyday life, objects in environments can be seen, touched, tasted, smelled, etc. by both hemispheres.

Thus, in order to tease out the true nature of the functional (cortical) specialization in split-brain patients, special experimental procedures are needed.

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