

A detailed illustration of a human brain, viewed from the top, showing the cerebral cortex with its characteristic folds and grooves. The brain is rendered in a semi-transparent, glowing yellow and orange color, set against a dark blue background that shows the faint outline of a human head and neck. The brain is the central focus of the cover.

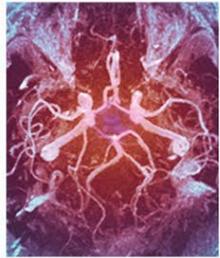
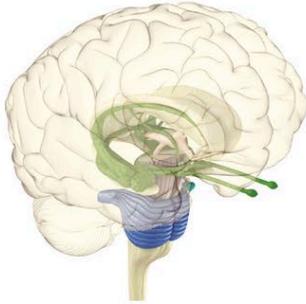
THE HUMAN BRAIN BOOK

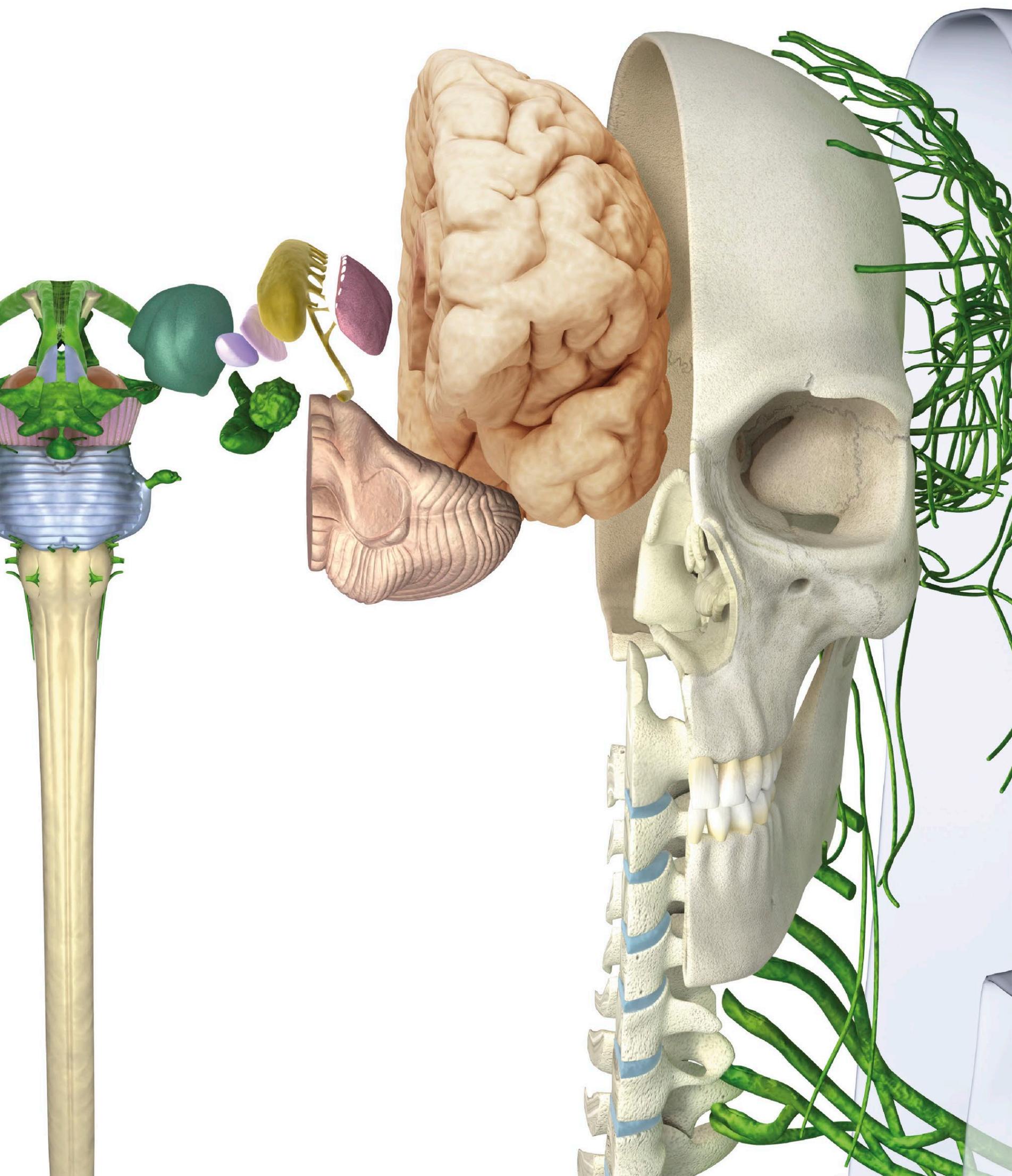
RITA CARTER



AN ILLUSTRATED GUIDE TO ITS STRUCTURE, FUNCTION, AND DISORDERS

THE HUMAN BRAIN BOOK







THE HUMAN BRAIN BOOK

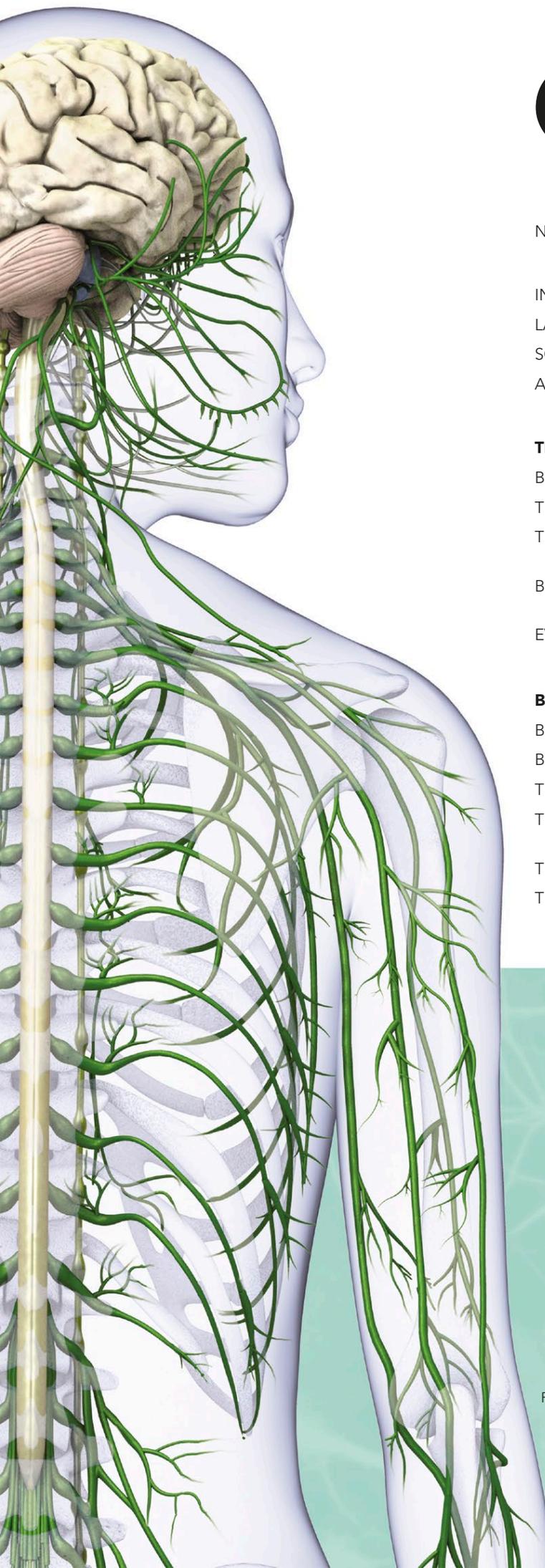
RITA CARTER

SUSAN ALDRIDGE

MARTYN PAGE

STEVE PARKER

CONSULTANTS Professor Chris Frith,
Professor Uta Frith, and Dr. Melanie Shulman



CONTENTS

NO ORDINARY ORGAN	6	THE CEREBRAL CORTEX	66
INVESTIGATING THE BRAIN	8	BRAIN CELLS	70
LANDMARKS IN NEUROSCIENCE	10	NERVE IMPULSES	72
SCANNING THE BRAIN	12	BRAIN MAPPING AND SIMULATION	74
A JOURNEY THROUGH THE BRAIN	14		
THE BRAIN AND THE BODY	36	THE SENSES	76
BRAIN FUNCTIONS	38	HOW WE SENSE THE WORLD	78
THE NERVOUS SYSTEM	40	THE EYE	80
THE BRAIN AND THE NERVOUS SYSTEM	42	THE VISUAL CORTEX	82
BRAIN SIZE, ENERGY USE, AND PROTECTION	44	VISUAL PATHWAYS	84
EVOLUTION	48	VISUAL PERCEPTION	86
		SEEING	88
BRAIN ANATOMY	50	THE EAR	90
BRAIN STRUCTURES	52	MAKING SENSE OF SOUND	92
BRAIN ZONES AND PARTITIONS	56	HEARING	94
THE NUCLEI OF THE BRAIN	58	SMELL	96
THE THALAMUS, HYPOTHALAMUS, AND PITUITARY GLAND	60	PERCEIVING SMELL	98
THE BRAIN STEM AND CEREBELLUM	62	TASTE	100
THE LIMBIC SYSTEM	64	TOUCH	102
		THE SIXTH SENSE	104
		PAIN SIGNALS	106
		EXPERIENCING PAIN	108



Penguin
Random
House

THIRD EDITION

DK DELHI

SENIOR EDITOR Rupa Rao
 ART EDITOR Sonakshi Singh
 MANAGING EDITOR Rohan Sinha
 MANAGING ART EDITOR Sudakshina Basu
 DTP DESIGNER Bimlesh Tiwary
 PICTURE RESEARCHER Sumedha Chopra
 PICTURE RESEARCH MANAGER Taiyaba Khatoun
 PREPRODUCTION MANAGER Balwant Singh
 PRODUCTION MANAGER Pankaj Sharma

DK LONDON

SENIOR EDITOR Peter Frances
 PROJECT EDITOR Ruth O'Rourke-Jones
 PROJECT ART EDITOR Francis Wong
 US EDITOR Jennette ElNaggar
 US EXECUTIVE EDITOR Lori Cates Hand
 MANAGING EDITOR Angeles Gavira Guerrero
 MANAGING ART EDITOR Michael Duffy
 JACKET DESIGN DEVELOPMENT
 MANAGER Sophia MTT
 PRODUCER, PREPRODUCTION Gillian Reid
 SENIOR PRODUCER Meskerem Berhane
 ASSOCIATE PUBLISHER Liz Wheeler
 ART DIRECTOR Karen Self
 DESIGN DIRECTOR Phil Ormerod
 PUBLISHING DIRECTOR Jonathan Metcalf

MOVEMENT AND CONTROL	110	MEMORY	154	BRAIN MONITORING AND STIMULATION	202
REGULATION	112	THE PRINCIPLES OF MEMORY	156	STRANGE BRAINS	204
THE NEUROENDOCRINE SYSTEM	114	THE MEMORY WEB	158		
PLANNING A MOVEMENT	116	LAYING DOWN A MEMORY	160	DEVELOPMENT AND AGING	206
EXECUTING A MOVEMENT	118	RECALL AND RECOGNITION	162	THE INFANT BRAIN	208
UNCONSCIOUS ACTION	120	UNUSUAL MEMORY	164	CHILDHOOD AND ADOLESCENCE	210
MIRROR NEURONS	122			THE ADULT BRAIN	212
		THINKING	166	THE AGING BRAIN	214
EMOTIONS AND FEELINGS	124	INTELLIGENCE	168	THE BRAIN OF THE FUTURE	216
THE EMOTIONAL BRAIN	126	CREATIVITY AND HUMOR	170		
CONSCIOUS EMOTION	128	BELIEF AND SUPERSTITION	172	DISEASES AND DISORDERS	220
DESIRE AND REWARD	130	ILLUSIONS	174	THE DISORDERED BRAIN	222
				DIRECTORY OF DISORDERS	224
THE SOCIAL BRAIN	132	CONSCIOUSNESS	176		
SEX, LOVE, AND SURVIVAL	134	WHAT IS CONSCIOUSNESS?	178	GLOSSARY	250
EXPRESSION	136	LOCATING CONSCIOUSNESS	180	INDEX	256
THE SELF AND OTHERS	138	ATTENTION AND CONSCIOUSNESS	182	ACKNOWLEDGMENTS	264
THE MORAL BRAIN	140	THE IDLING BRAIN	184		
		ALTERING CONSCIOUSNESS	186		
LANGUAGE AND COMMUNICATION	142	SLEEP AND DREAMS	188		
GESTURES AND BODY LANGUAGE	144	TIME	190		
THE ORIGINS OF LANGUAGE	146	THE SELF AND CONSCIOUSNESS	192		
THE LANGUAGE AREAS	148	THE INDIVIDUAL BRAIN	194		
A CONVERSATION	150	NATURE AND NURTURE	196		
READING AND WRITING	152	INFLUENCING THE BRAIN	198		
		PERSONALITY	200		

FIRST EDITION

SENIOR EDITOR Peter Frances
 SENIOR ART EDITOR Maxine Lea
 PROJECT EDITORS Nathan Joyce,
 Ruth O'Rourke, Miezán van Zyl
 EDITORS Salima Hirani, Katie John,
 Rebecca Warren
 PROJECT ART EDITORS Alison Gardner,
 Siân Thomas, Francis Wong
 DESIGNER Riccie Janus
 EDITORIAL ASSISTANT Elizabeth Munsey
 INDEXER Hilary Bird
 PROOFREADER Polly Boyd
 PICTURE RESEARCHER Liz Moore
 JACKET DESIGNER Duncan Turner
 SENIOR PRODUCTION CONTROLLER
 Inderjit Bhullar
 PRODUCTION EDITOR Tony Phipps

CREATIVE TECHNICAL SUPPORT
 Adam Brackenbury, John Goldsmid
 MANAGING EDITOR Sarah Larter
 SENIOR MANAGING ART EDITOR Phil Ormerod
 PUBLISHING MANAGER Liz Wheeler
 REFERENCE PUBLISHER Jonathan Metcalf
 ART DIRECTOR Bryn Walls
 ILLUSTRATORS Medi-Mation, Peter Bull Art Studio
 This American Edition, 2019
 First American Edition, 2009
 Published in the United States by DK Publishing
 345 Hudson Street, New York, New York 10014
 Copyright © 2009, 2014, 2019 Dorling Kindersley Limited
 DK, a Division of Penguin Random House LLC

19 20 21 22 23 10 9 8 7 6 5 4 3 2 1

001-306003-Jan/2019

All rights reserved.

Without limiting the rights under the copyright reserved above, no part of this publication may be reproduced, stored in or introduced into a retrieval system, or transmitted, in any form, or by any means (electronic, mechanical,

photocopying, recording, or otherwise), without the prior written permission of the copyright owner. Published in Great Britain by Dorling Kindersley Limited.

A catalog record for this book is available from the Library of Congress.

ISBN 978-1-4654-7954-9

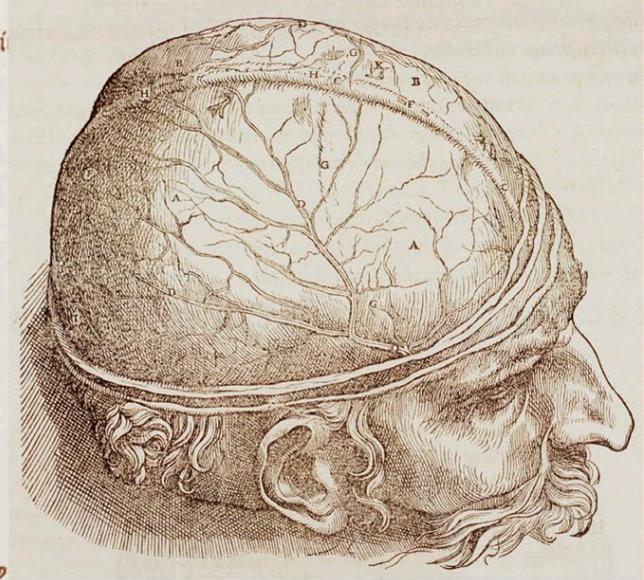
DK books are available at special discounts when purchased in bulk for sales promotions, premiums, fund-raising, or educational use. For details, contact: DK Publishing Special Markets, 345 Hudson Street, New York, New York 10014
 SpecialSales@dk.com

The Human Brain Book provides information on a wide range of medical topics, and every effort has been made to ensure that the information in this book is accurate. The book is not a substitute for medical advice, however, and you are advised always to consult a doctor or other health professional on personal health matters.

Printed in China

A WORLD OF IDEAS:
 SEE ALL THERE IS TO KNOW

www.dk.com



NO ORDINARY ORGAN

The human brain is like nothing else. As organs go, it is not especially prepossessing—3lb (1.4kg) or so of rounded, corrugated flesh with a consistency somewhere between jelly and cold butter. It doesn't expand and shrink like the lungs, pump like the heart, or secrete visible material like the bladder. If you sliced off the top of someone's head and peered inside, you wouldn't see much happening at all.

SEAT OF CONSCIOUSNESS

Given this, it is perhaps not surprising that for centuries the contents of our skulls were regarded as relatively unimportant. When they mummified their dead, the ancient Egyptians scooped out the brains and threw them away, yet carefully preserved the heart. The Ancient Greek philosopher, Aristotle, thought the brain was a radiator for cooling the blood. René Descartes, the French scientist, gave it a little more respect, concluding that it was a sort of antenna by which the spirit might commune with the body. It is only now that the full wonder of the brain is being realized.

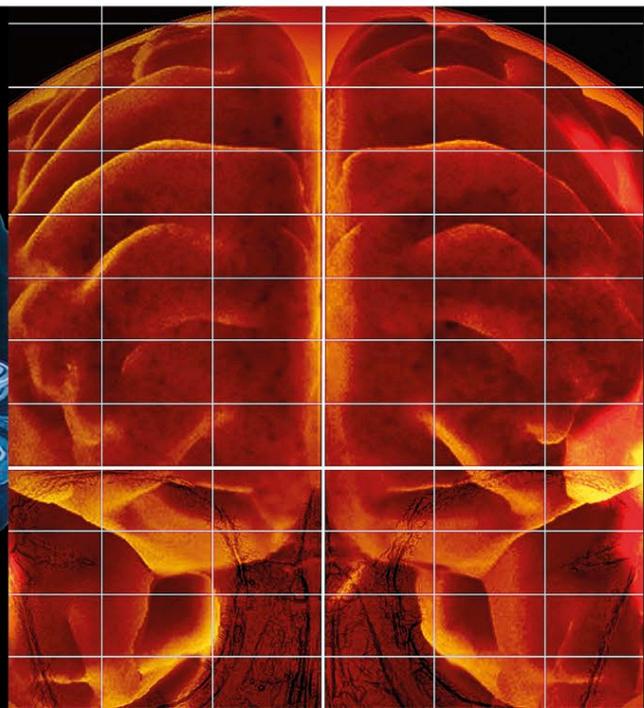
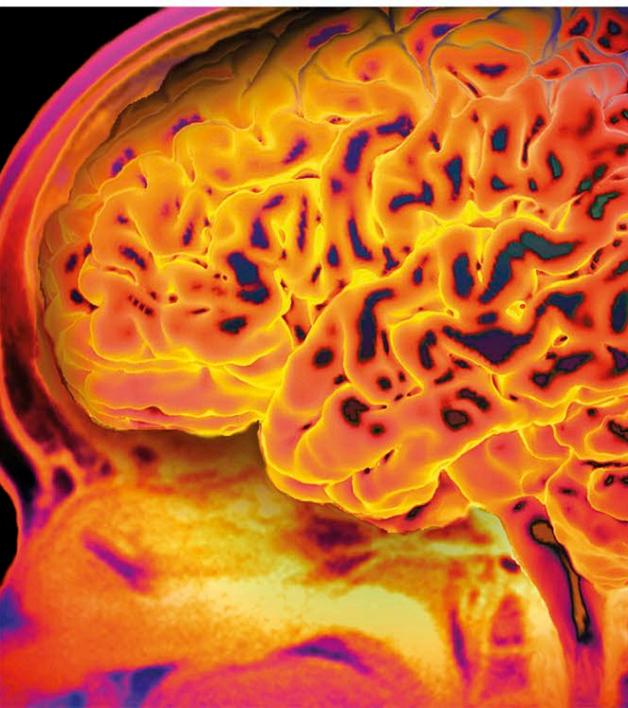
The most basic function of the brain is to keep the rest of the body alive. Among your brain's 100 billion neurons, some regulate your breathing, heartbeat, and blood pressure and others control hunger, thirst, sex drive, and sleep cycle. In addition to this, the brain

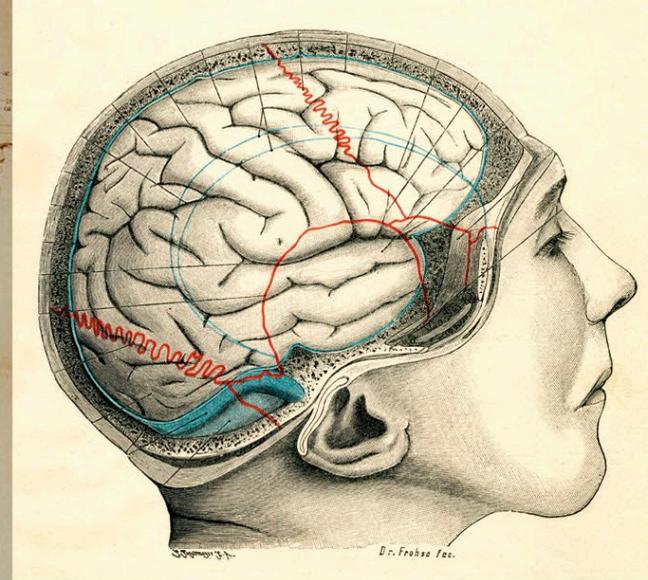
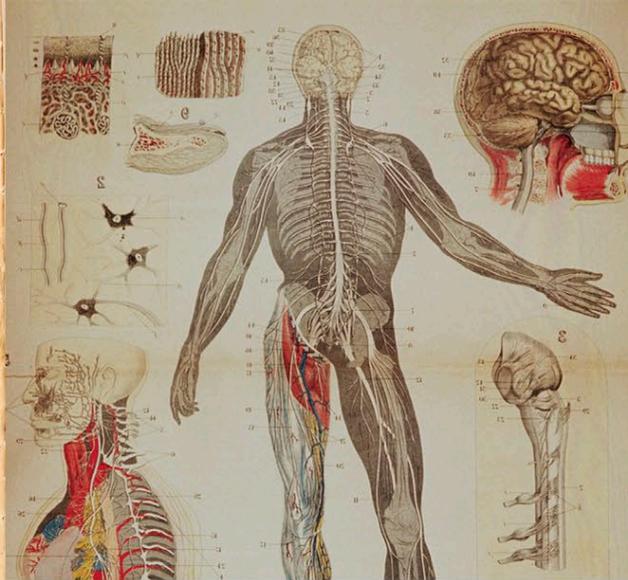
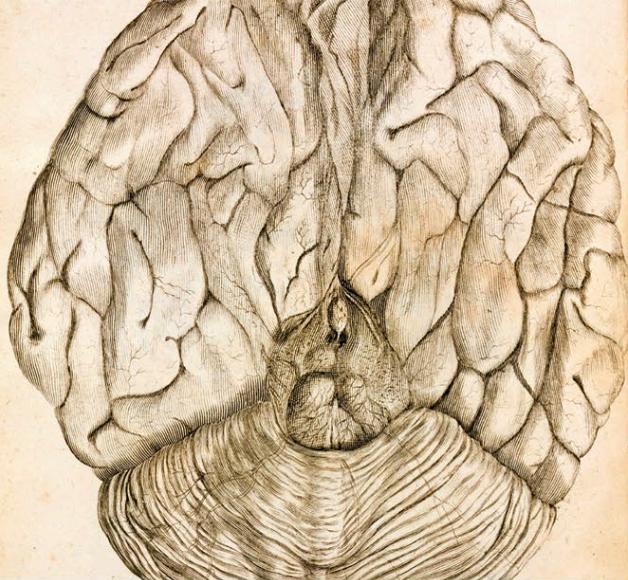
generates the emotions, perceptions, and thoughts that guide your behavior. Then it directs and executes your actions. Finally, it is responsible for the conscious awareness of the mind itself.

THE DYNAMIC BRAIN

Until about 100 years ago, the only evidence that brain and mind were connected was obtained from "natural experiments"—accidents in which head injuries created aberrations in their victims' behavior. Dedicated physicians mapped out areas of the cerebral landscape by observing the subjects of such experiments while they were alive—then matching their deficits to the damaged areas of their brains. It was slow work because the scientists had to wait for their subjects to die before they could look at the physiological evidence. As a result, until the early 20th century, all that was known about the physical basis of the mind could have been contained in a single volume.

Since then, scientific and technological advances have fueled a neuroscientific revolution. Powerful microscopes made it possible to look in detail at the brain's intricate anatomy. A growing understanding of electricity allowed the dynamics of the brain to be recognized and then, with the advent of electroencephalography (EEG), to be observed and measured. Finally, the arrival of





functional brain imaging machines allowed scientists to look inside the living brain and see its mechanisms at work. In the last 20 years, positron emission tomography (PET), functional magnetic resonance imaging (fMRI), and, most recently, magnetic encephalography (MEG) have among them produced an ever more detailed map of the brain's functions.

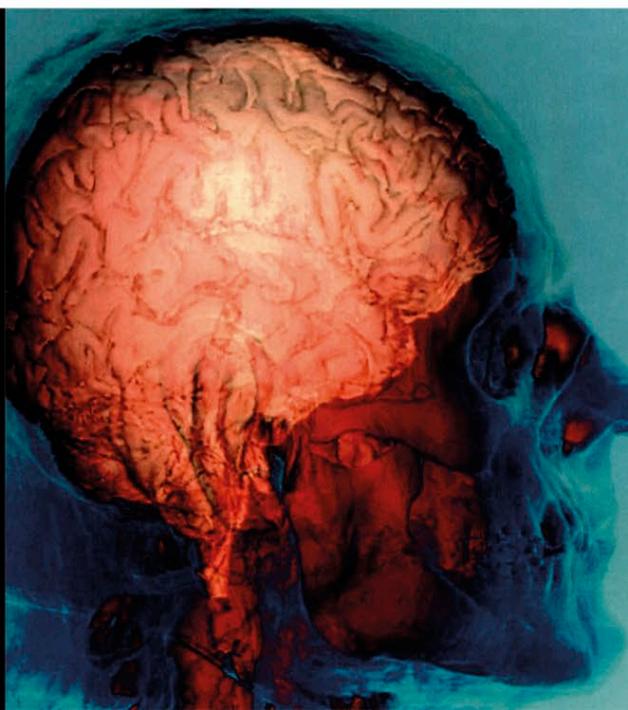
LIMITLESS LANDSCAPE

Today we can point to the circuitry that keeps our vital processes going, the cells that produce our neurotransmitters, the synapses where signals leap from cell to cell, and the nerve fibers that convey pain or move our limbs. We know how our sense organs turn light rays and sounds waves into electrical signals, and we can trace the routes they follow to the specialized areas of cortex that respond to them. We know that such stimuli are weighed, valued, and turned into emotions by the amygdala—a tiny nugget of tissue that punches well above its weight. We can see the hippocampus retrieve a memory, or watch the prefrontal cortex make a moral judgment. We can recognize the nerve patterns associated with amusement, empathy—even the thrill of *schadenfreude* at the sight of an adversary suffering defeat. More than just a map, the picture emerging from imaging

studies reveals the brain to be an astonishingly complex, sensitive system in which each part affects almost every other. “High level” cognition performed by the frontal lobes, for instance, feeds back to affect sensory experience—so what we see when we look at an object is shaped by expectation as well as by the effect of light hitting the retina. Conversely, the brain's most sophisticated products can depend on its lowliest mechanisms. Intellectual judgments, for example, are driven by the body reactions that we feel as emotions, and consciousness can be snuffed out by damage to the humble brainstem. To confuse things further, the system doesn't stop at the neck but extends to the tips of your toes. Some would argue it even goes beyond—to encompass other minds with which it interacts.

Neuroscientific investigation of the brain is very much a work in progress and no one knows what the finished picture will look like. It may be that the brain is so complicated that it can never understand itself entirely. So this book cannot be taken as a full description of the brain. It is a single view, from bottom to top, of the human brain as we know it today—in all its beauty and complexity. Be amazed.

Rita Carter

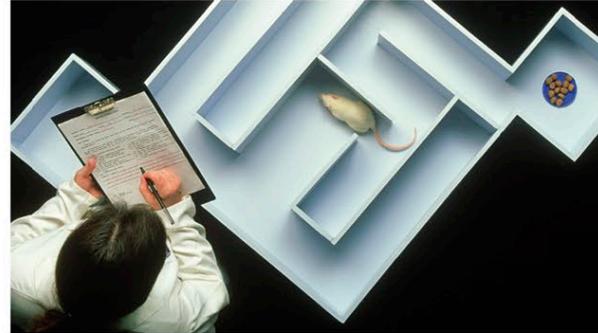


INVESTIGATING THE BRAIN

THE BRAIN IS THE LAST OF THE HUMAN ORGANS TO GIVE UP ITS SECRETS. FOR A LONG TIME, PEOPLE WERE NOT EVEN ABLE TO UNDERSTAND WHAT THE BRAIN IS FOR. THE DISCOVERY OF ITS ANATOMY, FUNCTIONS, AND PROCESSES HAS BEEN A LONG AND SLOW JOURNEY ACROSS THE MILLENNIA, AS HUMAN KNOWLEDGE ABOUT THIS MYSTERIOUS ORGAN HAS DEVELOPED AND ACCUMULATED.

EXPLORING THE BRAIN

The brain is particularly difficult to investigate because its structures are minute and its processes cannot be seen with the naked eye. The problem is compounded by the fact that its most interesting product, consciousness, does not feel like a physical process, so there was no obvious reason for our distant ancestors to associate it with the brain. Nevertheless, over the centuries, philosophers and physicians built up an understanding of the brain and, in the last 25 years with the advent of brain-imaging techniques, neuroscientists have created a detailed map of what was once an entirely mysterious territory.



USING RATS
The brains of rats are very similar to human brains. Until imaging techniques were developed, the only way scientists were able to look directly at brain tissue was by using the brains of rats and other non-human animals.



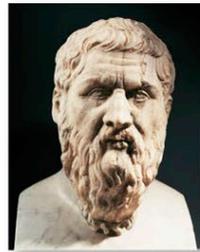
PAPYRUS

1700 BCE

Egyptian papyrus gives a careful description of the brain, but Egyptians do not rate this organ highly; unlike other organs, it is removed and discarded before mummification, suggesting that it was not considered to be of any use in future incarnations.

4000 BCE

Early Sumerian writing notes the euphoric effect of poppy seeds.



PLATO

387 BCE

The Greek philosopher Plato teaches at Athens; he believes the brain is the seat of mental processes.

450 BCE

Early Greeks begin to recognize the brain as the seat of human sensation.



DRAWING THE BRAIN

1543

Andreas Vesalius, a European physician, publishes the first "modern" anatomy, with detailed drawings of the human brain.

1664

Oxford physiologist Thomas Willis publishes the first brain atlas, locating various functions in separate brain "modules."



BRAIN ATLAS

1774

German physician Franz Anton Mesmer introduces "animal magnetism," later called hypnosis.

1848

Phineas Gage has his brain pierced by an iron rod (see p.141).

2500 BCE

Trepanation (boring holes into the skull) is a common surgical procedure across many cultures, possibly used for relieving brain disorders such as epilepsy, or for ritual or spiritual reasons.



TREPANNING

335 BCE

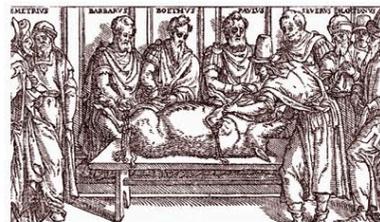
Greek philosopher Aristotle restates the ancient belief that the heart is the superior organ; the brain, a radiator to stop the body from overheating.



ARISTOTLE

170 BCE

Roman physician Galen theorizes that human moods and dispositions are due to the four "humors" (liquids that are held in the brain's ventricles). The idea persists for more than 1,000 years. Galen's anatomical descriptions, used by generations of physicians, were based mainly on work on monkeys and pigs.



GALEN AT WORK



RENÉ DESCARTES

1649

French philosopher René Descartes describes the brain as a hydraulic system that controls behavior. "Higher" mental functions are generated by a spiritual entity, however, which interacts with the body via the pineal gland.

1791

Luigi Galvani, an Italian physicist, discovers the electrical basis of nervous activity by making frogs' legs twitch.



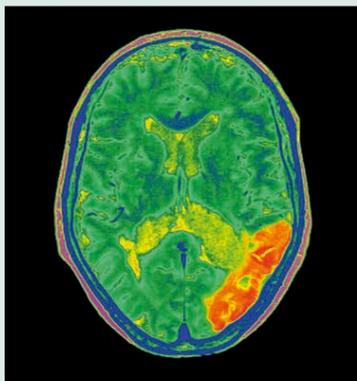
LUIGI GALVANI

1849

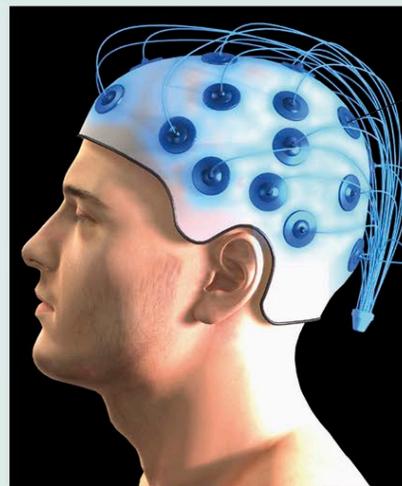
German physicist Hermann von Helmholtz measures the speed of nerve conduction and subsequently develops the idea that perception depends upon "unconscious inferences."

THE ADVENT OF IMAGING TECHNIQUES

Scientists were unable to find out much about the workings of the brain until relatively recently. The only way they were able to match functions such as sight, emotion, or speech to the locations in the brain in which they are controlled was to find a person in whom a faculty was disturbed due to injury, and then wait until they were dead in order to look at the location and extent of the brain damage. Otherwise, scientists could only guess at what was happening to the brain by observing people's behavior. Today, modern imaging techniques such as functional MRI and EEG (see p.12) allow neuroscientists to see the electrical activity in the brain as a person carries out various tasks or thought processes. This allows them to link types of actions, emotions, and so on, to specific types of activity in the brain. The freedom to observe the brain that imaging techniques have afforded has allowed for an explosion of knowledge within neuroscience, and has deepened our understanding of the brain and how it works.



MAGNETIC RESONANCE IMAGING
Brain scans can reveal damaged tissue—the red area in the MRI scan above indicates damage caused by a stroke.



Electrode "cap"

ELECTRODES
Neural activity can be measured by attaching electrodes to the scalp. These pick up electrical activity in the brain and transform it into a digital record.

1889

Santiago Ramón y Cajal proposes that nerve cells are independent elements and the basic units of the brain in *The Neuron Doctrine*. He wins the Nobel Prize in 1906.

Circa 1900

Sigmund Freud abandons an early career in neurology to study psychodynamics. The success of Freudian psychoanalysis eclipsed physiological psychiatry for half a century.



SIGMUND FREUD

1934

Portuguese neurologist Egas Moniz carries out the first leucotomy operations (later known as lobotomies, see p.11). He also invented angiography, one of the first techniques that allowed scientists to make images of the brain.



EGAS MONIZ

1981

Roger Wolcott Sperry is awarded the Nobel Prize for his work on the different functions of the two brain hemispheres (see pp.11 and 205).

2013

The United States and European Union start human brain simulation projects. The Connectome, a global cooperative endeavor, delivers its first charts of the connections between neurons.

1862–74

Broca and Wernicke (see p.10) discover the two main language areas of the brain.

1874

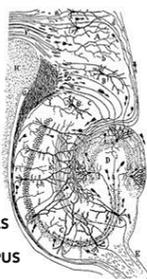
Carl Wernicke publishes on aphasia (language disorders after brain damage).

1859

Charles Darwin publishes *On the Origin of Species*.

1906

Santiago Ramón y Cajal describes how nerve cells communicate.



NERVE CELLS IN RODENT HIPPOCAMPUS

1919

Irish neurologist Gordon Morgan Holmes localizes vision to the striate cortex (the primary visual cortex).

1953

Brenda Milner describes patient HM (see p.157), who suffers memory loss after hippocampal surgery.

1983

Benjamin Libet writes on the timing of conscious volition (see p.11).

1900

2000

1850

Franz Joseph Gall finds phrenology (see p.10), which attributes different personality traits to specific areas of the head.

1906

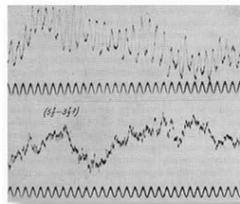
Alois Alzheimer describes presenile degeneration (see p.231).

1914

British physiologist Henry Hallett Dale isolates acetylcholine, the first of the neurotransmitters (see p.73) to be discovered. He wins the Nobel Prize in 1936.

1924

The first electroencephalograms are produced by Hans Berger.



ELECTROENCEPHALOGRAPHY

1970–80

Brain scanning is developed: PET, SPECT, MRI, and MEG all emerge during this decade.

1973

Timothy Bliss and Terje Lomo describe long-term potentiation (see p.158).

1991

Mirror neurons are discovered by Giacomo Rizzolatti in Parma (see pp.11 and 122–23).

1873

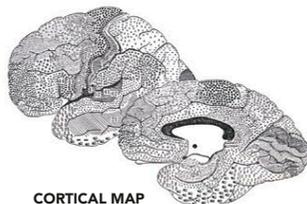
Italian scientist Camillo Golgi publishes the silver nitrate method, making it possible to see nerves in their entirety. He wins the Nobel Prize in 1906.



NERVE CELLS

1909

Korbinian Brodmann describes 52 discrete cortical areas based on neural structure. These areas are still used today (see p.67).



CORTICAL MAP

1957

W. Penfield and T. Rasmussen devise a motor and sensory homunculus (see pp.10 and 103).



EARLY MAGNETIC IMAGING

LANDMARKS IN NEUROSCIENCE

MOST OF THE KNOWLEDGE WE HAVE ABOUT THE BRAIN HAS BEEN GATHERED BY SLOW, PAINSTAKING RESEARCH INVOLVING LARGE TEAMS OF PEOPLE. HOWEVER, OCCASIONALLY THE HISTORY OF NEUROSCIENCE HAS BEEN PUNCTUATED BY DRAMATIC DISCOVERIES OR IDEAS, OFTEN ARISING FROM THE WORK OF A SINGLE SCIENTIST. SOME OF THESE SUBSEQUENTLY PROVED TO BE VALUABLE BREAKTHROUGHS WHILE OTHERS, ALTHOUGH INFLUENTIAL, PROVED TO BE DEAD ENDS.

PHRENOLOGY Franz Joseph Gall

Gall thought that personality could be read by feeling the contours of the skull. He theorized that various faculties were localized in the brain and that the strongest were correspondingly large, making the skull bulge measurably. It was hugely popular in nineteenth-century America and Europe—nearly every town had a phrenology institute. Although nonsense, Gall’s idea that brain functions are localized has turned out to be largely true. Imaging research aimed at locating brain functions is often called “modern phrenology.”

PHRENOLOGY HEAD
Models such as this claimed to show the bulges on the skull that revealed a person’s character. Categories included “blandness” or “benevolence.”



THE MAN WHO LOST HIMSELF Phineas Gage

This polite, well-liked American railroad foreman changed dramatically, becoming “grossly profane,” after an accident destroyed part of his brain (see p.141). His case was the first to show that faculties such as social and moral judgment can be localized to the frontal lobes.

FATEFUL INJURY
This reconstruction of Phineas Gage’s skull shows how an iron rod damaged the frontal lobes of his brain.



PAUL BROCA

CARL WERNICKE

LANGUAGE AREAS Broca and Wernicke

In 1861, French physician Paul Broca described a patient who he named “Tan,” as it was the only word “Tan” could say. When Tan died, Broca examined his brain and found damage to part of the left frontal cortex. This part of the brain became “Broca’s Area” (see p.148). In 1876, German neurologist Carl Wernicke found that damage to a different part of the brain (which became known as “Wernicke’s Area”) also caused language problems. These two scientists were the first to clearly define functional areas of the brain.

EARLY BRAIN IMPLANT José Delgado

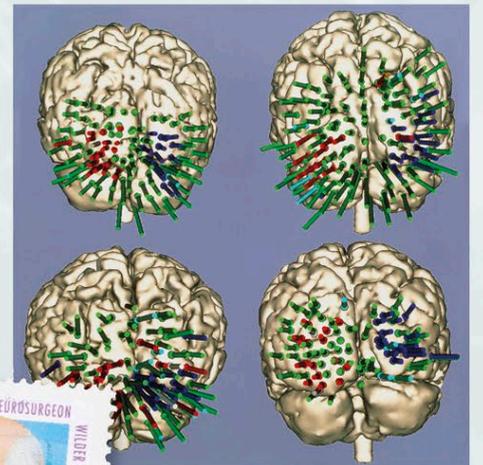
Spanish neurologist Dr. José Delgado invented a brain implant that could be remotely controlled by radio waves. He found that animal and human behavior could be controlled by pressing a button. In a famous experiment, conducted in 1964, Delgado faced a charging bull, bringing it to a halt at his feet by activating the implant in its brain. In another, he put a device in the brain of a chimp that was bullying its mate. He put the control in the cage where the victim chimp used it to “turn off” the bully’s bad behavior.



DELGADO AND THE BULL

MAPPING THE BRAIN Wilder Penfield

The first detailed maps of human brain function were made by Canadian brain surgeon Wilder Penfield. He worked with patients undergoing surgery to control epilepsy. While the brain was exposed, and the patient conscious, Penfield probed the cortex with an electrode and noted the responses of the patient as he touched each part. Penfield’s work was the first to reveal the role of the temporal lobe in recall and map the areas of the cortex that control movement and provide bodily sensations.



MODERN MAPPING
Today advanced imaging (see above) allows neural activity to be matched to mental tasks. However, much of the basic map was established by Penfield half a century earlier.



CANADIAN STAMP

LOBOTOMY

The first lobotomies were performed in the 1890s, but they only took off in the 1930s when the Portuguese neurosurgeon Egas Moniz found that cutting the nerves from the frontal cortex to the thalamus relieved psychotic symptoms in some patients. Moniz's work was picked up by US surgeon Walter Freeman, who invented the "ice pick lobotomy." From 1936 until the 1950s, he advocated lobotomy to cure for a range of problems, and 40,000 to 50,000 patients were lobotomized. The operation was overused and is now thought abhorrent. However, in many cases it eased suffering: a follow-up of patients in the UK found 41 percent were "recovered" or "greatly improved," 28 percent "minimally improved," 25 percent had "no change," 4 percent had died, and 2 percent were worse off.



TREPANATION

The practice of drilling holes in the head has been used since prehistoric times as a treatment for a vast array of illnesses. The modern equivalent, craniotomy, is carried out to relieve pressure within the skull.



"ICE PICK" LOBOTOMY

Walter Freeman, above, found he could perform a lobotomy under local anesthetic by hammering an ice pick above each eye of a patient and swishing the device back and forth like a windshield wiper.



ICE PICK

MAKING MEMORIES

Henry G. Molaison

In 1953, aged 27, "HM" underwent an operation in the US, to stem severe epilepsy. The surgeons, then unaware of the functions of the hippocampus, took out a large area of that part of his brain (see p.159). When he came round, he was unable to lay down new memories and remained so for the rest of his life. The tragic accident demonstrated the crucial role of the hippocampus in recall.



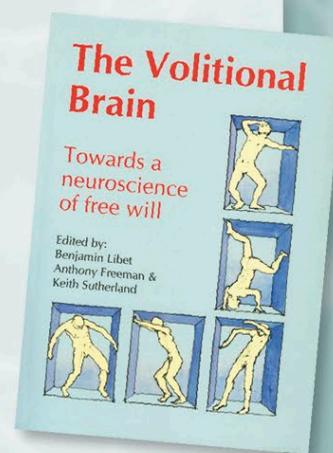
FROZEN IN TIME

Henry G. Molaison—generally known only as "HM"—was one of the most studied patients in the history of modern medicine.

CONSCIOUS DECISIONS

Benjamin Libet

A series of ingenious experiments by US neuroscientist Benjamin Libet (see p.191) in the early 1980s demonstrated that what we think are conscious "decisions" to act are actually just recognition of what the unconscious brain is already doing. Libet's experiments have profound philosophical implications because, on the face of it, the results suggest that we do not have a conscious choice about what we do, and therefore cannot consider ourselves to have free will.



INVESTIGATING FREE WILL

SPLIT-BRAIN EXPERIMENTS

Roger Sperry

Neurobiologist Roger Sperry conducted the split-brain experiments (see p.204) on people whose brain hemispheres were surgically separated in the course of treatment for epilepsy. They showed that, under certain conditions, each hemisphere could hold different thoughts and intentions. This raised the profound question of whether a person has a single "self."

ROGER SPERRY RECEIVES THE NOBEL PRIZE IN 1981



MIRROR NEURONS

Mirror neurons (see pp.122–23) were discovered in 1991—by accident. A group of researchers in Italy, led by Giacomo Rizzolatti, were monitoring neural activity in the brains of monkeys as they made reaching movements. One day a researcher inadvertently mimicked the monkey's movement while it was watching, and found that the neural activity in the monkey's brain that sparked up in response to the sight was identical to the activity that occurred when the monkey made the action itself. Mirror neurons are thought by some to be the basis of theory of mind, mimicry, and empathy.

MIMICKING MACAQUE

Mirror neurons produce automatic mimicry by producing a similar state in an observer's brain to the state of the person they are watching.



SCANNING THE BRAIN

BRAIN IMAGING TECHNIQUES CAN BE DIVIDED INTO TWO DIFFERENT TYPES: ANATOMICAL IMAGING, WHICH GIVES INFORMATION ABOUT THE STRUCTURE OF THE BRAIN, AND FUNCTIONAL SCANNING, WHICH ALLOWS RESEARCHERS TO SEE HOW THE BRAIN WORKS. USED TOGETHER, THESE TECHNIQUES HAVE REVOLUTIONIZED NEUROSCIENCE.

A WINDOW ON THE BRAIN

The structure of the brain is well known, but until recently the way it created thoughts, emotions, and perceptions could only be guessed at. Imaging technology has now made it possible to look inside a living brain and see it at work. The brain works



PET SCANNER
Positron emission tomography (PET) scanners detect signals from radioactive markers in tissues to show activity in the brain.

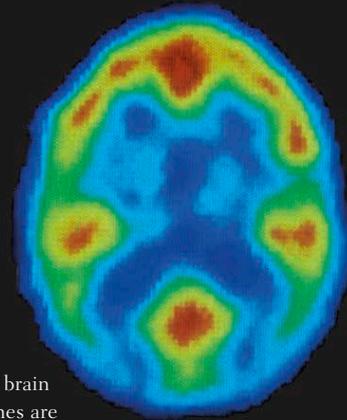
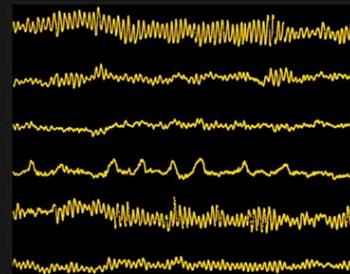
by generating tiny electrical charges. Functional imaging reveals which areas are most active. This may be done by measuring electrical activity directly (EEG), picking up magnetic fields created by electrical activity (MEG), or measuring metabolic side effects such as alterations in glucose absorption (PET) and blood flow (fMRI).

FUNCTION

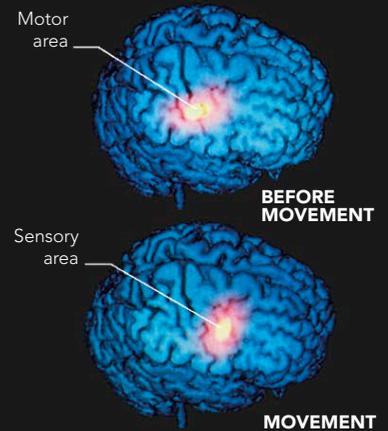
The brain is composed of modules that are specialized to do specific things. Functional brain imaging is largely about identifying which ones are most concerned with doing what. This has allowed neuroscientists to build a detailed map of brain functions. We now know where perceptions, language, memory, emotion, and movement occur. By showing how various functions work together, imaging also gives us a glimpse into some of the most sophisticated aspects of human psychology. For example, observing a person's brain making a decision, we see that apparently rational decisions are driven by the emotional brain. Imaging the brains of master chess players shows why expertise depends on practice. Watching the brain of a person seeing a frightened face shows that emotion is contagious.

BRAIN WAVES

Electroencephalographs (EEGs) show electrical activity caused by nerve cells firing. They record distinct "brain waves," which reflect the speed of firing in different states of mind.



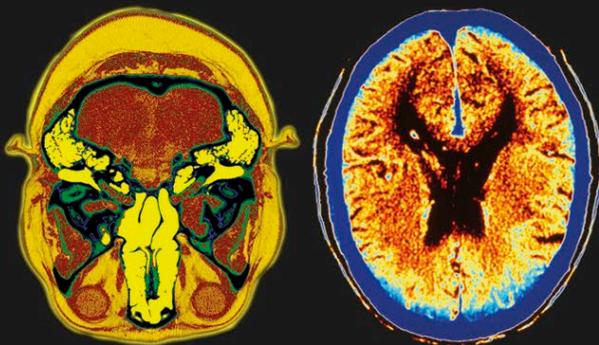
PET SCANS
These scans involve injecting a volunteer with a radioactive marker that attaches to glucose in the brain. Areas of high activity (red) attract glucose for fuel. The marker dye shows which parts of the brain are firing.



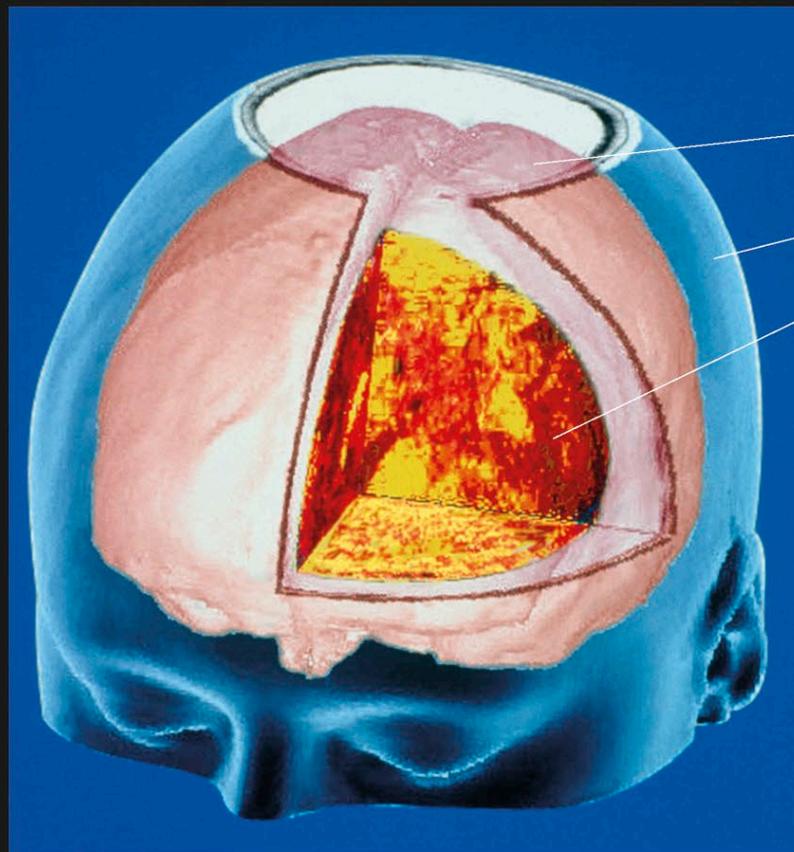
REAL-TIME ACTIVITY
Magnetoencephalography (MEG) picks up magnetic traces of brain activity. It is poor at showing where activity occurs, but good at pinpointing timing. Here, a brain plans a finger movement, then 40 milliseconds later its activity shifts as the movement is made.

ANATOMY

The brain looks very different according to how it is viewed. Computed tomography (CT) imaging combines the use of a computer and fine X-rays to produce multiple "slices" of the body. It allows you to see normally obscured body tissues, such as the inside of the brain, from any angle or level, with the delicate inner structures thrown into clear relief. Artificial coloring of the areas further distinguishes one part from another. CT scans are purely structural: they show the form of the organ but not how it works. They are very good at showing contrast between soft tissues and bone, and are therefore useful in diagnosing tumors and blood clots.



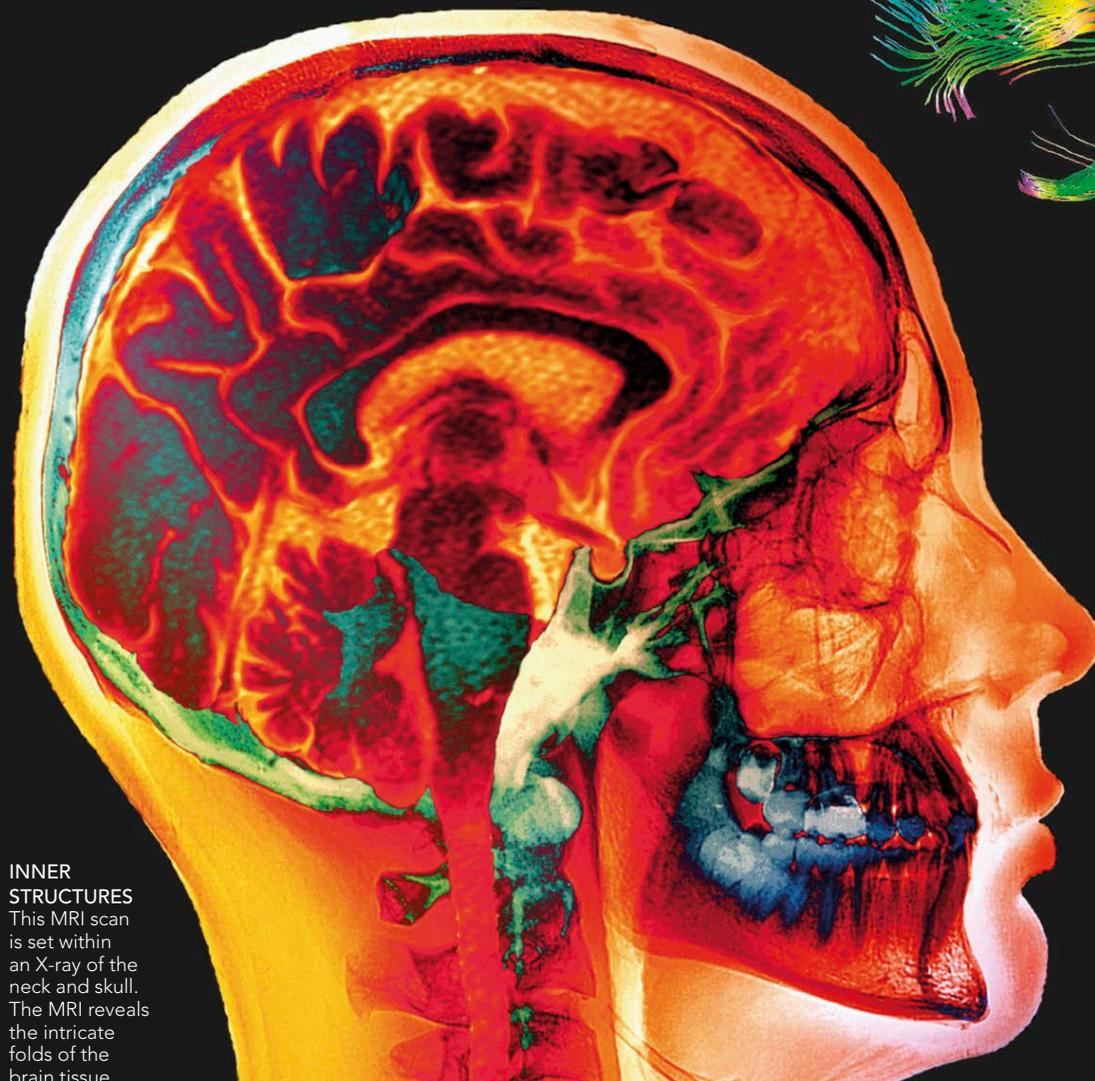
STRUCTURAL DETAILS
These CT images show different tissues in detail. The image on the left shows the cerebellum and eyeballs in red, the bones in blue and green, and the sinuses and ear cavities in bright yellow. The image on the right shows a healthy brain (front at bottom). The black areas are the fluid-filled ventricles.



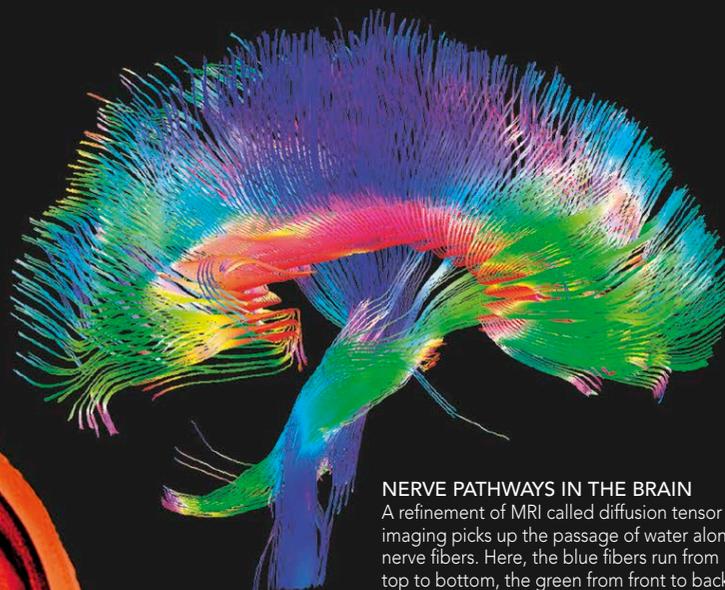
3-D BRAIN
CT allows pictures of brains to be displayed in three dimensions, and "sliced" to reveal the inner workings. Here, the front right quarter of the brain's coverings and surface are cut away to reveal the tissues beneath.

MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging (MRI) provides a better contrast between tissue types than CT. Instead of using X-rays, it uses a powerful magnetic field, which causes hydrogen atoms in the body to realign. The nuclei of the atoms produce a magnetic field that is “read” by the scanner and turned into a three-dimensional computerized image. The brain is scanned at a rapid rate (typically once every 2–3 seconds) to produce “slices” similar to those in CT scans. Increases in neural activity cause changes in the blood flow, which alter the amount of oxygen in the area, producing a change in the magnetic signal. Functional MRI (fMRI) involves showing differing levels of electrical activity in the brain, overlaid on the anatomical details.



INNER STRUCTURES
This MRI scan is set within an X-ray of the neck and skull. The MRI reveals the intricate folds of the brain tissue.



NERVE PATHWAYS IN THE BRAIN
A refinement of MRI called diffusion tensor imaging picks up the passage of water along nerve fibers. Here, the blue fibers run from top to bottom, the green from front to back, and the red between the two hemispheres.

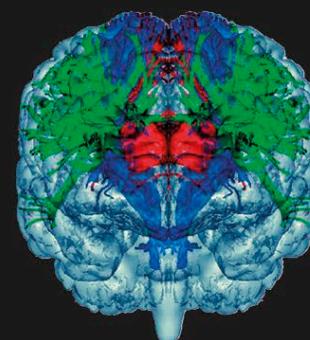
MOVEMENT

fMRI is very good at localizing brain activity. In this image (bottom of brain at top), the red area shows activity in the part responsible for moving the right hand. Each side of the body is controlled by the opposite hemisphere of the brain.



FIBER DETAIL

This diffusion tensor image shows another view of the nerve fibers. The green fibers link the various parts of the limbic system. The blue fibers run from the cerebellum, which joins onto the spine. The red fibers connect the two hemispheres.

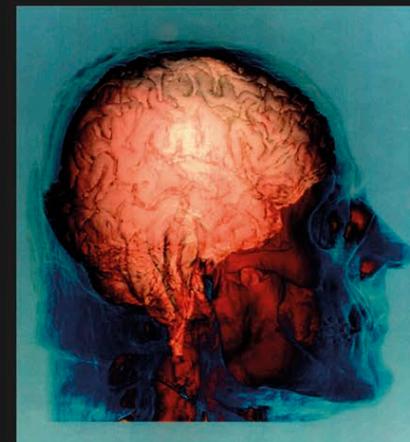


COMBINED IMAGING

Each type of imaging has its advantages. MRI is good on detail, for example, but is too slow to chart fast-moving events. EEG and MEG are fast but are not as good at pinpointing location. To get scans that show both fast processes and location, researchers use two or more methods to produce a combined image. Here (right), for example, high-resolution MRI, taking about 15 minutes to acquire, is combined with a low-resolution fMRI, which takes seconds to produce and shows the location of activity in the brain areas used in hearing language. The areas shift during a task like this that involves many aspects, and they have to work fast and in concert. The areas used in a task vary from person to person, so studies often combine data from volunteers to give an average.

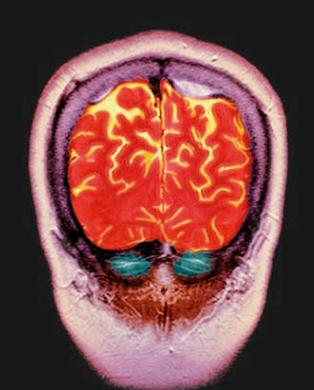
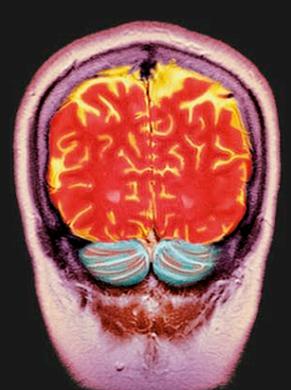
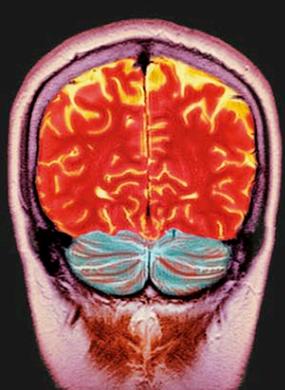
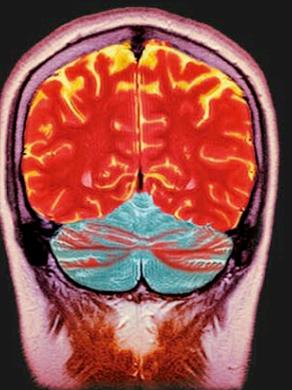
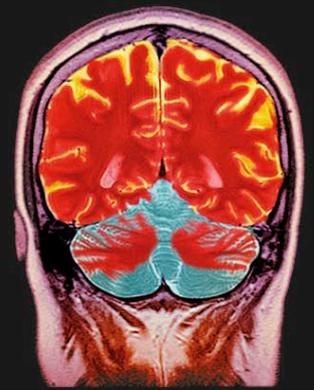
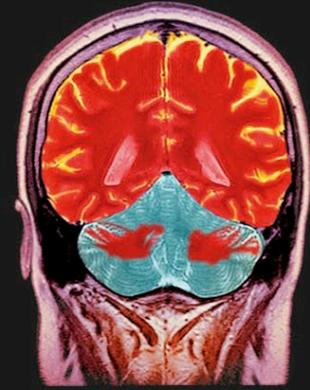
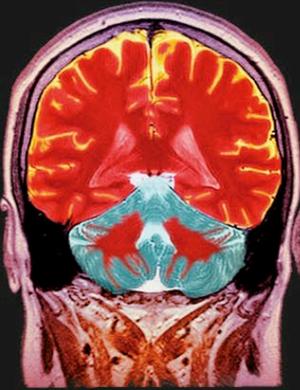
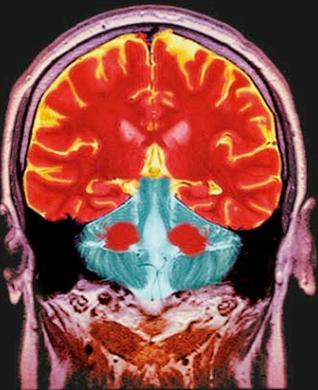
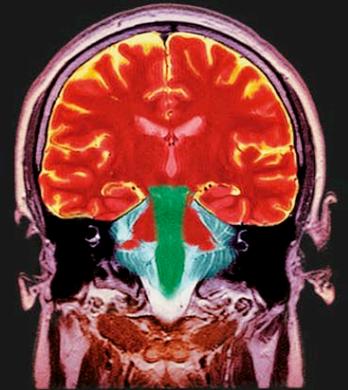
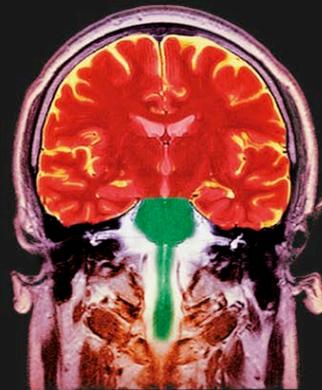
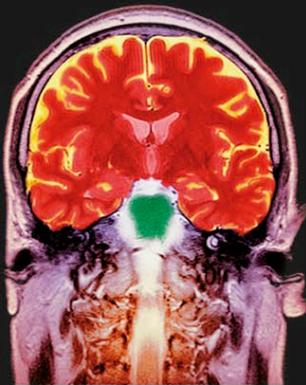
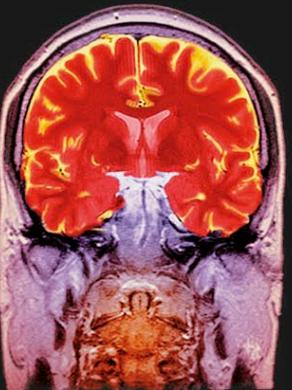
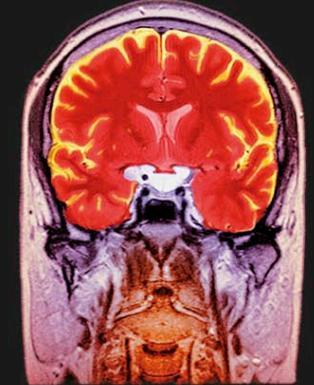
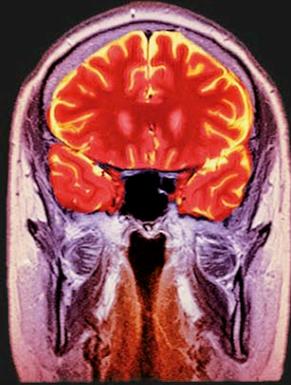
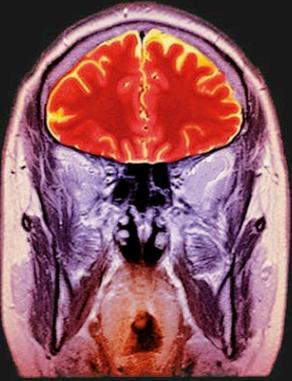
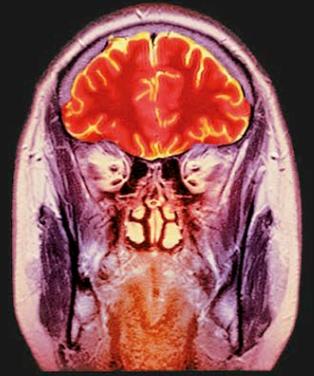
STUDYING LANGUAGE

In most people, the main language areas of the brain are located in the left hemisphere, so this area shows greater activity when a person listens to spoken words. The right hemisphere is also required for complete hearing, and for distinguishing tone and rhythm.



SLICED TOGETHER

Here, a combined CT and MRI scan shows the surface folds of the brain. It also reveals the skull bones and the top vertebrae.



A JOURNEY THROUGH THE BRAIN

THE BRAIN IS THE MOST COMPLEX ORGAN IN THE BODY AND IS PROBABLY THE MOST COMPLEX SYSTEM KNOWN TO HUMANKIND. OUR BRAIN CONTAINS BILLIONS OF NEURONS THAT ARE CONSTANTLY SENDING SIGNALS TO EACH OTHER, AND IT IS THIS SIGNALING THAT CREATES OUR MINDS. WITH THE HELP OF MODERN SCANNING TECHNOLOGY, WE NOW KNOW ABOUT BRAIN STRUCTURE IN GREAT DETAIL.

In the nineteenth century, much was learned about the structure of the brain by removing it from the body after death. Knowledge of the workings of the living human brain could only be gained by studying people with damaged brains, for example Phineas Gage (see p.141), but the precise location of this damage could not be known while the patient was still alive. Everything changed with the invention of brain scanners at the end of the twentieth century. In the following pages, we shall undertake a journey through the brain of a healthy, 55-year-old man revealed by magnetic resonance imaging (MRI). In these images, we can see the many components of the brain. We are

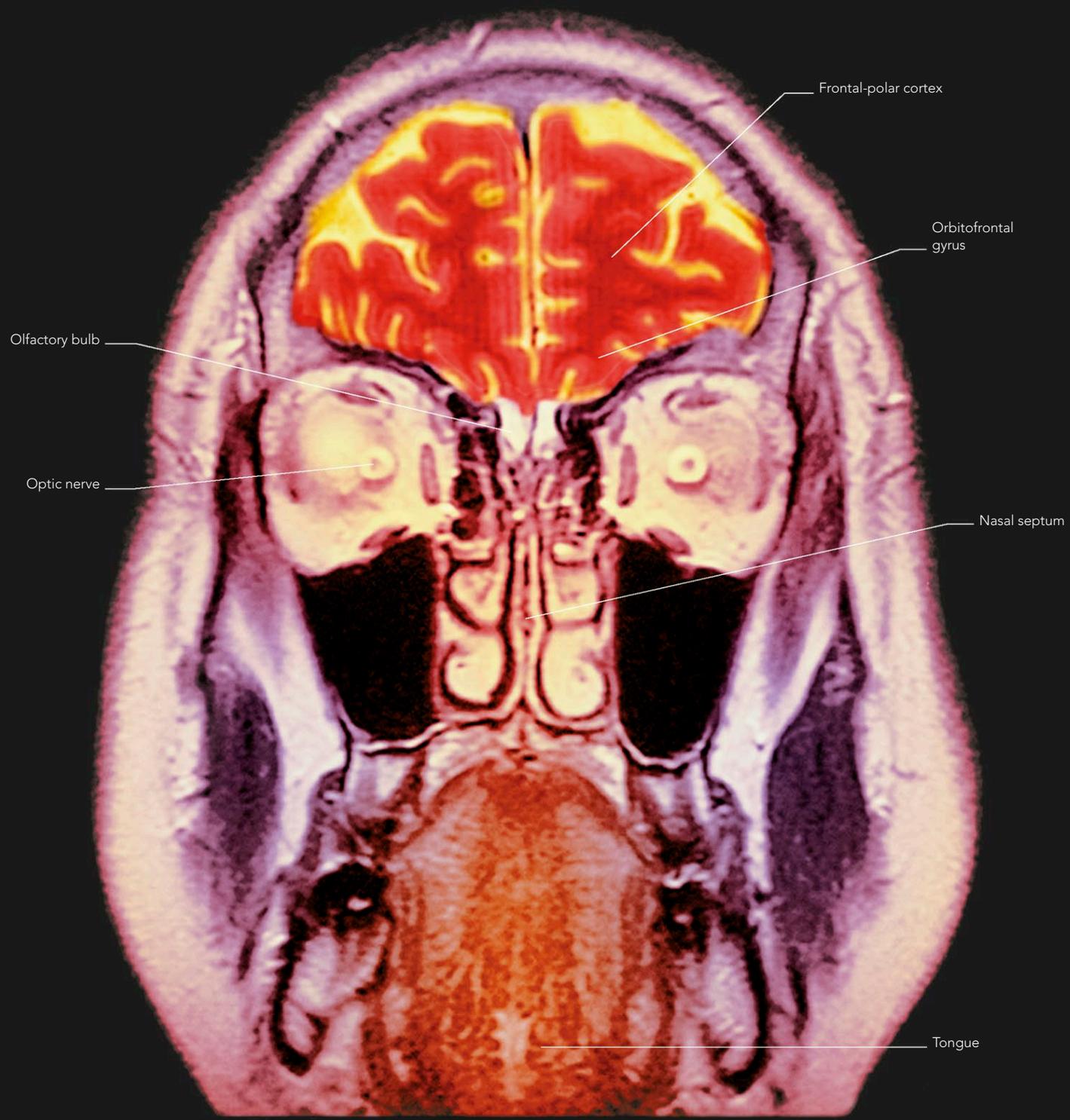
starting to understand the function of some of these, but we are only at the very beginning of this journey of understanding.

The captions that accompany the scans indicate the most likely function of various brain regions. But these regions often have many functions, and these functions depend upon interactions with other brain regions. Most structures in the brain are paired, with identical counterparts in the left and right hemispheres, so structures identified in one hemisphere are mirrored in the opposite one. The scans themselves have been colored, so that the cerebrum appears in red, the cerebellum in light blue, and the brainstem in green.

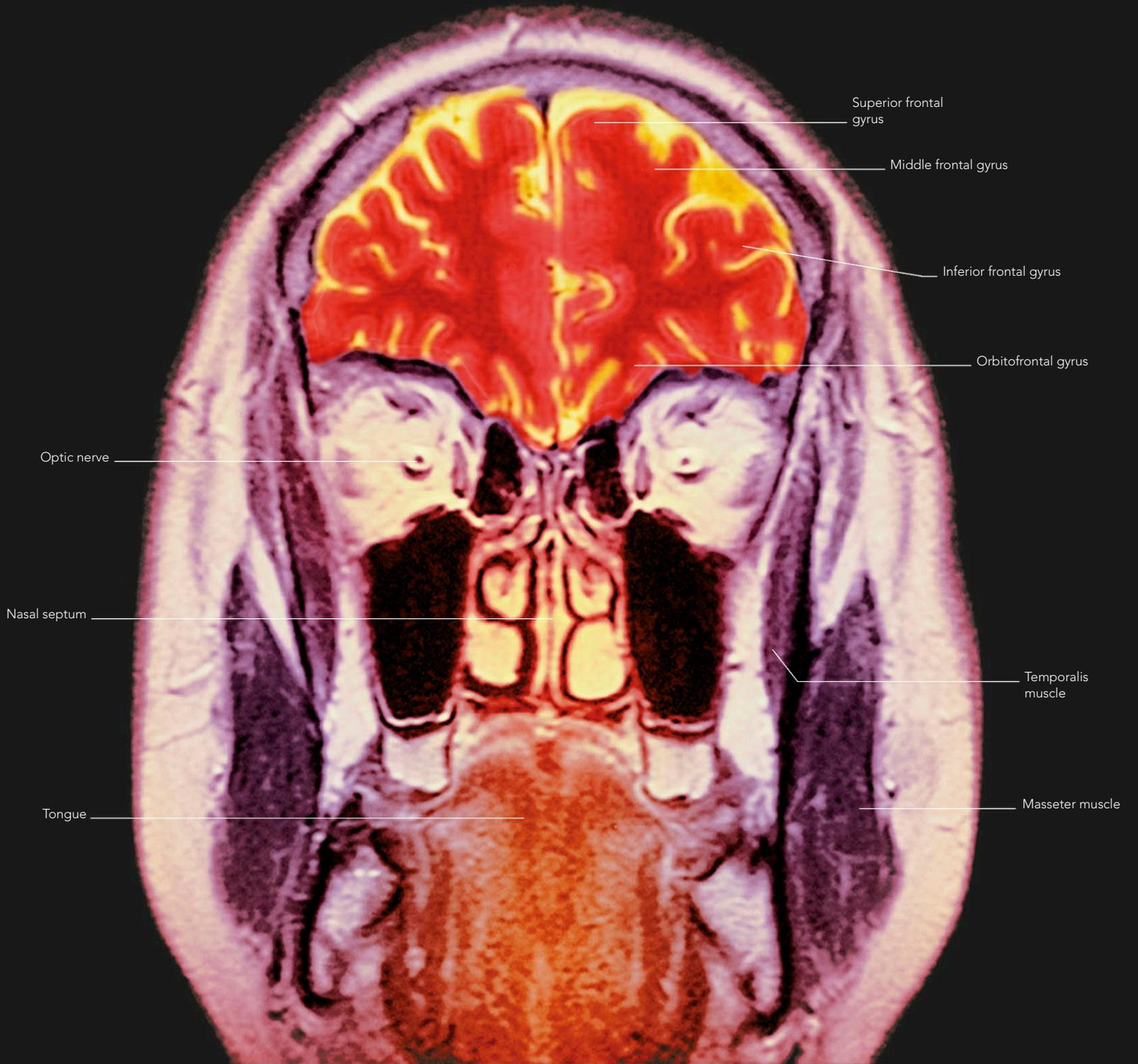


1 THE FRONTAL-POLAR CORTEX

The frontal-polar cortex is the most recently evolved part of the prefrontal cortex in the frontal lobe and is concerned with forward planning and the control of other brain regions. This slice, right at the front of the brain, also reveals other features of the skull, including the eyes, nasal cavity, maxillary sinus, and tongue.



2 THE FRONTAL LOBE
 The frontal lobe, of which the prefrontal cortex is the front part, is the largest of the brain's lobes and the latest to evolve. The frontal lobe is devoted to the control of action—precise control of muscles at the back, high-level planning at the front. In this slice, the optic nerve can also be seen carrying visual information from the eye to the brain.

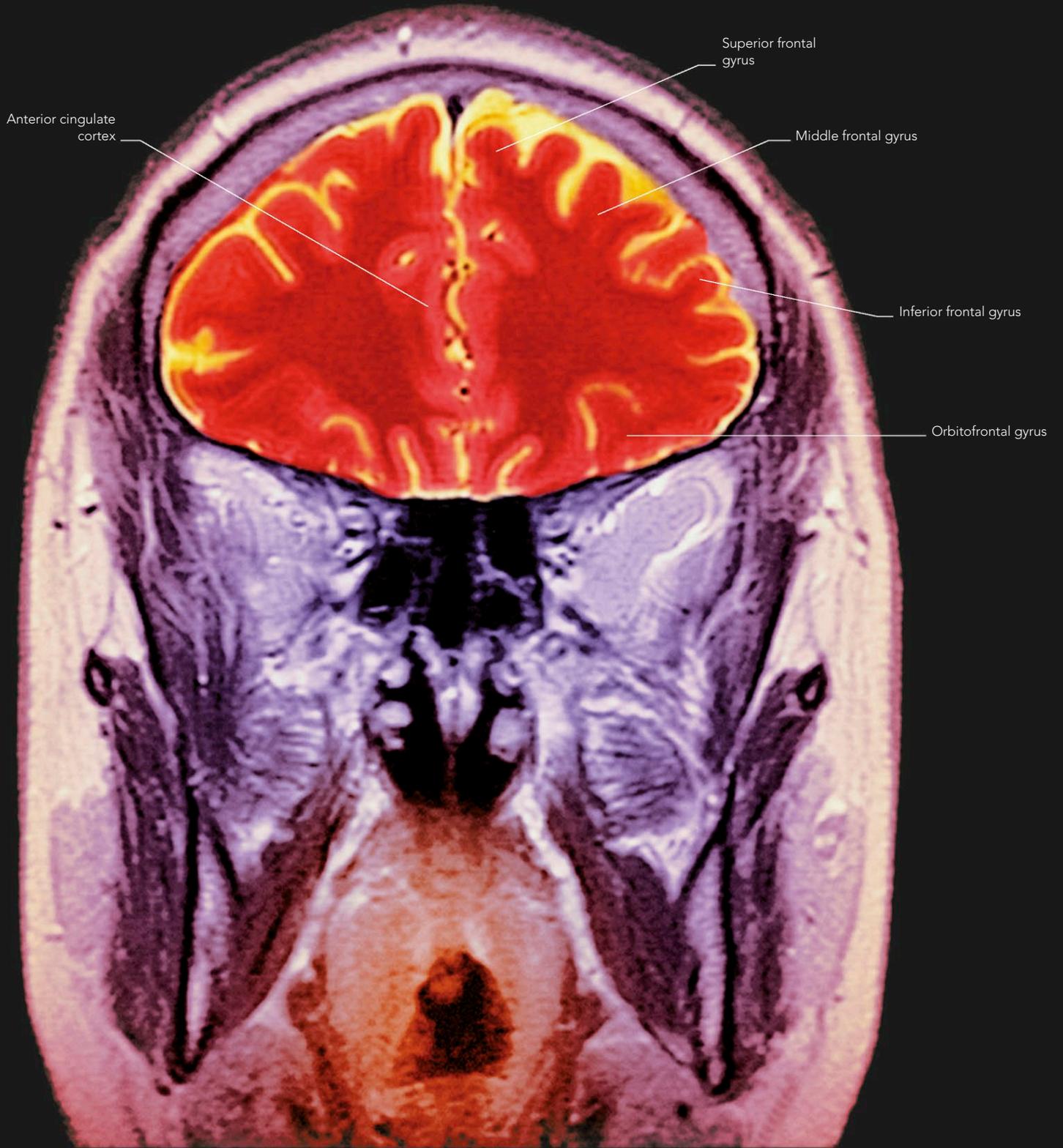


3 THE CORTEX

The cortex, which appears on these scans as yellow lines, is heavily folded, creating a large surface area. The major ingoing folds (sulci, singular sulcus) are used as landmarks to define brain regions. The bulges between the ingoing folds are known as gyri (singular, gyrus). The major components of the frontal lobe are the superior, middle, and inferior frontal gyri.



4 THE ORBITOFRONTAL GYRI
The orbitofrontal gyri, located at the bottom of the brain, receive signals about smell and taste. Like the rest of the prefrontal cortex, this area is concerned with predicting the future, but specializes in predictions about rewards and punishments and therefore emotions. This area is connected with the amygdala (see slice 9, p.24).



5 THE ANTERIOR CINGULATE CORTEX

Here we see the beginning of the anterior cingulate cortex, which lies between the two hemispheres. This sits alongside the limbic system. It is involved in linking emotions to actions and predicting the consequences of actions. The back part of the anterior cingulate cortex has direct connections with the motor system.



6 THE TEMPORAL LOBES
In this slice, the temporal lobes come into view for the first time. At the very front of the temporal lobes (the temporal poles), knowledge acquired from all the senses is combined, along with emotional tone. We can also see the lateral ventricles in the middle of the slice. These are parts of a system of fluid-filled spaces in the middle of the brain.



7 THE INSULA

The insula is a fold of cortex hidden deep in the brain between the frontal and temporal lobes. Signals about the internal state of the body—such as heart rate, temperature, and pain—are received here. Also visible in this slice is the corpus callosum, the band of nerve fibers that joins the brain's left and right hemispheres.

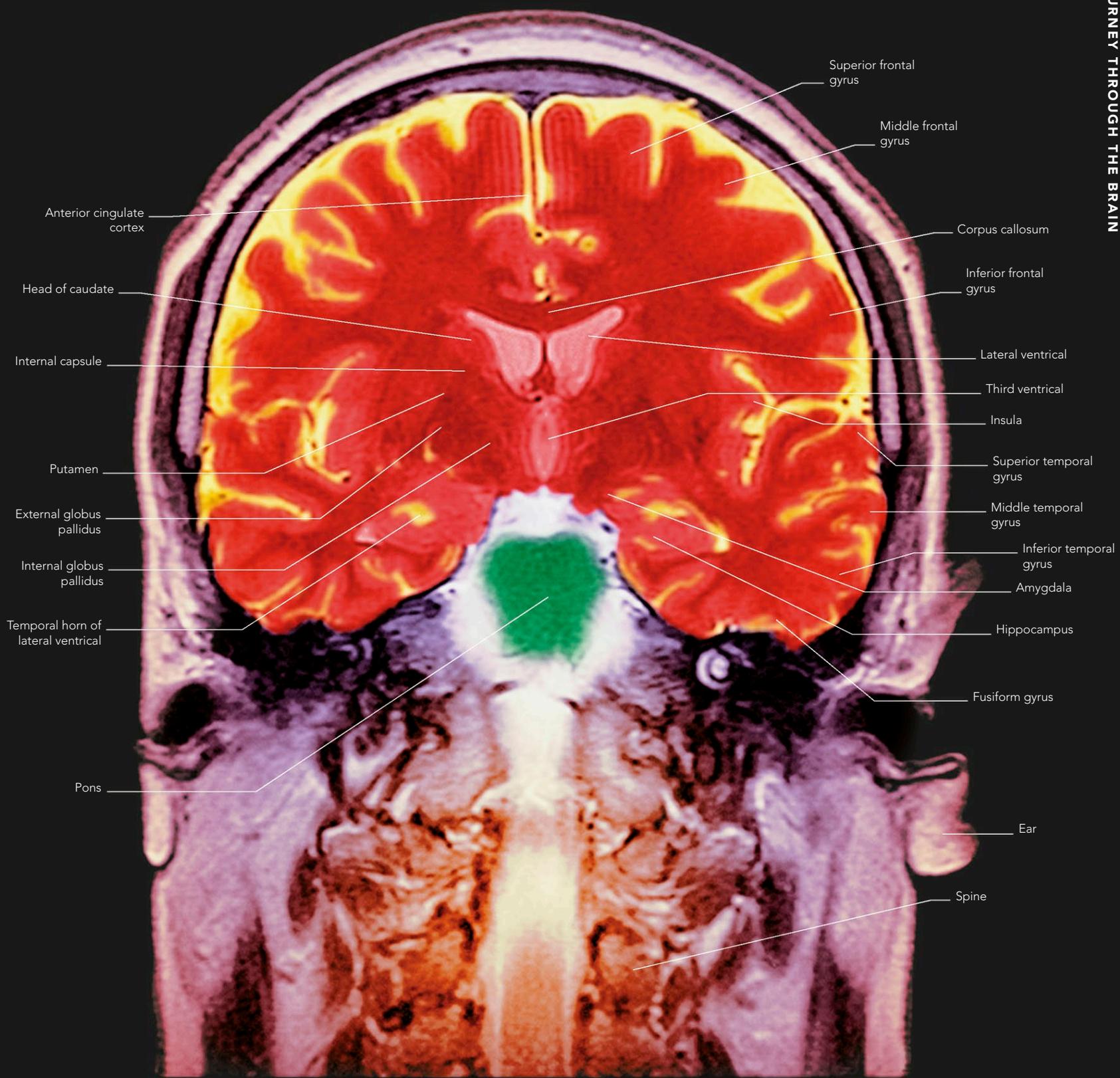


8 THE BASAL GANGLIA
 Located in the middle of the brain, the basal ganglia include the caudate, putamen, and globus pallidus. Also known as nuclei, ganglia are clumps of gray matter (or nerve-cell bodies) surrounded by white matter. The basal ganglia are linked to the cortex, the thalamus, and the brainstem and are concerned with motor control and decision making.

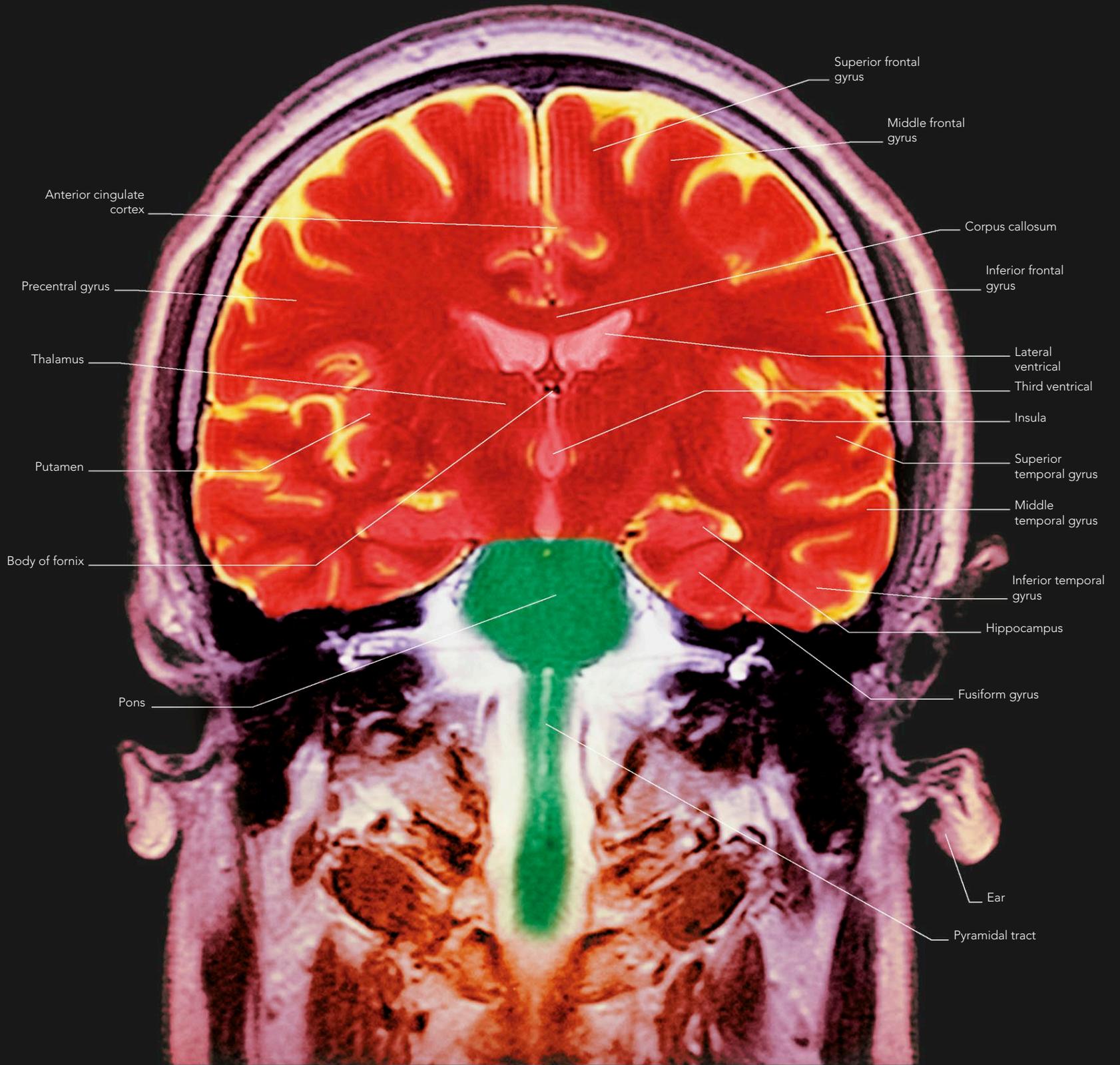


9 THE AMYGDALA AND HIPPOCAMPUS

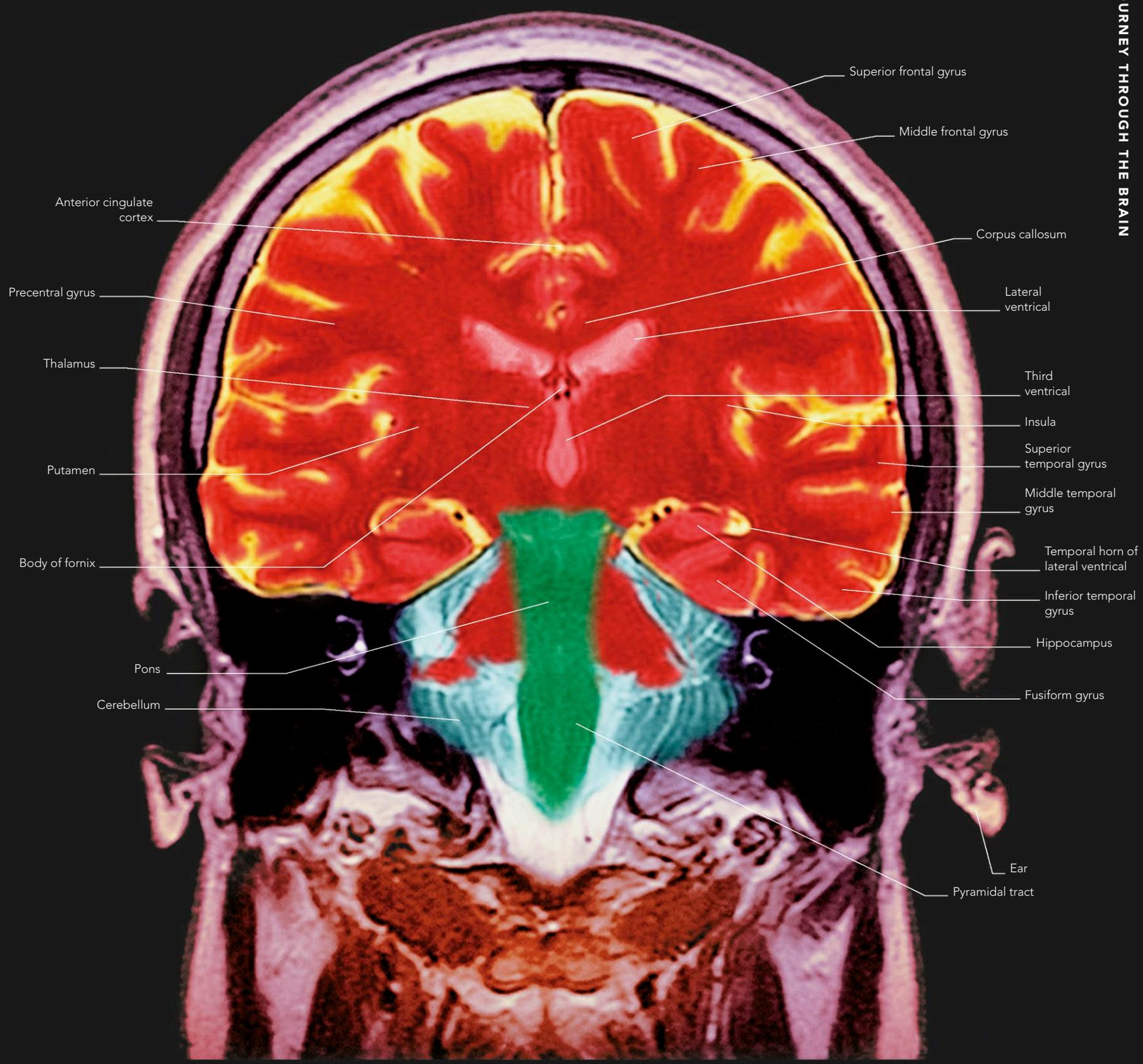
This slice includes the amygdala and the front part of the hippocampus. Both structures lie in the inner part of the temporal lobe. The amygdala is involved in learning to approach or avoid things and hence with emotion. The hippocampus has a critical role in spatial navigation and memory of past experiences, including routes between places.



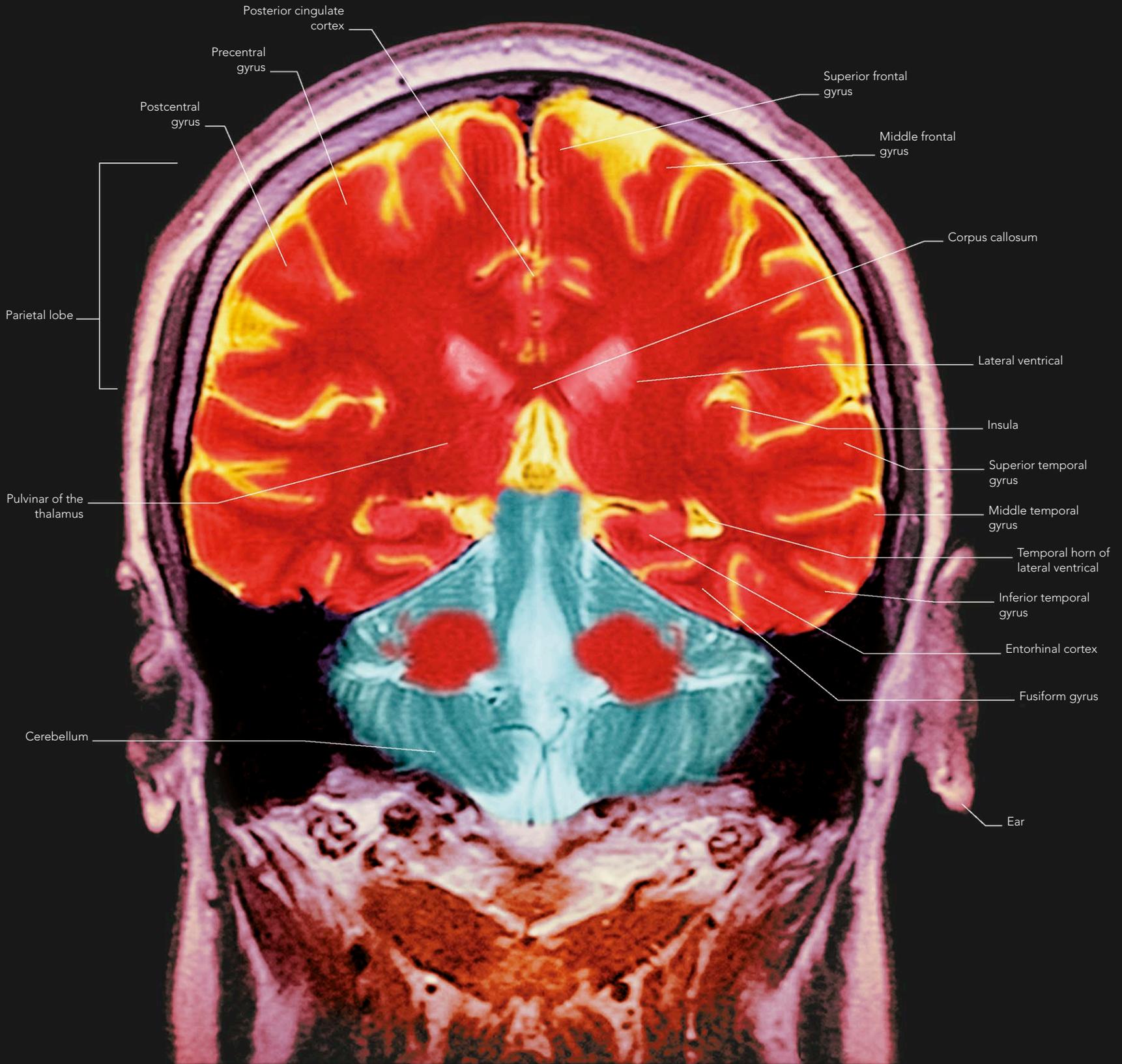
10 BROCA'S AREA Here we approach the back of the frontal lobe. The bottom of the inferior frontal gyrus in the left hemisphere, just above the insula, contains Broca's area, which has a critical role in speech and language. At the bottom of the slice, we see the front of the brainstem, the pons, which joins the brain to the spinal cord.



11 THE THALAMUS
 This slice includes the thalamus, which lies between the cerebrum and the brainstem. A complex structure, the thalamus is made up of more than 20 nuclei (see p.60). The thalamus acts as a relay station, taking in information from all of the senses (except smell) and sending them on to different parts of the cerebral cortex.

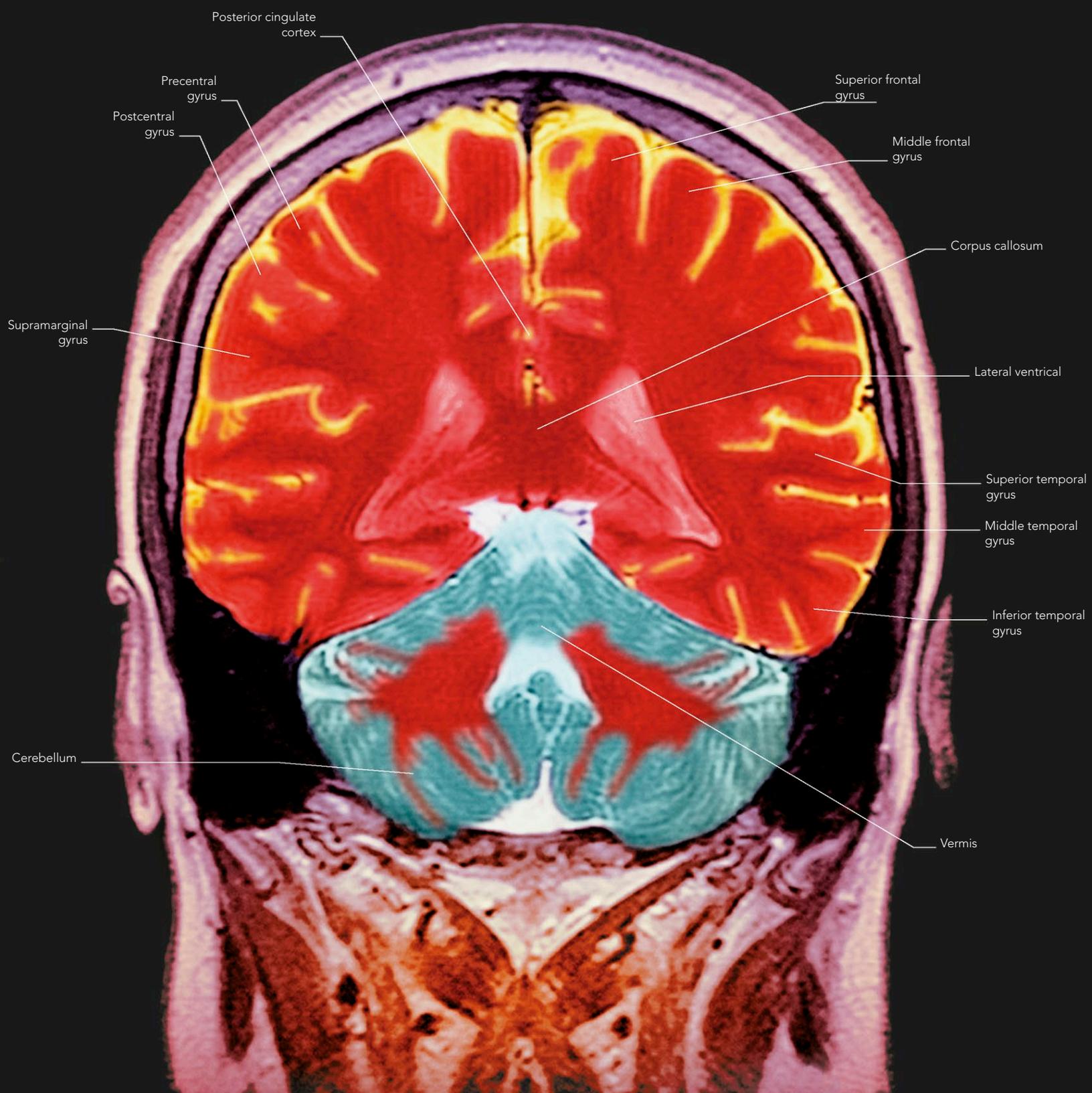


12 THE BRAINSTEM
 The brainstem (in green) joins the brain to the spinal cord and contains a number of structures such as the pons. The brainstem has a special role in the control of basic body functions, including the control of heart rate and breathing. It also relays signals from the brain to the muscles and from senses in all parts of the body to the brain.

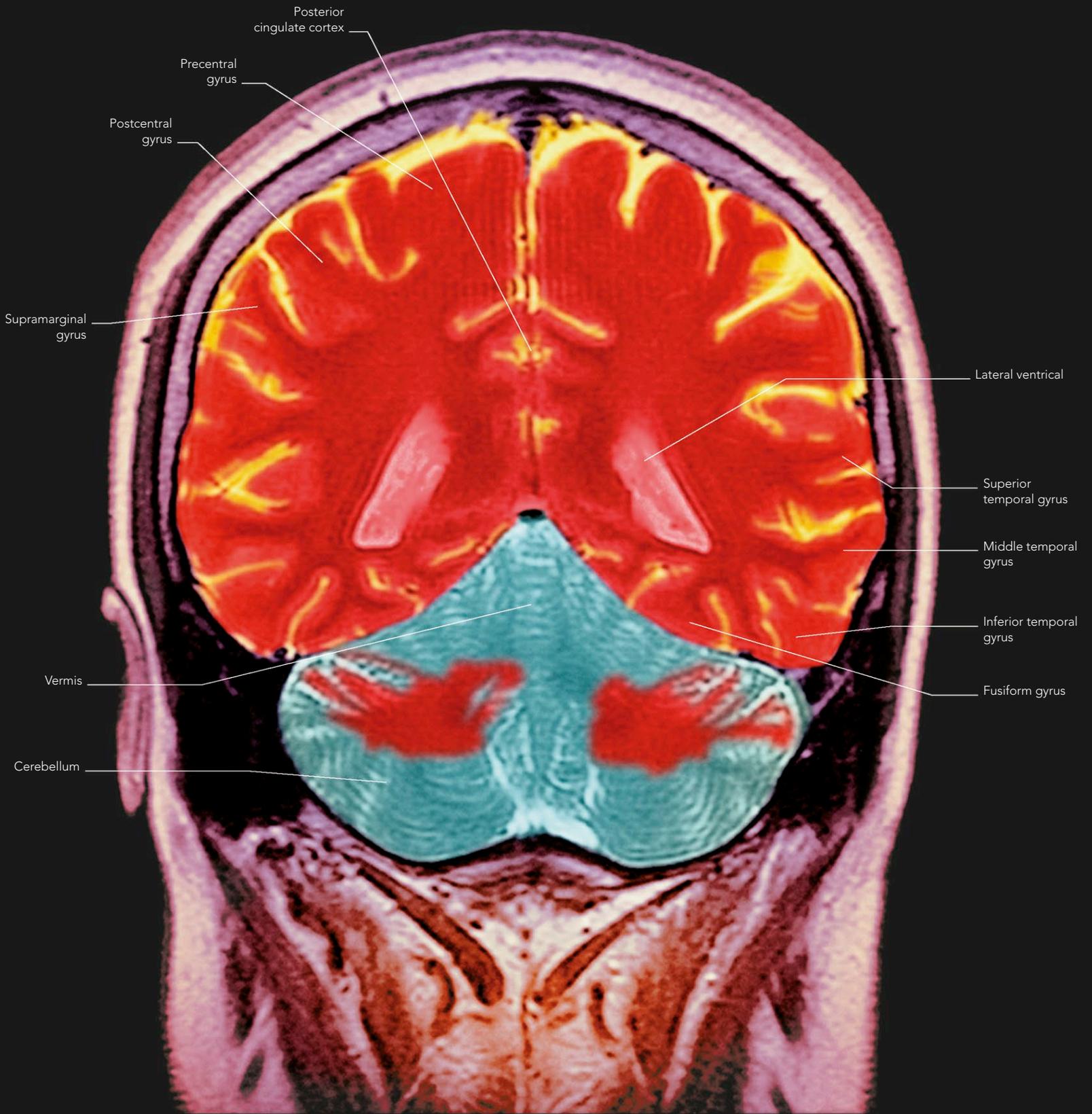


13 THE PARIETAL LOBE

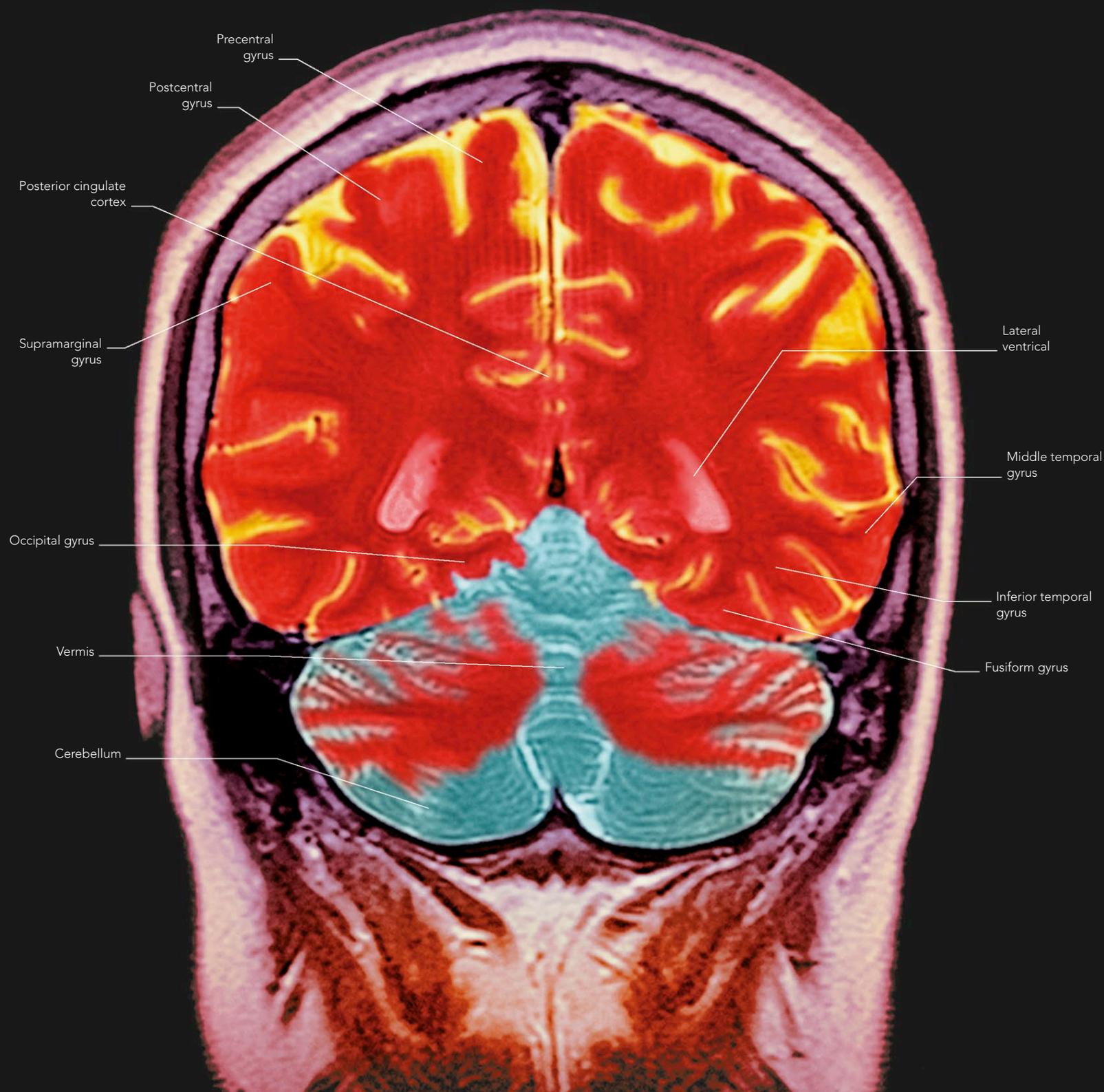
The parietal lobe includes the supramarginal gyrus and the angular gyrus (see slices 14–20, pp.29–35). The parietal lobe integrates signals from many of the senses (including visual information that arrives via the dorsal route, see pp.84–85) to estimate the position of the body and the limbs in space. This information is critical when we reach for and grasp objects.



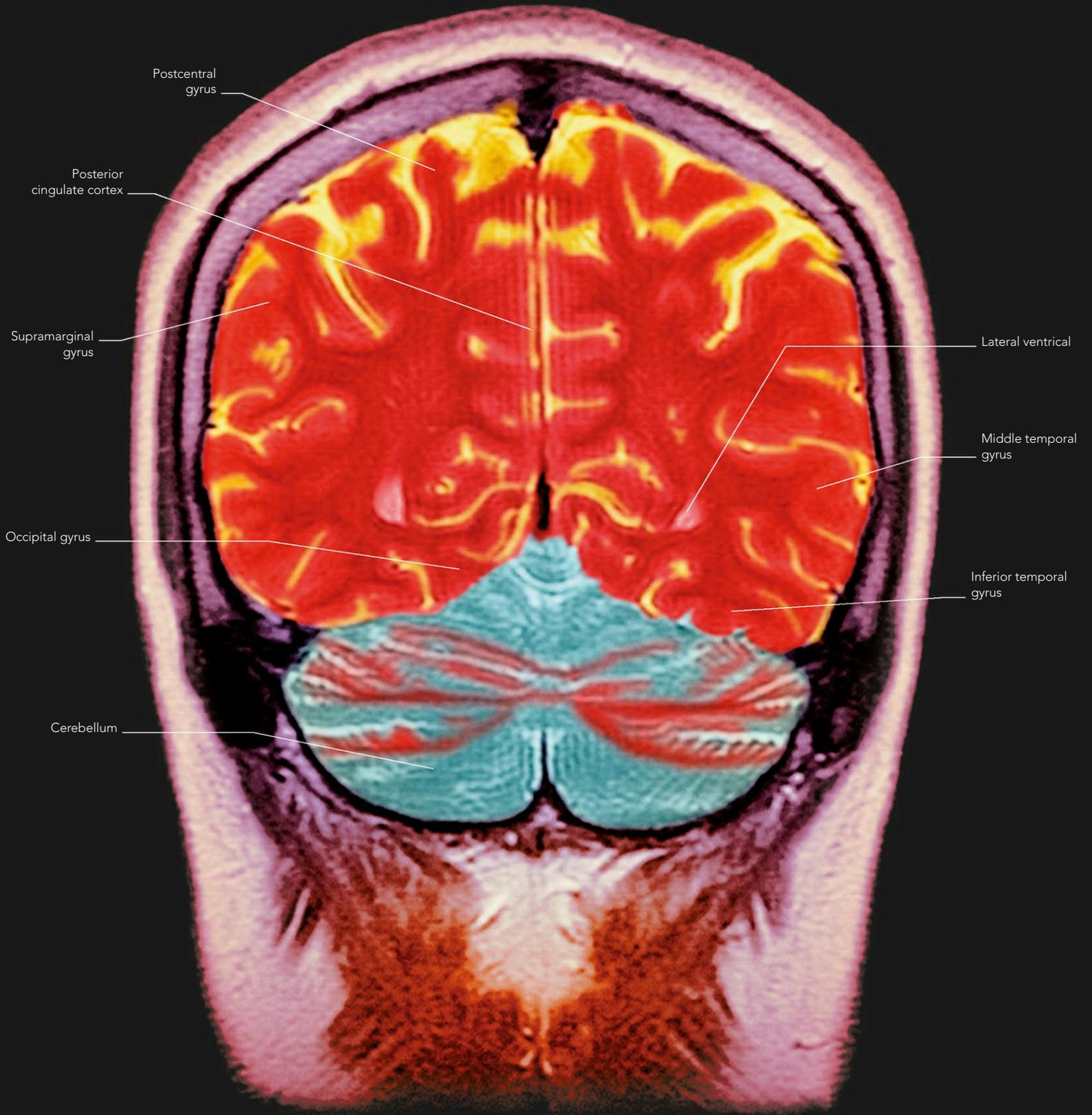
14 THE PRECENTRAL AND POSTCENTRAL GYRUS
 The last part of the frontal cortex is the precentral gyrus. This contains the motor strip, where different regions send signals to control different parts of the body. The immediately adjacent part of the parietal cortex (the postcentral gyrus) has a corresponding sensory strip, where sensory signals are received from different parts of the body.



15 THE PRIMARY AUDITORY CORTEX
The primary auditory cortex, where signals from the ears reach the cortex via the thalamus, lies along the very top of the superior temporal gyrus, in the fissure between the temporal lobe and the parietal lobe. Adjacent to the primary auditory cortex is Wernicke's area, where incoming sounds are turned into words.



16 THE FUSIFORM GYRUS The inferior temporal gyrus and the fusiform gyrus at the bottom of the temporal lobe are two areas concerned with recognition of objects. Part of the fusiform gyrus, known as the face-recognition area, is specialized for recognizing faces. It not only identifies facial features but also scrutinizes them for meaning, so it plays an important part in social interaction.



17 THE CEREBELLUM

The cerebellum (colored light blue) is the highly convoluted "little brain" that sits at the back and below the main brain (also known as the cerebrum). The cerebellum is concerned with fine motor control and the timing of movements. There are many connections between the cerebellum and the motor cortex.

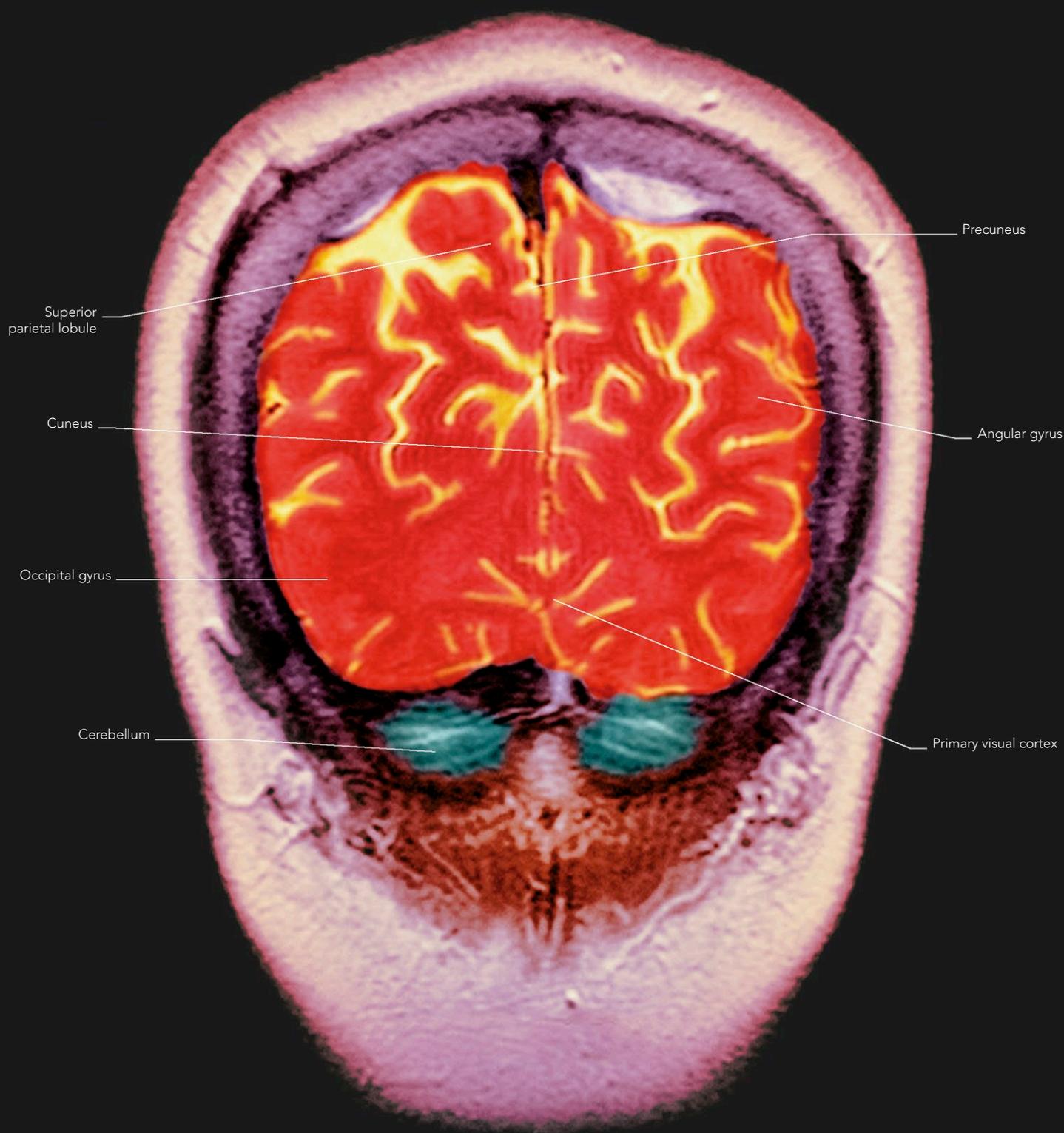


18 THE OCCIPITAL LOBE
The occipital lobe is concerned with vision. In the forward-most areas, signals from the primary visual cortex (see slice 20, p.35) are analyzed in terms of features such as shape and color. This information is then sent forward to the inferior temporal cortex (see slice 16, p.31), along a pathway called the ventral route, and used for object recognition.

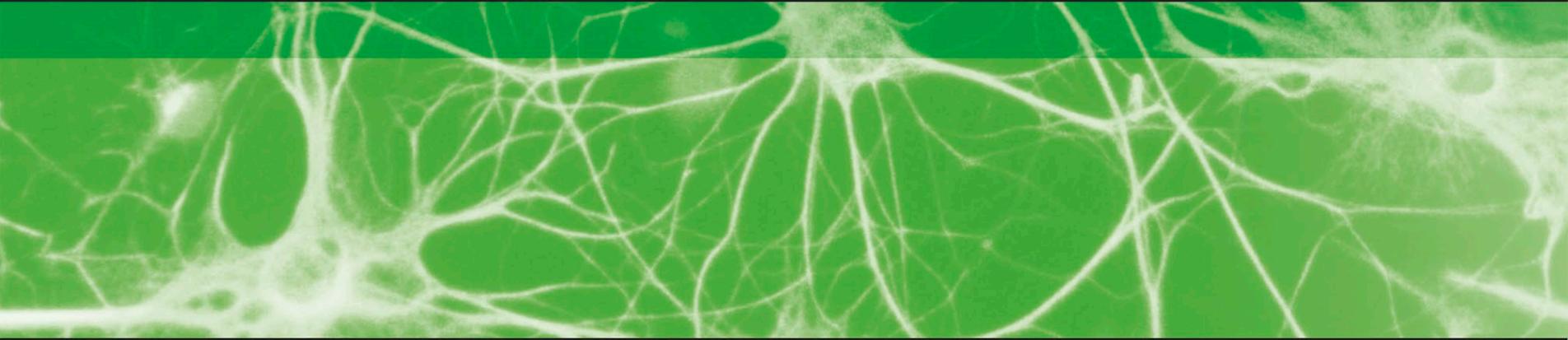


19 THE PRECUNEUS AND THE POSTERIOR CINGULATE CORTEX

The precuneus in the back part of the parietal lobe and posterior cingulate cortex (see slice 17, p.32) lie between the two hemispheres. These remain some of the more mysterious regions of the brain. They probably have a role in memory, especially memories about the self.



20 THE PRIMARY VISUAL CORTEX
 The primary visual cortex is right at the back of the brain and lies mostly on the inside of the two hemispheres. This is the first point in the cortex where signals arrive from the eyes via the thalamus. These signals are retinotopically mapped—that is, a signal from a particular point on the retina is sent to a corresponding point on the primary visual cortex.



THE HUMAN BRAIN KEEPS US PRIMED TO RESPOND TO THE WORLD AROUND US. IT IS AT THE HUB OF A VAST AND COMPLEX COMMUNICATIONS NETWORK THAT CONSTANTLY SEEKS AND COLLECTS INFORMATION FROM THE REST OF THE BODY AND THE OUTSIDE WORLD. AS THE BRAIN INTERPRETS THIS INFORMATION, IT GENERATES EXPERIENCES—SIGHTS AND SOUNDS, EMOTIONS AND THOUGHTS. BUT ITS PRIMARY FUNCTION IS TO PRODUCE CHANGES IN THE BODY. THESE INCLUDE LIFE-SUSTAINING BASICS SUCH AS THE REGULAR CONTRACTIONS OF THE HEART THROUGH TO THE COMPLEX ACTIONS THAT CONSTITUTE BEHAVIOR.

THE BRAIN AND THE BODY



BRAIN FUNCTIONS

THE PRIMARY TASK OF THE BRAIN IS TO HELP MAINTAIN THE WHOLE BODY IN AN OPTIMAL STATE RELATIVE TO THE ENVIRONMENT, IN ORDER TO MAXIMIZE THE CHANCES OF SURVIVAL. THE BRAIN DOES THIS BY REGISTERING STIMULI AND THEN RESPONDING BY GENERATING ACTIONS. IN THE PROCESS, IT ALSO GENERATES SUBJECTIVE EXPERIENCE.



WHAT THE BRAIN DOES

The brain receives a constant stream of information as electrical impulses from neurons in the sense organs. The first thing it does is determine whether the information warrants attention. If it is irrelevant or just confirmation that everything is staying the same, it is allowed to fade away and we are not conscious of it. But if it is novel or important, the brain amplifies the signals, causing them to be represented in various regions. If this activity is sustained for long enough, it will result in a

THE BRAIN AND BODY
The brain and spinal cord constitute the central nervous system, which is the body's main control center, responsible for coordinating all of the processes and movement in the body.

conscious experience. In some cases, thoughts are taken one step further, and the brain instructs the body to act on them, by sending signals to the muscles to make them contract.

KEY FEATURES OF THE BRAIN

FEATURE	DESCRIPTION
Processing information	The brain registers a vast amount of information. However, only a very small amount of this is actually selected for processing to the point at which it enters our consciousness and can be reported. Experience that cannot be reported is not conscious. Unconscious brain processing nevertheless guides and sometimes initiates actions (see p.116 and p.191).
Sending signals	The brain consists of about 1000 billion cells. Roughly 10 percent are specialized electrical cells called neurons, which send signals to one another; this signal transmission makes brain function different from any other bodily process. Although the signals are electrical, the mode of transmission between cells is chemical—the signals are passed on by substances called neurotransmitters.
Modules and connections	The brain is modular—different parts do different things. The modules are densely interconnected, however, and none works without the support of many others (and the rest of the body). Generally, lower-level functions, such as registering sensations, are strongly localized, but higher-level functions, such as memory and language, result from interconnections between brain areas.
Individuality	The basic “blueprint” of the brain is dictated by our genes. As with any other body feature, brains share a basic anatomy, but each one is also unique. Even identical twins have visibly different brains, right from the time they are born, because the brain is exquisitely sensitive to its environment. The differences between individual brains result in each person having a unique personality.
Plasticity	Brain tissue can be “strengthened” and built up like a muscle, according to how much it is exercised. So, if a person learns and practices a skill, such as playing a musical instrument or doing mathematics, the part of the brain concerned with that task will grow physically bigger. It also becomes more efficient and enables the person to perform the task more skillfully.

HOW THE BRAIN DOES IT

No one knows exactly how electrical activity turns into experience. That remains a famously hard problem, which has yet to be cracked (see p.179). However, much is now known about the brain processes that turn incoming information into the various components of subjective experience, such as thoughts or emotions. Much depends on where the information comes from. Each sense organ is specialized to deal with a different type of stimulus—the eyes are sensitive to light, the ears to sound waves, and so on. The sense organs respond to these stimuli in much the same way—they generate electrical signals, which are sent on for further processing. But the information from each organ is sent to a different part of the brain, and then processed along a different neural pathway. Where information is processed therefore determines what sort of experience it will generate.

ACTIONS

Certain brain areas are specialized to produce body movement. Brainstem modules control automatic internal actions, such as the lung and chest movements needed for breathing, the beating of the heart, and the constriction or dilation of blood vessels to control blood pressure. In conscious activities, the primary motor cortex sends messages (via the cerebellum and basal ganglia) to the muscles of the limbs, trunk, and head to create gross movements.

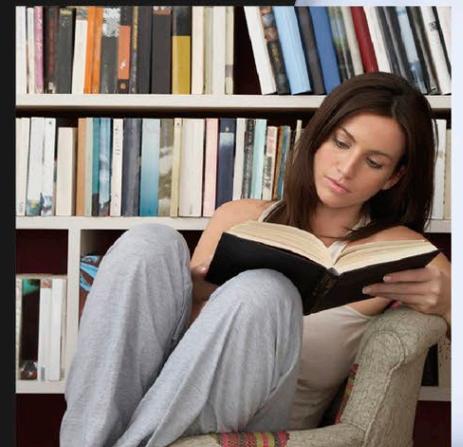


MEMORIES

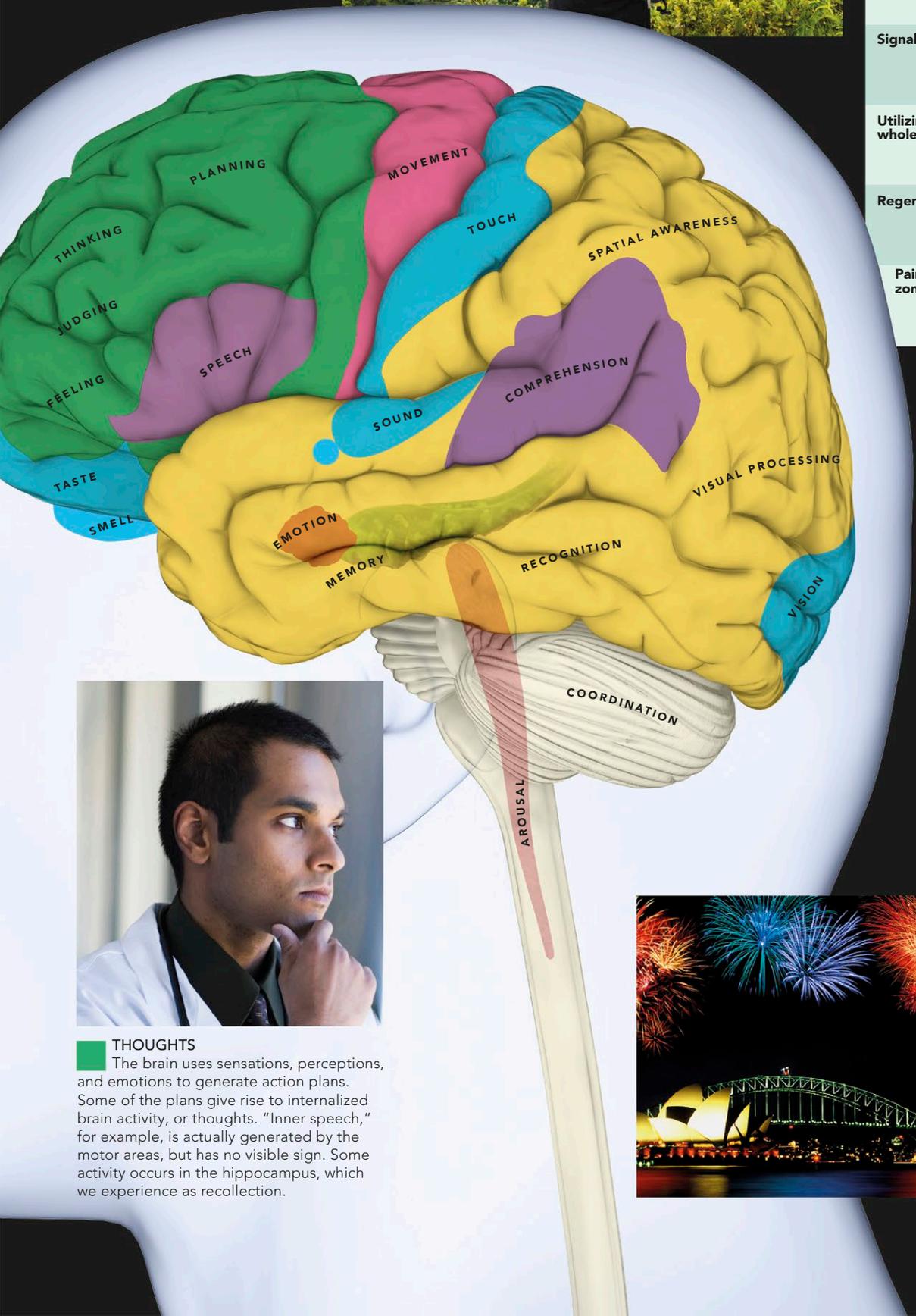
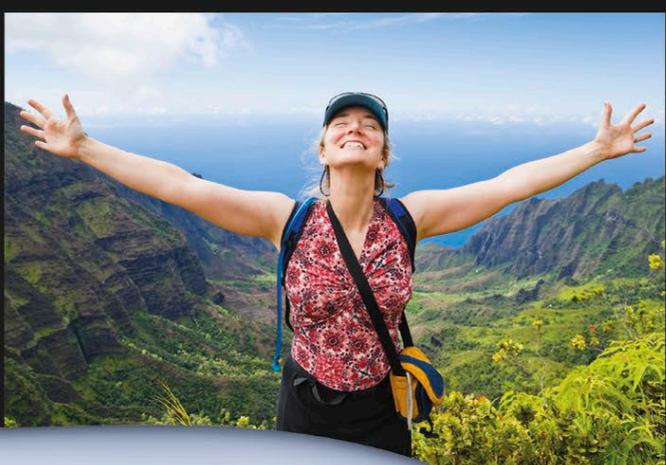
Some of the experiences we have change brain cells in such a way that the pattern of neural activity that produced the original experience can be replicated later in time. This process gives rise to recall, or memory, which enables us to use past experiences as a guide to how to behave in the present.

LANGUAGE

Language involves both producing speech and analyzing what others say to understand the meaning. It depends on the brain's ability to link objects with abstract symbols and then to convey the symbols—and thus the ideas they represent—to others via words. In addition to facilitating communication between people, language enables individuals to reflect on their own ideas.



EMOTIONS
 Certain stimuli (including some thoughts and imaginings) cause changes in the body by activating areas in the limbic system, especially the amygdala. Conscious "feelings" occur when signals from the limbic system are sent on to "association areas" in the prefrontal cortex that support consciousness. During adolescence, the amygdala is relied heavily upon for processing emotional information, because the prefrontal cortex only matures when a person reaches their late 20s.



THOUGHTS
 The brain uses sensations, perceptions, and emotions to generate action plans. Some of the plans give rise to internalized brain activity, or thoughts. "Inner speech," for example, is actually generated by the motor areas, but has no visible sign. Some activity occurs in the hippocampus, which we experience as recollection.

BRAIN FACTS	
FEATURE	FACT
Structure	The brain is highly compact. If you smoothed out all the wrinkles in the cortex, the brain would cover an area of about 2 1/2 square ft (2,300 square cm).
Connectivity	The brain has around 100 billion neurons. There are more potential connections between the neurons than there are atoms in the universe.
Growth	A fetus grows neurons at the rate of 250,000 a minute. A person is born with nearly all the neurons of an adult, but the neural networks are not mature yet.
Signaling speed	Information travels at different speeds within different types of neurons. Transmission speeds range from 3 to 330 feet/sec (1 to 100 meters/sec).
Utilizing the whole brain	The claim that we only use 10 percent of our brains is false—we use all of it. Some complex functions, such as memory, involve many areas at once.
Regeneration	You do not "lose" brain cells as you age, although some functions may decline. You can maintain the networks or even form new ones by exercising your brain.
Pain-free zone	Brain tissue has no pain receptors, so despite the fact it registers pain from all parts of the body, it does not actually feel pain itself.



SENSATIONS
 Information from the environment enters the brain via the different sense organs and is transmitted to specific areas of the cerebral cortex called the primary sensory areas. This information includes some input from the body itself. In the absence of external stimuli, the sensory areas continue to be active and are thought to generate the experiences that we know as dreams, hallucinations, and imagination.



PERCEPTIONS
 Most of the time we are receiving information from many sensory areas at once, as with the combination of auditory and visual signals at a fireworks display. These signals may be communicated to association areas, which bind all of this information together. If these items of "bound" information become conscious, they form what is known as a multisensory perception. There is a great deal of current neuroscientific research on how the binding process forms a unified perception, because it is still not fully understood.

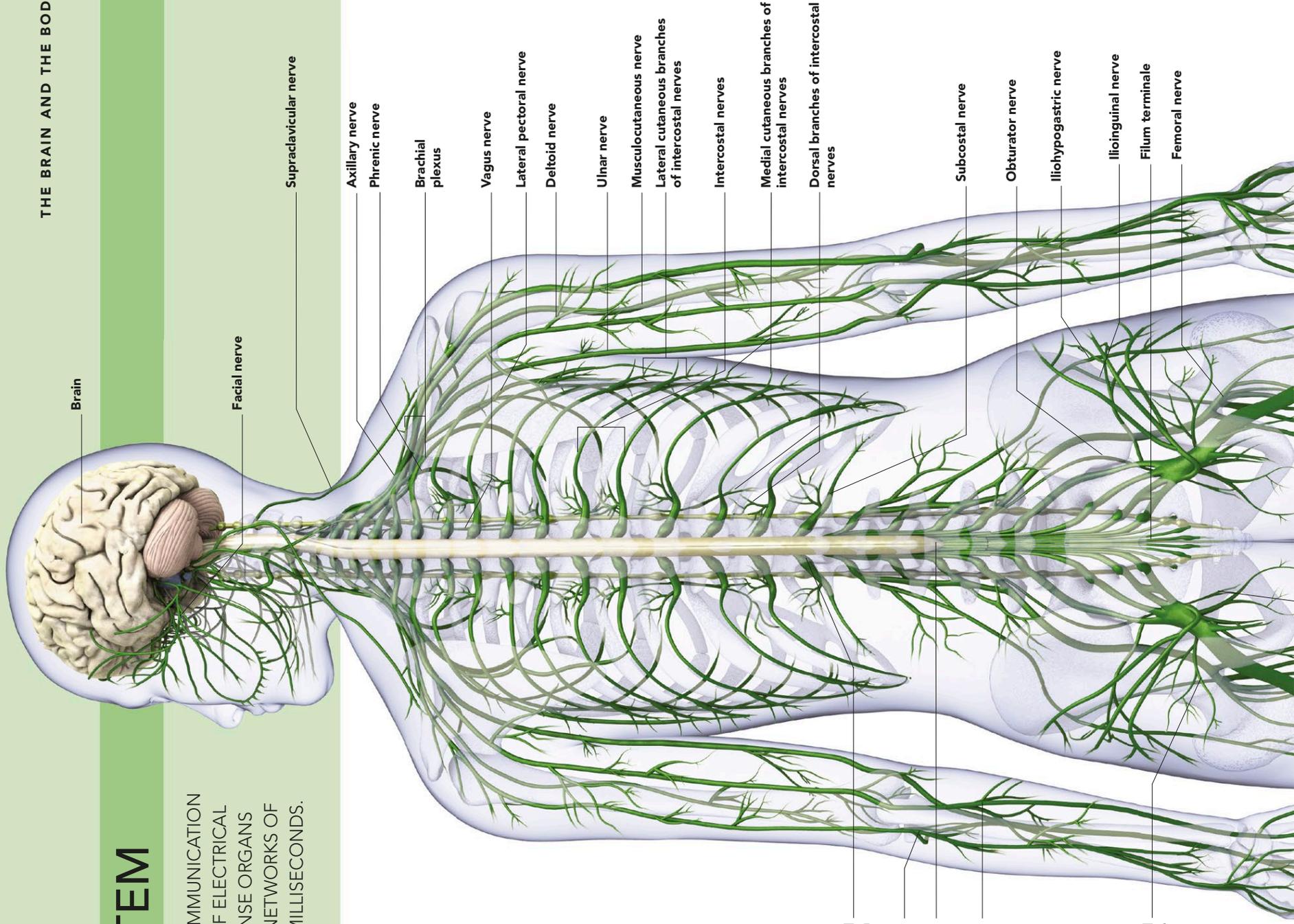
THE NERVOUS SYSTEM

THE NERVOUS SYSTEM IS THE BODY'S MAJOR COMMUNICATION AND CONTROL NETWORK. DATA, IN THE FORM OF ELECTRICAL SIGNALS, IS RELAYED CONSTANTLY FROM THE SENSE ORGANS TO AND FROM THE BRAIN, THROUGH COMPLEX NETWORKS OF NEURONS AND ON A TIMESCALE MEASURED IN MILLISECONDS.

Although it is a single, unified communications network, the nervous system consists of three anatomical and functional subdivisions. The central nervous system (CNS) is the coordinating system for the body. It comprises the brain and spinal cord, which are surrounded and protected by the skull and vertebral column respectively. The peripheral nervous system (PNS) is a complex network of nerves extending across the body, branching out from 12 pairs of cranial nerves originating in the brain and 31 pairs of spinal nerves emanating from the spinal cord. It relays information between the body and the brain in the form of nerve impulses. It has an afferent division (through which messages are sent to the brain) and an efferent division (which carries messages from the brain to the body).

Finally, there is the autonomic nervous system (ANS), which shares some nerve structures with both the CNS and PNS. It functions "automatically" without conscious awareness, controlling basic functions, such as body temperature, blood pressure, and heart rate.

Sensory input travels quickly from receptor points throughout the body via the afferent network of the PNS to the brain, which processes, coordinates, and interprets the data in just fractions of a second. The brain makes an executive decision that is conveyed via the efferent division of the PNS to muscles, which take the action needed to respond to environmental change rapidly.



Brain

Facial nerve

Suprascapular nerve

Axillary nerve

Phrenic nerve

Brachial plexus

Vagus nerve

Lateral pectoral nerve

Deltoid nerve

Ulnar nerve

Musculocutaneous nerve

Lateral cutaneous branches of intercostal nerves

Intercostal nerves

Medial cutaneous branches of intercostal nerves

Dorsal branches of intercostal nerves

Subcostal nerve

Obturator nerve

Iliohypogastric nerve

Ilioinguinal nerve

Filum terminale

Femoral nerve

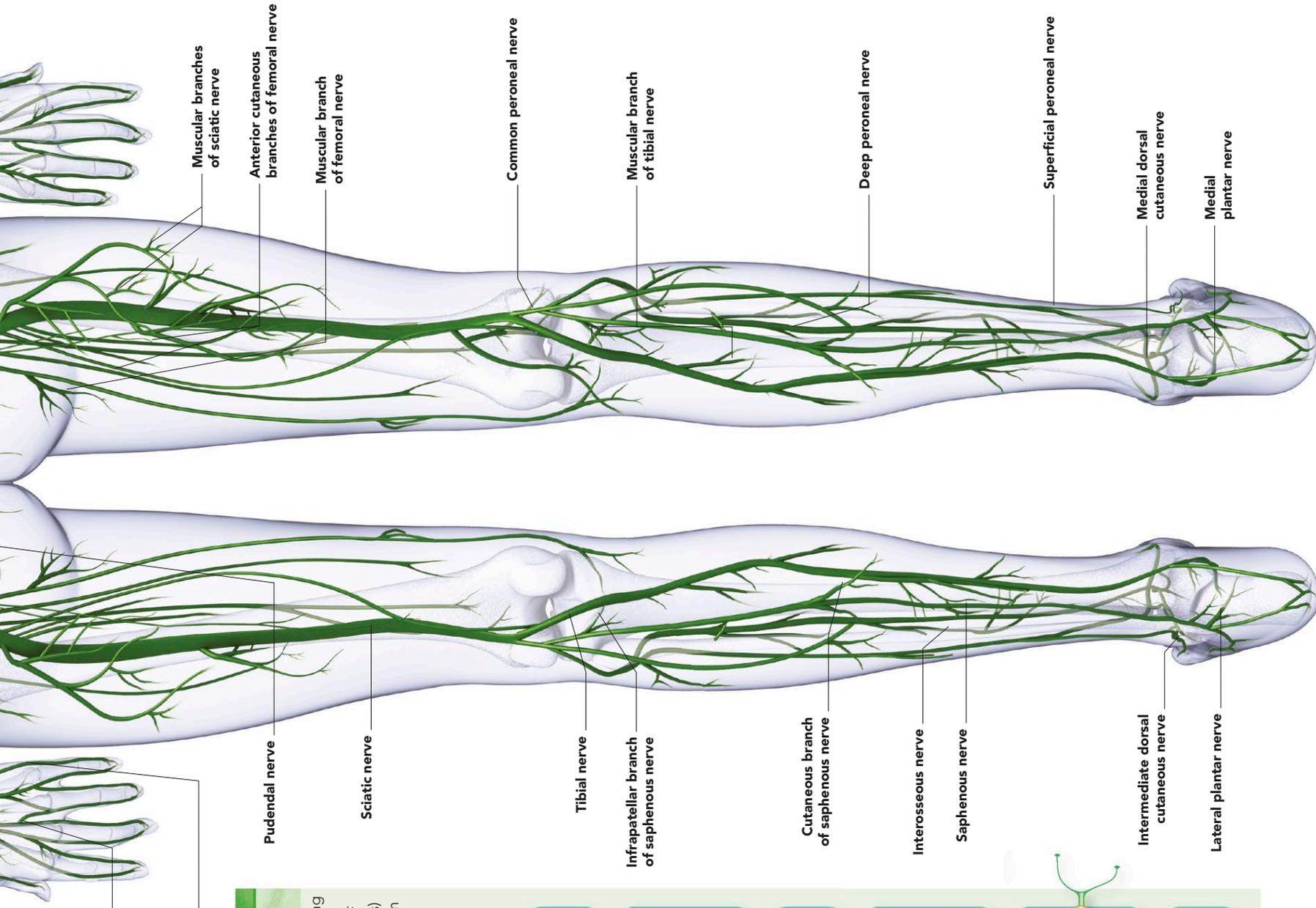
Spinal ganglion

Radial nerve

Spinal cord

Median nerve

Gluteal nerve



THE BRAIN AND THE BODY

Increasingly, the interaction between brain and body is being understood in much finer detail. The organization of the nervous system (and for that matter, all the other systems of the body, such as the cardiovascular and endocrine systems) can be considered at various different functional levels, from the entire system down to individual cells, the basic unit of all living things. The chart below shows six of these levels and their features. While it is possible to view organs with the naked eye, tissues, networks, cells, and molecules all have to be viewed with the aid of a microscope.

SYSTEM—THE CENTRAL NERVOUS SYSTEM

The brain and the spinal cord together make up the CNS.

ORGAN—THE BRAIN

The central organ of the CNS, the brain is a complex, integrated collection of tissues that controls the functions of the human body.



TISSUES—NUCLEI

These are groups of neurons (nuclei) that work together to perform specialized functions.

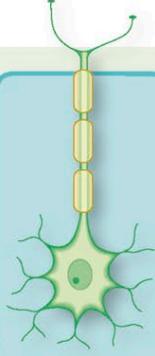
NETWORKS

Neural networks consist of thousands of neurons and the connections between them (synapses).



CELLS—NEURONS

Neurons are the basic units of the CNS. They transmit electrical signals, process the data, and communicate with each other via synapses.



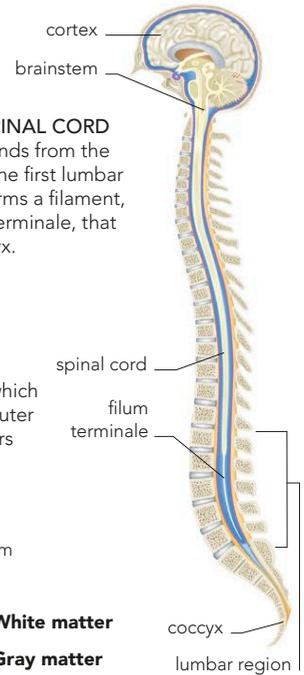
MOLECULES

These are the smallest recognized unit, comprising two or more atoms. All the body's cells contain working parts made of millions of them.

THE BRAIN AND THE NERVOUS SYSTEM

THE BRAIN SITS AT THE TOP OF THE BODY, DIRECTING AND COORDINATING ALL ACTION AND ACTIVITY THROUGHOUT ITS ENTIRETY. IT DOES SO VIA THE SPINAL CORD, AND THE NERVES THAT STEM FROM IT AT VARIOUS POINTS ALONG ITS LENGTH AND BRANCH OUT INTO A NETWORK THAT SPANS THE WHOLE BODY.

EXTENT OF THE SPINAL CORD
The spinal cord extends from the brainstem down to the first lumbar vertebra, where it forms a filament, known as the filum terminale, that extends to the coccyx.

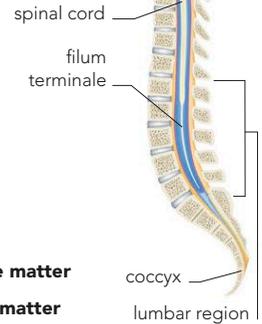


THE SPINAL CORD

The spinal cord carries information to and from the brain and all parts of the body except the head, which is served by the cranial nerves. The signals that travel along the spinal cord are known as nerve impulses. The cord itself comprises a bundle of nerve fibers, which are the long projections of nerve cells. They extend from the base of the brain to the lower region of the spine. The cord is roughly the width of a pencil, tapering at its base to a narrow bunch of fibers. Data from the sensory organs in different parts of the body is collected via the spinal nerves and transmitted along the spinal cord to the brain. The spinal cord also sends motor information, such as movement commands, from the brain out to the body, again transmitted via the spinal nerve network.

SPINAL CORD ANATOMY

The core of the spinal cord is gray matter, which is composed of nerve cells (neurons). The outer layer of white matter insulates the long fibers (axons) that extend from the nerve cells.



Nerve fibers

Bundles of nerve fibers carry signals to and from spinal cord and specific areas of the brain

White matter

Gray matter

Central canal

Filled with cerebrospinal fluid, which provides nourishment

Sensory root ganglion

Cluster of nerve cell bodies on each spinal nerve; partially processes incoming signals

Sensory nerve root

Nerve splits into rootlets that enter spinal cord at rear, carrying incoming signals about touch sensations to brain

Subarachnoid space

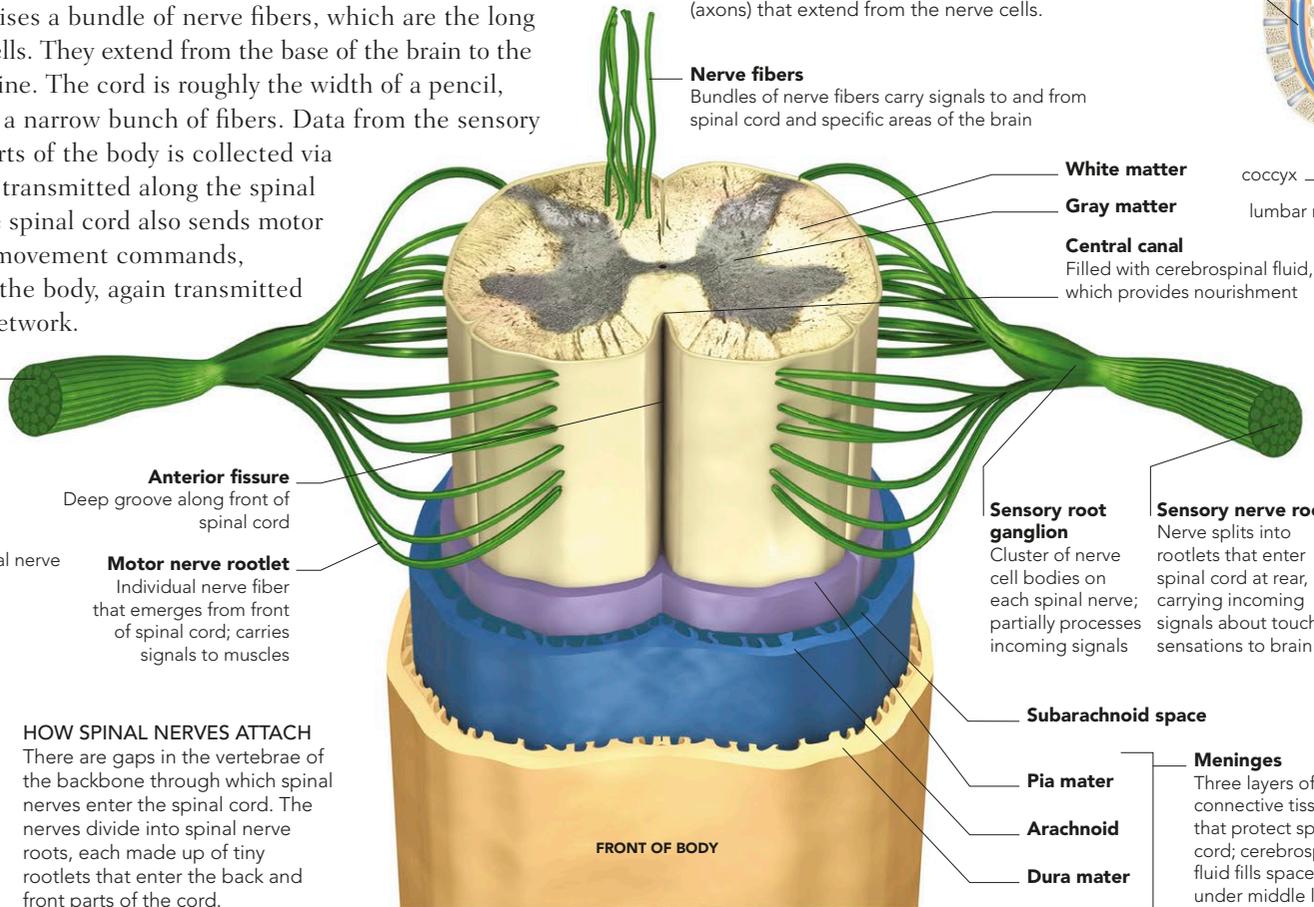
Pia mater

Arachnoid

Dura mater

Meninges

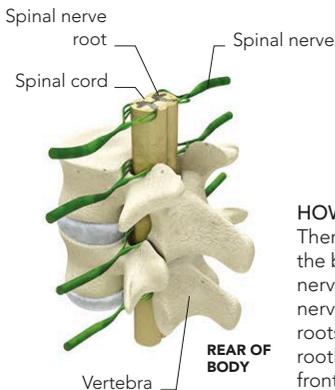
Three layers of connective tissues that protect spinal cord; cerebrospinal fluid fills space under middle layer



Spinal nerve
Carries both sensory and motor information between brain and body

Anterior fissure
Deep groove along front of spinal cord

Motor nerve rootlet
Individual nerve fiber that emerges from front of spinal cord; carries signals to muscles

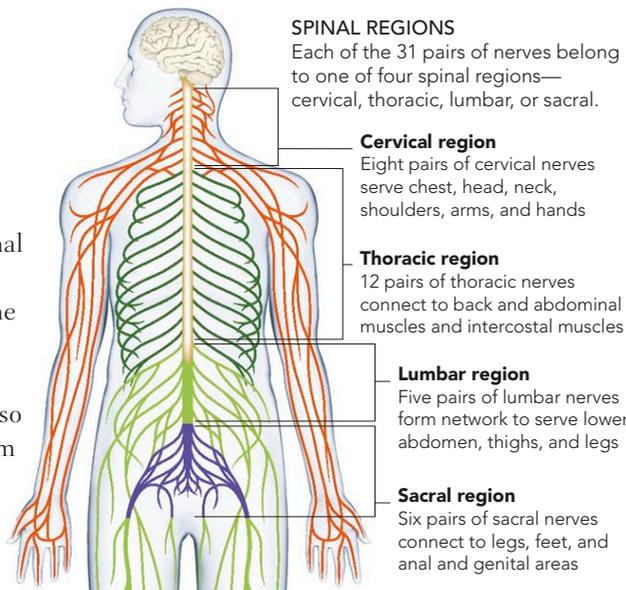


HOW SPINAL NERVES ATTACH

There are gaps in the vertebrae of the backbone through which spinal nerves enter the spinal cord. The nerves divide into spinal nerve roots, each made up of tiny rootlets that enter the back and front parts of the cord.

SPINAL NERVES

There are 31 pairs of spinal nerves. These branch out from the spinal cord, dividing and subdividing to form a network connecting the spinal cord to every part of the body. The spinal nerves carry information from receptors around the body to the spinal cord. From here the information passes to the brain for processing. Spinal nerves also transmit motor information from the brain to the body's muscles and glands so that the brain's instructions can be carried out swiftly.



SPINAL REGIONS

Each of the 31 pairs of nerves belong to one of four spinal regions—cervical, thoracic, lumbar, or sacral.

Cervical region

Eight pairs of cervical nerves serve chest, head, neck, shoulders, arms, and hands

Thoracic region

12 pairs of thoracic nerves connect to back and abdominal muscles and intercostal muscles

Lumbar region

Five pairs of lumbar nerves form network to serve lower abdomen, thighs, and legs

Sacral region

Six pairs of sacral nerves connect to legs, feet, and anal and genital areas

DERMATOMES

Spinal nerves contain a special fiber, the dorsal root, that sends sensory information from the skin to the brain. All but one pair of spinal nerves serves a specific area of the body, or dermatome. Nerve fibers in contact with skin receptors join up along the network of fibers in one dermatome to form the relevant dorsal root, which enters the spinal cord and conveys sensory impulses from that dermatome to the brain.

MAP OF DERMATOMES

This map shows the 30 dermatomes of the body. Each zone is served by a corresponding pair of spinal nerves.

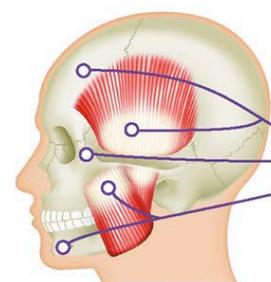
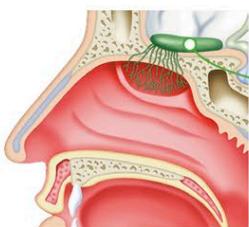


CRANIAL NERVES

There are 12 pairs of cranial nerves that are linked directly to the brain and do not enter the spinal cord. They allow sensory information to pass from the organs of the head, such as the eyes and ears, to the brain and also convey motor information from the brain to these organs—for example, directions for moving the mouth and lips in speech. The cranial nerves are named for the body part they serve, such as the optic nerve for the eyes, and are also assigned Roman numerals, following anatomical convention.

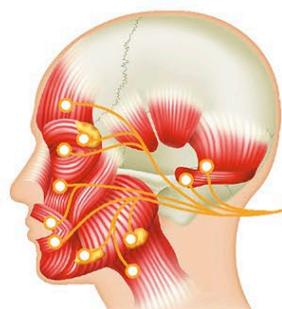
Olfactory nerve (I, sensory)

Smell molecules in nasal cavity trigger nerve impulses that pass along this nerve to olfactory bulb, then on to limbic areas (see pp.64–65) of brain



Trigeminal nerve (V, two sensory and one mixed branch)

Ophthalmic and maxillary branches of this nerve convey signals from eyes, teeth, and face, and other sensory fibers carry impulses from lower jaw; motor fibers control muscles involved with chewing

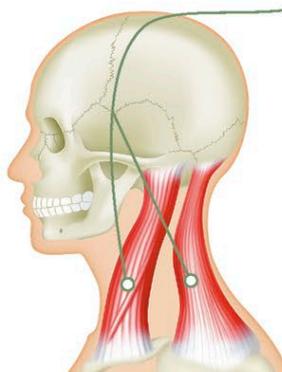


Facial nerve (VII, mixed)

Sensory fibers collect information from taste buds at front two-thirds of tongue; motor fibers are predominantly responsible for muscle movements controlling facial expression and also function of salivary gland and lacrimal gland, which secretes tears and lubricates the surface of the eye and conjunctiva of the eyelid

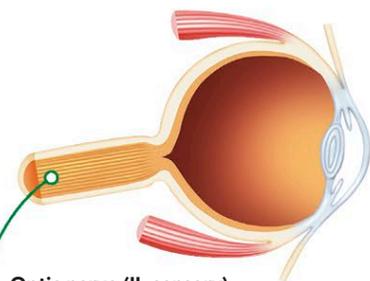
CRANIAL NERVE CONNECTIONS

The cranial nerves I and II connect to the cerebrum, while cranial nerves III to XII connect to the brainstem. The fibers of sensory cranial nerves each project from a cell body that is located outside the brain itself, in sensory ganglia or elsewhere along the trunks of sensory nerves.



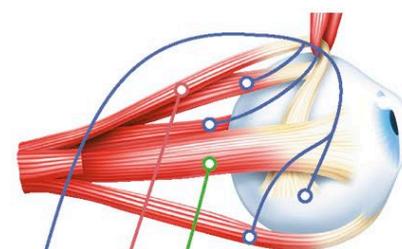
Spinal accessory nerve (XI, mixed)

Motor functions responsible for muscles and movements of head, neck, and shoulders; also stimulates muscles of larynx and pharynx, which are involved in swallowing; sensory functions unknown



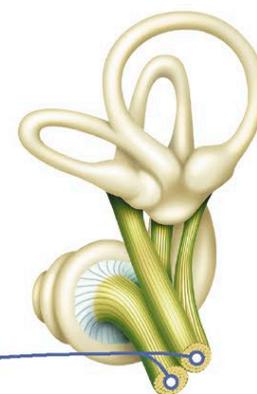
Optic nerve (II, sensory)

Visual information from retina is conveyed to brain by optic nerve at back of eye; optic nerves from both eyes meet at point known as optic chiasm, then signals from both visual fields are sent to opposite sides of brain



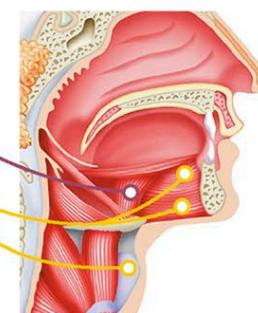
Oculomotor, trochlear, and abducens nerves (III, IV, VI, motor)

Three nerves regulating voluntary movements of eye muscles, allowing movement of eyeball and eyelids; oculomotor nerve also allows for pupil constriction



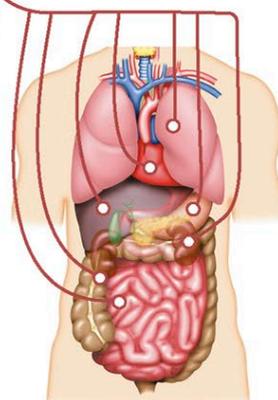
Vestibulocochlear nerve (VIII, sensory)

Vestibular branch of this nerve collects information from inner ear about head orientation and balance; cochlear branch is concerned with sound and hearing signals from ear



Glossopharyngeal and hypoglossal nerves (IX, XII, both mixed)

Motor fibers of these nerves control most of the muscles involved with tongue movement and swallowing; sensory fibers convey information on taste, touch, and temperature from tongue and pharynx and can trigger gag reflex if stimulated



Vagus nerve (X, mixed)

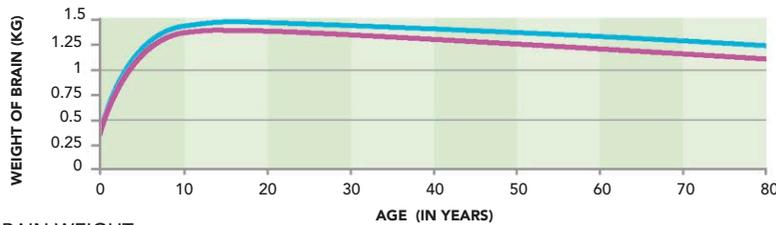
Longest and most branched of all cranial nerves, with autonomic, sensory, and motor fibers; serves lower part of head, throat, neck, chest, and abdomen, and plays role in many functions, including swallowing, breathing, heartbeat, and production of stomach acid

BRAIN SIZE, ENERGY USE, AND PROTECTION

THE BRAIN ACCOUNTS FOR AROUND 2 PERCENT OF TOTAL BODY WEIGHT, BUT CONSUMES A DISPROPORTIONATE AMOUNT OF FUEL TO SUPPORT ITS MANY ACTIVITIES. IT HAS SEVERAL FORMS OF PROTECTION—THE LAYERS OF MEMBRANE SURROUNDING IT, A BONY SKULL, AND FLUID PRODUCED IN ITS CHAMBERS (VENTRICLES) TO ABSORB THE IMPACT OF SHOCKS.

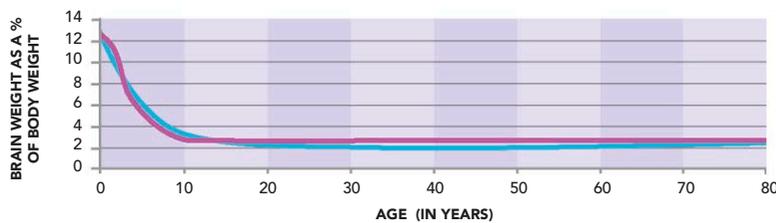
WEIGHT AND VOLUME

The average adult human brain weighs about 3¼ lb (1.5kg). Its volume and shape are similar to those of an average-sized cauliflower, and the consistency of its tissues is similar to stiff jelly. The size of a person's brain bears little relation to his or her intelligence, and every brain, whatever its weight and volume, has roughly the same number of neurons and synapses. After the age of 20 or so, brain mass decreases by about ½ oz (1g) per year. New neurons are made throughout life, but not enough to replace those that die off with age. This is generally no cause for concern, as there are plenty of neurons left to carry out the brain's functions.



BRAIN WEIGHT

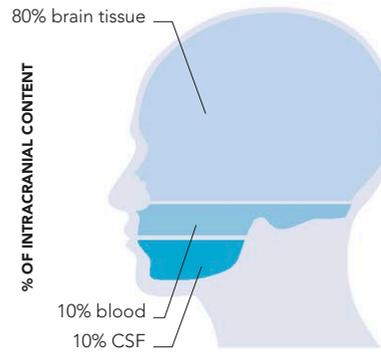
The brain's weight increases from birth and reaches its maximum during adolescence. The number of neurons is fixed in infancy but, as the body grows, they grow in size and form new connections. The male brain is consistently heavier than the female brain from birth.



BRAIN WEIGHT AND BODY WEIGHT

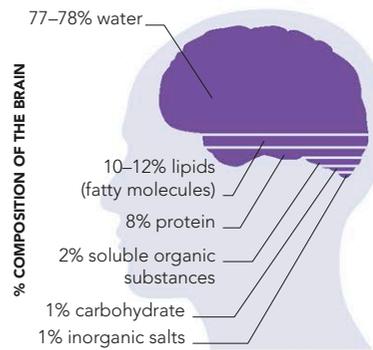
This graph shows brain weight as a percentage of total body weight over the course of a lifetime. Proportionally, a baby's brain is around six times larger than an adult's. Despite being lighter than the male brain overall, the female brain after the age of 13 is actually heavier than the male brain as a proportion of the entire body's weight.

KEY
— FEMALE
— MALE



INTRACRANIAL CONTENT

Brain tissue comprises gray and white matter, which consist of neurons and supporting glial cells respectively. A series of ventricles is filled with cerebrospinal fluid (CSF) and the brain is also richly supplied with blood vessels.

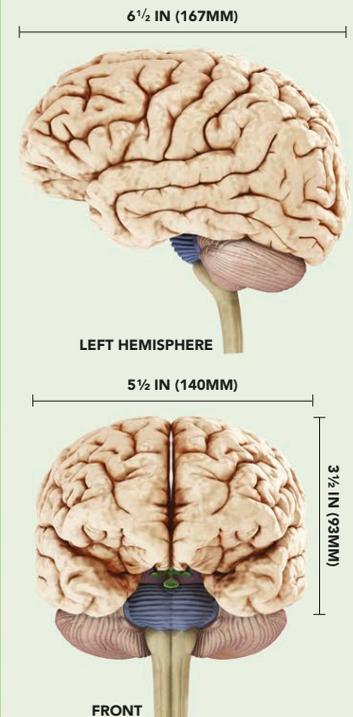


COMPOSITION OF THE BRAIN

The brain consists mainly of water, which occurs in the cytoplasm of neurons and glial cells, as well as being a major constituent of blood. The brain is also rich in lipids—fatty molecules that make up cell membranes.

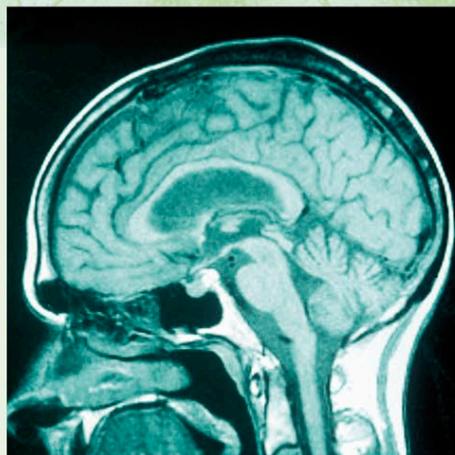
LENGTH, WIDTH, AND HEIGHT

The brain is housed within the intracranial cavity, so measurements of the skull effectively relate to the size of the brain. The actual length, width, and height of an individual human brain can be measured using MRI scanning. There is considerable variation in the size of the adult human brain, but the average dimensions are given against the diagrams below. Bear in mind that, because of the numerous complex folds within the cerebrum, the brain has a much larger surface area than is apparent from its overall shape.



BRAIN VOLUME AND LIFESTYLE

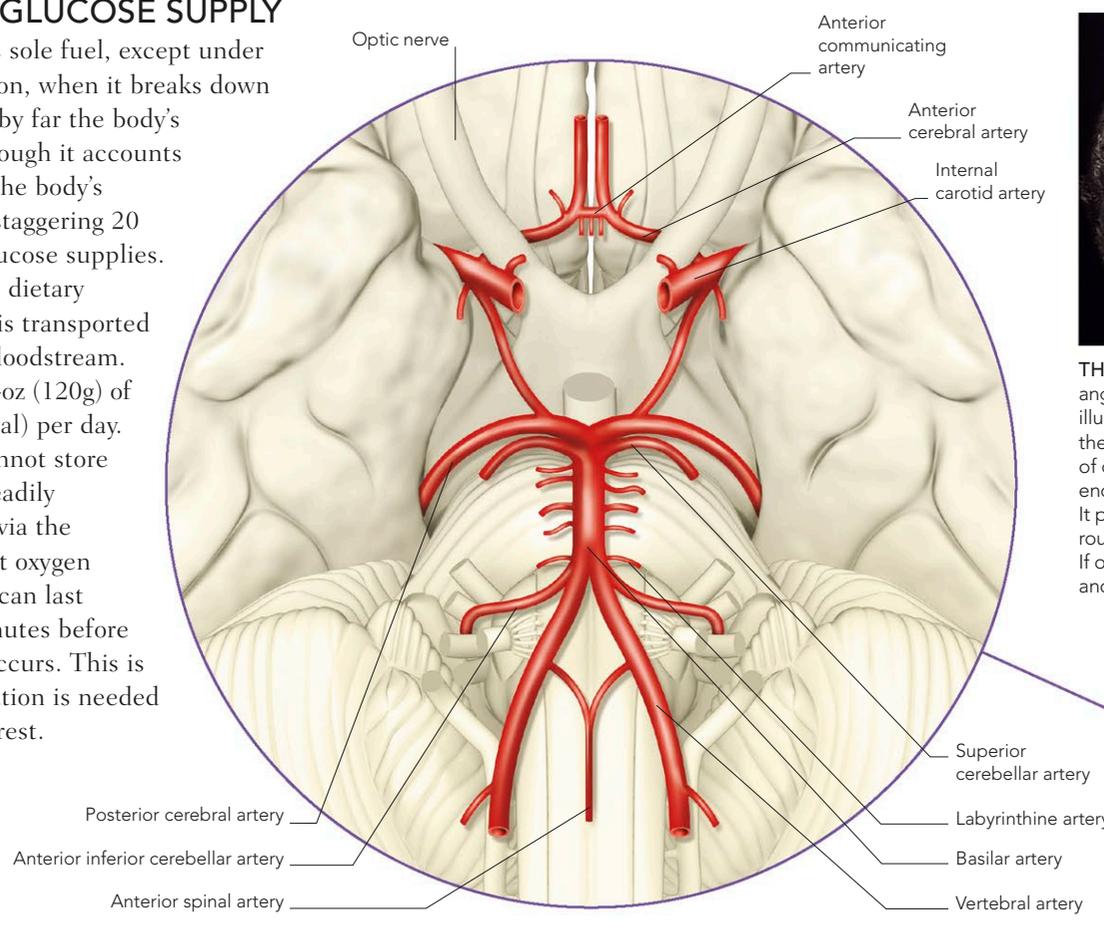
A recent study linked alcohol consumption to brain shrinkage. Participants disclosed their drinking habits and MRI scanning was used to measure each person's ratio of brain volume to skull size. It was found that abstainers had greater brain volumes than former drinkers, light drinkers, moderate drinkers, or heavy drinkers. On average, abstainers had 1.6 percent greater brain volume than heavy drinkers. Interestingly, the effects were most marked among elderly women. In another study, participants between the ages of 60 and 79 took up either regular aerobic exercise or toning and stretching exercises for six months. MRI scans of each participant taken both before and after the six-month period showed an increase in the brain volumes of those doing aerobic exercise, suggesting that aerobic exercise can help maintain the health of the brain in older adults.



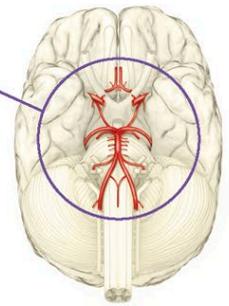
ALCOHOLISM AND BRAIN ATROPHY
 Alcoholism can lead to cerebellar degeneration as shown above. The low quality of the scan was due to the man's withdrawal symptoms, preventing him from sitting still.

OXYGEN AND GLUCOSE SUPPLY

Glucose is the brain's sole fuel, except under conditions of starvation, when it breaks down protein. The brain is by far the body's hungriest organ. Although it accounts for just 2 percent of the body's weight, it requires a staggering 20 percent of its total glucose supplies. This is obtained from dietary carbohydrate, which is transported to the brain via the bloodstream. It consumes roughly 4oz (120g) of glucose (about 420kcal) per day. Because the brain cannot store glucose, it must be readily available at all times via the blood supply. Without oxygen or glucose, the brain can last for only about 10 minutes before irreparable damage occurs. This is why prompt resuscitation is needed in cases of cardiac arrest.

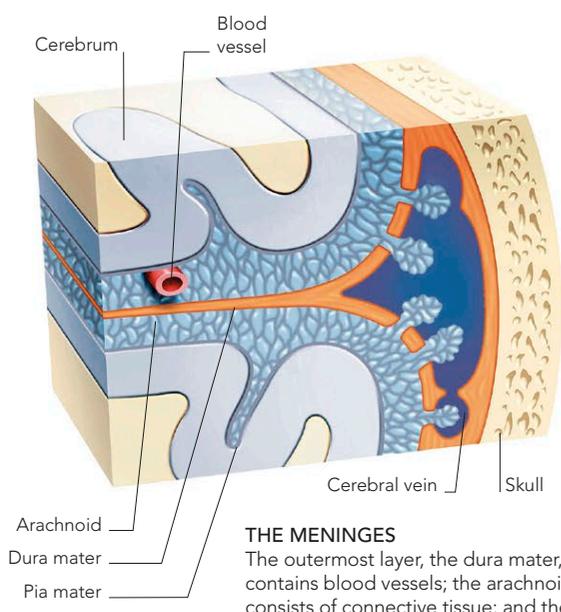


THE CIRCLE OF WILLISZThe angiogram above and the illustration to the left show the Circle of Willis, a ring of communicating arteries encircling the base of the brain. It provides the brain with supply routes for glucose and oxygen. If one route becomes blocked, another one compensates for it.



PROTECTING THE BRAIN

The brain has several defense mechanisms to protect it from damage. The bony skull acts as a box, containing the brain and buffering it against blows. The meninges are three layers of membranes that line the skull, enclosing the brain and providing extra layers of protection between the skull and the brain. Cerebrospinal fluid circulates within the brain, nourishing brain tissue and working as a shock absorber to reduce the impact of knocks.



THE MENINGES The outermost layer, the dura mater, contains blood vessels; the arachnoid consists of connective tissue; and the pia mater lines the brain's contours.

CEREBROSPINAL FLUID FLOW

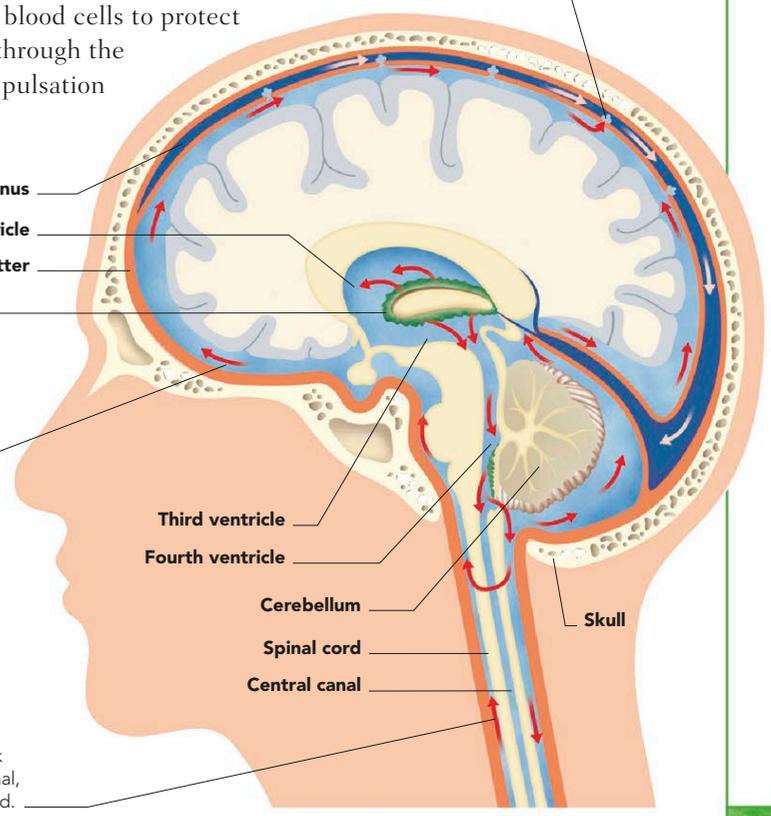
Brain tissue floats in cerebrospinal fluid (CSF) within the skull. CSF absorbs shocks from blows to the brain. It is produced in a series of connected chambers within the brain known as the ventricles, and is renewed four to five times per day. It contains proteins and glucose to nourish brain cells, as well as white blood cells to protect against infection. It moves through the ventricles, propelled by the pulsation of the cerebral arteries.

4 Site of reabsorption (arachnoid granulations) After traveling around the brain, the fluid is finally reabsorbed into the bloodstream through tiny arachnoid granulations (projections from the arachnoid layer of the meninges into the sagittal sinus).

1 Site of fluid production (choroid plexus) CSF is produced in the clusters of thin-walled capillaries (the choroid plexus) that line the walls of the ventricles.

2 Direction of flow CSF flows from the lateral ventricles into the third and fourth ventricles. It then continues up the back of the brain, down around the spinal cord, and to the front of the brain, as indicated by the arrows.

3 Circulation around spinal cord Helped by vertebral movement, fluid travels downward along the back of the spinal cord, into the central canal, and upward along the front of the cord.





CIRCLE OF WILLIS

The major arteries of the brain can be seen in this MRI scan. They include the Circle of Willis (below center) at the base of the brain, where arteries from the neck meet before branching.



OXYGEN SUPPLY

This arteriograph shows arteries carrying oxygen-rich blood to the brain. The arrangement of the arteries allows blood to be supplied by another route if one of the pathways is blocked.

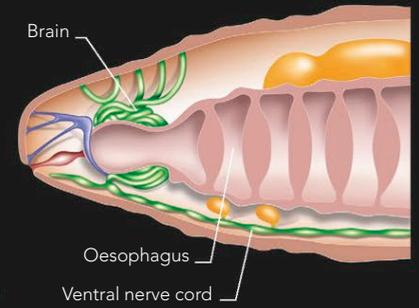
EVOLUTION

BRAINS EVOLVED TO ENABLE ANIMALS TO RESPOND TO ENVIRONMENTAL CHANGES. THE HUMAN BRAIN HAS EVOLVED TO ITS PRESENT COMPLEXITY THROUGH SEVERAL STAGES, MANY OF WHICH ARE COMMON TO ALL ANIMALS. ITS ORIGINS CAN BE SEEN IN THE BRAINS OF OTHER SPECIES, IN WHICH MORE PRIMITIVE STRUCTURES REMAIN.

EVOLUTION OF THE INVERTEBRATE BRAIN

All animals have to respond to changes in their internal and external environment in order to survive. To do this, they have evolved cells that are sensitive to stimuli such as light and to vibrations. The sensory cells are, in turn, connected to other cells that can move the organism or change its state in response to the stimulus. This system of interconnected nervous tissue is a crude form of brain. In invertebrates, such as worms, the nervous system is distributed throughout the creature's body, as a loose network of reactive fibers. Some of these networks contain small masses of nerves, known as ganglia. These are the forerunners of the structures that, in some species, have become the central nervous system or brain.

PRIMITIVE NERVOUS SYSTEM
The simplest system, as seen in this hydra (a tiny aquatic invertebrate), consists of a loose network of sensory cells with clumps of interconnected cells called ganglia.



EARTHWORM BRAIN
The earthworm has a crude brain, the cerebral ganglion, which is connected to a cord of nervous tissue (the ventral nerve cord) that runs the length of its body. Nerve fibers from the cord extend into each segment, so muscle contraction along the body can be coordinated to produce movement in response to stimuli.

EVOLUTION OF THE VERTEBRATE BRAIN

Through the course of evolution, the brain has undergone considerable changes. Compared to the primitive nervous systems of invertebrates, the brain of vertebrates is a well-developed, highly interconnected organ. The central nervous system is connected to the rest of the body by a peripheral nervous system that includes the fibers running to and from the sensory organs. The basic vertebrate brain—also sometimes referred to as the “reptilian brain”—

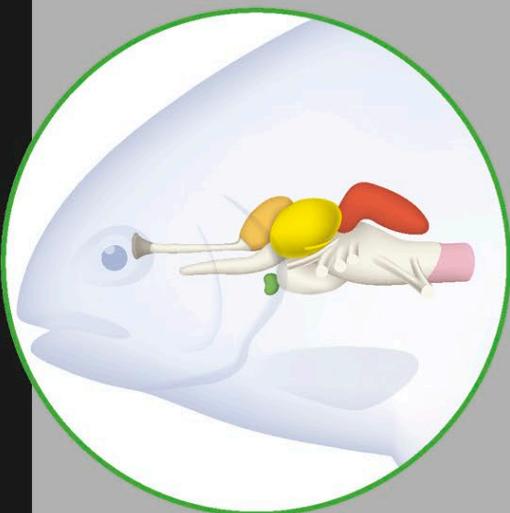
consists of the cluster of nuclei that lies just above the brainstem in humans. They include the modules that produce arousal, sensation, and reaction to stimuli. It is unlikely, however, that these nuclei alone are sufficient to produce consciousness. This basic vertebrate brain does not include more advanced features, such as the limbic system or cerebral cortex, which exist only in the brains of mammals.

KEY TO VERTEBRATE BRAIN AREAS

- Cerebellum
- Optic lobe
- Cerebrum
- Pituitary gland
- Medulla
- Olfactory bulb

FISH

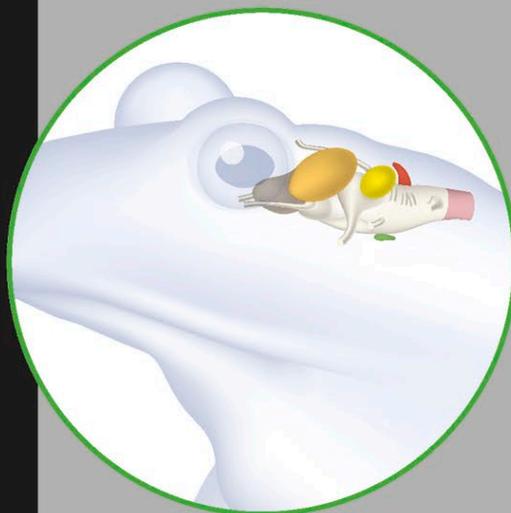
A fish's cerebrum receives sensory signals from the sense organs and combines them with information from the internal organs and nerves to guide action. Fish have a large cerebellum in order to coordinate movement and gauge pressure.



FISH

AMPHIBIANS

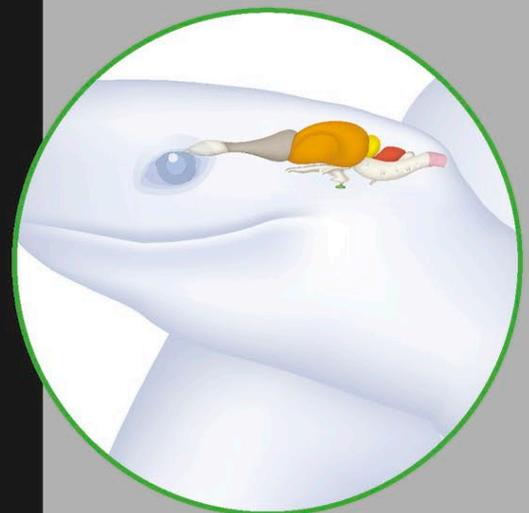
The amphibian brain resembles the fish brain except that the cerebrum is roofed over with nervous tissue. The main function of this region is to perceive smell, as reflected by the large olfactory bulb. The forebrain is much larger than the cerebellum.



FROG

REPTILES

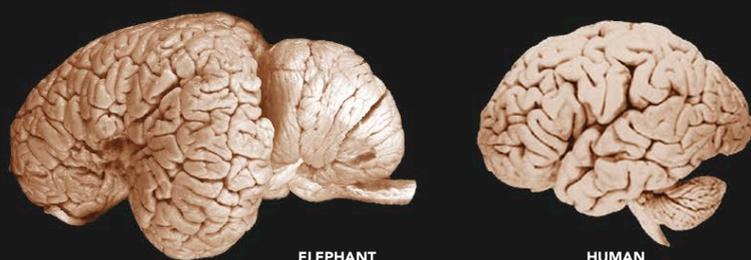
Modern reptiles show greater development in the basal parts of the forebrain, and the cerebrum is much larger than the optic lobe. The olfactory bulb is large in comparison with the other structures of the brain and is well developed.



TURTLE

MAMMAL BRAINS

The mammalian brain comprises a cluster of structures that evolved on top of the basic vertebrate brain, known as the limbic system, and a wrinkled covering called the cortex, which interconnects with the limbic structures beneath. The limbic system is the part of the brain that produces emotions. These are responses to stimuli that go beyond the basic “grab” or “avoid” reactions in the vertebrate brain, and produce subtle and complex actions that are not always predictable. The limbic system also contains structures that encode experiences as memories, to be recalled for use in guiding future actions. The emotional and memory faculties greatly increase the range and complexity of behavior that a mammal displays, because it is not governed purely by instinct.



ELEPHANT

HUMAN

BRAIN SIZE AND SHAPE

One striking aspect of mammalian brain evolution is the development of the cortex. This outer layer has evolved to serve the particular needs of each species, and therefore varies dramatically between one animal and another. A few mammals, such as humans, elephants, and dolphins, have a disproportionately large cortex compared to most mammals.



DOLPHIN



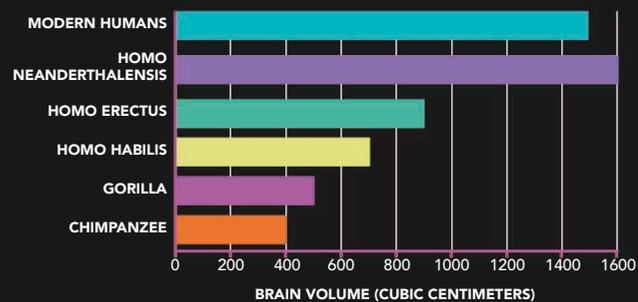
WOLF



CAT

HOMINID BRAINS

The brains of hominids (modern humans and their ancestors) underwent a surge of evolutionary changes that left them, in some ways, distinctly different even from their near relatives, such as chimpanzees and gorillas. The main distinction between human and other mammalian brains is the size and density of the cortex, and particularly of the frontal lobe, which is responsible for complex thought, conscious judgement, and self-reflection. No one knows why the human brain evolved as it did—it may have been due to some change in diet forced by the environment, or the product of living in groups (see p.138) that depended on close interdependence for survival.



DOES SIZE MATTER?

The growth of the human brain over the course of evolution is thought to be the reason why we are so dominant. However, size is not the only factor that matters for intelligence or survival—the way brains are wired up may be more important. Neanderthals had bigger brains than humans, but were less innovative and were finally superseded by other hominids.



NEANDERTHAL SKULL

BIRDS

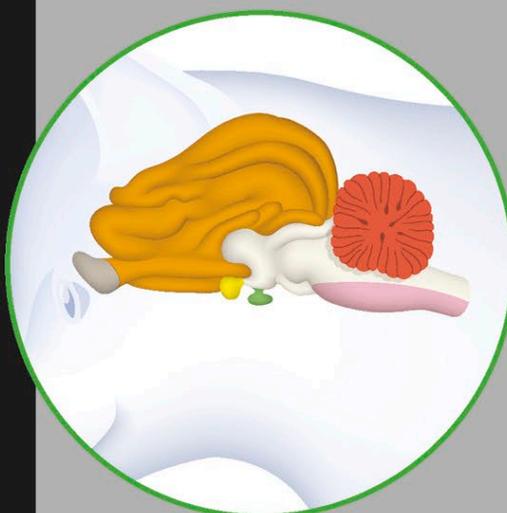
Birds' brains are similar to those of reptiles except that the cerebellum is highly developed to control balance and position in flight. Despite the size of the olfactory bulb, most birds have a poor sense of smell, with some exceptions, such as the kiwi.



THRUSH

MAMMALS

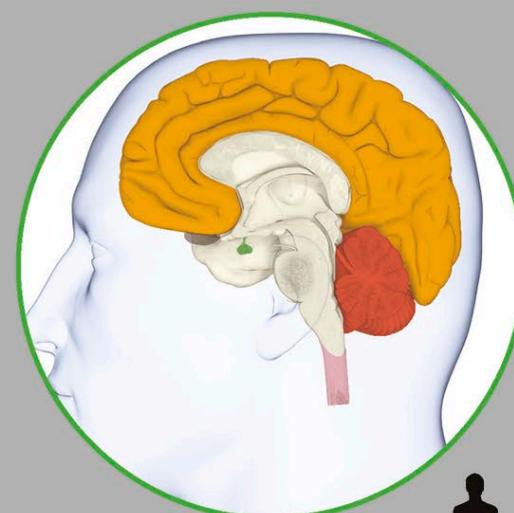
In mammals, the cerebellum is relatively small compared to the forebrain. The cerebrum is covered in wrinkled cortex; these wrinkles allow a greater volume of cortex to fit into the skull, compared to the smooth surface of the reptilian brain.



CAT

MAN

The human brain is completely dominated by the cerebrum, and the cortex is intricately folded to allow the maximum amount to be contained in the skull. The cerebellum remains large and active, however, to enable complex motor activity.

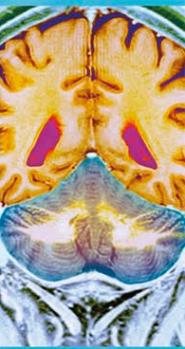


HUMAN



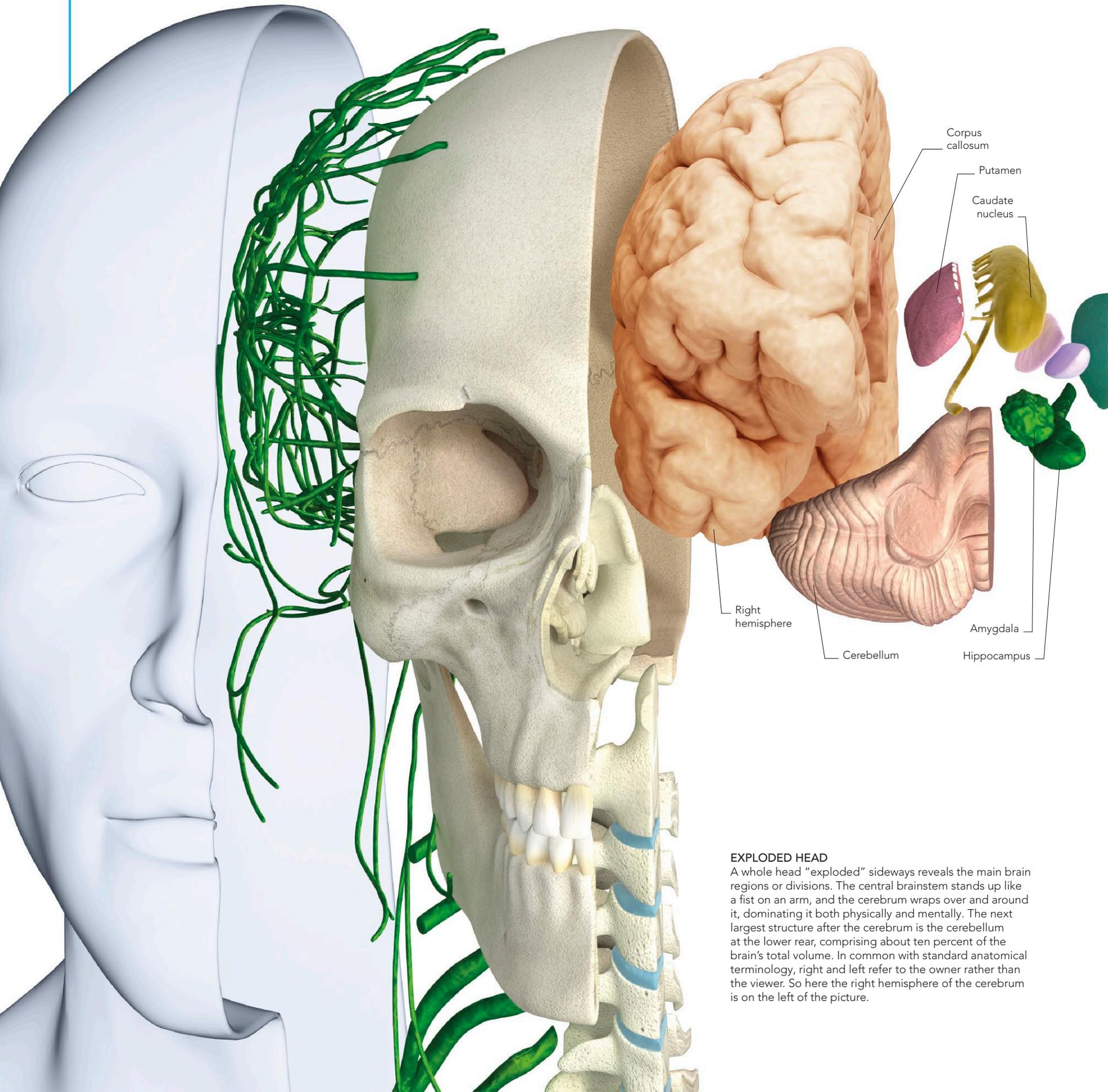
BRAIN ANATOMY IS HIDDEN, SECRET, AND MORE COMPLEX THAN ANY OTHER PART OF THE BODY. THE BASIC BUILDING BLOCK OF THE BRAIN IS THE CELL. SIGNALING CELLS KNOWN AS NEURONS FORM LARGER STRUCTURES CALLED NUCLEI THAT CARRY OUT PARTICULAR FUNCTIONS. THEY ALSO CLUSTER TOGETHER TO FORM THE THICK, LAMINATED SHEET OF GRAY MATTER FORMING THE COVERING OF THE BRAIN CALLED THE CORTEX. DEEP FISSURES IN ITS SURFACE DIVIDE THE BRAIN INTO TWO HALVES (THE HEMISPHERES), EACH WITH FIVE LOBES. THESE MAJOR DIVISIONS "SPECIALIZE" IN DIFFERENT TASKS, BUT ALSO INTERCONNECT AND INTERACT.

BRAIN ANATOMY



BRAIN STRUCTURES

THE BRAIN HAS A COMPLEX AND MANY-LAYERED ANATOMY. PEELING BACK THE DOMINANT CEREBRAL HEMISPHERES REVEALS A FURTHER SET OF STRUCTURES WITHIN. SOME ARE DISCRETE MASSES, SUCH AS THE CEREBELLUM AND THALAMUS, WHILE OTHERS ARE ZONES OF NERVE FIBERS OR NERVE CELLS WITHIN LARGER STRUCTURES, DISCERNIBLE ONLY BY MICROSCOPIC EXAMINATION.



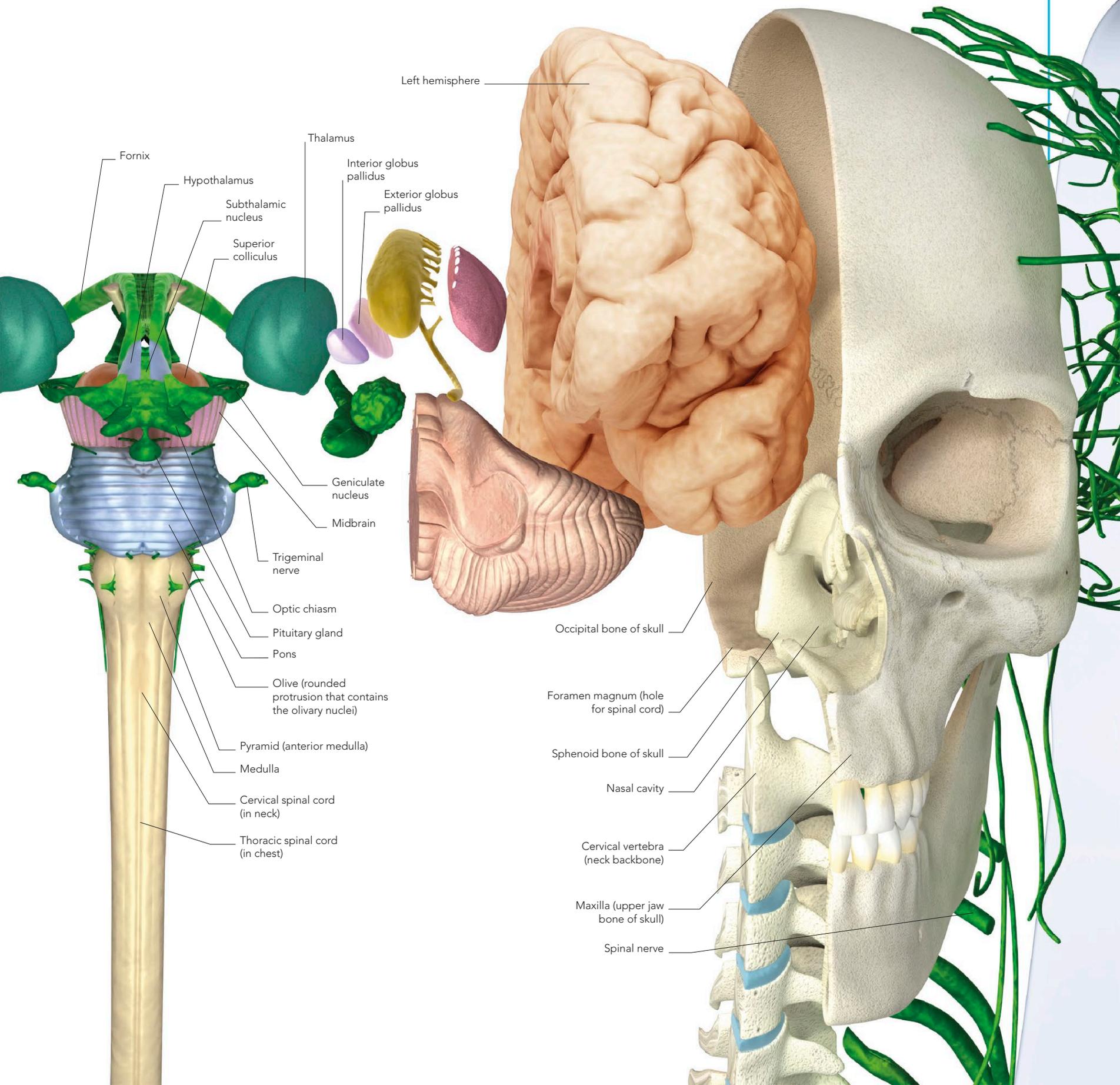
EXPLODED HEAD

A whole head "exploded" sideways reveals the main brain regions or divisions. The central brainstem stands up like a fist on an arm, and the cerebrum wraps over and around it, dominating it both physically and mentally. The next largest structure after the cerebrum is the cerebellum at the lower rear, comprising about ten percent of the brain's total volume. In common with standard anatomical terminology, right and left refer to the owner rather than the viewer. So here the right hemisphere of the cerebrum is on the left of the picture.

THE BRAIN HIERARCHY

The brain's major parts can be classified or categorized in several ways. In all of these systems, the dominant part is the cerebrum, the large pinky-gray wrinkled structure that forms more than three-quarters of the brain's total volume. The cerebrum is divided into left and right hemispheres, which are linked by a "bridge" of nerve fibers, the corpus callosum. The cerebrum, which includes the hippocampus and amygdala, is also known as the telencephalon. Together with the parts

it wraps around—the thalamus, hypothalamus, and associated parts, collectively known as the diencephalon—it comprises the major brain "division" known as the forebrain (prosencephalon). Below the forebrain is the midbrain (mesencephalon), a small division that includes groups of nerve-cell bodies known as nuclei, such as the basal ganglia. Below the midbrain is the hindbrain (rhombencephalon), with the pons as its uppermost part, and beneath it the cerebellum and the medulla, which tapers to merge with the spinal cord.



SCALP SKIN

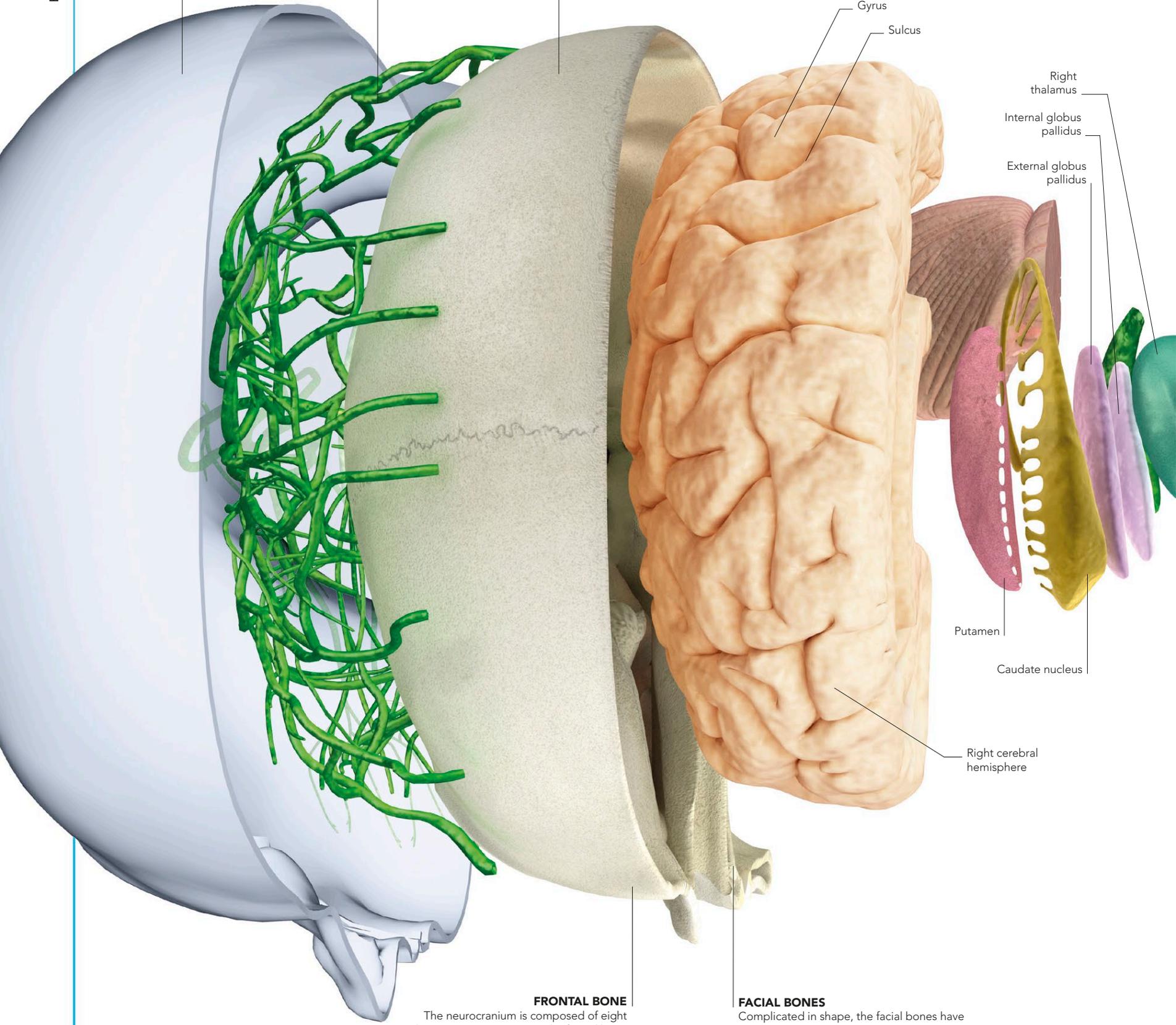
The skin of the scalp has only a thin underlying layer of subcutaneous fat and the hard skull is just beneath, so it wounds relatively easily and bleeds copiously.

SCALP NERVES

Many small peripheral nerves branch through and under the scalp skin from cranial nerves II, III, and V. Even faint contact registers, allowing us to react quickly and avoid injury.

SKULL

The upper domed part of the skull, called the neurocranium, forms a "braincase" to shield against knocks and jolts. This function is aided by the meninges (see p.56).

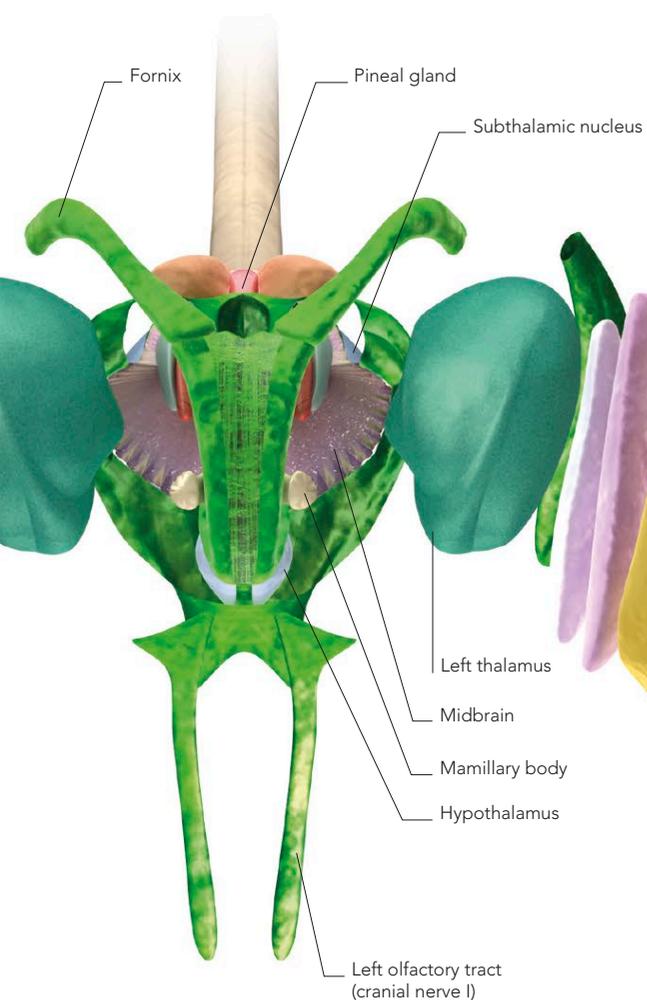


FRONTAL BONE

The neurocranium is composed of eight bones. Most prominent is the frontal bone under the forehead. The left and right parietals are behind it, the occipital below them at the lower rear, and the two temporals on the lower sides. The sphenoid and ethmoid bones are at the lower front, behind the nose area.

FACIAL BONES

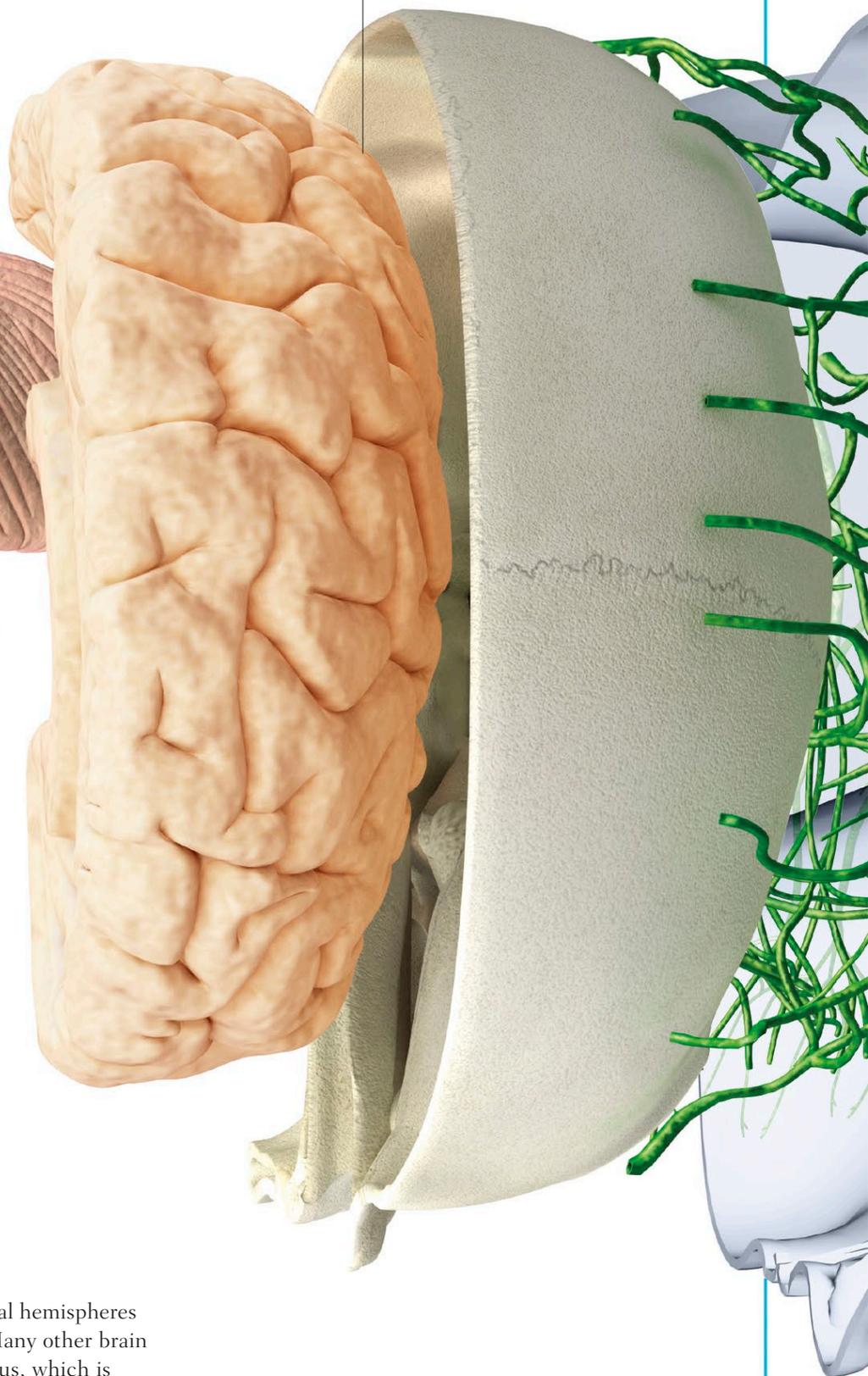
Complicated in shape, the facial bones have gaps (foramina) in them. Some allow cranial nerves to pass from the brain within the neurocranium, out to the nasal epithelium in the nose cavity, the eyes in their sockets, the inner ear, and other sensory parts. Blood vessels have similar sets of skull foramina.

**CEREBELLUM**

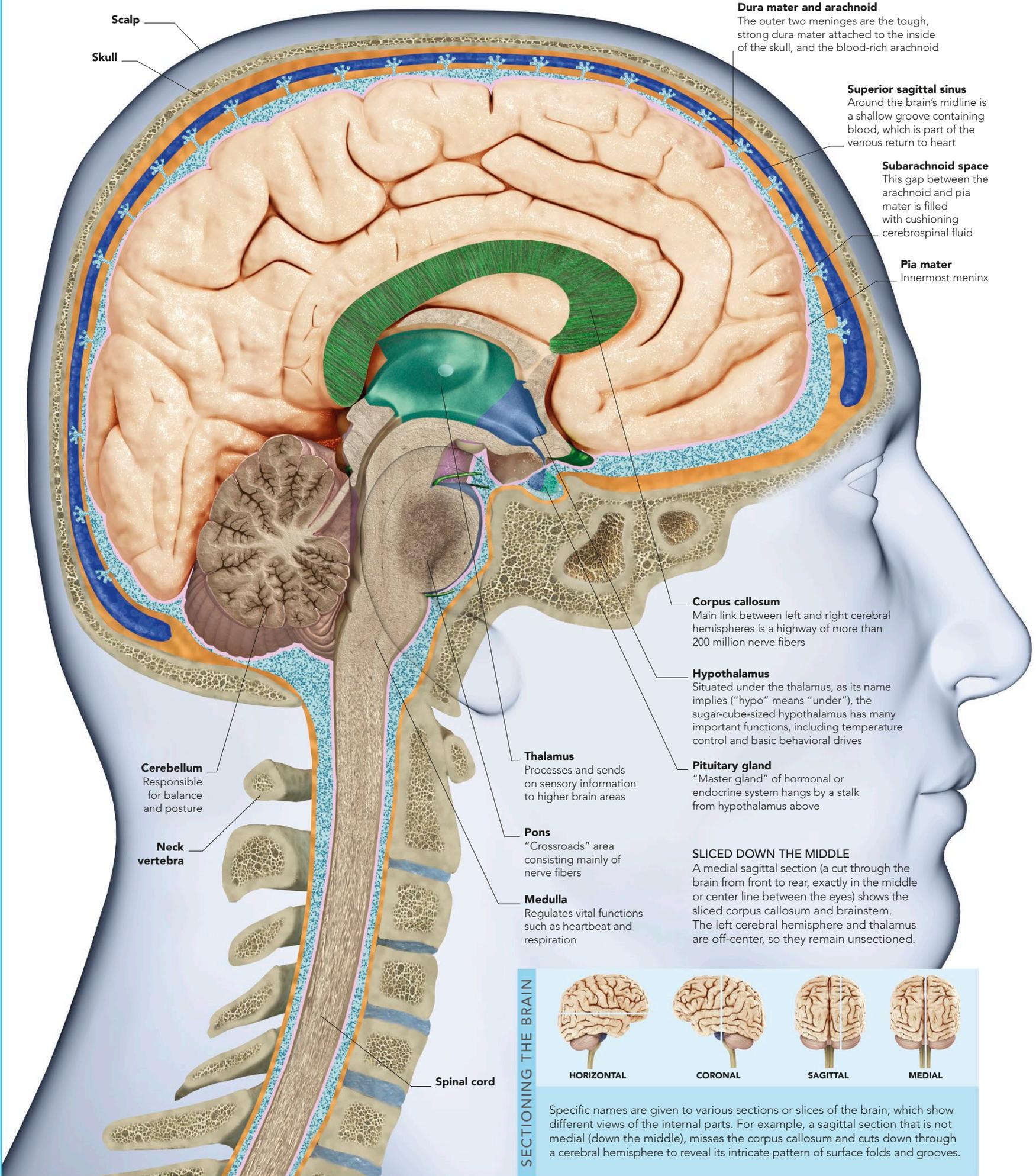
This name means “little brain,” referring to the pattern of grooves and bulges on the cerebellar surface, which reflects the external appearance of the cerebrum. The cerebellum is connected to the brainstem immediately in front of it by three pairs of thick, short, stalklike extensions, called the cerebellar peduncles.

CEREBRAL CORTEX

The thin grayish covering of each cerebral hemisphere is called the cerebral cortex. It has a characteristic pattern of bulges (gyri), shallower grooves (sulci), and deeper ones (fissures).

**LEFT AND RIGHT HEMISPHERES**

An overhead view of the “exploded” brain shows how the two cerebral hemispheres can be neatly separated by cutting through the corpus callosum. Many other brain structures are symmetrically paired in this way, such as the thalamus, which is sometimes described as “two hen’s eggs sitting side by side.” The cerebellum at the lower rear of the brain is accommodated within a bowl-like cavity of the skull known as the posterior cranial fossa. The cranial nerves (numbered I to XII, see p.43) enter the brain directly rather than connecting to the spinal cord.



Dura mater and arachnoid
The outer two meninges are the tough, strong dura mater attached to the inside of the skull, and the blood-rich arachnoid

Superior sagittal sinus
Around the brain's midline is a shallow groove containing blood, which is part of the venous return to heart

Subarachnoid space
This gap between the arachnoid and pia mater is filled with cushioning cerebrospinal fluid

Pia mater
Innermost meninx

Corpus callosum
Main link between left and right cerebral hemispheres is a highway of more than 200 million nerve fibers

Hypothalamus
Situated under the thalamus, as its name implies ("hypo" means "under"), the sugar-cube-sized hypothalamus has many important functions, including temperature control and basic behavioral drives

Pituitary gland
"Master gland" of hormonal or endocrine system hangs by a stalk from hypothalamus above

Thalamus
Processes and sends on sensory information to higher brain areas

Pons
"Crossroads" area consisting mainly of nerve fibers

Medulla
Regulates vital functions such as heartbeat and respiration

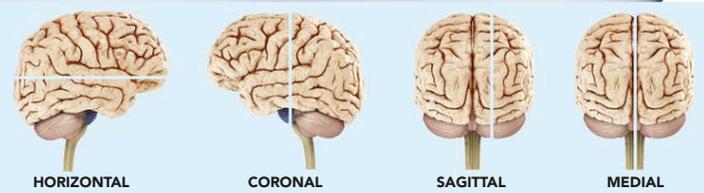
Cerebellum
Responsible for balance and posture

Neck vertebra

Spinal cord

SLICED DOWN THE MIDDLE
A medial sagittal section (a cut through the brain from front to rear, exactly in the middle or center line between the eyes) shows the sliced corpus callosum and brainstem. The left cerebral hemisphere and thalamus are off-center, so they remain unsectioned.

SECTIONING THE BRAIN



Specific names are given to various sections or slices of the brain, which show different views of the internal parts. For example, a sagittal section that is not medial (down the middle), misses the corpus callosum and cuts down through a cerebral hemisphere to reveal its intricate pattern of surface folds and grooves.

BRAIN ZONES AND PARTITIONS

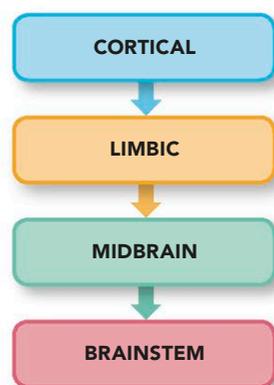
THE BRAIN'S PHYSICAL STRUCTURE BROADLY REFLECTS ITS MENTAL ORGANIZATION. IN GENERAL, HIGHER MENTAL PROCESSES OCCUR IN THE UPPER REGIONS, WHILE THE BRAIN'S LOWER REGIONS TAKE CARE OF BASIC LIFE SUPPORT.

VERTICAL ORGANIZATION

The uppermost brain region, the cerebral cortex, is mostly involved in conscious sensations, abstract thought processes, reasoning, planning, working memory, and similar higher mental processes. The limbic areas (see pp.64-65) on the brain's innermost sides, around the brainstem, deal largely with more emotional and instinctive behaviors and reactions, as well as long-term memory. The thalamus is a preprocessing and relay center, primarily for sensory information coming from lower in the brainstem, bound for the cerebral hemispheres above. Moving down the brainstem into the medulla are the so-called "vegetative" centers of the brain, which sustain life even if the person has lost consciousness.

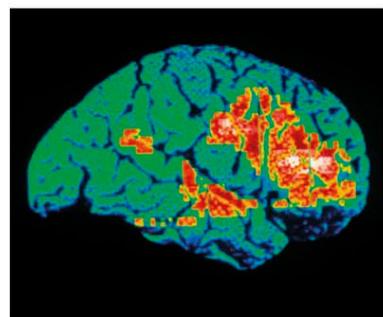
LESS CONSCIOUS, MORE AUTOMATIC

The brain's vertical zonation moves from high-level mental activity in the cerebral cortex gradually through to more basic or "primitive" lower functions, especially the autonomic centers of the medulla in the lower brainstem that deal with vital body functions, such as breathing and heartbeat.



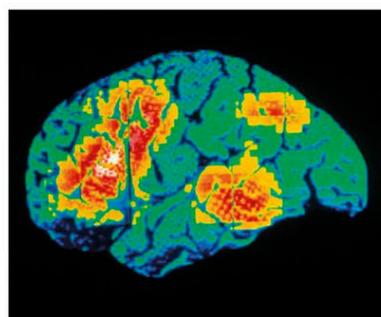
LEFT AND RIGHT

Structurally, the left and right cerebral hemispheres look broadly similar. Functionally, however, speech and language, stepwise reasoning and analysis, and certain communicating actions are based mainly on the left side in most people. Since nerve fibers cross from left to right at the base of the brain, this dominant left side receives sensory information from, and sends messages to, muscles in the right side of the body—including the right hand. Meanwhile, the right hemisphere is more concerned with sensory inputs, auditory and visual awareness, creative abilities, and spatial-temporal awareness (what happens in our surroundings, second by second).



LEFT-HANDED PERSON

In a PET brain scan where yellow and red show increasing activity, a left-handed person involved in word recognition has busy areas at the right front cerebral cortex.



RIGHT-HANDED PERSON

On the same test in a right-handed subject, the left side of the cortex shows a similar pattern, with activity largely in the frontal region and the temporal and parietal areas.

ANARCHIC HAND SYNDROME

In anarchic hand syndrome (AHS), a person has one hand that is no longer under conscious control and seems to move on its own, almost as if possessed by another intelligence. The problem is usually due to an abnormality in the motor center of the cortex on the opposite side of the brain to the hand. Nerve signals sent from here to control the hand do not register any conscious intention for the action.

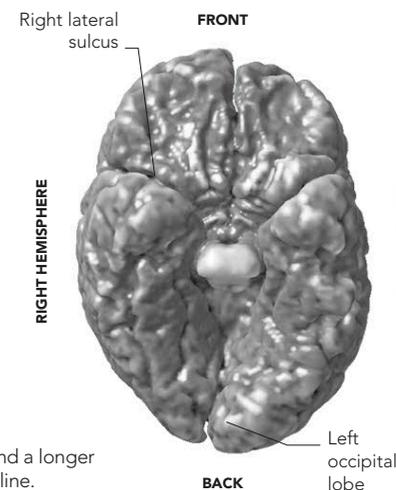
DR. STRANGELOVE

In this 1964 film the "hero" struggled with AHS as his leather-gloved right hand even tried to kill him.



THE ASYMMETRICAL BRAIN

In recent years, new and more accurate scanning techniques, especially MRI (see p.13), have shown that on average, brains are not as symmetrical in their left-right structure as was once believed. The scanning computer can be programmed to exaggerate any subtle departures from an exact mirror image. For example, near the lateral sulcus (Sylvian fissure), the part of the temporal lobe for understanding speech is slightly larger on the left than on the right. The lateral sulcus itself is also usually different in shape, being longer and less curved on the left than the right. This is partly due to a twisting effect known as Yakovlevian torque, which warps the right side of the brain forward.



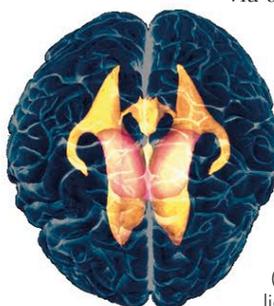
SEEN FROM BELOW

An asymmetry-enhanced MRI scan of the brain's underside reveals left-right differences, including a right frontal lobe that protrudes more than its counterpart, and a longer left occipital lobe that twists across the midline.

THE HOLLOW BRAIN

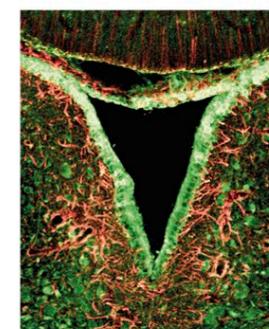
The brain has an internal system of chambers (ventricles), which are filled with a liquid—cerebrospinal fluid (CSF)—produced by the ventricle linings. The upper two chambers are the left and right lateral ventricles, one in each cerebral hemisphere, with hornlike forward- and side-facing projections. Small openings connect them to the third ventricle in the midbrain, which in turn links to the fourth ventricle in the pons and medulla. CSF flows slowly and continuously through the ventricles, then out

via small openings into the subarachnoid space around the brain and the spinal cord.



VENTRICLES

Two large lateral ventricles communicate along ducts with the third ventricle (yellow, upper center), which lies between and below them.



CEREBROSPINAL FLUID

CSF is made by the ventricle lining (green). It physically cushions the brain, distributes nutrients, and collects wastes.

THE NUCLEI OF THE BRAIN

IN THE BRAIN, NUCLEI ARE DISCRETE COLLECTIONS OF THE CELL BODIES OF NEURONS (NERVE CELLS). THEIR NERVE FIBERS OR AXONS SPREAD OUTWARD TO PROJECT, OR LINK, TO VARIOUS OTHER BRAIN PARTS. THE BRAIN HAS MORE THAN 30 SETS OF NUCLEI, MOSTLY PAIRED LEFT AND RIGHT.

GENERAL STRUCTURE

To the naked eye, most brain nuclei resemble “islands” of gray matter (nerve-cell bodies) within the white matter of nerve fibers. Many nuclei are unencapsulated—not contained within a membrane or covering—so they may lack sharp delineation from surrounding tissues. An older term for some of these nuclei is “ganglia.” However, this term is now usually reserved for similar structures in the peripheral nervous system, where groups of nerve-cell bodies are generally encapsulated into a discrete structure.

MAIN NUCLEI AND THEIR FUNCTIONS	
Basal	A system of nuclei (including some listed here) involved in motor control and learning.
Caudate	Involved in motor control and learning, especially processing feedback.
Subthalamic	Implicated in impulsive actions, including obsession–compulsion.
Thalamus	A major processing and relay area for inputs to the cerebral cortex (see pp.66–67).
Amygdala	Part of the limbic system, the amygdala is involved in learning, memory, and emotions.
Facial nucleus	One of several paired brainstem nuclei for cranial nerves, in this case nerve VII (facial).



CORPUS STRIATUM
This micrograph shows the nerve cell bodies (dark) and nerve fibers (pale) that make this brain region look striped or striated.

Together with the globus pallidus, or “pale sphere,” the putamen and caudate nuclei are known as the corpus striatum.

THE BASAL NUCLEI

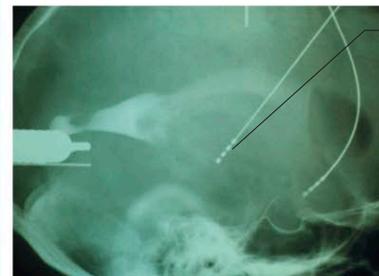
The basal nuclei (also known as the basal ganglia) is the collective name for several pairs of nuclei at the “base” of the cerebral hemispheres—adjacent to their inner surfaces, around and below the thalamus. They include the putamen, caudate nuclei, globus pallidus, subthalamic nuclei, and substantia nigra. The putamen and caudate nuclei are together called the dorsal striatum because of the striped or striated appearance of their tissues.

THE SUBTHALAMIC NUCLEI AND GLOBUS PALLIDUS

As the name implies, each one of the paired subthalamic nuclei is situated beneath the thalamus. They are also immediately above the substantia nigra. Each nucleus is about the size and shape of a partly squashed pea and is almost surrounded by nerve fibers passing to, from, or around it. Most of the incoming (afferent) nerve fibers are from the globus pallidus, along with some from the cerebral cortex and the substantia nigra. The majority of the outgoing (efferent) nerve fibers carry signals to the globus pallidus and the substantia nigra. The globus pallidus and the putamen are sometimes termed the lentiform or lenticular nucleus.

SUBSTANTIA NIGRA

The substantia nigra or “black substance” paired nuclei are among the lowest, or most basal, of the basal nuclei. Each is situated just beneath a subthalamic nucleus. The dark color that is characteristic of these nuclei is caused by the body pigment melanin (also found in the skin) that is part of the biochemical pathways involving the neurotransmitter dopamine. Degeneration of substantia nigra neurons is seen in Parkinson’s disease (see p.234).

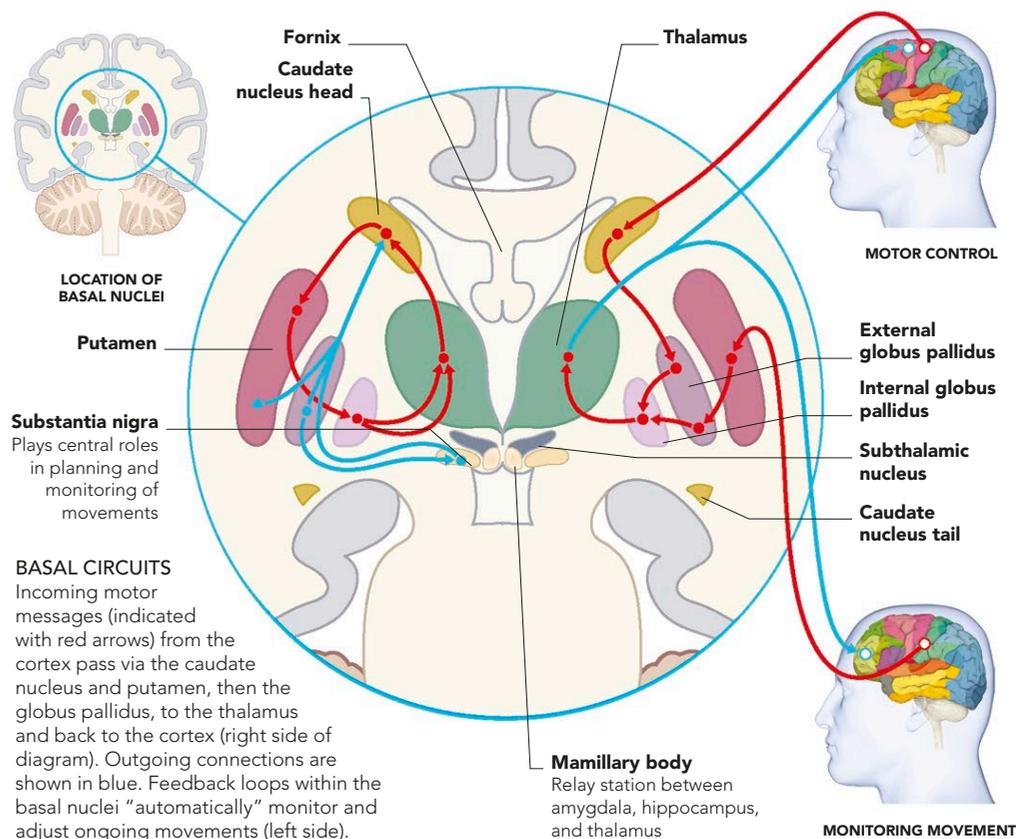


Electrode

STIMULATION
Deep brain stimulation of basal nuclei, such as the substantia nigra, using electrodes is part of research into and treatment for Parkinson’s.

CONNECTIONS AND FUNCTIONS

Most brain nuclei have multiple nerve connections, both inputs and outputs, and carry out wide-ranging functions. The C-shaped caudate nuclei above and to the side of the thalamus, and next to the lateral ventricle, have a head part, main body, and tapering tail. They are involved in motor (muscle) control and also in learning and memory. The rounded putamen, the outermost of the main basal ganglia, partly follows the shape of the caudate nucleus and is intricately linked anatomically to it. It, too, is heavily involved in motor control and movements, and in learning. The putamen has major nerve connections with the globus pallidus and substantia nigra. All of the basal ganglia work together as an integrated brain system to help ensure that physical movements are smooth and coordinated. Problems with one or more of the nuclei can lead to movement disorders such as tremors, tics, Parkinson’s disease (see p.234), Tourette syndrome (see p.243), and Huntington’s disease (see p.234). The subthalamic nuclei also have roles in impulsive actions and movement intentions.

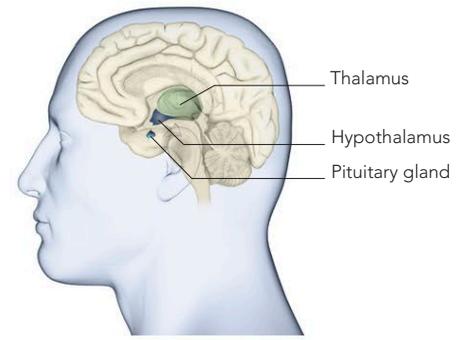




THE HIPPOCAMPUS
A micrograph of stained hippocampal tissue shows cellular organization that is similar to that in various brain nuclei. The neuron bodies are red, the axons (fibers) and other projections are blue. The glial cells, which provide support and nourishment, are green.

THE THALAMUS, HYPOTHALAMUS, AND PITUITARY GLAND

THE THALAMUS IS SITUATED AT THE ANATOMICAL CORE OF THE BRAIN. ITS POSITION MAKES IT PERFECTLY SITUATED TO ACT AS A RELAY STATION BETWEEN THE SENSE ORGANS AND THE BRAIN. SITTING BENEATH THE THALAMUS, THE HYPOTHALAMUS AND THE PITUITARY GLAND LINK THE CENTRAL NERVOUS SYSTEM AND THE ENDOCRINE SYSTEM.



LOCATOR

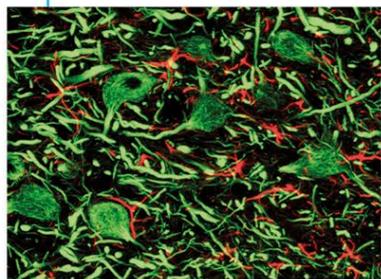
THE THALAMUS

Paired, egg-shaped masses that sit side by side make up the thalamus. In a typical brain, each mass is about 1¼ in (3cm) long and ½ in (1.5cm) across. There are no direct nerve connections from one mass to the other—in fact, the fluid-filled chamber of the third ventricle lies between them. The thalamus is the major relay station for nerve signals coming from all the senses except smell. It screens, sorts, and preprocesses this continuing torrent of sensory information and sends it on to the cerebral cortex.

INSIDE THE THALAMUS

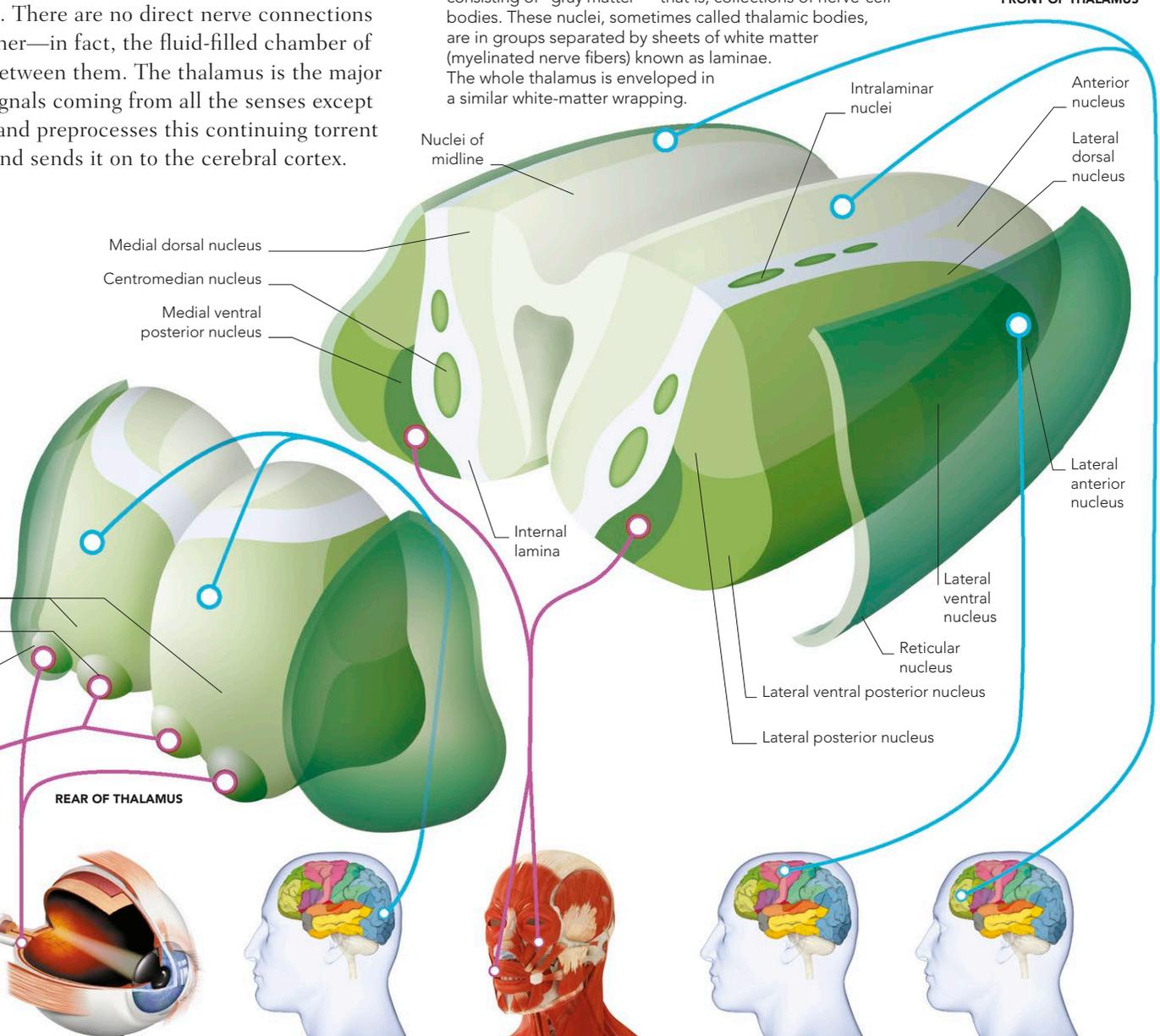
Each side (one of which is shown here) contains more than 20 nuclei consisting of “gray matter”—that is, collections of nerve-cell bodies. These nuclei, sometimes called thalamic bodies, are in groups separated by sheets of white matter (myelinated nerve fibers) known as laminae. The whole thalamus is enveloped in a similar white-matter wrapping.

FRONT OF THALAMUS



THALAMIC NEURONS

Densely interconnected neuron bodies and nerve fibers (green) receive physical and nutritional support from glial cells (red).



- Lateral nuclei (pulvinars)
- Medial geniculate nucleus
- Lateral geniculate nucleus

INNER EAR

Nerve impulses from the cochleas of the inner ears go mostly to the medial geniculate nuclei, which forward them on to the auditory cerebral cortex (Brodmann areas 41 and 42, see p.67).

RETINA

Information from the retinas, about what the eyes see, arrives at the lateral geniculate nuclei. After processing, it passes to the primary visual cortex (area 17) and visual association cortex.

VISUAL CORTEX

Working with the lateral geniculate nuclei, each much larger lateral nucleus (or pulvinar) sends accessory sensory information to several parts of the visual cortex (see pp.82–83).

FACE AND MOUTH

Sensory information from the facial skin and interior of the mouth travels along the trigeminal nerve and the trigeminothalamic tract to the medial ventral posterior nuclei.

PREMOTOR CORTEX

The thalamus has both incoming (afferent) and outgoing (efferent) nerve fibers. Many nerve fibers to the lateral anterior nuclei are afferent, from the premotor area of the cerebral cortex.

PREFRONTAL CORTEX

Most of the incoming signals for the medial dorsal nuclei are from the cerebral prefrontal cortex, and also from the hypothalamus when concerning emotions.

Fornix

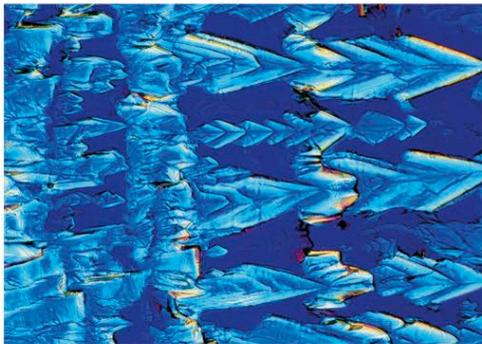
Paraventricular nucleus
Contains neurosecretory cells; also involved in control of blood pressure, body temperature, and appetite

Dorsomedial nucleus
Important in eating, drinking, and regulation and conscious awareness of body weight

Mamillothalamic tract
This bundle of nerve fibers conveys messages between parts of the limbic system

THE HYPOTHALAMUS

Not much larger than the end segment of the little finger, weighing just $\frac{5}{32}$ oz (4g), and comprising only 0.4 percent of total brain volume, the hypothalamus has many and varied vital roles—in conscious behavior, emotions and instincts, and automatic control of body systems and processes. It consists of more than a dozen paired nuclei (regions of interlinked nerve-cell bodies) clustered into the floor of the diencephalon and separated by the lateral ventricle. Its secretory cells make hormones (called releasing factors) that enter the bloodstream, and its neurosecretory cells produce hormonelike substances that travel along nerve axons down to the pituitary gland (see below).



OXYTOCIN CRYSTALS
This birth and breastfeeding hormone is manufactured by neurosecretory cells in the paraventricular and supraoptic nuclei of the hypothalamus.

Optic chiasm

Suprachiasmatic nucleus ("body clock")

Supraoptic nucleus

Two hormones, antidiuretic (ADH or vasopressin) and oxytocin, are produced by neurosecretory cells in the supraoptic nucleus

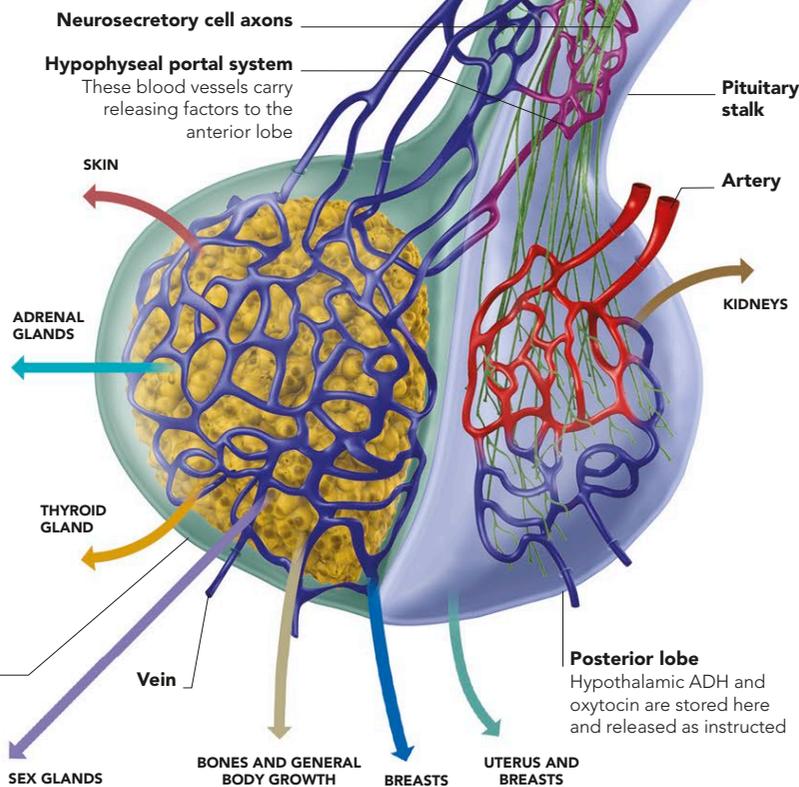
Posterior nucleus

Increases heart rate and blood pressure, dilates pupils, and other autonomic responses as part of "fight or flight" reaction

THE PITUITARY GLAND

The hypothalamus integrates the body's two systems for coordination and control: the nervous system around and above it; and the endocrine system (see p.114-15) via the pituitary just below it. The pea-sized pituitary (hypophysis), often called the body's "master hormone gland," has two distinct lobes. The anterior lobe (adenohypophysis) makes several hormones that release into the bloodstream to regulate other endocrine glands around the body, such as the thyroid. The posterior lobe (neurohypophysis) receives two hormones along axons from the hypothalamus.

Anterior lobe
Forming two-thirds of the pituitary bulk, the anterior lobe manufactures about eight major hormones; it is under the control of nerve messages and regulatory substances, called releasing factors, made in the hypothalamus



Neurosecretory cell axons

Hypophyseal portal system
These blood vessels carry releasing factors to the anterior lobe

Pituitary stalk

Artery

KIDNEYS

ADRENAL GLANDS

THYROID GLAND

SEX GLANDS

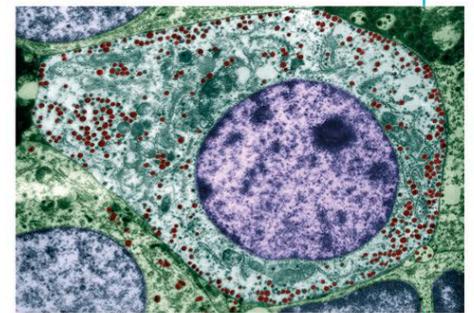
Vein

BONES AND GENERAL BODY GROWTH

BREASTS

UTERUS AND BREASTS

Posterior lobe
Hypothalamic ADH and oxytocin are stored here and released as instructed



ENDOCRINE CELL
This micrograph shows somatotroph cells in the anterior pituitary. These cells store their growth hormone as granules (red dots) ready for export.

KEY TO PITUITARY HORMONES

- Melanocyte-stimulating hormone (MSH)
- Adrenocorticotropic hormone (ACTH)
- Thyroid-stimulating hormone (TSH)
- Follicle-stimulating hormone (FSH), Luteinizing hormone (LH)
- Growth hormone (GH)
- Oxytocin
- Antidiuretic hormone (ADH)
- Prolactin

THE BRAINSTEM AND CEREBELLUM

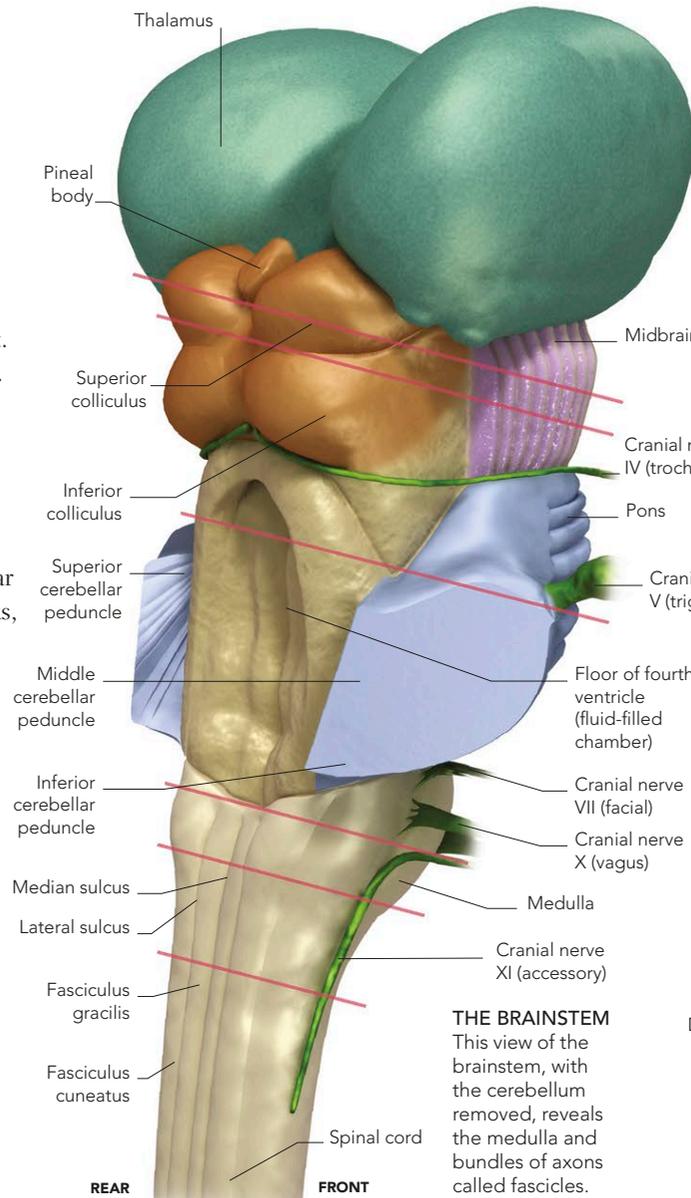
THE BRAINSTEM IS PERHAPS MISNAMED. IT IS NOT A STEM LEADING TO THE SEPARATE BRAIN ABOVE, BUT AN INTEGRAL PART OF THE BRAIN ITSELF. IT IS SHAPED RATHER LIKE A WIDENING UPRIGHT STALK, ON TOP OF WHICH ARE THE THALAMUS AND THE DOME OF THE CEREBRAL HEMISPHERES. CURLED AROUND THE LOWER BRAINSTEM, AT THE REAR OF THE BRAIN, SITS THE CEREBELLUM.

BRAINSTEM ANATOMY

The brainstem includes almost all of the brain except for the highest parts, which make up the forebrain (cerebrum and diencephalon, see p.52). Its uppermost region is the midbrain comprising an upper “roof” or tectum incorporating the superior and inferior colliculi or bulges at the rear, and the tegmentum to the front. Below the midbrain is the hindbrain. At its front is the large bulge of the pons. Behind and below this is the medulla, which narrows to merge with the uppermost end of the body’s main nerve, the spinal cord. The cerebellum joins to the rear of the medulla by three pairs of stalks, known as the cerebellar peduncles.



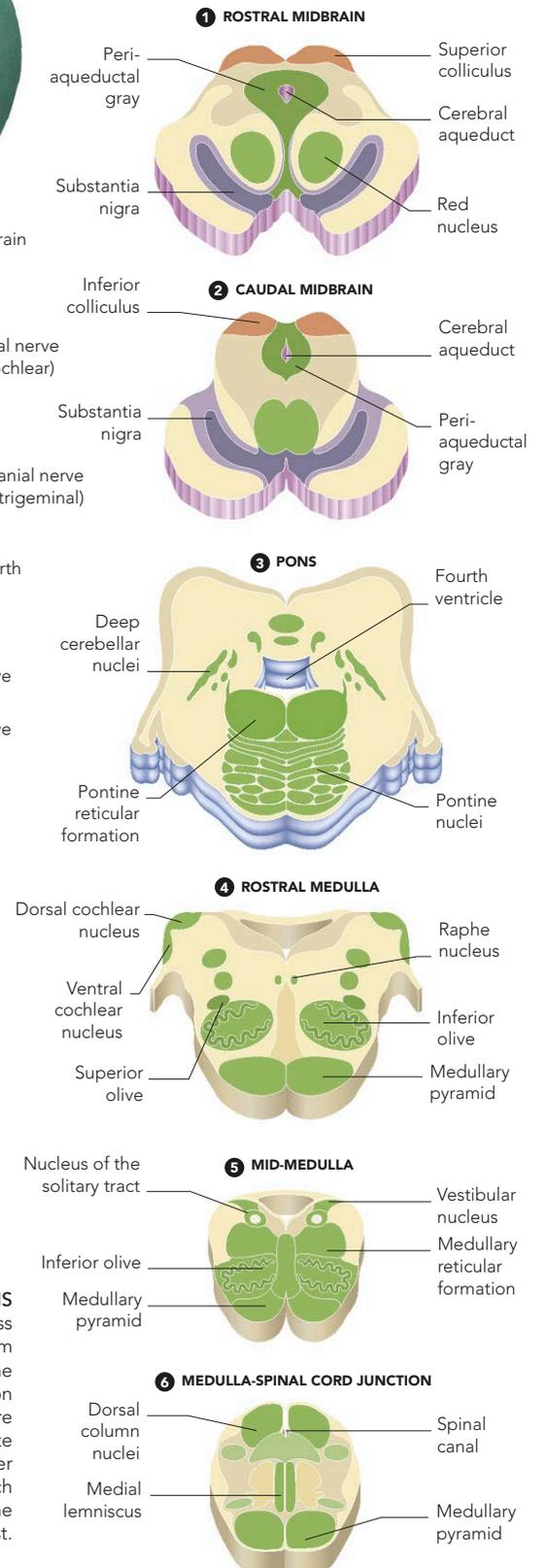
CONNECTING THE BRAIN
This MRI scan shows how the upper brainstem is at about level with the eyes, and its lower region joins the spinal cord at a gap through the base of the skull, the foramen magnum.



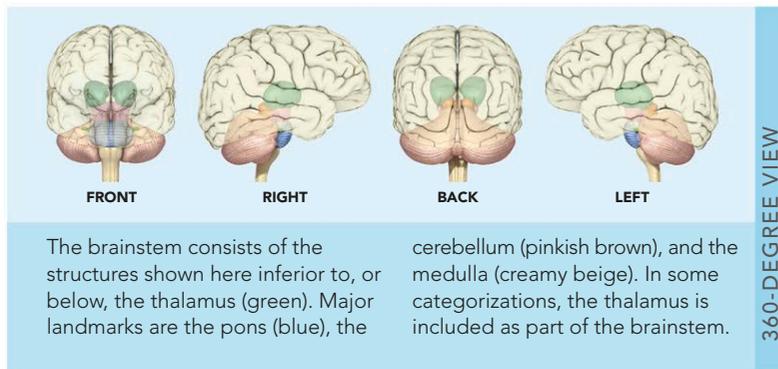
THE BRAINSTEM
This view of the brainstem, with the cerebellum removed, reveals the medulla and bundles of axons called fascicles. The cranial nerves join various parts of the brainstem.

INTERNAL STRUCTURE

Within the brainstem are groupings of nerve-cell bodies known as nuclei (see pp.58–59) and numerous bundles of nerve fibers or axons, called nerve tracts. For example, the pontine nuclei of the front or ventral pons are involved in learning and remembering motor skills—they act as relay stations for nerve signals from the motor cortex, which are traveling to the cerebellum behind the pons (see panel, opposite).

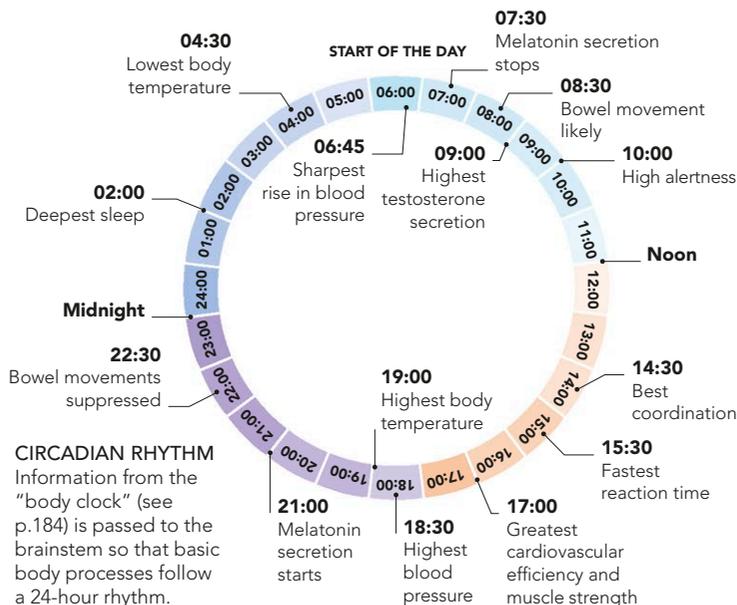


BRAINSTEM SECTIONS
The horizontal cross sections of the brainstem shown here match the numbers in the illustration above left. Nuclei are shown in green; the white matter of nerve fiber tracts is pale. In each section, the rear of the body is uppermost.



BRAINSTEM FUNCTIONS

The brainstem is highly involved in mid- to low-order mental activities, for example, the almost “automatic” scanning movements of the eyes as we watch something pass by, rather than higher activities such as abstract thought. It is also the site of subconscious or autonomic control mechanisms, of which we are usually unaware. The medulla, in particular, houses groups of nuclei that are centers for respiratory (breathing), cardiac (heartbeat), and vasomotor (blood pressure) monitoring and control, as well as for vomiting, sneezing, swallowing, and coughing.



LOCKED-IN SYNDROME

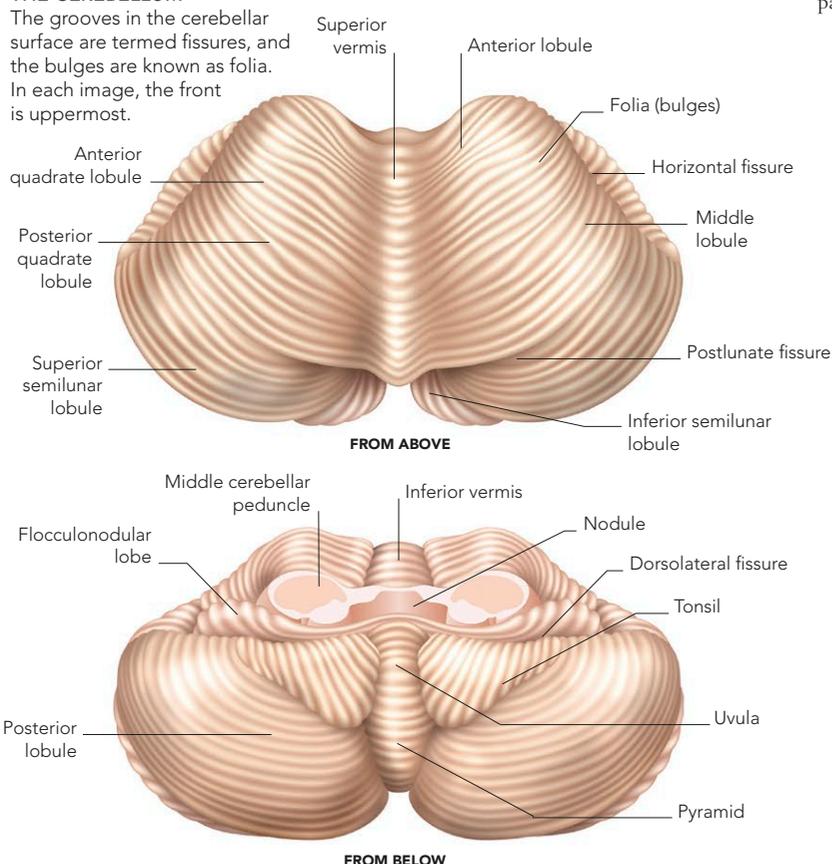
Damage to certain parts of the brainstem, especially the forward-facing area of the pons, can produce a condition known as “locked-in” or ventral pontine syndrome. The sufferer is aware of his or her surroundings and able to see and hear, but cannot activate any voluntary muscles—those that are under conscious control—and so is unable to move or react. Damage may be due to injury or the lack of blood supply during a stroke. In some cases, the eye muscles continue to function, allowing communication by eye movements.

THE CEREBELLUM

The “little brain” is the lower, rearmost part of the entire brain. It resembles the wrinkled appearance of the cerebrum above, but its grooves and bulges are finer and organized into more regular patterns. Major anatomical parts of the cerebellum include: the long, slim vermis (“worm”) in the center; two flocculonodular lobes beneath, one on each side; and outside these, two much larger lateral lobes, each of which is divided into several lobules. The two lateral lobes are reminiscent of the two hemispheres of the cerebrum and are sometimes termed cerebellar hemispheres. The cerebellum’s main functions are to coordinate body movements through integrated control of muscles, including balance and posture, and equilibrium.

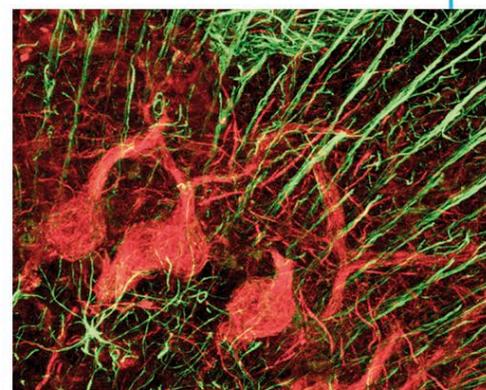
THE CEREBELLUM

The grooves in the cerebellar surface are termed fissures, and the bulges are known as folia. In each image, the front is uppermost.

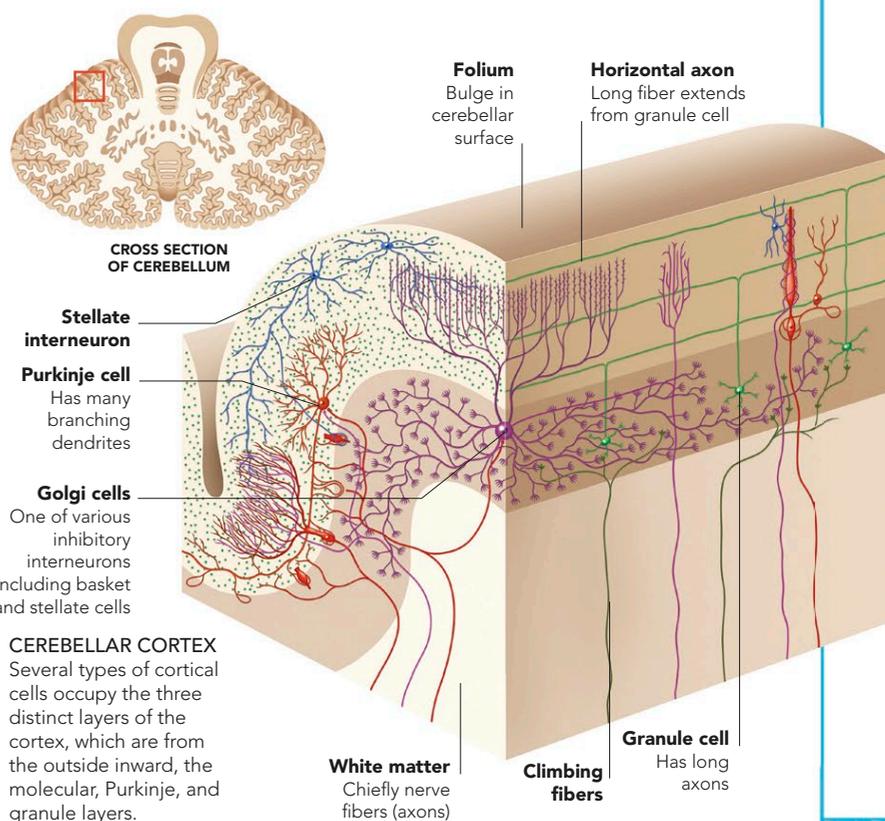


INTERNAL STRUCTURE

The cerebellum has a similar layered microstructure to the cerebrum. The outer layer, or cerebellar cortex, is gray matter composed of nerve-cell bodies and their dendrite projections. Beneath this is a medullary area of white matter consisting largely of nerve fibers. Toward the center are collections of more nerve-cell bodies known as deep cerebellar nuclei. Nerve fibers run from these nuclei to the cerebral cortex high above. In a cross section at almost any angle through the cerebellum, the white matter between the cortex and deep nuclei forms a complex branching pattern known as the arbor vitae.



CEREBELLUM CELLS
The main types of nerve cells in the cerebellar cortex are known as Purkinje cells (red), supported by glial cells (green).

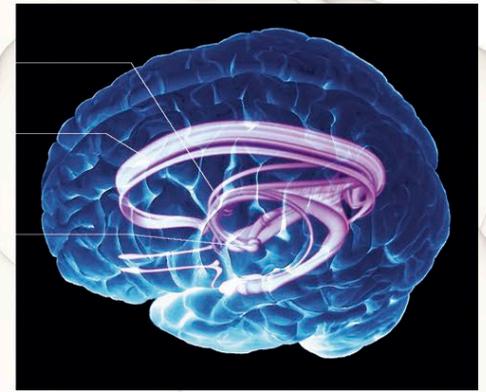


THE LIMBIC SYSTEM

THE LIMBIC SYSTEM IS INVOLVED IN INSTINCTIVE BEHAVIORS, DEEP-SEATED EMOTIONS, AND BASIC IMPULSES SUCH AS SEX, ANGER, PLEASURE, AND GENERAL SURVIVAL. IT ALSO FORMS A LINK BETWEEN CENTERS OF HIGHER CONSCIOUSNESS, IN THE CEREBRAL CORTEX, AND THE BRAINSTEM, WHICH REGULATES THE BODY'S SYSTEMS.

COMPONENTS OF THE LIMBIC SYSTEM

The limbic system includes the areas of the cortex and adjacent parts known as the limbic lobe (see opposite page), along with the amygdala, hypothalamus, thalamus, mamillary bodies, and other deeper, more central brain structures. The system is also "hard-wired" into parts of the sensory system, especially the sense of smell. Nerve fibers link all of these parts intimately and also connect them to other areas of the brain, particularly the lower frontal cortex, with its roles in expectation, reward, and decision-making.



AT THE BRAIN'S CORE
Situating approximately in the anatomical center or core of the brain, the limbic system is a varied collection of structures extending from the cerebrum inward and down to the brainstem.

Cingulate gyrus
Part of limbic cortex just above corpus callosum

Column of fornix

Mamillary bodies
Small lumps of nerve cells, these relay signals to thalamus, contributing to alertness and memory formation

Olfactory bulbs
Tracts of sensory nerve cells extend from nasal cavity into the brain; they part-process smell information before it enters conscious awareness

Fornix
This tract of nerve fibers connects the mamillary bodies and hippocampus

LIMBIC STRUCTURES
The name of this system is derived from the Latin *limbus*, meaning "border" or "edge." Its major structures form a circular, beltlike transition zone between the relatively plain-looking main cerebral cortex and the more distinctive bodies, tracts, and nuclei of the inner, lower brain.

Hypothalamus
Chief link and mediator between nervous system and hormonal or endocrine system (see p.61)

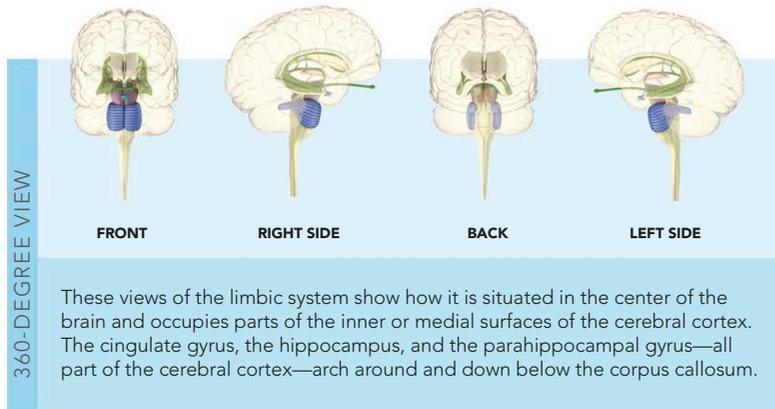
Pons

Hippocampus
Named after its vague S-shaped resemblance to a seahorse, this part is involved in memory and spatial awareness

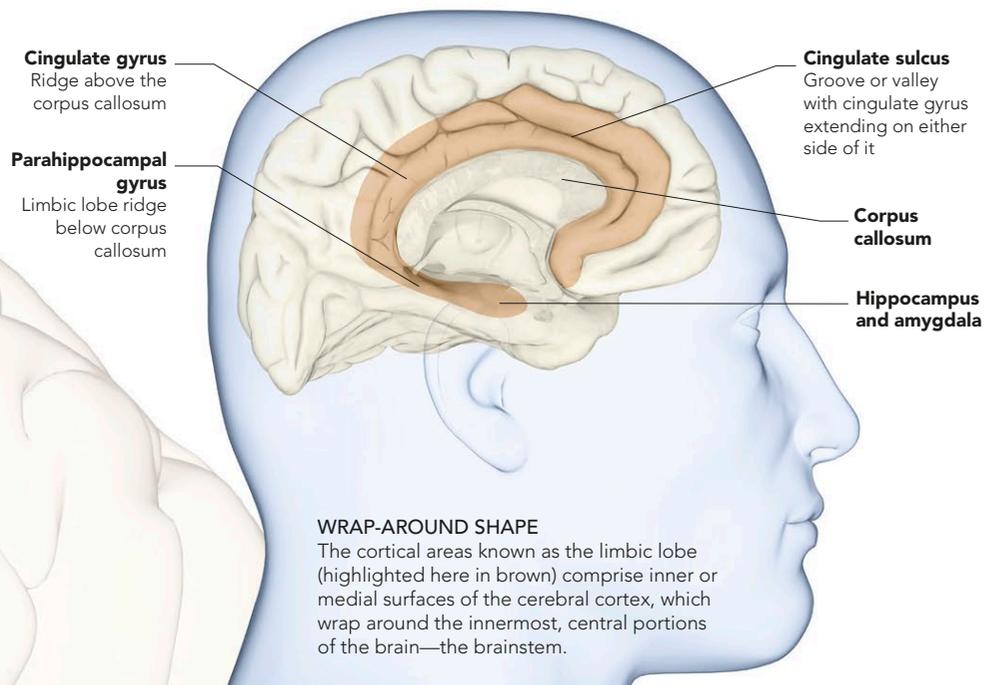
Midbrain
The limbic system extends nerve fibers from thalamus and other higher parts into this uppermost part of the brainstem and also to the basal nuclei

Parahippocampal gyrus
This area of cortex flanking the hippocampus is active when viewing scenes and places

Amygdala
Almond-shaped neuron clusters that are heavily involved in memory and emotional responses



These views of the limbic system show how it is situated in the center of the brain and occupies parts of the inner or medial surfaces of the cerebral cortex. The cingulate gyrus, the hippocampus, and the parahippocampal gyrus—all part of the cerebral cortex—arch around and down below the corpus callosum.



THE LIMBIC LOBE

The structures of the limbic system are surrounded by an area of the cortex referred to as the limbic lobe. The lobe forms a collarlike or ringlike shape on the inner surfaces of the cerebral hemispheres, both above and below the corpus callosum. The upper part is the cingulate gyrus, on either side of the cingulate sulcus. The lower part is the parahippocampal gyrus, delineated below by the collateral fissure and rhinal sulcus. The cingulate and parahippocampal gyri are together known as the fornicate gyrus. As such, the limbic lobe comprises the inward-facing parts of other cortical lobes, including the temporal, parietal, and frontal, where the left and right lobes curve around to face each other. The hippocampus and amygdala are not integral to this split-ring shape, but are considered as anatomically part of the limbic lobe as well as components of the limbic system.

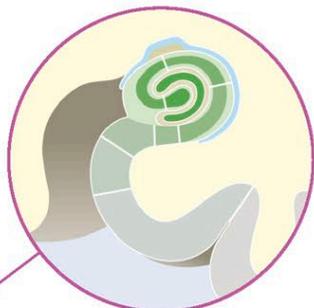
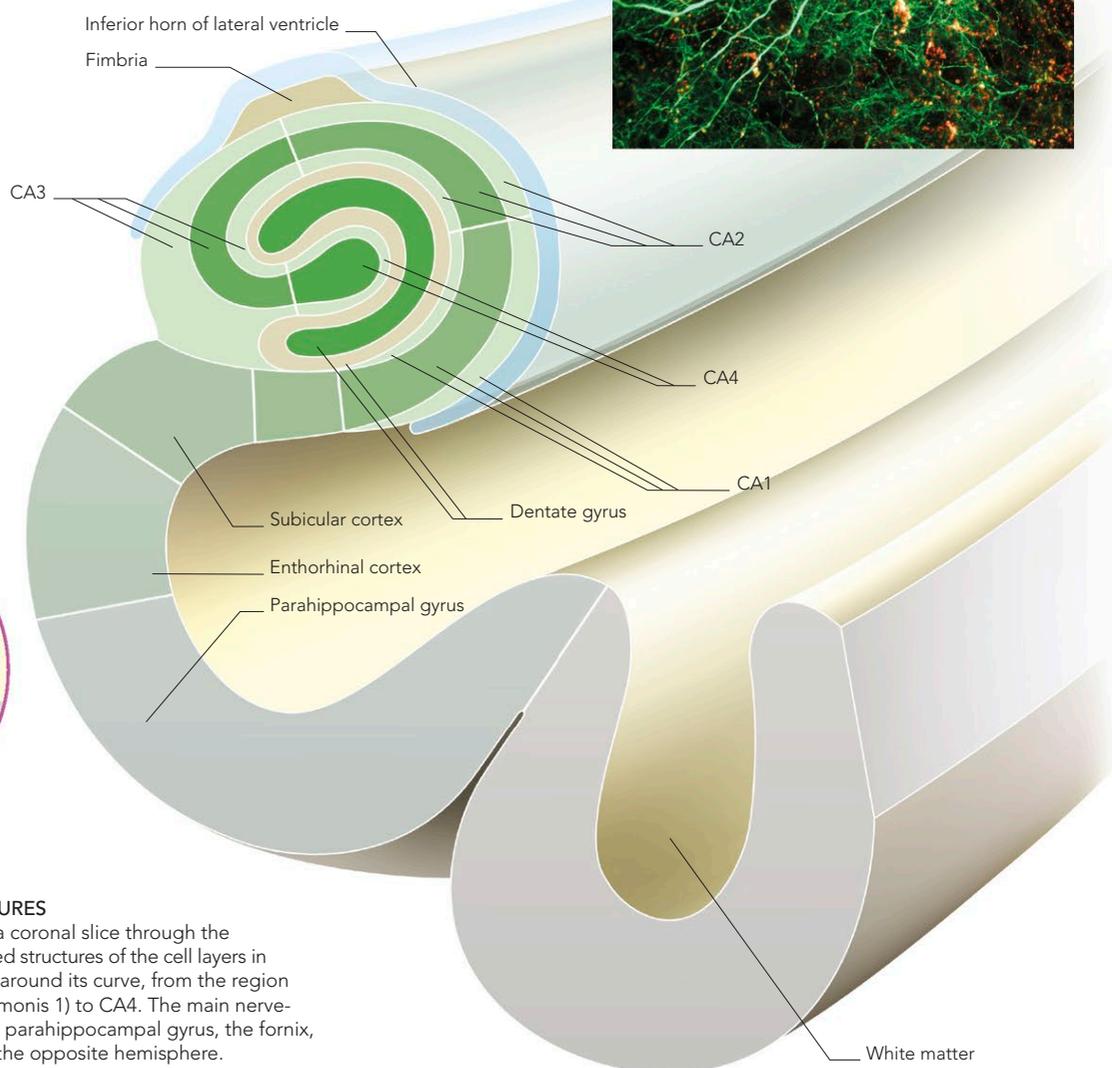
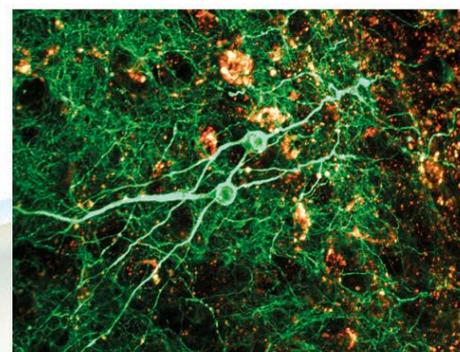
THE HIPPOCAMPUS

The hippocampus is strung along the upper edge of the parahippocampal gyrus. The hippocampus interlocks with another ridge, known as the dentate gyrus—together the two form the hippocampal–dentate complex. It is part of the cerebral cortex, but it has only one, two, or three layers of cells, rather than the usual six layers found in most of the more “advanced” regions of the cortex.

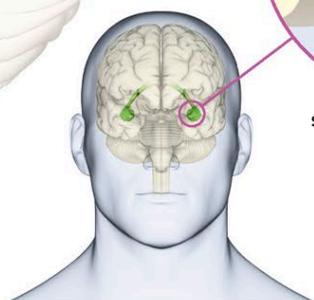
The main functions of the hippocampus include spatial awareness, and memory formation and recall. In particular, the hippocampus helps select transient information for memorizing and then pass it through to longer-term memory areas. Damage to it can prevent a person from forming new memories, even though memories from before the damage are intact.

NEURONS

A light micrograph of a section through the hippocampus reveals neurons that have been labeled with green fluorescent protein. Also seen are ion channels (colored gold) that allow the exchange of sodium and calcium ions across the cell membrane. This exchange propagates nerve impulses.



SECTION OF HIPPOCAMPUS



LOCATION OF HIPPOCAMPUS

HIPPOCAMPAL STRUCTURES

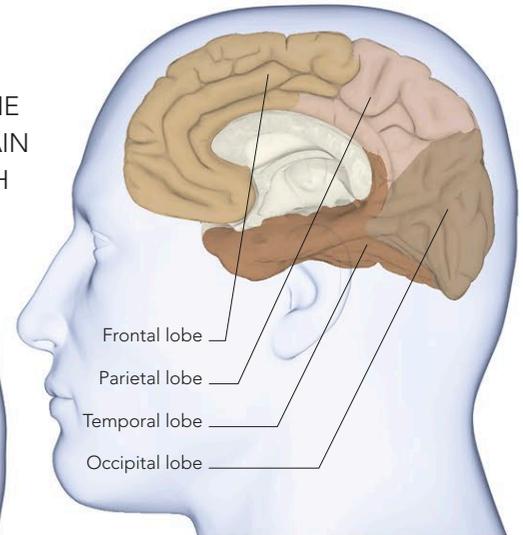
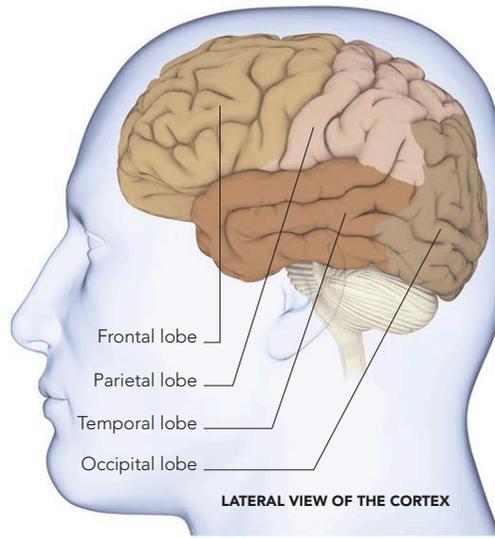
This cross section shows a coronal slice through the hippocampus. The detailed structures of the cell layers in the hippocampus change around its curve, from the region known as CA1 (cornu ammonis 1) to CA4. The main nerve-signal inputs are from the parahippocampal gyrus, the fornix, and the hippocampus in the opposite hemisphere.

THE CEREBRAL CORTEX

THE CEREBRAL CORTEX IS THE OUTER LAYER OF THE BRAIN'S MOST DOMINANT PART, THE CEREBRUM. IT IS THE BULGING WRINKLED SURFACE WE SEE WHEN LOOKING AT THE BRAIN FROM ANY ANGLE. IT IS COMMONLY KNOWN AS GRAY MATTER FROM ITS COLOR, WHICH CONTRASTS WITH THE WHITE MATTER IN THE LAYER BELOW.

THE CEREBRAL LOBES

Bulges and grooves help divide the cortex into four to six paired lobes, according to the anatomical system used. The main and deepest groove is the longitudinal fissure that separates the cerebral hemispheres. Both the extent and the names of the lobes are also partly related to the overlying bones of the skull, known as the neurocranium. For example, the two frontal lobes are approximately beneath the frontal bone, and likewise for the occipital lobes under the occipital bone. In some naming systems, the limbic lobe (see p.65) and the insula, or central lobe, are distinguished as separate from other lobes.

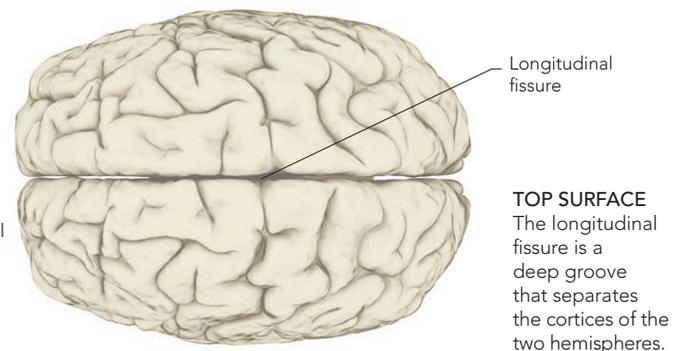
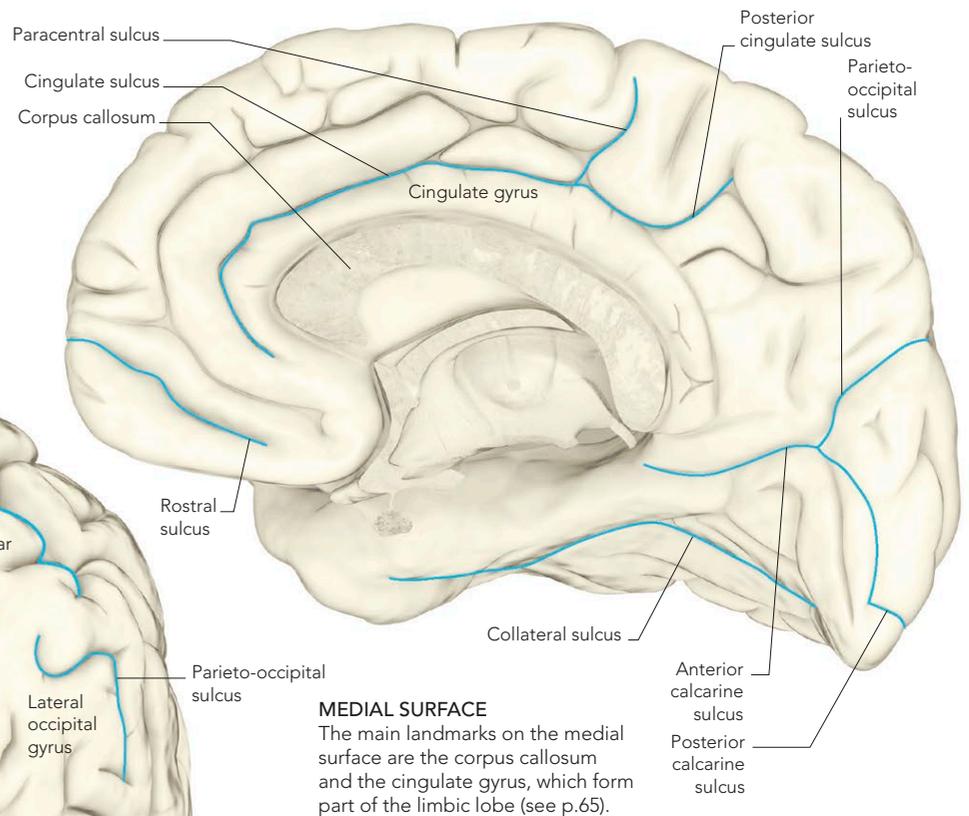
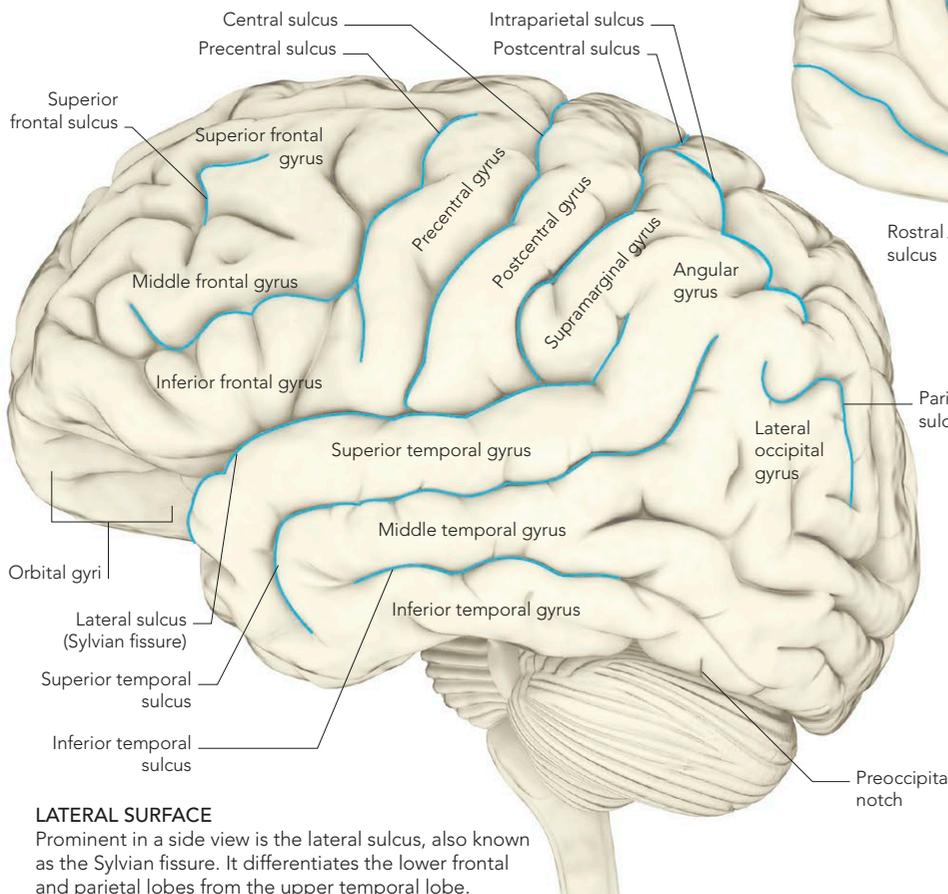


LOBE DIVISIONS

The cortex can be divided into four areas called lobes (shown here). In some classifications, the forward part of the frontal lobe is separated as the prefrontal lobe, but the term prefrontal cortex is more generally accepted.

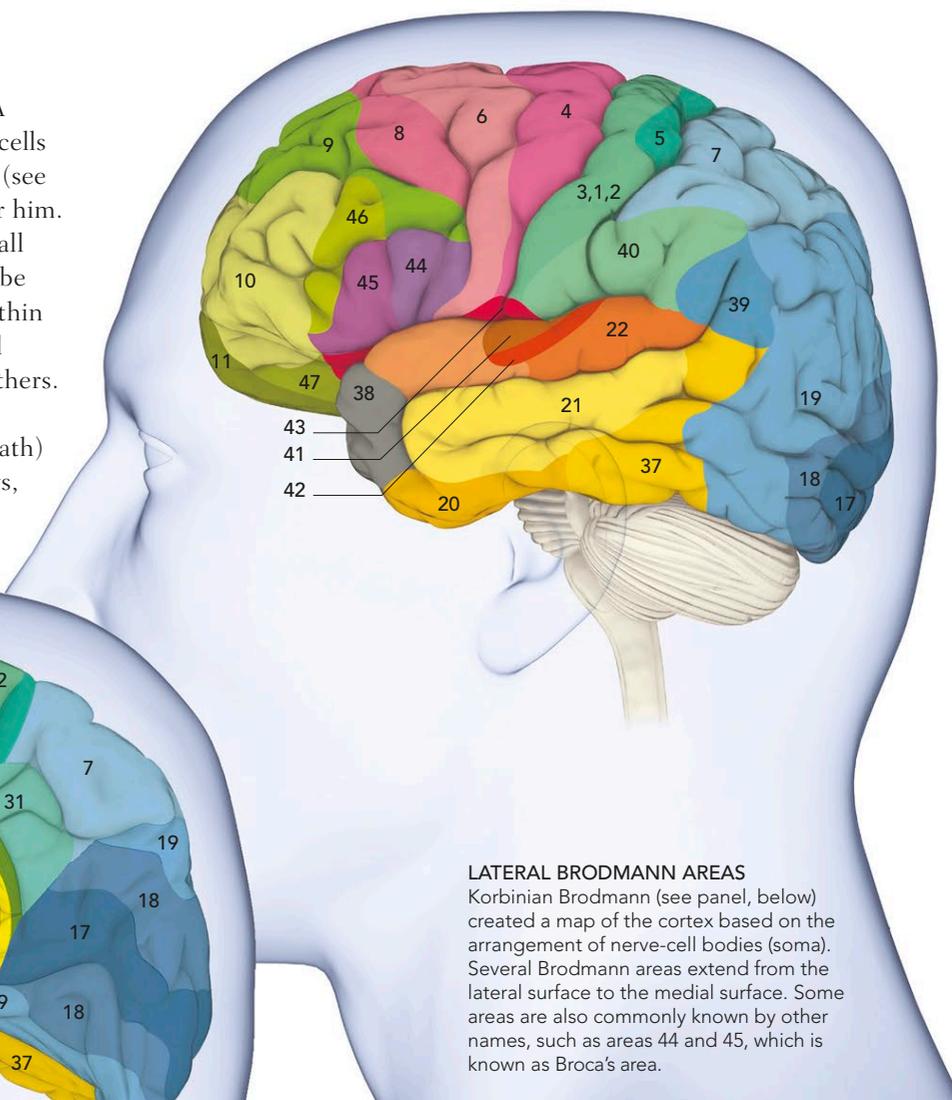
CORTICAL LANDMARKS

Rounded bulges of the cortex are known as gyri; grooves are termed sulci when relatively shallow and fissures when deeper. The overall patterns of gyri and sulci are similar but rarely identical among normal brains—individual variations occur. They are also similar for the left and right of an individual's brain, although there are minor asymmetries (see p.57).



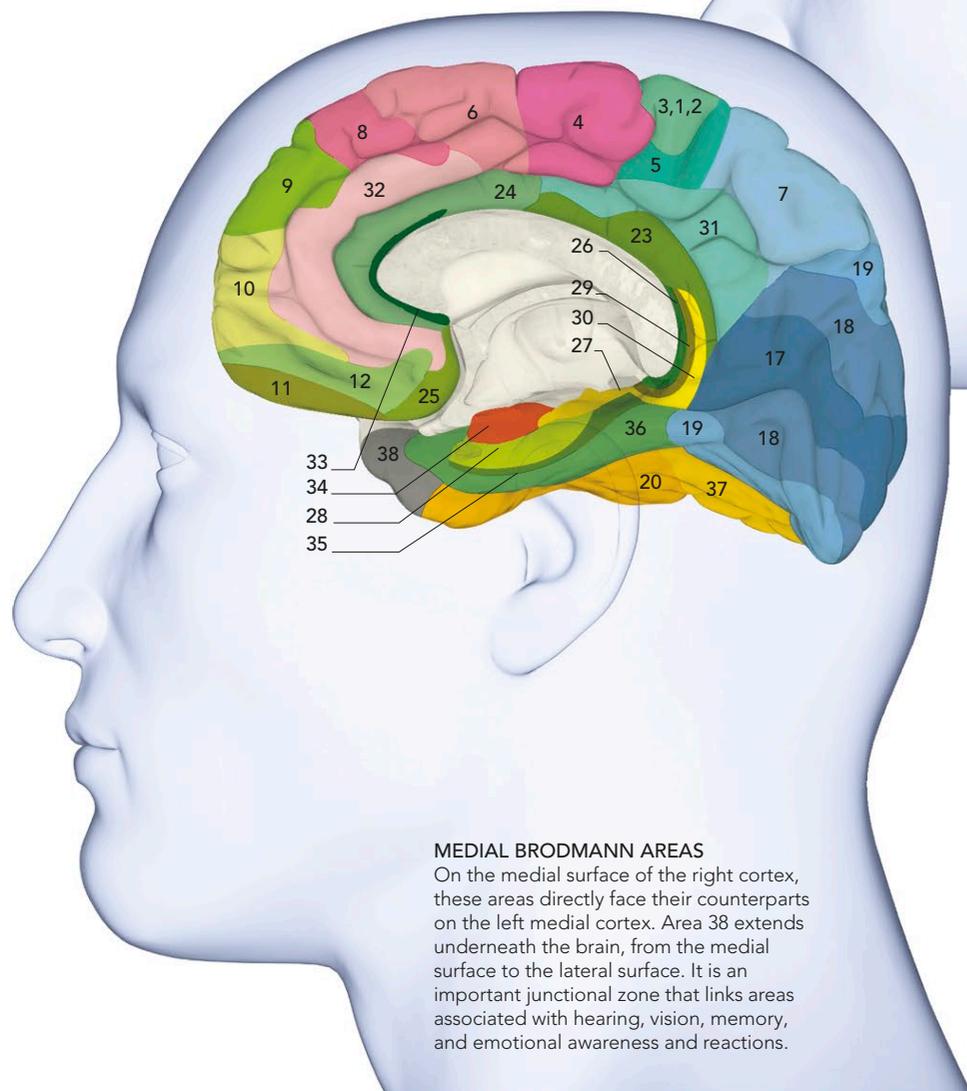
FUNCTIONAL AREAS

The cortex can be “mapped” in three ways. One is by gross anatomy, as defined by sulci and gyri (see opposite page). A second is by microscopic anatomy—the shapes and types of cells and their connections, as pioneered by Korbinian Brodmann (see panel, below). The map of areas shown here is named after him. The third method is by neurological function, in which small areas are correlated with what they do. For example, the lobe at the back of the brain is mainly devoted to vision, and within it smaller areas are responsible for various aspects of visual processing—determining color, shape, or motion, among others. The earliest parts of this functional “map” were created by matching damage in a person’s brain (usually after their death) with cognitive deficits they displayed when alive. Nowadays, it is mainly done by stimulating small areas and noting the effect. The three “maps” only partially coincide.



LATERAL BRODMANN AREAS

Korbinian Brodmann (see panel, below) created a map of the cortex based on the arrangement of nerve-cell bodies (soma). Several Brodmann areas extend from the lateral surface to the medial surface. Some areas are also commonly known by other names, such as areas 44 and 45, which is known as Broca’s area.



MEDIAL BRODMANN AREAS

On the medial surface of the right cortex, these areas directly face their counterparts on the left medial cortex. Area 38 extends underneath the brain, from the medial surface to the lateral surface. It is an important junctional zone that links areas associated with hearing, vision, memory, and emotional awareness and reactions.

APPROXIMATE FUNCTIONS

AUDITION

Temporal lobe

- 22 41
- 38 42

BODY SENSATION

Parietal lobe

- 1, 2, 3
- 5 39
- 7 40
- 31

EMOTION

Anterior cingulate and orbital cortex

- 11 32
- 12 33
- 24 38
- 25

GUSTATION

Insula

- 43

OLFACTION

Medial temporal cortex

- 28 34

MEMORY

Medial temporal lobe, posterior cingulate cortex

- 23 30
- 26 35
- 27 36
- 29

MOTOR

Frontal lobe

- 4 44
- 6 45
- 8 46
- 9 47
- 10

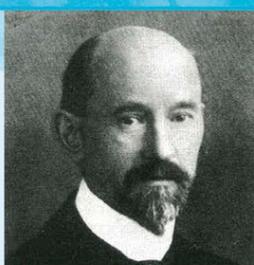
VISION

Occipital cortex and temporal cortex

- 17 21
- 18 37
- 19 38
- 20

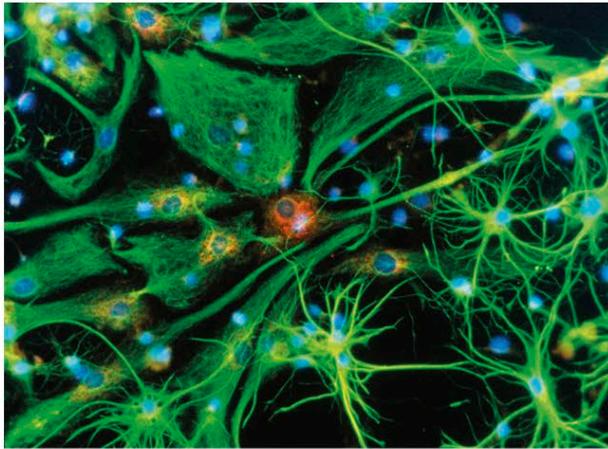
KORBINIAN BRODMANN

A German neurologist, Brodmann (1868–1918) made a detailed study of the cortex, looking at the way its layers, tissues, and individual neurons and other cells vary in their structure and size. He identified and numbered different areas in the brains of humans, monkeys, and other mammals, ending the considerable confusion in naming parts of the cortex that existed at the time.



ASSOCIATION AREAS

Some parts of the cortex, called association areas, are composed of neurons that are connected to two or more functional areas. This means that they receive different types of information—for example, visual and auditory. Their role is to combine this information. It is part of the construction process that allows us to see the world as an integrated whole rather than discrete bits. The adjoining edges of the visual and parietal areas, for example, combine visual information with body awareness to work out the position of a visually perceived object in relation to the body. The frontal cortex may be considered an association area because it receives information from all other areas of the brain and combines it. The product of this mix is thoughts, judgments, and conscious feelings.



GLIAL CELLS

In this light micrograph, star-shaped astrocytes (lighter green) can be seen along with other support cells, or neuroglia. They make up the brain's connective tissue and provide protection to neurons. Connective tissue supports the neurons transmitting information between cortical areas.

Frontal lobes

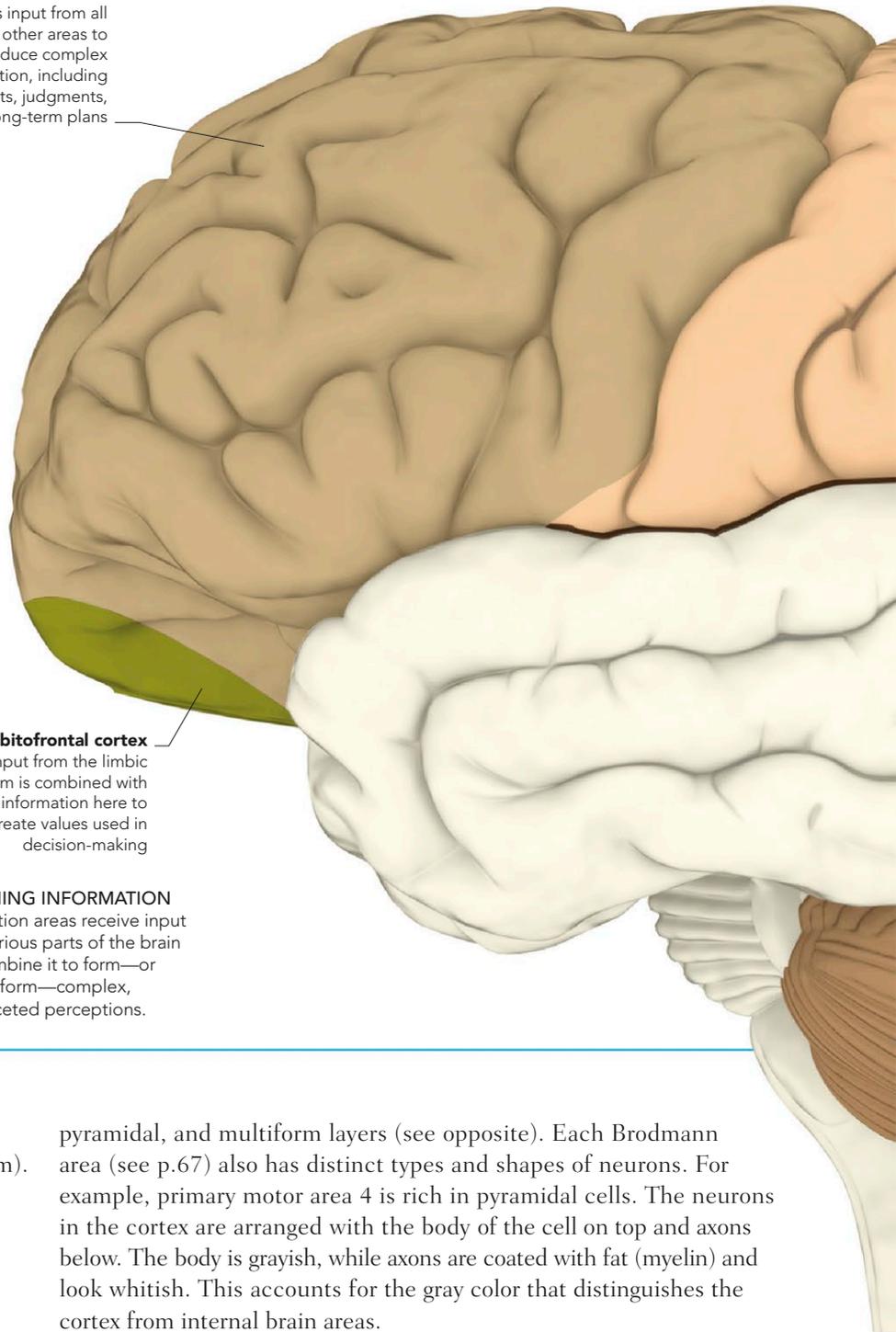
The front of the brain gathers input from all other areas to produce complex cognition, including thoughts, judgments, and long-term plans

Orbitofrontal cortex

Input from the limbic system is combined with other information here to create values used in decision-making

INCOMING INFORMATION

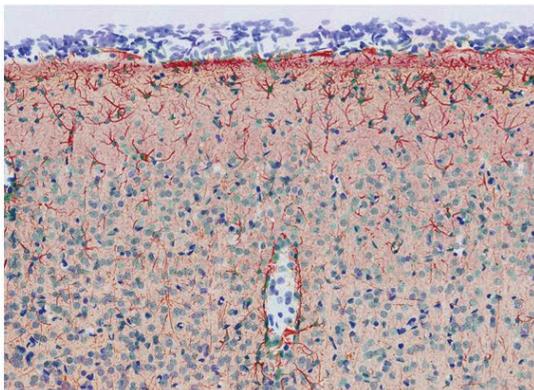
Association areas receive input from various parts of the brain and combine it to form—or start to form—complex, multifaceted perceptions.



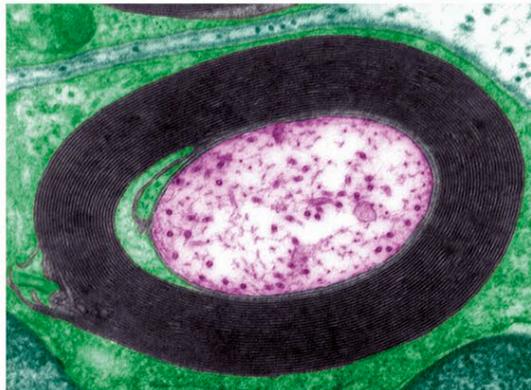
STRUCTURE OF THE CORTEX

The highly convoluted sheet of gray matter that constitutes the cerebral cortex varies in thickness from about $\frac{1}{16}$ to $\frac{3}{16}$ in (2 to 5mm). Estimates of its cell numbers vary from 10 billion to more than 50 billion neurons and about 5 to 10 times this number of glial (supporting) and other cells. The neurons are organized into six layers, known generally from the outside inward as the molecular, external granular, external pyramidal, internal granular, internal

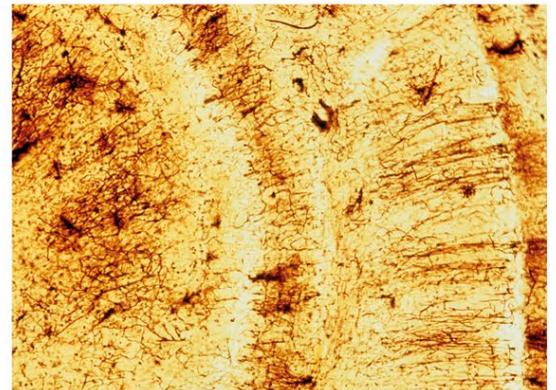
pyramidal, and multiform layers (see opposite). Each Brodmann area (see p.67) also has distinct types and shapes of neurons. For example, primary motor area 4 is rich in pyramidal cells. The neurons in the cortex are arranged with the body of the cell on top and axons below. The body is grayish, while axons are coated with fat (myelin) and look whitish. This accounts for the gray color that distinguishes the cortex from internal brain areas.



CORTEX TISSUE



NERVE FIBER



CEREBRAL LAYERS



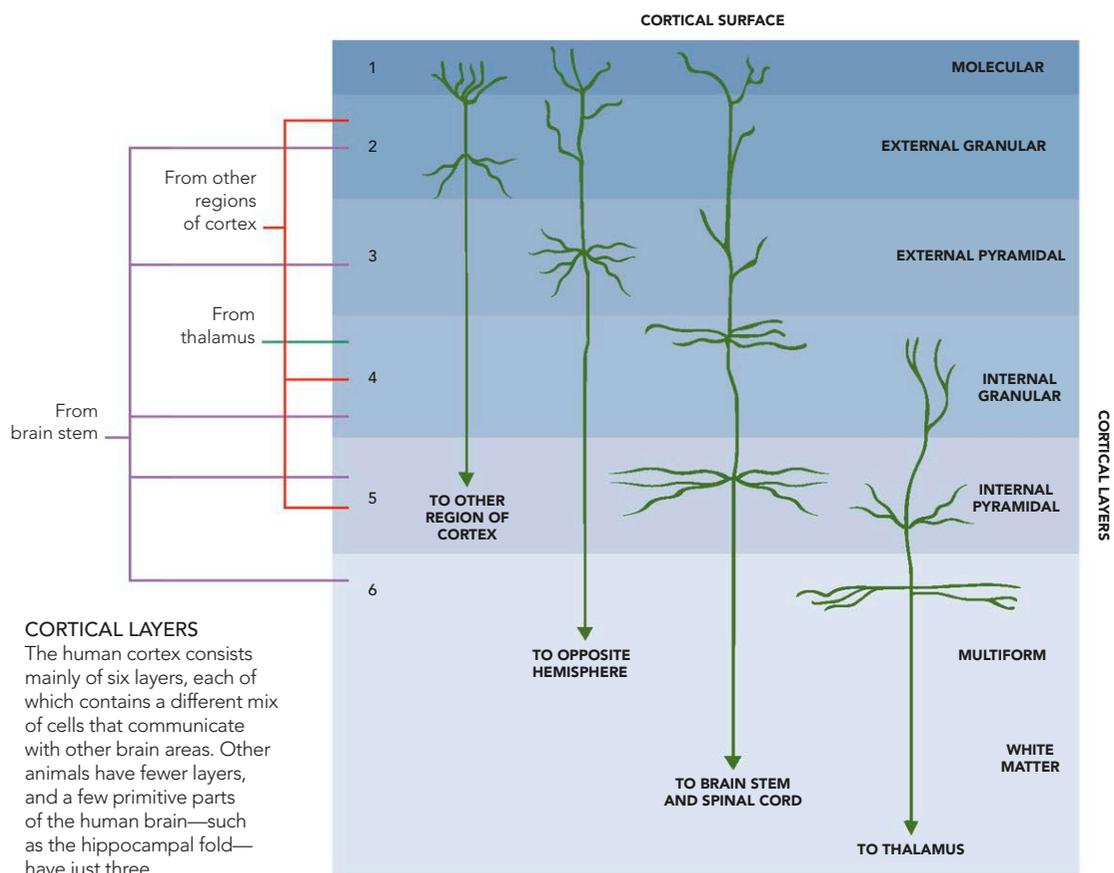
Parietal lobes
Inputs arrive here from visual, auditory, and emotional areas to produce body-centered understanding of the current environment

Temporoparietal junction
This area puts together perceptual information to give a "whole" knowledge of what is happening at any moment

Cerebellum
The back of the brain combines input from perceptual areas to guide fine motor actions

CORTICAL FUNCTIONING

Most of the human cortex comprises six layers, each of which contains a distinct pattern of neuron types. Cortical neurons receive and send signals to other brain areas, including other parts of the cortex. This to and fro of messages keeps all parts of the brain aware of what is going on elsewhere. Neurons in the cortex are "head down"—their receiving parts (dendrites) point up to the surface, while threads that carry messages to other cells (axons) are oriented down. Some axons extend below the cortex and form part of the "white matter"—connective tissue that carries information to distant brain areas. Other axons travel through the lower layers of cortex to connect with other cortical cells.



OLIGODENDROCYTE CELL

CORTEX COMPONENTS
Relatively low magnification of cortical tissue shows neurons (far left, blue-gray) packed among supporting glial cells (red). Higher magnification reveals an individual axon at the cortex base (second from left). Different laboratory stains show four of the six cortical layers (third from left) and fatty myelin wrapped around an axon.

THE FOLDED BRAIN

The scrunched-up structure of the cortex is one of the features that distinguishes the human brain most clearly from that of other species. Most of the cortical surface is tucked into grooves, and if it could be flattened out, it would cover the size of a small tablecloth. The dense cortical folding seen in humans may have evolved along with the shift from walking on all fours to bipedalism. To allow an upright stance, our ancestors evolved a narrow pelvis, which hampered childbirth. It might be that babies with small heads were more likely to survive and that their head size was due to a genetic mutation that caused the brain to fold up, allowing the skull to stay relatively small. Apart from packing in more neurons, cortical folding creates shorter nerve pathways, which in turn create faster data processing.



FLAT CORTEX
Computer software can "flatten" the surface of a brain to show the tissue that is normally hidden in the sulci. Here, the green areas are the surface (gyri), and the red areas are those normally tucked inside.

BRAIN CELLS

THERE ARE OVER A THOUSAND TYPES OF BRAIN CELL, WHICH FALL INTO TWO BROAD GROUPS: NEURONS AND GLIAL CELLS. NEURONS SEND ELECTRICAL SIGNALS, OR "FIRE," IN RESPONSE TO STIMULI. THERE ARE ABOUT 86 BILLION NEURONS IN AN AVERAGE HUMAN BRAIN AND TEN TIMES AS MANY GLIAL CELLS.

NEURONS

Like hepatocyte cells in the liver, osteocytes in bone, or erythrocytes (red cells) in blood, each neuron is a self-contained functioning unit. Its internal components, the organelles, include a nucleus harboring the genetic material (DNA), energy-providing mitochondria, and protein-making ribosomes. As in most other types of cells, the organelles are concentrated in the main cell body. In addition, characteristic features of neurons are neurites—long, thin, fingerlike or threadlike extensions from the cell body (soma). The two main types are dendrites and axons. Usually dendrites receive nerve signals, while axons send them onward.

MICROANATOMY OF A NEURON

The cell body of a neuron is about 10–100 micrometers across, that is, $\frac{1}{100}$ th to $\frac{1}{10}$ th of one millimeter. The axon is 0.2–20 micrometers in diameter; dendrites are usually slimmer. In the central nervous system, dendrites are typically 10–50 micrometers long, and axons can be up to a few centimeters (inches) in length.

Axon (nerve fiber)

Most neurons have just one main axon or sending neurite, also called an axonal process or nerve fiber; it is usually much longer and thicker than the dendrites

Myelin sheath

Spiral wrapping of myelin around certain axons helps speed and insulate the nerve impulses they carry

Oligodendrocyte

Manufactures myelin sheaths for axons of brain neurons

Neuron cell body

Axon end bulb

Synapse

Communication point between neurons

Dendrite

Microtubules

Flexible, rod-like assemblies form the structural "scaffolding" of the cell

Golgi complex

Stores and processes proteins made by the ribosomes, ready for export from the cell

Vacuoles

Baglike containers inside the cell that store various substances such as wastes or excess water

Cell membrane

Outer covering or "skin" of the cell; in neurons, it is specialized to convey or propagate nerve impulses (see p.72)

Cytoplasm

The cell's individual organelles are suspended in this jellylike, solute-packed fluid

Rough endoplasmic reticulum

Sheets of membrane are folded, stacked into piles, and studded with tiny, spherical ribosomes

Mitochondrion

Cellular "power station" that splits apart sugar and fat molecules to release their chemical energy

Ribosomes

Ball-like structures that assemble proteins

Smooth endoplasmic reticulum

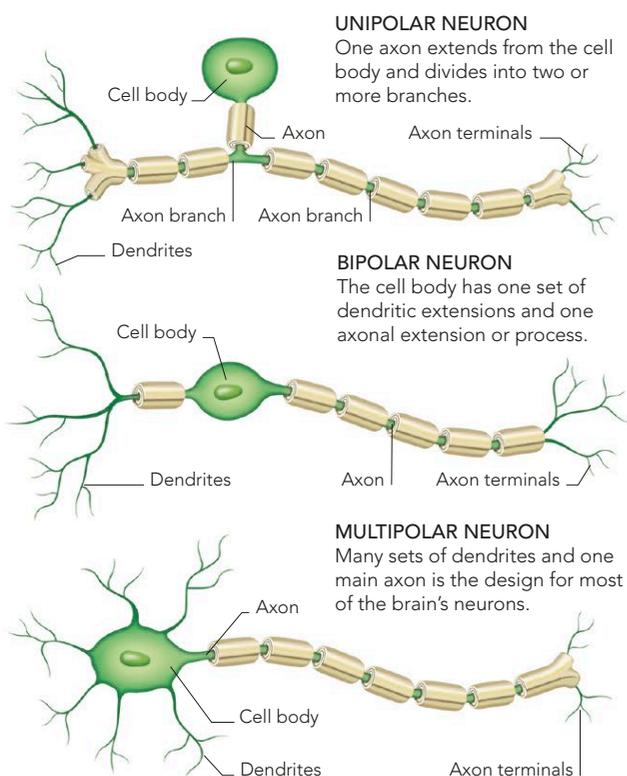
Tubes and layers that help transport and store materials

Nucleus

Contains DNA that instructs how the cell develops and functions

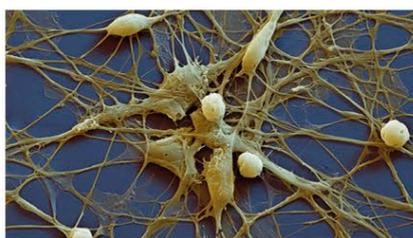
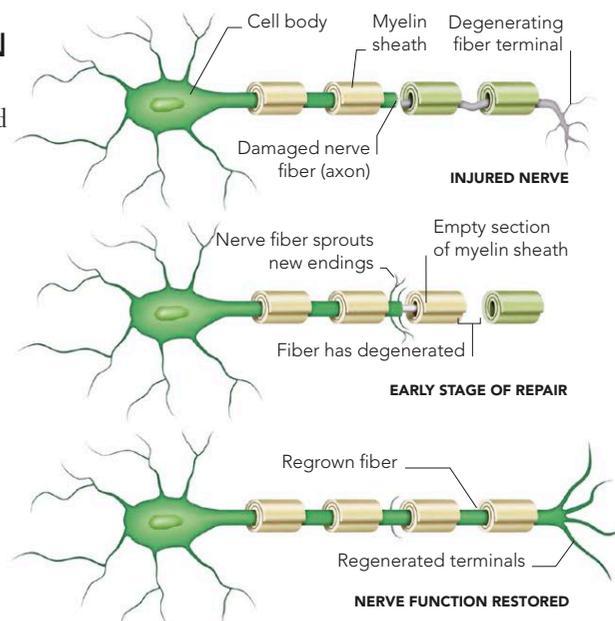
TYPES OF NEURON

Neurons can be categorized structurally according to the location of the cell body in relation to the axon and dendrites, and also the number of dendrites and axon branches (see illustration, below). In some regions of the brain, peripheral nervous system, and sense organs, neuron types are organized and easily recognized. For example, the retina of the eye contains ranks of bipolar neurons (see p.80). However, in many other regions, the neurons are mixed in shape and form a complex, interconnected web. In the cortex, one neuron may receive signals from many thousands of other neurons via its multitudinous branching dendrites. Signals are conducted to the soma, around this, and then away along the axon—always by the cell membrane, not through the cytoplasm.



NEURON REGENERATION

Each neuron has its own immensely complex, highly individual shape and sets of connections, via synapses, to other neurons. Its links are shaped by its history and how it is used over time, as some of its connections weaken and fade while others strengthen. This uniqueness makes any disease or damage very serious. The neuron is unlikely to reform all of its extensions and their links. Even if regrowth occurs, it is slow and at first random, as the dendrites and axon “feel” their way according to the nerve signals being received and sent.

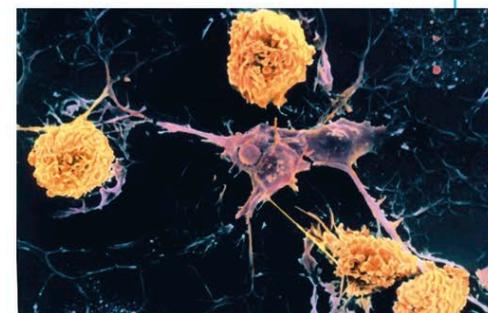


NEUROGENESIS
The brain can form new nerve cells. Neural progenitor cells (shown in this micrograph) are a stage in specialization between stem cells and fully formed nerve cells. At this stage they can specialize into neurons or support cells.

REPAIRING NERVE FIBERS
Nerve cell repair is a very slow process, if it occurs at all. The damaged or severed end of the axon (fiber) can be encouraged to send out new sprout growths by treating it with substances called nerve growth factors. A sprout that finds an empty myelin sheath may then grow through it.

GLIAL CELLS

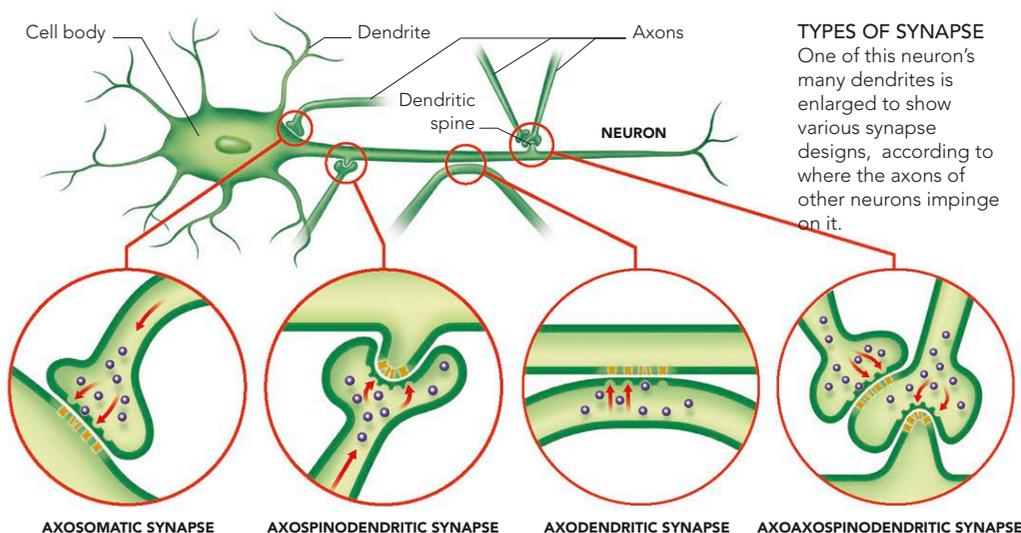
Glial cells give physical support to neurons (glia means “glue” in Greek) but they are also thought to influence neurons’ electrical activity. They provide physical support for the thin dendrites and axons that wind their way around the neural network, and supply nutrition for neurons in the form of sugars and raw materials for growth and repair. There are several types of glial cells. Oligodendrocytes make myelin sheathing, a task performed in peripheral nerves by Schwann cells. Microglia destroy invading microbes and clear up debris from degenerating neurons. Astrocytes are thought to affect neuronal behavior and play a role in memory and sleep.



OLIGODENDROCYTES UNDER ATTACK
In multiple sclerosis (MS) oligodendrocytes (purple), which normally make insulating myelin sheaths around nerve axons in the brain and spinal cord, are attacked and destroyed by microglia (yellow).

SYNAPSES

Synapses are communication sites where neurons pass nerve impulses among themselves. Many neurons do not actually touch one another, but pass their signals via chemicals (neurotransmitters) across an incredibly thin gap, called the synaptic cleft (see pp.72–73). Microanatomically, synapses are divided into types according to the sites where the neurons almost touch. These sites include the soma, the dendrites, the axons, and tiny narrow projections called dendritic spines found on certain kinds of dendrites (see illustration, right). Axospinodendritic synapses form more than 50 percent of all synapses in the brain; axodendritic synapses constitute about 30 percent.

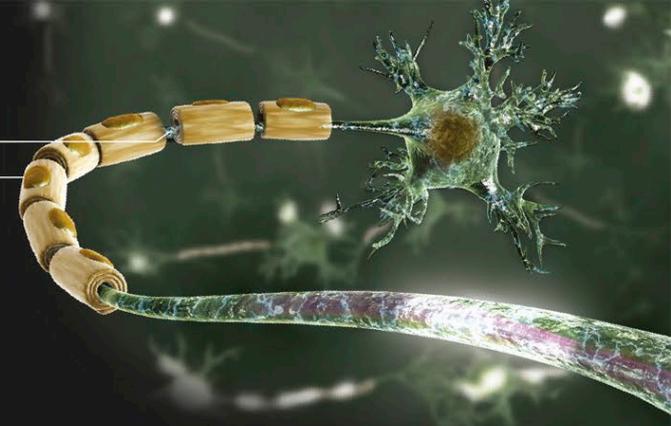


NERVE IMPULSES

A NERVE IMPULSE OR SIGNAL CAN BE THOUGHT OF AS A TINY, BRIEF “SPIKE” OF ELECTRICITY TRAVELING THROUGH A NEURON. AT A MORE FUNDAMENTAL LEVEL, IT CONSISTS OF CHEMICAL PARTICLES MOVING ACROSS THE CELL’S OUTER MEMBRANE, FROM ONE SIDE TO THE OTHER.

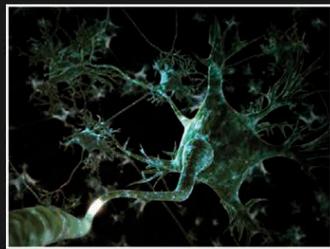
ANATOMY OF AN IMPULSE

Nerve signals are composed of series of discrete impulses, also known as action potentials. A single impulse is caused by a traveling “wave” of chemical particles called ions, which have electrical charges and are mainly the minerals sodium, potassium, and chloride. In the brain, and throughout the body, most impulses in most neurons are of the same strength—about 100 millivolts (0.1 volt). They are also of the same duration—around one millisecond (1/1,000 of a second)—but travel at varying speeds. The information they convey depends on how frequently they pass in terms of impulses per second, where they came from, and where they are heading.

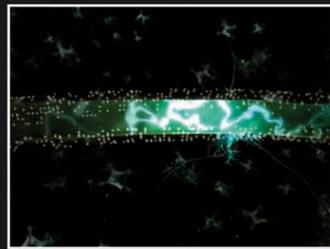


SPEED OF CONDUCTION

Impulses travel at widely differing rates, from 3 to more than 330ft/s (1–100m/s), depending on the type of nerve carrying them. They are fastest in myelinated axons. Here the impulse “jumps” rapidly between the myelin-coated sections from one gap (neurofibril node), to the next node.



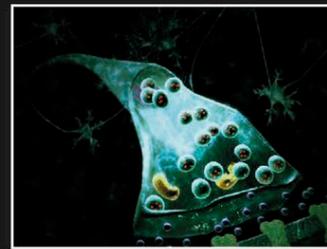
IMPULSE HEADS TOWARD SYNAPSE



AXON IS POLARIZED AT REST



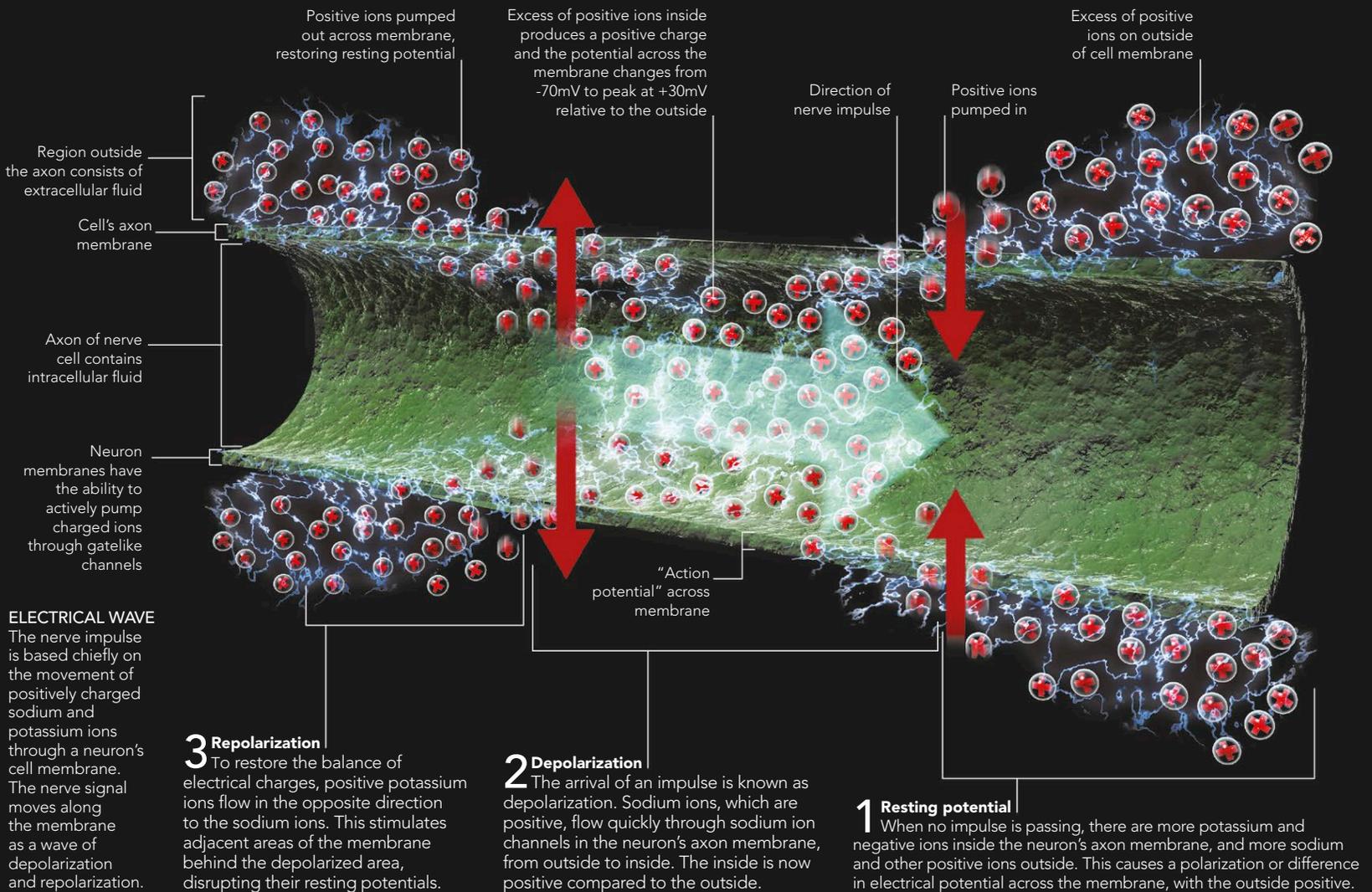
AXON DEPOLARIZES AS IMPULSE PASSES



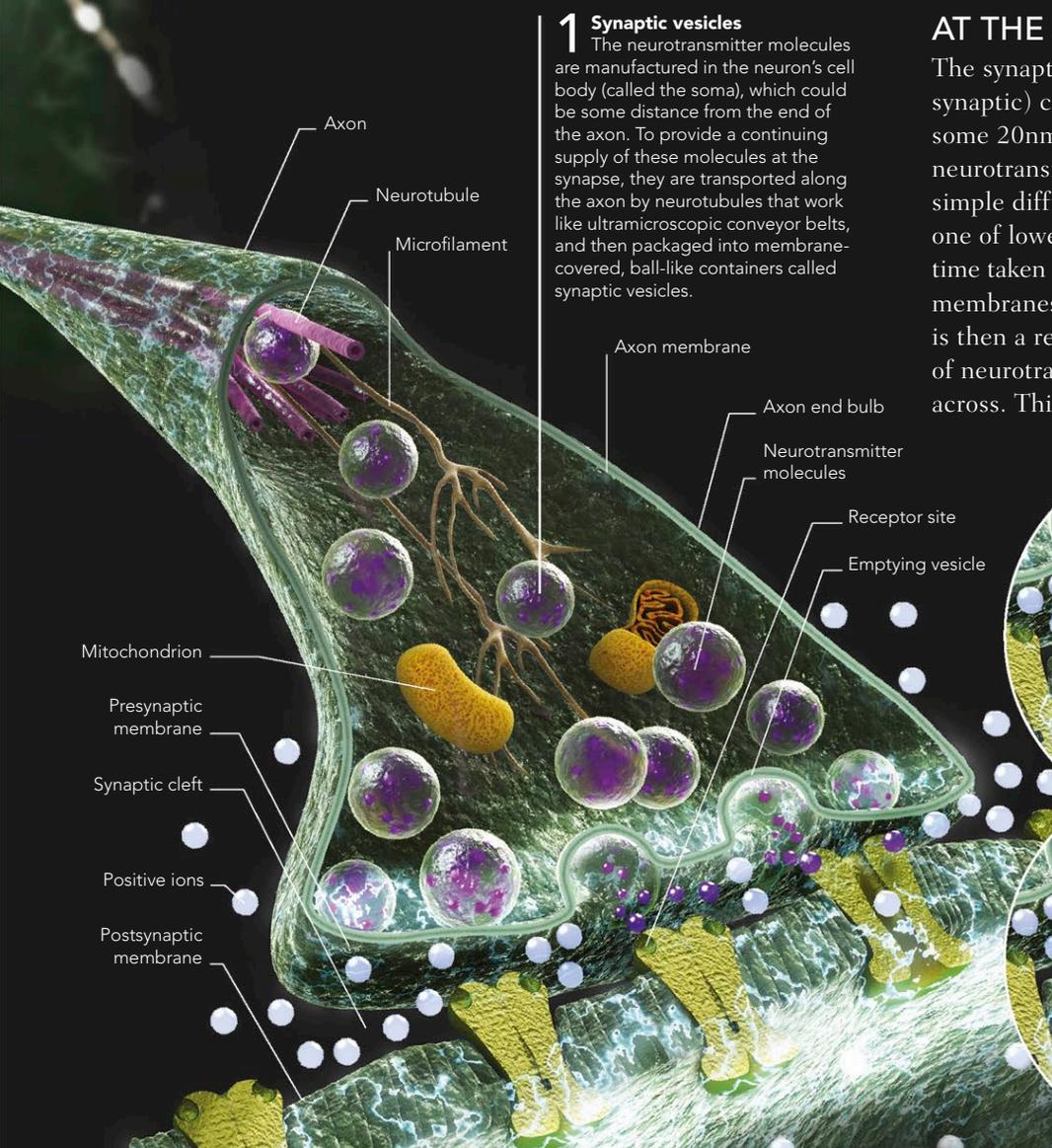
IMPULSE ARRIVES AT SYNAPSE

CHANGING FORM

A nerve impulse is always based on chemical particles. As it passes along a dendrite or axon, it consists of moving electrically charged ions, but at a synapse, it relies more on the structural shape of the chemical neurotransmitter.



ELECTRICAL WAVE
The nerve impulse is based chiefly on the movement of positively charged sodium and potassium ions through a neuron's cell membrane. The nerve signal moves along the membrane as a wave of depolarization and repolarization.

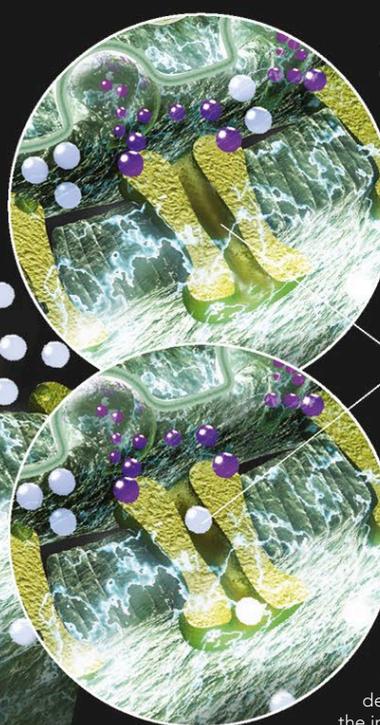


1 Synaptic vesicles
The neurotransmitter molecules are manufactured in the neuron's cell body (called the soma), which could be some distance from the end of the axon. To provide a continuing supply of these molecules at the synapse, they are transported along the axon by neurotubules that work like ultramicroscopic conveyor belts, and then packaged into membrane-covered, ball-like containers called synaptic vesicles.

AT THE SYNAPSE

The synaptic cleft separating the membranes of the sending pre-synaptic cell and the receiving (postsynaptic) cell has a width of some 20nm (20 billionths of a meter). This is so narrow that the neurotransmitter molecules can pass across it extremely quickly by simple diffusion—moving from a region of higher concentration to one of lower concentration. Depending on the neurotransmitter, the time taken for the impulse to pass from the pre- to the postsynaptic membranes is typically less than 2ms ($1/500$ of a second). There is then a recovery delay or clearance time, as the concentrations of neurotransmitter subside, before the next impulse can be sent across. This may last several tenths of a second.

2 Discharge of neurotransmitter
When the nerve impulse or action potential reaches the presynaptic membrane of the axon end bulb, it causes synaptic vesicles to fuse or merge with the membrane. This releases the neurotransmitter molecules to pass or diffuse across the synaptic cleft to the post-synaptic membrane and slot into receptor sites.

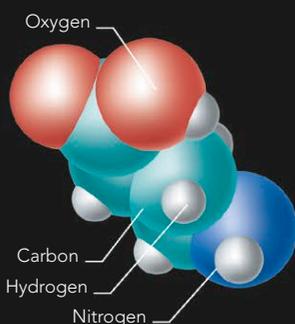


Membrane channel opens
Ions pass through channel

3 Post-synaptic excitation
Neurotransmitter molecules slot into the same-shaped receptor sites of gatelike membrane channels in the postsynaptic membrane (such as the dendrite of the next nerve cell). When this happens, the channel opens and allows positive ions to flow from the outside to the inside of the post-synaptic cell. This triggers a new wave of depolarization, which continues the impulse if it is strong enough.

NEUROTRANSMITTERS

Neurotransmitters are chemicals that allow signals to pass between a neuron and another cell. There are several groups of neurotransmitter molecules. One contains only acetylcholine. A second is known as biogenic amines, or monoamines, and includes dopamine, histamine, norepinephrine, and serotonin. The third group is composed of amino acids, such as GABA, glutamic acid, aspartic acid, and glycine. Many of these substances have other roles in the body. For example, histamine is involved in the inflammatory response. Amino acids (apart from GABA) are also very common, being the building blocks for hundreds of kinds of protein molecules.



GABA MOLECULE
GABA is the chief inhibitory neurotransmitter throughout much of the human brain and nervous system.

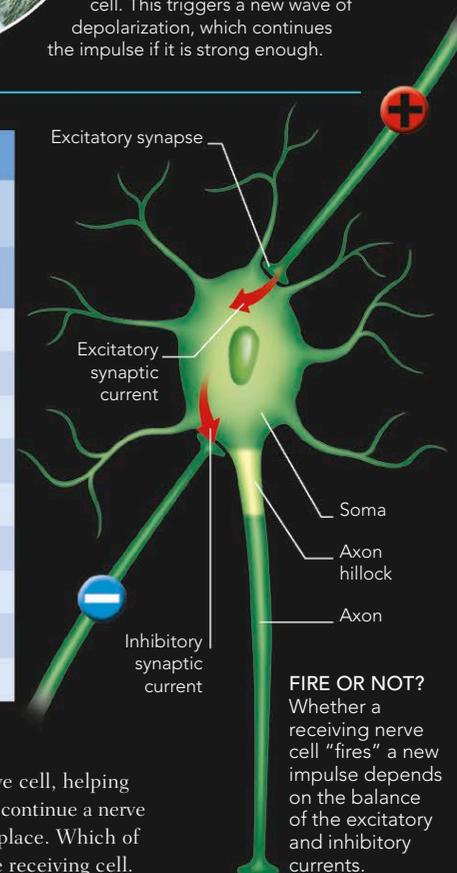
SMALL MOLECULE NEUROTRANSMITTER

Several common examples of neurotransmitters are listed together with their typical effects at synapses.

NEUROTRANSMITTER CHEMICAL NAME	USUAL POST-SYNAPTIC EFFECT
Acetylcholine	Mostly excitatory
Gamma aminobutyric acid (GABA)	Inhibitory
Glycine	Inhibitory
Glutamate	Excitatory
Aspartate	Excitatory
Dopamine	Excitatory and inhibitory
Noradrenaline	Mostly excitatory
Serotonin	Inhibitory
Histamine	Excitatory

EXCITATION AND INHIBITION

A particular neurotransmitter can either excite a receiving nerve cell, helping depolarize the axon hillock (where the soma and axon meet) and continue a nerve impulse, or inhibit it by preventing depolarization from taking place. Which of these occurs depends on the type of membrane channel on the receiving cell.



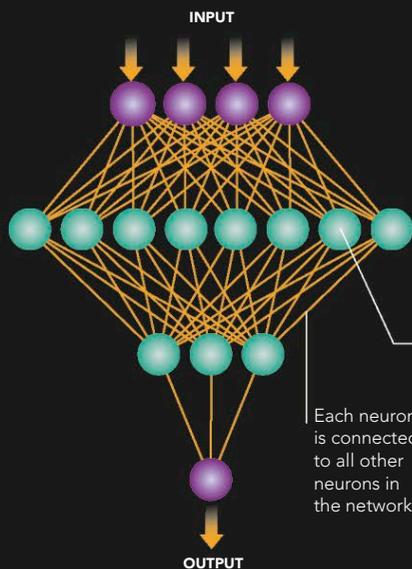
FIRE OR NOT?
Whether a receiving nerve cell "fires" a new impulse depends on the balance of the excitatory and inhibitory currents.

BRAIN MAPPING AND SIMULATION

CREATING AN ARTIFICIAL BRAIN IS A LONG-HELD DREAM THAT IS FINALLY BEING MADE POSSIBLE THANKS TO ADVANCES IN COMPUTER POWER. TWO GLOBAL PROJECTS ARE NOW UNDERWAY TO REPRODUCE A DIGITAL SIMULATION OF THE HUMAN ORGAN. IF THIS IS ACHIEVED, IT WILL EFFECTIVELY BE A BRAIN, ALTHOUGH WHETHER IT WILL BE CONSCIOUS AND WHAT SORT OF EXPERIENCE IT MIGHT HAVE ARE UNKNOWN.

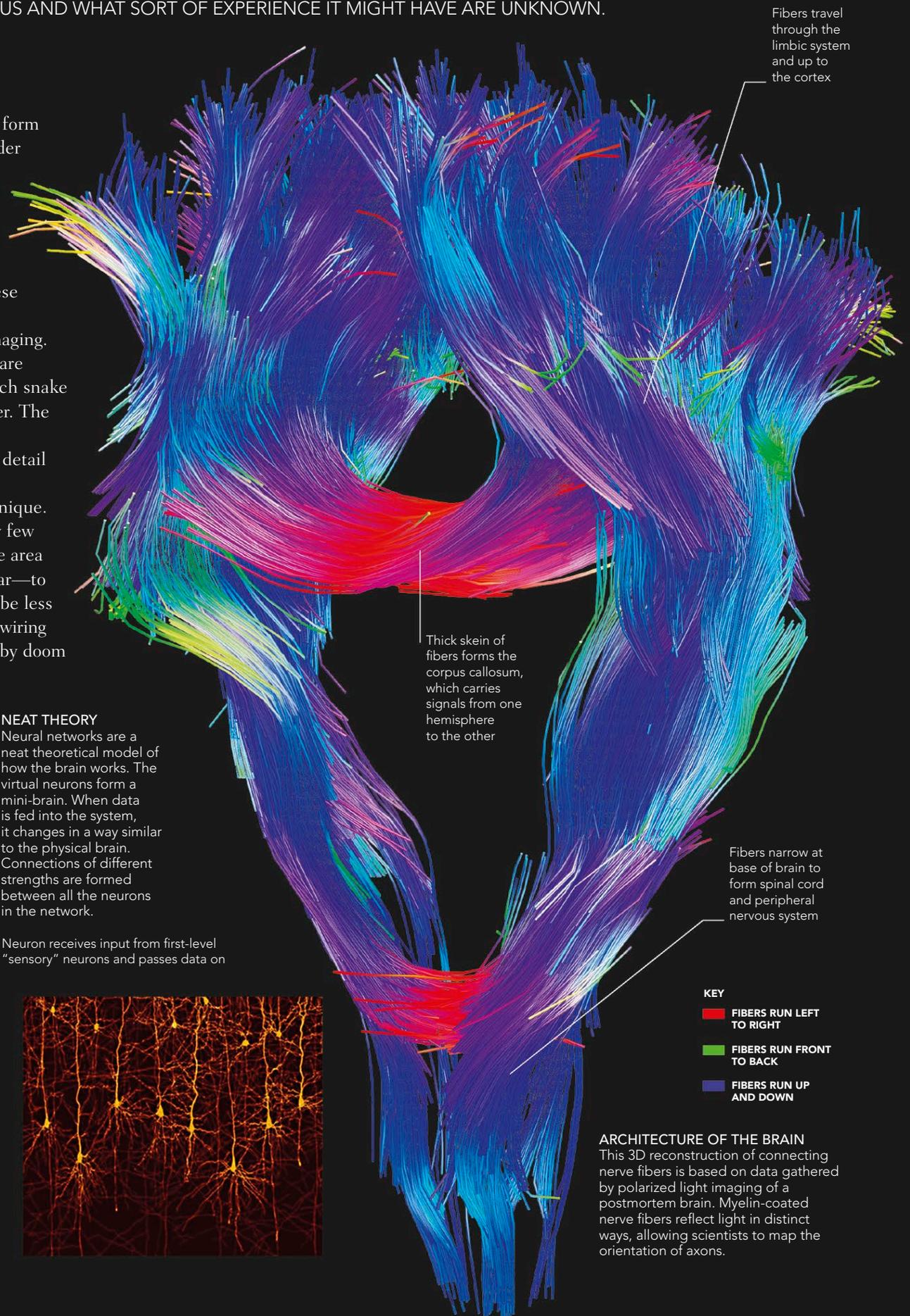
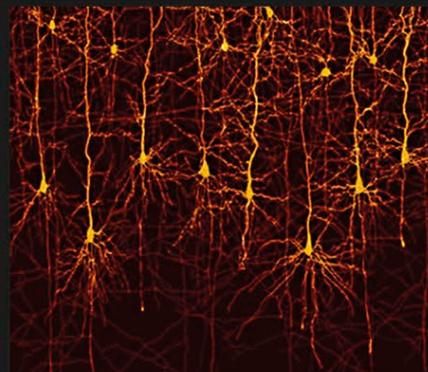
THE CONNECTOME

The connections between neurons form the “wiring” of the brain, and in order to recreate a working simulation, it is essential to know in detail the route taken by information passing from one neuron to another. A global initiative called the Connectome project charts these pathways using a form of MRI scanning called diffusion tensor imaging. The connecting fibers of the brain are skeins of myelin-coated axons, which snake out from one cell to contact another. The overall pattern of neural pathways is similar in all of us, but differs in detail from person to person. It is these differences that make each of us unique. For instance, people with relatively few pathways from their amygdala—the area deep in the brain that generates fear—to their prefrontal cortex are likely to be less nervous than people whose neural wiring allows their forebrain to be deluged by doom alerts from the amygdala.



NEAT THEORY
Neural networks are a neat theoretical model of how the brain works. The virtual neurons form a mini-brain. When data is fed into the system, it changes in a way similar to the physical brain. Connections of different strengths are formed between all the neurons in the network.

COMPLEX WEB
This image of cells in a minute section of neocortex reveals that the network of fibers in the brain is incredibly complex. To produce a model of a brain that really behaves like a human one involves tracing each and every fiber.

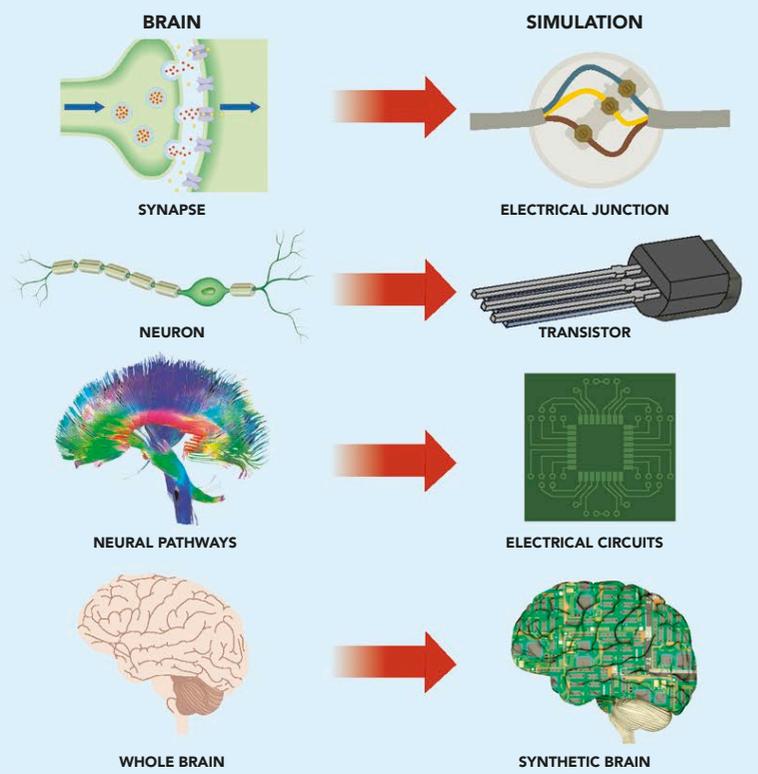


ARCHITECTURE OF THE BRAIN
This 3D reconstruction of connecting nerve fibers is based on data gathered by polarized light imaging of a postmortem brain. Myelin-coated nerve fibers reflect light in distinct ways, allowing scientists to map the orientation of axons.

MAKING A BRAIN

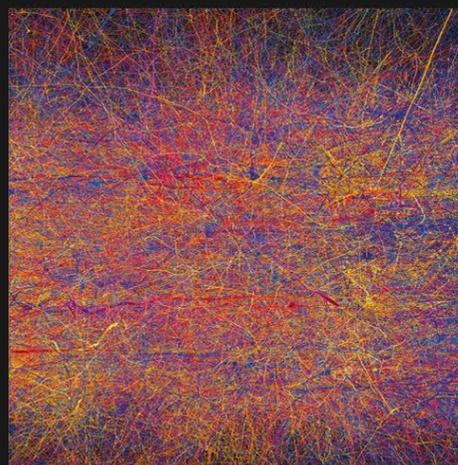
Researchers are working on digital simulations of the brain by mapping its electrical circuitry then modeling it by substituting electrical devices for biological mechanisms (see below). An electrical brain is unlikely to be

conscious or to fulfill all the functions of a real brain because it would need to be embedded in a body and exist in an environment in which to learn. Nor does it include nonelectrical elements, such as hormones.



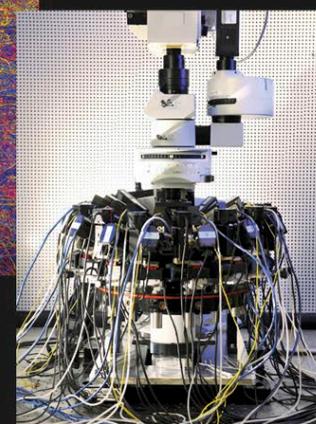
DIGITAL MODELING

The biggest challenge facing neuroscientists is to simulate an entire human brain. The current approach is to identify every neuron in a normal brain and then trace all the connections between them. Bit by bit, the entire organ and its wiring will be determined and the information converted to a digital model, which will be stored on one or more supercomputers. The system could then be run on demand, fed by digital input that mimics sensations triggered by the environment. This should, in theory, function like a real brain. In Europe, this mammoth task is being undertaken by the European Union flagship Human Brain Project (HBP), and a similar endeavor, Brain Research through Advancing Innovative Neurotechnologies (BRAIN), is underway in the US.



PATCH CLAMP

The electrical output of neurons is recorded using a 12-patch clamp instrument (below). The patch clamp allows 12 living neurons to be studied at the same time.



BLUE BRAIN PROJECT

Neurons in the cortex are so dense it is almost impossible to visualize them. The Swiss Blue Brain Project has produced the digital equivalent of around one million neurons and their billion interconnections, as seen here.

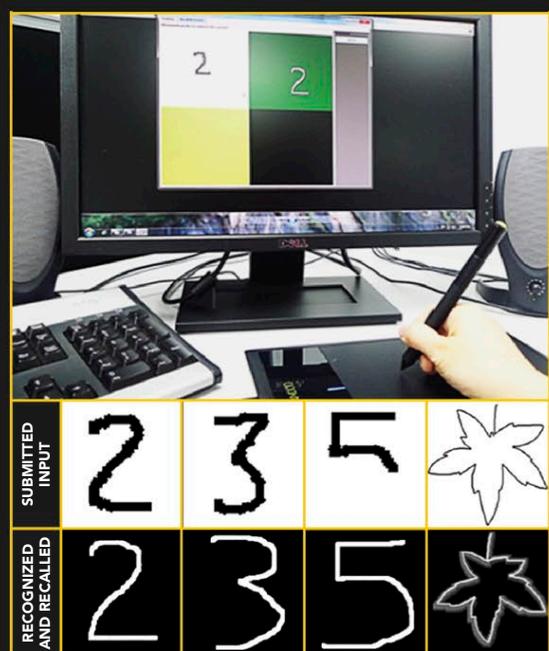
THE SELF-BUILD BRAIN

Another approach to brain simulation is to let a virtual brain grow digitally. The idea is to create a neural network—a system of computer-based information nodes organized to communicate with one another—that will restructure itself as it receives new data. NeuraBASE, for example, is a computer-based artificial-intelligence system that starts with

virtual motor and sensory neurons, each of which responds to an element of information. Real-life stimuli are fed into the system, much as the brain is fed with experiences through the senses. The neurons in NeuraBASE form associations as neurons in the brain do. The virtual links form networks that become denser as more stimuli is fed in, just as biological brains learn through experience. Given enough computer resources, NeuraBASE could in theory grow itself to function like a brain.

LEARNING PROGRAM

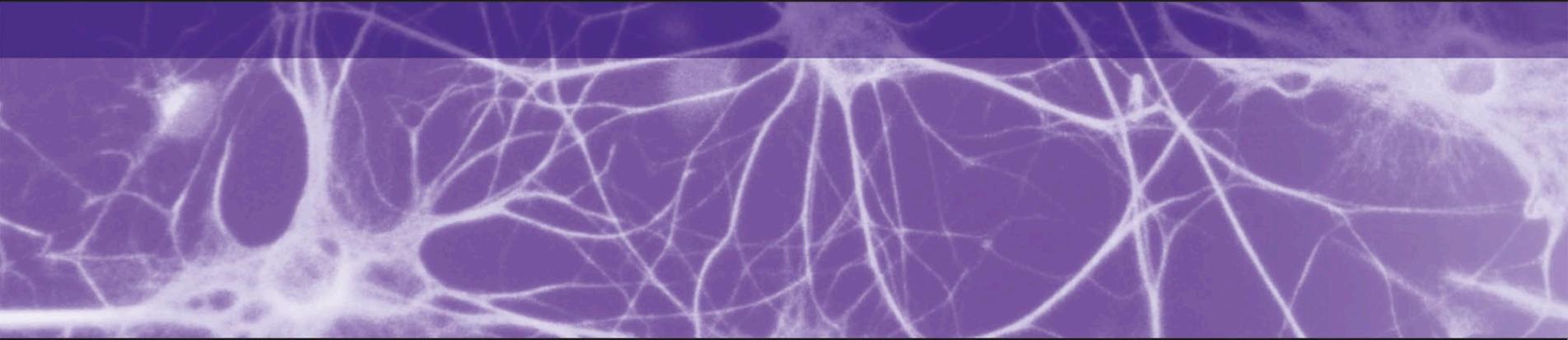
NeuraBASE learns to recognize hand-drawn figures and reproduce them. It does not just copy the input, but, like a human brain, it recognizes the idea encapsulated in the input even when—like the 5 here—it is incomplete.



AUTOMATA

Attempts to replicate brainlike systems go back a long way. Automata—apparently driven by internal intelligence—were popular entertainments in the 18th century and are the forerunners of today's robots. Lifelike figures had hidden clockwork mechanisms. These moved their limbs and allowed them to carry out seemingly intelligent actions like writing. Although the workings of such mechanical “brains” seem crude today, the idea—to make an artificial system that functions like a human being—is the same as that driving today's huge projects.





THERE ARE NO SIGHTS, SOUNDS, TASTES, OR SMELLS IN THE WORLD—JUST VARIOUS TYPES OF WAVES AND MOLECULES. SENSATIONS, THEREFORE, ARE “VIRTUAL” CONSTRUCTS CREATED BY THE BRAIN. THE SENSE ORGANS BEGIN THIS EXTRAORDINARY ACT OF TRANSFORMATION BY TURNING STIMULI, SUCH AS LIGHT WAVES OR THE TOUCH OF CERTAIN MOLECULES, INTO ELECTRICAL SIGNALS THAT ARE CARRIED TO BRAIN AREAS DEDICATED TO DEALING WITH THAT TYPE OF INPUT. SOME STIMULI ALSO ORIGINATE FROM WITHIN THE REST OF THE BODY. ALTHOUGH SOME SENSATIONS ARE CONSCIOUSLY EXPERIENCED, MANY REMAIN UNCONSCIOUS.

THE SENSES



HOW WE SENSE THE WORLD

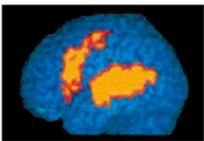
THE BRAIN REACHES OUT TO THE ENVIRONMENT VIA OUR SENSE ORGANS, WHICH RESPOND TO VARIOUS STIMULI SUCH AS LIGHT, SOUND WAVES, AND PRESSURE. THE INFORMATION IS TRANSMITTED AS ELECTRICAL SIGNALS TO SPECIALIZED AREAS OF THE CEREBRAL CORTEX (THE OUTER LAYER OF THE CEREBRUM) TO BE PROCESSED INTO SENSATIONS SUCH AS VISION, HEARING, AND TOUCH.

MIXED SENSES

Sensory neurons respond to data from specific sense organs. Visual cortical neurons, for example, are most sensitive to signals from the eyes. But this specialization is not rigid. Visual neurons have been found to respond more strongly to weak light signals if accompanied by sound, suggesting that they are activated by data from the ears as well as the eyes. What you see also influences what you hear. In a phenomenon known as the McGurk effect, if someone says “ba,” while you watch someone mouthing “ga,” you hear a third sound, “da.” This is the brain’s attempt to

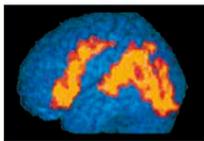
make sense of conflicting inputs. Other studies show that in people who are blind or deaf, some neurons that would normally process visual or auditory stimuli are “hijacked” by the other senses. Hence, blind people hear better and deaf people see better.

HEARING PERSON PROCESSES SPEECH

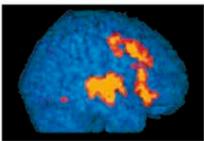


LEFT SIDE OF BRAIN

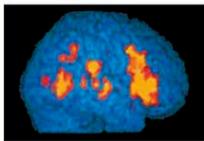
DEAF PERSON PROCESSES SIGNING



LEFT SIDE OF BRAIN



RIGHT SIDE OF BRAIN



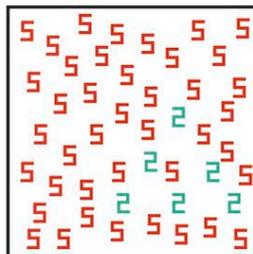
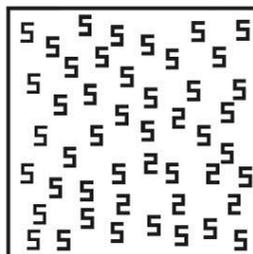
RIGHT SIDE OF BRAIN

“HEARING” WITHOUT SOUND
These fMRI scans of human brains show some sensory neurons that are activated by speech in hearing people being used in deaf people to process sign language.

SYNESTHESIA

Most people are aware of only a single sensation in response to one type of stimulus. For example, sound waves make noise. But some people experience more than one sensation in response to a single stimulus. They may “see” sounds as well as hear them, or “taste” images. Called synesthesia, this sensory duplication occurs when the neural pathway from a sense organ diverges and carries data on one type of stimulus to a part of the brain that normally processes another type.

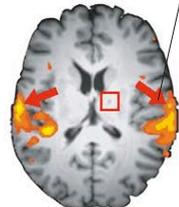
NUMBER TEST
Some synesthetes see numbers as having different colors. Variations in shape “pop out” (bottom) for them.



CONTROL GROUP



SYNESTHETES



Larger area responds to sounds

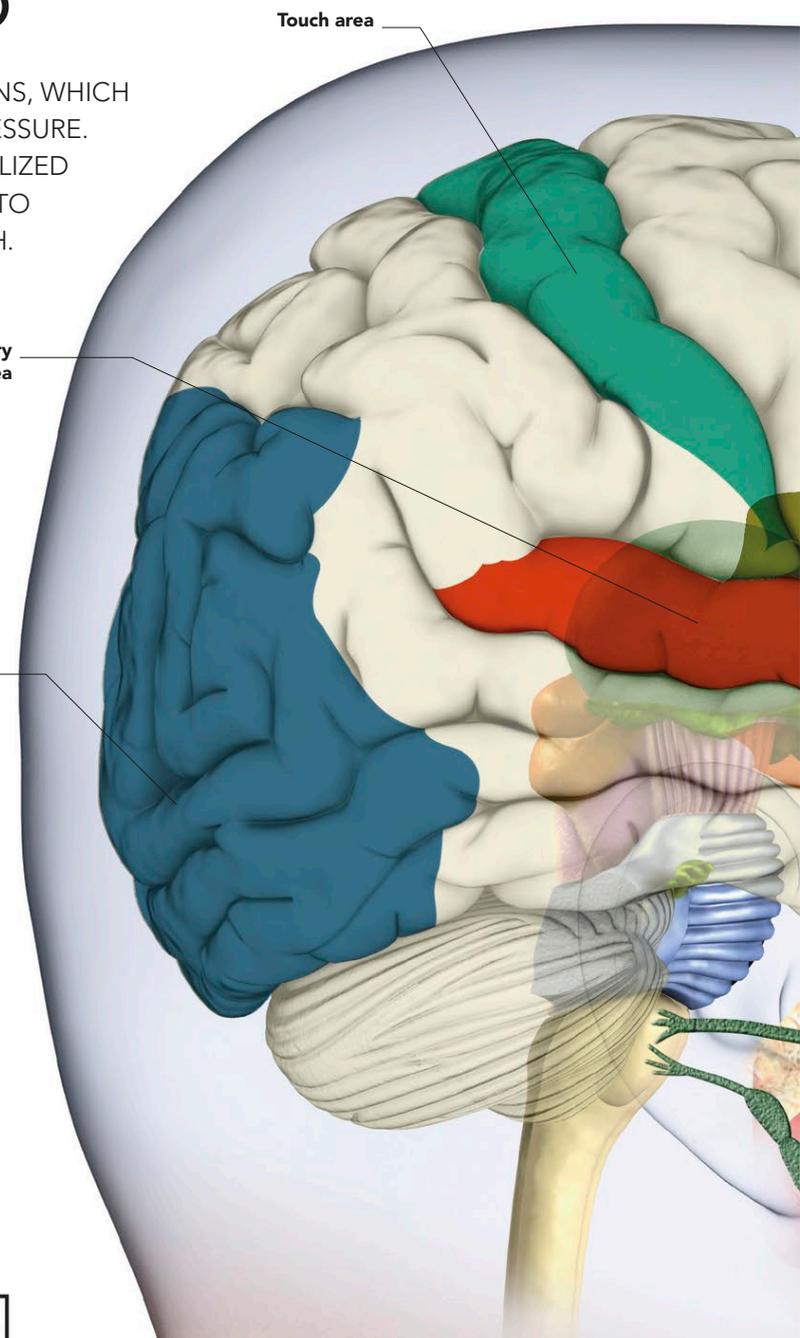
Increased activity

RICHER EXPERIENCE
These fMRI scans show brain activity in people listening to sounds. In response, those with synesthesia generate more sensations than others, suggesting that the condition enriches everyday experiences by increasing sensation.

Auditory area

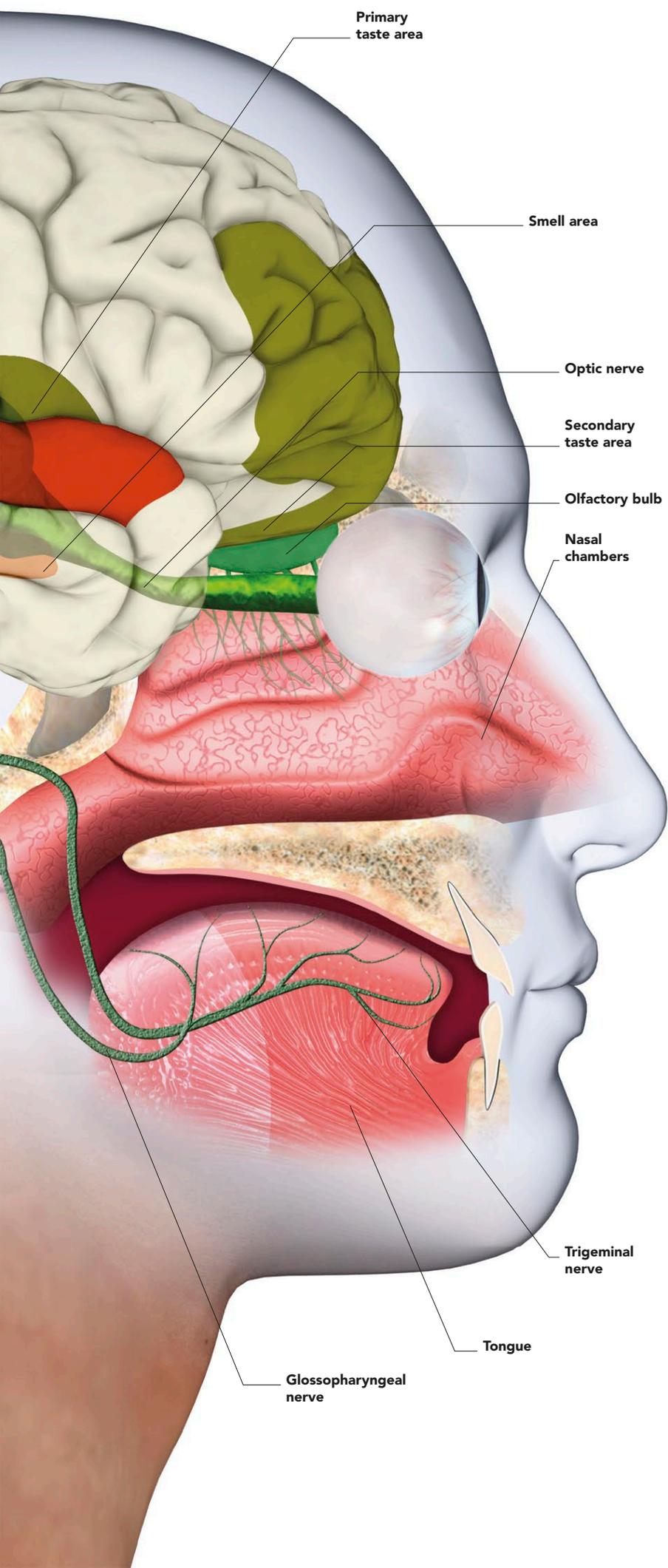
Visual area

Touch area



ROUTES TO SENSATION

Sense organs detect stimuli, turn the information into electrical signals, and transmit these to areas of the brain that are specialized to process specific types of sensory information into sensations such as sound, vision, taste, smell, touch, and pain. Some of this data is then “forwarded” to areas of the brain that make it conscious.



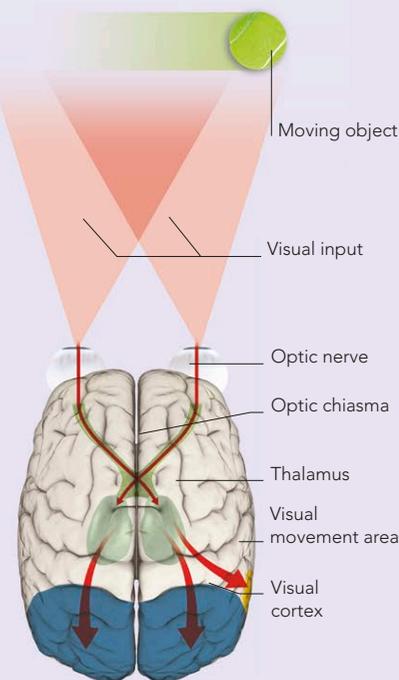
CONSCIOUS AND UNCONSCIOUS SENSATION

Our brains are bombarded with sensory information, but only a fraction of it reaches consciousness. Most sensory signals fizzle out unnoticed. Especially “loud” or important data grabs our attention (see pp.182–183), and we become conscious of it. Sensations we are not conscious of may still guide our actions. For example, unconscious sensations relating to our body position allow us to move without thinking about it. Also, sights and sounds that we fail to notice may nevertheless influence our behavior.

BLINDSIGHT

Blindsight gives visual knowledge without conscious vision. It is likely that we all have it, but it is most easily measured in people who are blind due to cortical damage. Such people cannot knowingly see, but if something is put in front of them they can correctly “guess” what it looks like, without knowing how. Most blindsight studies use moving objects. The subjects say they can’t see the objects but can usually “guess” the direction of movement correctly.

“GUESSING” MOVEMENT
Blindsight for movement is probably due to information from the eyes stimulating the visual movement area directly via an unconscious route. Conscious vision depends on activation in the primary visual cortex, stimulated via another pathway.



BOTTOM-UP AND TOP-DOWN PROCESSING

Sensations are triggered externally, by an occurrence that impacts on a sense organ, and internally, by memory or imagination. The former is known as “bottom-up” and the latter as “top-down” processing (see p.87). The two combine to create our experience of reality. Each person’s experience of a given event is different. Physiological differences affect bottom-up processing. One person’s color-processing area in the brain may be highly sensitive, for example, so that colors are more vibrant than average. Also, an individual’s own memories, knowledge, and expectations affect top-down processing.



LETTER OR NUMBER?

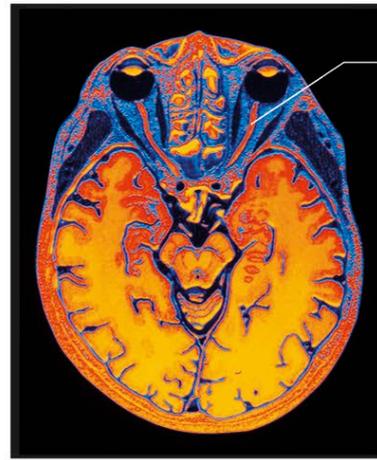
The symbol in the center is identical in these two images, and our “bottom-up” visual process sees it as such. However, expectation, or “top-down” visual processing, leads to us seeing it as different. The context in which it appears on the left causes us to see it as the letter “B,” while we see it as “13” in the right-hand image.

THE EYE

THE EYE IS AN EXTENSION OF THE BRAIN. IT CONTAINS ABOUT 125 MILLION LIGHT-SENSITIVE NERVE CELLS, KNOWN AS PHOTORECEPTORS, WHICH GENERATE ELECTRICAL SIGNALS THAT ALLOW THE BRAIN TO FORM VISUAL IMAGES.

THE STRUCTURE OF THE EYE

The eyeball is a fluid-filled orb with a hole in the front (the pupil); a sheet of nerve cells (the retina), some of which are light-sensitive, at the back; and a lens in between. The pupil is surrounded by pigmented fibers (the iris) and covered by a sheet of clear tissue (the cornea) that merges with the tough outer surface or the “white” of the eye (the sclera). The optic nerve passes through a hole in the back of the eye (the optic disk) to enter the brain.



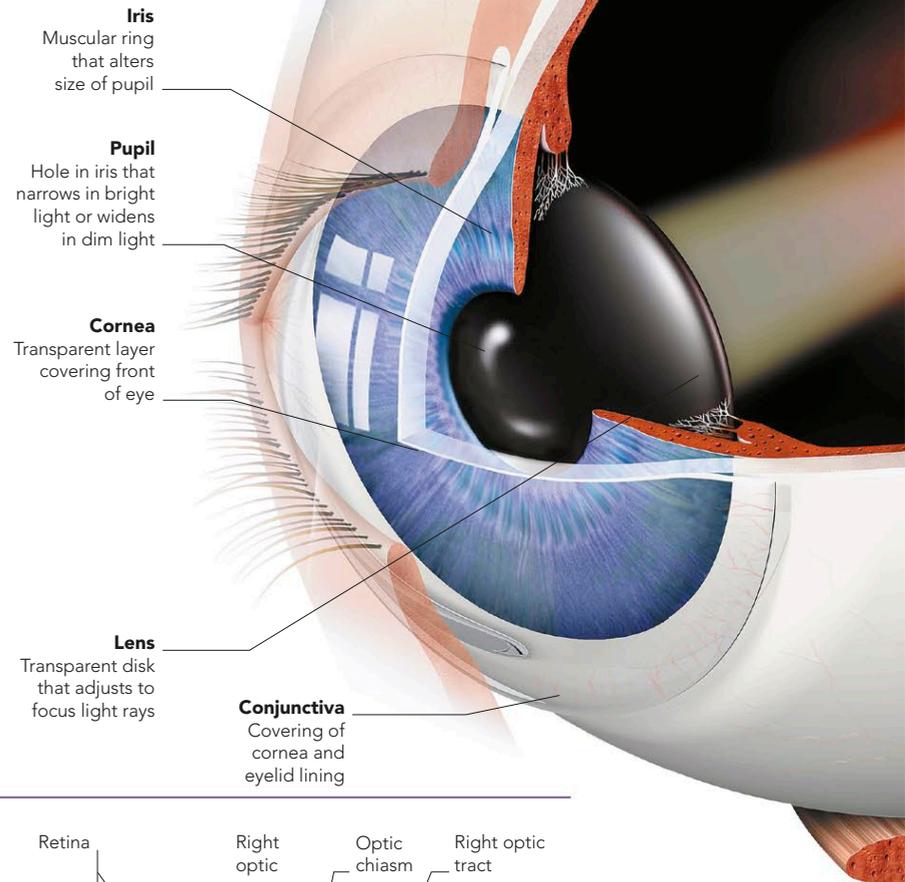
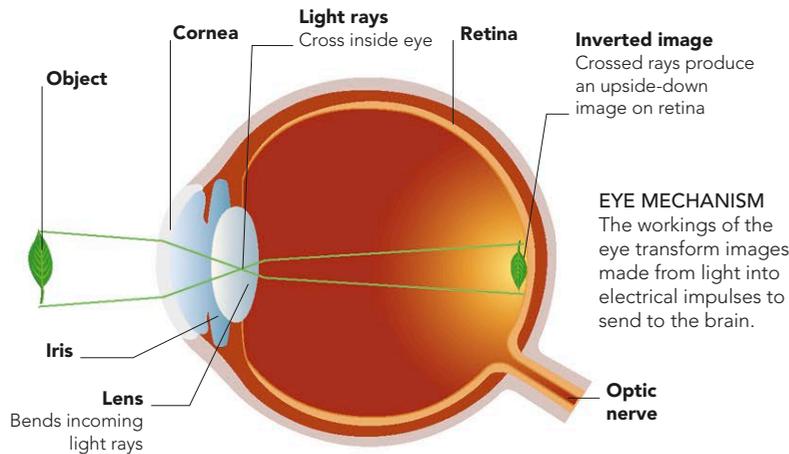
Optic nerve

OPTIC NERVE

This colored MRI scan shows the thick bundle of fibers, the optic nerve, that connects each eye to the brain.

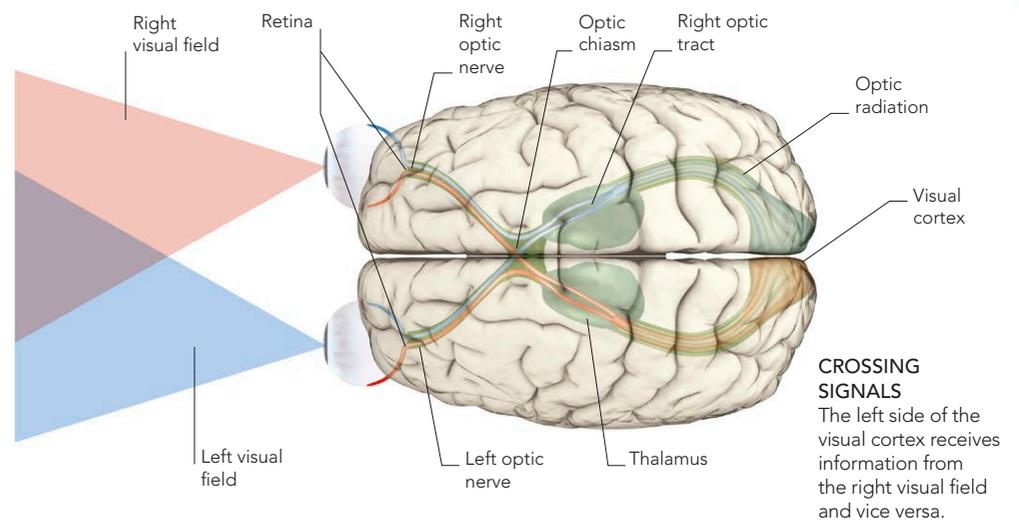
SEQUENCE OF VISION

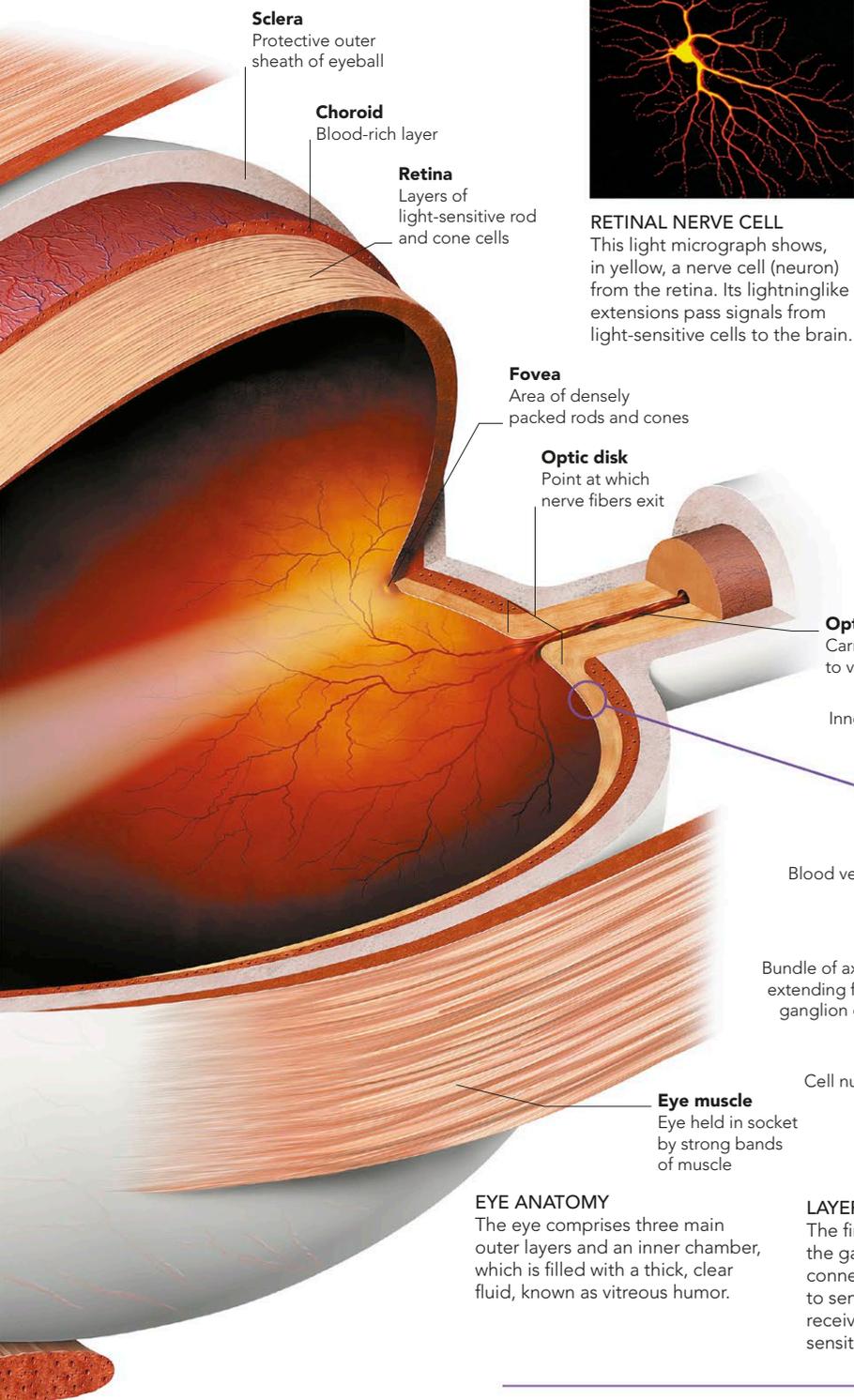
Light passes through the cornea and enters the eye through the pupil. The iris controls how much enters by changing shape, so the pupil appears smaller in bright light and expands in shade. Light rays then pass through the lens, which bends (refracts) the light so it converges on the retina. If focusing on a near object, the lens thickens to increase refraction, but if the object is distant, the lens needs to flatten. The light then hits the photoreceptors in the retina, some of which fire, sending electrical signals to the brain via the optic nerve.



VISUAL PATHWAYS

Information from the eyes has to travel right to the back of the brain before it starts to be turned into conscious vision. En route, it passes through two major junctions, and half of it crosses from one side of the brain to the other. Signals from the two optic nerves first converge at a crossover junction called the optic chiasm. Fibers carrying information from the left side of each retina join up and proceed as the left optic tract, while fibers carrying information from the right side form the right optic tract. Each tract ends at the lateral geniculate nucleus, which is part of the thalamus, but their signals continue to the visual cortex via bands of nerve fibers, called the optic radiation.

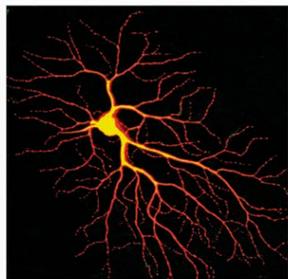




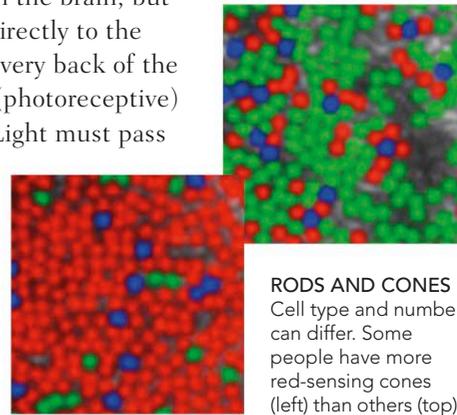
EYE ANATOMY
The eye comprises three main outer layers and an inner chamber, which is filled with a thick, clear fluid, known as vitreous humor.

THE RETINA

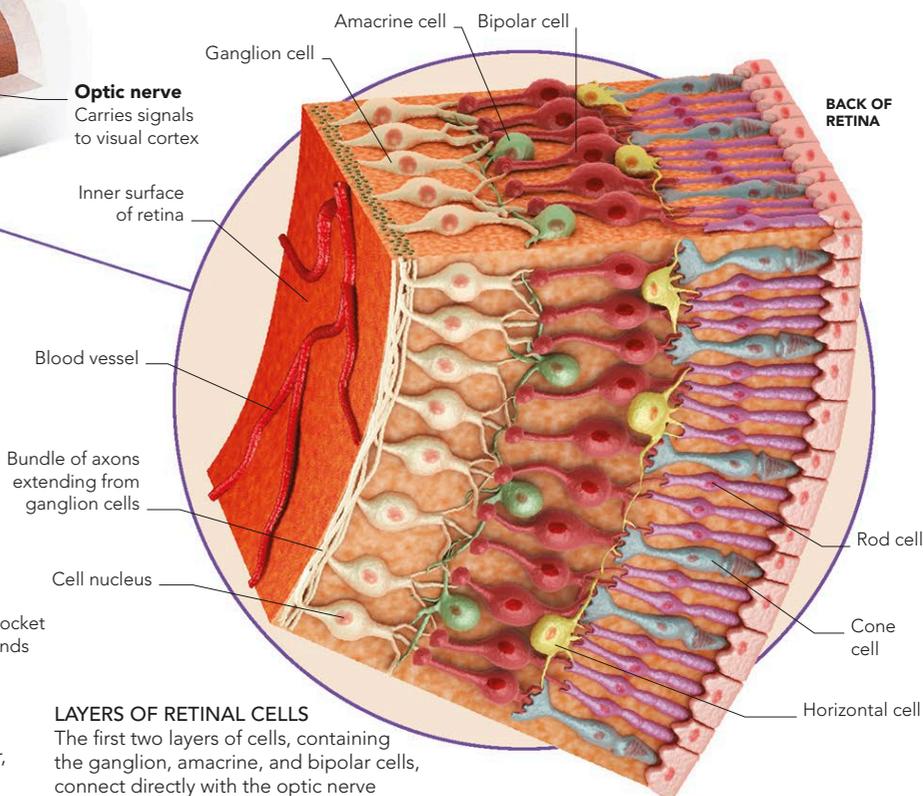
The retina contains three layers of cells, each one connecting to the next via junctions between neurons (synapses), through which information (electrical impulses) can pass. The first two layers send signals to the visual cortex in the brain, but these cells do not respond directly to the light. The third layer, at the very back of the retina, bears light-sensitive (photoreceptive) cells—the rods and cones. Light must pass over the first two layers to trigger any neural activity. Rods, which make up 90 percent of photoreceptors, are responsible for vision in dim light. Cones detect fine detail and color.



RETINAL NERVE CELL
This light micrograph shows, in yellow, a nerve cell (neuron) from the retina. Its lightninglike extensions pass signals from light-sensitive cells to the brain.



RODS AND CONES
Cell type and number can differ. Some people have more red-sensing cones (left) than others (top).



LAYERS OF RETINAL CELLS
The first two layers of cells, containing the ganglion, amacrine, and bipolar cells, connect directly with the optic nerve to send signals to the brain. Horizontal cells receive and regulate input from the light-sensitive rods and cones in the third layer.



THE FOVEA

The central part of the retina allows for far sharper vision than the periphery because it contains more cones (which pick up detail and color) than rods. Right in the center of the retina is the fovea, a tiny pitted area where cones are most densely packed. In addition to being more numerous, foveal cones can also pass on more detail, because almost every one has a dedicated signal-sending pathway to the brain.

FOVEAL MAGNIFICATION
This electron micrograph shows the part of the retina that gives sharpest vision, the foveal pit.

Light-sensitive cells elsewhere on the retina must share these means of output.

BLIND SPOT

Signal-carrying nerve fibers bundle together at the optic disk in the back of the eye to form the optic nerve. Consequently, this area has no light-sensitive cells, so it forms a “blind spot.” We are unaware of this gap in our vision because the brain “fills in” the area we can’t see.



OPTIC DISK
This ophthalmoscope image of a retina shows the optic disk, the site of the blind spot.

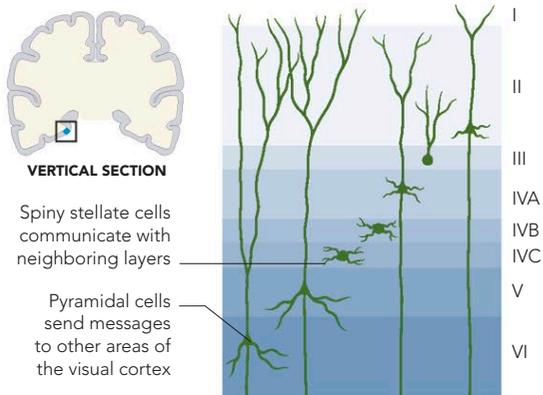
THE VISUAL CORTEX

THE VISUAL AREAS OF THE BRAIN ARE AT THE BACK OF THE BRAIN; THEREFORE, INFORMATION FROM THE EYES HAS TO TRAVEL THE FULL DEPTH OF THE SKULL BEFORE IT BEGINS TO BE PROCESSED INTO SIGHT. VISUAL INFORMATION CAN GUIDE ACTIONS WITHIN ONE-FIFTH OF A SECOND, BUT IT TAKES ABOUT HALF A SECOND FOR US TO SEE AN OBJECT CONSCIOUSLY.

VISUAL AREAS

The visual cortex is divided into several functional areas, each of which specializes in a particular aspect of vision (see table, right). The process is similar to assembly-line production: raw material is checked in by V1, then sent on to other vision areas, which contribute shape, color, depth, and motion. These components are then combined to form a whole image. Because of the modular nature of vision, if one of the sight areas is damaged, a particular visual component may be lost while the others remain intact. Cell death in the motion-detecting area, for example, may cause the world to be seen as a series of still snapshots.

AREAS OF THE VISUAL CORTEX	
AREA	FUNCTION
V1	Responds to visual stimuli
V2	Passes on information and responds to complex shapes
V3A, V3D, VP	Registers angles and symmetry, and combines motion and direction
V4D, V4V	Responds to color, orientation, form, and movement
V5	Responds to movement
V6	Detects motion in periphery of visual field
V7	Involved in perception of symmetry
V8	Probably involved in processing of color

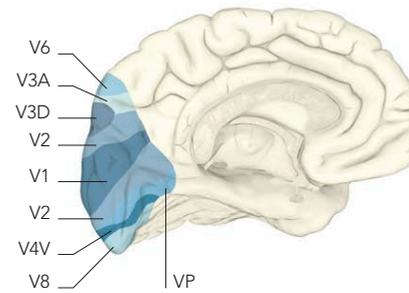
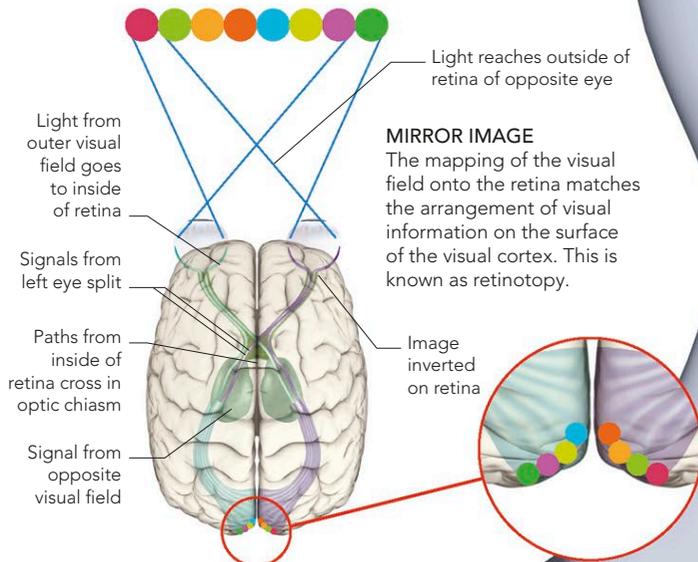


CORTICAL LAYERS

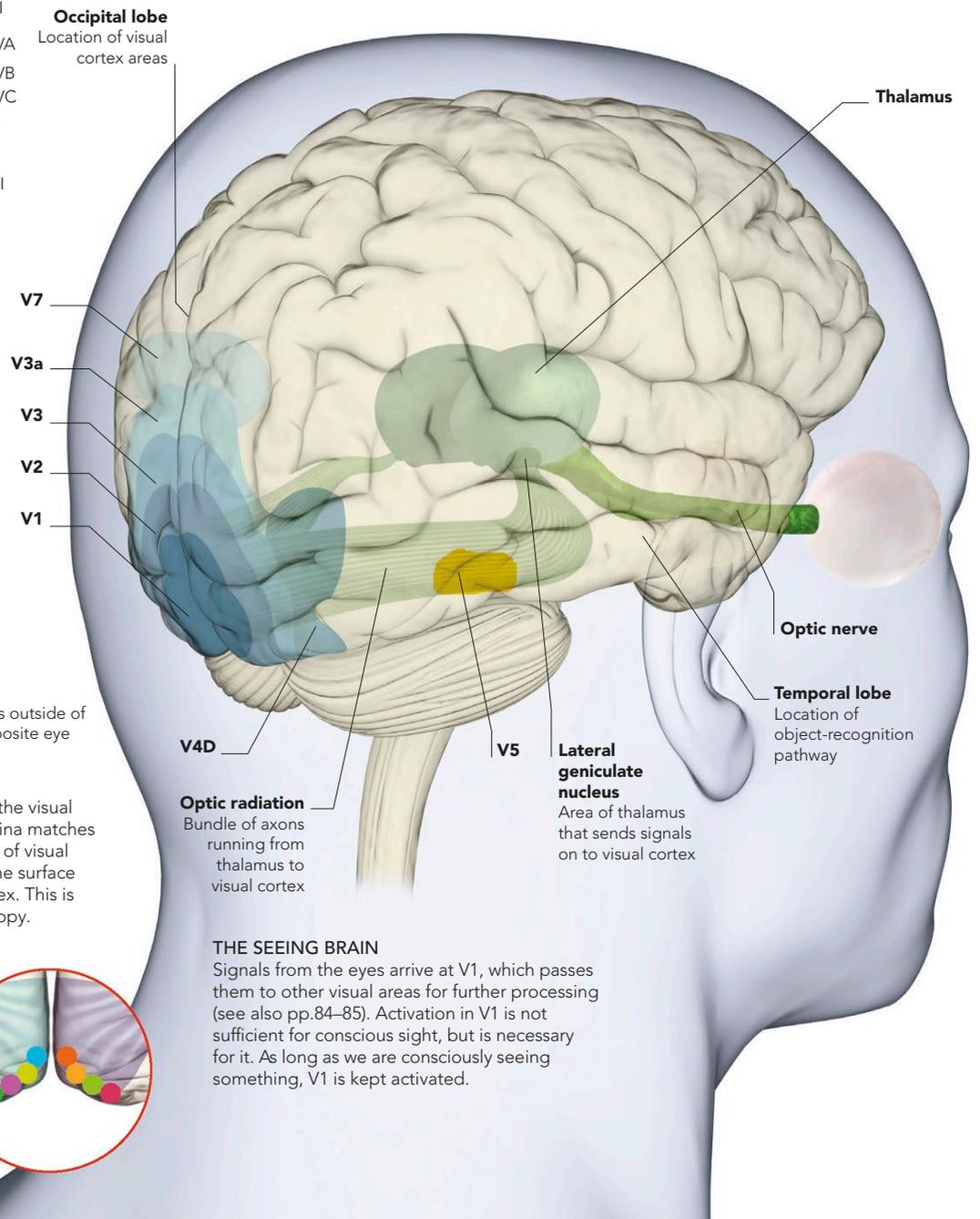
The primary visual cortex consists of several cell layers, numbered I to VI, each of which contains a special mix of cells. Each layer sends and receives signals to and from different parts of the brain.

THE MIND'S MIRROR

The crisscrossed layout of the visual pathways (see p.80) causes the view seen by the eyes to be reversed, so it registers on the primary visual cortex (V1) as a mirror image. Signals from the left field of vision end up in the right hemisphere and vice versa. The information is passed between the two sides to give a shared view. In certain rare conditions, each side of the brain sees something different—the person appears to be in “two minds” (see p.11, p.199).



INTERIOR CORTEX
Some, but not all, of the visual processing areas curve around the back of the brain and into the groove between the hemispheres.

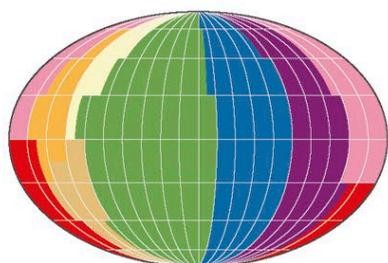


THE SEEING BRAIN

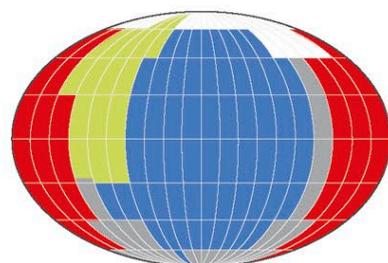
Signals from the eyes arrive at V1, which passes them to other visual areas for further processing (see also pp.84–85). Activation in V1 is not sufficient for conscious sight, but is necessary for it. As long as we are consciously seeing something, V1 is kept activated.

DISTINGUISHING COLORS

In theory the human visual system can distinguish millions of colors, but in practice the number of colors we see depends on whether we have learned to see them. Presented with a globe showing all possible colors, people can easily distinguish those for which they have distinct names. But if a range of hues is lumped together under a single name, they often find it hard to see the differences.



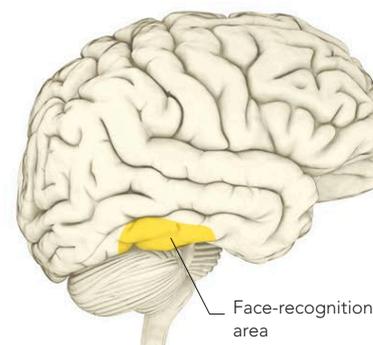
ENGLISH HUES
This globe shows the spectrum of color, which is divided into eight basic categories (red, orange, green, blue, purple, yellow, and brown) in the English language.



OTHER HUES
Studies suggest that language affects how people see the globe. For example, the Berinmo tribe of Papua New Guinea split colors into five categories, each of which relates to a different hue from those above.

RECOGNIZING OBJECTS

Conscious sight requires the brain to recognize what it is seeing. To achieve this, the image is forwarded from the occipital lobe to other brain areas concerned with emotion and memory. Here it gains information relating to its function, its identity, and its emotional significance. One of the first steps is in the object-recognition area, which runs along the bottom rim of the temporal lobe. Human faces are dealt with in a particular subregion that has evolved to make fine distinctions. Its ability to distinguish tiny differences between individual faces makes nearly all of us “experts” at recognizing one another.



FACE-RECOGNITION AREA
Part of the brain's object-recognition path scrutinizes things of importance. This area processes objects that call for fine discrimination, such as faces.

GREEBLES

Greebles are organic-looking objects used in studies that, like faces, are each slightly different from one another. At first sight the differences are easily overlooked, but as people become familiar with Greebles their brains start processing the sight of them in the face-recognition area. This allows them to see the tiny differences very clearly and they become Greeble “experts.”



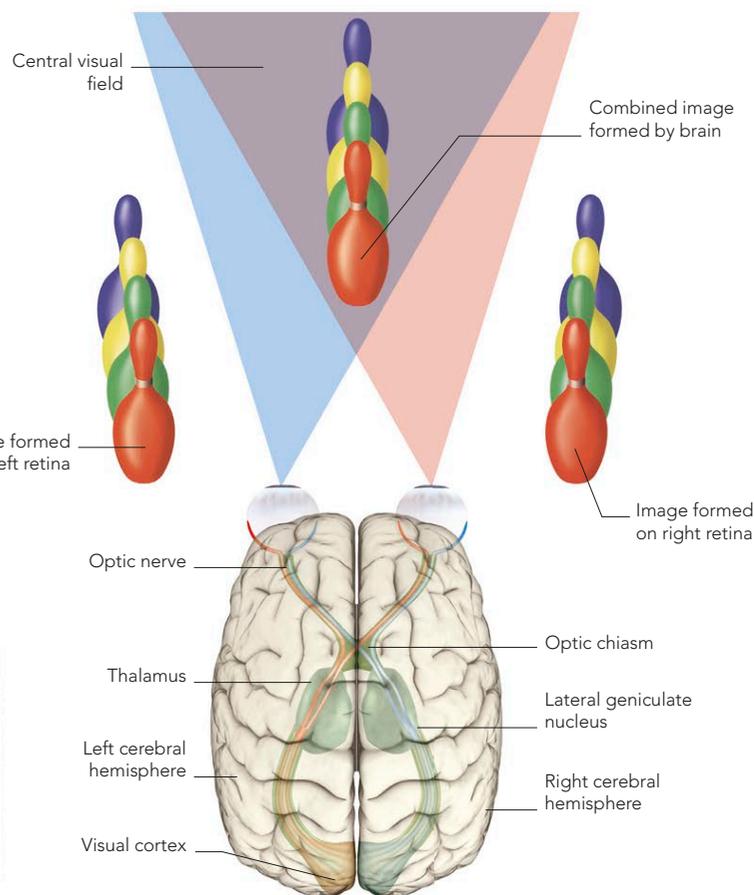
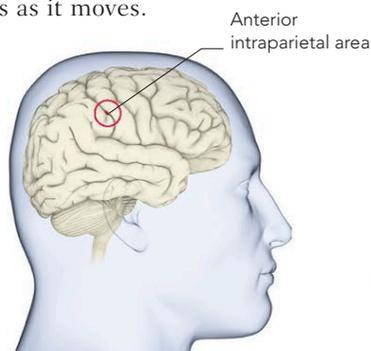
DEPTH AND DIMENSION

The brain uses two types of cues to produce our three-dimensional view of the world. One is the slightly different image recorded by each eye (spatial binocular disparity), and the other is the way the perceived shape of an object shifts as it moves.

Both cues come together in an area of the brain called the anterior intraparietal area (AIP), which lies between the visual processing areas and the part of the brain devoted to monitoring our position in space.

DEPTH AREA

The AIP combines two types of visual cue to calculate distance and depth. This information guides the movements involved in reaching out and grasping objects.



STEREOGRAM

Stereoscopic images make use of the way the brain processes visual information to trick it into seeing a three-dimensional image when in fact there is only a flat plane. One way to do this is to present, side by side, two minutely differing images of the same scene. The difference between them is that which would normally be perceived by each eye—a tiny shift of perspective equal to the distance between the eyes. These illusions were popular in Victorian times.



PHANTOM IMAGE

If you can force your eyes to cross or to diverge, so that each eye sees just one picture, a ghostly third image appears in the center in three dimensions.

3-D VISION

The slightly differing views provided by each eye, combined with information about how shapes change as they move across the visual field, produce a three-dimensional view of the world.

VISUAL PATHWAYS

CONSCIOUS VISION IS THE FAMILIAR PROCESS OF SEEING SOMETHING, WHILE UNCONSCIOUS VISION USES INFORMATION FROM THE EYES TO GUIDE BEHAVIOR WITHOUT OUR KNOWING IT IS HAPPENING. THE TWO TYPES OF VISION ARE PROCESSED ALONG SEPARATE PATHWAYS IN THE BRAIN. THE UPPER (DORSAL) ROUTE, IS UNCONSCIOUS AND GUIDES ACTION, WHILE THE LOWER (VENTRAL) PATH IS CONSCIOUS AND RECOGNIZES OBJECTS.

DORSAL AND VENTRAL ROUTES

Electrical signals from the eyes travel to the primary visual cortex, where the brain begins to process them into vision. The signals are then sent on to other brain regions via the two separate dorsal and ventral pathways.

DORSAL

THE "WHERE" PATHWAY

The dorsal, or "where," pathway carries signals triggered by a visual stimulus—for example, the light bouncing off a nearby object—from the visual cortex to the parietal cortex. Along the way, it passes through areas that calculate the object's location in relation to the viewer and creates an action plan in relation to it. The dorsal path gathers information about motion and timing that is integrated into the action plan. All the information needed to, say, duck a flying object, is gathered along this path with no need for conscious thought.

Parietal lobes
Depth and position of object in relation to observer are gauged

V7
Contributes to perception of symmetry

V3a
Information on motion and direction is collated here

VENTRAL

THE "WHAT" PATHWAY

The ventral, or "what," pathway follows a route that takes it first through a series of visual processing areas, each of which adds a specific aspect of perception, such as shape, color, depth, and so on (see pp.88–89). The loosely formed representation then passes into the bottom edge of the temporal lobe, where it is matched or compared to visual memories in order to achieve recognition. Some information continues along this pathway to the frontal lobes, where it is assessed for meaning and significance. At this stage, it becomes a conscious perception.

V3
Angles and orientation analyzed—paths split here

V2
Information passed on through secondary visual cortex—complex shapes are registered here

V1
Signals from eyes received in primary visual cortex

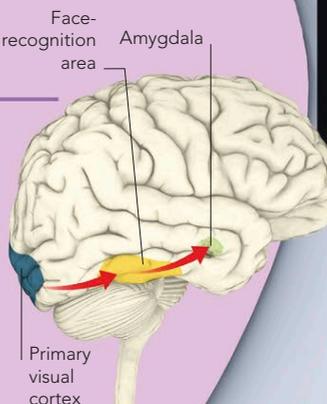
V4D
Involved in perception of color, orientation, form, and movement

V5
Direction of movement detected here

RECOGNIZING FACES

Different types of visual stimuli are processed in different parts of the brain. Faces, which are recognized by the pattern of human facial features, activate the face-recognition area. This extracts information about facial expression and forwards it to relevant brain areas. When a face matches a memory, the information is sent to the frontal lobes for further processing.

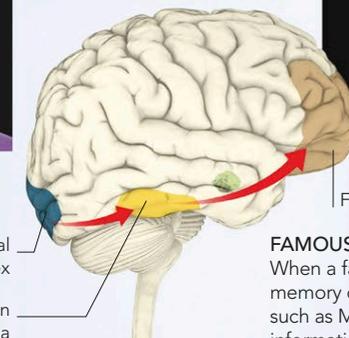
FAMILIAR PERSON
Emotional recognition is near-instant. The pathway runs from the visual cortex via the face area to the amygdala.



SEEING SOMEONE FAMILIAR



EMOTIONAL



SEEING SOMEONE FAMOUS



FACTUAL

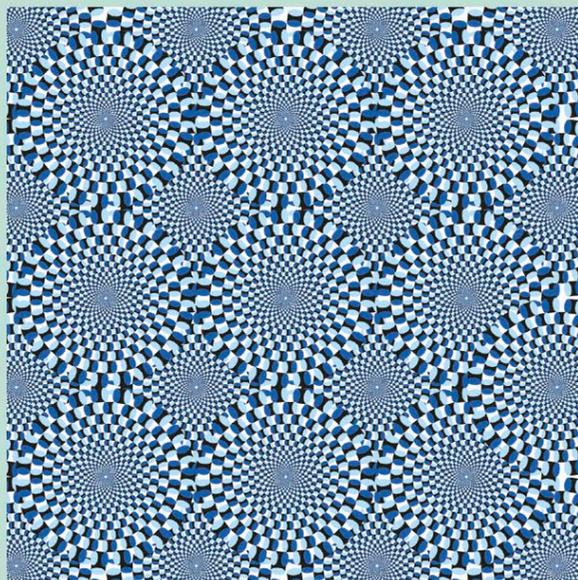
FAMOUS PERSON
When a face matches a memory of a famous person, such as Marilyn Monroe, the information is shunted to the frontal lobes for processing.

DAMAGE TO THE DORSAL PATHWAY

Damage to the dorsal visual pathway causes a number of disorders, all of which affect the ability to deal with objects in space. A person may, for example, be unable to see that two objects are in different places or to correctly see their spatial relationship, one to the other. They may find it impossible to reach out and grasp an object accurately or to know where it lies in relation to themselves. For example, a person may say something like, "I know there is a banana there but I don't know where it is." Patients may also suffer visual attention defects (see pp.182–83).

Frontal lobe

Some information from dorsal route arrives in frontal lobes, where it is consciously perceived

**STILL LIFE**

The ability to see movement is vital for survival. Many animals, such as frogs, can only see things in motion. The motion area of the human brain is tiny and more than 90 percent of neurons here are specialized to detect direction of movement. It is generally well protected from injury but, very rarely, a person may lose motion vision due to a stroke. The effect is profoundly disturbing, reducing the world to a series of snapshots. Day-to-day life becomes difficult—crossing the road, for example, is perilous as approaching traffic appears first to be distant and then suddenly close. Pouring a cup of tea is difficult because the column of liquid seems to be frozen and then overflowing.

ILLUSORY MOTION

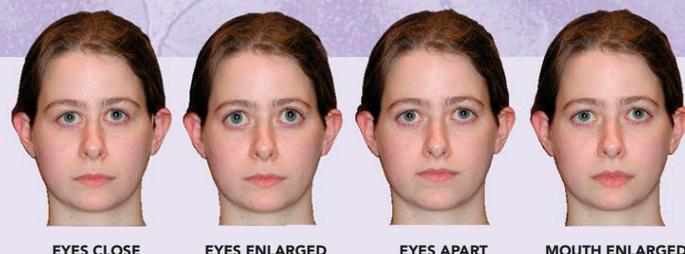
The brain frequently detects motion where there is actually none. Many different types of illusions can do this. Most of them depend on exciting motion-detecting neurons, causing them to fire and thus create the effect of movement.

Inferior temporal lobe

Fusiform gyrus involved in recognizing objects, especially faces

PROSOPAGNOSIA

If the face-recognition area is damaged, or fails for some reason to develop normally, people may be unable to recognize people they know—even their closest friends and members of their own family. Prosopagnosia is severely socially disabling. Affected people may get quite good at identifying people by features other than their face (by voice or clothing) but these techniques are slower and less reliable than normal face recognition. Face recognition relies on detailed information about distances between features. In the faces above, the shape of the features or the distance between them have been manipulated. People with prosopagnosia are unable to spot the differences.



EYES CLOSE

EYES ENLARGED

EYES APART

MOUTH ENLARGED

ALTERED IMAGES

These photographs have had features, such as mouth or eye size, altered or have been changed configurally—the eyes moved together or further apart.

**MONA LISA ILLUSION**

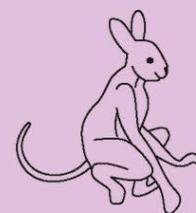
The face-recognition area only processes stimuli that have the pattern of facial features. So a picture of an upturned face is not processed here but is dealt with by an area that is not sensitive to facial expression. The upturned image of Mona Lisa seems at first to be normal. Turn it the right way up, though, and the face area alerts you to something very wrong!

DAMAGE TO THE VENTRAL PATHWAY

Damage to the ventral pathway results in one or another form of visual agnosia—the inability to recognize what one is seeing. Prosopagnosia, the inability to recognize faces (see panel, above), is one type of agnosia, but there are many others. Visual agnosia is generally divided into two categories: apperceptive and associative. The first type results from damage to the parts of the pathway in the occipital lobe and manifests itself as an inability to form a properly constructed perception. Hence a person with apperceptive agnosia cannot copy or draw an object, even though they may be able to see the parts of it quite clearly. Associative agnosia is an inability to identify objects. The person sees the object and may be able to mime an appropriate action in relation to it—for example, using a fork to raise food to the mouth—yet be unable to say what it is.



LETTER



FANTASY OBJECT

AGNOSIA TESTS

Tests for agnosia include recognizing objects from their silhouettes, telling fantasy objects from real ones, or identifying an incomplete letter.



SILHOUETTE

So strong is the attraction of faces that even the portraits within the picture get close and repeated study

Eye gaze and mouth are scrutinized for clues to the intentions and inner states of the characters in the picture



The viewer's gaze lingers here to scrutinize the interplay between the "main" characters

The eye passes straight across the floor, pausing briefly when the pathway is obstructed, but not stopping long enough to see it

Openings are scanned, perhaps for the possibility of others intruding on the scene and altering the human dynamics within it



Pointing to an object increases its significance and makes it worthy of a look

TUNING IN TO DETAIL

The white lines on this image track the viewer's eyes as they navigate around the scene. The circles represent where the gaze rests—the larger the circle, the longer the eye lingers.

VISUAL PERCEPTION

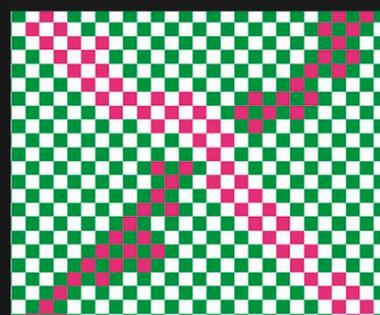
WE DO NOT SEE WHAT WE THINK WE SEE. WHEN WE LOOK AT A SCENE WE HAVE THE IMPRESSION OF SEEING ALL OF IT IN ONE GLANCE, BUT IN PRACTICE WE TYPICALLY PICK OUT JUST A FEW TINY DETAILS.

TOP-DOWN AND BOTTOM-UP PROCESSING

Visual perception is momentary, partial, and fragmentary. “Bottom-up” visual processing presets the brain with information about the whole field of vision, but “top-down” processes select which parts of the scene to make conscious. When we look at a picture, our eyes typically alight on a few thumbnail-size areas that we scan in sequence repeatedly. The rest of the image remains a blur unless we deliberately turn our attention to it. Eye-tracking studies (see left) show that the parts of a scene that we look at most closely are those that relate to other people. Although this visual selection is determined by “higher” brain functions—those involved in social concerns rather than, say, ducking a low branch—people are often unaware of what they are looking at. When asked, they may say they are looking at one thing when in fact their eyes have been resting on another.

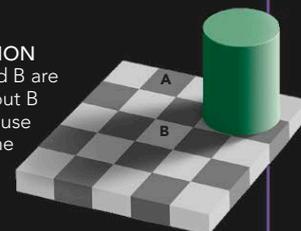
MAKING SENSE OF PICTURES

The brain works hard to make sense of visual information. Looking at a complex scene (see left) activates processes that distinguish target objects, such as people, from the background and then selects which bits of the target to focus on. These details are then scrutinized while the conscious brain pieces together the story. This interpretation begins unconsciously. Colors and shades are not recognized just by the type and amount of light reflected from them. The unconscious brain works out an object's most likely color or shade from its context.



CYLINDER ILLUSION

The squares A and B are identical shades but B looks lighter because we assume that the cylinder is casting a shadow over it.

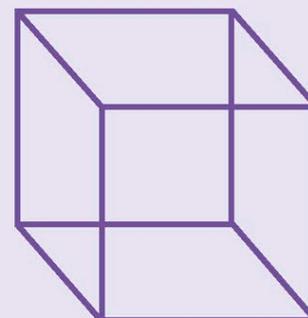


COLOR ILLUSION

The color you see depends on those around it. Pink next to white looks paler than pink next to green. This is due to “lateral inhibition,” which defines objects from their surroundings.

LAUGHTER PLAYS TRICKS ON THE EYES

Laughing literally changes the way you see the world. Normally, when you look at a Necker cube the image switches between two competing 3-D images, a situation known as binocular rivalry. This rivalry occurs because each eye sends a slightly different image to each side of the brain (see p.83), and the brain switches conscious awareness of one to the other. One theory on why switching stops during laughter is that amusement is a state in which information from both halves of the brain merges more than usual.



NECKER CUBE

SEEING

SEEING SEEMS TO BE INSTANTANEOUS AND EFFORTLESS, AND VISUAL IMAGES ALWAYS APPEAR FULLY FORMED. UNCONSCIOUSLY, HOWEVER, THE BRAIN IS CONSTANTLY UNDERTAKING A MAJOR FEAT OF CONSTRUCTION TO PRESENT US WITH OUR VIEW OF THE WORLD.

VISUAL PERCEPTION

One way of thinking about visual perception is to see it as the end product that emerges from a long and complicated assembly line. The construction process begins in earnest when information from the eyes—the raw material—reaches the primary visual cortex at the back of the brain. This is then sent along two main pathways (see pp.84–85), through a number of cortical and subcortical areas. Each of these responds by creating neural activity that generates various aspects of vision such as color, form, location, and movement. Eventually, the various elements are bound together and we become conscious of a meaningful sight.

2 Retinal cells
The light passes through the lens and then through two layers of retinal cells before hitting the light-sensitive rods and cones at the back.

1 Light enters the eye
Light waves enter the eye through the pupil, a hole in the center of the iris. The pupil expands to let in more light in shady conditions, and contracts when the light is bright, so a relatively constant amount of light is allowed in.

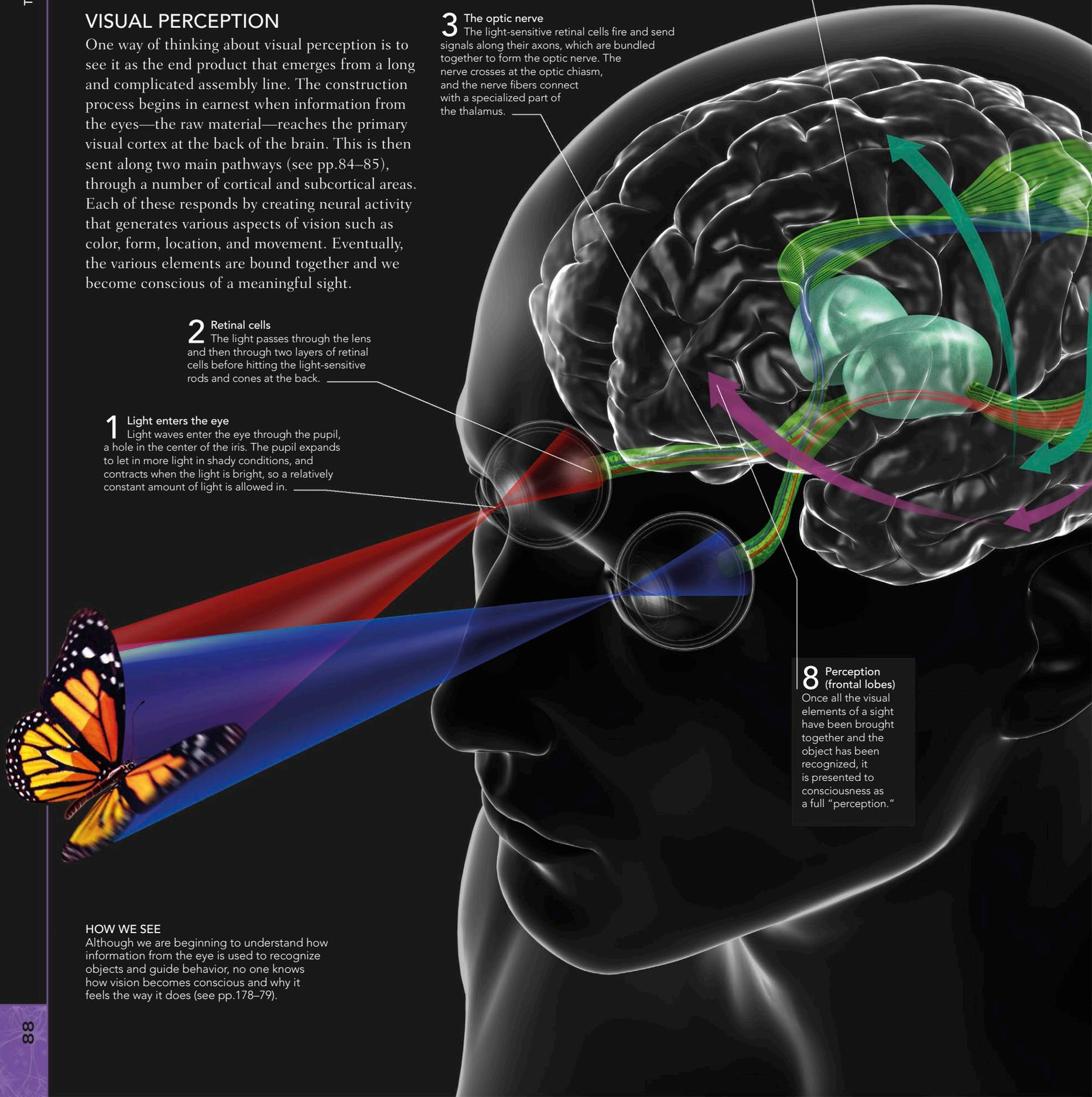
3 The optic nerve
The light-sensitive retinal cells fire and send signals along their axons, which are bundled together to form the optic nerve. The nerve crosses at the optic chiasm, and the nerve fibers connect with a specialized part of the thalamus.

4 The optic radiation
The signals are then sent from the thalamus on to the visual cortex via a thick band of tissue known as the optic radiation.

8 Perception (frontal lobes)
Once all the visual elements of a sight have been brought together and the object has been recognized, it is presented to consciousness as a full "perception."

HOW WE SEE

Although we are beginning to understand how information from the eye is used to recognize objects and guide behavior, no one knows how vision becomes conscious and why it feels the way it does (see pp.178–79).



5 THE DORSAL ROUTE

Information from the eyes is registered by the primary visual cortex and then sent forward along two pathways for further processing. The dorsal route takes it up through areas that are concerned with charting the location of the target object in relation to the viewer. Along this route, neuronal activity encodes the object's position, movement, and some aspects of its size and shape. The dorsal route ends in the parietal areas, which construct action plans relative to the viewed object. This process occurs unconsciously.

Motion

Movement is processed along the dorsal pathway. It is an essential component of any "action plan" (see p.121), and the brain not only notes current motion, but also predicts where an object will be in a split second. This ensures that any action plan is well timed.

**Depth**

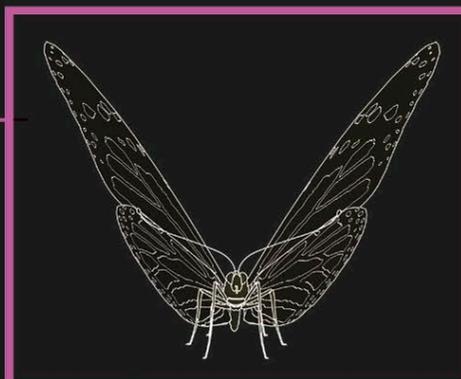
In order to calculate the depth of an object, the brain combines visual signals from both eyes—each of which has a slightly different view (see p.83)—along with information about how the shape of the image alters as the eyes move.



DORSAL

6 THE VENTRAL ROUTE

The ventral route carries information from the primary visual cortex down through the temporal lobes, where the neural activity identifies the sights and "clothes" them with meaning. A face, for example, is distinguished and recognized here (see p.84), and information about it such as the name of the person is recalled from memory (see p.163). Information traveling along the ventral path is brought together with that from the dorsal path in the frontal lobes—resulting in conscious perception rather than action.

**Form**

The brain has many different ways of "seeing" form. These include registering the orientation of light waves hitting an object and processing information about the way the waves reflect from its surfaces or outlines.

**Color**

Color discrimination begins in the retinal cells, some of which are tuned to fire in response to specific light wavelengths. Color processing continues in the brain, especially in an area known as V4 (see pp.82–83), which contains the majority of color-sensing neurons.

VENTRAL

7 Recognition path

In order to see something properly, a person needs to have some idea of what is being seen. If an image is not recognized, it is less likely to be consciously registered and may be overlooked altogether. Recognition is not purely visual, but involves clothing the perception with knowledge—such as who or what it is, what its intention is (if it is sentient), why it is there, and what it is called. Some of these elements may be missing—you may see someone you know but fail to recall his or her name, for example. By contrast, the purely visual elements of a perception are nearly always intact.

SEEING WITH SOUND?

A device that turns visual information into sound has been reported to create visual experience in at least one user, who is otherwise blind. The device involves mounting a small camera on a person's head, which captures a moment-by-moment view of what would normally be the person's visual field. This information is then turned into a "soundscape" that is played into the user's ears. As the person learns to recognize the physical qualities matching the sounds—for example, that a single high-pitched tone signifies a vertical surface—they seem to cease to hear it as a noise and instead experience it much like normal vision. One woman claims that her experience of "hearing" the environment is sometimes indistinguishable from seeing it.

**SOUNDSCAPE**

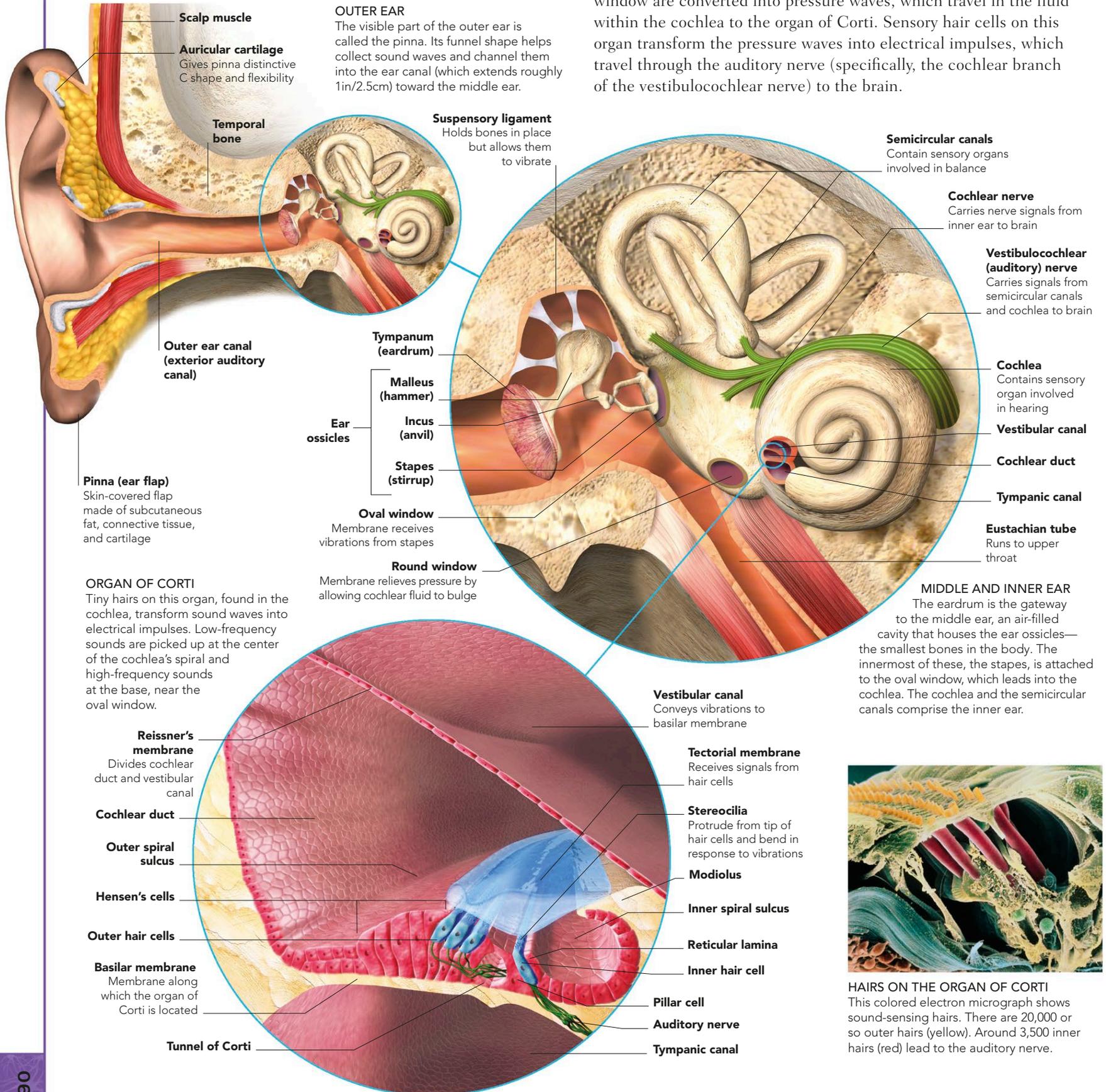
This image is a computer reconstruction of one second of sound, as "seen" by the system that builds soundscapes from camera images.

THE EAR

THE EAR PICKS UP SOUND WAVES IN THE ENVIRONMENT AND TRANSLATES THIS INFORMATION INTO NERVE IMPULSES, WHICH ARE SENT TO THE BRAIN FOR PROCESSING. THE EAR ALSO SENSES THE MOTION AND POSITION OF THE BODY, WHICH ALLOWS THE BRAIN TO REGULATE BALANCE.

THE ANATOMY OF HEARING

The ear is divided into three sections: the outer ear, middle ear, and inner ear. The outer ear funnels sound waves along the ear canal to the eardrum (tympanic membrane)—the start of the middle ear. The sound waves cause the eardrum to vibrate, which in turn causes a chain of bones, known as the ear ossicles, to vibrate. One of these, the stapes, is attached to a membrane known as the oval window—the start of the inner ear. Beyond this is the maze of fluid-filled chambers of the spiral-shaped cochlea. The vibrations of the stapes on the oval window are converted into pressure waves, which travel in the fluid within the cochlea to the organ of Corti. Sensory hair cells on this organ transform the pressure waves into electrical impulses, which travel through the auditory nerve (specifically, the cochlear branch of the vestibulocochlear nerve) to the brain.



OUTER EAR
The visible part of the outer ear is called the pinna. Its funnel shape helps collect sound waves and channel them into the ear canal (which extends roughly 1in/2.5cm) toward the middle ear.

Suspensory ligament
Holds bones in place but allows them to vibrate

Semicircular canals
Contain sensory organs involved in balance

Cochlear nerve
Carries nerve signals from inner ear to brain

Vestibulocochlear (auditory) nerve
Carries signals from semicircular canals and cochlea to brain

Cochlea
Contains sensory organ involved in hearing

Vestibular canal

Cochlear duct

Tympanic canal

Eustachian tube
Runs to upper throat

MIDDLE AND INNER EAR
The eardrum is the gateway to the middle ear, an air-filled cavity that houses the ear ossicles—the smallest bones in the body. The innermost of these, the stapes, is attached to the oval window, which leads into the cochlea. The cochlea and the semicircular canals comprise the inner ear.

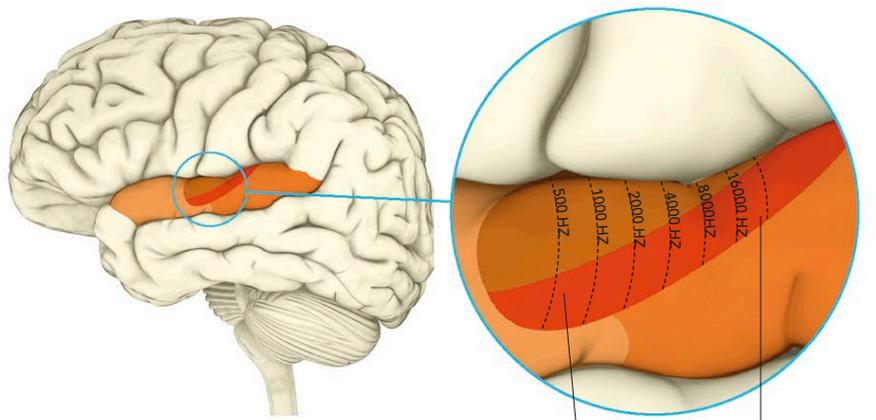
ORGAN OF CORTI
Tiny hairs on this organ, found in the cochlea, transform sound waves into electrical impulses. Low-frequency sounds are picked up at the center of the cochlea's spiral and high-frequency sounds at the base, near the oval window.



HAIRS ON THE ORGAN OF CORTI
This colored electron micrograph shows sound-sensing hairs. There are 20,000 or so outer hairs (yellow). Around 3,500 inner hairs (red) lead to the auditory nerve.

THE AUDITORY CORTEX

Sound information, in the form of electrical impulses, travels from the ear along the auditory nerve to the auditory cortex (situated in the temporal lobe, beneath the temples) for processing. In one of its three areas, the primary auditory cortex, different auditory neurons respond to specific sound frequencies. Also, some respond to the intensity of a sound rather than to its frequency, while others respond to more complex sounds, such as clicks, animal noises, and bursts of noise. It is thought that the secondary auditory cortex plays a part in processing harmony, rhythm, and melody, while the tertiary auditory cortex is involved in integrating the variety of sounds into a whole impression.



PERCEIVING SOUND FREQUENCIES
In the primary auditory cortex, neurons are sited according to the frequency each responds to, as are the sensory cells in the cochlea.

Corresponds to apex of cochlea
Corresponds to base of cochlea

AUDITORY RANGES	
SPECIES	FREQUENCY (HERTZ)
Elephant	16–12,000
Goldfish	20–3,000
Human	64–23,000
Dog	67–45,000
Porpoise	75–150,000
Bullfrog	100–3,000
Owl	200–12,000
Bat	2,000–110,000

AUDITORY RANGES

Many animals can hear sounds that humans cannot, both at higher and lower frequencies. Some animals pick up frequencies significantly higher than those humans can detect. For example, bats using echolocation can detect reflected sounds in the 14,000–100,000 Hertz range. The lower limit of the human auditory frequency range is fixed throughout life, but the upper limit begins to fall from adolescence. The maximum frequency heard by a normal middle-aged adult is between 14,000 and 16,000 Hertz.



HAIR CELLS AND FREQUENCY
This colored electron micrograph shows V-shaped sensory hair cells on the organ of Corti (see opposite page), each with multiple strands (yellow) or stereocilia. Cells are arranged within the cochlea according to the frequency of the sound each is able to detect.

THE COCHLEAR IMPLANT

Rather than restore hearing, this device helps the wearer have a perception of sound with no time lag, which can help with lip-reading. A microphone picks up sounds and passes them to a sound processor, which turns them into digital electrical signals. The transmitter conveys the signals, in the form of radio waves, to the implanted receiver, located beneath the skin. This receiver communicates via electrodes with the sensory hair cells in the cochlea, which pass the information on to the brain.

EXTERNAL APPARATUS
A transmitter, microphone, and sound processor convert environmental sounds into digital signals.

INTERNAL APPARATUS
Surgery is required to insert the receiver and electrodes that convey the sound information to the inner ear.

AUDITORY DISORDERS

Hearing loss is common but total deafness is rare and usually results from a congenital problem. Mild or severe hearing loss can result from ear disease, injury, or degeneration of the hearing system with age. Hearing loss is either conductive (a fault in the transferral of sound from the outer to inner ear) or sensorineural (sometimes known as nerve deafness, involving damage to the auditory nerves, or to the sensory parts of the inner ear). Common hearing disorders include otitis media and otosclerosis. Otitis media mainly affects young children and is an inflammation of the middle ear caused by a bacterial infection. Otosclerosis occurs when there is abnormal bone growth on the stapes bone of the middle ear, which stops it from vibrating and conducting sound waves on to the inner ear.



PERFORATED EARDRUM
The eardrum may become perforated due to infection, injury, or sudden exposure to an explosive noise that causes excessive vibration. Perforations can heal naturally.



NORMAL EARDRUM
The eardrum consists of a thin layer of fibrous tissue continuous with the skin of the outer ear and the mucous membrane of the middle ear.

MAKING SENSE OF SOUND

SOUND VIBRATIONS ARE TURNED INTO ELECTRICAL IMPULSES IN THE COCHLEA. FROM THERE, THEY TRAVEL TO THE AUDITORY CORTEX AND ITS ASSOCIATION AREAS VIA THE MEDULLA AND THE THALAMUS.

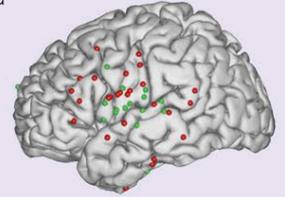
PERCEPTION OF SOUND

Sounds start as vibrations entering the ears. In the inner ear, receptor cells in the cochlea transform these vibrations into electrical signals, which pass along the cochlear nerve to the medulla in the brainstem, and then to the inferior colliculus. The cochlear nerve fibers divide so that most of the input from each ear can go to both hemispheres. At this stage, the source of the sound is determined by areas of the brainstem that compare input from both ears and analyze the delay (of about $\frac{1}{1,500}$ of a second) between the receipt of the signal by the ear nearest to the source and the ear farther away.

The signals reach the auditory cortex via the thalamus, where frequency, quality, intensity, and meaning are perceived. The left auditory cortex is more concerned with the meaning and identification of sound; the right, with quality.

THE COCKTAIL-PARTY EFFECT

The brain not only receives signals from the ears, it also sends signals to them, creating a circuit that modulates input. Background noise is dimmed, and the longer a person concentrates on a single strand of conversation, the greater the effect of filtering. This makes it easy to hear words you are interested in but may reduce the background so much that important messages fail to get through. If your brain registers an important sound, such as your name, it will instantly identify the source of that sound and upgrade it from heard to listened to. This is known as the cocktail-party effect.



HEAR OR LISTEN

When in a noisy environment, such as at a party, the brain can tune in to listen to a particular conversation while still hearing the noise of background speech. Green areas in the scan above register the sound of speech while red areas process speech to the level of understanding—it is “listened to” as well as heard.



Sound crosses to right hemisphere

Most signals from left cochlea travel to right side of cortex

Right auditory cortex

Corpus callosum

Part of thalamus that receives signal

Left auditory cortex

Sound crosses to left hemisphere

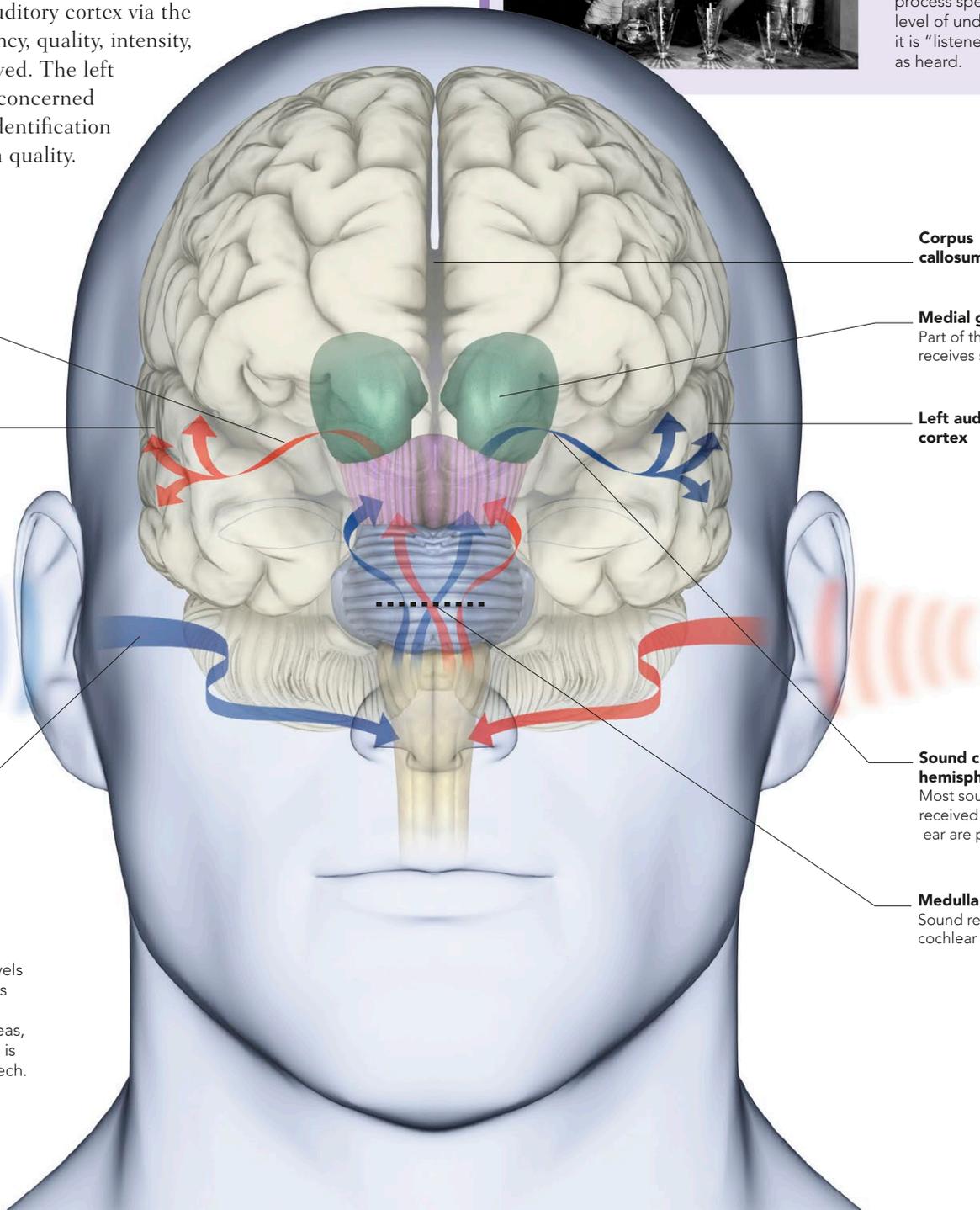
Most sound signals received from right ear are processed here

Medulla in brainstem
Sound received in cochlear nucleus

Sound travels through ear along cochlear nerve

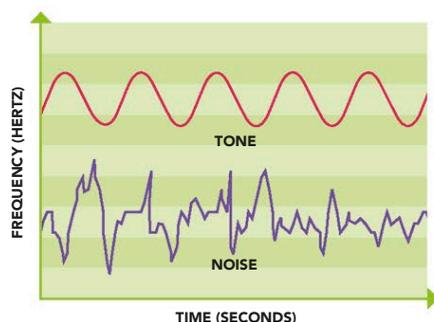
THE HEARING BRAIN

Sound enters the ears and travels via the brainstem and thalamus to the auditory cortex. Here, it is processed by association areas, such as Wernicke’s area, which is involved with interpreting speech.



NOISE OR MUSIC?

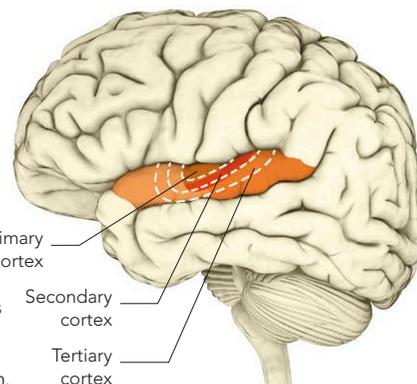
Sound consists of waves, or vibrations, whose characteristics are determined by the source of the sound. The main characteristics influencing our perception of sound are frequency (number of vibrations per second) and amplitude (the size of the waves' "peaks" and "troughs"). Frequency influences pitch, and amplitude governs loudness. Irregular sound-wave patterns tend to be experienced as



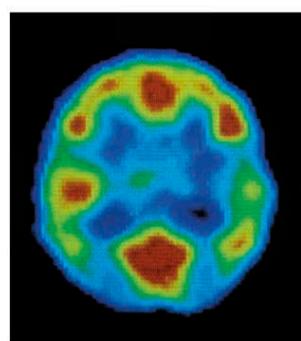
noise; in contrast, music produces regular patterns. Music is hard to define precisely, but the quality of musical notes depends upon

NOISE OR NOTE
Analysis of the wave forms of sounds reveals pure tones to be regular in frequency and amplitude, while noise is irregular.

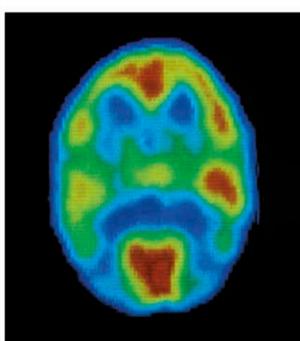
their sound source—a musical instrument—and how it is being played. Another important factor in music is timbre, or the "quality" of a sound. Timbre depends upon how many different frequencies of the note are heard at once; multiple frequencies or overtones (harmony) make a richer timbre. The auditory cortex responds to different qualities in music. The primary region responds to frequencies and the secondary area to harmony and rhythm, while the tertiary area adds higher levels of appreciation and integration.



AUDITORY CORTEX
The inner primary auditory cortex has areas associated with specific frequencies. The secondary and tertiary regions tune into more complex aspects of sound perception.



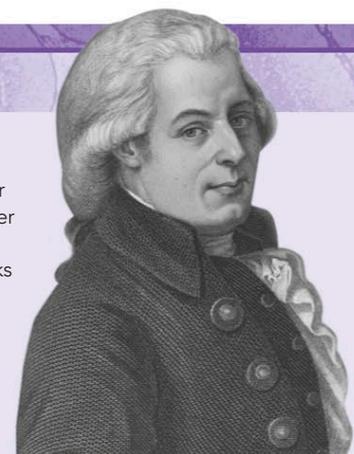
ACTIVITY DURING SPEECH
Speech tends to produce more intense activity in the left-hand side of the auditory cortex.



ACTIVITY DURING MUSIC
Music produces more pronounced activity on the right-hand side of the auditory cortex.

THE MOZART EFFECT

The French child-development expert Alfred Tomatis first described the "Mozart effect" in 1991. He claimed that listening to the music of the 18th-century classical composer Mozart could help the mental development of children under three. Researchers have also demonstrated that students listening to Mozart could improve their performance on tasks involving spatial reasoning and show a temporary increase in IQ. Recent research has given mixed results, but the idea has gained in popularity. The Mozart effect may, however, have more to do with changes in mood and arousal affecting mental performance than any direct impact on intelligence.



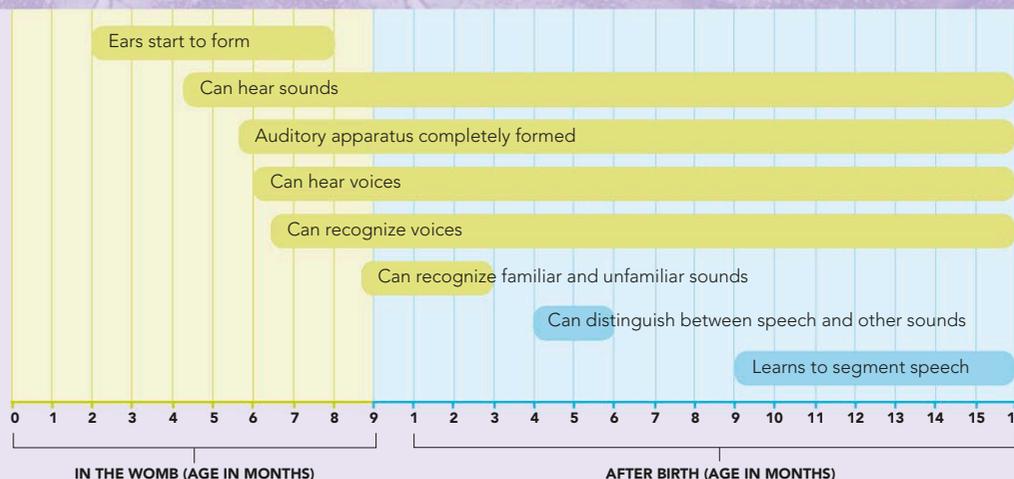
DEVELOPMENT OF HEARING

The development of hearing is a gradual process that begins in the womb and is complete by about the end of the first year of a baby's life. Research shows that the unborn child is capable of hearing by about the fourth month of gestation, but the auditory apparatus is not fully formed until about the sixth month. At birth, hearing is the most developed of the senses, so it is of prime importance to the baby in

exploring its world. Studies have shown how the baby learns to recognize sounds in its first few months, gradually becomes able to distinguish between speech and non-speech sounds, and then begins to understand words. Children also lose the ability to hear differences between sounds that are not important in their native language. Many Japanese children, for example, can no longer hear the difference between "l" and "r," which they could distinguish at an earlier age.

DEVELOPMENT BEFORE AND AFTER BIRTH

The human fetus has some basic hearing capacity from the age of about 18 weeks. This ability matures and develops over the next few weeks, with low-frequency sounds outside the mother's body being heard better than those of high frequency. From birth up to four months, the baby starts to respond to loud or sudden sounds, beginning to localize them by turning the head. From three to six months, the baby begins to recognize and also make sounds. Between six and 12 months, he or she begins to babble, recognizes basic words like "mommy," and starts to recognize voices. The baby begins to form words from the age of about one year. Each child reaches these milestones in hearing and speech development at different times, but very slow development may indicate some problem with the hearing apparatus.



HEARING

HEARING INVOLVES MECHANICAL VIBRATIONS FROM THE ENVIRONMENT—SPEECH, MUSIC, AND EVERYDAY NOISE—TRAVELING THROUGH THE OUTER, MIDDLE, AND INNER EAR. THE VIBRATIONS ARE TRANSFORMED TO ELECTRICAL SIGNALS, WHICH TRAVEL TO THE BRAIN TO BE INTERPRETED AS SOUND.

PATHWAY OF SOUND

The ear is a complex, exquisitely designed instrument for the capture of sound and its transport to the brain. Once mechanical vibrations from sound sources reach the inner ear, they are transformed into electrical impulses that shoot along the cochlear nerve to the brainstem. Here, they follow complex pathways up to the thalamus before arriving at the auditory cortex. Processing in the brain allows perception of the meaning, direction, and volume of a sound.

5 The cochlea
The cochlea contains three fluid-filled ducts. The vestibular canal transmits sound vibrations (blue) to the basilar membrane of the organ of Corti. Residual vibrations (red) travel back along the tympanic canal to the round window.

1 The outer ear
Sound waves are caught in the funnel-like curves of the outer ear that comprises the exterior "flap" of the pinna and the auditory canal.

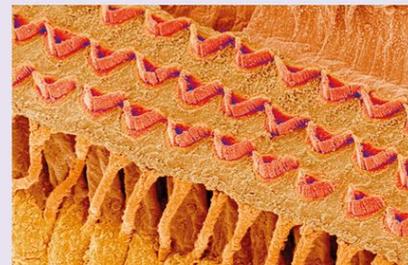
2 The auditory canal
The sound waves continue along the 1-in- (2.5cm)-long auditory canal, which extends from the concha (inner curve) of the outer ear to the eardrum and is lined with tiny hairs that protect it from the entry of foreign objects.

3 The eardrum
The eardrum, or tympanic membrane, vibrates as sound waves enter the auditory canal. It is a thin layer of fibrous tissue that forms a barrier between the outer ear and the middle ear.

4 Ossicles
Vibrations are passed on to a set of tiny bones called ossicles (see p.90), which act as a chain of levers. The stapes pushes and pulls on the oval window at the entrance to the cochlea, transmitting sound to the inner ear.

HEARING LIGHT

Hair cells turn sound vibrations into electrical signals that stimulate neurons in a healthy ear. Damage to the hair cells can lead to loss of hearing. However, research shows that infrared light is also capable of stimulating ear neurons. A team at Northwestern University in Chicago exposed inner-ear neurons in guinea pigs to infrared light. This resulted in electrical activity in the inferior colliculus suggesting that the light had caused soundlike input to be sent to the brain. This discovery could be turned into a new type of cochlear implant if fiber optics targeting light to the inner ear are developed.



HAIR CELLS
Each hair cell is topped by about 100 projections called cilia. It is the movement of these in response to vibrations that generates electrical signals.

11 The thalamus
Nerve impulses are received and processed by specialized neurons in the medial geniculate nucleus of the thalamus. These signals are then sent to the primary auditory cortex, which also feeds information back to the thalamus.

12 The primary auditory cortex
The characteristics of a sound input are finally interpreted, after intermediate processing at the primary auditory cortex, which works with other cortical areas on sound perception.

10 The inferior colliculus
All the ascending auditory pathways—some of which bypass the superior olives—converge upon the inferior colliculi at the top of the brainstem and their input is then passed on toward the thalamus.

9 The superior olive
Cells in the ventral cochlear nucleus send signals up to the superior olives on both sides of the brainstem. Here the brain interprets the direction of sounds (see p.92). The superior olive then sends signals on to the midbrain.

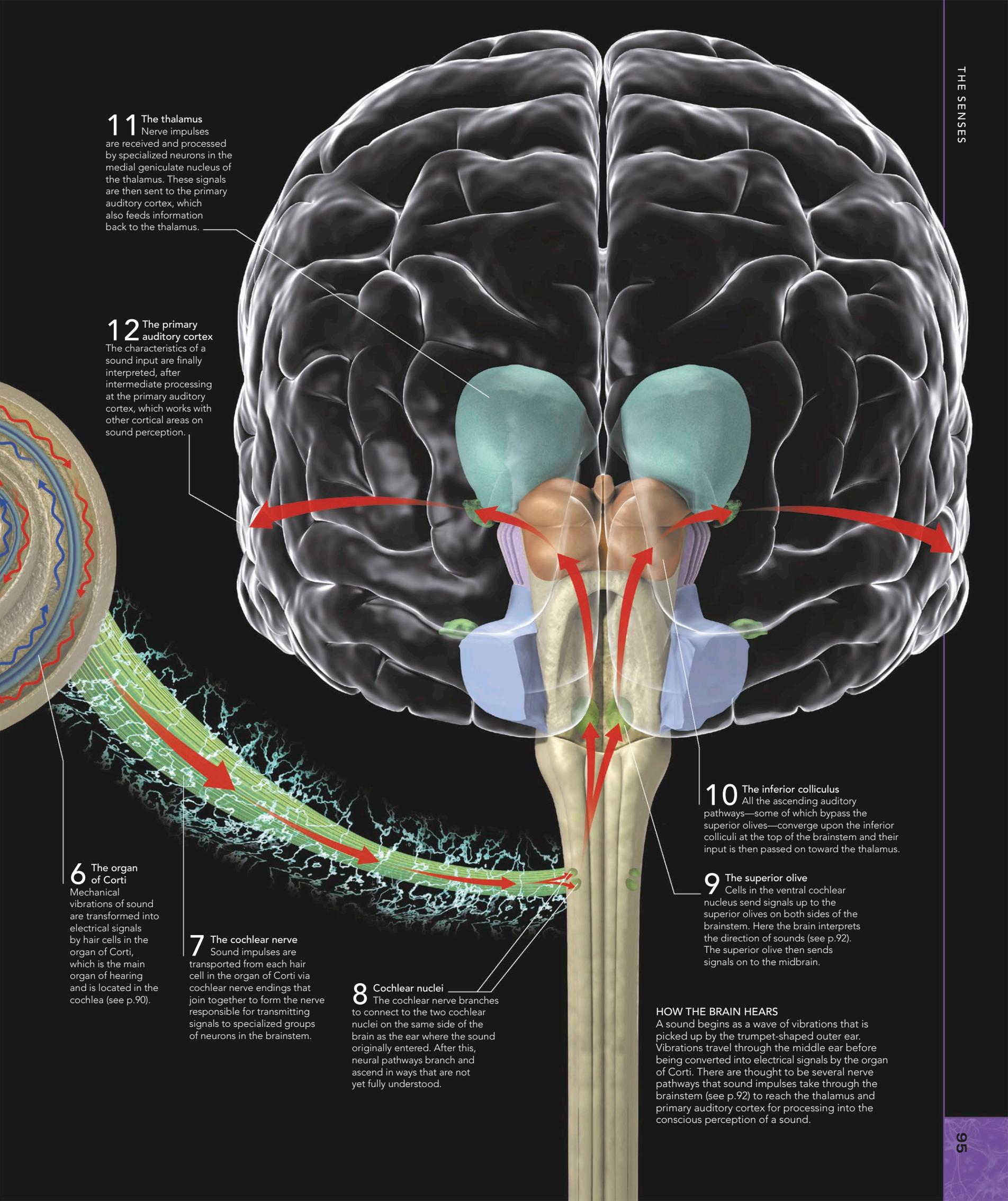
HOW THE BRAIN HEARS

A sound begins as a wave of vibrations that is picked up by the trumpet-shaped outer ear. Vibrations travel through the middle ear before being converted into electrical signals by the organ of Corti. There are thought to be several nerve pathways that sound impulses take through the brainstem (see p.92) to reach the thalamus and primary auditory cortex for processing into the conscious perception of a sound.

6 The organ of Corti
Mechanical vibrations of sound are transformed into electrical signals by hair cells in the organ of Corti, which is the main organ of hearing and is located in the cochlea (see p.90).

7 The cochlear nerve
Sound impulses are transported from each hair cell in the organ of Corti via cochlear nerve endings that join together to form the nerve responsible for transmitting signals to specialized groups of neurons in the brainstem.

8 Cochlear nuclei
The cochlear nerve branches to connect to the two cochlear nuclei on the same side of the brain as the ear where the sound originally entered. After this, neural pathways branch and ascend in ways that are not yet fully understood.

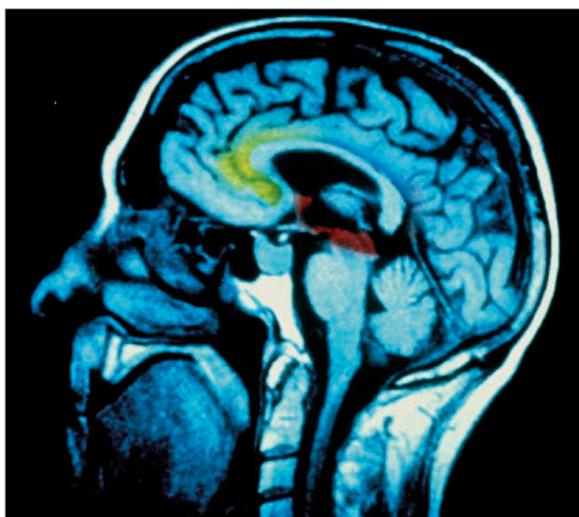


SMELL

ALTHOUGH VISION HAS BECOME THE DOMINANT SENSE IN HUMANS, THE SENSE OF SMELL (OLFACTION) REMAINS IMPORTANT TO SURVIVAL BECAUSE IT CAN WARN US OF HAZARDOUS SUBSTANCES IN OUR ENVIRONMENT. THE SENSES OF SMELL AND TASTE ARE CLOSELY LINKED.

DETECTING SMELL

Like the sense of taste, smell is a chemical sense. Specialized receptors in the nasal cavity detect incoming molecules, which enter the nose on air currents and bind to receptor cells. Sniffing sucks up more odor molecules into the nose, allowing you to “sample” a smell. It is a reflex action that occurs when a smell attracts your attention, and can help warn of danger, such as smoke from a fire or rotting food. Olfactory receptors located high up in the nasal cavity send electrical impulses to the olfactory bulb, in the limbic area of the brain, for processing.



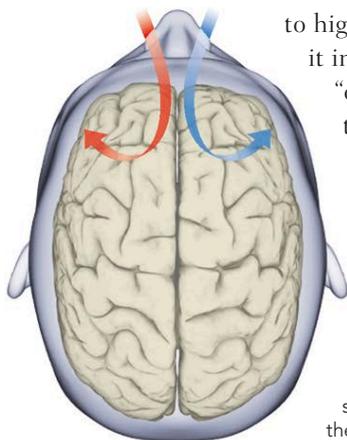
SMELL CENTERS IN THE BRAIN

The olfactory bulb is the smell gateway to the brain. Here, data about smells is processed in the forebrain (yellow), then sent to various areas of the brain, including the olfactory cortex adjacent to the hippocampus (red).

SMELL PATHWAYS

Odors are initially registered by receptor cells in the nasal cavity. These send electrical impulses along dedicated pathways to the olfactory bulb (each nostril connects to one olfactory bulb). The olfactory bulb is part of the brain’s limbic system, the seat of our emotions, desires, and instincts, which is why smells can trigger strong emotional reactions. Once processed by the olfactory bulb, data is transmitted via three olfactory pathways to higher centers in the brain that process it in different ways. This process is called

“orthonasal” smelling, in which smell data travels along pathways directly from the nose (see opposite). In “retronasal” smelling (see p.101), odors also have a flavor component that enters the olfactory pathways via the mouth.



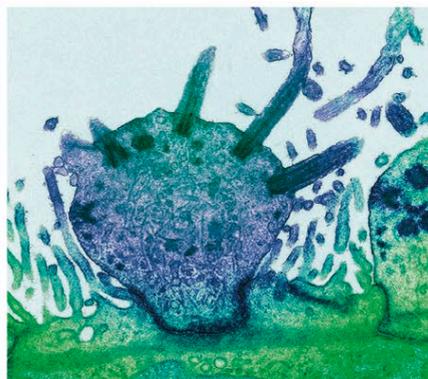
SAME-SIDE PROCESSING

Unlike data gathered by the other sense organs, odors are processed on the same, not opposite, side of the brain as the nostril the sensory data was sent from.

RECEPTOR ARRAYS

There are around 1,000 types of receptor cell in the nasal cavity, but we can distinguish around 20,000 different smells so, clearly, there is more to smell reception than “one receptor, one smell.” Research shows that each receptor has zones on it, each of which responds to a number of smell molecules. Also, multiple receptors respond to the same smell molecule—it may be that each receptor binds to a different part of it. A specific smell will activate a specific pattern or

“array” across the receptors, so that each smell has its own “signature.” When the receptors forming a specific pattern are activated, this signature is sent to the brain for processing.



OLFACTORY RECEPTOR CELL

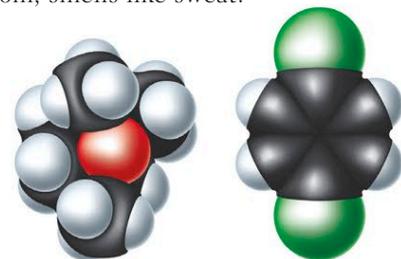
This colored electron micrograph shows tiny cilia projecting from a receptor cell. Odor molecules bind to the cilia and activate the receptor.

THE CHEMISTRY OF SMELL

There is still much to be learned about the relationship between chemical structure and smell. Scientists have identified eight primary odors (rather like the three primary colors): camphorous, fishy, malty, minty, musky, spermatic, sweaty, and urinous. Smells are often produced by a combination of many different smell molecules, often from different categories. Comparisons of the structures of smell molecules within each category have shown some similarities—for example, minty smelling compounds often share a similar molecular structure. However, tiny differences in molecular structure can produce very different smells. Octanol, a fatty alcohol, smells like oranges, while octanoic acid, a saturated fatty acid that differs from octanol by only one oxygen atom, smells like sweat.

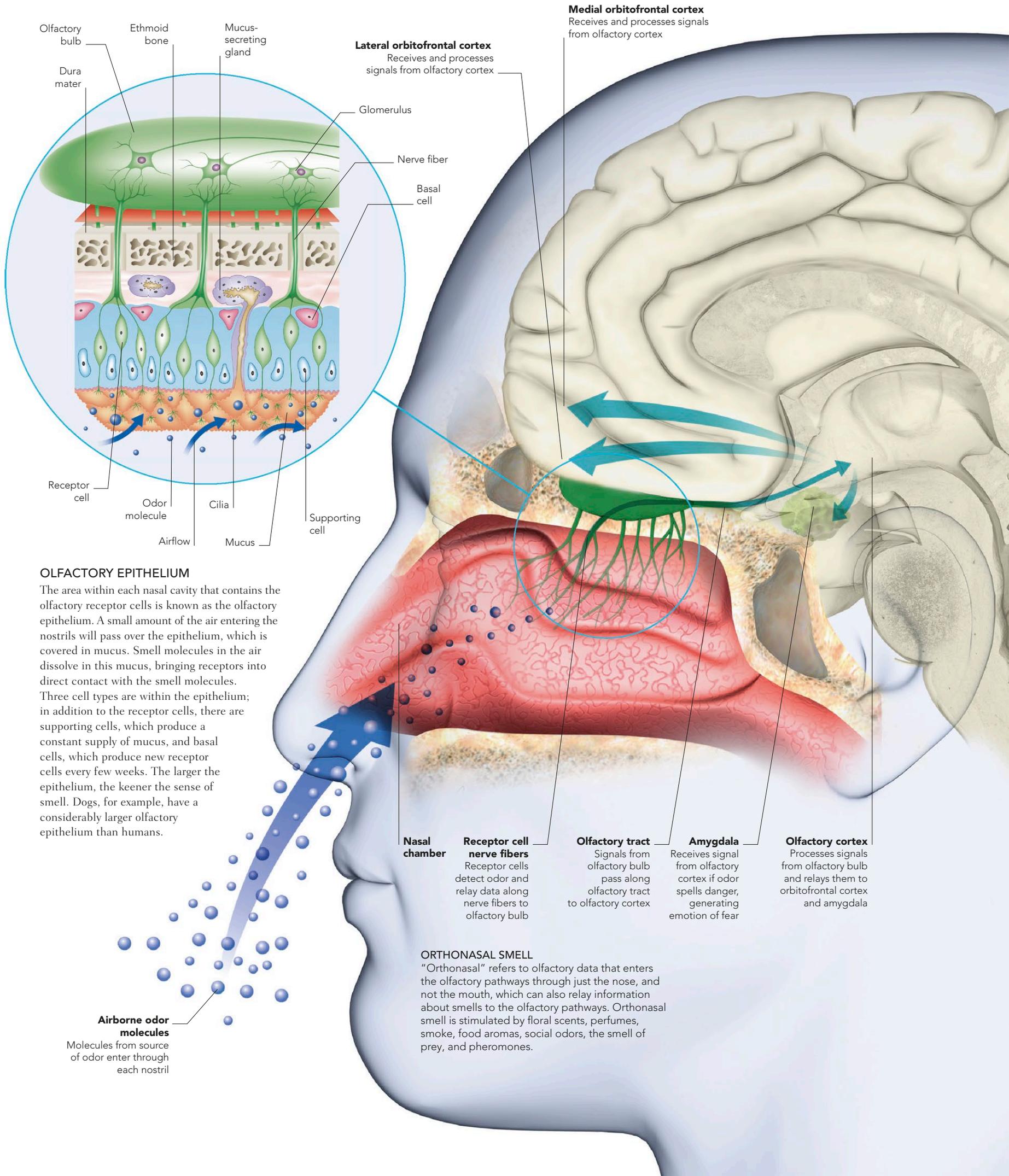
SMELL AND MOLECULAR STRUCTURE

These two molecules differ significantly in their chemical structure, yet both of them conjure the same characteristic “mothball” smell of camphor. One theory is that it is not the shape of molecules that causes them to smell, but the frequency at which their atoms vibrate.



PRIMARY SMELLS

Scientists investigating the perception of smell have attempted to identify primary odors, which can be combined with one another to produce the much larger range of smells that we experience. To date, eight primary odors have been identified, including the distinctive smell of fish.



OLFACTORY EPITHELIUM

The area within each nasal cavity that contains the olfactory receptor cells is known as the olfactory epithelium. A small amount of the air entering the nostrils will pass over the epithelium, which is covered in mucus. Small molecules in the air dissolve in this mucus, bringing receptors into direct contact with the smell molecules. Three cell types are within the epithelium; in addition to the receptor cells, there are supporting cells, which produce a constant supply of mucus, and basal cells, which produce new receptor cells every few weeks. The larger the epithelium, the keener the sense of smell. Dogs, for example, have a considerably larger olfactory epithelium than humans.

Nasal chamber

Receptor cell nerve fibers
Receptor cells detect odor and relay data along nerve fibers to olfactory bulb

Olfactory tract
Signals from olfactory bulb pass along olfactory tract to olfactory cortex

Amygdala
Receives signal from olfactory cortex if odor spells danger, generating emotion of fear

Olfactory cortex
Processes signals from olfactory bulb and relays them to orbitofrontal cortex and amygdala

ORTHONASAL SMELL

"Orthonasal" refers to olfactory data that enters the olfactory pathways through just the nose, and not the mouth, which can also relay information about smells to the olfactory pathways. Orthonasal smell is stimulated by floral scents, perfumes, smoke, food aromas, social odors, the smell of prey, and pheromones.

PERCEIVING SMELL

SMELL IS MORE LIKELY TO EVOKE EMOTION AND MEMORY THAN THE OTHER SENSES. THE FACT THAT OLFACTORY AREAS OF THE BRAIN EVOLVED EARLY ON AND ARE WIRED INTO THE PRIMITIVE BRAIN SUGGESTS THAT SMELL IS VITAL FOR OUR SURVIVAL, AS WELL AS THE SURVIVAL OF OTHER ANIMALS.

THE EVOLUTION OF SMELL

The smell brain, centered around the olfactory bulb in the limbic system, is of ancient origin, having evolved about 50 million years ago in fish. The sense of smell was overtaken in importance by the sense of vision when humans began to walk on two legs, although it is still dominant for many animals. But smell

is an important aspect of survival for humans, shown in the fact that we take prompt action if we smell gas or smoke, for example.

It also plays an important role in sexual selection, emotional responses, and forming preferences for food and drink. All of these factors were probably of key importance in the lives of our ancestors.



DISGUST
When a bad odor is detected, such as that of rotting meat, it is natural to both feel and express disgust. Avoidance of the source of the odor follows, and it is almost impossible to eat food that smells bad.

OLFACTION IN ANIMALS

Although humans can smell some odors at a concentration as low as one part per trillion, our sense of smell is weak compared to that of other animals. The size of the surface area of the olfactory epithelium (see p.97) and the density of smell receptor cells indicate how sensitive an animal's sense of smell is. Dogs, for example, can identify a particular human from just a few odor molecules. Northern dogs, such as huskies and jackals, are renowned for their sense of smell. Hunting dogs and grayhounds have a weaker sense of smell—in the chase, they don't have time to distinguish prey from background smells.

SNIFFER DOG

A breed combining the behavioral characteristics of a domestic dog and a jackal's sense of smell makes an ideal sniffer dog for security work.



SMELL ACROSS SPECIES

SPECIES	NUMBER OF OLFACTORY RECEPTOR CELLS	AREA OF OLFACTORY EPITHELIUM
Human	12 million	1 1/2 square in (10 square cm)
Cat	70 million	2 1/4 square in (21 square cm)
Rabbit	100 million	Data not available
Dog	1 billion	26 1/2 square in (170 square cm)
Bloodhound	4 billion	59 square in (381 square cm)

SMELL PREFERENCES

Whether we find a smell nice, nasty, or neutral is very subjective and depends upon familiarity, intensity, and perception as pleasant or unpleasant. It is not clear if preferences are innate or learned, but much experimental evidence supports the latter possibility.

Associative learning links pleasant smells to pleasant experiences, and vice versa. For example, people who fear the dentist do not like the clovelike smell of eugenol, which is used in dental cement; those without a fear of the dentist react positively or neutrally to this odor.



SUBJECTIVE RESPONSES

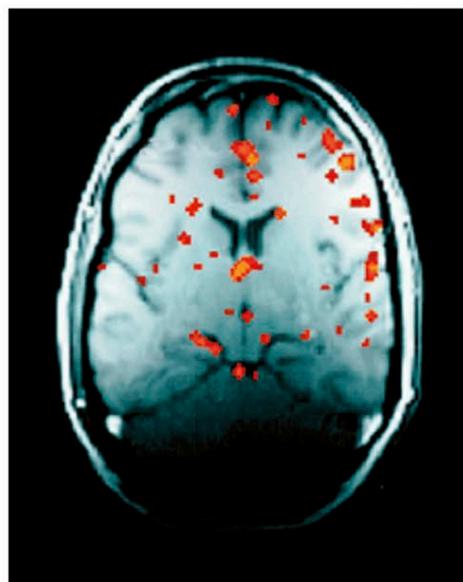
The distinctive smell of the durian fruit is perceived by some as revolting but others find it extremely tempting.

THE SIX WORST SMELLS IN THE WORLD

SMELL	DESCRIPTION
Decaying flesh	Repulsive to most people; may evoke thoughts of death
Skunk odor	Horrible to most, but a few people find it "interesting"
Vomit	Often associated with illness, which may heighten disgust
Feces or urine	Caused by gas released as bacteria break down food residue
Decaying food	Triggers an "adaptive" response to food that could cause illness
Isonitriles	Chemicals in nonlethal weapons described as "world's worst smell"

STEREOSCOPIC AND BLIND SMELL

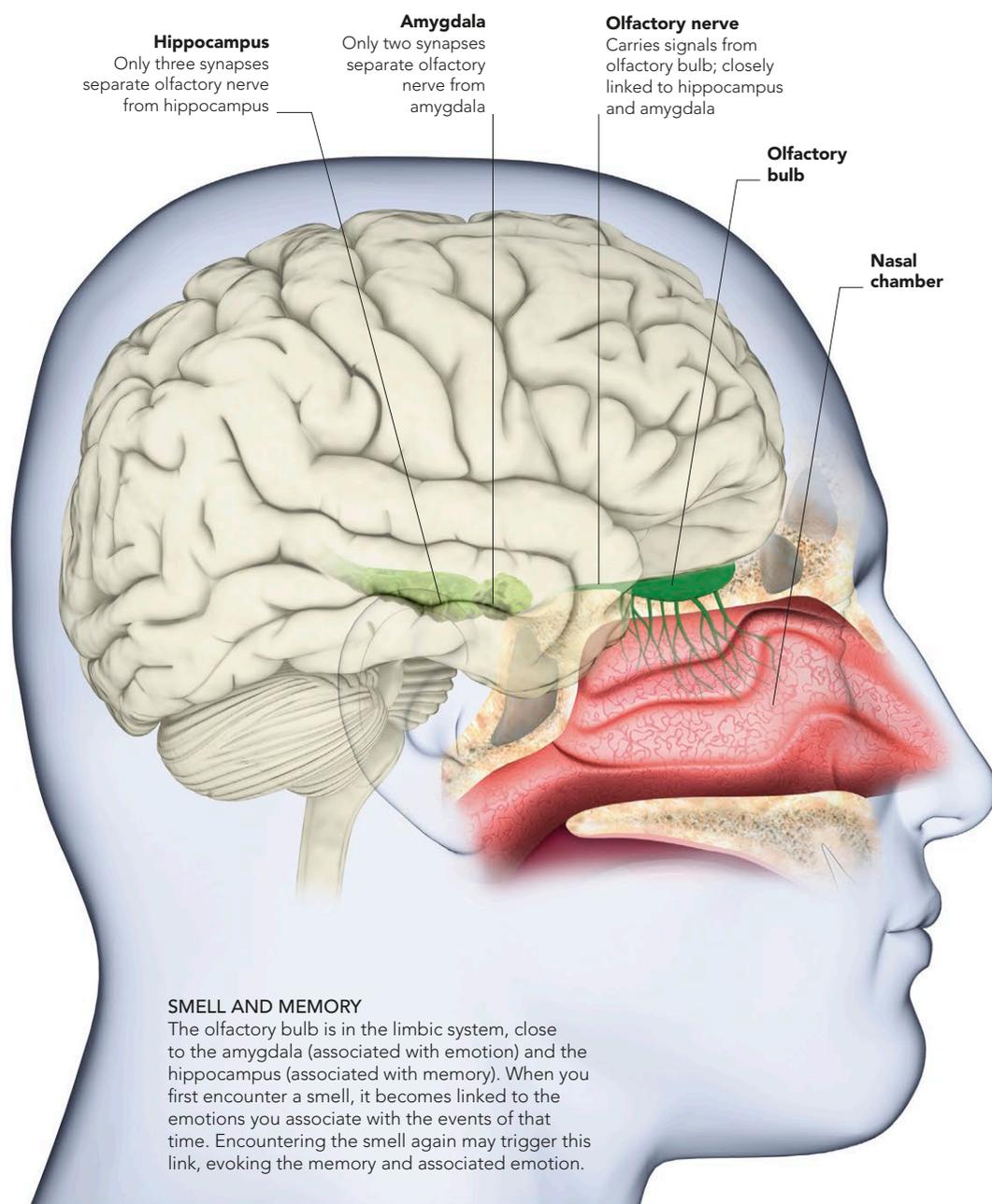
It is generally believed that the human sense of smell has atrophied in relation to our other senses, but recent research shows that humans can still effectively track a scent. Using both nostrils to sample a smell, the human brain uses both sets of data to accurately pinpoint the location of the source of the odor. Therefore, as with vision and hearing, smell can be "stereoscopic," relying on both nostrils for a full understanding



of a scent. "Blind" smell refers to the ability of the brain to detect a smell without being consciously aware of it, which has been demonstrated in experiments using fMRI scans showing how olfactory areas are activated without the participant's knowledge.

BLIND SMELL ACTIVATION

This fMRI scan shows widespread activity throughout the brain in areas including the thalamus (just above center), on exposure to an odor at concentrations that cannot be detected consciously.

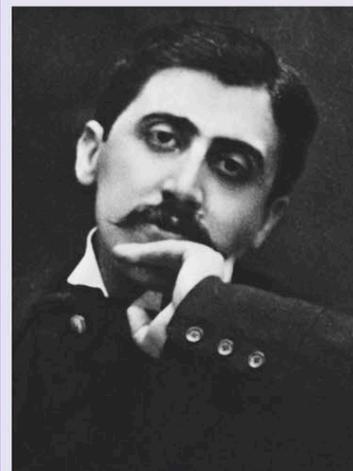


SMELL AND MEMORY

An event is associated with input from all the senses, co-ordinated by the hippocampus. Reexperiencing any of the sight, smell, or sound inputs may trigger a memory of the event, but smell seems most strongly associated with memory. This may be because olfactory regions are linked to all emotional areas in the limbic system. Research shows that a memory of a visual image is likely to fade within days, but the memory of a smell may persist for up to a year or even decades. The hippocampus may not even be crucial for the link, because people who sustain damage to this region can still recall scents from their childhood, even though suffering from general memory loss.

THE MADELEINE EFFECT

The madeleine effect is named after an episode in Marcel Proust's epic *Remembrance of Things Past*. As a mature adult, the novel's hero eats a madeleine soaked in lime-blossom tea and is mentally transported to his childhood and the house of his aunt, who used to serve madeleines before Sunday mass. Long before the effect



was investigated scientifically, Proust recognized that taste and olfactory memories can take us further back than visual or auditory cues.

PROUST
French novelist Marcel Proust (1871–1922) wrote “the smell and taste of things remain poised a long time, ready to remind us....”



MALE BODY SMELL

Male sweat contains androstenone, a musky compound. When sprayed on a waiting-room chair, most women choose that one. Androstadienone, another compound, affects men, making them more helpful. It is likely to stem from the need for men to hunt cooperatively.

SMELL AND COMMUNICATION

Animals emit compounds called pheromones that are used as communication signals and detected by an accessory olfactory system in the brain. Humans recognize each other in a similar way—for example, infants prefer the smell of their mother's breast to that of other women. Research into the existence of pheromones in humans has found that women's menstrual cycles can synchronize when one woman is exposed to odorless compounds (supposing that these are pheromones) emitted from the underarms of another woman. In animals, the accessory olfactory system is linked to the vomeronasal region (VMO), an area in the nasal cavity that responds to pheromones. The VMO's existence in humans remains debatable.



USING SMELL COMMERCIALY

Some estate agents claim that the smells of baking bread, cinnamon, and coffee can help sell a house by evoking a good feeling in potential purchasers. Equally, they advise banishing pets, so that animal smells do not put off buyers.

TASTE

LIKE SMELL, TASTE HAS A SURVIVAL VALUE—POISONOUS SUBSTANCES TEND TO TASTE BAD (USUALLY BITTER) WHILE THOSE THAT ARE NOURISHING TASTE PLEASURABLE (USUALLY SWEET OR SAVORY). TOGETHER, TASTE AND SMELL ALLOW ANIMALS TO EVALUATE AND RECOGNIZE WHAT THEY EAT AND DRINK.

THE EVOLUTION OF TASTE

The sense of taste enables animals, including humans, to make the most of the variety of foods available to them. Many plants that look tempting are toxic, so genes that enable us to detect (and therefore avoid) these toxins have an obvious survival value. One such gene that has been identified affords taste sensitivity to phenylthiocarbamide (PTC), an organic compound that resembles many toxic compounds found in plants.



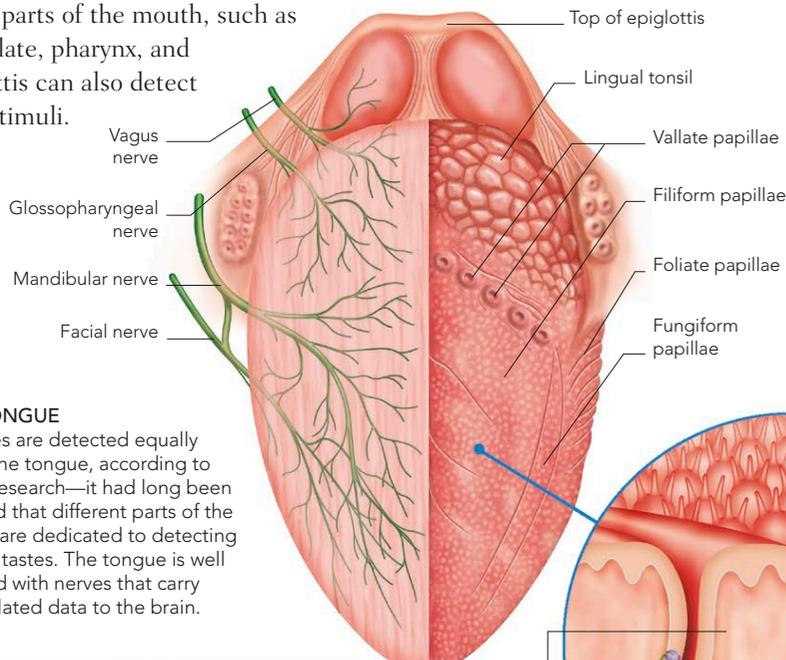
EVOLVED TO REACT TO TASTES

Herbivores, such as deer, with fewer bitter-taste genes than omnivores, are less selective and therefore benefit from an increased food supply. They can tolerate more toxins because they have larger livers than omnivores, such as chimpanzees.

THE TONGUE

The tongue is the main sensory organ for taste detection. It is the body's most flexible muscular organ, as revealed by its work in both nutrition and communication. It has three interior muscles and three pairs of muscles connecting it to the mouth and throat. Its surface is dotted with tiny, pimplelike structures called papillae.

Other parts of the mouth, such as the palate, pharynx, and epiglottis can also detect taste stimuli.



THE TONGUE

All tastes are detected equally across the tongue, according to recent research—it had long been believed that different parts of the tongue are dedicated to detecting specific tastes. The tongue is well supplied with nerves that carry taste-related data to the brain.

SUPERTASTERS

Around a quarter of the population are “supertasters,” which means they have an overall higher level of tasting ability. They are very sensitive to a chemical called propylthiouracil (PROP), finding it incredibly bitter. Half the remaining population find PROP moderately bitter, and the final quarter cannot taste it at all. Supertasters find bitter compounds such as coffee too strong. They seem to have more fungiform papillae on the tongue, which may explain the increased sensitivity.



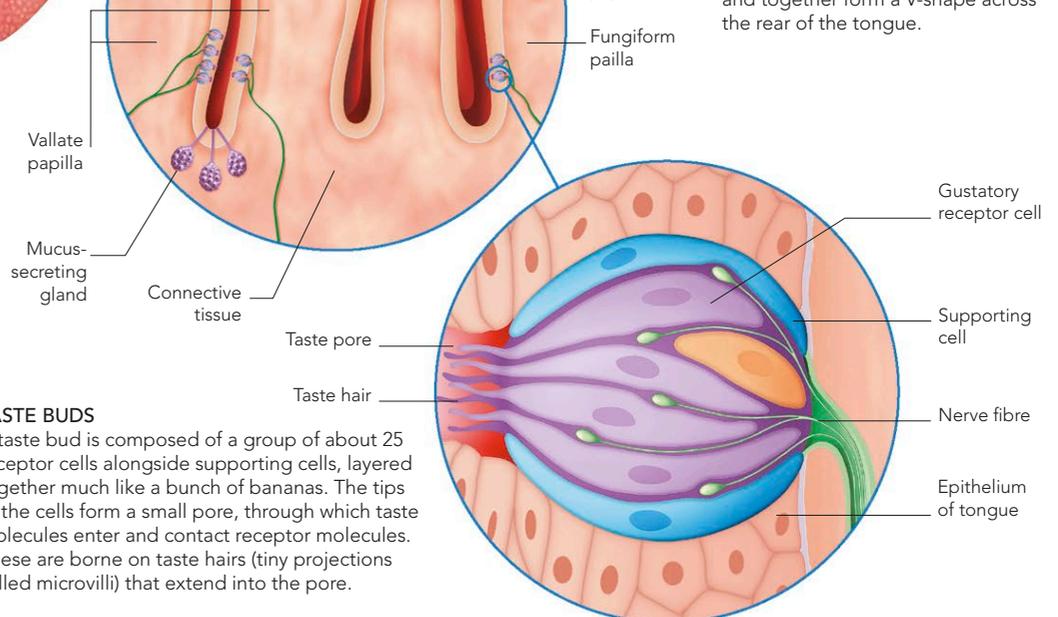
PAPILLAE

Papillae contain taste buds and are distributed across the tongue. Four types of papillae have been distinguished—vallate, filiform, foliate, and fungiform. Each type bears a different amount of taste buds. Fungiform and filiform are the smallest papillae, and vallate are the largest, and together form a V-shape across the rear of the tongue.

THE FIVE BASIC FLAVORS

Along with the basic tastes, people can detect other substances, such as fatty acids, through receptors in the upper airways. This suggests that taste is a part of smell, just as smell is a part of taste.

NAME	DESCRIPTION
Sweet	Often linked to energy-rich, high-calorie foods.
Sour	May be a danger sign, signaling unripe or “off” foods.
Salty	Most chemical salts, including sodium chloride, taste salty.
Bitter	May be linked to natural toxins, and is best avoided.
Umami	Savory (“umami” means “delicious” in Japanese).

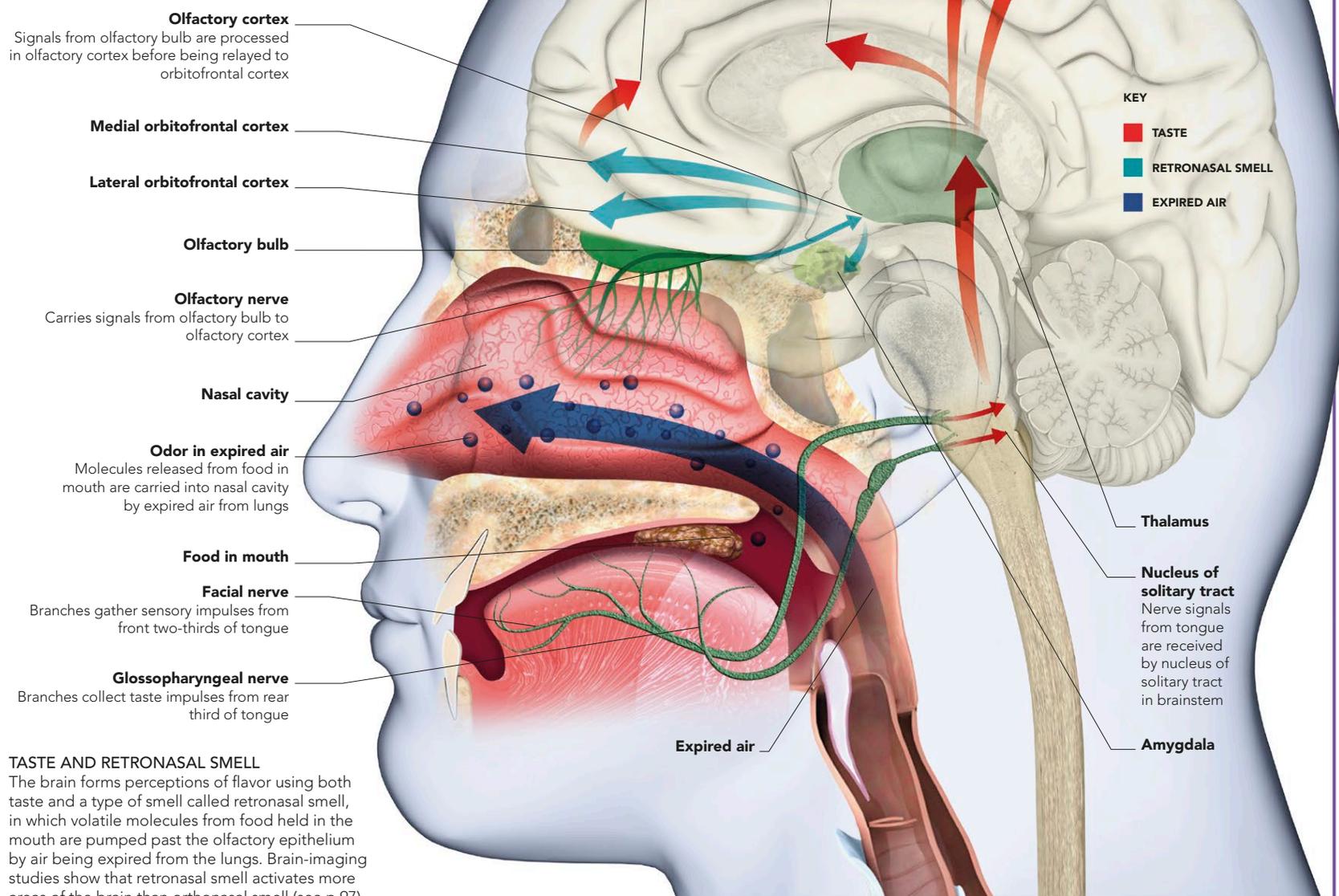


TASTE BUDS

A taste bud is composed of a group of about 25 receptor cells alongside supporting cells, layered together much like a bunch of bananas. The tips of the cells form a small pore, through which taste molecules enter and contact receptor molecules. These are borne on taste hairs (tiny projections called microvilli) that extend into the pore.

TASTE AND SMELL BRAIN AREAS

Taste and smell are both chemical senses—receptors in the nose and mouth bind to incoming molecules, generating electrical signals to send to the brain. Both sets of signals pass along the cranial nerves. Smell-related (olfactory) signals travel from the nose to the olfactory bulb, then along the olfactory nerve to the olfactory cortex in the temporal lobe for processing (see also pp.96–97). The pathway of taste-related (gustatory) data travels from the mouth along branches of the trigeminal and glossopharyngeal nerves to the medulla, continues to the thalamus, then to primary gustatory areas of the cerebral cortex.



TASTE AND RETRONASAL SMELL

The brain forms perceptions of flavor using both taste and a type of smell called retronasal smell, in which volatile molecules from food held in the mouth are pumped past the olfactory epithelium by air being expired from the lungs. Brain-imaging studies show that retronasal smell activates more areas of the brain than orthonasal smell (see p.97).

TASTE ASSOCIATIONS

When a food makes you ill (spoiled seafood, for example), the association can linger for a long time, making even the thought of that food repulsive. The phenomenon, known as flavor-aversion learning, has been demonstrated by researchers at Harvard Medical School who fed rats a sweet liquid with a substance that made them briefly ill. Thereafter, the rats avoided the liquid despite its tempting sweetness. When a food is paired with nausea, flavor-aversion learning has a survival value in teaching animals to avoid attractive-looking foods that may be toxic. It is a robust form of learning—occurring after one episode only, but lasting for many years.



TASTE AVERSION

As an alternative to killing coyotes that prey on domestic sheep, some farmers in the western US place lamb bait laced with an illness-inducing drug around their ranches. The coyotes learn to avoid lamb meat and therefore stop approaching sheep.

TOUCH

THERE ARE MANY KINDS OF TOUCH SENSATIONS. THESE INCLUDE LIGHT TOUCH, PRESSURE, VIBRATION, AND TEMPERATURE AS WELL AS PAIN (SEE PP.106–107), AND AWARENESS OF THE BODY’S POSITION IN SPACE (PROPRIOCEPTIONS, SEE PP.104–105). THE SKIN IS THE BODY’S MAIN SENSE ORGAN FOR TOUCH.

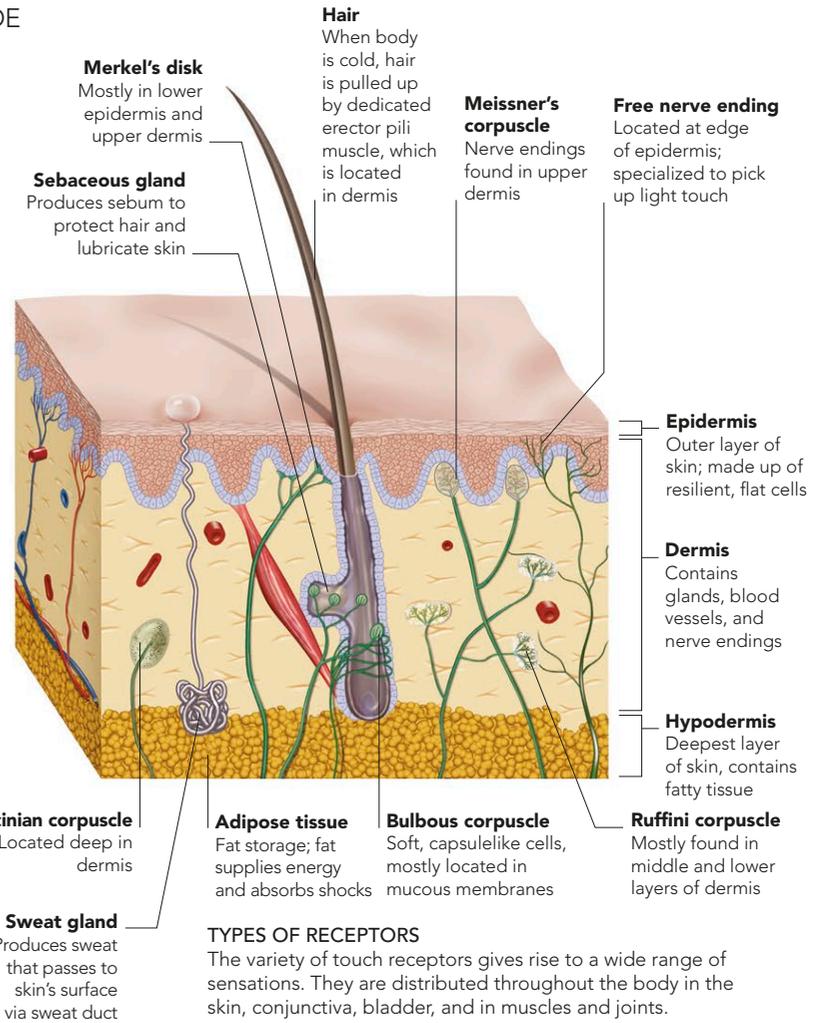
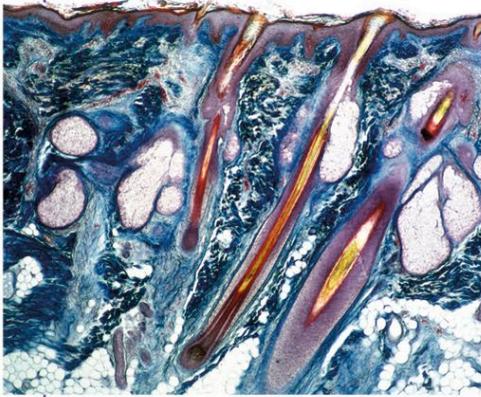
TOUCH RECEPTORS

There are around 20 types of touch receptor that respond to various types of stimuli. For instance, light touch, a general category that covers sensations ranging from a tap on the arm to stroking a cat’s fur, is detected by four different types of receptor cells: free nerve endings, found in the epidermis; Merkel’s disks, found in deeper layers of the skin; Meissner’s corpuscles, which are common in the palms, soles of the feet, eyelids, genitals, and nipples; and, finally, the root hair plexus, which responds when the hair moves. Pacinian and Ruffini corpuscles respond to more intense pressure. The

sensation of itching is produced by repetitive, low-level stimulation of nerve fibers in the skin, while feeling ticklish involves more intense stimulation of the same nerve endings when the stimulus moves over the skin.

SKIN STRUCTURE

Skin is the largest sense organ and allows us to interact fully with our surroundings. This light micrograph reveals how the skin is embedded with nerves, receptors, glands, hair follicles, and a rich blood supply.



TYPES OF TOUCH

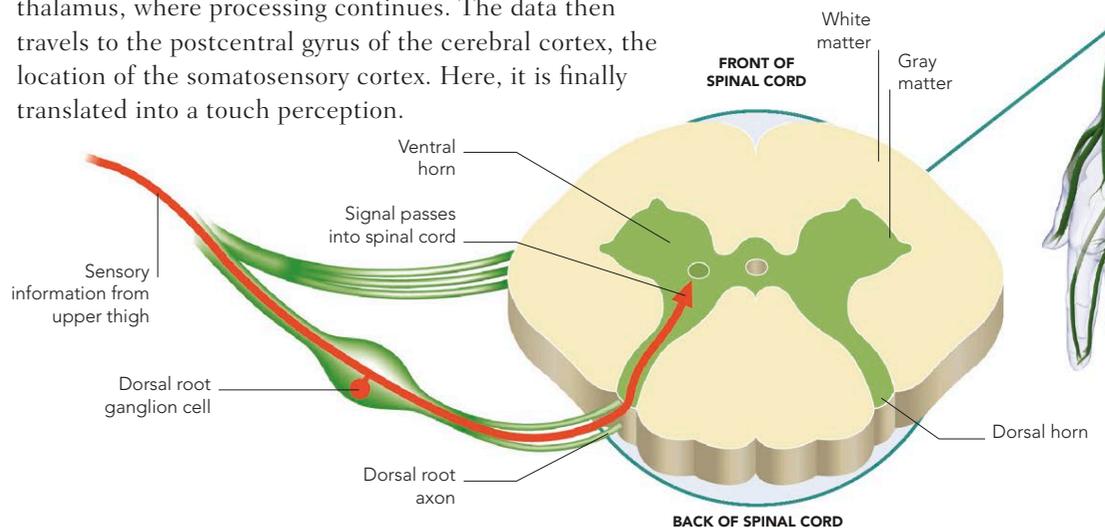
The different types of touch sensation convey detailed, complex information about the world around us and can act as a warning signal. Touch is essential for experiencing the texture and “feel” of objects. It also plays a vital role in communicating with others.

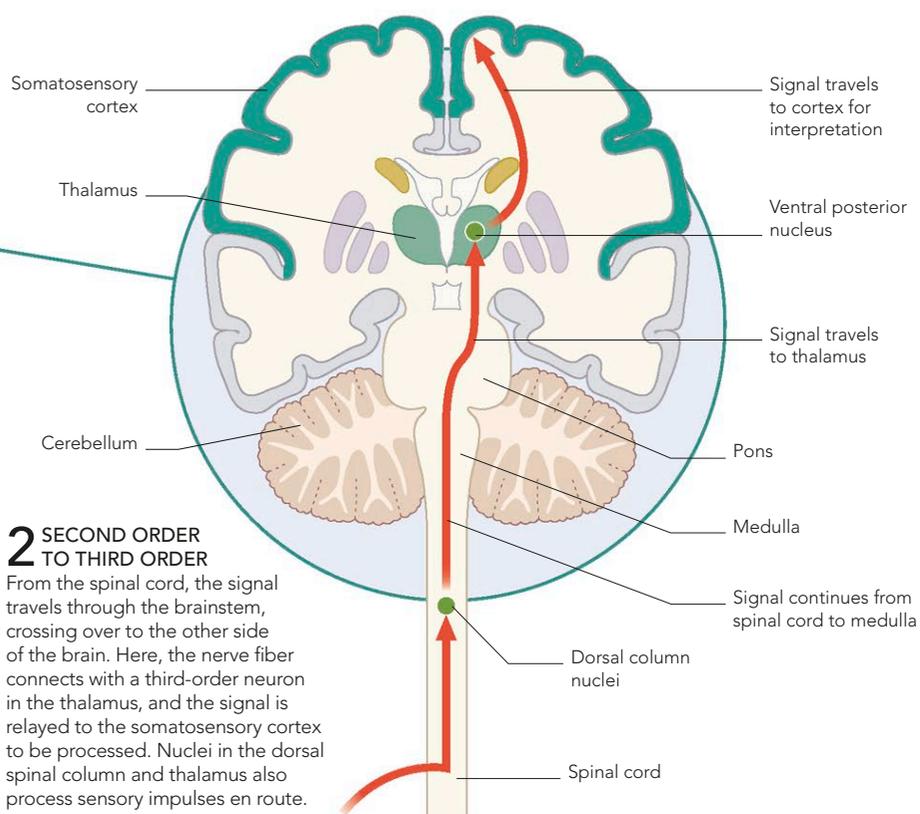
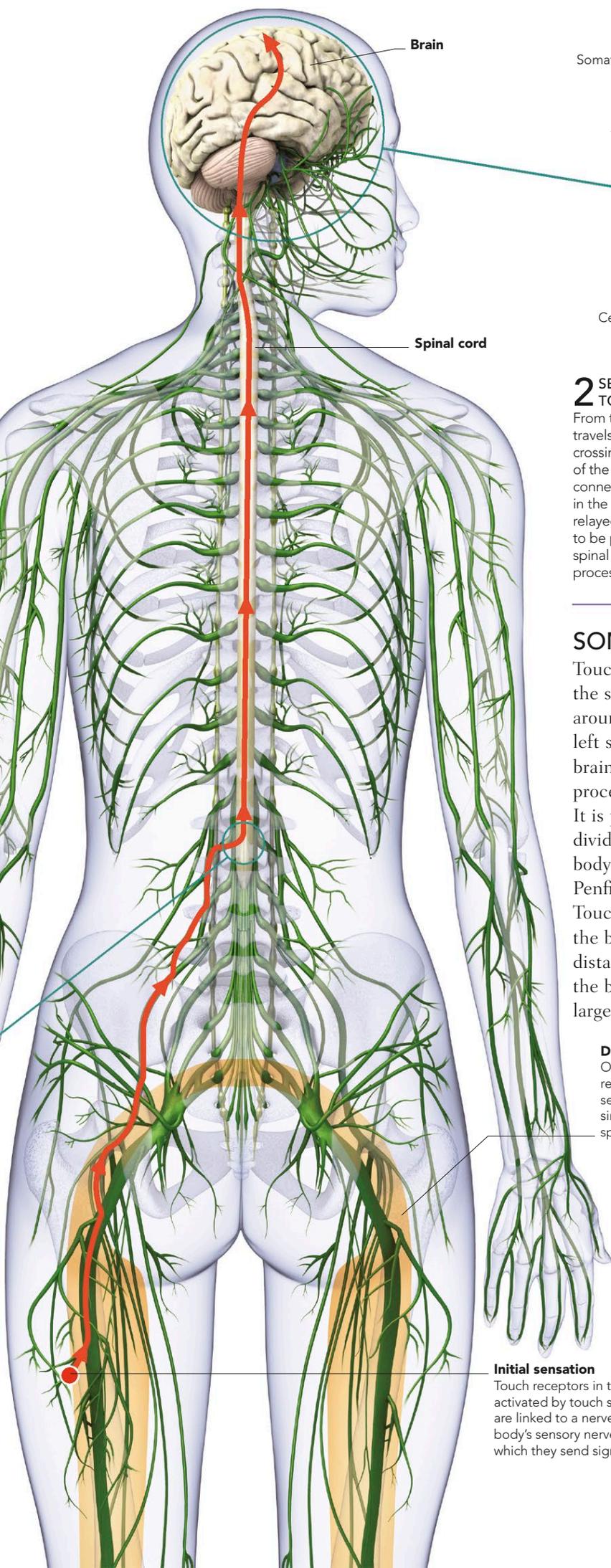
SENSATION	RECEPTORS
Light touch	The skin is not deformed by light touch, for example a handshake or a kiss. Free nerve endings in the skin respond to light-touch stimuli.
Touch pressure	Pressure entailing short-lived skin deformation stimulates Pacinian corpuscles and Ruffini corpuscles, located deep in the skin.
Vibration	Pacinian corpuscles and Meissner's corpuscles (mechanoreceptors, detecting mechanical movements) respond to vibrations.
Heat and cold	Receptors are sensitive to either hot or cold, not temperature itself. Heat and cold receptors occur in specific spots on the skin.
Pain	Pain signals come from damaged tissue and stimulate nociceptors (see pp.106–107), which consist of free nerve endings.
Proprioception	Receptor cells located in muscles and joints send information to the brain about the position and movement of the body.

TOUCH PATHWAY

When a sense receptor is activated, it sends information about touch stimuli as electrical impulses along a nerve fiber of the sensory nerve network to the nerve root on the spinal cord. The data enters the spinal cord and continues upward to the brain. The processing of sensory data is begun by nuclei in the upper (dorsal) column of the spinal cord. From the brainstem, sensory data enters the thalamus, where processing continues. The data then travels to the postcentral gyrus of the cerebral cortex, the location of the somatosensory cortex. Here, it is finally translated into a touch perception.

1 FIRST ORDER TO SECOND ORDER
First-order neurons carry data from the touch receptors of the upper thigh to the spinal cord. Their cell bodies are found in the dorsal root ganglia of the spinal cord. On entering the spinal cord, they connect with second-order neurons, most of which are located in the gray matter of the spinal cord, before traveling up the spinal cord along the pathway known as the ascending anterior spinothalamic tract.





2 SECOND ORDER TO THIRD ORDER

From the spinal cord, the signal travels through the brainstem, crossing over to the other side of the brain. Here, the nerve fiber connects with a third-order neuron in the thalamus, and the signal is relayed to the somatosensory cortex to be processed. Nuclei in the dorsal spinal column and thalamus also process sensory impulses en route.

SOMATOSENSORY CORTEX

Touch sensations are turned into perceptions in the somatosensory cerebral cortex, which curls around the brain like a horseshoe. Data from the left side of the body ends on the right side of the brain, and vice versa. Each part of the cortex processes data from a different part of the body. It is possible to make a map of the cerebral cortex, dividing it into regions that correspond to distinct body parts. Such a map was first drawn by Wilder Penfield, a renowned Canadian neurosurgeon. Touch receptors are unevenly distributed across the body. For example, experiments show that the distance between touch receptors is far greater on the back than on the lips. The hands have the largest proportion of touch receptors in the body.



HOMUNCULUS

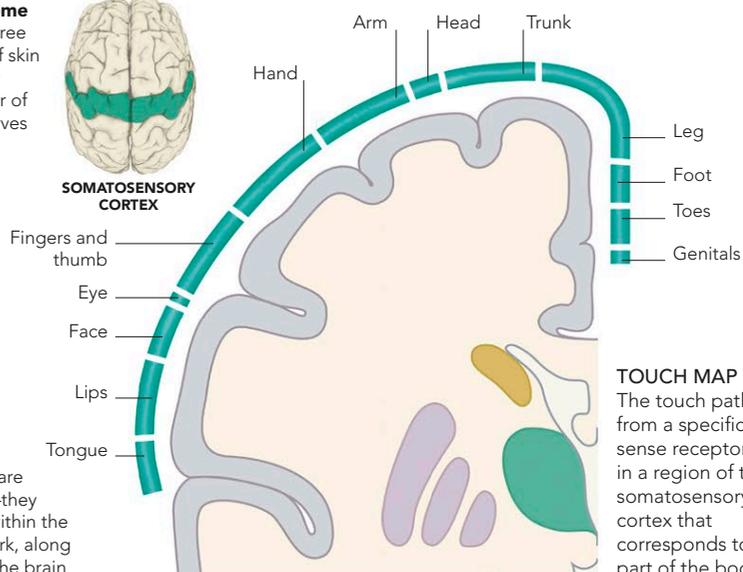
The size of the body parts with the most sensory and motor connections with the brain are proportionally enlarged on this distorted figure.

Dermatome

One of three regions of skin served by single pair of spinal nerves



SOMATOSENSORY CORTEX



TOUCH MAP

The touch pathway from a specific sense receptor ends in a region of the somatosensory cortex that corresponds to that part of the body.

Initial sensation

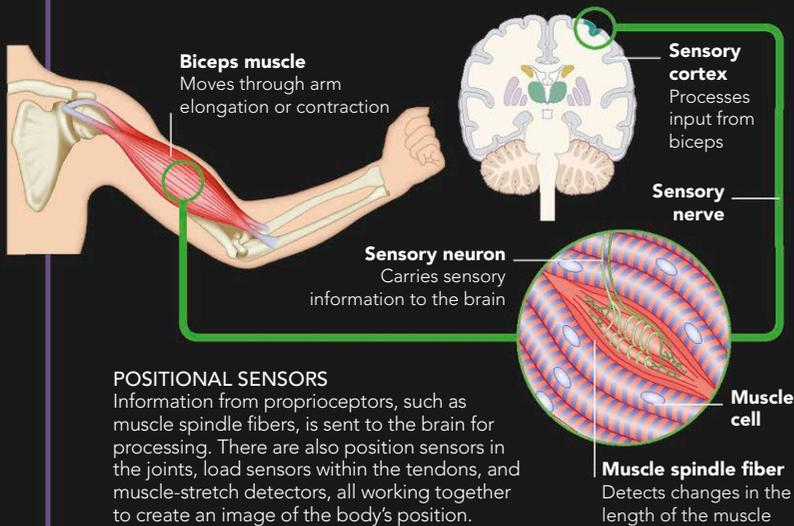
Touch receptors in the skin are activated by touch stimuli—they are linked to a nerve fiber within the body's sensory nerve network, along which they send signals to the brain

THE SIXTH SENSE

PROPRIOCEPTION—FROM *PROPRIO*, THE LATIN FOR “SELF”—IS SOMETIMES REFERRED TO AS THE SIXTH SENSE. IT IS THE SENSING OF BODY POSITION, MOVEMENT, AND POSTURE, INVOLVING FEEDBACK TO THE BRAIN FROM THE BODY. HOWEVER, THIS INFORMATION IS NOT ALWAYS MADE CONSCIOUS.

WHAT IS PROPRIOCEPTION?

Proprioception is our sense of how our bodies are positioned and moving in space. This “awareness” is produced by part of the somatic sensory system, and involves structures called proprioceptors in the muscles, tendons, joints, and ligaments that monitor changes in their length, tension, and pressure linked to changes in position. Proprioceptors send impulses to the brain. Upon processing this information, a decision can be made—to change position or to stop moving. The brain then sends signals back to the muscles based on the input from the proprioceptors—completing the feedback cycle.



POSITIONAL SENSORS

Information from proprioceptors, such as muscle spindle fibers, is sent to the brain for processing. There are also position sensors in the joints, load sensors within the tendons, and muscle-stretch detectors, all working together to create an image of the body's position.

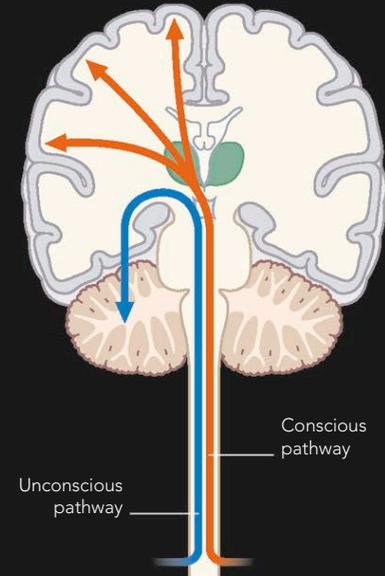
FIELD SOBRIETY TESTS

Proprioception is impaired when people are under the influence of alcohol or certain other drugs. The degree of impairment can be tested by field sobriety tests, which have long been used by the police in cases of suspected drink-driving. Typical tests include asking someone to touch their index finger to their nose with their eyes closed, to stand on one leg for 30 seconds, or to walk heel-to-toe in a straight line for nine steps.



TYPES OF PROPRIOCEPTION

Proprioceptive information is either made conscious or processed unconsciously. For example, keeping and adjusting balance is generally an unconscious process. Conscious proprioception usually involves some kind of cortical processing, resulting in decision-making. This normally ends in a command to the muscles to perform a movement. The sheer amount of proprioceptive input means that much is processed unconsciously.

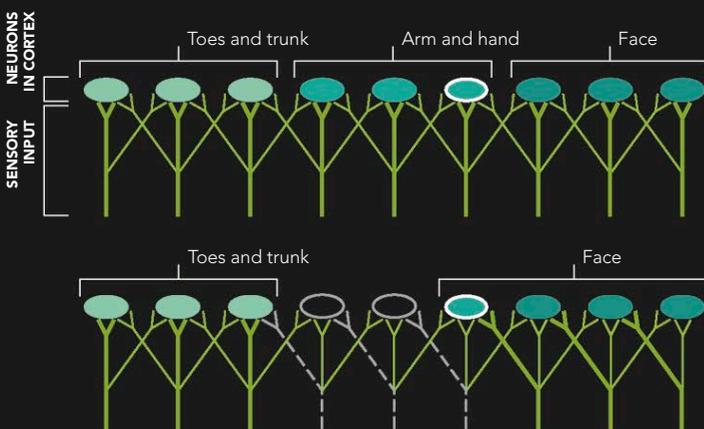


PROPRIOCEPTION PATHWAYS

Conscious proprioception uses the dorsal column–medial lemniscus pathway, which passes through the thalamus, and ends in the parietal lobe of the cortex. Unconscious proprioception involves spinocerebellar tracts, and ends in the cerebellum, the part of the brain at the back of the skull involved with the control of movement.

PHANTOM LIMBS

When someone has a part of the body amputated or removed—be it a limb, an extremity, or an organ, such as the appendix—they sometimes continue to have sensations, often including pain, in that area. Research has linked this to changes in the sensory cortex. Specifically, the somatosensory cortex undergoes a remapping process in which the areas near the “dead” area “take over”, so that stimuli in these areas are felt as sensations in the area that has been lost. This reorganization of the cortex has been confirmed through imaging studies.



PHANTOM-LIMB-PAIN TREATMENT

Research has shown that the development of phantom-limb pain is linked to the plasticity of the sensory cortex. Trying to reverse the changes in the cortex can actually reduce the pain sensation for the patient. For instance, use of an electric prosthetic limb that is moved by signals from the patient's muscles was helpful. Brain scans revealed that this was linked with reversion of the cortex to its original state, maybe by replacing some of the original input.



MIRROR TREATMENT
When a patient's remaining arm is shown as a mirror image and moved, it looks as though the missing arm is moving. Somehow, this illusion can relieve phantom-limb pain.

FINE BALANCE

Proprioceptors in the muscles, tendons, and skin work together with hair cells in the vestibule and semicircular canals of the inner ear to maintain balance. A gymnast will work on all aspects of strength, movement, and body coordination to achieve feats involving fine balance.



PAIN SIGNALS

PAIN IS PRIMARILY A WARNING SIGNAL. IT TELLS YOU THERE IS SOMETHING WRONG AND FORCES YOU TO TAKE ACTION. PAIN USUALLY OCCURS AS A RESULT OF STIMULATION OF SPECIALIZED NERVE FIBERS THAT EXTEND THROUGHOUT THE BODY.

PAIN PATHWAYS

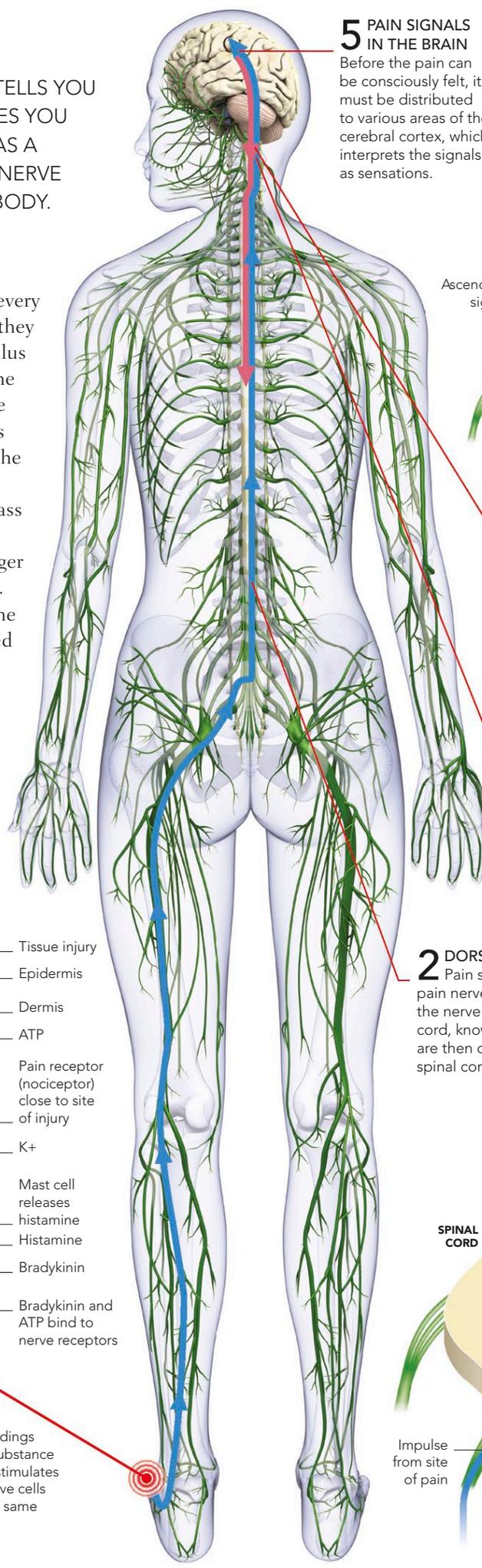
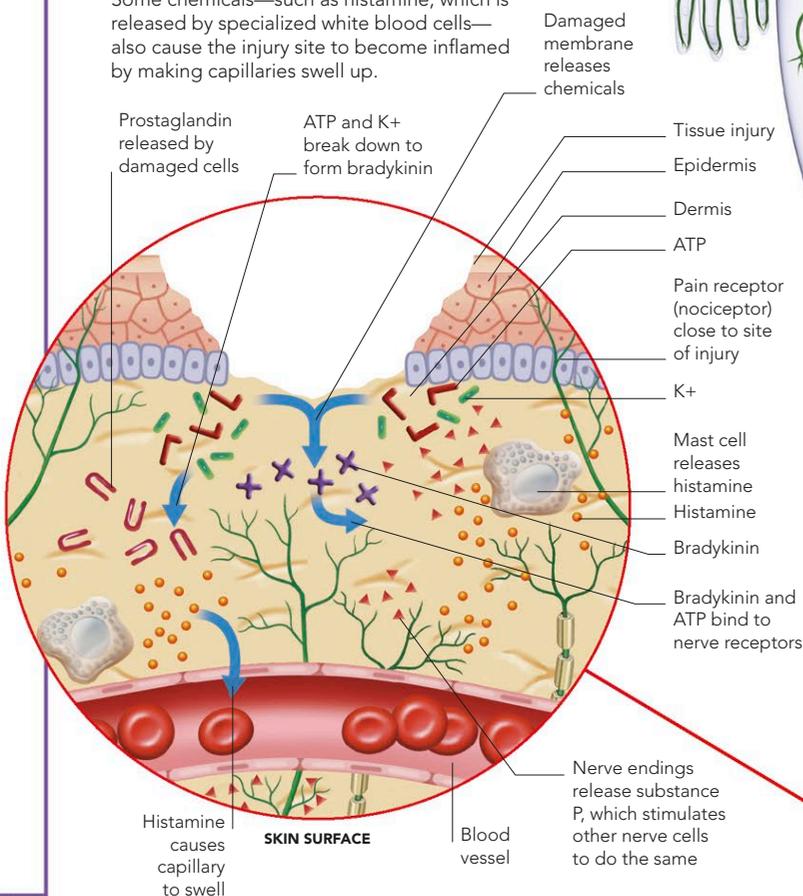
Pain-transmitting nerve fibers permeate almost every part of the body. When stimulated by an injury, they send electrical signals from the site of the stimulus to the spinal cord. The signals then cross over the cord and continue up to the brain. This crossover means that pain from one side of the body activates the opposite side of the brain. As they pass through the medulla in the brainstem, pain signals trigger automatic bodily responses. The signals then arrive at the thalamus and are distributed to various regions of the brain to be processed.



FEELING PAIN
Pain is not felt until the brain has processed signals indicating injury.

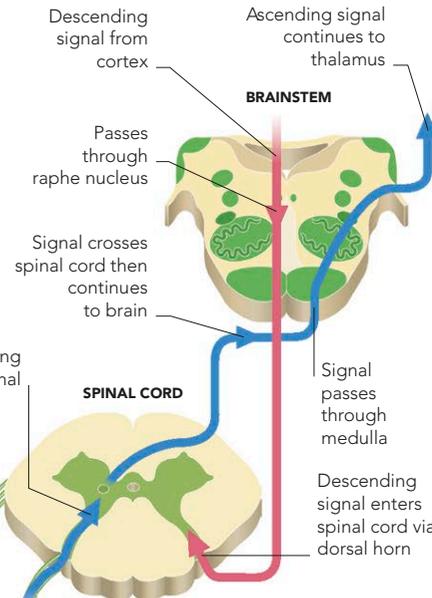
1 INFLAMMATORY "SOUP"

Injury sets off the release of chemicals, such as bradykinin and ATP, which trigger the nerve impulses that are experienced as pain. Some chemicals—such as histamine, which is released by specialized white blood cells—also cause the injury site to become inflamed by making capillaries swell up.



5 PAIN SIGNALS IN THE BRAIN

Before the pain can be consciously felt, it must be distributed to various areas of the cerebral cortex, which interprets the signals as sensations.



4 DESCENDING CONNECTIONS

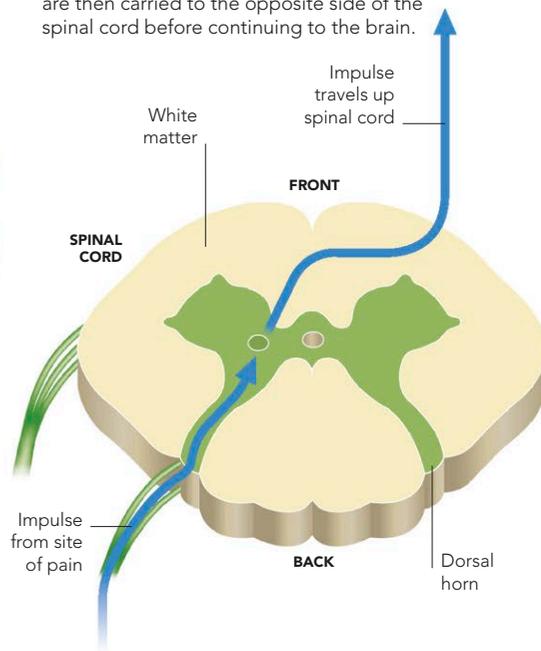
Nerve fibers descending from pain-registering regions of the brain intercept the ascending pain signals and modify them, by triggering the release of analgesic chemicals in the brainstem and spine in order to reduce pain.

3 MEDULLA

As the pain signals pass through the medulla, a part of the brainstem, they trigger activity in the autonomic nervous system (see pp.112–13). This results in an increase of blood pressure, heart and breathing rates, and sweating.

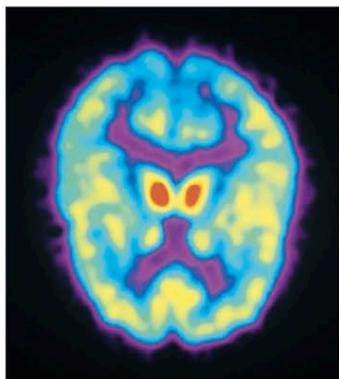
2 DORSAL HORN

Pain signals travel to the spine along pain nerve fibers. Most pain fibers enter the nerve tract at the back of the spinal cord, known as the dorsal horn. The signals are then carried to the opposite side of the spinal cord before continuing to the brain.



THE CHEMISTRY OF PAIN RELIEF

The body has a natural opioid (pain relief) system that acts in much the same way as opiate drugs, such as heroin and morphine. Natural opioids, which include endorphins and enkephalins, are produced by the thalamus and pituitary gland during stress and pain. These substances are also produced in situations associated with feeling a natural “high,” such as strenuous exercise and sexual activity. Nerve endings in the brain and throughout the body have special receptors on them that bind to opiate substances. The opiates then dampen the pain signals carried in those nerve endings, thus reducing pain.



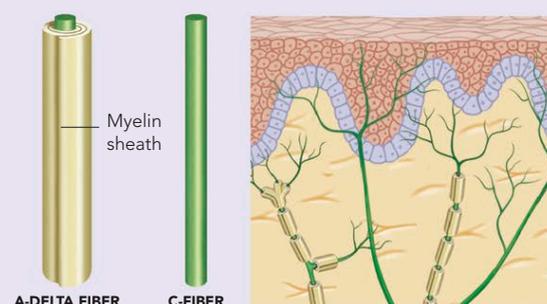
OPIOID RECEPTORS

This PET scan shows the concentration of opioid receptors in a normal brain. Red areas show where they are highest, through yellow and green, to blue, which indicates the lowest concentration.

PAIN FIBERS

There are two main types of nerve fiber that detect pain: A-delta and C. A-delta fibers are thin and carry sharp, localized pain signals to the brain. The site of the injury will be within a millimeter of these nerve fibers, so the site is easily identified. These nerve

fibers are covered in a fatty myelin sheath that aids the transmission of signals. C-fibers are not insulated by a myelin sheath. The source of pain transmitted by a C-fiber is difficult to pinpoint because its nerve endings are spread out over a relatively large area.



C-FIBERS AND A-DELTA FIBERS

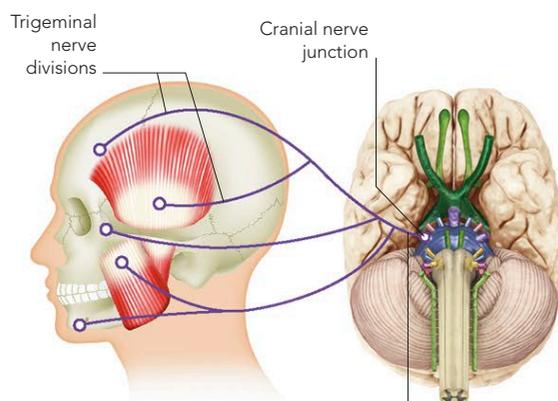
A-delta fibers are found mostly in subcutaneous tissue. C-fibers accompany all the blood vessels, lymphatic, sensory, and motor nerves, and the peripheral autonomic nerves.

TYPES OF PAIN

Pain usually arises when pain receptors are stimulated by heat, cold, vibration, overstretching, or by chemicals released from damaged cells. Specialized nerve fibers (see panel, above) transmit this information to the brain. However, certain types of pain are processed and experienced in different ways, for example the facial nerves connect directly to the cranial nerves (see below), whereas visceral pain, from internal organs such as the heart (see right), can be difficult to locate. Damage to the nervous system itself, such as a trapped nerve, is known as neuropathic pain (see bottom).

FACIAL PAIN

Stimulation of trigeminal nerves usually causes facial pain. It often affects only one side of the face and can be felt on the skin or in the mouth and teeth. It comes and goes unpredictably and its nature is variously described as stabbing, lacerating, like an electric shock, and shooting. It can range in severity from mild to excruciating. There are frequently “trigger points” on the skin, which, if touched, will bring on a violent pain spasm. People may experience pain daily for weeks and months, then it may disappear for months or even years.

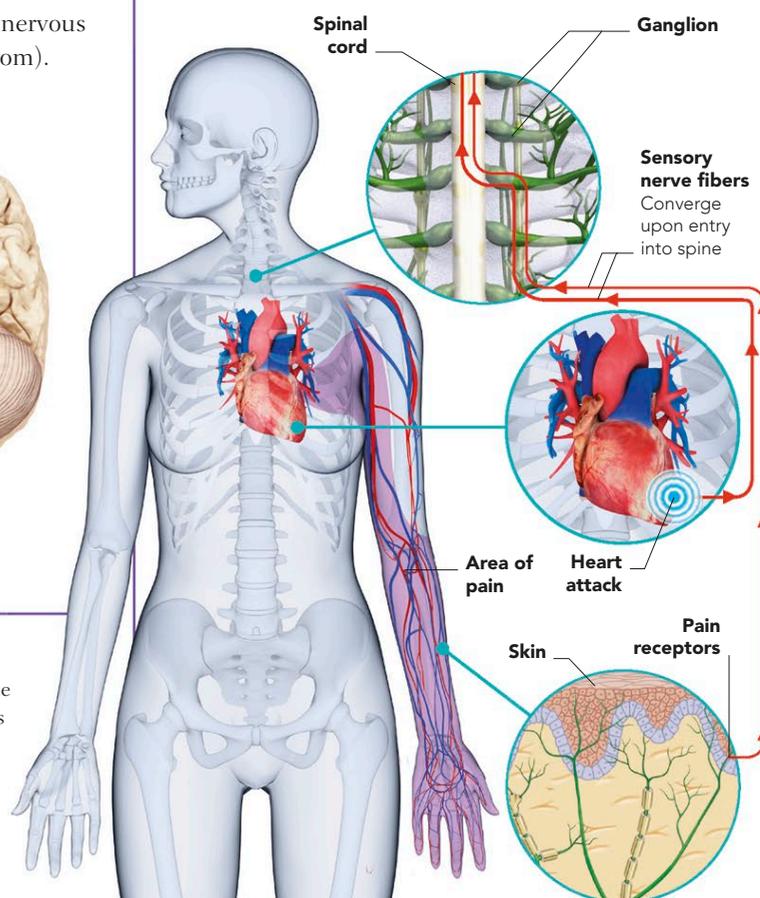


TRIGEMINAL NERVES

There are two trigeminal nerves, one on each side of the face, each has branches to the forehead, cheek, and jaw.

REFERRED PAIN

Referred pain occurs when nerve fibers from areas of high sensory input (such as the skin) and nerve fibers from areas of low sensory input (such as internal organs) enter the spinal cord at the same location. Since the brain expects to be receiving the data from high-sensory areas, it misinterprets the location of the pain.



HEART ATTACK

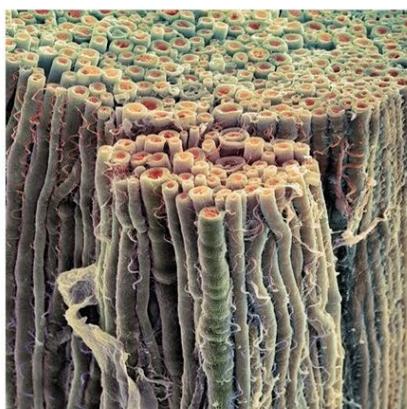
Pain-signaling nerves from the heart converge with those from the arm as they enter the spinal cord. The brain interprets the signals as coming from the arm rather than the heart.

NEUROPATHIC PAIN

Pain that is caused by damage or malfunction in the nervous system itself rather than injury is known as neuropathic pain. A pain-transmitting nerve may be severed, or be stimulated so often that it gets into the “habit” of sending pain signals to the brain. Pain-registering neurons in the cortex can become sensitized so that they produce the experience of pain even when there is no external cause.

SEVERED NERVE BUNDLE

This colored electron micrograph shows a severed bundle of nerves. These may continue to send pain signals to the brain even when the cause of the damage itself is long gone.

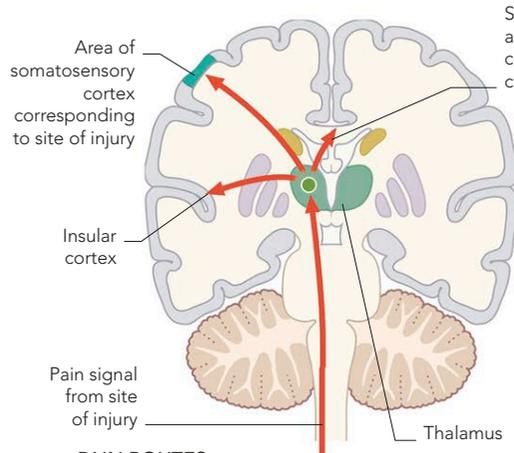


EXPERIENCING PAIN

THE FEELING OF PAIN IS NOT ACTUALLY CAUSED BY AN INJURY IN ITSELF. IN ORDER TO EXPERIENCE PAIN, IT MUST BE MADE CONSCIOUS. THIS REQUIRES ACTIVITY IN BRAIN AREAS INVOLVED IN EMOTION, ATTENTION, AND ASSESSING SIGNIFICANCE. SUCH ACTIVITY CAN CREATE THE PAIN EXPERIENCE IN THE ABSENCE OF A CAUSE.

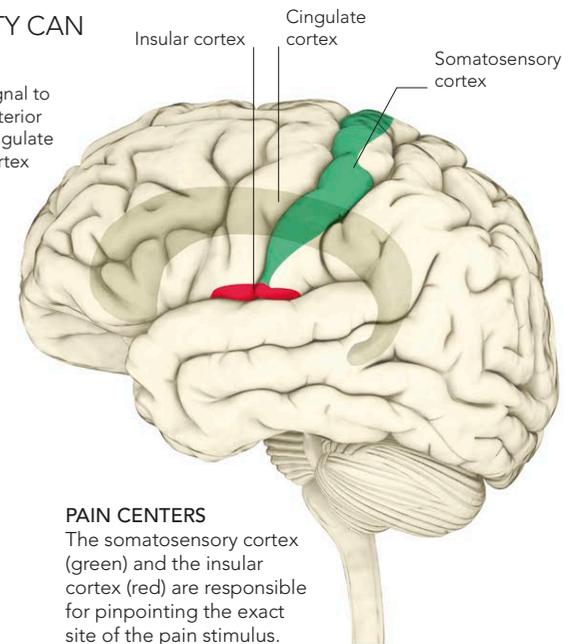
PATHWAY OF PAIN

Pain signals are transmitted to several areas of the cortex, where they activate neurons that monitor the state of the body. Two such areas are the somatosensory cortex, which lets the brain know which part of the body the pain stems from, and the insular cortex—the deep fold that divides the temporal and frontal lobes. The other cortical site associated with pain experience is the anterior (front) of the cingulate cortex (ACC), which lies in the groove between the hemispheres. The ACC seems to be particularly concerned with the emotional significance of pain and with determining how much attention an injury should command.



PAIN ROUTES

Pain signals from the body ascend to the brain via the spinal cord, then rise through the brainstem to the thalamus. Thereafter, they are distributed to various cortical areas for processing.

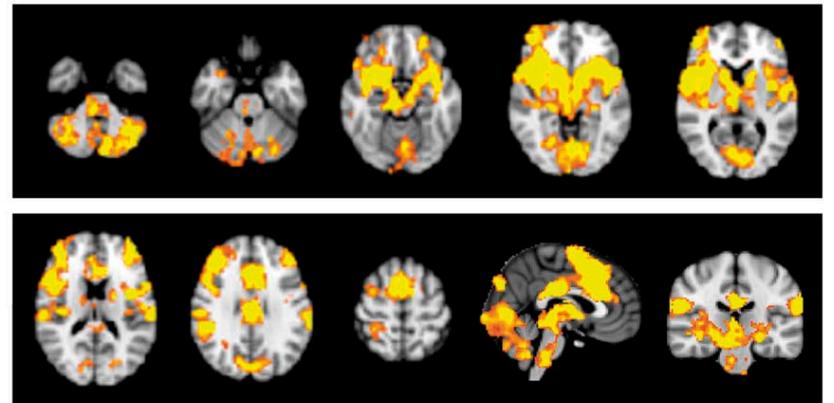
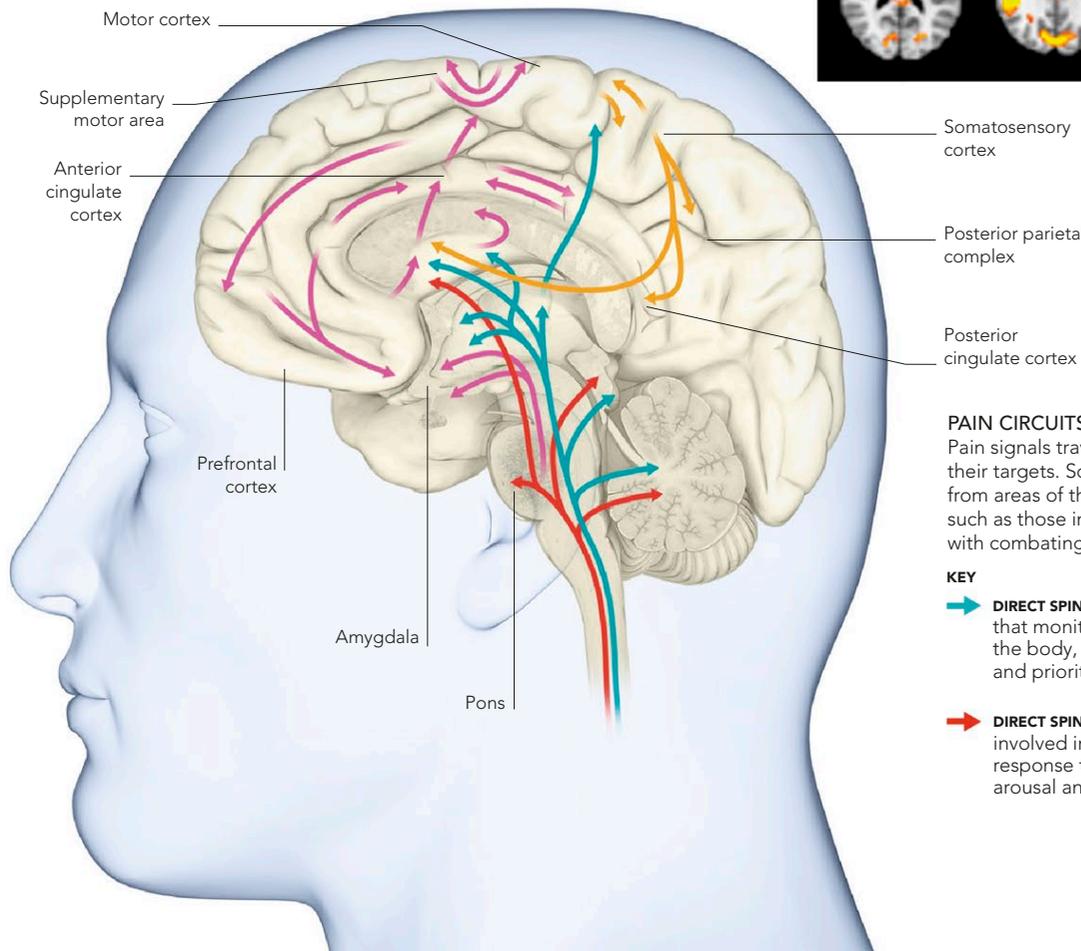


PAIN CENTERS

The somatosensory cortex (green) and the insular cortex (red) are responsible for pinpointing the exact site of the pain stimulus.

A WHOLE-BRAIN AFFAIR

Pain is so important to our survival that it may involve practically every part of the brain. Three main “pain areas” (see above) register and assess pain signals, and pinpoint the site of their source, but other areas also come into play. The supplementary motor area and motor cortex may plan and execute movement aimed at escaping the pain stimulus. Parts of the parietal cortex may direct attention to the threat, and several parts of the frontal cortex may be involved in working out the significance of the pain and what to do about it.



PAIN STUDY

The fMRI scans above show various “slices” through the brain of a healthy person who is being subjected to a painful stimulus on the arm. The regions highlighted in yellow show areas of neural activity in response to the stimulus, revealing how widespread the effects of pain are on the brain.

PAIN CIRCUITS

Pain signals travel along many different neural circuits to hit their targets. Some follow the paths of nerves that ascend from areas of the body, while others stem from brain nuclei, such as those in the hypothalamus, which are concerned with combating the effects of the pain stimulus.

KEY

- **DIRECT SPINAL INPUT** to areas that monitor the state of the body, direct attention, and prioritize response.
- **DIRECT SPINAL INPUT** to areas involved in automatic response to pain, such as arousal and movement.

- **CIRCUIT THROUGH** cortical and limbic areas involved in evaluation and monitoring of pain.
- **CIRCUIT THROUGH** cortical and limbic areas that affect pain, including intensity, emotion, and pain memory.

BRAIN OVER PAIN

Part of the role of higher brain areas is to modify pain. Nerve signals that travel from the brain into the body interrupt pain signals traveling up from the site of the injury before they reach the brain. This reduces the number of pain signals reaching the brain and, therefore, the amount of pain felt. Also, our thoughts, expectations, and emotions can all have a profound effect on the degree to which a person is “pained” by pain. People can affect pain consciously by directing attention away from it, or imagining that they are pain-free. An intensely imagined experience generates almost identical brain activity to the equivalent “real” experience, so an imagined state of physical ease may be achieved even as pain fibers in the body are being stimulated.

PLACEBO AND NOCEBO

Pain may be exacerbated or reduced as a result of the way in which we think about it. Believing that pain is being alleviated, by surgical intervention or a drug, for example, can help ease the pain. This is known as the placebo effect. Expecting pain to be intractable or bad does the opposite, known as the nocebo effect.

FREEZING OUT PAIN

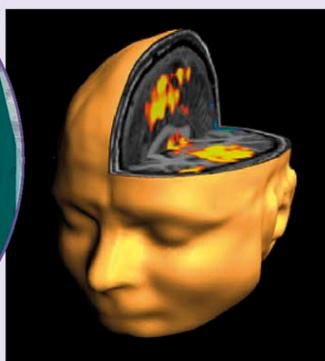
The cingulate cortex is an area of the brain that is partly concerned with determining how much attention to give to a pain stimulus. People can develop the ability to tone down activity in this region by learning to shift attention away from the pain stimulus, creating an analgesic effect. Using virtual reality as a focus point has been found to help distract attention away from pain.



DISTRACTING ATTENTION
Burns victims have been found to experience pain relief when immersed in a cooling virtual environment, which is thought to work by distracting attention away from pain.



VIRTUAL ENVIRONMENT
Virtual reality is so distracting, it leaves less attentional resources available for the brain to process pain signals.

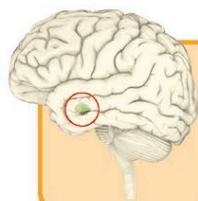


NO VIRTUAL REALITY

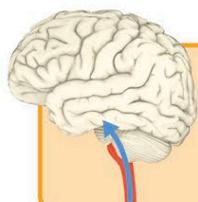


USING VIRTUAL REALITY

PAIN-RELATED BRAIN ACTIVITY
The areas colored yellow in these images show activity related to pain. The distraction provided by virtual reality significantly reduces activity in these areas (right).



Anxiety signals from the amygdala—pain-related or otherwise—spark brain activity in a way that is associated with the experience of pain.



Pain stimulus arrives in the brain via the spinal cord, causing levels of anxiety to become increased.

PAIN

Nocebo effect
Anxiety plus pain input from the body produces a pain-related experience that is more intense than if either factor occurred alone. Anxiety is therefore an example of the nocebo effect—an intensification of pain due to the effects of negative thoughts, beliefs, or expectations.

Placebo effect
The belief that an intervention such as a drug or a medical procedure will alleviate pain is itself able to reduce a pain experience. This is because experience is subjective so, if you think you do not feel pain, you don't. The process by which belief becomes fact is known as the placebo effect.

Descending signals from the prefrontal cortex of the brain can interrupt incoming pain signals. This can be unconscious or consciously controlled.

The anterior cingulate cortex can play a role in directing attention away from pain. Deliberately diverting attention from pain reduces activity here.

PAIN AND THE BRAIN

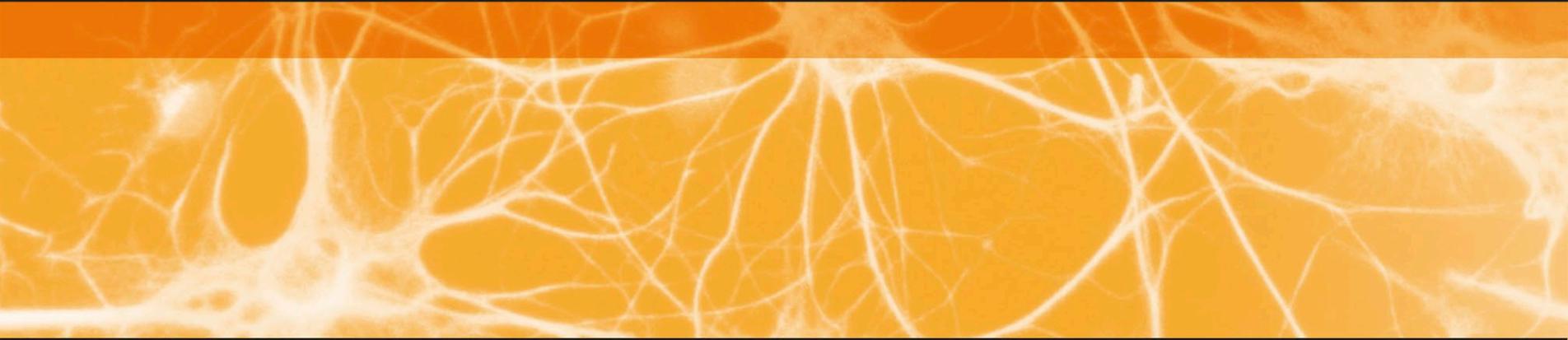
Although the brain is responsible for the experience of pain, it does not feel pain itself because it contains no pain receptors. This fact becomes very useful during brain surgery, because it allows surgeons to operate while the patient is conscious. The patient can report their experiences when different areas of the brain are stimulated and, therefore, help the surgeons recognize areas of the brain that have crucial functions. In this way, surgeons can carefully work their way toward, say, a brain tumor without damaging important and healthy brain tissue.

BRAIN SURGERY
Patients who remain conscious during brain operations can tell surgeons when the scalpel is close to a crucial area by responding to questions.



LIFE WITHOUT PAIN

A very few people—probably about one in 125 million—are born without the ability to feel pain. The condition is caused by a genetic disorder, congenital analgesia, that results in a lack of pain-sensitive nerve endings in the body. Some people with this condition are able to feel touch or pressure, which relies on different types of nerves. Although the idea of not feeling any pain may, at first, sound rather desirable, the effect is disastrous. Pain normally warns people that they are in danger and forces them to take action to protect themselves. Without it, physical perils are likely to be unnoticed or ignored, leading to lethal injuries and often to premature death.



THE BRAIN IS IN CONSTANT COMMUNICATION WITH THE REST OF THE BODY, CONTROLLING EVEN ITS MOST BASIC PROCESSES. IN DOING SO, IT INITIATES MANY MOVEMENTS THAT WE ARE NOT AWARE OF, SUCH AS SPEEDING UP OR SLOWING DOWN OUR RATE OF BREATHING. SOME OTHER MOVEMENTS ARE MADE AS REFLEX ACTIONS, WITHOUT ANY SIGNALS REACHING THE BRAIN AT ALL. SUCH UNCONSCIOUS ACTION LEAVES THE CONSCIOUS BRAIN FREE TO DIRECT ITS ATTENTION TO OTHER THINGS, INCLUDING MOVEMENTS THAT REQUIRE GREAT CONCENTRATION AS WELL AS CAREFUL PLANNING.

MOVEMENT AND CONTROL



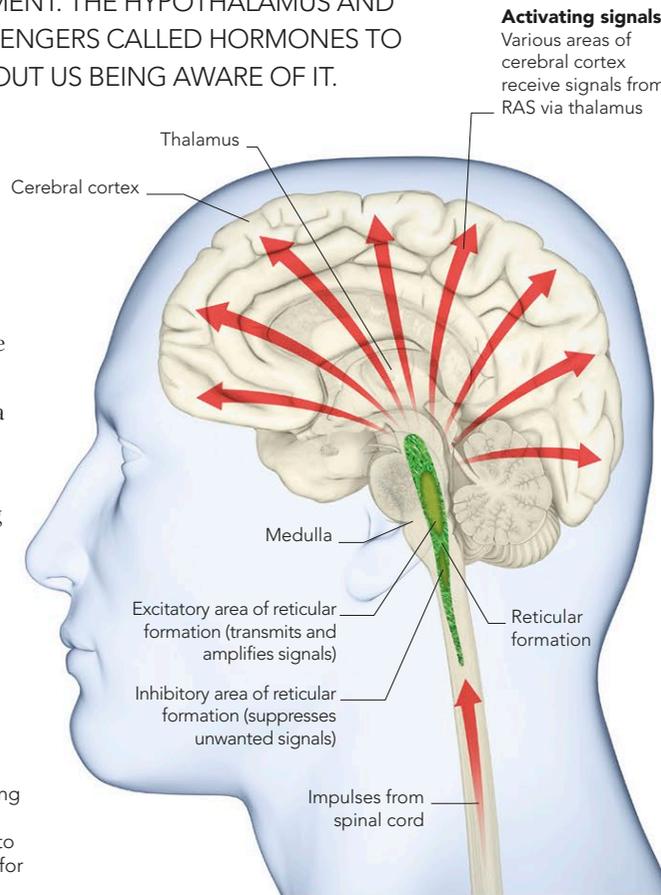
REGULATION

THE BODY'S BASIC FUNCTIONS ARE CAREFULLY CONTROLLED IN ORDER TO MAINTAIN A STABLE INTERNAL ENVIRONMENT. THE HYPOTHALAMUS AND BRAINSTEM WORK WITH CHEMICAL MESSENGERS CALLED HORMONES TO KEEP THE BODY TICKING—MOSTLY WITHOUT US BEING AWARE OF IT.

THE RETICULAR FORMATION

The reticular formation is located in the brainstem and is made up of a series of long nerve pathways that modulate sensory inputs and carry information to and from the cerebral cortex. It also plays an important role in regulating the autonomic nervous system (ANS), which is responsible for maintaining a balanced internal environment. The reticular formation contains neuronal centers that manage various functions, such as controlling the heart rate and rate of respiration. It is also involved in regulating other basic functions such as digestion, salivation, perspiration, urination, and sexual arousal. The reticular formation and its connections constitute the reticular activating system (RAS), an arousal mechanism that keeps the brain alert and awake.

THE RETICULAR ACTIVATING SYSTEM
The RAS receives incoming sensory information and transfers it to the cortex to keep it alert and primed for environmental changes.



Activating signals
Various areas of cerebral cortex receive signals from RAS via thalamus

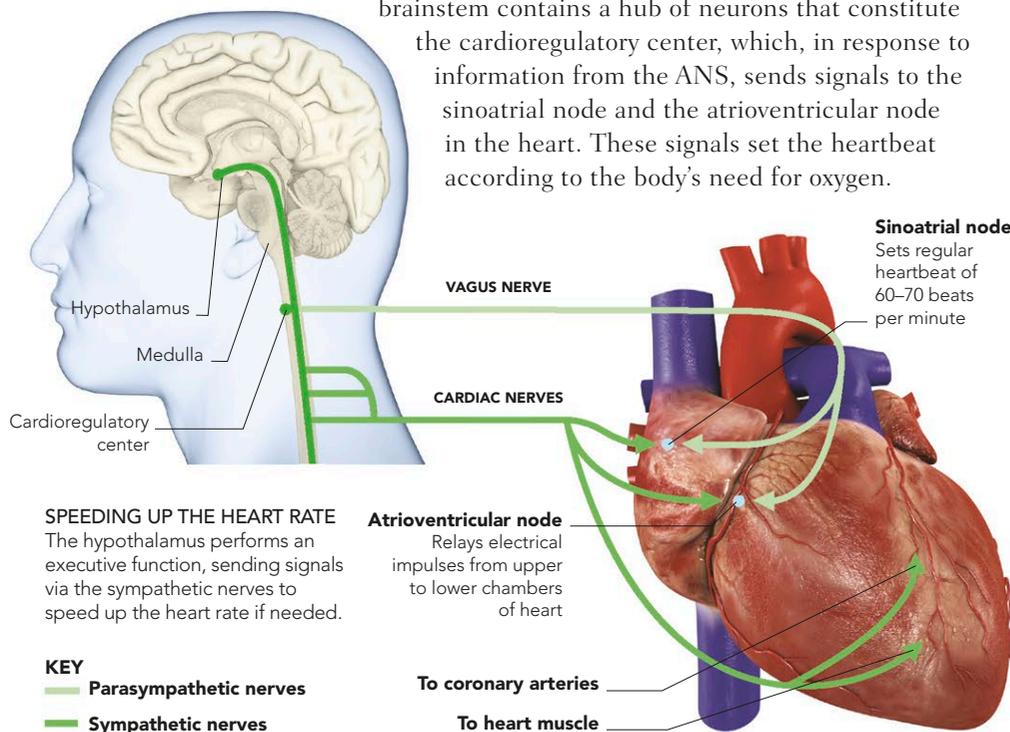
GENERAL ANESTHETICS

A cornerstone of modern medicine, general anesthetics allow surgeons to carry out operations that were previously unfeasible. Yet the way in which an anesthetic causes loss of consciousness in a controlled and reversible way is still not fully understood. Ether, chloroform, and halothane act on neurons in the reticular activating system, suppressing alertness and awareness, and also on neurons in the hippocampus, temporarily wiping out memories. These substances also affect the nuclei in the thalamus, by interrupting the flow of sensory information from the body to the brain. Together, the actions of anesthetics on the brain produce an experience of deep oblivion.



REGULATION OF HEART RATE

The heart rate is regulated by the hormonal action of the ANS, which, in turn, is regulated by the reticular formation. The sympathetic branch of the ANS speeds up the heart rate and the parasympathetic branch slows it down. The medulla in the brainstem contains a hub of neurons that constitute the cardiorespiratory center, which, in response to information from the ANS, sends signals to the sinoatrial node and the atrioventricular node in the heart. These signals set the heartbeat according to the body's need for oxygen.



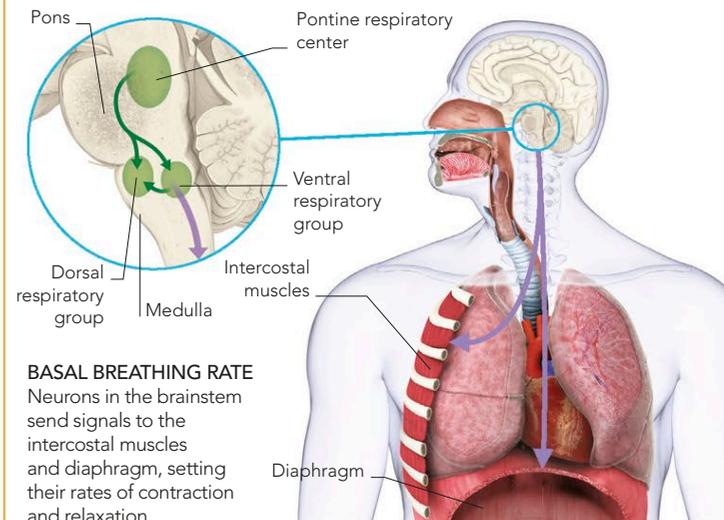
SPEEDING UP THE HEART RATE
The hypothalamus performs an executive function, sending signals via the sympathetic nerves to speed up the heart rate if needed.

Atrioventricular node
Relays electrical impulses from upper to lower chambers of heart

KEY
— Parasympathetic nerves
— Sympathetic nerves

REGULATION OF BREATHING

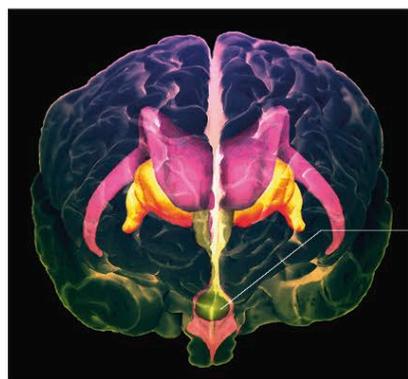
The rate of breathing in and out is regulated by collections of neurons in the reticular formation, called the dorsal and ventral respiratory groups. These respond to levels of oxygen and carbon dioxide in the blood and regulate the breathing rate accordingly to maintain constant levels. The basal rate of breathing can also be adjusted (in response to increased activity or metabolism) through electrical impulses sent by the pontine respiratory center.



BASAL BREATHING RATE
Neurons in the brainstem send signals to the intercostal muscles and diaphragm, setting their rates of contraction and relaxation.

FUNCTIONS OF THE HYPOTHALAMUS

The hypothalamus contains many minute clusters of neurons, called nuclei, which perform specific functions, including controlling body temperature, eating and drinking behavior, water balance, hormonal levels, and sleep-wake cycles. Among other things, the hypothalamus is regarded as the major coordinating center of the limbic system, and it has extensive connections with the pituitary gland and autonomic nervous system. Through these connections, it produces vital responses to body conditions and initiates feelings such as hunger, anger, and fear. The functions of the hypothalamus are essential to life, so even subtle damage can have dramatic effects on behavior and survival.

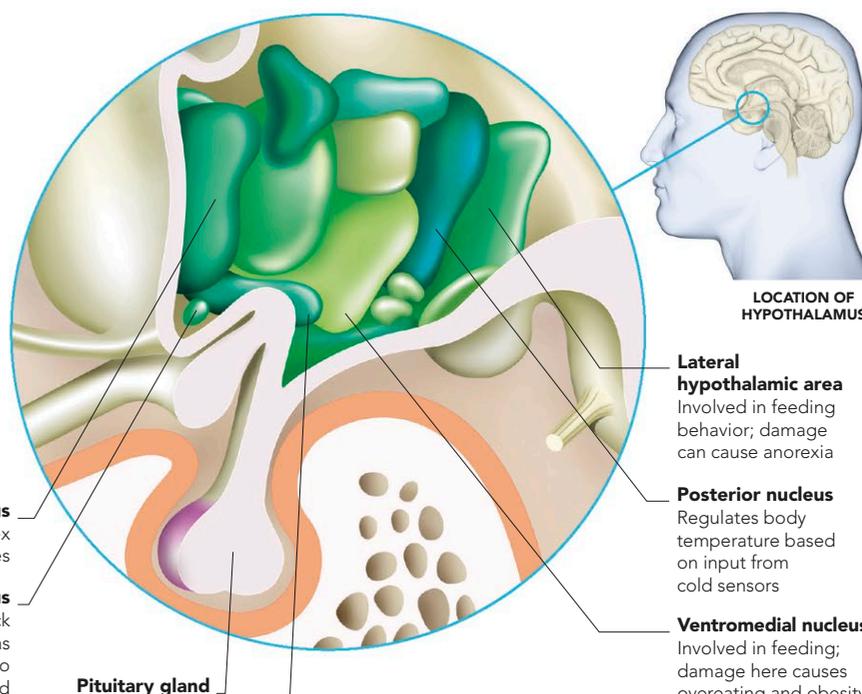


Hypothalamus

HYPOTHALAMUS
This illustration shows the location of the hypothalamus. It lies beneath the thalamus, near the brainstem, and is about the size of a sugar cube.

Medial preoptic nucleus
Regulates production of sex hormones

Suprachiasmatic nucleus
Helps regulate body clock and circadian rhythms; has numerous connections to pituitary gland



LOCATION OF HYPOTHALAMUS

Lateral hypothalamic area
Involved in feeding behavior; damage can cause anorexia

Posterior nucleus
Regulates body temperature based on input from cold sensors

Ventromedial nucleus
Involved in feeding; damage here causes overeating and obesity

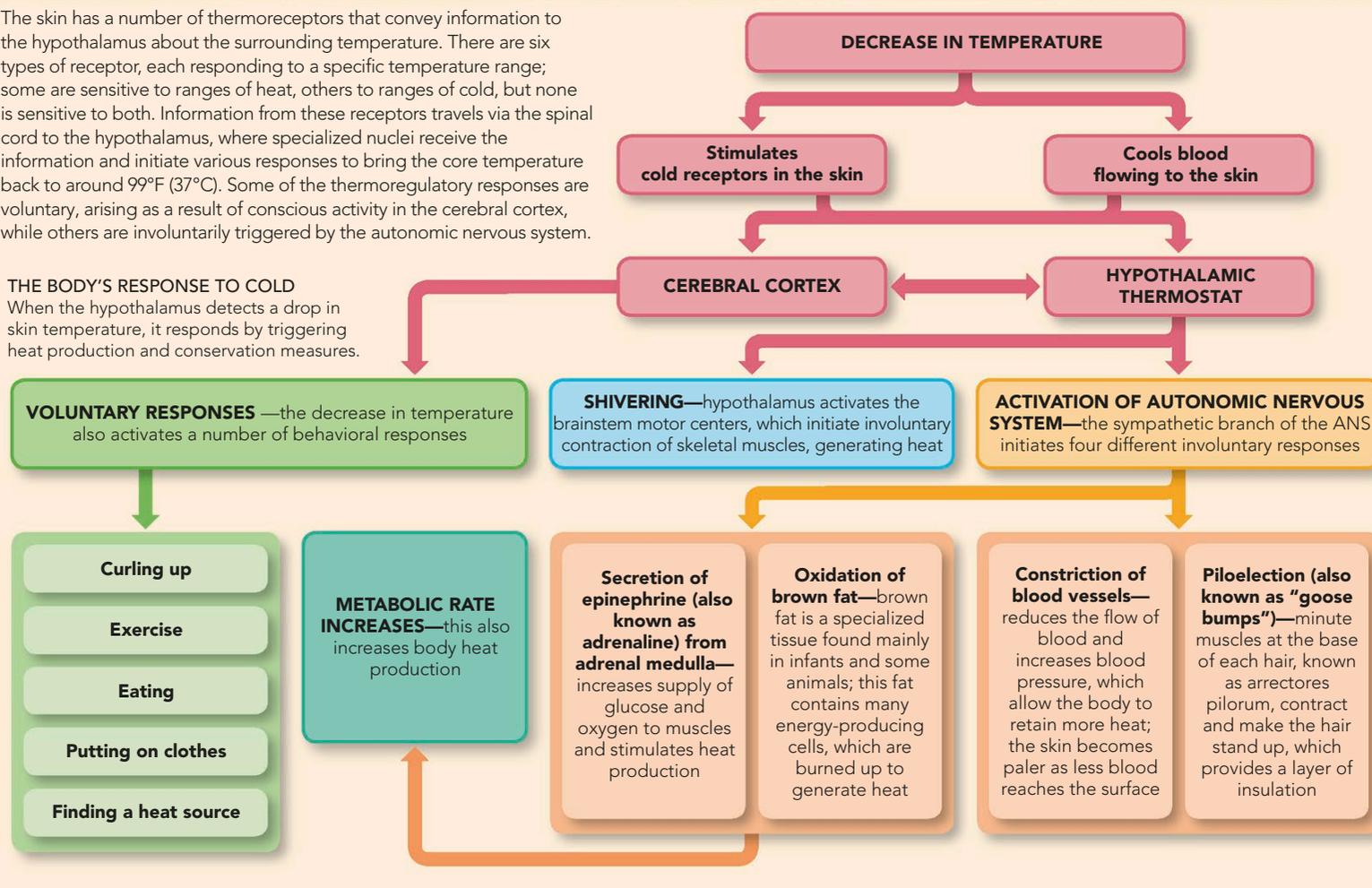
Anterior nucleus
Neurons in this region are concerned with temperature control and process data from body's heat sensors

HYPOTHALAMIC NUCLEI
Groups of neurons (nuclei) within the hypothalamus have specialized roles in controlling specific responses and regulating the body's systems. Their complete range of functions is not fully known, but some functions have been identified and isolated to specific regions.

THE BODY'S THERMOSTAT

The skin has a number of thermoreceptors that convey information to the hypothalamus about the surrounding temperature. There are six types of receptor, each responding to a specific temperature range; some are sensitive to ranges of heat, others to ranges of cold, but none is sensitive to both. Information from these receptors travels via the spinal cord to the hypothalamus, where specialized nuclei receive the information and initiate various responses to bring the core temperature back to around 99°F (37°C). Some of the thermoregulatory responses are voluntary, arising as a result of conscious activity in the cerebral cortex, while others are involuntarily triggered by the autonomic nervous system.

THE BODY'S RESPONSE TO COLD
When the hypothalamus detects a drop in skin temperature, it responds by triggering heat production and conservation measures.



THE NEUROENDOCRINE SYSTEM

THE BRAIN MAINTAINS THE BODY'S STABLE INTERNAL STATE, KNOWN AS HOMEOSTASIS, THROUGH THE ACTION OF HORMONES. NEURAL-CONTROL CENTERS IN THE BRAIN INFLUENCE THE BODY'S GLANDS TO PRODUCE AND RELEASE THE HORMONES THAT ARE NEEDED TO MAINTAIN THIS VITAL BALANCE.

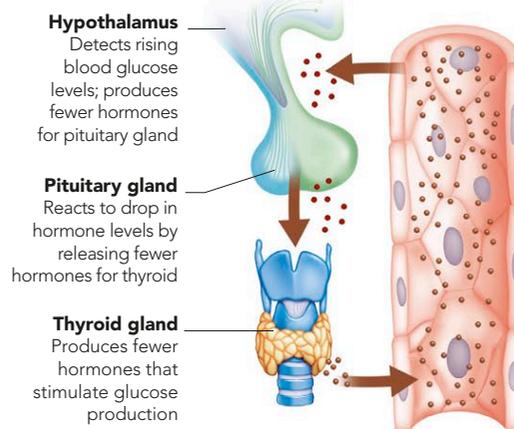
HORMONE SYNTHESIS AND CONTROL

Glands are organs that respond to imbalances in the body in order to regulate internal activities, such as the absorption of nutrients, and influence activities such as the intake of food or water. They react by increasing or decreasing their production of hormones, which then travel to a target organ, where they lock onto specialized receptors on the surface of cells. This binding triggers a physiological change that restores homeostasis. The hypothalamus is the crucial link between the nervous system and endocrine system, releasing hormones that, in turn, trigger the pituitary gland to either stop or start secreting its hormones.

HORMONES RELEASED BY THE PITUITARY GLAND	
Melanocyte-stimulating hormone (MSH)	Stimulates the production and release of melanin, the determinant of skin and hair color
Adrenocorticotropic hormone (ACTH)	Triggers the adrenals to produce steroid hormones that control stress response
Thyroid-stimulating hormone (TSH)	Increases the activity of thyroid gland, which controls metabolism
Growth hormone (GH)	Acts on entire body, but especially important for growth and development in children
Luteinizing and follicle-stimulating hormone	Triggers the sex glands in males and females to make their own hormones
Oxytocin	Causes contractions during labor; also involved in the release of milk from the mammary glands
Prolactin	Stimulates the production of milk from the mammary glands
Antidiuretic hormone (ADH)	Controls amount of water removed from the blood by microfilters in the kidneys

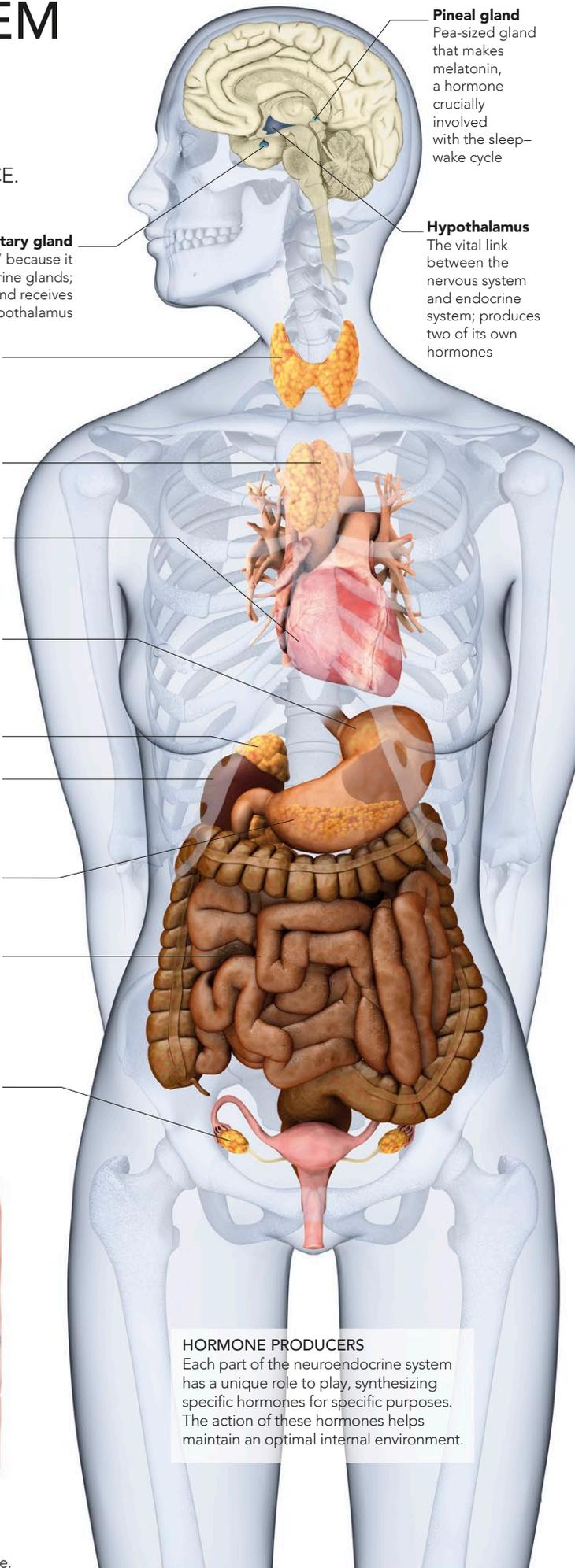
FEEDBACK MECHANISMS

Imbalances in the body are detected and corrected using feedback mechanisms, or loops. Levels of a hormone within the bloodstream are gauged and the information is sent to the control unit in charge of that hormone, which in most cases is the hypothalamus-pituitary unit. If the level of a hormone is high, the control unit responds by reducing the production of that hormone to achieve balance. If the level is low, the control unit initiates an increase in production. Feedback mechanisms are also used to trigger rare homeostatic functions, such as contractions during labor.



NEGATIVE FEEDBACK

In response to a rise in blood glucose, the hypothalamus triggers a chain reaction of reduced hormone production that results in a fall in glucose levels, which restores balance.



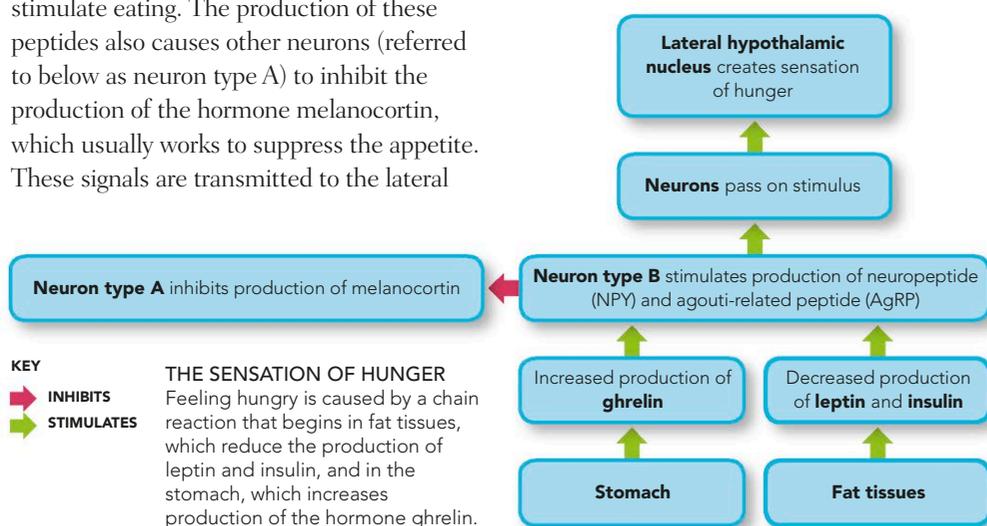
HORMONE PRODUCERS

Each part of the neuroendocrine system has a unique role to play, synthesizing specific hormones for specific purposes. The action of these hormones helps maintain an optimal internal environment.

HUNGER

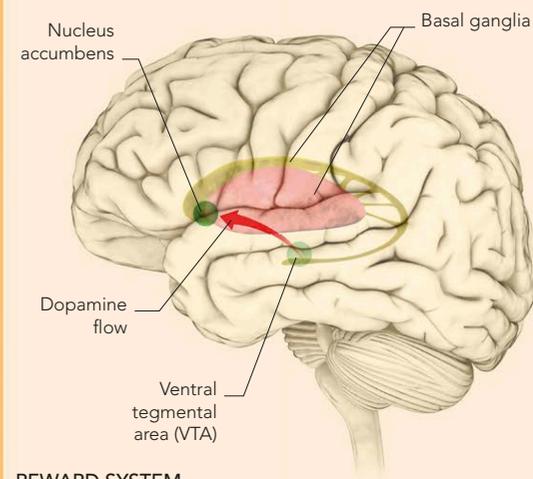
The body maintains its weight at a set point by using hormones to trigger the sensations of either hunger or satiety. To stimulate the appetite, the stomach produces the hormone ghrelin, while fat tissues decrease their production of leptin and insulin. These changes signal to specific neurons (referred to as neuron type B on the chart below) to start producing more neuropeptide (NPY) and agouti-related peptide (AgRP), which stimulate eating. The production of these peptides also causes other neurons (referred to below as neuron type A) to inhibit the production of the hormone melanocortin, which usually works to suppress the appetite. These signals are transmitted to the lateral

hypothalamic nucleus (via other neurons), which generates the sensation of hunger. To suppress the appetite, the body's fat tissues increase production of leptin and insulin. These hormones signal to neuron type B to inhibit production of NPY and AgRP. At the same time, the increased leptin and insulin trigger neuron type A to produce melanocortin. These signals reach the ventromedial nucleus in the hypothalamus, which creates the feeling of satiety.



SUGAR ADDICTION

As a "reward" for performing functions essential for the survival of both the individual and the species, such as eating or reproducing, the brain releases opiates, which create sensations of pleasure. Sugar-rich diets generate heightened reward signals, so that the more sugar you have, the more you want. This can override self-control mechanisms and lead to addiction.

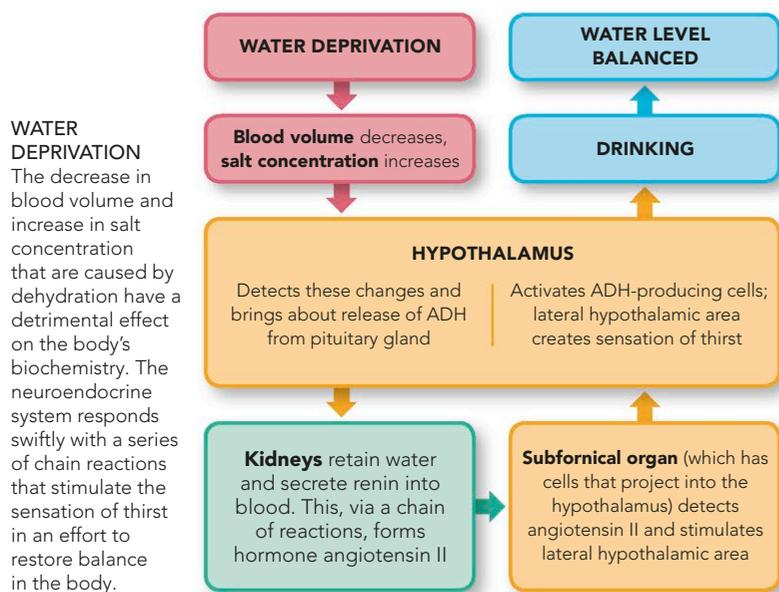


REWARD SYSTEM

The VTA in the midbrain processes information about how well various needs are being met and transfers this data to the nucleus accumbens in the basal ganglia, via the neurotransmitter dopamine. The more dopamine, the greater the pleasure, and the more likely the action will be repeated in the future.

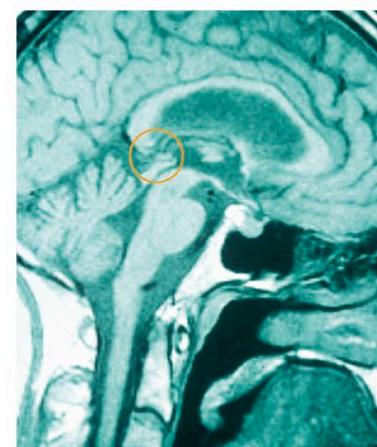
THIRST

When the body's water levels fall, salt concentration increases and blood volume decreases. Pressure receptors in the cardiovascular system and salt-concentration-sensitive cells in the hypothalamus detect these changes. In response, the pituitary gland releases antidiuretic hormone (ADH), which acts on the kidneys to retain water and produce less urine. The kidneys secrete the enzyme renin into the blood which, through a series of reactions, forms the hormone angiotensin II. This is detected by the subfornical organ, which is connected to the hypothalamus, which in turn activates more ADH-producing cells and creates the sensation of thirst, leading to drinking.



SLEEP-WAKE CYCLES

The suprachiasmatic nucleus (SCN) in the hypothalamus plays a key role in sleep-wake cycles. Light levels are sensed by the retina, and this information is relayed to the SCN, which then sends a signal to the pineal gland. This triggers the release of melatonin, the hormone that tells the body when to sleep. At this point, the brain becomes less alert and fatigue starts to take over. When melatonin levels fall in response to increased light, the waking part of the cycle begins.

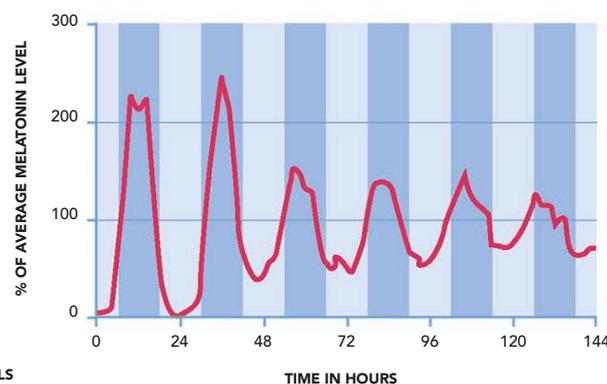


PINEAL GLAND

The circle on this lateral MRI scan of the brain pinpoints the pineal gland, a pea-sized gland located beneath the thalamus. It is responsible for the secretion of melatonin.

MELATONIN
 Falling levels of light trigger the production of melatonin, which forms a link between the external environment and the brain's sleep-wake cycles.

KEY
 NIGHT (Dark blue)
 DAY (Light blue)
 MELATONIN LEVELS (Red line)



PLANNING A MOVEMENT

MOVEMENTS MAY BE PLANNED EITHER CONSCIOUSLY OR UNCONSCIOUSLY, AND BOTH TYPES MAY PRODUCE COMPLEX ACTIONS THAT LOOK VERY MUCH ALIKE. ALL PLANNED MOVEMENTS INVOLVE THE BRAIN, ALTHOUGH CONSCIOUS MOVEMENTS ARE HATCHED IN A DIFFERENT AREA FROM UNCONSCIOUS MOVEMENTS. THE MORE SKILLED WE ARE AT MAKING A PARTICULAR MOVEMENT, THE LESS LIKELY IT IS TO REQUIRE CONSCIOUS PLANNING.

CONSCIOUS AND UNCONSCIOUS MOVEMENT

Many of our actions are conscious—thinking about picking up an object, for example, and then actually picking it up. However, there are many actions that take place without our awareness, such as blinking. Some unconscious actions may be triggered directly by environmental stimuli—the sight of food may trigger an automatic reaching movement, for example. Whether a complex movement is conscious or unconscious depends largely on the individual’s level of skill. As an action becomes increasingly familiar, it can become “automatic.” However, these movements can also be performed consciously if the individual turns attention to them.



COMPLICATED ACTIONS

Even advanced movements, such as juggling and unicycling simultaneously, can be performed unconsciously.

SKILL AND FAMILIARITY

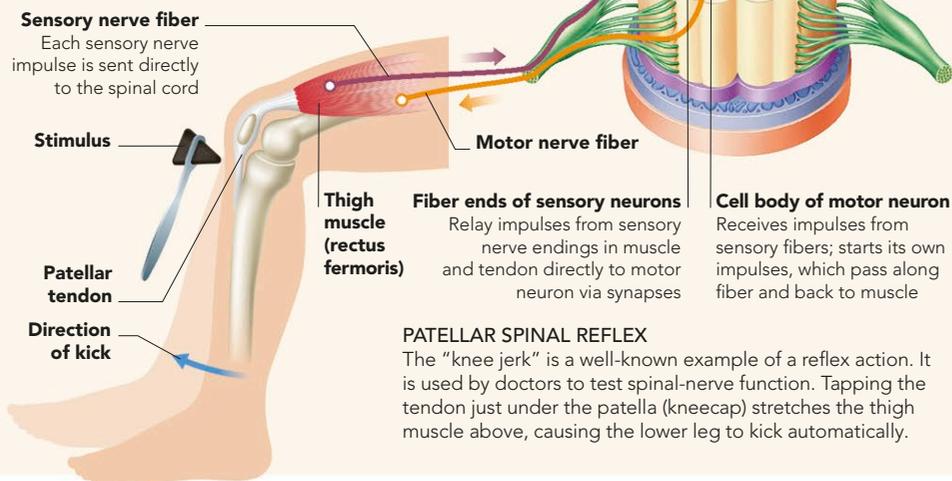
The chart to the left shows that a skilled driver on a familiar route will carry out all of the individual actions involved with turning the car unconsciously, while a learner will be conscious of all the actions. A skilled driver on an unfamiliar route will only be conscious of looking for the turn.

SKILLED DRIVER familiar route	SKILLED DRIVER unfamiliar route	LEARNING DRIVER
LOOKING FOR THE TURN	LOOKING FOR THE TURN	LOOKING FOR THE TURN
CHECK MIRROR	CHECK MIRROR	CHECK MIRROR
CHANGE GEAR	CHANGE GEAR	CHANGE GEAR
TURN WHEEL	TURN WHEEL	TURN WHEEL

KEY
 CONSCIOUS
 UNCONSCIOUS

REFLEX ACTIONS

Reflex actions are motor actions that are programmed into the spinal cord. The brain is not involved, and the actions cannot be controlled consciously. Most reflex actions protect the body by producing rapid reactions to escape from potentially damaging stimuli. In each case, the stimulus causes sensory nerve endings to fire; these signals pass through the nerve fibers to the spine, and trigger firing in the adjacent motor neurons, which then feed back to the relevant area and cause it to move.



COMPLEX PLANNING

Some actions require lengthy conscious deliberation. If a person is highly skilled at doing something—a professional golfer putting a ball, for example—the execution of the action will be relegated to unconscious areas of the brain. This “frees up” higher cognitive regions to concentrate on planning where to strike the ball and how hard to strike it.

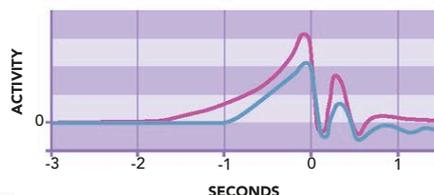


BRAIN AREAS AND MOVEMENTS

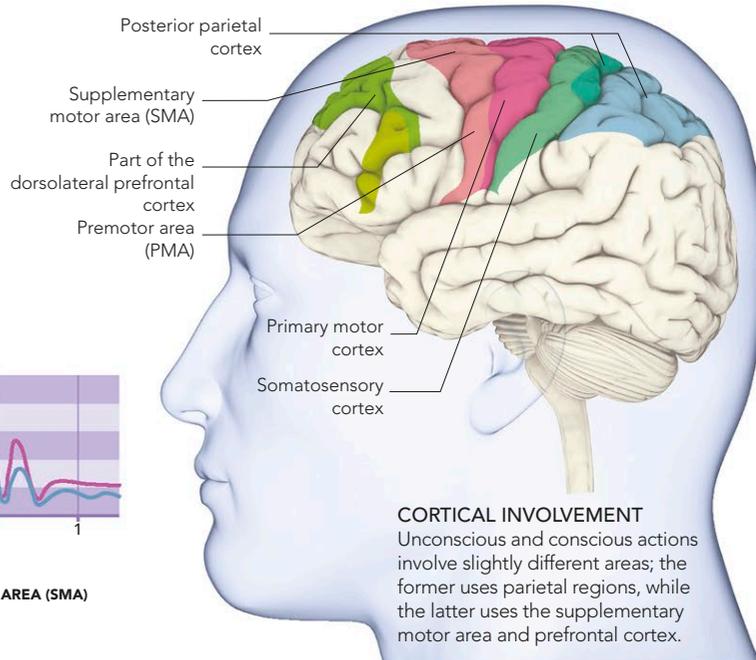
Both conscious and unconscious actions involve the primary motor cortex, which sends the “go” signals that contract the muscles (via the spinal cord and motor nerves). However, while unconscious movements are planned by areas in the parietal lobe, conscious actions involve “higher” frontal brain areas, including the premotor and supplementary motor cortices. They may also involve prefrontal areas, such as the dorsolateral prefrontal cortex, where actions are consciously assessed. It may feel as though conscious actions result from a decision. In fact, unconscious areas of the brain plan and start to execute movements before we consciously decide to do them. The “decision” may, therefore, merely be the conscious recognition of what the unconscious mind is planning to do.

READINESS POTENTIAL

Unconscious activity in the SMA and PMA starts two seconds before an action. The “decision” to act occurs only a fraction of a second before the action.



KEY
 — SUPPLEMENTARY MOTOR AREA (SMA)
 — PREMOTOR AREA (PMA)



CORTICAL INVOLVEMENT

Unconscious and conscious actions involve slightly different areas; the former uses parietal regions, while the latter uses the supplementary motor area and prefrontal cortex.

THE BASAL GANGLIA

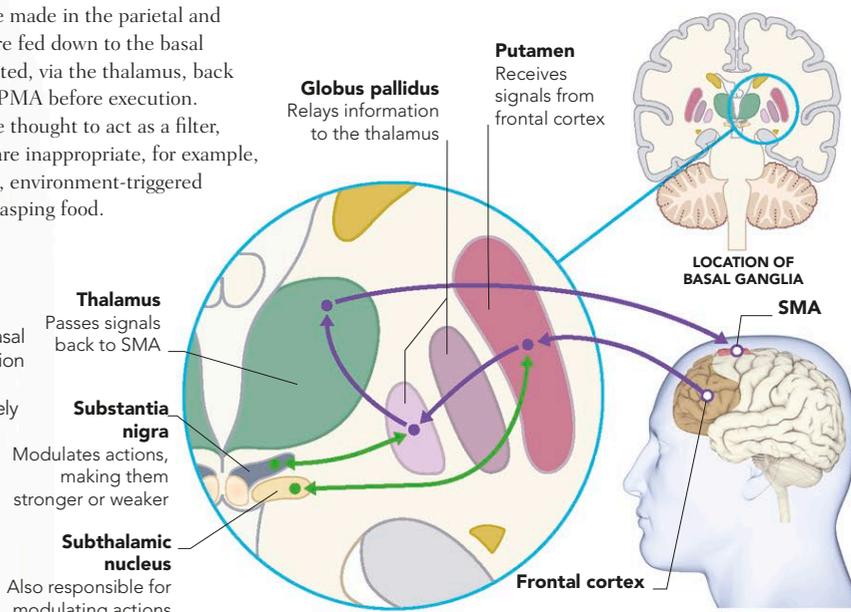
Action plans that are made in the parietal and frontal brain areas are fed down to the basal ganglia and then routed, via the thalamus, back up to the SMA and PMA before execution. The basal ganglia are thought to act as a filter, blocking plans that are inappropriate, for example, inhibiting automatic, environment-triggered responses such as grasping food.

RESPONSE CONTROL

As action plans are routed around the basal ganglia, the information is made more or less potent—and thus likely to be executed—by the action of various neurotransmitters.

KEY

— BASAL GANGLIA LOOP
 — MODULATING CIRCUITS



THE CEREBELLUM

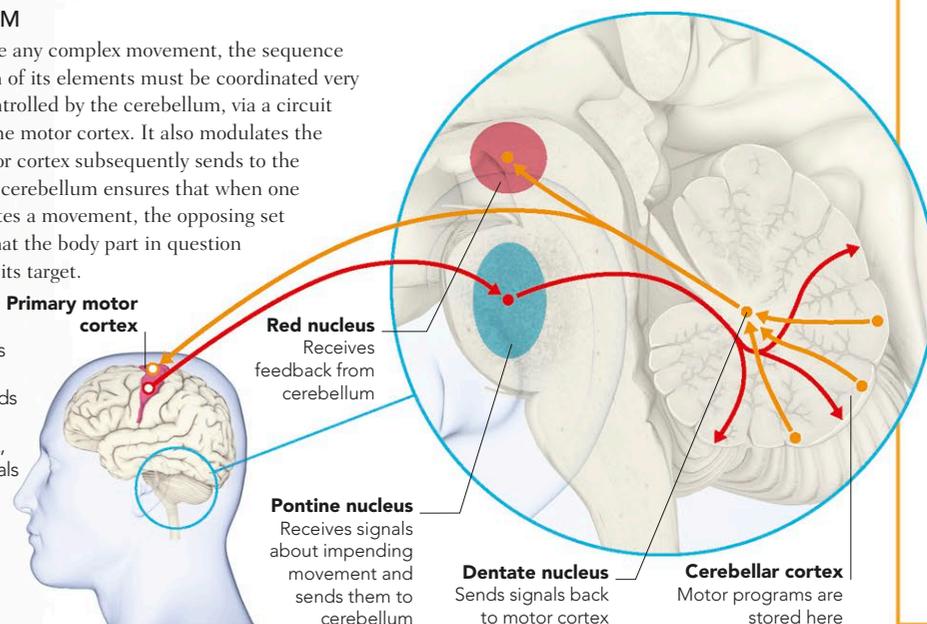
For the body to make any complex movement, the sequence and duration of each of its elements must be coordinated very precisely. This is controlled by the cerebellum, via a circuit that connects it to the motor cortex. It also modulates the signals that the motor cortex subsequently sends to the motor neurons. The cerebellum ensures that when one set of muscles initiates a movement, the opposing set acts as a brake, so that the body part in question arrives accurately at its target.

PRECISE TIMING

The cerebellar circuits include a system that measures time. It feeds its calculations to the primary motor cortex, which sends the signals to the muscles.

KEY

— SIGNALS FROM CEREBELLUM
 — SIGNALS TO CEREBELLUM

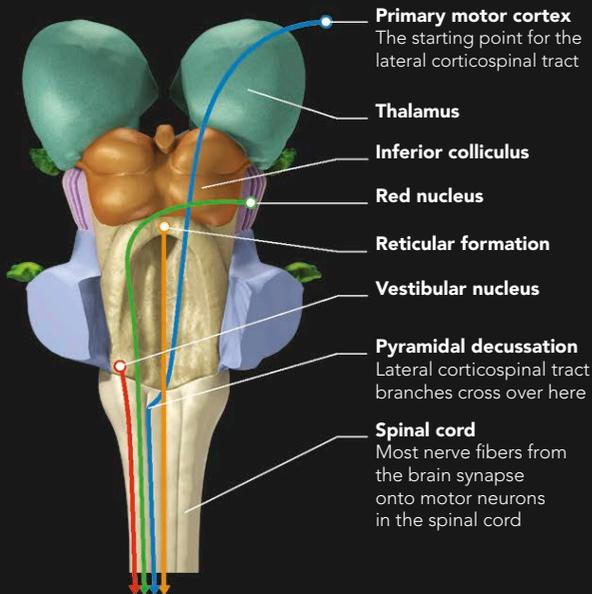


EXECUTING A MOVEMENT

ONCE A MOVEMENT HAS BEEN PLANNED, THE BRAIN AREAS RESPONSIBLE SEND SIGNALS TO THE MUSCLES TO EXECUTE THE ACTION. SOME OF THESE SIGNALS ARE SENT FIRST TO THE MOTOR CORTEX, AND THEN ONWARD THROUGH THE SPINAL CORD. OTHERS TRAVEL BY MORE DIRECT ROUTES. MOVEMENT OCCURS WHEN THE SIGNALS REACH THE MUSCLE FIBERS, CAUSING THEM TO CONTRACT.

SPINAL TRACTS

Action plans generated in the supplementary, premotor, and parietal cortices are forwarded to the motor cortex for execution. The motor cortex is made up of about one million neurons, which send long axons down the spinal cord. These are bundled together, along with axons that come directly from the somatosensory cortex, to form the lateral corticospinal tract. Just before entering the spinal cord, the nerves from each hemisphere of the brain separate and cross over, so the fibers from the left hemisphere of the cortex go down the right side of the spinal cord, and vice versa. The rubrospinal tract originates from the red nucleus in the midbrain, and helps to produce fine movements. The vestibulospinal and reticulospinal tracts start lower down in the brainstem and help control balance and orientation.



- KEY**
- VESTIBULOSPINAL TRACT
 - RUBROSPINAL TRACT
 - LATERAL CORTICOSPINAL TRACT
 - RETICULOSPINAL TRACT

LIMB CONTROL
The lateral corticospinal tract is the only spinal tract to originate in the cerebral cortex and is mostly responsible for controlling limb movements.



BALANCING ACT
The reticulospinal and vestibulospinal tracts help control balance and orientation, and neutralize the effects of gravity.

SPINE TO MUSCLE

The axons of the motor neurons, which receive signals from the spinal tracts, emerge from between the vertebrae and travel to the muscles. The nerve endings infiltrate the muscle fibers at neuromuscular junctions, and when they fire they release the neurotransmitter acetylcholine. This diffuses across the narrow “synaptic cleft” connecting the muscle to the nerve and binds to acetylcholine receptors in the muscle cell membrane, which, by a series of reactions, makes the specific muscle contract. Muscles required to carry out fine movements have correspondingly higher numbers of neurons than those required to perform gross movements.

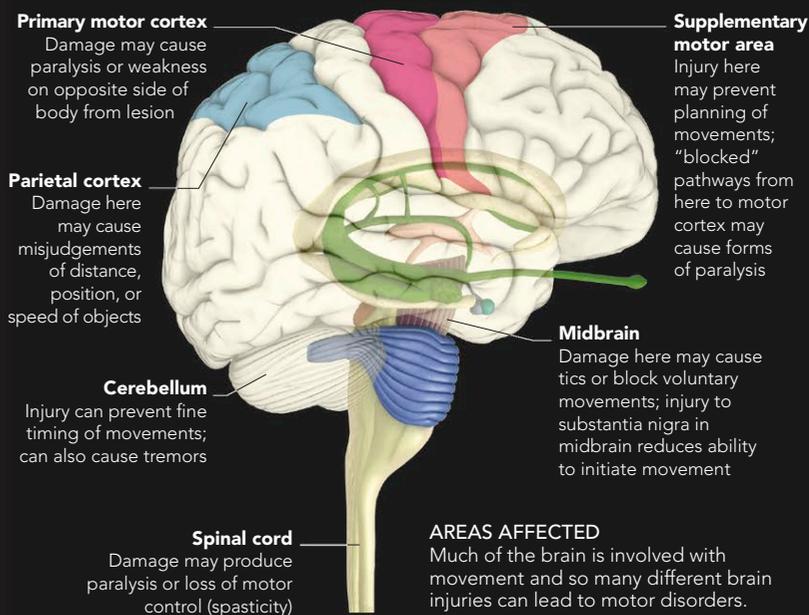


NEUROMUSCULAR JUNCTION
When stimulated by a motor nerve, electrical changes in the muscle cause the release of calcium ions inside the muscle. This causes the filaments of the muscle to slide against each other and contract.

PRECISE SEQUENCE
After receiving the order to move from the primary motor cortex, a rapid, precisely timed sequence of motor-neuron firings causes specific muscles to contract.

MOTOR DISORDERS

Motor disorders can be divided into two principal groups: hyperkinesia (overactivity) and hypokinesia (too little movement). The former includes a wide range of motor disorders, from involuntary, slow shaking of various body parts to tics, which are uncontrollable, rapid, disjointed movements and/or sounds. Sudden, shocklike muscle contractions are symptoms of myoclonus, while quick, random, usually jerky limb movements are caused by chorea and ballism. Hyperkinesia disorders include: general slowness of movement (bradykinesia); “freezing” or inability to begin a movement or involuntary arrest of a movement; rigidity—an increase in muscle tension when a limb encounters force; and postural instability, which is the loss of ability to maintain an upright posture.

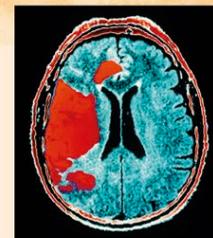


MOTOR RECOVERY

Movement disorders may result from damage to many different areas of the brain, and it is very common for one of these to follow a stroke. Damage to the motor cortex, for example, may cause whole or partial paralysis of the opposite side of the body, and strokes in subcortical areas may lead to loss of control of voluntary movements. The affected neural pathways can, however, rebuild to a certain extent, reducing the long-term effect of the damage. Studies show that damaged midbrain-cortical motor pathways form new connections in as little as three months after remedial therapy.

STROKE REHABILITATION
The neural pathways damaged by a stroke do rebuild themselves to a limited degree. Physical therapy encourages the rewiring of motor circuits, and recovery is often directly related to the intensity of the therapy.

STROKE
This CT scan shows the extent of internal bleeding in the brain caused by a stroke.



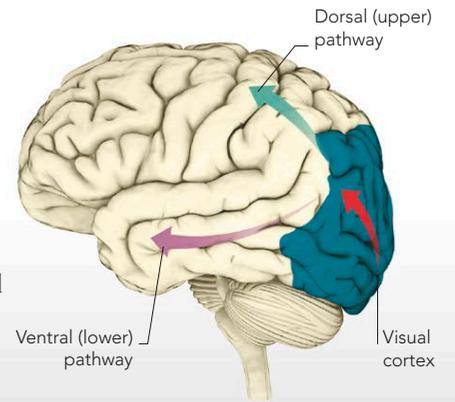
UNCONSCIOUS ACTION

THE BRAIN REGISTERS EVENTS VIA THE SENSE ORGANS ALMOST IMMEDIATELY, BUT IT TAKES UP TO HALF A SECOND TO BECOME CONSCIOUS OF THEM. IN ORDER TO GENERATE EFFECTIVE RESPONSES IN A FAST-CHANGING ENVIRONMENT, THE BRAIN MUST PLAN AND EXECUTE MOMENT-BY-MOMENT ACTIONS UNCONSCIOUSLY.

REACTION PATHWAYS

It takes up to 400 milliseconds (ms) for the brain to process incoming information to the stage where it may become conscious. It takes a similar length of time to prepare the body for action. So if we waited to be conscious of a sight or sound

before starting to respond to it, our behavior would lag almost a second behind the events to which we are responding. By the time we leapt out of the path of a speeding car, it is likely to have run us over. The brain speeds up our physical responses by fast-tracking sensory information to the motor-



DORSAL AND VENTRAL ROUTES
Visual stimuli are processed along parallel pathways. The unconscious dorsal route generates physical responses while the ventral route creates conscious perception.

RETURNING A SERVE

Professional tennis players can plan and initiate the complex moves required to return a fast service before they are consciously aware that the ball is on its way. Unlike novice players, they do not have to think consciously about each muscle movement because practice has turned the relevant action sequences into automated motor programs that are stored and run unconsciously. Familiarity with their opponents' body language also allows them to make well-informed unconscious predictions about where the ball will land.

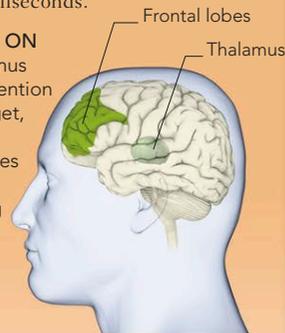
EVENTS IN RECEIVER'S BRAIN

0ms Attention

The player's brain prepares for action by focusing attention on his opponent. This prevents the brain from responding to irrelevant stimuli and amplifies information coming from the part of the visual field containing the target of attention. If the player is familiar with the opponent's playing style, his brain will register the movements made by the opponent as he serves and compare them with previous observations to help predict where the ball will land. Attention to such cues may speed up reactions by 20–30 milliseconds.

LOCKING ON

The thalamus directs attention to the target, while the frontal lobes inhibit distracting thoughts.

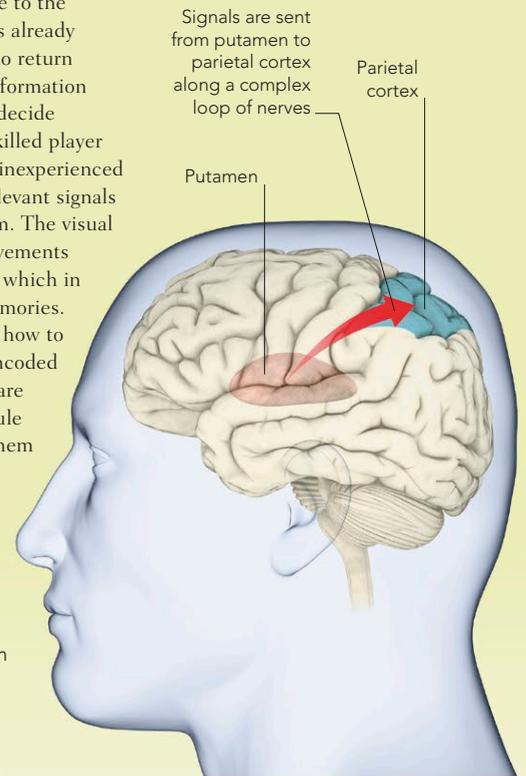


70ms Body memory

The ball is not yet consciously visible to the player, but unconsciously his brain is already planning the actions he must make to return it. At this stage he is mainly using information about his opponent's movements to decide how his own body should move. A skilled player processes fewer visual cues than an inexperienced one because the brain identifies irrelevant signals at a very early stage and ignores them. The visual information from his opponent's movements activates the player's parietal cortex, which in turn calls up relevant procedural memories. These are learned actions—such as how to return a serve—that have become encoded as automatic motor programs. They are stored in an unconscious brain module called the putamen, which replays them as the situation demands.

MOVEMENT MEMORY

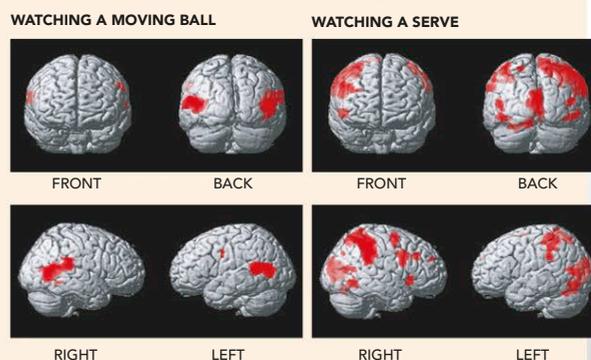
Part of the basal ganglia, the putamen acts as a store of memories about deeply ingrained habits of movement. Signals from the putamen are passed to the parietal cortex.



planning areas along an unconscious pathway. A visual stimulus such as a moving object prompts neural activity that works out where it is in relation to the body. Various parts of the occipital and parietal cortex, between them, calculate the object's shape, size, relative motion, and trajectory. This information is then brought together and used to form an action plan, which might involve hitting (swatting a fly, for example), avoidance (ducking or jumping out of the way of a missile), or grabbing (a falling fruit or a stumbling child). The chosen response is largely learned; for example, a skilled athlete is likely to catch or hit a speeding ball while an unpracticed player might just duck it.

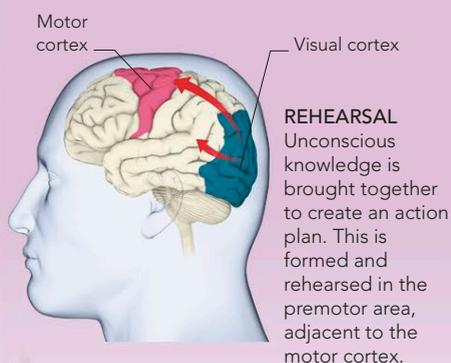
TENNIS PLAYERS UNDER OBSERVATION

Tennis players watching a video of another player serving a ball imagine themselves making the action. These fMRI images show that watching a moving ball (left) activates areas of the brain that track visual objects, but watching someone serve a ball (right) activates visual areas plus large parts of the parietal cortex. The additional activation shows the viewer's brain is "acting out" the moves seen in the video. This information helps the viewer predict where the ball will go.



250ms Action plan

The receiving player's brain brings together the information that has been registered so far to construct a response to the fast-approaching ball. The plan is informed by information gathered from the opponent's body movements, the (still unconscious) knowledge of the ball's speed and trajectory, and the procedural memories triggered by these stimuli. The plan is held in the premotor area, which lies just in front of the motor cortex. This is like a rehearsal stage, allowing action to be played out as a pattern of neuronal activity without affecting the muscles.

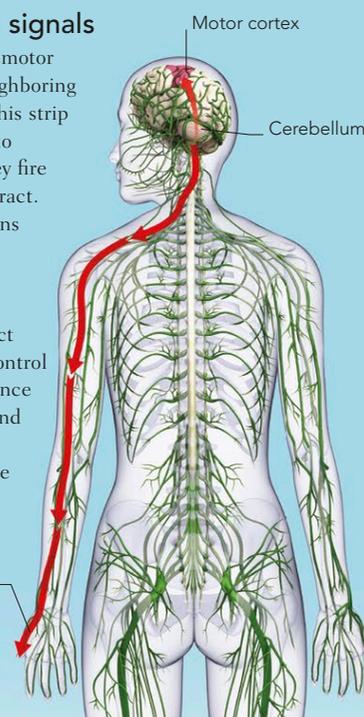


355ms Sending signals

The action plan held in the premotor cortex is transmitted to the neighboring motor cortex. The neurons in this strip of brain connect via the spine to skeletal muscles, and when they fire they cause the muscles to contract. In this case, the firing of neurons about halfway down the right side of the motor strip move the player's left arm and hand to position the racket to connect with the ball. Other neurons control the rest of the body. The sequence in which these neurons fire—and therefore the sequence of limb movement—is controlled by the cerebellum.

Signal from motor cortex travels to player's hand

INSTRUCTION TO MOVE
Neural signals from the motor cortex are sent along the spine, causing muscles to contract and leading to overt movement.



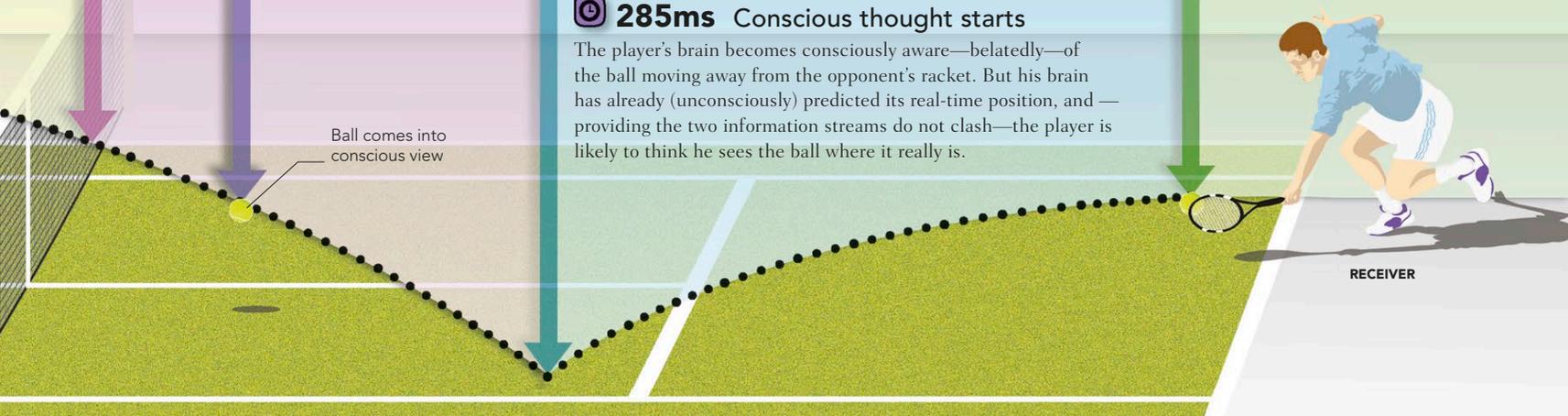
500ms Conscious act

If the player's conscious perception of the ball's trajectory differs markedly from his earlier, unconscious prediction he may veto the earlier action plan and start to construct an alternative, or try to adjust the current plan to take into account the new information. It takes another 200–300ms, however, to incorporate the new, conscious information into a revised action plan and by then the ball has traveled too far for any player to be able to return it.

The situation is similar to the one that occurs when a person steps forward onto what the brain predicted was flat ground, but which is actually a downward step. The resultant physical catastrophe, in both cases, triggers a further cascade of signals that may generate a wide range of emotions, including anger, embarrassment, and a feeling of failure.

285ms Conscious thought starts

The player's brain becomes consciously aware—belatedly—of the ball moving away from the opponent's racket. But his brain has already (unconsciously) predicted its real-time position, and—providing the two information streams do not clash—the player is likely to think he sees the ball where it really is.



MIRROR NEURONS

CERTAIN NEURONS ARE ACTIVATED WHEN YOU MOVE, AND ALSO WHEN YOU SEE SOMEONE ELSE MOVING. THIS MEANS WE UNCONSCIOUSLY MIMIC THE ACTIONS OF OTHERS, AND THUS SHARE, TO SOME EXTENT, THEIR EXPERIENCE. MIRROR NEURONS ALSO ALLOW US TO KNOW WHAT ANOTHER PERSON IS FEELING, WITHOUT HAVING TO THINK ABOUT IT. THESE FINDINGS ARE AMONG THE MOST SIGNIFICANT NEUROSCIENTIFIC DISCOVERIES IN RECENT YEARS.



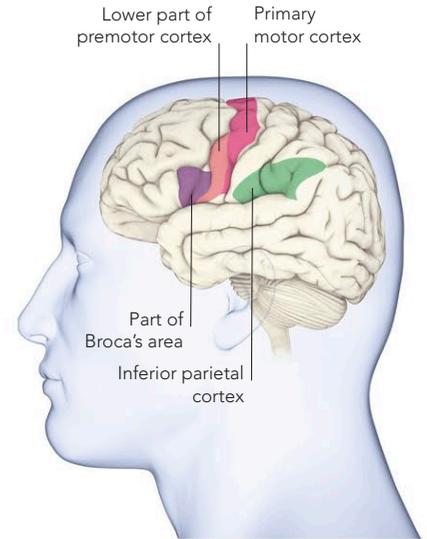
WHAT ARE THEY?

Mirror neurons were first discovered in the motor-planning area in the brains of macaques (a species of monkey) and subsequent brain-imaging studies suggest that they exist in humans too. The human mirror system seems to be broader in scope than that of monkeys, in that mirror neurons exist not only in movement areas, but also in areas concerned with emotions, sensations, and even intentions. They

HOW THEY WERE DISCOVERED

Mirror neurons were discovered in a monkey whose brain was wired up to show which nerve cells lit up as it reached out to grasp food. When laboratory staff made the same movement while the monkeys sat and watched, the same neurons lit up.

provide people with immediate knowledge of what is going on in another's mind; this ability to know what another person is feeling or doing is thought to be the basis of mimicry.

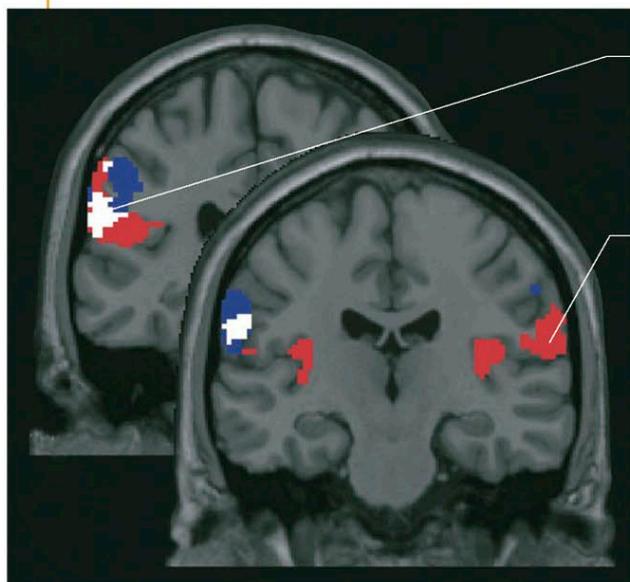


WHERE THEY ARE

In humans, mirror neurons seem to extend into the areas of the frontal lobe that are concerned with intentions, such as part of the premotor cortex. They are also found in the parietal lobe, which is involved with sensations. However, the full extent of these neurons is still being researched.

MIRRORING TOUCH

Mirror neurons also seem to operate in the somatosensory cortex—the area of the brain that registers touch. In one study, subjects' brains were scanned, first while their leg was brushed, and then while they watched a video of someone else's leg being touched. Activity in their brains revealed that some parts of the somatosensory areas are activated only by direct touch and others are activated by the sight of another being touched. A third group of neurons, however, are activated both by direct touch and by seeing others being touched. These mirror neurons—shown in white on the scans below—were limited to the left hemisphere in this study, though in other experiments they have been detected in both hemispheres.



Somatosensory areas in left hemisphere activated by both touch and vision-of-touch

Activity only arises in the right hemisphere from direct touch, but mirror neurons have been detected here in similar experiments

ACTIVATED AREAS

These MRI scans are coronal sections taken from the same brain. They show the areas stimulated by touch, watching another being touched, and the overlap between the two.

- KEY**
- AREAS ACTIVATED BY TOUCH
 - AREAS ACTIVATED BY VISION-OF-TOUCH
 - AREAS ACTIVATED BY BOTH

MIRRORING SPEECH

Mirror neurons may help people communicate by “syncing” their brains when they talk together. Partners in a conversation unconsciously imitate one another, adopting a similar rate of speaking and the same sort of grammatical structures. This helps one person predict what the other is going to say next, making communication quicker and smoother. Speech is combined with body movements and facial expressions to convey full meaning, and these tiny movements amplify the perception of another's voice. Watching a speaker's face is equivalent in effect to turning up the volume by 15 decibels.



BODY LANGUAGE

As well as mirroring speech patterns and speed, people unconsciously adapt their body language to match whoever they are speaking to. Partners in a conversation focus on each other's faces, picking up minute movements that help express meaning.

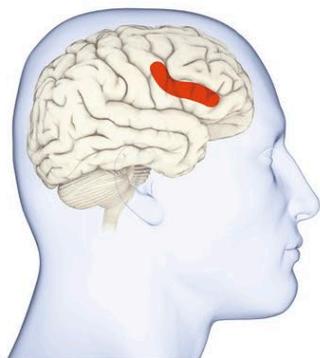


KNOWING HOW IT FEELS

To mirror another's actions, the brain must "know" how it feels to do it. For example, to mirror expert dance moves, you would have to have some idea of how to go about doing them, even if you could not reproduce them perfectly.

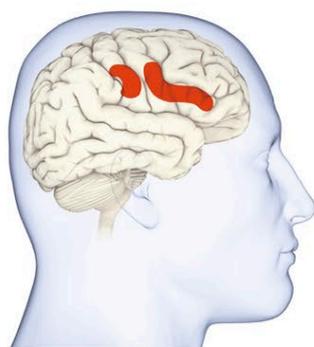
MIRRORING MOVEMENT

Recent studies have found that a certain, as yet unknown, proportion of mirror neurons are active both when moving and when watching movement. Neurons in the premotor cortex concerned with planning to move the legs are activated when you watch a person running, for example. In other words, when you see someone doing something, in your brain you do it too. However, in order to mirror another's action, the sight of the action must "resonate" with a motor program that the brain has already learned.



WATCHING CHEWING

Simply watching another person chewing shows activity in both the premotor cortex and the part of the primary motor cortex concerned with mouth and jaw movements.



ACTING ON AN OBJECT

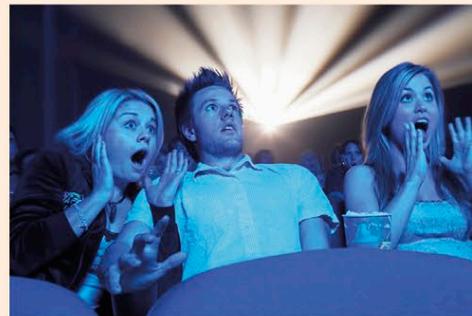
When the movement involves acting on an object—biting an apple, for example, rather than just simply chewing—areas of the parietal cortex also light up.

MIRRORING EMOTIONS

When one person sees another expressing an emotion, the areas of the brain that are associated with feeling that emotion are activated, making emotions transmittable. In one study, volunteers inhaled a disgusting smell, and later, watched a video of someone else smelling something and expressing disgust. Both produced neuronal activity in the area of the brain associated with feeling disgust. Emotion mirroring is thought to be the basis of empathy. Autistic people, who tend to lack empathy, have been found to show less mirror-neuron activity.

HORROR MOVIE

Seeing someone else looking frightened makes you feel scared yourself. Mirror neurons, therefore, help whip up emotion in audiences.



MIRRORING INTENTIONS

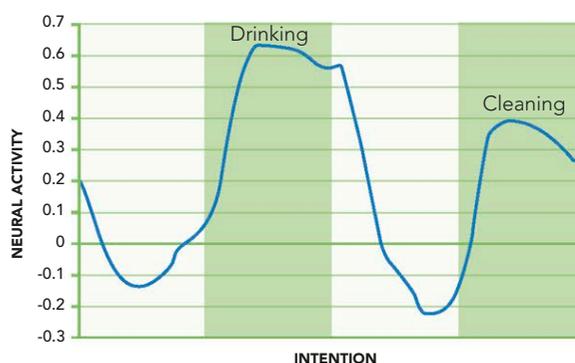
Two movements may be identical, but may signal very different things in different contexts. Human mirror neurons seem to take this into account. When one person sees another picking up a cup in order to drink from it, a different set of neurons are activated from those that light up at the sight of a person making the identical movement but in a context that suggests they are clearing the cup away. Hence, the observer's brain does not just generate a faint idea of what the other person is doing with their body, but also an echo of their intention

in doing it. This allows us to get a glimpse of another individual's plans and thought processes without consciously having to work it out.



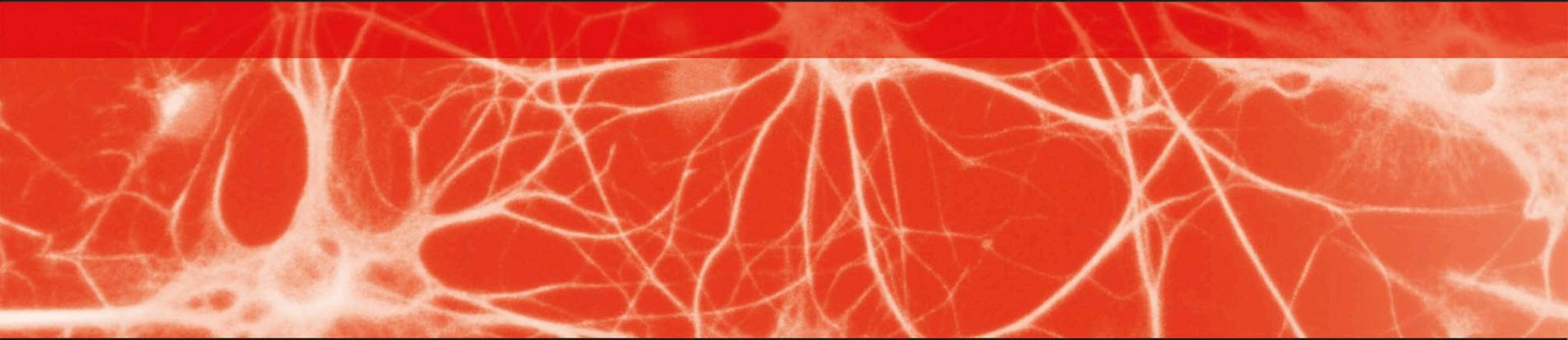
DRINKING AND CLEANING UP

The top image shows a table set for breakfast, while the image at the bottom shows the table after the meal has been finished. The action of grasping the cup can be exactly the same in both, but our brains take into account the difference in contexts and therefore we automatically "know" that each one signals a different intention.



LEVEL OF ACTIVITY

The increased activity associated with watching the intention to drink is thought to be because it is more commonly practiced than the intention to clear up.



EMOTIONS CAN BE THOUGHT OF AS BODY CHANGES THAT PROMPT US TO ACT. THEY HAVE EVOLVED TO GET US TO DO WHAT WE HAVE TO IN ORDER TO SURVIVE AND PASS OUR GENES ON TO THE NEXT GENERATION. TO REINFORCE THEIR EFFECTIVENESS, EMOTIONALLY TRIGGERED ACTIONS ARE ASSOCIATED WITH PLEASANT OR UNPLEASANT CONSCIOUS FEELINGS. EMOTIONS TEND TO BE SHORT-LIVED, LASTING A FEW HOURS AT MOST, BUT THEY CAN LEAD TO MORE PERSISTENT CONDITIONS CALLED MOODS.

EMOTIONS AND FEELINGS



THE EMOTIONAL BRAIN

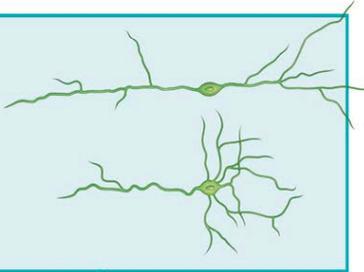
EMOTIONS MAY SEEM TO BE CONSCIOUS FEELINGS, BUT THEY ARE, IN FACT, “INNER MOTIONS”—PHYSIOLOGICAL RESPONSES TO STIMULI, DESIGNED TO PUSH US AWAY FROM DANGER AND TOWARD REWARD. EMOTIONS ARE GENERATED CONSTANTLY, BUT MUCH OF THE TIME WE ARE UNAWARE OF THEM.

ANATOMY OF EMOTION

Emotions are generated in the limbic system: a cluster of structures that lies beneath the cortex. The system evolved very early in mammalian history. In humans, it is closely connected with the more recently evolved cortical areas. The two-way traffic between the limbic system and the cortex allows emotions to be consciously felt and conscious thoughts to affect emotions. Each emotion is produced by a different network of brain modules, including the hypothalamus and pituitary gland; these control the hormones that produce physical reactions such as increased heart rate and muscle contraction.

Cingulate cortex

This part of the cortex is closest to the limbic system. Performing difficult tasks, or experiencing intense love, anger, or lust, causes activity in the anterior cingulate cortex (ACC) to increase; this area has been found to be active when mothers hear infants cry. The ACC contains unusual neurons called spindle cells (right), which may be particularly concerned with detecting how others feel and reacting to their emotions.



Stria terminalis

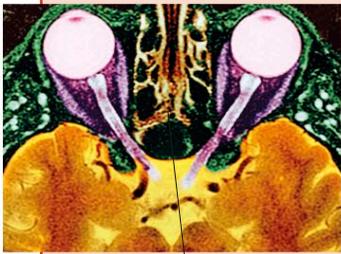
This is part of the network of pathways that link the amygdala to other parts of the brain. The stria terminalis plays a part in anxiety and stress responses. Cell density differs in men and women, and may play a part in gender identification—for example, transsexuals have been found to have a cell structure that matches the typical pattern of the sex to which they are changing.

Frontal cortex

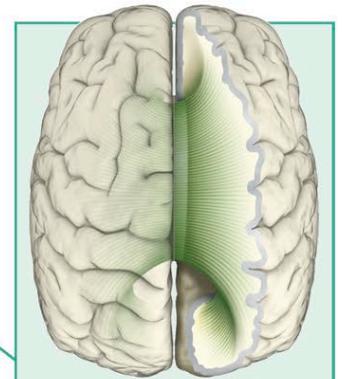
Information from the limbic system is fed to the frontal cortex to produce conscious feelings, while conscious knowledge about the environment is fed from the cortex back to the limbic system in a continuous loop. The effect of emotion on thought is stronger than vice versa, probably because there are more nerve tracts carrying signals up from the limbic system than passing signals back down.

Olfactory complex

The olfactory bulbs carry messages about smell straight to the limbic areas—unlike the pathways serving the other senses, which pass signals via the thalamus to the cortex for processing. This is why scents create such an intense, instant emotional response. The olfactory complex is thought to be the brain’s original “emotional” center, and probably evolved before sight and hearing.



Nasal bones

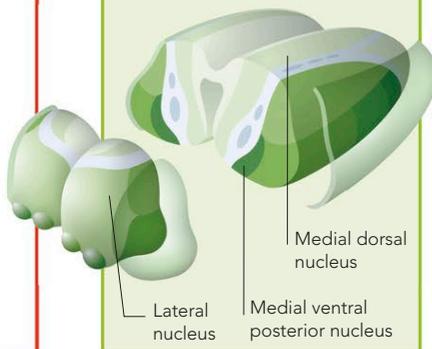


Corpus callosum

The corpus callosum (CC) plays an important role in transmitting emotions between the left and right hemispheres. Women, on average, have a greater density of fibers in the CC than men; this may account for some differences between the sexes in emotional response.

Thalamus

The thalamus acts as a distribution center for incoming information and is therefore involved in more or less every activity. However, some of the thalamic nuclei (dark green) have a particularly strong influence on emotions because they shunt emotionally salient stimuli to the appropriate limbic areas, such as the amygdala and the olfactory cortex, for further processing.

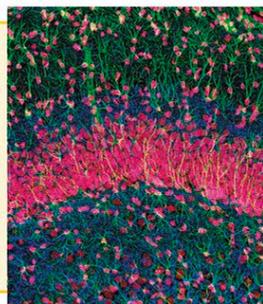


Lateral nucleus

Medial dorsal nucleus
Medial ventral posterior nucleus

Hippocampus

The hippocampus is mainly concerned with encoding and retrieving memories. Personal or “episodic” memories include an emotional component, so the hippocampus, by calling these up, creates a replay of emotions from the past. These may blend with current emotions, or they may override them—as when a sudden memory of something sad “blights” a happy moment.

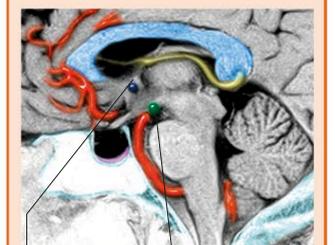


Amygdala

The amygdala is a tiny part of the brain that is most centrally and exclusively concerned with emotion. This area assesses both external and internal information for threat level and emotional significance (see opposite page).

Hypothalamus and mammillary body

The hypothalamus is a tiny part of the brain but it has complex and widespread effects. It acts as a hormonal signaler and transmitter, affecting bodily reactions to the environment and causing the sensations we feel as emotion. It also mediates the fear reaction made by the amygdala. The mammillary bodies, which connect to the hippocampus via the fornix, lie at the interface between memory and emotion.



Hypothalamus

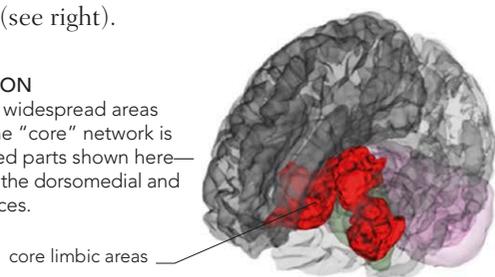
Mammillary body

AMYGDALA

The amygdala “tastes” all stimuli and signals other areas to produce appropriate emotional reactions. It contains distinct regions called nuclei, which generate different kinds of responses to fear. The central nucleus generates the fear response of freezing, while the basal nucleus generates the fear response of flight. The nuclei are affected by sex hormones, and are therefore different in men and women. Activation of the amygdala can be modulated by the hypothalamus (see right).

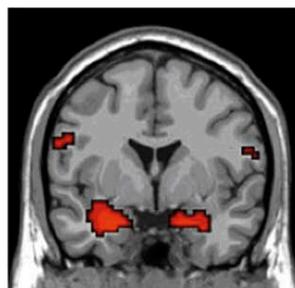
CORE OF EMOTION

Emotions engage widespread areas of the brain but the “core” network is centered on the red parts shown here—the amygdala and the dorsomedial and orbitofrontal cortices.

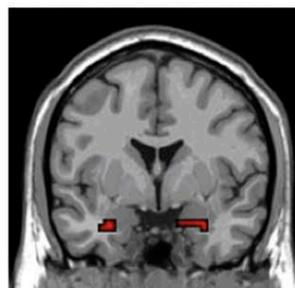


MEDIATING AMYGDALA RESPONSE

The amygdala is activated by frightening stimuli (left). However, the hormone oxytocin, secreted by the hippocampus, dampens down amygdala activity (right) and with it the feeling of fright.



FEAR RESPONSE



WITH OXYTOCIN

POSITIVE EMOTION

Limbic system structures next to the amygdala are involved in feelings of pleasure, mainly by reducing activity in the amygdala and in cortical areas concerned with anxiety. Anticipation and pleasure-seeking are influenced by the “reward” circuit. This acts on the hypothalamus and amygdala: it secretes dopamine, which provides anticipation and drive, and GABA, which inhibits neurons from firing.

PLEASURE AND THE BRAIN

Pleasurable stimuli, such as watching your soccer team score a goal, activate brain areas close to the limbic system.

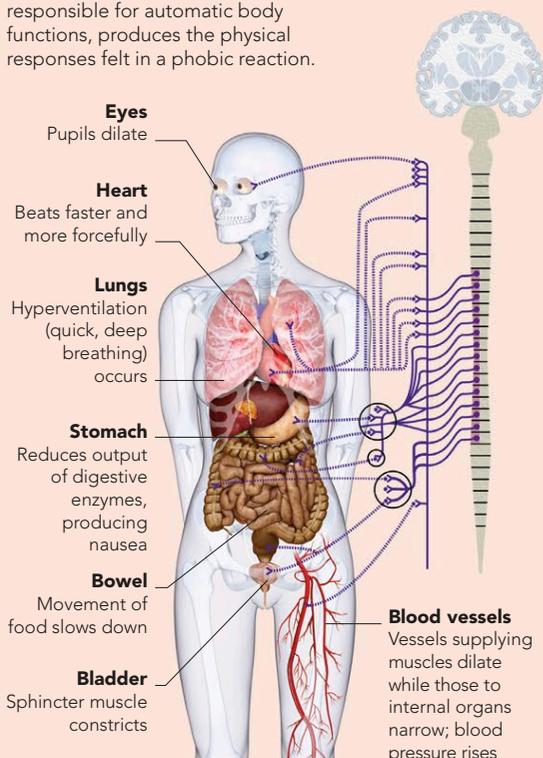


FEELING FEAR

The amygdala acts as a store for good and bad memories, especially emotional traumas. It is also “hard-wired” to fear certain stimuli, such as low-flying birds, spiders, and snakes. For a phobia to develop, however, there also needs to be an environmental trigger, such as a nasty encounter with a “hard-wired” stimulus, or the sight of someone else being frightened by it. It is often very hard to get rid of a phobia because the amygdala is not under conscious control. It can, however, “learn” to reduce its reaction to the stimulus.

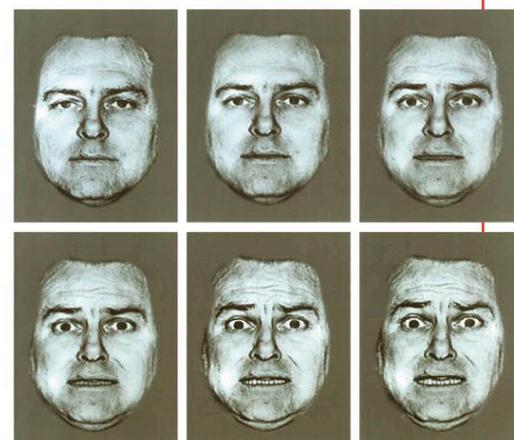
PANIC RESPONSES

The autonomic nervous system, responsible for automatic body functions, produces the physical responses felt in a phobic reaction.



UNCONSCIOUS EMOTION

We have evolved a conscious emotional system, but we retain the primitive, automatic responses at the heart of emotion. A frightening sight or sound, for example, registers in the amygdala before we are even conscious of it. While the sensory information is sent to the cortex to be made conscious, the amygdala sends messages to the hypothalamus, which triggers changes that ready the body for flight, fight, or appeasement. This “quick and dirty” route allows us to take instant action to save ourselves. When we “start” at a loud noise, then relax on realizing that it is harmless, we are experiencing both stages—unconscious reaction and conscious response.

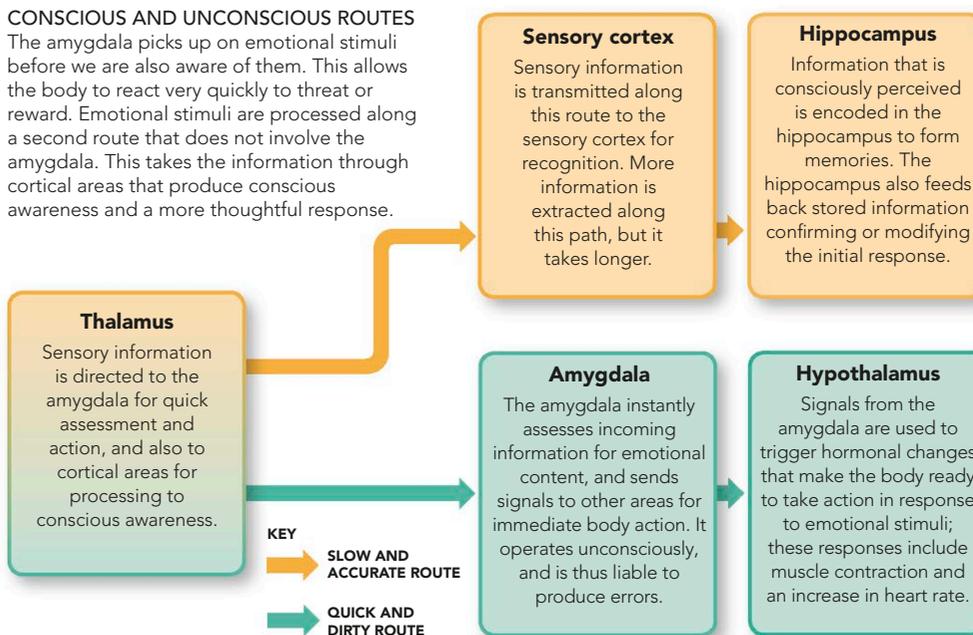


FACES OF FEAR

This series of images shows the onset of fear. The amygdala registers the emotional facial expressions of others, and produces a reaction before we even know we have seen them.

CONSCIOUS AND UNCONSCIOUS ROUTES

The amygdala picks up on emotional stimuli before we are also aware of them. This allows the body to react very quickly to threat or reward. Emotional stimuli are processed along a second route that does not involve the amygdala. This takes the information through cortical areas that produce conscious awareness and a more thoughtful response.



CONSCIOUS EMOTION

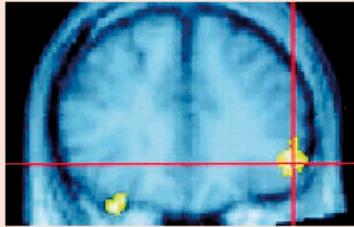
EMOTIONS ARE GENERATED IN THE LIMBIC SYSTEM, WHICH DOES NOT SUPPORT CONSCIOUSNESS ITSELF. INTENSE EMOTIONS CREATE “KNOCK-ON” ACTIVITY IN THE CORTEX, ESPECIALLY IN THE FRONTAL LOBES, WHICH WE EXPERIENCE AS A CONSCIOUS “FEELING” OR MOOD. SOMETIMES, AN EMOTION IS CLEARLY LINKED TO AN EXPERIENCE. AT OTHER TIMES, THE CAUSE IS NOT OBVIOUS, BUT BEING AWARE OF THE EMOTION MAKES IT EASIER TO UNDERSTAND WHAT IS HAPPENING TO US.

FEELING EMOTION

Emotions are primarily unconscious physical reactions to threat or opportunity. The sight of a snake, for example, automatically prepares the body for flight. In humans, emotions are consciously experienced as powerful “feelings” that give our lives meaning and value. The unconscious physiological component of emotion is generated in deep brain areas as signals that are then sent to the body to prepare it for action. Some signals travel upward to activate cortical areas, and this activation produces the feeling of emotion. The type of emotion experienced depends on which parts of the cortical areas are activated.

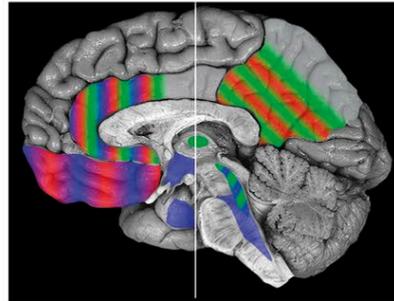
RIGHT HEMISPHERE

The right hemisphere generates more negative emotions than the left, and recognition and consciousness of sadness and fear depend on signals from the right hemisphere being received and processed by the left hemisphere. If the signals do not get through, a person may remain unconscious of their emotions, even though their behavior may be affected by them.



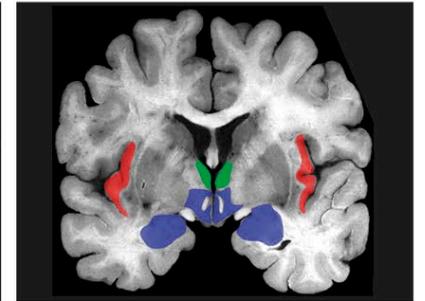
INCREASED ACTIVITY

This PET scan shows brain activity in a volunteer who is watching a person display various emotional facial expressions and gestures. These stimulate far more activity in the right frontal cortex (targeted) than in the same area in the left hemisphere.



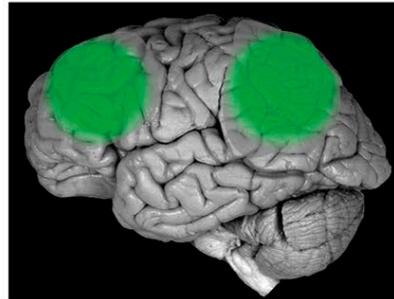
EMOTIONS INITIATED

Emotions arise in the amygdala, brainstem, and hypothalamus (blue). Conscious feelings (red) involve the orbitofrontal and cingulate cortex.



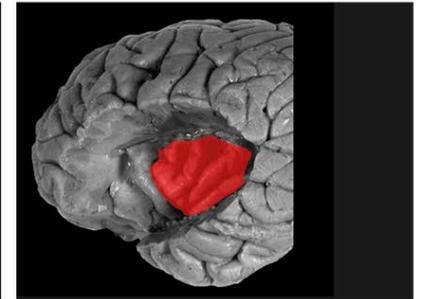
CONSCIOUS EXPRESSION

The amygdala and hypothalamus (blue) are active in expressing emotion, while the thalamus (green) maintains consciousness.



EMOTIONS BECOME CONSCIOUS

Large areas of the frontal and parietal lobes (green) are involved in making emotions conscious and mediating their intensity.



DISGUST

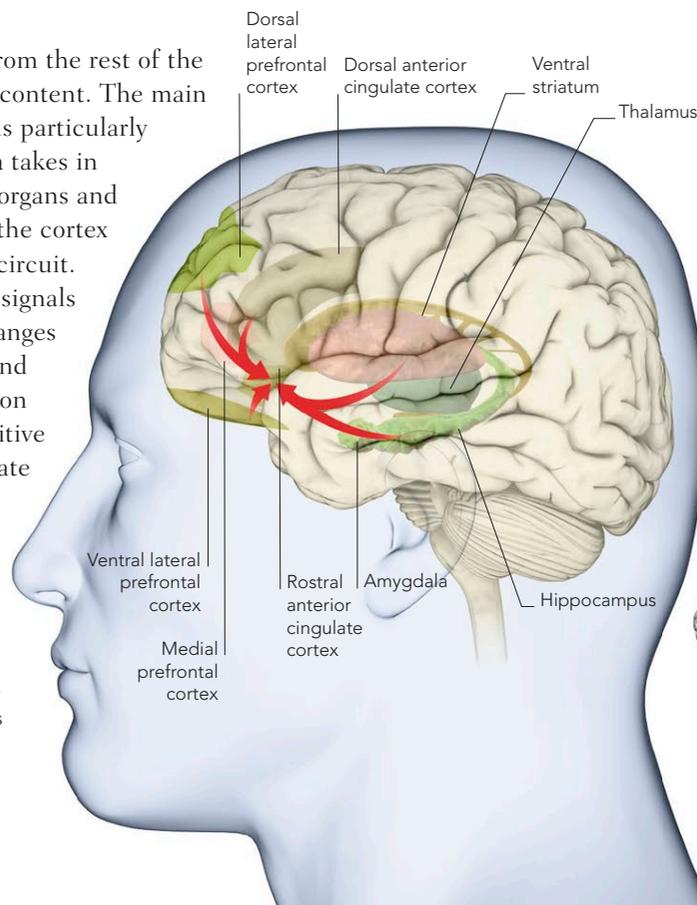
This cutaway shows the insula (red—also in top scan), part of which is active during the generation of emotion, particularly disgust.

EMOTION CIRCUITS

Information from the environment, and from the rest of the body, is constantly “tasted” for emotional content. The main emotion “sensor” is the amygdala, which is particularly sensitive to threat and loss. The amygdala takes in information both directly from the sense organs and via the sensory cortices, and connects to the cortex and also to the hypothalamus, creating a circuit. When the amygdala is activated, it sends signals around this circuit. These trigger body changes as they pass through the hypothalamus, and create conscious recognition of the emotion as they pass through the frontal lobe. Positive emotions are passed along a slightly separate circuit, which takes in an area of the brainstem that produces the mood-lifting neurotransmitter dopamine.

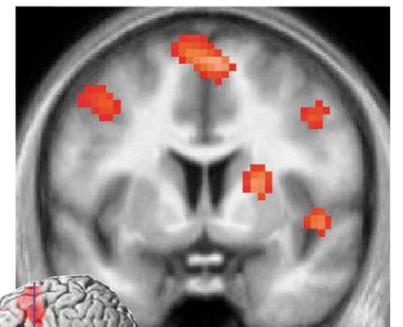
PROCESSING EMOTION

Information about the identification and orientation of emotion travels from the thalamus, ventral striatum, and amygdala to the rostral (lower) anterior cingulate cortex. Regulatory signals travel from areas of the frontal and prefrontal cortices to meet them.



FEELING HATRED

Each emotion sparks a slightly different pattern of activity in certain brain areas. Hatred, for example, activates the amygdala (which responds to all negative emotion), the insula (which is associated with disgust and rejection), and also areas of the brain concerned with action and calculation.

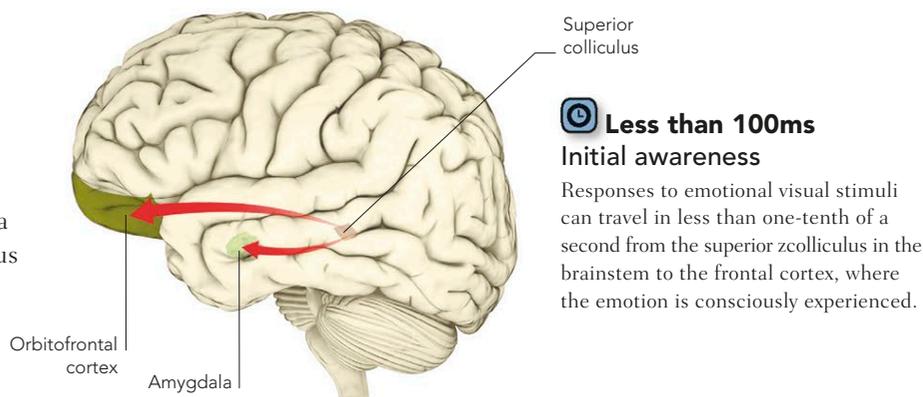


HATE CIRCUITS

Feeling hatred involves areas linked to calculation (shown in the left fMRI scan) and action (top). This pattern may reflect plotting, followed by attack.

TIMING EMOTION

Things that we find emotionally moving grab our attention rapidly (see illustrations, right) compared with things that we do not. The sight of something that poses a threat, for example, is brought to conscious awareness faster than a nonemotional stimulus. This may be because the amygdala unconsciously picks up the threat and primes the conscious brain to “expect” an important perception. Good things also attract attention fast. Research shows that people react as quickly to an image of a smiling baby as they do to one of an angry face—both elicit quicker reactions than nonemotional stimuli.

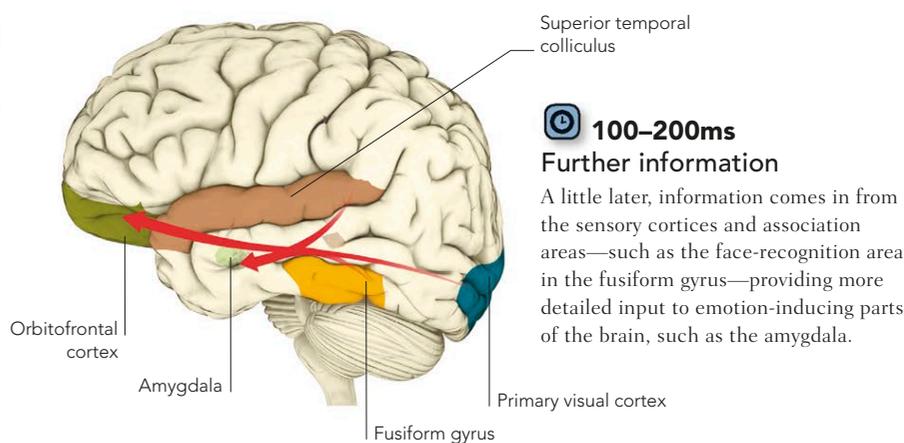


⌚ Less than 100ms Initial awareness

Responses to emotional visual stimuli can travel in less than one-tenth of a second from the superior colliculus in the brainstem to the frontal cortex, where the emotion is consciously experienced.

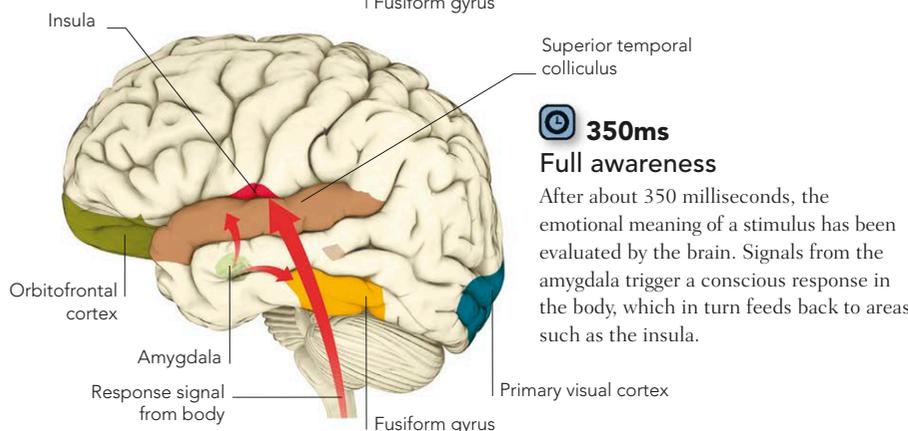
WEARING YOUR EMOTIONS

Scientists have developed clothing that can project the emotion of the wearer. Biometric sensors that pick up minute changes or detect EEG signals are being incorporated into garments next to the skin. The clothes then change color according to the information received. This futuristic dress developed by Philips shines bright white when the wearer is happy but turns blue when she is sad. It has a corset layer containing sensors that send information to an outer skirt layer causing it to change color.



⌚ 100–200ms Further information

A little later, information comes in from the sensory cortices and association areas—such as the face-recognition area in the fusiform gyrus—providing more detailed input to emotion-inducing parts of the brain, such as the amygdala.



⌚ 350ms Full awareness

After about 350 milliseconds, the emotional meaning of a stimulus has been evaluated by the brain. Signals from the amygdala trigger a conscious response in the body, which in turn feeds back to areas such as the insula.

EMOTIONS AND FEELINGS

An emotion is usually transient and arises in response to the thoughts, activities, and social situations of the day. Emotions act as cues that prompt adaptive behavior (see table, right). Moods, in contrast, may last for hours, days, or even months, in the case of some illnesses. Thus, the emotional state of distress, when extended over time, is called sadness; if it persists, unrelenting, for a period of weeks, it is referred to as depression (see p.239). Moods can be initiated very quickly by things that we are not even aware of. One study, for instance, found that flashing pictures of a disgusting nature for a split second—too fast to be seen consciously—made those who were subjected to them more sensitive to other stimuli of a similar nature afterwards. The feelings elicited by these unconscious stimuli were described by the volunteers as “moods” rather than emotions.



TELLING THE DIFFERENCE
Emotions are sudden, intense reactions to events, such as unexpected bad news, whereas moods are more diffuse and tend to last longer.

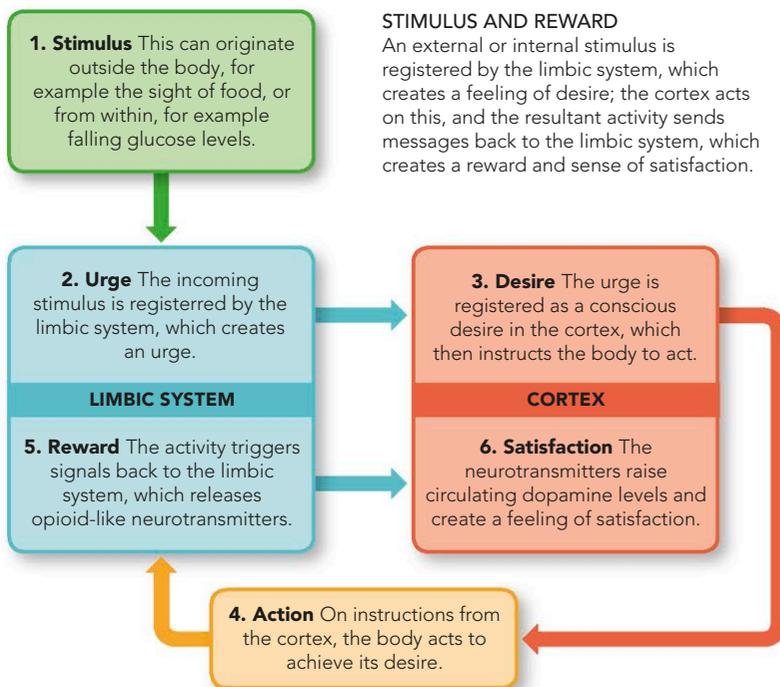
ADAPTIVE BEHAVIORS		
EMOTION OR FEELING	POSSIBLE STIMULUS	ADAPTIVE BEHAVIOR
Anger	Challenging behavior from another person	“Fight” reaction prompts dominant and threatening stance or action
Fear	Threat from stronger or dominant person	Flight, to avoid the threat, or appeasement, to show a lack of challenge to the dominant person
Sadness	Loss of loved one	Backward-looking state of mind and passivity, to avoid additional challenge
Disgust	Unwholesome object (e.g. rotting food or unclean surroundings)	Aversion behavior—remove oneself from the unhealthy environment
Surprise	Novel or unexpected event	Focus attention on the object of surprise, ensuring maximum information input to guide further actions

DESIRE AND REWARD

DESIRE IS HARD TO DEFINE PRECISELY, BUT IT CAN BEST BE DESCRIBED AS WANTING OR YEARNING FOR SOMETHING THAT YOU FEEL WILL BRING PLEASURE OR SATISFACTION ONCE YOU OBTAIN IT. THERE ARE SPECIFIC BRAIN CIRCUITS LINKED TO DESIRE AND REWARD (PLEASURE). DESIRE FOR FOOD AND SEX HAS A SURVIVAL VALUE, BUT DESIRE CAN ALSO BE DESTRUCTIVE IF IT FUELS AN ADDICTION.

DESIRE

Desire is a complex drive that strongly reflects personal preferences. It is made up of two different components—liking and wanting. Put simply, liking is linked to getting pleasure, while wanting is linked to an actual need for something. With some activities, such as eating, sleeping, and sexual activity, liking and wanting overlap, and the resulting desire has survival value. However, an individual with an addiction may want and “need” a drug, but not particularly like or enjoy it, so the resulting pleasure is tainted with destruction. Liking and wanting seem to use somewhat different brain circuits, although dopamine is the most important neurotransmitter in both cases.



ANTICIPATION

Learning and memory clearly play an important role in shaping desires and preferences. This leads to the possibility of anticipation, which is the expectation of a reward. Anticipation has been studied by researchers using a game of chance. In the anticipation phase, where participants were told they might win money, fMRI scans showed that cerebral blood flow in the amygdala and orbitofrontal cortex increased, indicating activity in the nucleus accumbens and the hypothalamus—all rich in dopamine receptors. The bigger the potential reward, the greater the brain activity.



LEFT INTRAPARIETAL CORTEX

REWARD ANTICIPATION

This fMRI scan shows activity in the left intraparietal cortex. Activity in the anterior cingulate cortex and intraparietal cortex show that greater attention is paid to a task when a person is anticipating a reward.

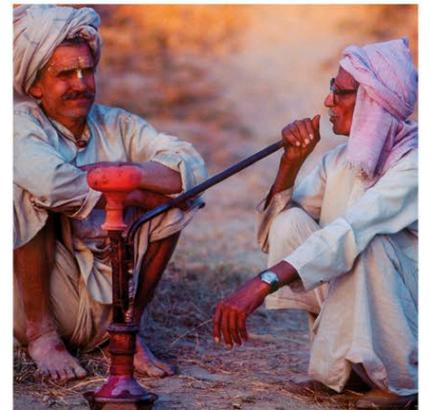
COMPLICATED GRIEF

Losing a loved one is hard, but most people do recover in time. For about 10 to 20 percent of bereaved people, grief endures and is referred to as “complicated.” In one fMRI study, it was revealed that in such people, reminders of the deceased activate a brain area associated with reward processing, pleasure, and addiction. A group of women were shown pictures and words linked to a loved one lost to breast cancer. Brain networks associated with social pain became activated in all women, but in those with complicated grief, the reminders also excited the nucleus accumbens, suggesting that grief was linked, somehow, with pleasure.

PLEASURE-SEEKING AND ADDICTION

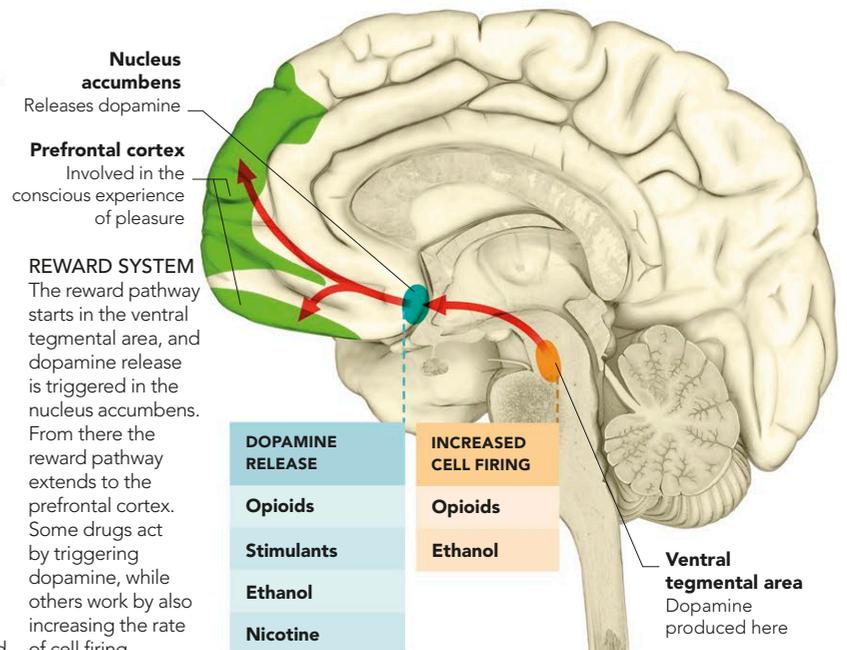
Addictive substances can activate the dopamine reward system, providing pleasure, even though the substances are not essential to survival.

Chronic exposure to drugs leads to the suppression of reward circuits, increasing the amounts needed to get the same effect. The opiate system is involved in pain and anxiety relief. Heroin and morphine lock onto the opiate receptors, creating a sense of euphoria. The cholinergic circuits—where nicotine acts—are involved in memory and learning. Cocaine acts at the noradrenergic receptors, which are involved in stress responses and anxiety.



CULTURAL EXPOSURE

Smoking is regarded as a highly social activity in many cultures. Prolonged exposure to addictive substances may lead to increasing dependence, drug-seeking behavior, and withdrawal problems.



THRILL SEEKERS

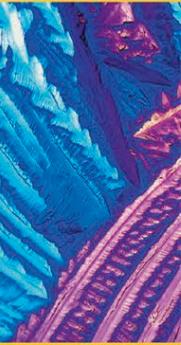
Thrilling or dangerous experiences can cause a rush of epinephrine and dopamine in brain circuits. This rush may lead us to seek out such activities as an easy way of generating intense feelings of pleasure, be it through extreme sports or fairground rides.





HUMANS ARE EXCEPTIONALLY SOCIAL CREATURES. WE NEED EACH OTHER FOR MUTUAL SUPPORT AND PROTECTION, AND TO THIS END WE HAVE EVOLVED BRAINS THAT ARE EXQUISITELY SENSITIVE TO OTHERS OF OUR KIND. THE SOCIAL BRAIN IS A SET OF FUNCTIONS THAT BETWEEN THEM ENSURE THAT WE CAN OPERATE IN A TIGHTLY KNIT COMMUNITY. IT INCLUDES THE ABILITY TO COMMUNICATE WITH AND TO UNDERSTAND OTHER PEOPLE, AND TO KEEP TRACK OF OUR SOCIAL POSITION IN RELATION TO THEM. IN ORDER TO ACHIEVE THIS, WE ALSO NEED TO BE ABLE TO GENERATE A SENSE OF BEING A DISTINCT SELF.

THE SOCIAL BRAIN

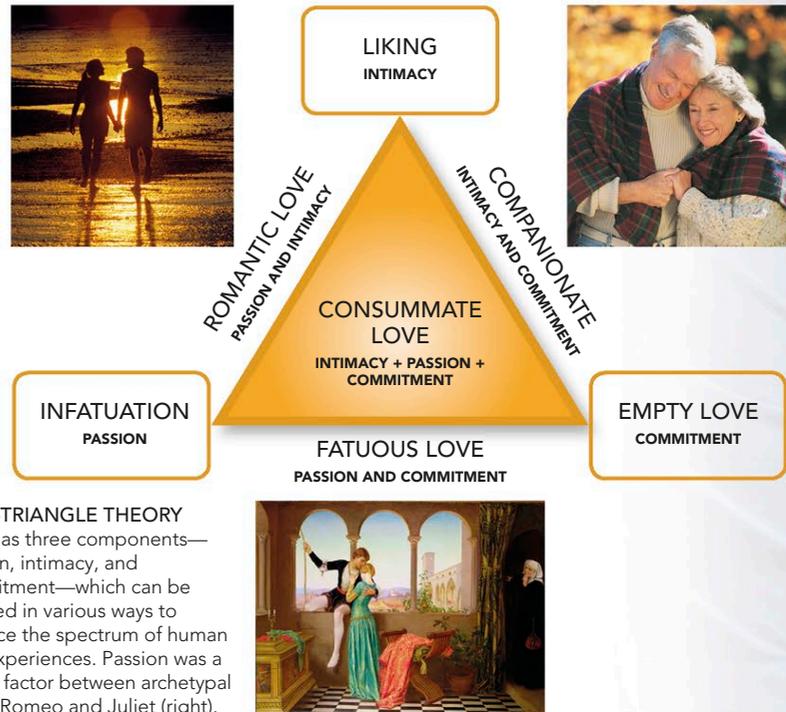


SEX, LOVE, AND SURVIVAL

SEX HAS A SURVIVAL VALUE IN THAT IT DRIVES REPRODUCTION. SEXUAL ACTIVITY STIMULATES THE BRAIN'S REWARD SYSTEM—IF IT DID NOT, PEOPLE MIGHT NOT BOTHER WITH IT AND HUMANITY WOULD DIE OUT. RECENT RESEARCH HAS SHED LIGHT ON THE BRAIN CIRCUITS INVOLVED IN SEX AND LOVE. ROMANTIC LOVE, WHICH BRINGS COUPLES TOGETHER, AND MATERNAL LOVE, WHICH BINDS MOTHER AND CHILD, ALSO HAVE SURVIVAL VALUE.

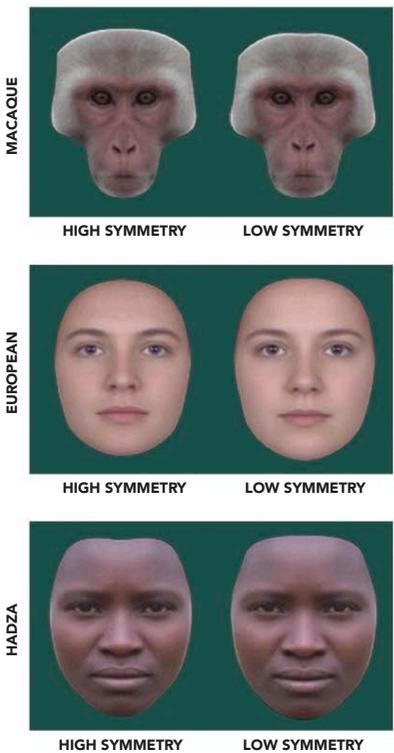
DIFFERENT TYPES OF LOVE

Love is a complex phenomenon, encompassing sex, friendship, intimacy, and commitment. Not only does it have a survival value for the individual as well as the species, but it also adds greatly to quality of life. As far as sex is concerned, humans engage in it whenever they wish, unlike most other species who undertake sex only when the female is ready to conceive. Therefore, sex has become disconnected from reproduction in humans. Romantic love, which is what many people mean by “love,” has a survival advantage because it promotes pair bonding—an ideal setting for the care and protection of young children. Friendship and social networks are also important for promoting health and well-being. We know a little about the neurotransmitters involved in “falling in love,” but not much about corresponding brain circuits. Phenylethylamine and dopamine are involved in the initial euphoria, which probably act in the pathways between the limbic system (concerned largely with emotions) and cortical areas (concerned with reason).

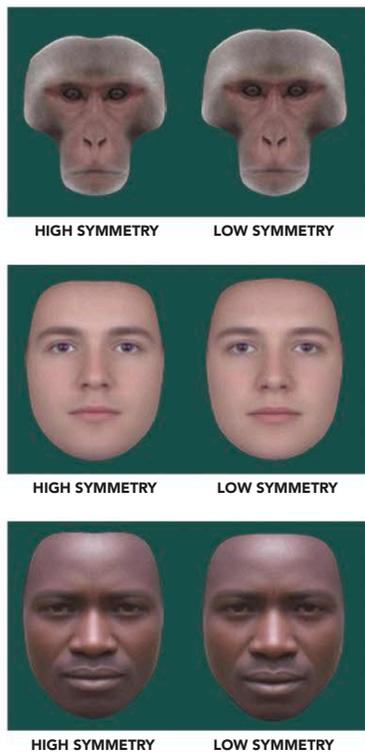


LOVE-TRIANGLE THEORY
Love has three components—passion, intimacy, and commitment—which can be blended in various ways to produce the spectrum of human love experiences. Passion was a strong factor between archetypal lovers Romeo and Juliet (right).

FEMALE



MALE



GENDER AND SYMMETRY

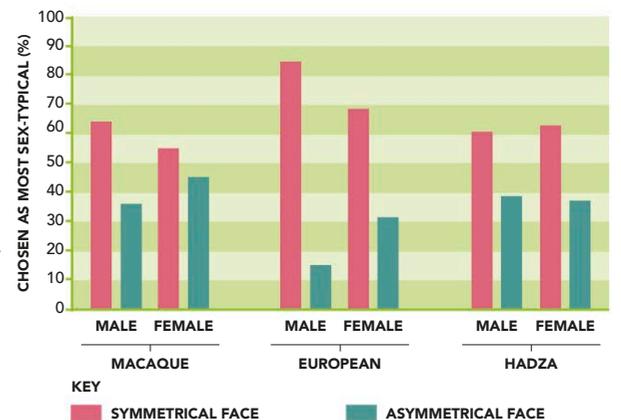
These composite faces, from photos of individuals from three groups, represent high- and low-symmetry faces for each group. High-symmetry faces are often selected as most gender-typical.

SEXUAL ATTRACTION

An individual's face is an important element in how attractive they appear to others and whether they are instinctively considered a good mating prospect. The degree of symmetry, which is linked to how masculine or feminine they appear, has been shown to be an important aspect of facial attractiveness. A recent study shows that these properties are involved in sexual pairings in groups of Europeans, African hunter-gatherers, and one group of nonhuman primates (see below and left). Because the relationship is common to two human groups and one primate group, it may be universal. It seems, therefore, that symmetry and how masculine or feminine a face appears are linked to an underlying biological mechanism that could advertise a person's level of attractiveness and genetic fitness as a mate.

FACIAL SYMMETRY

This graph charts high and low levels of facial symmetry in two human and one primate group. Ratings of faces as more or less masculine or feminine depends on the degree of symmetry measured.





TWO-WAY BOND
Cuddling triggers oxytocin release in both babies and parents, forming a mutual bond. Physical intimacy is vital for a baby. Those reared without it—in some orphanages, for example—may suffer long-term emotional problems.

OXYTOCIN—THE FEEL-GOOD FACTOR

Oxytocin is a hormone produced by the hypothalamus and released by stimulation of the sex and reproductive organs, during orgasm and in the final stages of childbirth. It produces a pleasurable feeling that promotes bonding. This could be because, like the closely related hormone vasopressin, oxytocin helps the processing of social cues involved in the recognition of individuals and may play a role in laying down shared memories. It is possible that oxytocin has a somewhat “addictive” effect, like dopamine. This may explain why people feel anguish at being parted from loved ones—they miss the oxytocin “rush” involved in being with them.



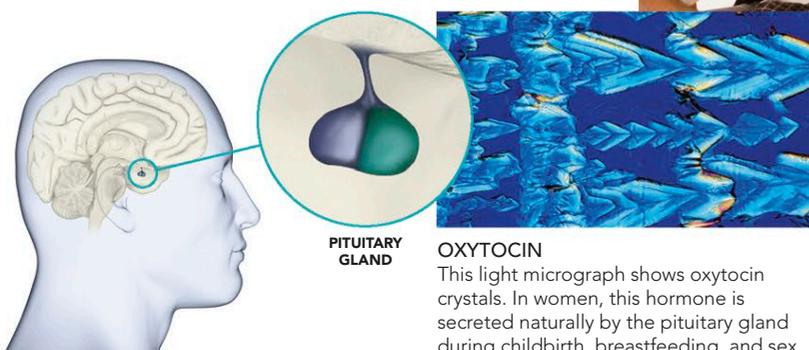
FEELING CLOSE
Kissing and cuddling trigger the release of oxytocin into the bloodstream. This may help heighten feelings of closeness and strengthen the bond between partners.

THE DARK SIDE OF OXYTOCIN

Oxytocin creates trust and kindness among “bonded” individuals, but it amplifies distrust and aggression toward those outside a bonded group. Experiments show that volunteers who are given a dose of oxytocin before playing a trading game are more generous than others to those players who “play fair” but more punitive to others who try to cheat. And one effect of military “bonding sessions”—in which oxytocin is probably engaged—is to make teams of soldiers fight enemies more fiercely.



BONDING SESSION
Soldiers who train together form a tight social bond, which is likely to engage oxytocin. This helps forge trust among the unit but also increases aggression toward perceived outsiders.



PITUITARY GLAND

OXYTOCIN
This light micrograph shows oxytocin crystals. In women, this hormone is secreted naturally by the pituitary gland during childbirth, breastfeeding, and sex.

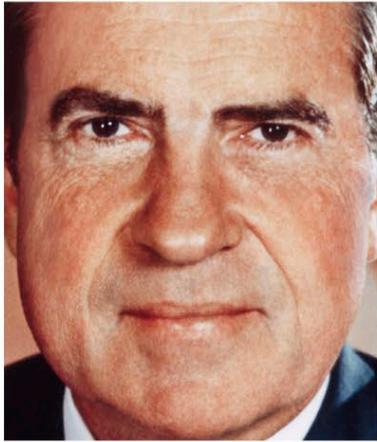
EXPRESSION

HUMANS ARE HIGHLY INTERDEPENDENT—WHAT ONE DOES INVARIABLY AFFECTS WHAT HAPPENS TO OTHERS. IT IS THEREFORE VERY USEFUL FOR US TO BE ABLE TO READ EACH OTHERS' EMOTIONS IN ORDER TO PREDICT WHAT SOMEONE MIGHT DO NEXT. WE ALSO NEED TO SIGNAL OUR OWN EMOTIONS IN ORDER TO NUDGE OTHERS TO DO WHAT WE WANT.

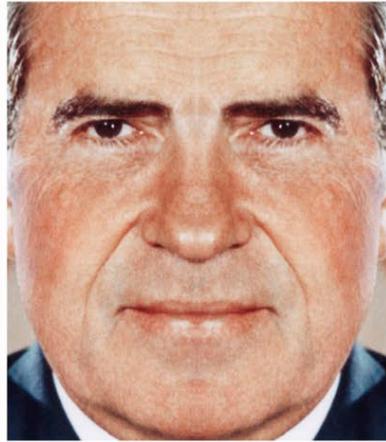
EXPRESSING EMOTION

Expressions are more than just signals; they are an extension of the emotion itself. When we feel something, the neural activation pattern associated with the emotion includes the firing of neurons, which, if not inhibited, cause face and body muscles to contract in

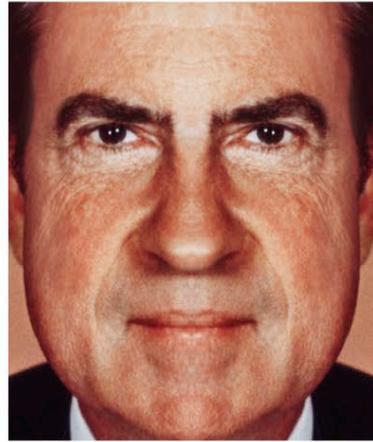
characteristic ways. There are six basic, or universal, emotions (see bottom). Recent studies have looked at the range of expressions used by people who have been blind since birth and found that they are similar or identical to those displayed by sighted people. This suggests that learning plays quite a small part in expression.



TRUE EXPRESSION?
The left hemisphere controls movement on the right side of the face, while the more emotional right brain controls the left side.



RIGHT AND RIGHT
The two right sides of former US president Richard Nixon's face hint at his unconscious feelings. Here the eyes appear less engaging.



LEFT AND LEFT
The two left halves together give a clearer picture of the intended or "social" facial expression that looks more eager to please.

MICROEXPRESSIONS

As well as making the obvious "macro" expressions, people make facial changes that are tiny or momentary (or both) and that they can't easily control and are probably unaware of. These "micro" and "subtle" expressions occur when people are trying not to show what they are thinking or feeling. It is easy to miss these fleeting giveaways, but when you know what to look for, you can learn to spot and decode them. Microexpressions come and go in a fraction of a second, while subtle expressions may last throughout a conversation, but the muscular changes may be so slight as to be barely visible.

SURPRISE

Brows lowered
Eyes bulging
Arched brows
Eyes open wide
Jaw dropped

ANGER

Cheeks raised
Nose wrinkled
Lips pressed

DISGUST

Raised brows
Eyes widened
Mouth open
Upper lip raised

FEAR

SIX EMOTIONS
Surprise, anger, disgust, fear, happiness, and sadness are all universal emotions. Each produces a distinct facial expression, which is almost identical across every culture.

ANATOMY OF A SMILE

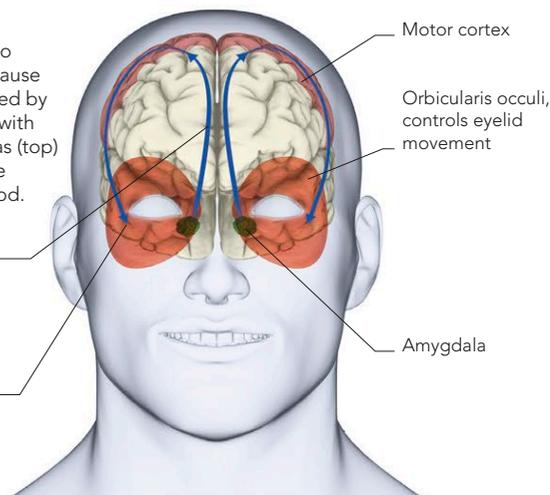
There are two fairly distinct types of human smile: the conscious “social” smile, and the genuine “Duchenne” smile, which is named after the French neurologist Guillaume Duchenne, who first described it. The first involves consciously activating the muscles that stretch the mouth sideways. The second involves an additional set of muscles, which are mainly controlled by unconscious brain processes. These muscles make the lower lids of the eyes swell and the edges crinkle into “crow’s-feet”. Expressions not only show what a person is feeling but they can also actually bring about the feeling that they are associated with. In laboratory tests, consciously producing a smile was found to produce a weak but detectable sense of happiness in those who displayed it. So, even producing a “fake” social smile can promote a faint but real sensation of happiness in the person expressing it.

SMILING

A heartfelt smile is hard to produce on demand because it requires and is controlled by emotion. The real smile, with both mouth and eye areas (top) activated, is usually a true reflection of a happy mood.

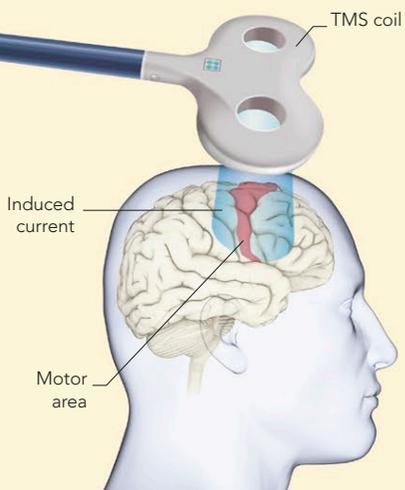
In “genuine” smile, signals are sent from areas of brain, such as amygdala, and are transmitted to motor cortex without awareness

Signal causes small muscles surrounding eye socket to contract, creating characteristic “wrinkles”



READING EMOTIONS

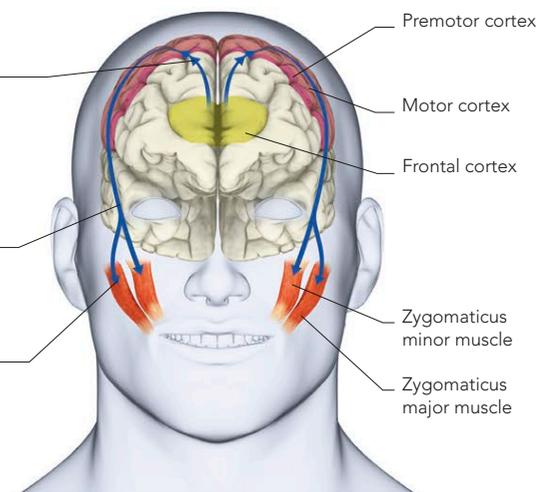
When we read somebody’s expression, we automatically make it ourselves. We can hide this echo by consciously inhibiting the muscular change. Because expressions cause, as well as transmit, our feelings, this mimicry creates an echo of the emotion we see and tells us how the other person is feeling. This is shown by experiments in which people are stopped from echoing expressions by temporarily paralyzing an area of the motor cortex with transcranial magnetic stimulation. When volunteers were unable to mimic expressions, they were less accurate at reading them in others.



In “social” smile we are aware of signals being sent to premotor and motor cortex

Signal bypasses eyes

Signal causes large muscles around mouth to contract, pulling lips sideways



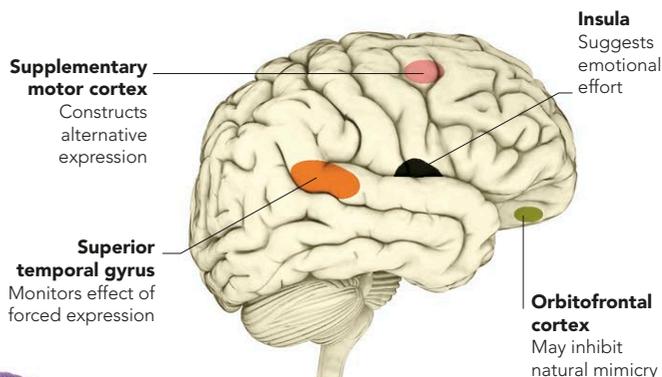
HAPPINESS

SADNESS



CONFLICTING EMOTIONS

Expressions have a direct effect on those who see them (see pp.122–123), so they are useful to get others to serve our needs. However, in social situations, we sometimes have to make a conscious effort to stop making the expression that matches either what we spontaneously feel or what we see in others. Because expressing an emotion creates that emotion, when we do this, we have to override one emotion with another, creating emotional conflict. Humans are probably unique in using facial expressions dishonestly, and we have become experts at doing so, but we are also very good at scrutinizing the expressions of others to discern the genuine from the fake.



AREAS OF CONFLICT

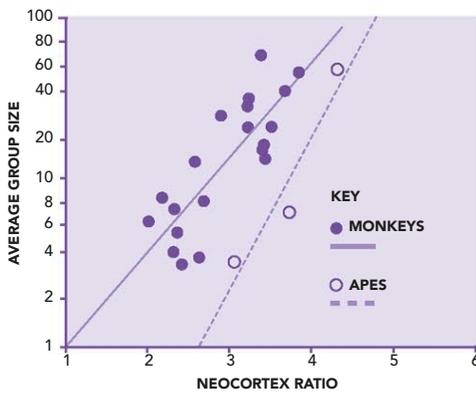
Trying to override natural mimicry of an emotion by expressing a conflicting one engages various brain areas.

THE SELF AND OTHERS

THE HUMAN ANIMAL IS AN INTENSELY SOCIAL SPECIES, AND OUR SURVIVAL DEPENDS LARGELY ON SUCCESSFUL INTERACTIONS WITH OUR NEIGHBORS. AS WITH OTHER SOCIAL ANIMALS, WE HAVE EVOLVED DISTINCT BRAIN CIRCUITS DEDICATED TO BONDING, COOPERATION, AND PREDICTING THE ACTIONS OF OTHERS. WE CAN ALSO RECOGNIZE THAT OTHER PEOPLE HAVE THEIR OWN THOUGHTS AND FEELINGS.

MADE TO BE SOCIABLE

One of the most distinctive features of the human brain is the large area of neocortex, its relatively recently developed outer layer. The frontal cortex (the part of the neocortex that surrounds the frontal lobe) is responsible for abstract reasoning, conscious thought and emotion, planning, and organization, and is highly developed in humans. One reason for the substantial growth of the neocortex may be that humans adapted this way in response to the demands of living in large, close-knit groups. Social living creates challenges such as moderating one's own behavior in order to accommodate others, competing subtly for reproductive rights, and predicting how others will behave, all of which need neocortical activity. Spending time in social activity also seems to grow the areas of the brain responsible for understanding and dealing



with others. People who have large numbers of friends on social networking sites have correspondingly large social brain regions.

GROUP SIZE MATTERS
In primates, the size of the neocortex relative to other brain areas increases in almost direct proportion to the average size of the social group.

SOCIAL ANIMAL

Animals that live in large groups are socially smarter than those that don't. A study found that ring-tailed lemurs, which live in big groups, learned to steal food from people only when they were not looking. Other animals with comparable intelligence failed to do this.



CONTAGIOUS YAWNING

Social behaviors can be deliberate or unconscious. For example, it is thought that "catching" a yawn is an unconscious way of synchronizing group behavior. One theory about yawning is that, when one person does it, it signals that it is time for the entire group to sleep. By mimicking the yawn, other members implicitly agree. Another theory is that yawning keeps the brain alert. Its contagious nature ensures that each member of the group sharpens up.



SOCIAL AWARENESS

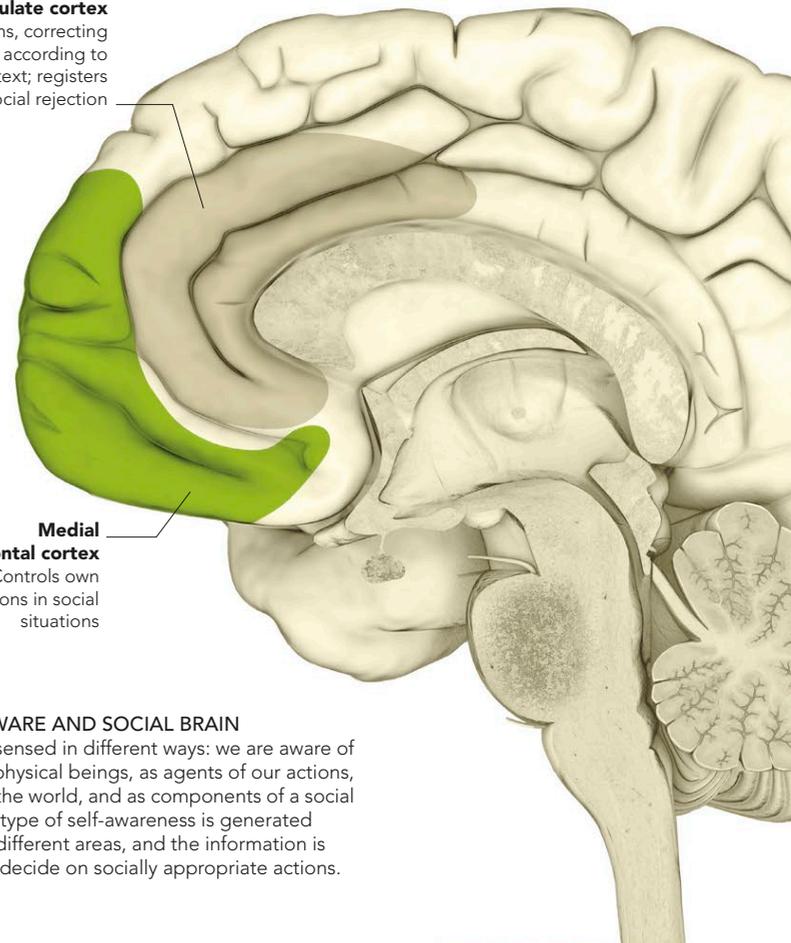
Social awareness covers a wide range of cognition that generates a sense of a "self" as well as of that self in a social context. For example, we adapt our behavior to cooperate with others, we predict what other people are likely to do and their reasons for doing it, we understand that others may hold different ideas and beliefs from our own, we are able to imagine how other people see us, and we can scrutinize our own minds. The range and diversity of skills required means that several areas of the brain are involved.

Anterior cingulate cortex

Selects actions, correcting intentions according to social context; registers social rejection

Medial prefrontal cortex

Controls own emotions in social situations



THE SELF-AWARE AND SOCIAL BRAIN

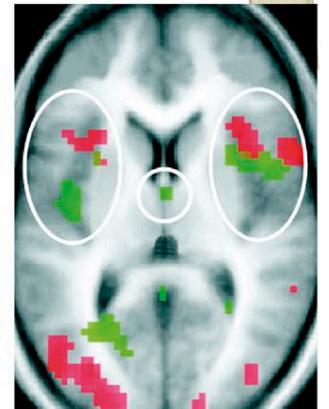
The "self" is sensed in different ways: we are aware of ourselves as physical beings, as agents of our actions, as objects in the world, and as components of a social system. Each type of self-awareness is generated by activity in different areas, and the information is combined to decide on socially appropriate actions.

THE INSULA

The insula may be responsible for humans experiencing the feeling of a "self" and having a sense of the boundary of that self, allowing for the distinction between "me" and "you." According to a school of thought known as "embodied cognition," which proposes that rational thought cannot be separated from emotions and their impact on the body, the insula detects body states that are induced by emotions as part of a process that brings our emotional experiences into our consciousness.

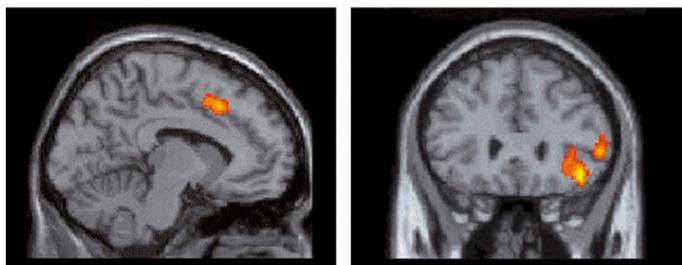
OBSERVING PAIN

Tests using fMRI scans show insula activity (green) in participants watching a person in pain, suggesting that the insula triggers empathic feelings.



THE PAIN OF REJECTION

In one study, fMRI scans were conducted on people playing a virtual ball game from which they were progressively excluded. Upon awareness of rejection, the anterior cingulate cortex (ACC) was activated, an area that also registers body pain, suggesting that the emotional impact of the two is similar. Part of the prefrontal cortex that helps control emotions was also activated, which seemed to reduce feelings of rejection.



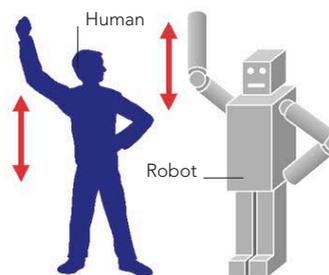
ANTERIOR CINGULATE CORTEX
Social rejection causes the same type of activity in the anterior cingulate cortex (ACC) as physical pain.

PREFRONTAL CORTEX
The ventral prefrontal cortex then interacts with the ACC, which seems to reduce the pain of social rejection.

CONGRUENCE

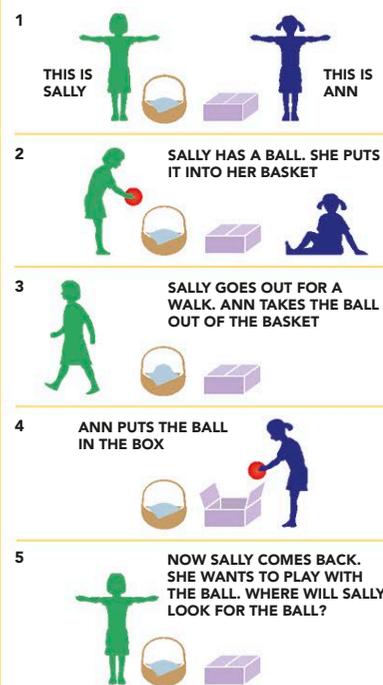
Our brains are highly sensitive to the movements of other animals, especially other humans. The mirror neuron system (see pp.122–23) automatically makes us mirror the actions of others. The effect is so strong that when one person notices another not mirroring their own actions, it often makes them falter in their own actions. This “interference effect” applies only to biological motion—when participants observe a robot, no such interference occurs, even if the actions are humanlike.

MIRRORING
A person is discomfited if someone fails to mirror their actions, but whether or not a robot does so has no effect.

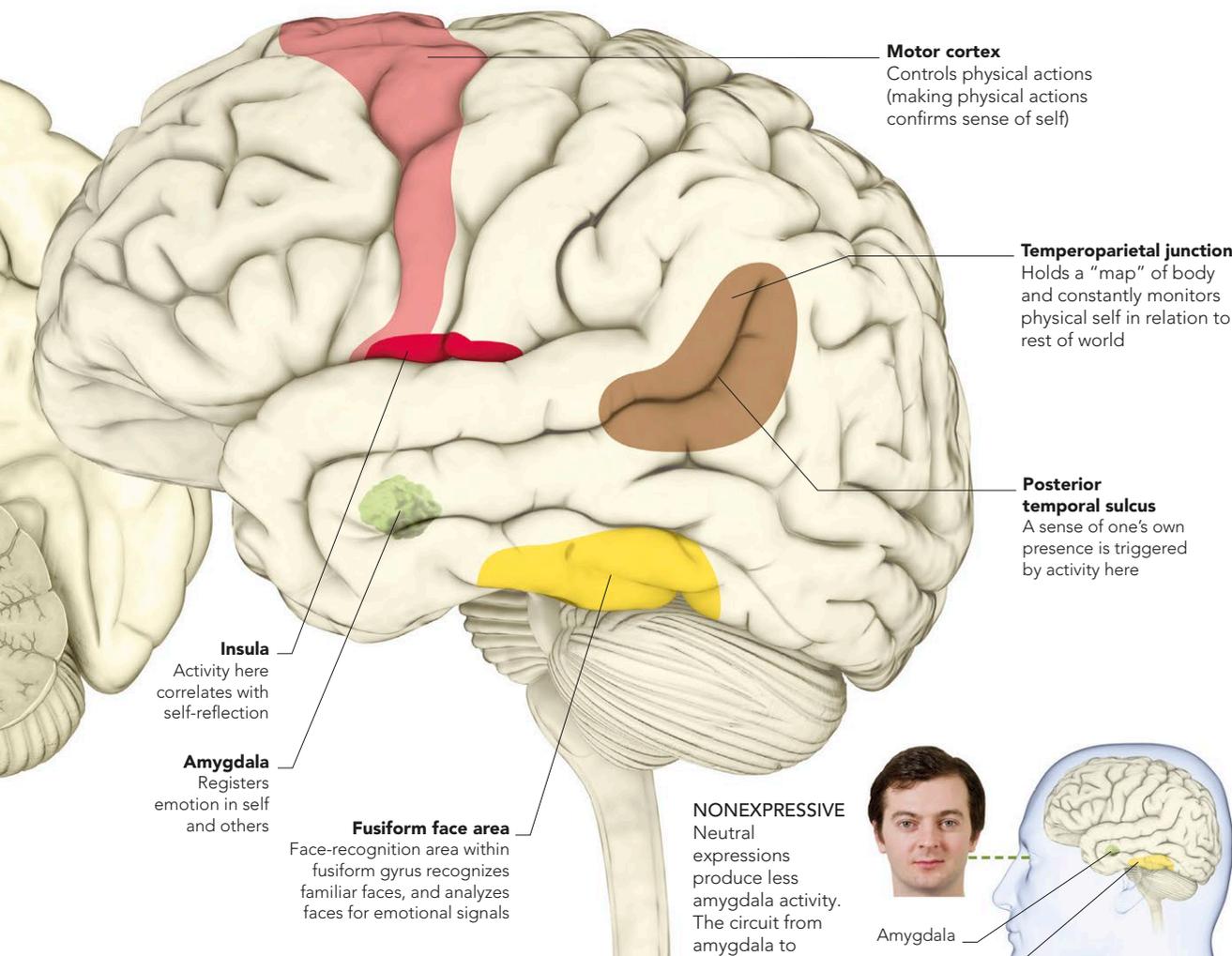


THEORY OF MIND

Theory of mind (ToM) refers to the instinctive “knowledge” that other people may hold different beliefs than one’s own, and that it is those beliefs, not the facts of a situation, that inform and determine their behavior. One way to test for ToM is the Sally-Ann test (see diagram, below). Recent studies have shown that infants as young as 10 months may “pass” the Sally-Ann test.



SALLY-ANN TEST
If children indicate that, on her return, Sally will look in the place she expects the ball to be (in the basket), they appear to have ToM.



Motor cortex
Controls physical actions (making physical actions confirms sense of self)

Temporoparietal junction
Holds a “map” of body and constantly monitors physical self in relation to rest of world

Posterior temporal sulcus
A sense of one’s own presence is triggered by activity here

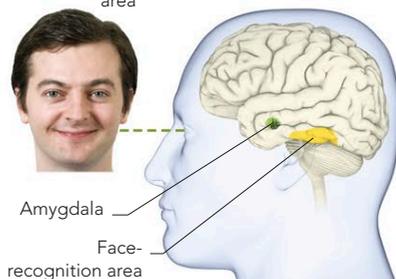
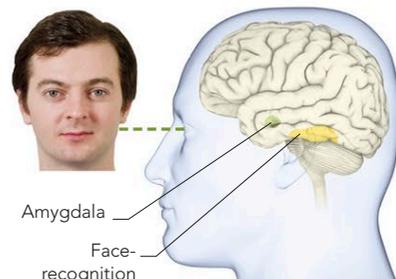
Insula
Activity here correlates with self-reflection

Amygdala
Registers emotion in self and others

Fusiform face area
Face-recognition area within fusiform gyrus recognizes familiar faces, and analyzes faces for emotional signals

NONEXPRESSIVE
Neutral expressions produce less amygdala activity. The circuit from amygdala to face-recognition area is toned down and the brain takes in less information.

EXPRESSIVE
The amygdala reacts to facial expressions by “mirroring” the emotion. A smile, for example, triggers signals that begin the process of smiling back.

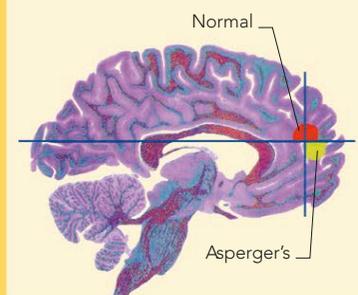


RESPONDING TO EMOTION

Facial expression is a signal—of intention and state of mind—and also a means of achieving empathy between people. Expressions are initially processed unconsciously by the amygdala, which monitors incoming data for emotional content. It responds by generating the emotion that has been observed. A fearful expression, for example, produces amygdala activation that triggers fear in the observer. Soon after the amygdala activation, the expression registers in the face-recognition area situated in the fusiform gyrus. Studies suggest that if a face expresses emotion, the amygdala signals this area to scrutinize it for meaning.

AUTISM AND THE MIND

Autism is marked by the absence of ToM. Rather than just “knowing” why Sally acts according to a false belief, people with Asperger’s syndrome (a form of autism) consciously “work out” what is happening using part of the brain (yellow) that is thought to be more recently evolved than the area that generates ToM (red).



THE MORAL BRAIN

NORMAL PEOPLE BROUGHT UP IN A NORMAL ENVIRONMENT DEVELOP AN INSTINCTIVE SENSE OF RIGHT AND WRONG THAT SEEMS TO BE, AT LEAST IN PART, “HARDWIRED” INTO THE BRAIN. THIS NATURAL “MORALITY” IS NOT NECESSARILY RATIONAL OR FAIR, AND PROBABLY EVOLVED BECAUSE BEHAVIOR PROMOTING SOCIAL COHESION ALSO, INDIRECTLY, AIDS SELF-SURVIVAL.

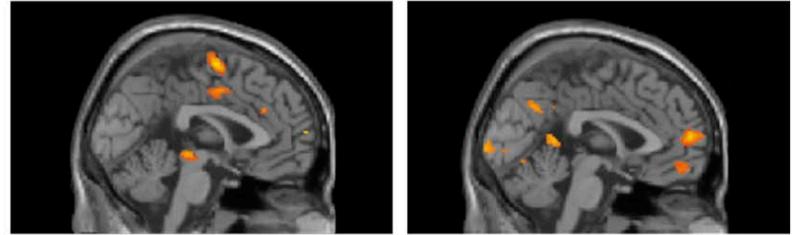
EMPATHY AND SYMPATHY

“Feeling” for another person—experiencing a faint version of their sorrow or flinching when you see them hurt—seems to be largely instinctive. It depends partly on theory of mind (see pp.138–139), which ensures that we “know” what is likely to be going on in other people’s minds. Empathy goes a step further, in that it also involves “echoing” the emotions of another person. When a person is told a story about someone experiencing emotional trauma, the activated areas in the listener’s brain come into play when he or she is in such a situation.



SYMPATHETIC STANCE

Being able to put yourself into someone else’s situation, to experience an echo of what they feel, and sympathize with them appears to be an instinctive human trait.



WITNESSING ACCIDENTAL PAIN

This fMRI scan shows that seeing someone hurt by accident produces similar brain activity as if the viewer was accidentally hurt.



WITNESSING INTENTIONAL INJURY

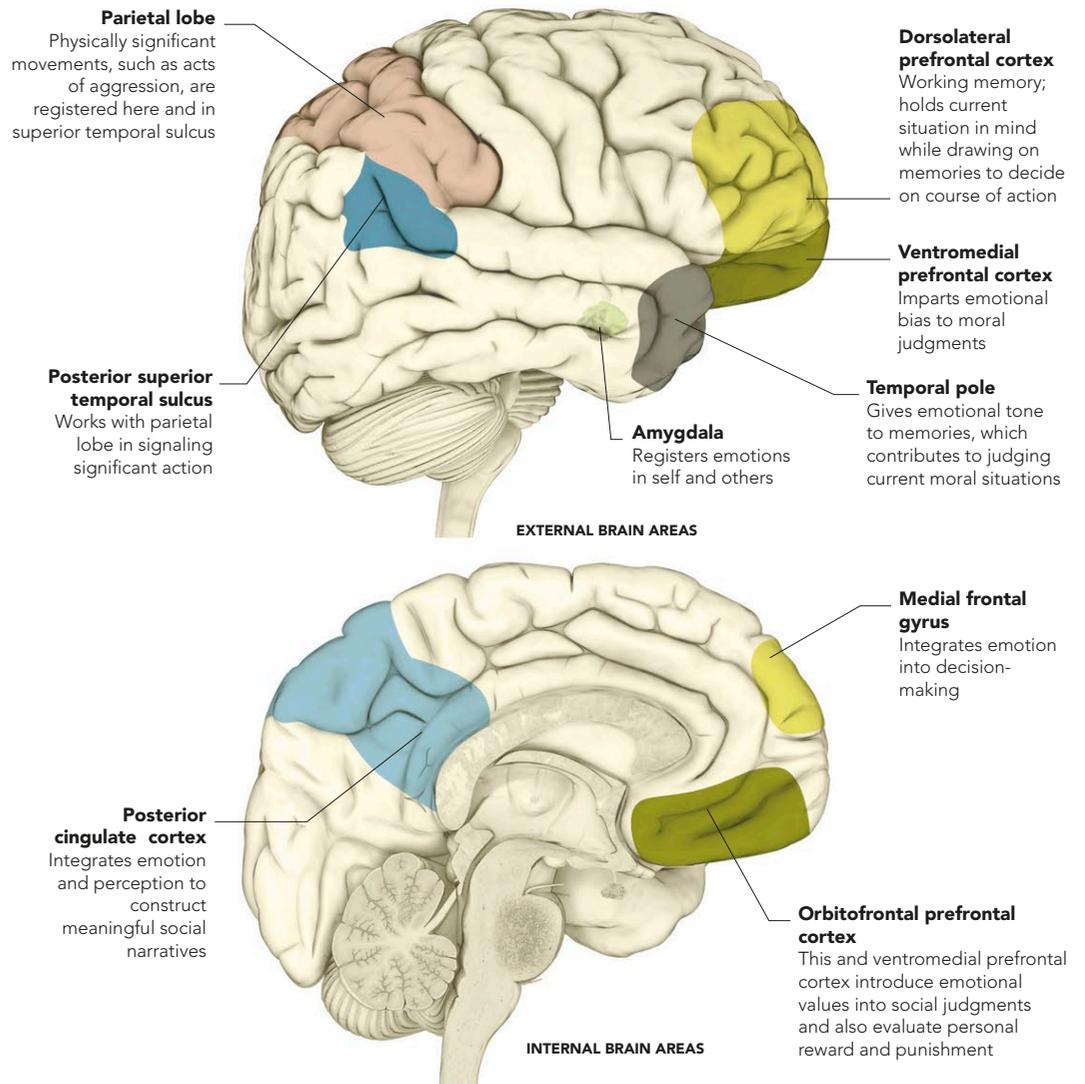
When witnessing someone hurt intentionally, brain areas concerned with judgments and moral reasoning (above) are also activated.

MORALITY

Our sense of right and wrong permeates all our social perceptions and interactions. Moral decision-making is partly learned, but it also depends on emotions, which give “value” to actions and experiences. When making moral judgments, two overlapping but distinct brain circuits come into play. One is a “rational” circuit, which weighs up the pros and cons of an action objectively. The other circuit is emotional. It generates a fast and instinctive sense of what is right and wrong. The two circuits do not always arrive at the same conclusion, because emotions are biased toward self-survival and/or protecting those who are loved or related to oneself. Emotional bias in moral judgments seems to rely on activity in the ventromedial and orbitofrontal prefrontal cortex. Studies of people with damage to this area have found that their moral judgments are more rational than those of others, suggesting that human “morality” is hardwired into the brain and evolved more to protect ourselves than to “do good.”

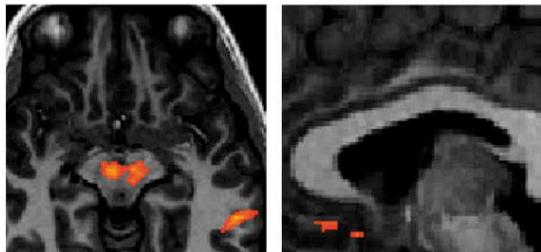
MORAL JUDGMENT CIRCUITS

Emotions play a crucial role in moral decision-making (see p.169). In order to arrive at moral decisions, areas of the brain associated with emotional experience work alongside those that register facts and consider possible actions and outcomes.



ALTRUISM

The notion of altruism assumes that people can do things for others with no motivation of a direct reward for themselves. However, brain scans show that doing “good” things is personally rewarding. One fMRI study was conducted while participants made or withheld donations to real charities. The participants could keep any donations they refused to make. The result showed that both keeping the money and giving it away activated the brain’s “reward” pathways.



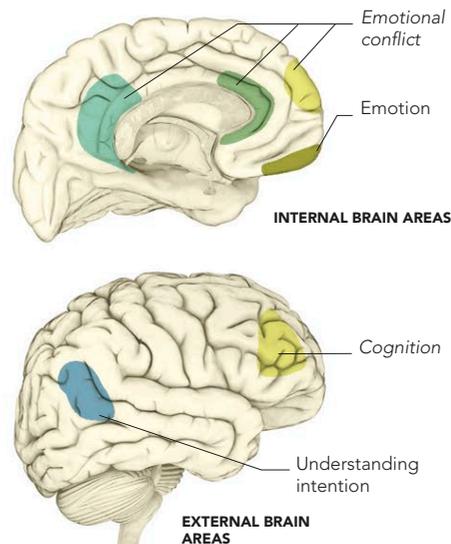
RECEIVING

GIVING

Giving away money also enhanced activity in areas concerned with belonging and group bonding.

REWARD AREAS

Giving and receiving activate areas linked to pleasure and satisfaction. Areas linked to bonding and social cohesion are active when giving.



BRAIN DAMAGE AFFECTS MORALS

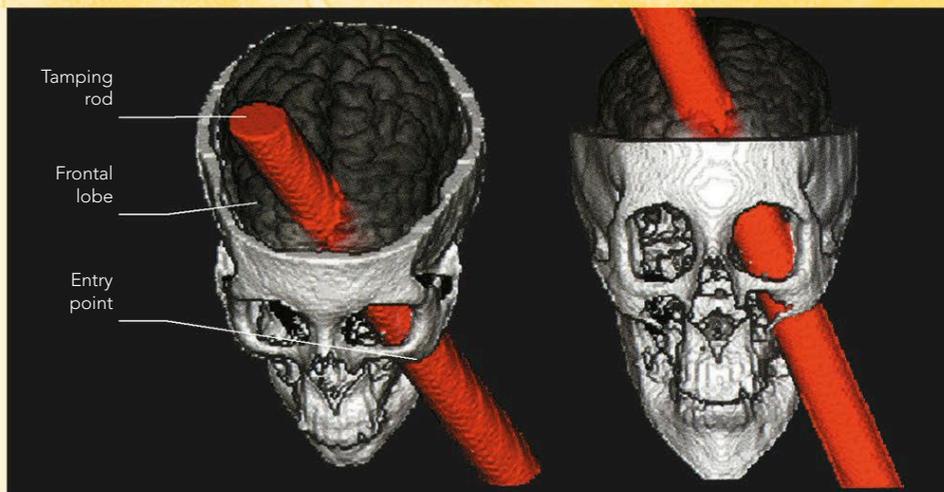
Damage to any one of several brain areas can affect moral judgment. They include: areas involved in feeling emotion and assessing emotional intent and conflict; the frontal areas involved in thinking about current situations and assessing action; and the area at the junction of the parietal and temporal lobes, which allows for understanding others’ intentions.

PHINEAS GAGE

The idea that our moral sense may have a biological basis in the brain arose largely as a result of a freak accident in 1848. A railroad worker named Phineas Gage blew a hole in the front of his brain with a tamping rod. He survived with little damage to most of his faculties, but his behavior changed dramatically. From being polite and thoughtful, Gage was described by his doctor as “fitful, irreverent, indulging at times in the grossest profanity (which was not previously his custom), manifesting but little deference for his fellows, impatient of restraint of advice when it conflicts with his desires, at times pertinaciously obstinate, yet capricious and vacillating... his mind was radically changed, so decidedly that his friends and acquaintances said he was ‘no longer Gage.’”

RECONSTRUCTION

Computer-generated images reveal the exact location of the damage to Phineas Gage’s brain. Apart from going blind in one eye, he suffered few physical effects, but his behavior changed dramatically.

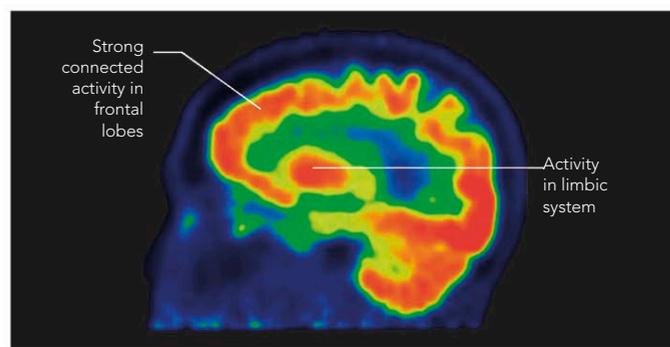


PSYCHOPATHY

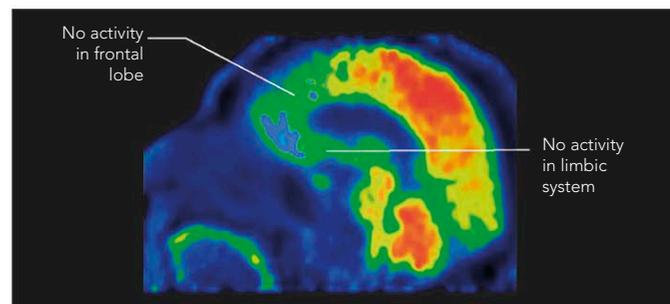
Psychopaths are marked by an abnormal lack of empathy, to the extent that some even enjoy seeing others suffer. They may, however, be charming, intelligent, and capable of mimicking normal emotions so well that they are difficult to spot. Psychopathic behavior is linked to risk-taking, irresponsible, and generally selfish behavior, but those with high intelligence can curb these tendencies and become very successful. A large number of leading businesspeople show psychopathic tendencies, as well as a large proportion of criminals. The brains of people who have psychopathic tendencies show less emotional response to images of people being hurt, and the emotional parts of their brains have fewer connections with the frontal areas that consciously “feel” for others.

PSYCHOPATHIC BRAINS

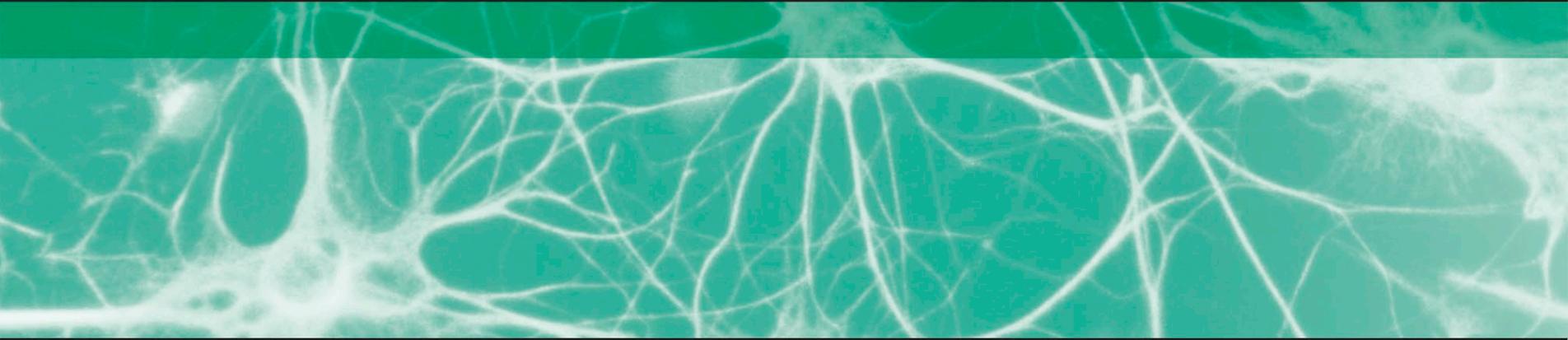
Psychologist James Fallon studied psychopathic prisoners and scanned their brains (bottom right) as they viewed emotional images. Professor Fallon found that his own brain has psychopathic markers, which he acknowledges reflects his lack of empathy. His intelligence and insight allow him to overcome his emotional dysfunctions.



NORMAL BRAIN

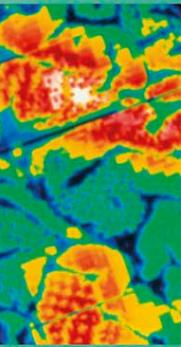


PSYCHOPATHIC BRAIN



WE SIGNAL OUR INTENTIONS TO EACH OTHER IN VARIOUS WAYS. A SURPRISINGLY LARGE AMOUNT OF INFORMATION CAN BE TRANSMITTED BY GESTURES AND BODY LANGUAGE. THIS IS AN ABILITY THAT HUMANS SHARE WITH MANY OTHER ANIMALS, BUT WE CAN ALSO COMMUNICATE IN WAYS THAT ARE UNIQUE TO OUR SPECIES. ONLY THE HUMAN BRAIN HAS AREAS DEDICATED TO LANGUAGE. WE USE THESE TO SPEAK AND TO READ AND WRITE. ALTHOUGH READING AND WRITING HAVE TO BE LEARNED, WE SEEM TO BE BORN WITH THE ABILITY TO SPEAK AND TO FOLLOW COMPLEX RULES OF GRAMMAR.

LANGUAGE AND COMMUNICATION

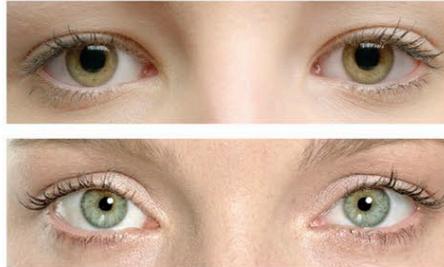


GESTURES AND BODY LANGUAGE

WE SIGNAL OUR THOUGHTS, FEELINGS, AND INTENTIONS BY GESTURE AND BODY LANGUAGE AS WELL AS BY SPEECH. HALF OF OUR COMMUNICATION IS TYPICALLY NONVERBAL, AND WHEN THEY CONFLICT, GESTURES “SPEAK” LOUDER THAN WORDS.

EYE TALK

Human eyes convey information through facial expression and movement. Unlike in most species, the visible white of the human eye makes it easy to see in which direction a person is looking and thus where their attention is directed. People have a strong instinct to follow another’s eye gaze, and this simple mechanism ensures that when someone is in sight of another person, they can manipulate each other’s attention and share information without even having to communicate with words.



STRONG SIGNALERS

Pupils dilate when a person has an emotional reaction. Some drugs have a similar effect—belladonna was once used by women to send signals of sexual excitement.

MIRRORING PARENTS

By three months old, babies have the ability to follow another person’s eye gaze, and they are quick to pick up any emotion contained in a look. Experiments show that if a parent looks toward something and displays fear, for example, by widening their eyes, the child is very likely to mirror this reaction and be scared too, even if the object is clearly harmless.

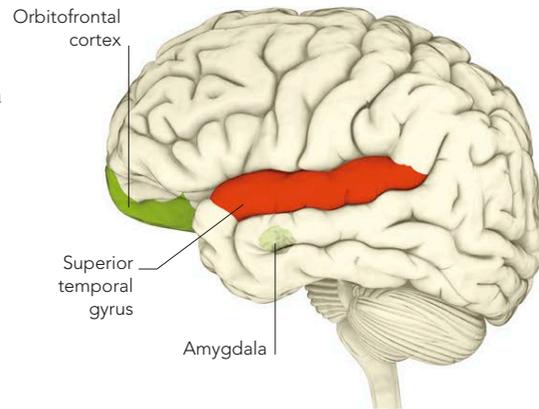


BODY LANGUAGE

Body language is mostly instinctive, consisting largely of unconscious “breakthrough” acts. Some of these are remnants of primitive reflexes, when other living things were often seen primarily as either predator or prey. These ancient reflexes program us to approach small, soft stimuli, which suggest prey, and to withdraw from strong, hard stimuli, which suggest a predator. Aggression is usually shown through tensed muscles and an upright or forward-leaning stance, indicating that a predator is ready to pounce. Fear is displayed by a softer body contour and backward stance, indicating that the prey is preparing to flee. When emotions are mixed,

EXPRESSION AND BODY LANGUAGE STUDY
When body language and facial expression do not match each other, we are biased toward the emotion signaled by the body, rather than the expression on the face.

a person may take up a midway stance from which they can shift quickly from one posture to another.



BRAIN PROCESSES
Giveaway eye, mouth, hand, and body movements, as well as deliberate gestures, are registered in the superior temporal sulcus, a brain area concerned with the self in relation to others. The amygdala notes the emotional content, and the orbitofrontal cortex analyzes it.



ANGRY EXPRESSION;
ANGRY BODY LANGUAGE

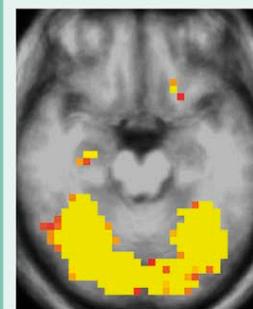
FEARFUL EXPRESSION;
ANGRY BODY LANGUAGE

ANGRY EXPRESSION;
FEARFUL BODY LANGUAGE

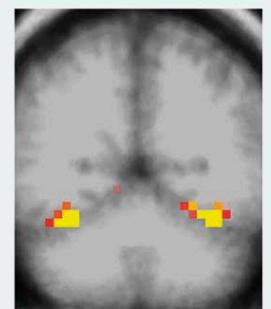
FEARFUL EXPRESSION;
FEARFUL BODY LANGUAGE

REACTING TO BODY LANGUAGE

Body language showing fear or anger sparks activity in brain areas involved in movement, while that expressing happiness stirs activity in the visual cortex. In one study, subjects’ brains were scanned while they were shown images of actors with blurred faces in fearful, happy, or neutral poses. Happy gestures, such as arms spread in welcome, spurred activity in the visual cortex. Fearful ones, like cowering, caused activity in emotional centers and in areas involved in movement. This might explain how fear spreads in a crowd and prepares the body to flee.



HAPPINESS



FEAR

GESTURES

Although body language is mostly unconsciously performed, we have a greater degree of conscious control over its more refined form—gestures. Many parts of the body can be involved with making gestures, but most tend to include hand and finger movements, which can display complex spatial relations, issue directions, and show the shape of imagined objects. They can help convey emotions and thoughts, insults, and invitations. Gestures are used throughout the world, although they by no means have universal meanings. Even

THREE MAIN CATEGORIES

“Natural” gestures tend to be used for three main purposes: to tell a story, to convey a feeling or idea, or to emphasize a spoken statement. Invented gestures, such as the Masonic handshake, may be completely arbitrary or developed from natural body language.

simple gestures, such as pointing at a person, which is commonly used in many parts of the world, can be highly offensive in parts of Asia.

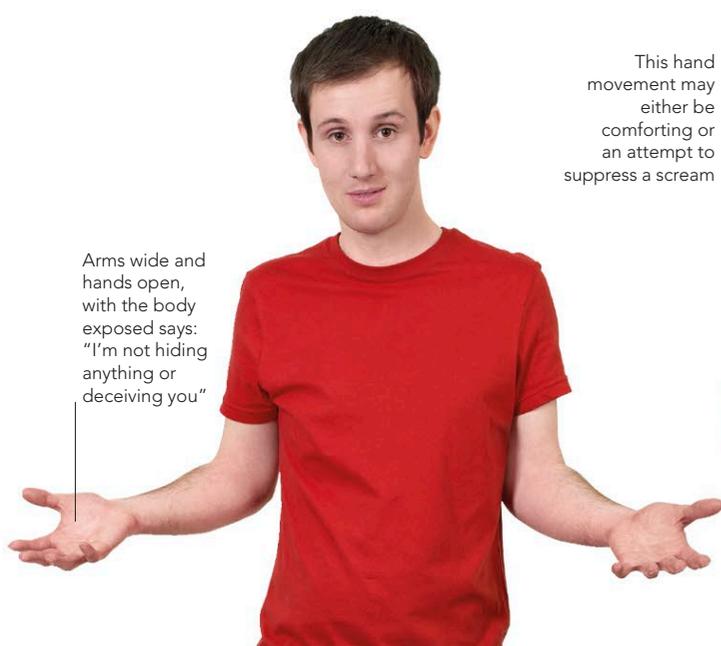


INTRICATE GESTURES

Statues of Hindu deities often convey symbolic meanings through the specific positioning of their hands. With his outward-facing palm, the god Shiva is assuring protection.

THE GRAMMAR OF GESTURE

Unlike the rules of speech, which vary from language to language, gesturing seems to have a universal “grammar”. Asked to communicate a simple statement using words of their native languages, English, Chinese, and Spanish speakers started with the subject, then the verb and finally the object, whereas Turkish speakers used the subject, object, then the verb. However, when just using gestures, speakers of all of these languages placed the subject, object, and verb in that order.



Arms wide and hands open, with the body exposed says: “I’m not hiding anything or deceiving you”

PROTESTING INNOCENCE

This hand movement may either be comforting or an attempt to suppress a scream



SHOCK

Aggressive, rigid hand movement suggests anger or rejection of another person



ANNOYANCE



Raising to full height with clenched fists suggests victory

JUBILATION



Hands may convey a more precise measurement than the speaker might be able to get across verbally

MEASURING WITH HANDS

Pulling fingertips together suggests accuracy, cohesion, and concentration; may be used to focus listener’s attention on words



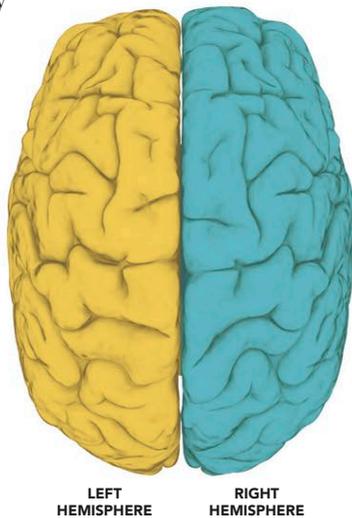
REINFORCING A POINT

THE ORIGINS OF LANGUAGE

HUMANS HAVE AN INNATE CAPACITY FOR LANGUAGE—A FACULTY THAT SEEMS TO RELY ON ONE OR MORE GENES THAT ARE UNIQUE TO OUR SPECIES. IT IS NOT KNOWN, THOUGH, WHETHER LANGUAGE AROSE AS A DIRECT RESULT OF GENETIC MUTATION OR AS A RESULT OF THE INTERACTION BETWEEN SUBTLE BIOLOGICAL CHANGES AND ENVIRONMENTAL PRESSURES.

HEMISPHERE SPECIALIZATION

Compared to the brains of other species, human brains are less symmetrical in terms of functions. Language is the most obvious example of this lopsidedness, and the vast majority of people have the main language areas on the left side of the brain, although a few seem to have language functions distributed on both sides, and some have it only on the right. Generally, language is associated with the “dominant” side of the brain—that is, the one that controls the most competent hand. Language is thought by some to be the mechanism that elevates the brain to full consciousness, and before language evolved, it is possible that our ancestors were not consciously aware of themselves. Because language is so important, disruptions have awful consequences, so brain surgeons have to be very careful to avoid damaging the language areas. This is one of the reasons for the Wada test.



LANGUAGE FUNCTIONS	
The three principal language areas are usually found in the left hemisphere, while four other important language areas are located in the right hemisphere.	
HEMISPHERE	FUNCTION
Left	Articulating language
Left	Comprehending language
Left	Word recognition
Right	Recognizing tone
Right	Rhythm, stress, and intonation
Right	Recognizing the speaker
Right	Recognizing gestures

AREAS INVOLVED

The main language skills of recognizing, understanding, and generating speech are situated in the left hemisphere in most people. The right hemisphere, however, processes aspects of language that are needed to obtain “full” comprehension.

THE WADA TEST

The Wada test, named after Canadian neurologist Juhn Wada, involves anesthetizing one hemisphere of the brain while leaving the other fully active. This is possible because each hemisphere of the brain has its own blood supply. If the patient is able to speak when one brain hemisphere is asleep, the principal language areas must be on the conscious side. This information is vital for surgeons to plan operations. The Wada test will eventually be replaced by advanced scanning techniques.



CAROTID ARTERIES

This colored magnetic resonance angiogram (MRA) shows the arteries that supply the head and neck. The Wada test involves injecting one of the internal carotid arteries to put one brain hemisphere to sleep.

SILBO LANGUAGE

Most languages use words—that is, noises made by exercising muscles in the throat and mouth that chop up (articulate) and vary the sound of the passage of air from the lungs. Silbo, however, is a language made up entirely of whistles, used by the inhabitants of La Gomera in the Canary Islands. Brain-imaging studies show that Silbo-users process the whistles in the main language areas of their brains, whereas those who do not know the language process the whistles simply as a collection of sounds, which are registered in other areas of the brain.

WHISTLE WHILE YOU WORK

Silbo developed among islanders who needed to communicate in a landscape where deep ravines made shouting impractical—their whistles carry farther than words and with less distortion.

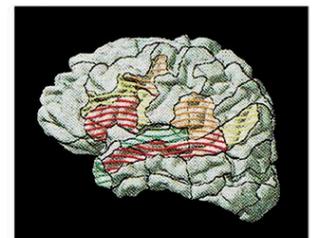


WHAT IS LANGUAGE?

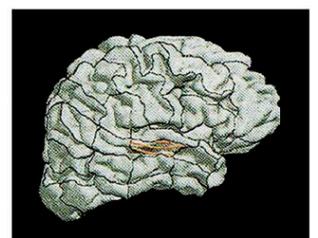
Language is not just a matter of stringing symbols together to convey meaning. Language is governed by a complex set of rules, known as grammar. The details of these rules differ from language to language, but they share a similar type of complexity. Simple, wordlike sounds do not engage language areas in the same way that words that form part of a language do—the brain just treats them as noises. Some theorists believe that the overarching rules of language—the structure that is common to them all—is embedded in the human brain and is instinctive rather than learned. Although primates have learned how to link visual symbols on keyboards to objects and some can understand sign language, it has not been possible to teach another species spoken language.

SENTENCES AND CONSONANT STRINGS

Several areas in the brain’s left hemisphere become active when people hear a familiar language spoken to them, compared to a small area of the right hemisphere that is active when they hear strings of consonants that do not make any sense.



LEFT HEMISPHERE



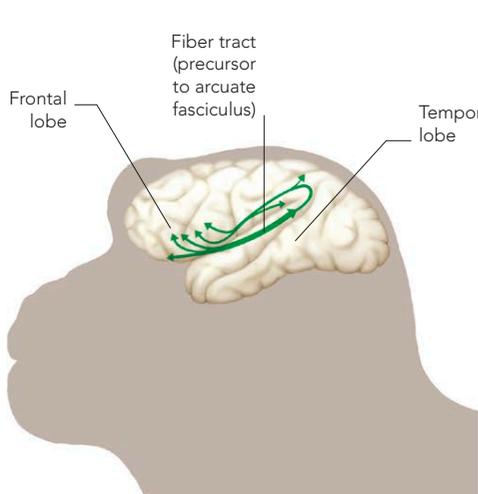
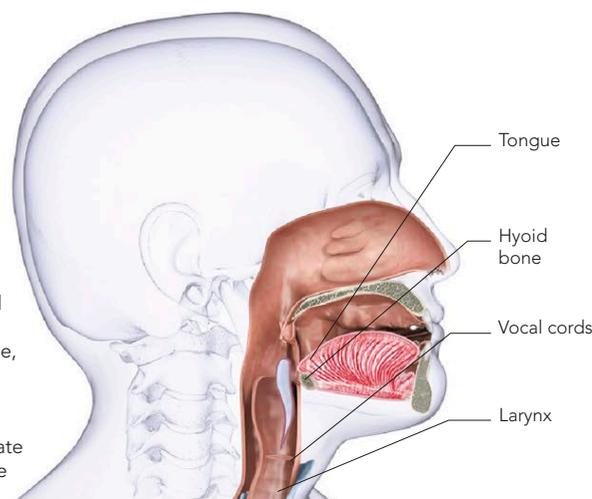
RIGHT HEMISPHERE

THE EVOLUTION OF LANGUAGE

Spoken language leaves no traces in the historic record, so we shall probably never know how or even exactly when it originated. The ability to generate speech and understand language is something only humans possess, although some primates' brains have regions that may function as primitive language areas. An important factor in the evolution of language took place in the throat and larynx, around the time that our ancestors started walking upright. These changes affected the variety and intricacy of the sounds they could produce. This improved ability to communicate probably increased the chances of survival for those who used it most effectively and therefore the chances of it being passed on to subsequent generations.

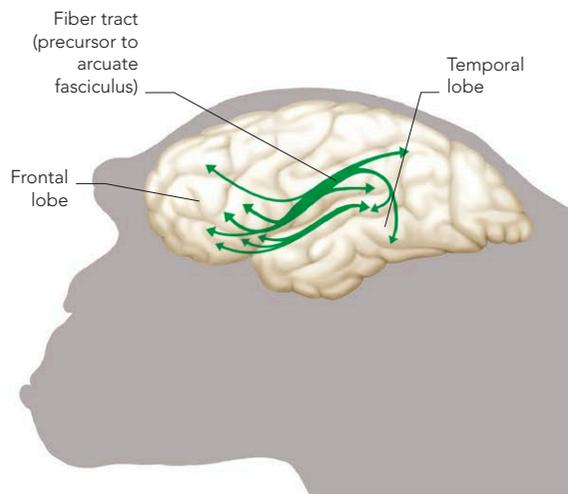
THE ANATOMY OF SPEECH

The altered larynx in upright hominids allowed them to make more inventive noises. It also meant they could no longer swallow and breathe at the same time, leading to an increased risk of choking. The descended hyoid bone is also thought to facilitate the production of a wide range of sounds.



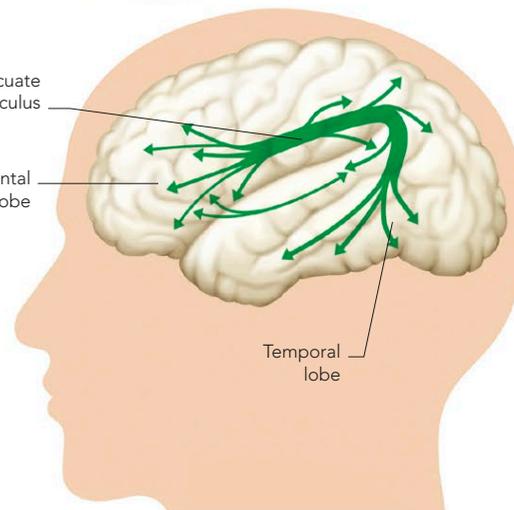
MACAQUE FIBER TRACT

Macaques have simple language areas. A crucial part of this region is a thick bundle of fibers, which links the areas associated with understanding language in the temporal lobe with the areas that generate it, in the frontal lobe.



CHIMPANZEE FIBER TRACT

The connections between the frontal lobe and the temporal lobe are more advanced than in macaques, allowing for improved cognitive abilities, but they do not have such prominent temporal-lobe projections of the fiber tract.



HUMAN FIBER TRACT

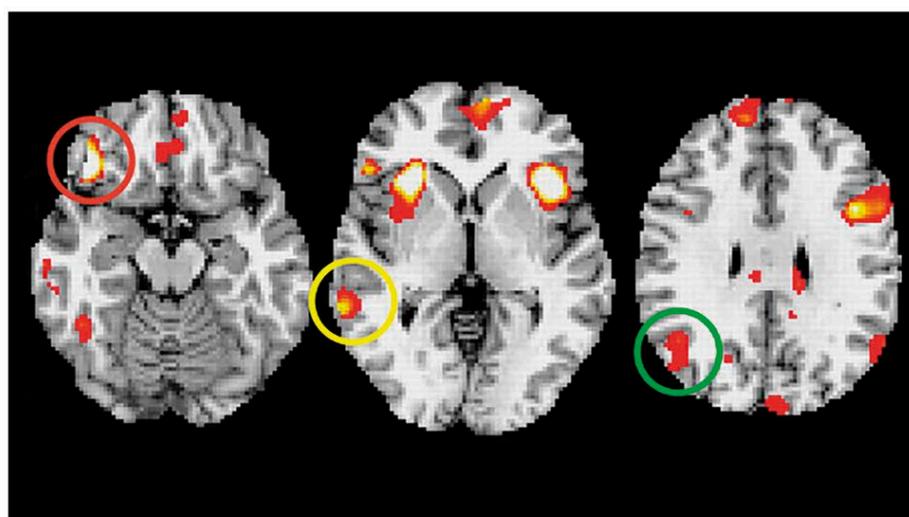
In the human brain, the tract is known as the arcuate fasciculus, connecting two areas crucial for speech and comprehension. It is one of the specializations thought to have led to the evolution of language.

LANGUAGE GENES

Hundreds of genes combine to make language possible, but one gene in particular is associated with the normal development of speech and language. FOXP2 is a gene that helps to connect the many brain areas that work together to produce fluent speech. People with a particular mutation on this gene have a condition known as childhood apraxia of speech. Those affected have problems producing words and in some cases may also have difficulty understanding speech. Animals that communicate through sound, including songbirds, mice, whales, and other primates, also have the FOXP2 gene. However, in humans, it is thought to have evolved further and faster, resulting in the formation of more complex connections in the brain. Certain mutations to the FOXP2 gene—in both the human and animal versions—may produce comparable problems, however. In mice, for instance, a particular change in the gene makes them “stutter” in their squeaking “songs,” just as it does in people.

LANGUAGE AND PERCEPTION

Language is much more than just a way of signaling things to one another—evidence shows that it shapes the way we perceive the world. If your language makes a distinction between blue and green, for example, you will be less likely to confuse a blue color chip with a green one when recalling them, because you will have been able to attach a mental label to each of them. If a language does not distinguish between colors in the same way, it will be more difficult to recall which is which. Similarly, the Amazonian Piraha tribe do not have words for numbers above two and are unable to reliably tell the difference between four and five objects placed in a row.



COLOR STUDY

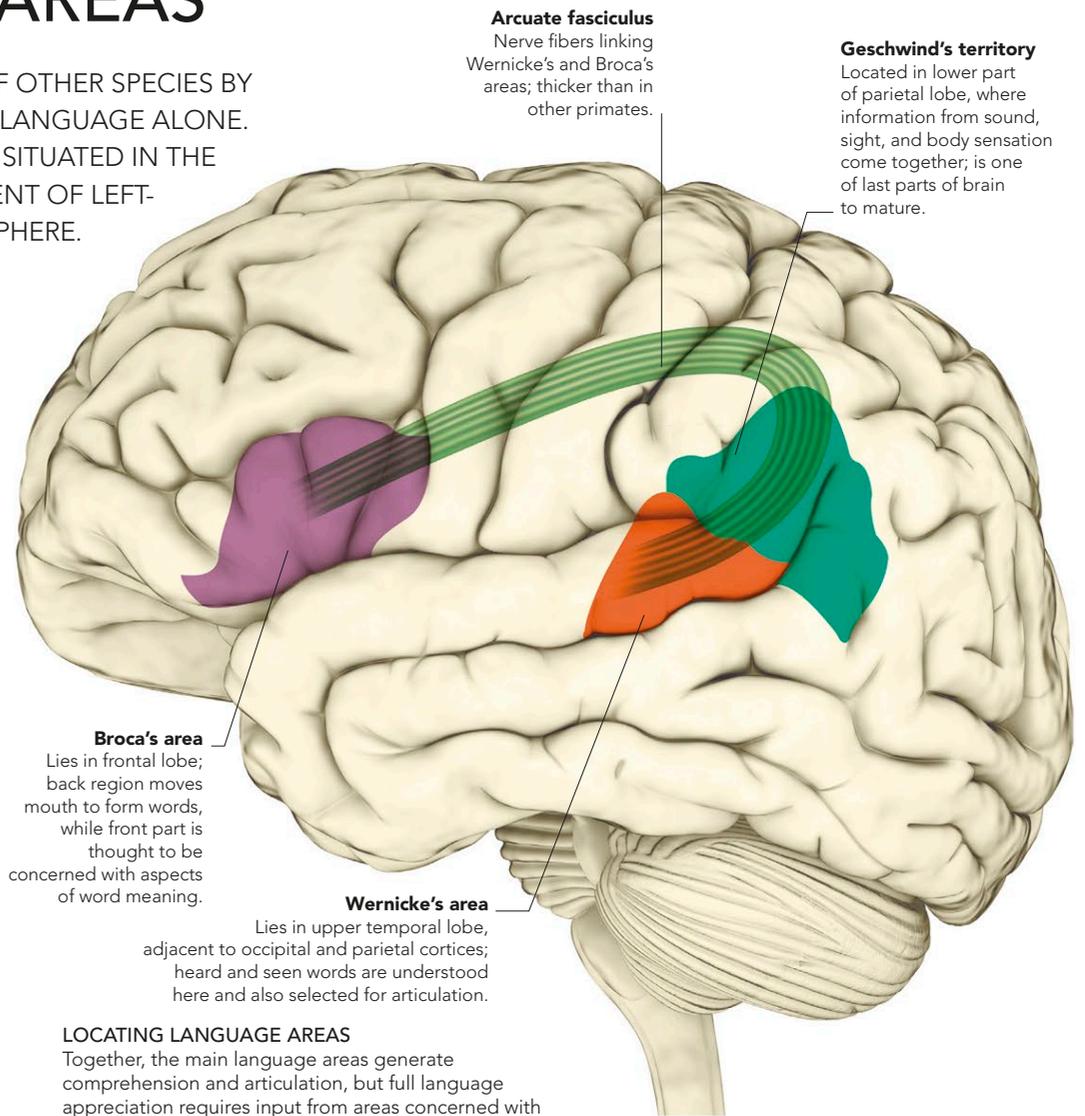
Areas of the brain involved in recognition and word retrieval (circled, left) are engaged more when people distinguish between colors that have different names than between colors that share a name, even if they are visually distinctive.

THE LANGUAGE AREAS

THE HUMAN BRAIN DIFFERS FROM THAT OF OTHER SPECIES BY HAVING A REGION THAT IS DEDICATED TO LANGUAGE ALONE. IN THE VAST MAJORITY OF PEOPLE, THIS IS SITUATED IN THE LEFT HEMISPHERE, BUT IN ABOUT 20 PERCENT OF LEFT-HANDED PEOPLE, IT IS IN THE RIGHT HEMISPHERE.

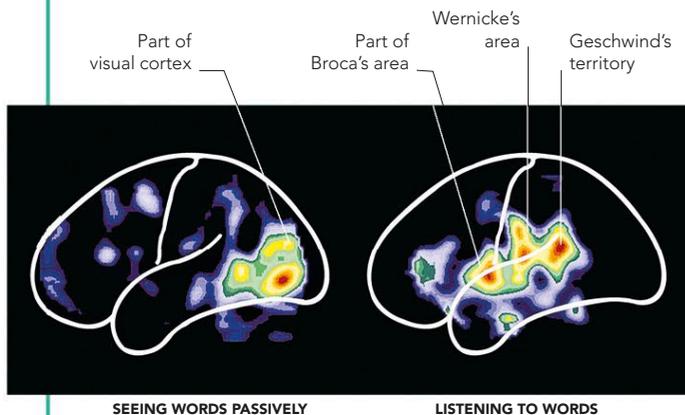
MAIN LANGUAGE AREAS

Language processing occurs mainly in Broca's and Wernicke's areas. Broadly speaking, words are comprehended by Wernicke's area and articulated by Broca's. A thick band of tissue called the arcuate fasciculus connects these two areas. Wernicke's area is surrounded by an area known as Geschwind's territory. When a person hears words spoken, Wernicke's area matches the sounds to their meaning, and special neurons in Geschwind's territory are thought to assist by combining the many different properties of words (sound, sight, and meaning) to provide full comprehension. When a person speaks, the process happens in reverse: Wernicke's area finds the correct words to match the thought that is to be expressed. The chosen words then pass to Broca's area via the arcuate fasciculus (or, possibly, via a more circuitous route through Geschwind's territory). Broca's area then turns the words into sounds by moving the tongue, mouth, and jaw into the required position and by activating the larynx.



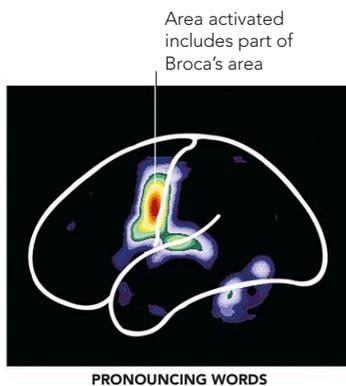
LOCATING LANGUAGE AREAS

Together, the main language areas generate comprehension and articulation, but full language appreciation requires input from areas concerned with tone, emotion, and rhythm.



AREAS ACTIVATED IN DIFFERENT TASKS

These fMRI scans show distinct patterns of activity in the three main language areas, depending on whether the person undertaking the task is listening to speech or pronouncing words. Simply looking at words passively does not involve much activity in the language areas.

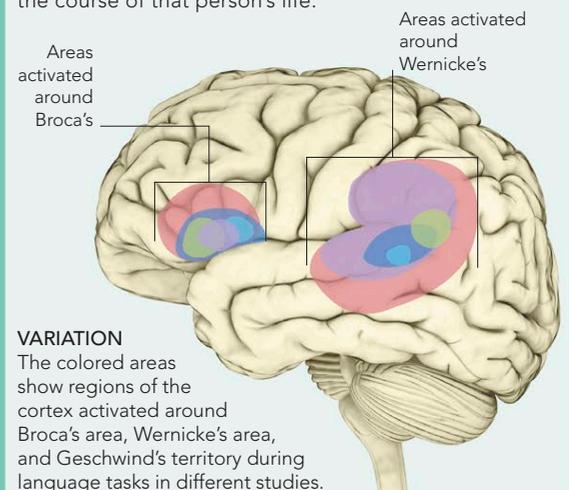


LANGUAGE TASKS

Different types of language tasks activate a number of different areas of the brain. However, the key language areas only become active when language is turned into meaning. So merely looking at words as marks on a page involves areas of the brain such as the visual cortex, which is responsible for processing incoming visual information, whereas listening to spoken words triggers activity in Wernicke's area and Geschwind's territory, signifying that the sounds are being turned into meaningful information. Broca's area is significantly involved in listening, too, because understanding words involves, to some extent, articulating them "in your head" (also referred to as "sounding out"). Broca's area is strongly activated when the task involves pronouncing words, while generating words involves both Wernicke's and Broca's areas, as well as Geschwind's territory.

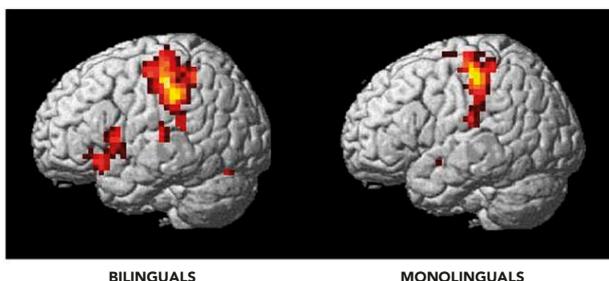
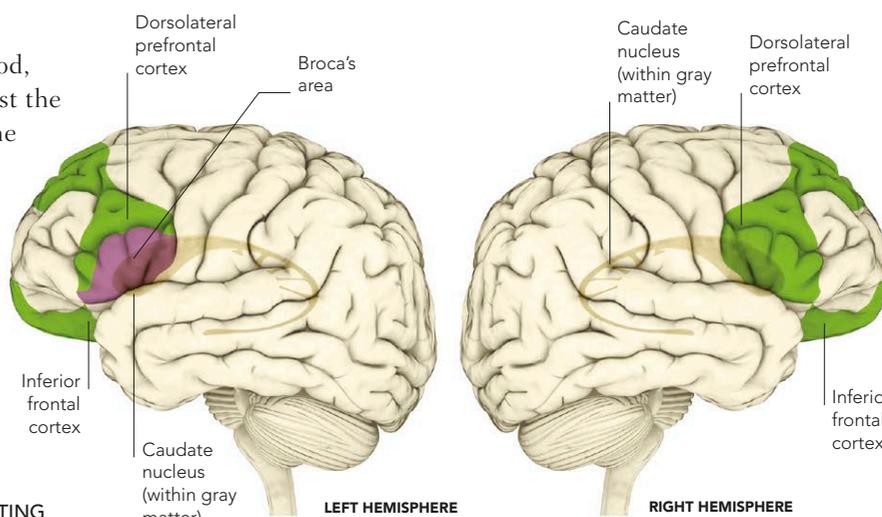
SHIFTING GROUND

Wernicke's and Broca's areas are now well defined, but immediately around them lie large regions of the cortex that become active during a variety of different language studies. Their precise functions remain unclear, and their shapes and locations differ from person to person. Even with a single individual, the peripheral areas engaged in language may shift over the course of that person's life.



THE MULTILINGUAL BRAIN

Being fluent in two languages, particularly from early childhood, enhances various cognitive skills and might also protect against the onset of dementia and other age-related cognitive decline. One reason for this may be that speaking a second language builds more connections between neurons. Studies show that bilingual adults have denser gray matter, especially in the inferior frontal cortex of the brain's left hemisphere, where most language and communication skills are controlled. The increased density was most pronounced in people who learned a second language before the age of five.



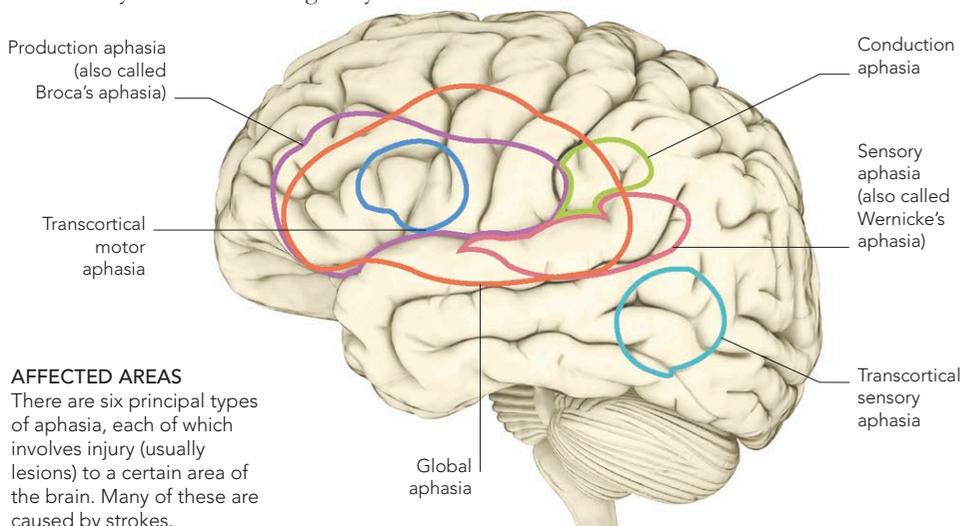
CONTRASTING ACTIVATION
These scans show the brains of bilingual and monolingual individuals when hearing the same language.

NEURAL SIGNATURE OF BILINGUALISM

The purple area is used by both mono- and bilingual individuals when speaking one language; areas in green are activated when bilingual speakers switch languages. The caudate nucleus is also activated during the switch.

LANGUAGE PROBLEMS

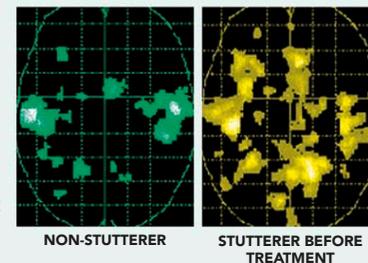
There are a wide range of speech and language problems that can arise from a correspondingly varied number of injuries and impairments. Some problems affect only comprehension, whereas others specifically hinder expression; learning disabilities, such as dyslexia (see p.153) and specific language impairment (see p.248), can affect both. Traumatic brain injuries and strokes can lead to aphasia, which is the loss of the ability to produce and/or comprehend language. By contrast, dysphasia is the partial loss of the ability to communicate, although these terms are often incorrectly used interchangeably.



AFFECTED AREAS
There are six principal types of aphasia, each of which involves injury (usually lesions) to a certain area of the brain. Many of these are caused by strokes.

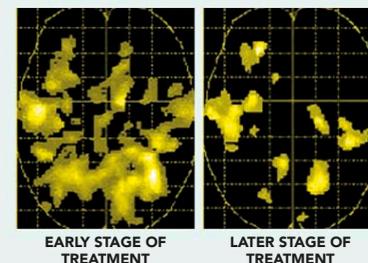
STUTTERING

About 1 percent of people (75 percent of them men) stutter. In most cases, stuttering (also known as stammering) begins between the ages of two and six. Imaging studies have shown that the brains of stutters behave differently from those of non-stutterers when processing speech, in that many more areas of the brain are activated during speech production. It may be that these interfere with one another and cause the stuttering, or it may be the result of stuttering.



TREATMENT FOR STUTTERING

Speech therapy is often successful, as these PET scans show. As treatment progresses, brain activity during speech dies down to near normal.



TYPES OF APHASIA

Aphasia is usually associated with a brain injury (such as a stroke), which affects the brain's language areas. Depending on the type of damage the area affected (see right), and the extent of damage, those suffering from aphasia may be able to speak, yet have little or no comprehension of what they or others are saying. Or they may be able to understand language yet be unable to speak. Sometimes, sufferers can sing but not speak or write but not read.

Production aphasia (damage to Broca's area) Inability to articulate words or string them together; if words can be uttered, they tend to be verbs or nouns, with abnormal tone and rhythm.

Conduction aphasia (damage to link between Wernicke's and Broca's areas) Speech errors include substituting sounds, but good comprehension and fluent speech production.

Global aphasia (widespread damage) General deficits in comprehension, repetition, naming, and speech production; automatic phrases (e.g. reciting numbers) may be spared.

Transcortical sensory aphasia (damage to temporal-occipital-parietal junction) Inability to comprehend, name, read, or write, but with normal ability to recite previously learned passages.

Transcortical motor aphasia (damage around Broca's) Good comprehension but nonfluent speech, often limited to two words at a time. Sufferers retain the ability to repeat words and phrases.

Sensory aphasia (damage to Wernicke's area) Inability to understand language, often combined with general comprehension problems and lack of awareness of own deficiency.

A CONVERSATION

CONVERSATION COMES NATURALLY TO MOST OF US, BUT IN TERMS OF BRAIN FUNCTION IT IS ONE OF THE MOST COMPLICATED CEREBRAL ACTIVITIES WE ENGAGE IN. BOTH SPEAKING AND LISTENING INVOLVE WIDESPREAD AREAS OF THE BRAIN, REFLECTING MANY DIFFERENT TYPES AND LEVELS OF COGNITION.

LISTENING

The sound of spoken words take a short time—about 150 milliseconds—to pass from the speaker's mouth to the listener's ear, for the ear to turn this stimulus into electrical signals, and for this to be processed as sound by the auditory cortex. Words are decoded in Wernicke's area in the left hemisphere, but other areas are also at work to provide full comprehension, including parts of the right hemisphere concerned with tone, body language, and rhythm. If any of these areas are damaged, a person may be left with an incomplete understanding of what is being communicated.

1 50–150 MS AFTER WORDS ARE SPOKEN SOUND REGISTERED

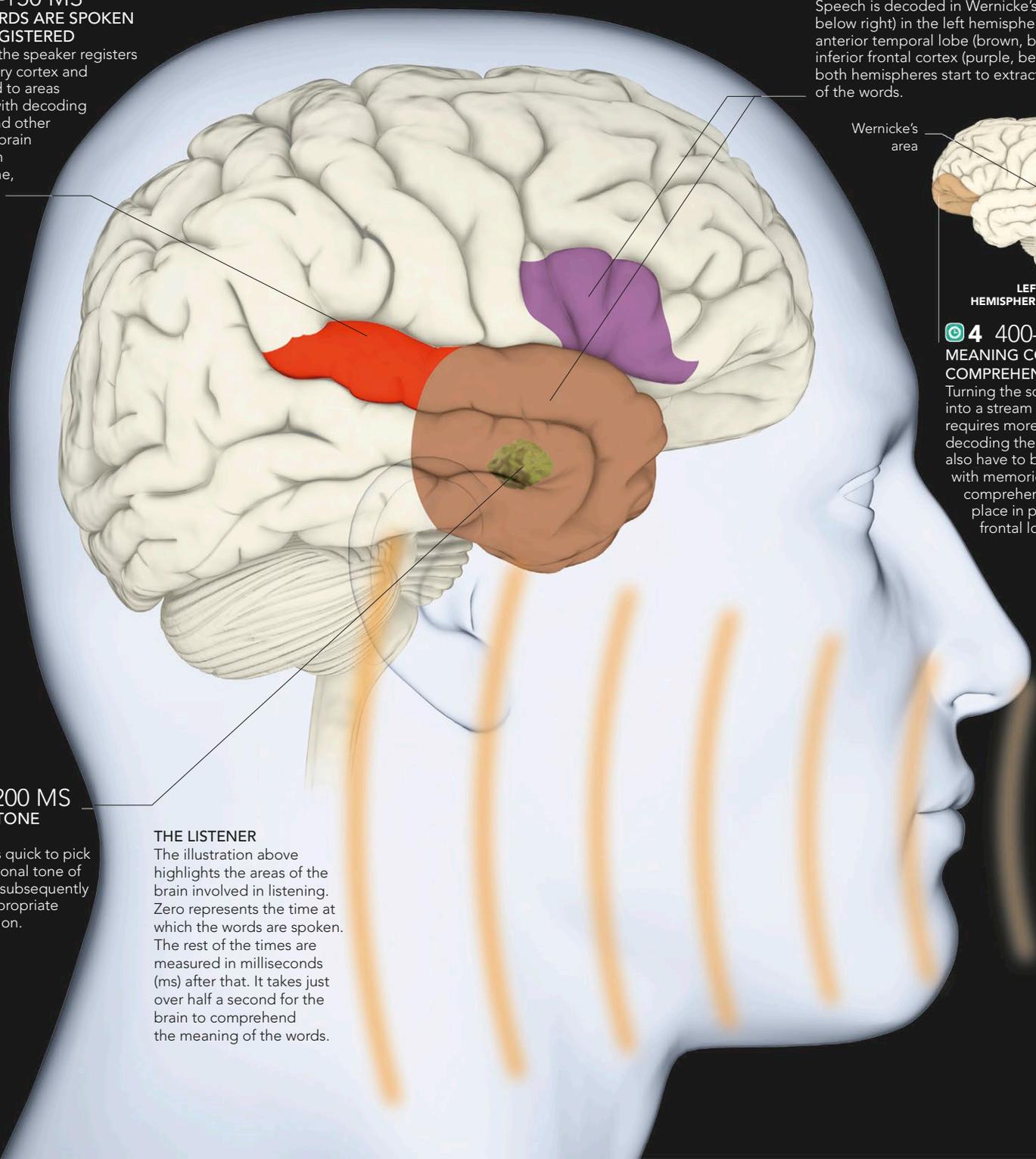
Sound from the speaker registers in the auditory cortex and is distributed to areas concerned with decoding the words and other areas of the brain involved with emotion, tone, and rhythm.

2 150–200 MS EMOTIONAL TONE REGISTERED

The amygdala is quick to pick up on the emotional tone of the speech and subsequently produces an appropriate emotional reaction.

THE LISTENER

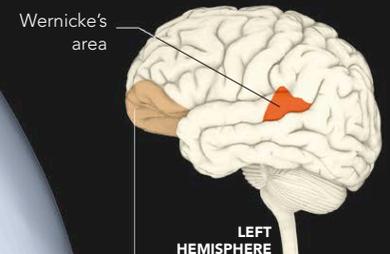
The illustration above highlights the areas of the brain involved in listening. Zero represents the time at which the words are spoken. The rest of the times are measured in milliseconds (ms) after that. It takes just over half a second for the brain to comprehend the meaning of the words.



MORE THAN WORDS

Face-to-face conversations involve more than just decoding words—tone and body language are also part of “understanding.”

3 250–350 MS
**STRUCTURE OF WORD STREAM ANALYZED
AND MEANING OF WORDS EXTRACTED**
Speech is decoded in Wernicke's area (orange, below right) in the left hemisphere. Then, the anterior temporal lobe (brown, below left) and inferior frontal cortex (purple, below left) in both hemispheres start to extract the meaning of the words.



4 400–550 MS
**MEANING CONSCIOUSLY
COMPREHENDED**
Turning the sound of speech into a stream of meaning requires more than just decoding the words—they also have to be associated with memories to give full comprehension. This takes place in part of the frontal lobe.

SPEAKING

The speech process starts about a quarter of a second before words are actually uttered. This is when the brain starts to select the words that are to convey whatever the person wants to say. The words then have to be turned into sounds, and are finally articulated. Most of this complicated activity occurs in specific language areas, which in most people are on the left side of the brain. However, in a minority of people they are situated in the right, or spread between both hemispheres. Right-hemisphere language dominance is more prevalent among left-handers (see p.199).

CRUCIAL PATHWAY

"Prepared" words are transmitted to Broca's area via a bundle of nerve fibers called the arcuate fasciculus. It is much thicker and better developed in humans than in other species, and is thought to be key to the development of language.

2 -200 MS WORDS TO PHONOLOGY

Shortly after they have been retrieved from memory, the words are matched to the sounds in Wernicke's area, which is adjacent to the auditory cortex, where sounds are distinguished.

3 -150 MS PHONOLOGY TO SYLLABLES

Broca's area is the part of the brain most closely associated with speech. It matches the sounds of words to the specific mouth, tongue, and throat movements required to actually voice them.

4 -100 MS ARTICULATION

The mouth, tongue, and throat movements needed to articulate the selected words are directed by the part of the motor cortex that controls these parts of the body.

5 UNDER 100 MS FINE CONTROL OF ARTICULATION

The cerebellum is concerned with orchestrating the timing of speech production. The right cerebellar hemisphere connects to the left cerebral hemisphere, and this shows greatest activation during speech, whereas the left cerebellar hemisphere is more active during singing.

SHIFTING FUNCTIONS

Speech and comprehension problems often result from strokes, which damage the language areas. If the damage happens early in life, the speech functions may shift to the opposite hemisphere. In older people, this is less likely to be successful, but undamaged areas can still take on some functions of the damaged areas.



SPEECH AND LANGUAGE THERAPY

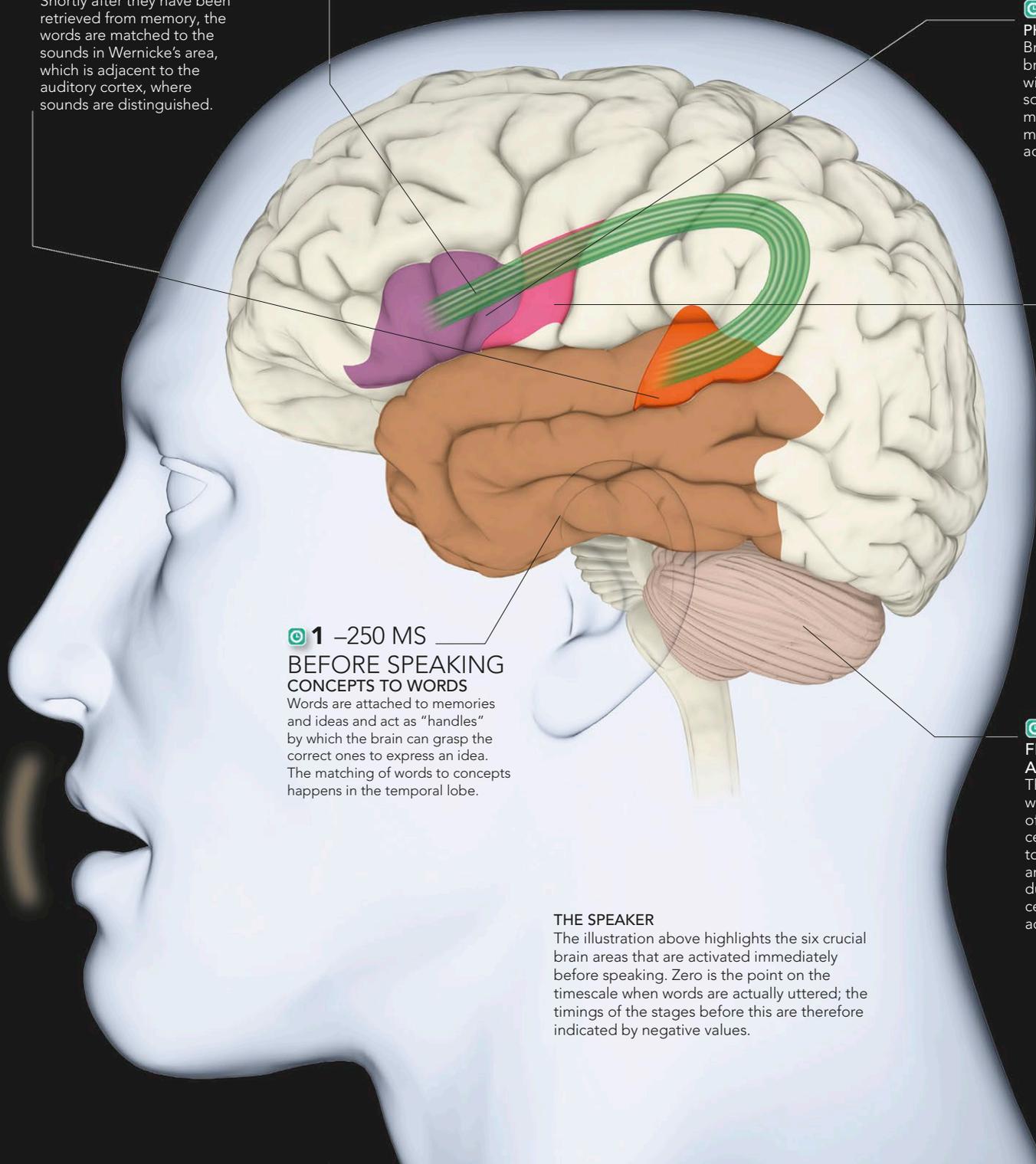
It is possible for people who suffer from aphasia as a result of a stroke to recover some language functions through intense speech and language therapy.

1 -250 MS BEFORE SPEAKING CONCEPTS TO WORDS

Words are attached to memories and ideas and act as "handles" by which the brain can grasp the correct ones to express an idea. The matching of words to concepts happens in the temporal lobe.

THE SPEAKER

The illustration above highlights the six crucial brain areas that are activated immediately before speaking. Zero is the point on the timescale when words are actually uttered; the timings of the stages before this are therefore indicated by negative values.

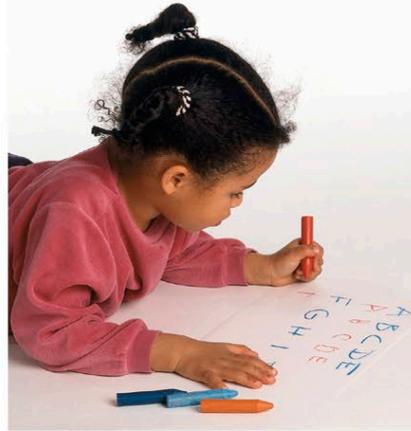


READING AND WRITING

OUR ABILITY TO SPEAK AND TO UNDERSTAND THE SPOKEN WORD HAS EVOLVED SO THAT OUR BRAINS ARE WIRED FOR SPEECH. READING AND WRITING, HOWEVER, DO NOT NATURALLY COME TO US IN THE SAME WAY. IN ORDER TO LEARN TO READ AND WRITE, EACH INDIVIDUAL HAS TO TRAIN THE BRAIN TO DEVELOP THE NECESSARY SKILLS.

LEARNING TO READ AND WRITE

To learn how to read and write, a child has to translate the shapes of letters on the page into the sounds they make if they are spoken aloud. The word “cat,” for instance, must be broken down into its phonological components—“kuh,” “aah,” and “tuh.” Only when the word on the page is translated into the sound that is heard when the word is spoken can the child match it to its meaning. Learning to write uses even more of the brain. In addition to the language areas concerned with comprehension, and the visual areas concerned with decoding text, writing involves integrating the activity in these areas with those concerned with manual dexterity, including the cerebellum, which is involved with intricate hand movements.



VISUAL DISTINCTIONS
Distinguishing between written letters uses a part of the brain that evolved to make detailed visual distinctions between natural objects. This may be why many letters resemble shapes seen in nature.

3 THE AUDITORY CORTEX

Written words are broken into their phonological elements and “sounded out” so they can be “heard”; the auditory cortex allows the reader to recognize each word by the way it sounds.

4 BROCA'S AREA

Once a word has been recognized, it is also “sounded out” in Broca’s area, linking the written word to the spoken word.

5 THE TEMPORAL LOBE

This area helps match the words to their meanings by retrieving memories. Full appreciation of written text—especially fiction—may involve recalling personal memories from the hippocampus.

Hippocampus

2 THE VISUAL WORD-RECOGNITION AREA

This area, which evolved to make fine visual distinctions between different objects, is “hijacked” by the reader’s brain when it is trained to recognize written text.

1 THE VISUAL CORTEX

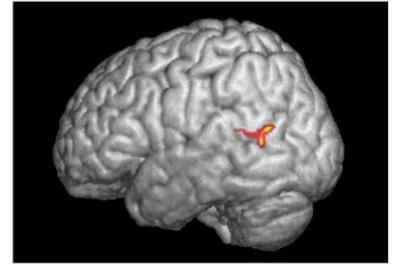
The text is initially processed in the visual cortex, which sends the information along the recognition—processing route toward the language areas of the brain.

BRAIN AREAS USED IN READING

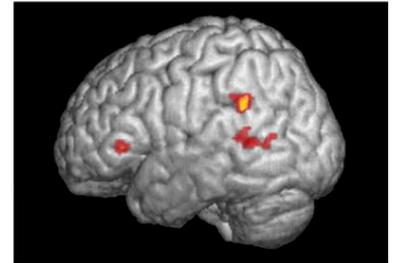
Reading uses various areas across the brain, from the visual cortex at the back to areas of the frontal lobes so that the sound, spelling, and meaning of a word are linked together.

SKILLED READERS

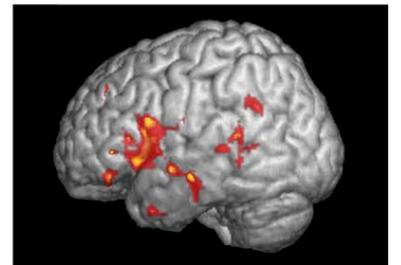
While we are learning to read, our brains have to work very hard to translate the symbols on the page into sounds. This activates an area in the upper rear of the temporal lobe, in which sounds and vision are brought together. The process becomes automatic with practice, and the brain becomes more concerned with the meaning of the words. Hence, the areas concerned with meaning are more active in a skilled reader’s brain (usually an adult’s) during reading.



6-9 YEARS



9-18 YEARS



20-23 YEARS

READING DEVELOPMENT

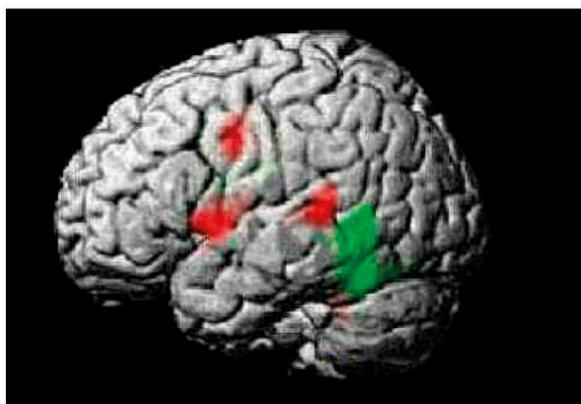
These fMRI scans show that children learning to read rely on a brain area that matches written symbols to sounds (top). As skill develops, areas involving meaning (middle and bottom) become more active.

HOW LITERACY AFFECTS THE BRAIN

Learning to read and write involves building complex new neural connections in many different parts of the brain. This improves a person’s ability to distinguish speech sounds and encourages more and wider mental connections, effectively increasing imagination. Reading people-based fiction has also been found to improve empathy.

DYSLEXIA

Dyslexia is a language-development disorder with a genetic basis. It may affect 5 percent of the population and is most obvious when a language, such as English, has a complex mapping system between speech sounds and letters of the alphabet. One explanation for dyslexia, known as the phonological deficit hypothesis, is that dyslexics cannot analyze and remember the sounds contained in words. This slows down the learning of spoken language and makes it very difficult to map sounds to their corresponding letters of the alphabet when learning to read.



HOW DYSLEXICS DIFFER

Dyslexics differ mostly in the brain area in which words are translated from visual symbols into sounds (shown in green on this fMRI scan). Research has found that dyslexics have more gray matter in this area than nondyslexics, but the significance of this finding is not fully understood.

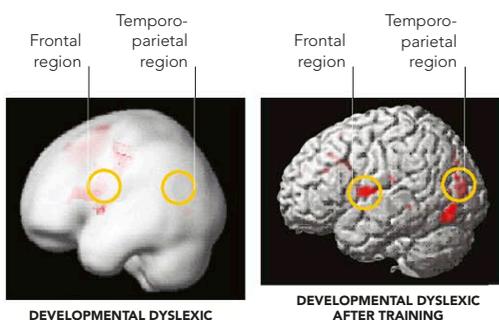
TREATING DYSLEXIA?

There is no cure for dyslexia, but dyslexics can improve reading skills through compensatory learning, using the help of specialist teachers to find ways to remember spellings. While reading is likely to remain slow and spelling error-prone, audio books, spell-checkers, and voice-recognition programs can help circumvent the problems of dyslexia.



VISUAL TECHNIQUES

Some cases of dyslexia are thought to be improved by using colored glasses or by wearing a patch over one eye.



REMIEDIATION

Early studies suggest that a process of listening to slowed-down sounds can aid dyslexics. The circles in the left-hand scan show inactivity in crucial reading areas of a dyslexic's brain; the more detailed right-hand scan shows greater activity in reading areas after training.

HYPERLEXIA

Hyperlexic children exhibit extremely advanced reading and writing skills but may experience difficulty in understanding spoken words. They often have problems with social interaction and may have symptoms of autism. Some hyperlexics learn to spell fairly long words before the age of two and to read sentences by three. Brain scans of one such child suggest that hyperlexia is neurologically opposite to dyslexia in that, when the child was reading, brain areas that are sluggish in dyslexic children were overactive.



PRECOCIOUS READERS

Hyperlexic children are fascinated by letters and numbers and learn how to read from an early age but sometimes find it hard to understand spoken language.

LANGUAGE DIFFERENCES

English speakers have a particularly hard time learning to read. English spelling rules are notoriously difficult to master, and skilled readers know that they cannot rely on letter-to-sound decoding rules, as there are too many exceptions—for example, “i” is pronounced differently in “ice” and “ink.” For dyslexics, these exceptions are difficult to master, and learning to read and spell takes years longer than it does for nondyslexics.



ENGLISH-SPEAKING DYSLEXICS

Learning to read English can be challenging for dyslexics due to the number of words that do not follow standard spelling rules.



ITALIAN-SPEAKING DYSLEXICS

Italian dyslexics are more accurate at word recognition than their English counterparts, since Italian spelling rules are less complex.

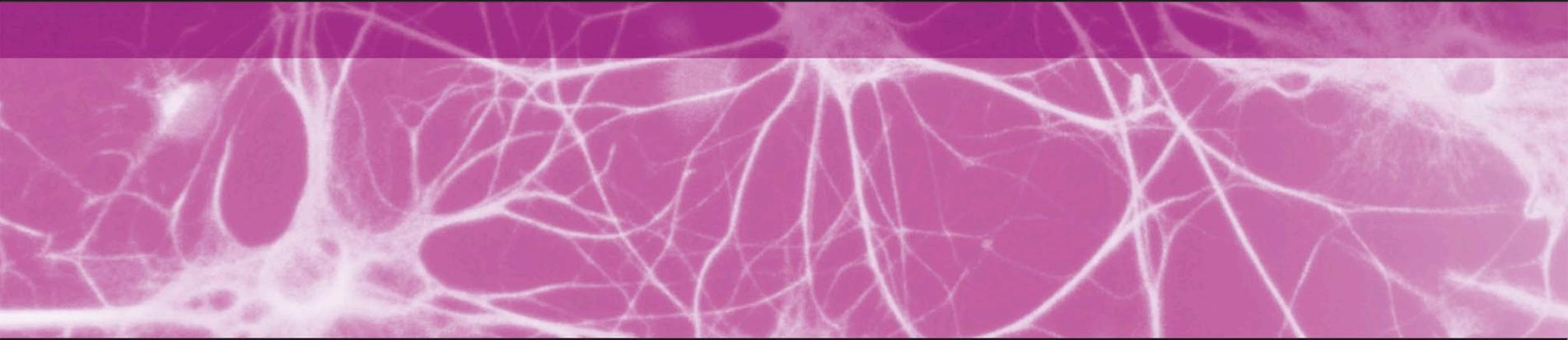
DYSGRAPHIA

Some people have great difficulty writing, even though they may read well. Known as dysgraphia, this may be language- or motor-based. The first is due to difficulty turning sounds into visual marks, while the second is a problem making the fine movements needed to write or difficulty flowing from one such movement to another. Both show up as wobbly, indistinct, or mangled handwriting—far worse than normal. Some letter reversal is normal in young children, but it usually disappears well before adulthood.

tanw zi zint
enitirw rorrim
!skil rtkool

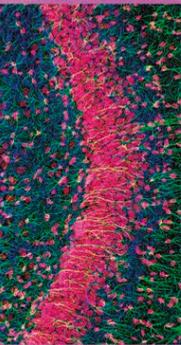
MIRROR WRITING

Fluent mirror writing, in which all the letters are reversed, is very rare and extremely difficult for normal writers to do. It may reflect an abnormal layout of language areas in the brain.



MOST OF OUR MOMENT-TO-MOMENT EXPERIENCES PASS RAPIDLY INTO OBLIVION, BUT A TINY FEW ARE ENCODED IN THE BRAIN AS MEMORIES. WHEN WE REMEMBER AN EVENT, THE NEURONS INVOLVED IN GENERATING THE ORIGINAL EXPERIENCE ARE REACTIVATED. HOWEVER, RECOLLECTIONS ARE NOT REPLAYS OF THE PAST, BUT RECONSTRUCTIONS OF IT. THE PRIMARY PURPOSE OF MEMORY IS TO PROVIDE INFORMATION TO GUIDE OUR ACTIONS IN THE PRESENT, AND TO DO THIS EFFICIENTLY WE GENERALLY RETAIN ONLY THOSE EXPERIENCES THAT ARE IN SOME WAY USEFUL. OUR RECALL OF THE PAST IS THEREFORE SELECTIVE AND UNRELIABLE.

MEMORY



THE PRINCIPLES OF MEMORY

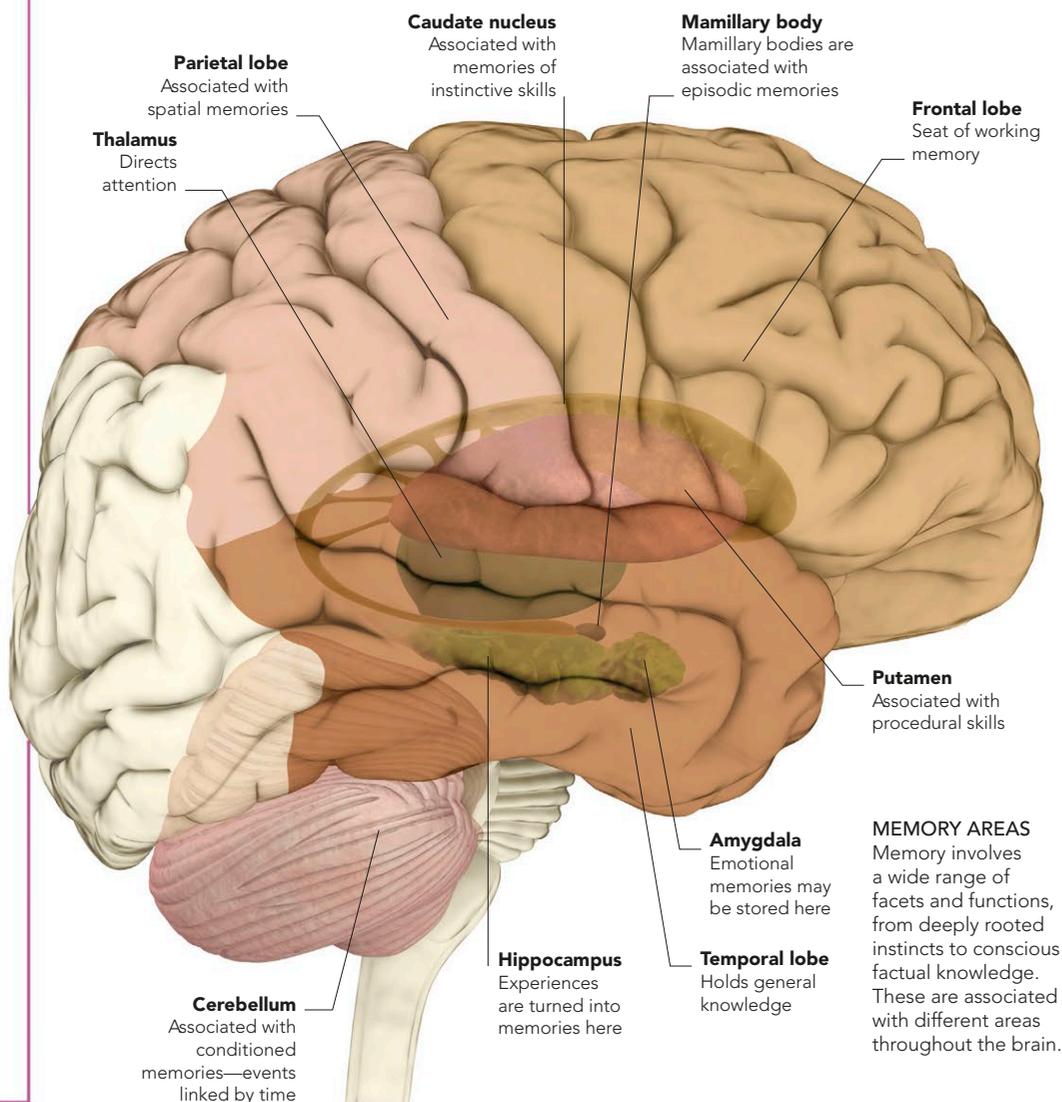
MEMORY IS A BROAD TERM USED TO REFER TO A NUMBER OF DIFFERENT BRAIN FUNCTIONS. THE COMMON FEATURE OF THESE FUNCTIONS IS THE RE-CREATION OF PAST EXPERIENCES BY THE SYNCHRONOUS FIRING OF NEURONS THAT WERE INVOLVED IN THE ORIGINAL EXPERIENCE.

WHAT IS MEMORY?

A memory may be the ability to recall a poem or recognize a face on demand; a vague vision of some long past event; the skill required to ride a bike; or the knowledge that your car keys are on the table. What all these phenomena have in common is that they involve learning, and total or partial reconstruction of a past experience.

Learning is a process in which neurons that fire together to produce a particular experience are altered so that they have a tendency to fire together again. The subsequent combined firing of the neurons reconstructs the original experience, producing a “recollection” of it. The act of recollecting makes the neurons involved even more likely to fire again in the future, so repeatedly reconstructing an event makes it increasingly easy to recall.

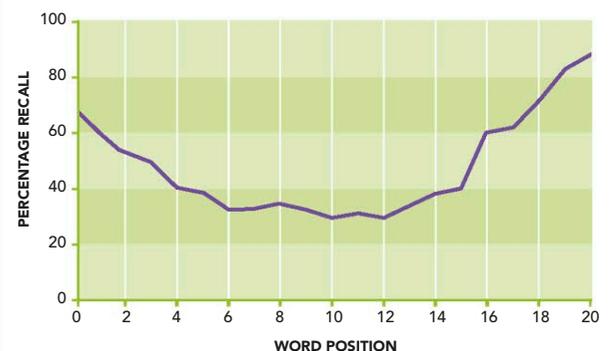
MEMORY PROCESS		
The process of memory formation has several natural stages, from the initial selection and retention of information to recollection and, sometimes, eventual change or loss of the memory. Each stage has particular characteristics—and things that can go wrong.		
STAGE	WHAT'S MEANT TO HAPPEN	WHAT CAN GO WRONG
Selection	The brain is designed to store information that will be useful at a later date and allow the rest to pass by unnoted.	Important events are neglected or irrelevant ones retrieved. You might fail to recall a person's name, but remember the mole on their nose.
Lay-down	Experience selected for memorizing is stored so that it is associated with relevant pre-existing memories and retained for an appropriate period.	Information may be “mis-filed,” with faulty links between items. Or new items are not laid down, so it is hard to learn or to retain new memories.
Recollection	Current events should stimulate the recollection of appropriate memories—i.e. information that can guide future actions.	Current events fail to prompt useful memories, such as words, names, events—you know the information is there but you cannot grasp it.
Change	Each time a memory is recalled it is altered slightly to accommodate new information.	Alteration may create false memories.
Forgetting	Items start to be forgotten as soon as they have been registered, unless they are regularly refreshed. Unnecessary information is deleted.	Important or useful information is forgotten. Alternatively, unnecessary or even damaging memories are not.



SHORT- AND LONG-TERM MEMORY

Short-term memories generally stay with us only as long as we need them. A telephone number you use just once is an example. Short-term memories are held in the mind by a process of “working” memory (see opposite page). Long-term memories, in contrast, can be recalled years or even decades later. The address of your childhood home may be such a memory. In between these extremes, we have many medium-term memories, which may last for months or years and finally fade away.

Many different factors determine whether an experience or item of knowledge is destined to be a short- or a long-term memory. These include their emotional content, novelty, and the amount of effort that we make to practice recalling them.



FIRST AND LAST

If we are asked to learn a list of words, we are more likely to remember the first and last items than those in the middle. This is thought to be because we give the first greater attention, so it “sticks,” while the last may be repeated more than the others because we can do this without another item crowding in behind.

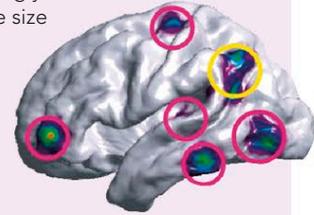
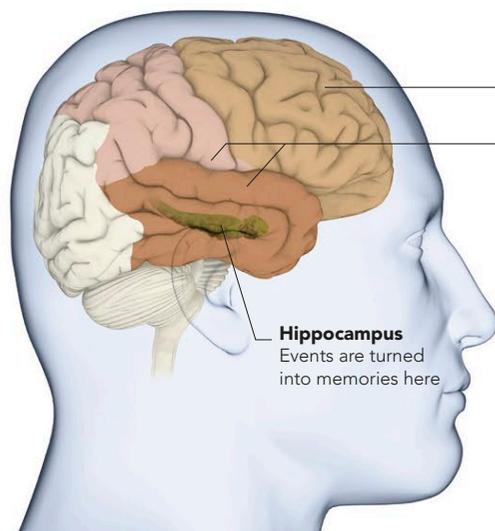
TYPES OF MEMORY

We have five different types of memory, for particular purposes. Episodic memory comprises reconstructions of past experiences, including sensations and emotions; these usually unfold like a movie and are experienced from one's own point of view. Semantic memory is non-personal, factual knowledge that "stands alone." Working memory is the capacity to hold information in mind for just long enough to use it. Procedural "body" memories comprise learned actions, such as walking, swimming, or riding a bicycle. Implicit memories are those we don't know we have. They affect our actions in subtle ways; for example, you might take an inexplicable dislike to a new person because they remind you of someone nasty.

LEARNING IS GOOD FOR YOU

Learning involves making new connections between clusters of neurons in different parts of the brain. This builds up the brain, making it fitter. For example, practicing spatial skills such as finding your way around a city has been shown to increase the size of the rear hippocampus. The more connections you create, the better you can use what you learn and the longer it takes you to forget it.

ENLARGED AREAS
This image shows areas to do with implicit learning (red) and explicit skills (yellow) that have grown denser with practice.

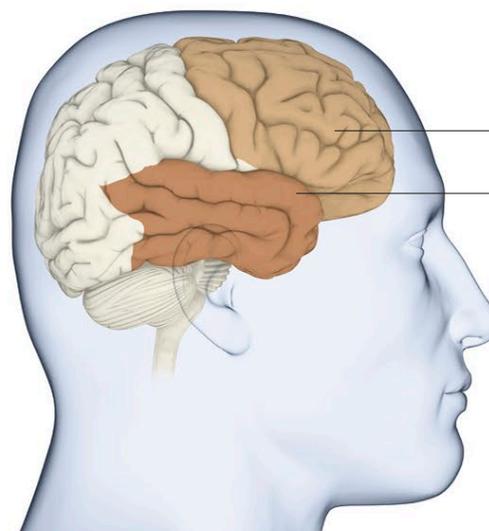
Frontal lobe
Activity here ensures that episodic memories are not mistaken for real life

Cortical areas
Episodic memories activate the areas originally involved in the experience that is being recalled

Hippocampus
Events are turned into memories here



EPISODIC MEMORY
The parts of the brain involved in episodic memories depend on the content of the original experience. Highly visual experiences, for example, will activate visual areas of the brain, while remembering a person's voice will activate the auditory cortex.

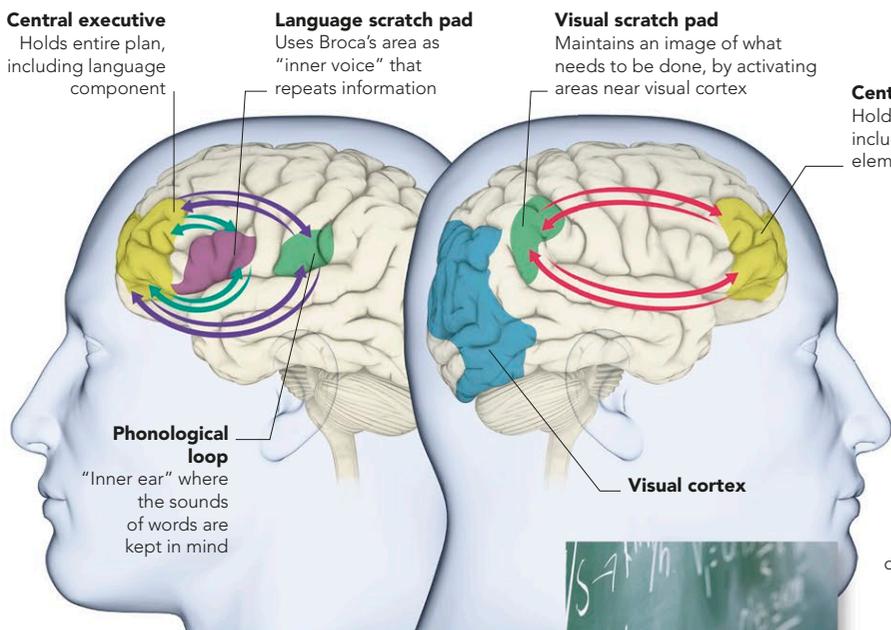


Frontal lobe
Semantic memories are activated by frontal lobe areas that draw on stored knowledge to guide current behavior

Temporal lobe
The temporal lobes encode factual information, and activity here is a marker of facts being recalled



SEMANTIC MEMORY
Semantic memories are facts that may once have had a personal context but now stand as simple knowledge. The fact that a man once walked on the Moon, for example, may once have been part of your personal experience, but now it is just "knowledge."



Central executive
Holds entire plan, including language component

Language scratch pad
Uses Broca's area as "inner voice" that repeats information

Visual scratch pad
Maintains an image of what needs to be done, by activating areas near visual cortex

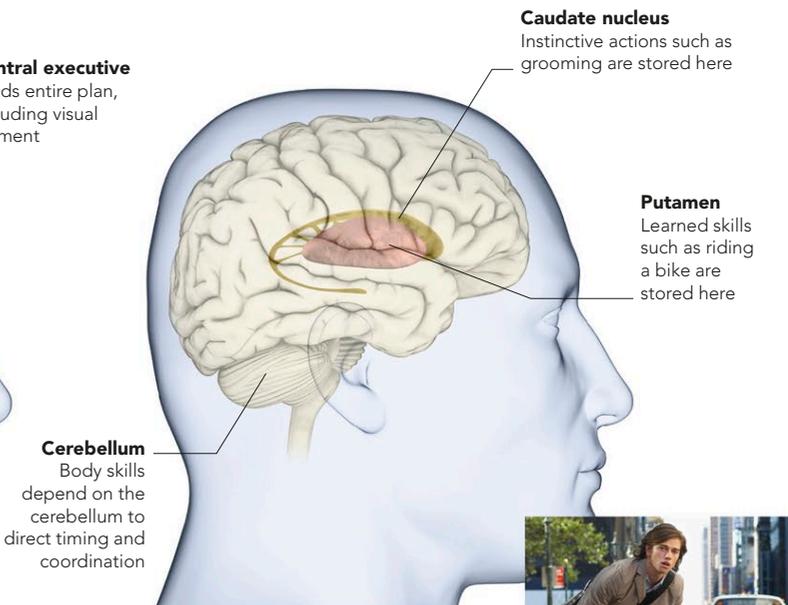
Phonological loop
"Inner ear" where the sounds of words are kept in mind

Central executive
Holds entire plan, including visual element

Visual cortex



WORKING MEMORY
One part of the frontal lobes, the central executive, holds a plan of action while calling up items from the rest of the brain. There are also two neural loops, for visual data and for language; these act as scratch pads, temporarily holding data until it is erased by the next job.



Caudate nucleus
Instinctive actions such as grooming are stored here

Putamen
Learned skills such as riding a bike are stored here

Cerebellum
Body skills depend on the cerebellum to direct timing and coordination



PROCEDURAL MEMORY
"Body" memories allow us to carry out ordinary motor actions automatically, once we have learned them. Such skills are stored in brain areas that lie beneath the cortex. They can be recalled to mind, but usually remain unconscious.

THE MEMORY WEB

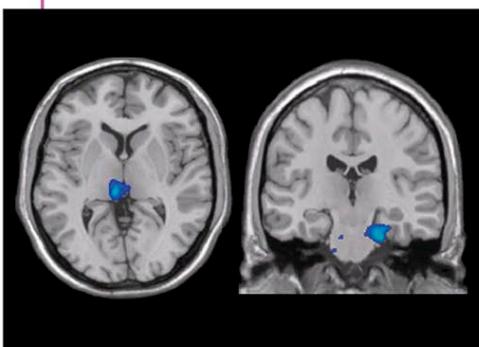
MEMORIES ARE STORED IN FRAGMENTS THROUGHOUT THE BRAIN. ONE WAY TO ENVISAGE THE PATTERN OF MEMORIES IN THE BRAIN IS AS A COMPLEX WEB, IN WHICH THE THREADS SYMBOLIZE THE VARIOUS ELEMENTS OF A MEMORY THAT JOIN AT THE NODES, OR INTERSECTION POINTS, TO FORM A WHOLE, ROUNDED MEMORY OF AN OBJECT, PERSON, OR EVENT.

BRAIN-WIDE WEB

“Declarative” memories—episodes and facts you can bring to mind consciously—are laid down and accessed by the hippocampus but are stored throughout the brain. Each element of a memory—the sight, sound, word, or emotion that it consists of—is encoded in the same part of the brain that originally created that fragment. When you recall the experience, you recreate it in essence by reactivating the neural patterns generated during the original experience that was encoded to memory. Take, for example, the memory of a dog you once owned. Your recall of his color is created by the “color” area of the visual cortex; the recollection of walking with him is reconstructed (in part at least) by the motor area of your brain; his name is stored in the language area, and so on.

RECALLING MEMORY

The fMRI scan to the left shows activity in the sensory cortex when sensory aspects of a memory are recalled. The scan to the right shows the hippocampus, which plays a central role in memory management. Here, the person being scanned is actively suppressing a memory—note the lack of activity in the sensory cortex.



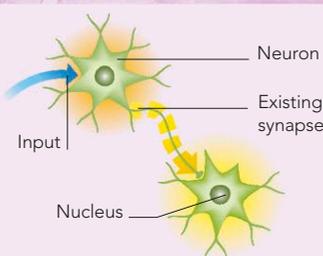
FACETS OF A MEMORY

Once a memory is sparked off, the hippocampus triggers various aspects of it in unison. If you remember a pet dog, different brain areas recall a variety of memories of the dog and peripheral items such as dog bowls, as well as memories of things connected to the idea of “dog.”

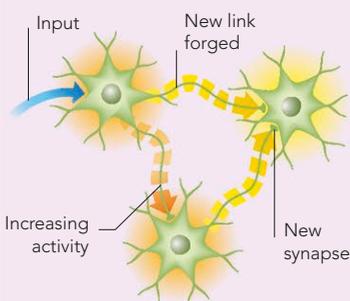


FORMING MEMORIES

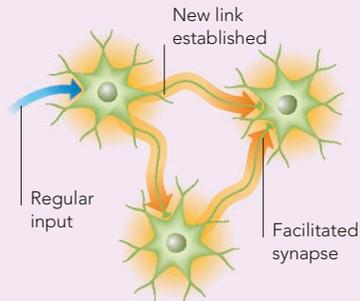
The initial perception of an experience is generated by a subset of neurons firing together. Synchronous firing makes the neurons involved more inclined to fire together again in the future, a tendency known as “potentiation,” which recreates the original experience. If the same neurons fire together often, they eventually become permanently sensitized to each other, so that if one fires, the others do as well. This is known as “long-term potentiation.”



1 INPUT An external stimulus triggers two neurons to fire simultaneously. In future, if one fires, the other is likely to fire, too.



2 CIRCUIT FORMATION A third neuron fires. One of the initial pair is stimulated to fire with it, triggering the second, so the three become linked.



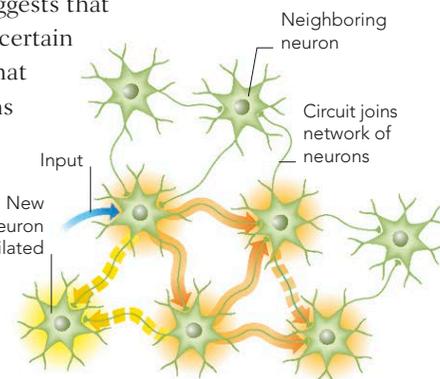
3 INCREASING ACTIVITY The three neurons are now sensitized to one another, so that if one fires, the other two are likely to fire as well.

DISTRIBUTED MEMORIES

Our memories are distributed throughout the brain, so even if one part of an experience is lost, many others will remain. One benefit of such a distributed storage system is that it makes long-term memories more or less indestructible. If they were held in a single brain area, damage to that place—for example, from a stroke or head injury—would eradicate the memory completely. As it is, brain trauma and degeneration may nibble away at memories but rarely destroy them entirely. You may lose a person’s name, for example, but not the memory of their face. Some studies have found that memories persist even when the synapses encoding them are broken. This suggests that neurons themselves may also store certain aspects of memory. One theory is that dendrites—the branches on neurons that receive information from other cells—change sensitivity if they are repeatedly stimulated.

EXPANDING WEB

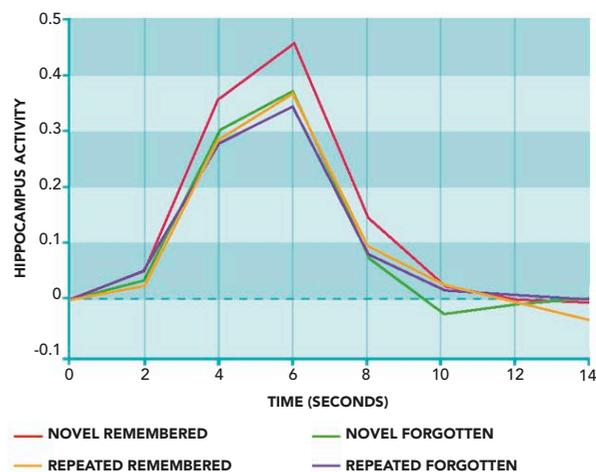
The memory web spreads through the brain as existing neurons make connections with new neurons by firing together.



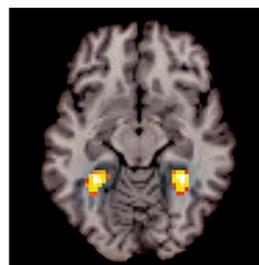


ACCESSING MEMORIES

Events that are destined to be recalled are more strongly encoded to begin with than events that are later forgotten. In one study, 16 people viewed 120 photographs and answered which pictures were taken indoors or outdoors. Each image was then shown once again. After 15 minutes, the subjects were shown the photos again, along with some new ones, and asked if they remembered them. Scans taken during the test show strong activation of the hippocampus in response to recalled photos at the first viewing but less activity in this area when the photos were repeated. This pattern is a “marker” for familiarity (see below).



HIPPOCAMPAL ACTIVITY AND MEMORY FORMATION
 Things that get remembered are marked by high activity in the hippocampus when they are first experienced but less activity when they are seen a second time. This distinguishes the recalled scenes from those that are new or forgotten.

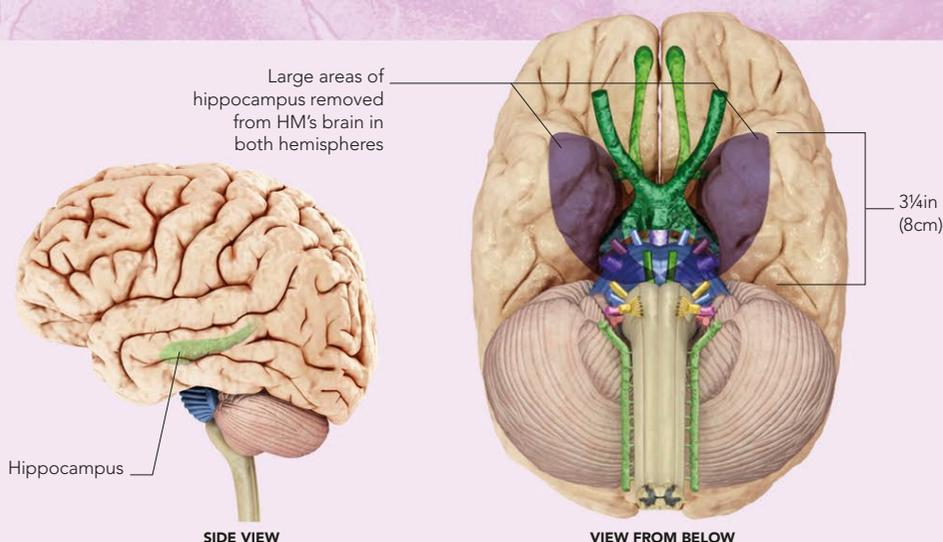


PARAHIPPOCAMPAL ACTIVITY
 When you recall an episode from your life, the hippocampus and the area around it (shown in yellow on this fMRI scan) are activated. During memory recall, the hippocampus is busy pulling together the various facets of the memory from widely distributed areas of the brain.

INABILITY TO STORE

In 1953 surgery was performed on a patient known as HM to relieve the symptoms of severe epileptic seizures. The operation involved removing a large part of the hippocampus. This controlled the seizures, but it also produced a severe memory deficit. From the time HM woke up from the operation, he was unable to lay down conscious memories. Day-to-day events remained in his mind for only a few seconds or minutes. When he met someone, even a person he had seen many times a day, year after year, he did not recognize them. HM believed himself to be a young man right into his 80s, because the years since his operation did not, effectively, exist for him. His case shows how essential the hippocampus is for memory storage.

THE MISSING PIECE
 The hippocampus is embedded deep in the temporal lobes. Experiences “flow through it” constantly, and some of them are encoded in memory through a process of long-term potentiation. Thereafter, the hippocampus is involved in retrieving most types of memory.

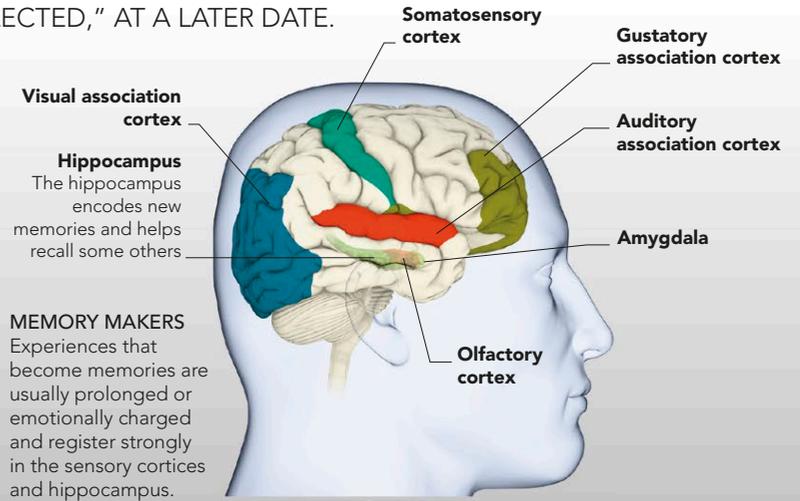


LAYING DOWN A MEMORY

MOST EXPERIENCES LEAVE NO PERMANENT TRACE. A FEW, THOUGH, ARE SO STRIKING THAT THEY ALTER THE STRUCTURE OF THE BRAIN BY FORGING NEW CONNECTIONS BETWEEN NEURONS. THESE CHANGES MAKE IT POSSIBLE FOR THE NEURAL ACTIVITY THAT GENERATED THE INITIAL EXPERIENCE TO BE RECONSTRUCTED, OR “RECOLLECTED,” AT A LATER DATE.

THE ANATOMY OF MEMORY

Only experiences giving rise to unusually prolonged and/or intense neural activity become encoded as memories. It takes up to two years to consolidate the changes that create a long-term memory (see sequence below), but, once encoded, that memory may remain available for life. Long-term memories include events from a person’s life (episodic memories) and impersonal facts (semantic memories). Together, these are termed “declarative memories,” since they can be recalled consciously (“declared”). Procedural (body) memories and implicit (unconscious) memories may also be stored long term.



FORMING A LONG-TERM MEMORY

🕒 0.2 seconds Attention

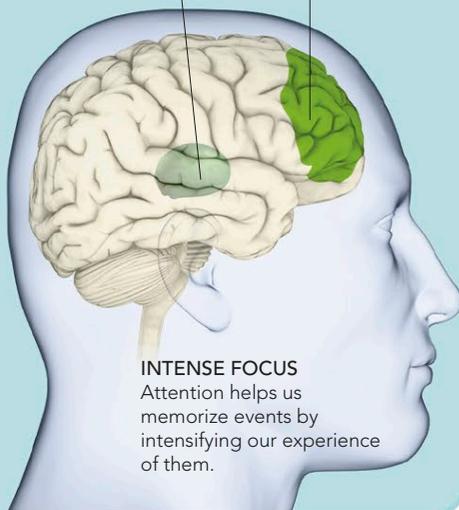
The brain can absorb only a finite amount of sensory input at any point. It can sample a little input about several events at once or focus attention on one event and extract lots of information from that alone. Attention causes the neurons that register the event to fire more frequently. Such activity makes the experience more intense; it also increases the likelihood that the event will be encoded as a memory. This is because the more a neuron fires, the stronger connections it makes with other brain cells.



MEMORABLE EVENT
Zooming in on an event helps capture it as a memory, like a camera taking a snapshot.

Thalamus
Maintains activity in brain regarding target of attention

Frontal lobe
Keeps attention locked to target by inhibiting distractions



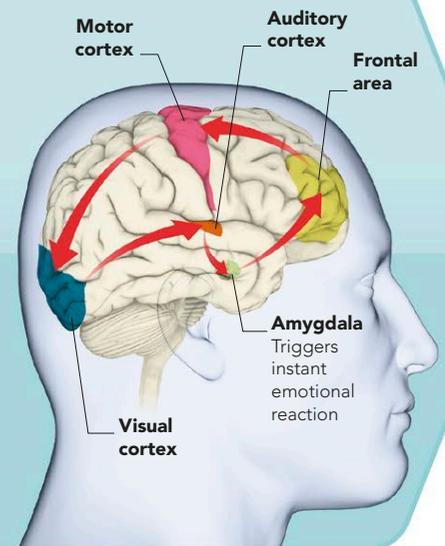
🕒 0.25 seconds Emotion

Intensely emotional experiences, such as the birth of a child, are more likely to be laid down in memory because emotion increases attention. The emotional information from a stimulus is processed initially along an unconscious pathway that leads to the amygdala; this can produce an emotional response even before the person knows what they are reacting to, as in the “fight or flight” response. Some traumatic events may be permanently stored in the amygdala.



EMOTIONAL EVENTS
Personal interactions and other emotional events “grab” attention so are more likely to be stored.

EMOTION PATHWAY
The amygdala helps keep an emotional experience “live” by replaying it in a loop, which begins the encoding of a memory.



🕒 0.2–0.5 seconds Sensation

Most memories derive from events that included sights, sounds, and other sensory experiences. The more intense the sensations, the more likely it is that the experience is remembered. The sensational parts of such “episodic” memories may later be forgotten, leaving only a residue of factual knowledge. For example, a person’s first experience of seeing the Washington Monument may be reduced to the simple “fact” of what the tower looks like.

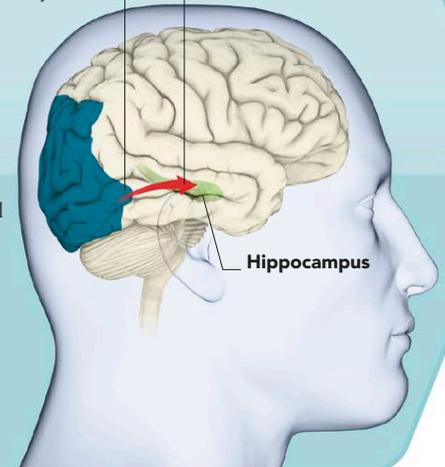


TASTE
Sensory perceptions, such as taste, sight, or smell, form the raw material of memories.

FORMING PERCEPTIONS
Sensations are combined in association areas, to form conscious perceptions.

Sensory cortices
Perceptions start to be formed in sensory cortices

Sensory signal
Information flows to hippocampus

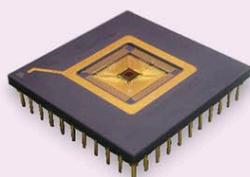


HIPPOCAMPUS REPLACEMENT

Neuroscientists from the University of Southern California, in Los Angeles, have developed an artificial hippocampus that may one day help people with dementia halt memory loss. A small pilot study in which people were fitted with the implant showed that their memory for images improved over their previous performance by nearly 40 percent. The researchers first devised a model of how the hippocampus performs by observing the input-output patterns of the real thing. Then they built the model into a silicon chip designed to interface with the brain, taking the place of damaged tissue. One side of the chip records the electrical activity coming in from the rest of the brain, while the other sends appropriate electrical instructions back out to the brain.

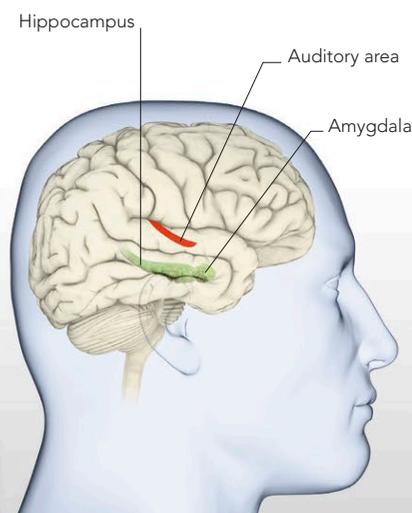
MEMORY CHIP

The chip is designed to be spliced into the hippocampus and communicate with the brain through two arrays of electrodes, placed on either side of the damaged area.



THE LOCATION OF MEMORIES

After consolidation, long-term memories are stored throughout the brain as groups of neurons that are primed to fire together in the same pattern that created the original experience. “Whole” memories are divided into their components (sensations, emotions, thoughts, and so on); each component is stored in the brain area that initiated it. Groups of neurons in the visual cortex, for instance, will encode a sight, and neurons in the amygdala will store an emotion. The simultaneous firing of all these groups constructs the memory in its entirety.



LASTING IMPRESSION
Some memories seem to be cast in stone. In fact, no recollection is ever perfectly sharp or complete.

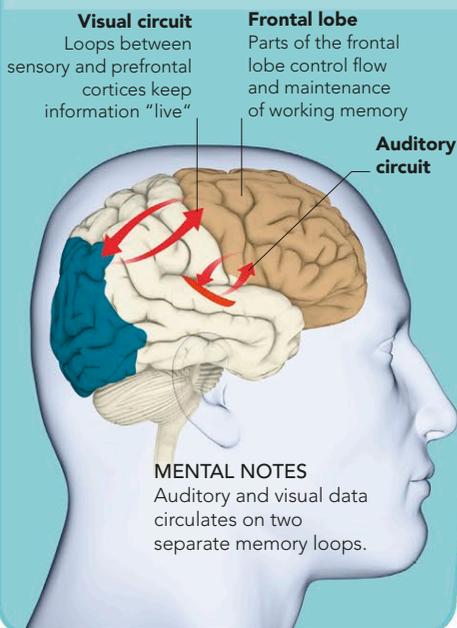


MEMORY STORE

Memories are encoded in the neurons that created them: for example, sounds in the auditory cortex and emotions in the amygdala. The hippocampus pulls them together.

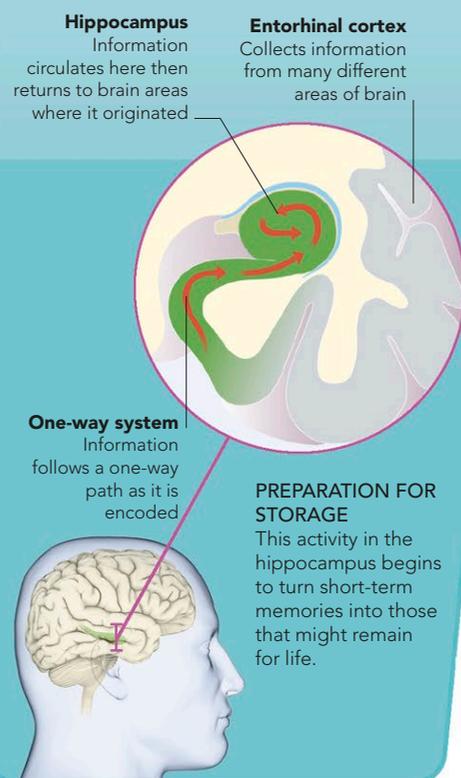
0.5 seconds–10 minutes Working memory

Short-term, or “working,” memory is like text on a blackboard that is constantly refreshed. It begins with an experience and continues as that experience is “held in mind” by repetition. A telephone number, for example, may be repeated for as long as it takes to dial. Working memory is thought to involve two neural circuits (see p.157), around which the information is kept alive for as long as it is needed. One circuit is for visual and spatial information, and the other for sound. The routes of the circuits encompass the sensory cortices, where the experience is registered, and the frontal lobes, where it is consciously noted. The flow of information into and around these circuits is controlled by neurons in the prefrontal cortex.



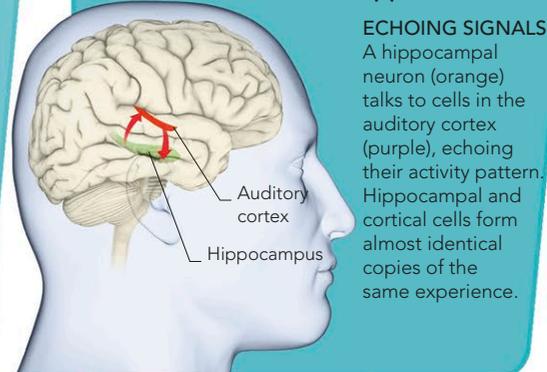
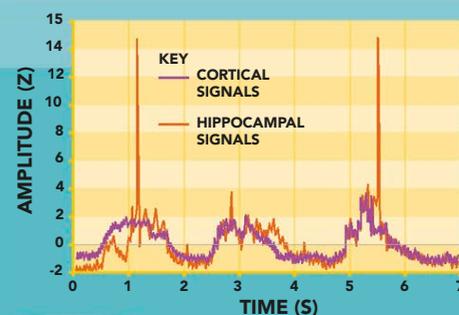
10 minutes–2 years Hippocampal processing

Particularly striking experiences “break out” from working memory and travel to the hippocampus where they undergo further processing. They cause neural activity that loops around coiled layers of tissue; the hippocampal neurons start to encode this information permanently by a process called long-term potentiation (see p.158). The strongest information “plays back” to the parts of the brain that first registered it. A sight, for example, returns to the visual cortex, where it is replayed as an echo of the original event.



2 years onward Consolidation

It takes up to two years for a memory to become firmly consolidated in the brain, and even after that, it may be altered or lost. During this time, the neural firing patterns that encode an experience are played back and forth between the hippocampus and the cortex. This prolonged, repetitive “dialogue” causes the pattern to be shifted from the hippocampus to the cortex; this may happen in order to free up hippocampal processing space for new information. The dialogue takes place largely during sleep. The “quiet” or slow-wave phase of sleep is thought to be more important to this process than rapid eye movement sleep (see p.188).



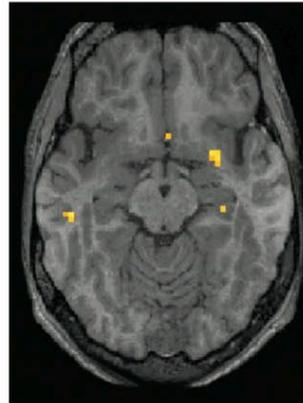
RECALL AND RECOGNITION

MEMORIES OCCUR WHEN THE BRAIN “REPLAYS” A PATTERN OF NEURAL ACTIVITY THAT WAS ORIGINALLY GENERATED IN RESPONSE TO A PARTICULAR EVENT. SO SIMILAR IS THE PATTERN TO THE ORIGINAL THAT THE MEMORY ECHOES THE BRAIN’S PERCEPTION OF THE REAL EVENT. BUT THESE REPLAYS ARE NEVER IDENTICAL TO THE ORIGINAL—IF THEY WERE, WE WOULD NOT KNOW THE DIFFERENCE BETWEEN THE GENUINE EXPERIENCE AND THE MEMORY.

THE NATURE OF MEMORIES

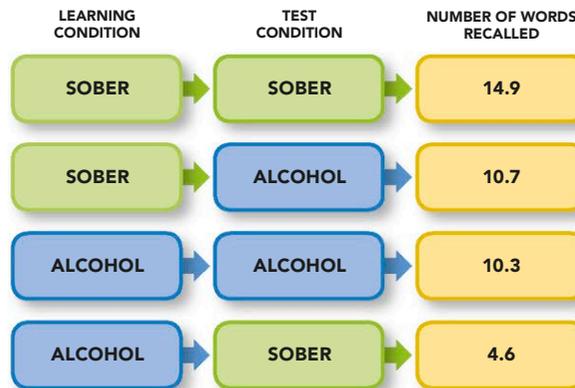
When we recall an event, we reexperience it—but only up to a point. Even when “lost” in reminiscence, we maintain some awareness of the present moment, so the neural activity is not identical to the one that produced the remembered event. Rather, the experience is that of the original mixed with an awareness of the current situation. This experience of remembering “overwrites” the memory, so each time an event is brought to mind it is really a recollection of the last time we remembered it. Hence, memories gradually change over the years, until eventually they might bear very little resemblance to the original event.

SENSORY MEMORY
Tests using fMRI scans show that objects we associate with specific smells spark activity in the olfactory cortex (largest yellow area). In this way, cues trigger all senses, conjuring detailed memories.



STATE-DEPENDENT MEMORY

If you learn or experience something when in a certain state of mind or while concurrently experiencing a particular sensation, you will subsequently recall it more readily when you are again in that state. For example, if you read a book on a sunny beach during a vacation, you may seem to forget it completely when you get home. But years later, on another sunny beach, the plot may come flooding back. Similarly, certain behaviors may be learned when in a particular situation or state of mind, and subsequently displayed only when in the same situation or state of mind, and “forgotten” at other times, giving the impression that the person has more than one personality.



INTOXICATION AND MEMORY
Subjects drank a nonalcoholic or alcoholic beverage prior to studying a list of words, later recalling them while sober or intoxicated. Those intoxicated in both phases recalled more words than those intoxicated in the study phase only.

MEMORY AIDS
Memories of past events are often “jogged” into consciousness when we re-encounter some of the sensations involved in the original experience. Photographs and similar memory aids work in this way. Even if the sensations they trigger are not identical to the original ones, they are likely to be similar enough to jog memories of the same period.

SPATIAL MEMORY

The structure of the human brain reveals just how important spatial orientation and memory are for our species. The whole parietal lobe of the brain—the area under the crown of the skull—is given over to “maps” of our bodies and of our position in space. Also, a sizeable part of the hippocampus is concerned with registering the landscape through which we travel and laying down memory maps. Damage to either of these areas can seriously affect a person’s ability to find his or her way around. If the “navigation” area of the hippocampus is affected by stroke or injury, for instance, a person may lose the ability to remember new routes.



MAZE-MINDED
People who can find their way out of mazes use the hippocampus in both hemispheres. Those who remain lost use one side only.

“THE KNOWLEDGE”

Some people have better memories for places than others. In part, this is a matter of habit and training—those whose lives depend on their ability to find their way around vast tracts of land naturally attend more closely to landmarks. London taxi drivers, for example, are famously adept at finding their way around the city’s labyrinthine streets. Their skill is developed during a two-year training, known as “the knowledge,” during which time they “exercise” the part of the hippocampus responsible for spatial memory. The training seems to increase the size of the hippocampus, much as a muscle is enlarged by weight training.



NATURAL NAVIGATORS
A brain-scanning study found that the rear hippocampus, which encodes spatial memories, is larger in taxi drivers.

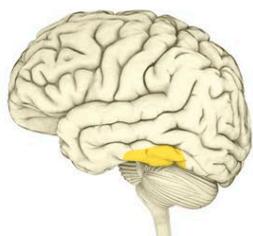


DÉJÀ VU AND JAMAIS VU

Déjà vu is characterized by a sudden, intense feeling of familiarity and the sense that you have experienced the same moment before. One explanation for this is that the new situation triggers a memory of a similar experience in the past, but the recollection is confused with the present as it is recalled, creating a sense of recognition without bringing to mind the previous event. Research suggests that déjà vu occurs when a new situation is mistakenly “marked” as familiar when processed in the limbic system. Jamais vu, by contrast, occurs when one is in a situation that should be familiar, but which seems strange. You might suddenly find your own home to be unfamiliar, for instance. Jamais vu is thought to be a glitch in recognition, whereby the emotional input that usually accompanies familiar experiences fails to occur.

RECOGNITION

Recognizing a person fully involves collating a huge number of memories. They include different types of facts about the person—I know him/he owns a dog/he walked right past me the last time I saw him/his name is Bill. At the same time, you have an emotional reaction to the person based on memories, which produces the feeling of familiarity. Most or all of this happens unconsciously—you see the person and immediately “know” who it is.



FACE-RECOGNITION AREA

SITE OF RECOGNITION

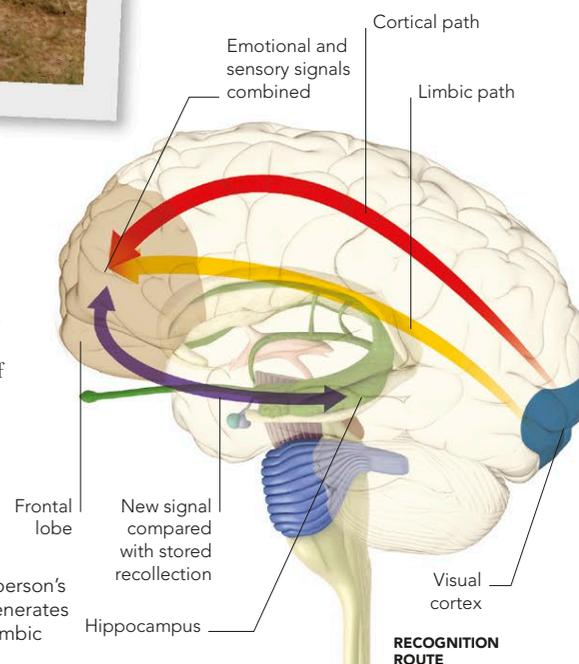
This area processes the sight of a face (see p.84) by extracting information about expression and familiarity.

EMOTIONAL RECOGNITION

When you spot someone you know, the information is first processed by the visual cortex, and is then shunted through the brain along different pathways (see pp.84–85) as shown on this diagram (right). One path travels through the limbic areas that generate a sense of familiarity—separate from conscious recognition—when a familiar person is seen. If this route is blocked, a person may recognize consciously that they know a person, but feel strangely detached from them. Without this input, even one’s nearest relatives would feel like strangers.

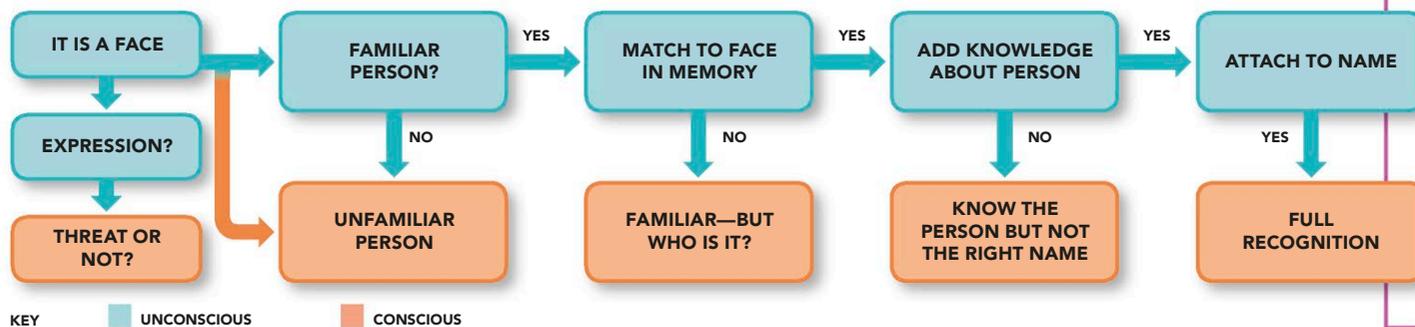
RECOGNITION PATHWAYS

The cortical path (red) processes data about a person’s movements and intentions. Another (purple) generates conscious knowledge of who a person is. The limbic path (yellow) generates a sense of familiarity.



RECOGNIZING A PERSON

Recognizing a person and assigning them their correct name is a complicated process. When it works properly, it seems easy, because it happens unconsciously and apparently instantly. But if the process fails at any stage, recognition is incomplete.



UNUSUAL MEMORY

“BAD” MEMORY USUALLY MEANS FORGETTING. BUT THERE ARE MANY OTHER TYPES OF MEMORY PROBLEMS: CLEAR BUT FALSE RECOLLECTION, BLURRED MEMORIES, AND INTRUSIVE FLASHBACK MEMORIES OF TRAUMATIC EVENTS. IT IS EVEN POSSIBLE TO REMEMBER THINGS TOO CLEARLY.

FORGETTING

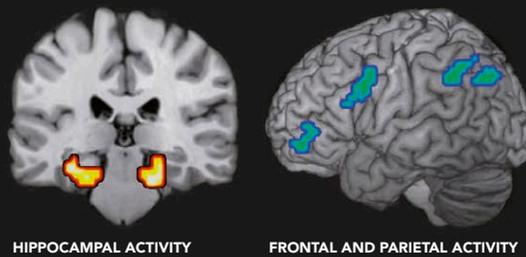
The purpose of human memory is to use past events to guide future actions, and keeping a perfect and complete record of the past is not a useful way to achieve this. It is more important to be able to generalize from experience. When you first drive a car, for example, you learn the pedal positions of the first vehicle you use. Subsequently, when you get in any car, you assume that the pedal positions are the same. The specific memory of the layout of one particular car is lost while the general knowledge, the position of the pedals, is retained. Forgetting specifics is not a fault—it is essential.

FALSE MEMORY

Our brains sometimes lay down memories that are false from the start. This usually happens because an event is misinterpreted. For example, if you expect to see a particular thing, something similar may easily be mistaken for it. The memory will be of what was assumed to be there, rather than what really was. False memories can also be created during what seems like recall. If a person is persuaded that a given thing happened, the event may be “patched together” from scraps of other memories and then experienced as a “real” recollection.

CONFIDENT RECALL

True memory (left) sparks activity in the hippocampus, which “lays down” memory. Confident recall of false memory (right) activates frontal areas associated with familiarity rather than precise recollections.

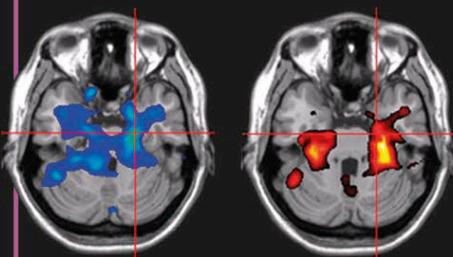


HIPPOCAMPAL ACTIVITY

FRONTAL AND PARIETAL ACTIVITY

TRAUMATIC MEMORY

Post Traumatic Stress Disorder (PTSD) is a condition in which people have vivid “flashback” memories of a traumatic experience (see p.233). Such memories can ambush a person out of the blue—the sound of a car backfiring, for example, may plunge a soldier back into the middle of a gunfight, complete with the emotions experienced at the time. Emotionally traumatic experiences are by their nature more likely to be remembered because emotion amplifies experience. Yet there is also a strong incentive to put such events “out of mind,” and it seems the brain has a mechanism that can make this possible. Experts have found that the brain is able to block memories at will (see below).



ACTIVE SUPPRESSION

ACTIVE RECALL

ACTIVE MEMORY

Emotional memory recall activates the hippocampus and amygdala (emotion). If the memory is suppressed, there is less activity in these areas and in brain areas that recreate the sensations associated with the recalled event.

SUPER MEMORY

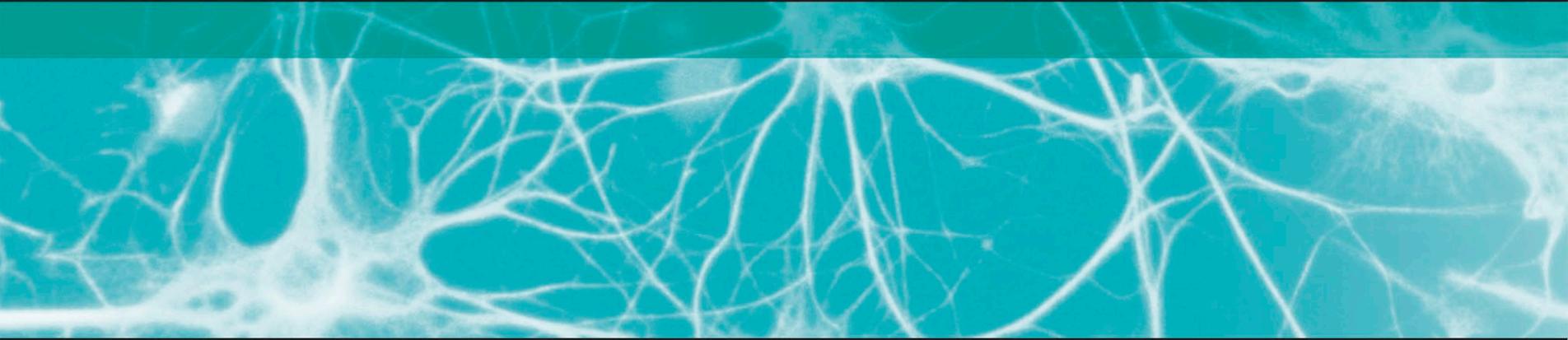
Some people have extraordinarily clear memories for events that happened to them or that were of particular interest to them. For example, an American woman can recount details of every TV program she has seen, and an Australian woman recalls every birthday she’s had since she was one. The condition is called hyperthymesia, and brain scans of people who display it often show markers suggestive of synesthesia or obsessive-compulsive disorder. It is also associated with autism, though not exclusively.





REMEMBERING IN DETAIL

A small number of people known as autistic savants remember things in such detail that they can reproduce them perfectly, even years later. This drawing of Westminster and the Thames River, by Stephen Wiltshire, was produced from memory after a brief tour of London.



DECIDING WHAT TO DO IN A COMPLEX WORLD TAKES THOUGHT. BY THINKING WE CAN EXPLORE THE POTENTIAL CONSEQUENCES OF OUR ACTIONS IN OUR IMAGINATION. THIS, IN TURN, INVOLVES HOLDING ONE OR MORE IDEAS IN MIND AND MANIPULATING THEM. THINKING IS AN ACTIVE, CONSCIOUS, ATTENTION-DEMANDING PROCESS THAT USUALLY DRAWS ON SEVERAL AREAS OF THE BRAIN. THINKING UNDERPINS SOME PARTICULARLY HUMAN ABILITIES AND TENDENCIES, INCLUDING CREATIVITY AND THE CONSTRUCTION OF IMAGINATIVE EXPLANATIONS FOR OUR EXPERIENCES.

THINKING

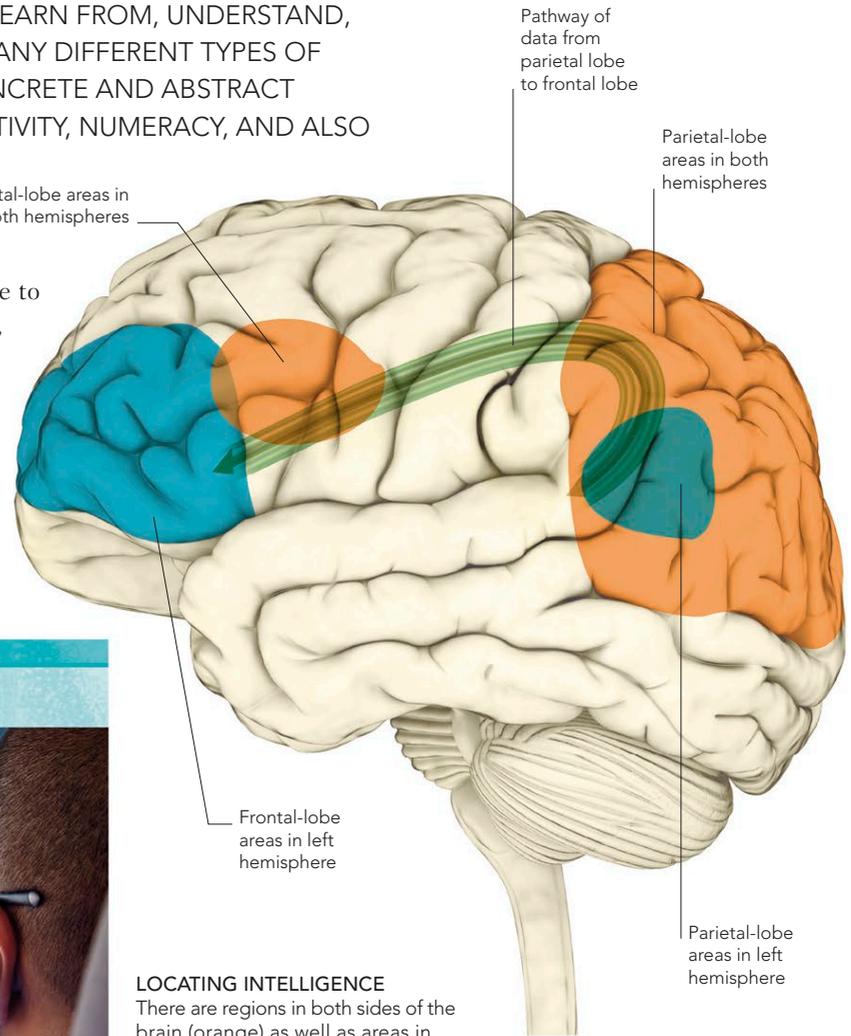


INTELLIGENCE

“INTELLIGENCE” REFERS TO THE ABILITY TO LEARN ABOUT, LEARN FROM, UNDERSTAND, AND INTERACT WITH ONE’S ENVIRONMENT. IT EMBRACES MANY DIFFERENT TYPES OF SKILLS, SUCH AS PHYSICAL DEXTERITY, VERBAL FLUENCY, CONCRETE AND ABSTRACT REASONING, SENSORY DISCRIMINATION, EMOTIONAL SENSITIVITY, NUMERACY, AND ALSO THE ABILITY TO FUNCTION WELL IN SOCIETY.

THE BRAIN’S SUPERHIGHWAY

The frontal lobes are thought to be the seat of intelligence, as damage to these areas affects the ability to concentrate, make sound judgments, and so on. Yet frontal-lobe damage does not always affect a person’s IQ (“intelligence quotient,” measured by testing spatial, verbal, and mathematical dexterity), so other brain areas must also be involved. Research suggests that intelligence relies on a neural “superhighway” linking the frontal lobes, which plan and organize, with the parietal lobes, which integrate sensory information. The speed at which the frontal lobes receive ready-to-use data via this route may affect IQ, as does the extent to which frontal-lobe activity is enhanced by education.



LOCATING INTELLIGENCE

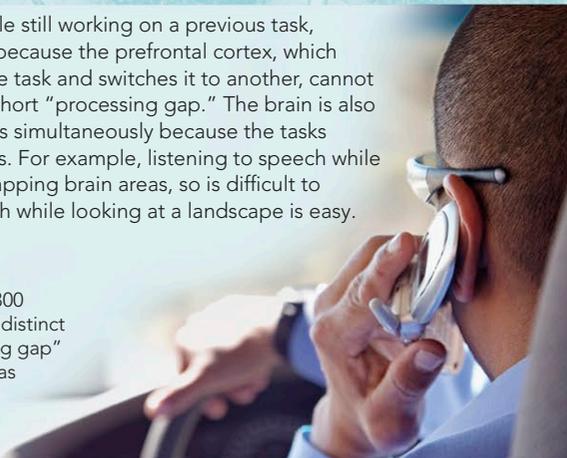
There are regions in both sides of the brain (orange) as well as areas in the left hemisphere only (blue) that are strongly associated with intelligence and reasoning. The arcuate fasciculus (green), a thick bundle of nerve fibers, provides a neural link between the parietal and frontal lobes.

WHY WE CAN'T DO TWO THINGS AT ONCE

If you try to do something while still working on a previous task, your brain stalls. This may be because the prefrontal cortex, which disengages attention from one task and switches it to another, cannot do so instantly, resulting in a short “processing gap.” The brain is also unable to do two similar things simultaneously because the tasks compete for the same neurons. For example, listening to speech while reading words activates overlapping brain areas, so is difficult to achieve, but listening to speech while looking at a landscape is easy.

JUGGLING TASKS

The brain needs a minimum of 300 milliseconds to switch from one distinct task to the next. This “processing gap” makes a task combination such as talking on a phone while driving potentially lethal.

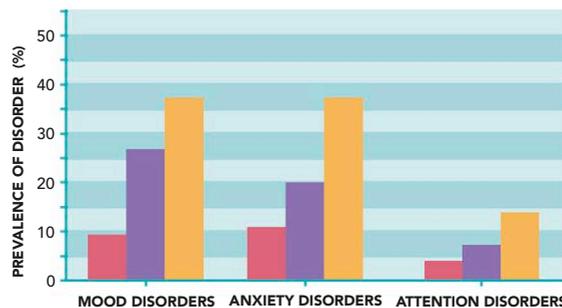
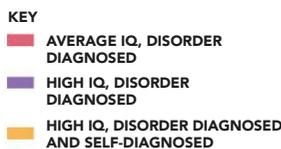


THE DARK SIDE OF BEING BRIGHT

Having a high IQ is generally advantageous, but it is associated with mental ill health. A study of members of MENSA, a club for people with high IQ, found a disproportionate number suffered mental problems. The reason for the link is not clear—it might be because intelligence often coincides with creativity, which is associated with abstract thoughts rather than practical matters. Grappling with big ideas may create stress, which triggers some conditions. Studies suggest high IQ is a sign of hyper brain activity, which also manifests as mental instability.

SUPERCHARGED

Some researchers think high IQ may signify a hyperactive brain, within a hyperactive body. This may result in vulnerability to a range of conditions.



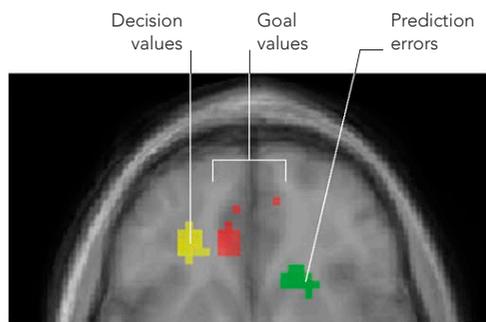
WHAT CONTRIBUTES TO INTELLIGENCE?

Tests for IQ measure general intelligence rather than quantity of knowledge or the level of a specific skill. A score of 100 is average, and the vast majority of people fall in the range of 80–120. High scores are correlated with a number of both social and physical factors.

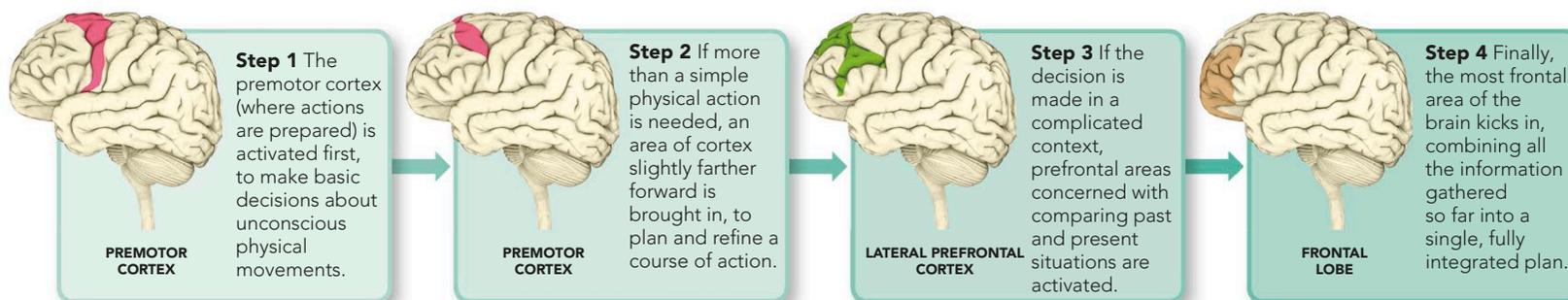
FACTOR	EFFECT
Genes	There are thought to be about 50 different genes related directly to IQ, but so far very few have been identified. Identical twins raised apart typically have very similar IQs, even when raised in strikingly different environments.
Brain size	Those with bigger brains compared to other members of the same sex seem to have a slight intelligence advantage. Overall size, however, may be less important than the size, or neural density, of areas concerned with reasoning.
Signaling efficiency	The smoothness and speed of neural signaling may determine how much information is available for action and how well it can be integrated into plans. Depression, fatigue, and some types of illness reduce efficiency.
Environment	A stimulating social environment in infancy is essential for normal brain development and continues to be important throughout childhood. Verbal interaction seems to be especially useful for IQ.

MAKING DECISIONS

Intelligence is largely the ability to make sensible decisions, which involves calculating pros and cons. First, the brain assesses the “goal value”—the reward expected as a result of the decision. Next, it calculates the “decision value”—the net outcome, or the reward minus the cost. Finally, the brain makes a prediction of how likely it is that the decision will deliver the reward envisaged, which can be compared with the actual outcome, giving a “prediction error.” The more complex the problem, the more the frontal areas of the brain are involved.



ACTIVATION MAP
Activity in the medial orbito-frontal cortex correlates with goal values (red); activity in the central orbitofrontal cortex (yellow) correlates with decision values; and activity in the ventral striatum, part of the caudate nucleus and putamen, correlates with prediction errors (green).

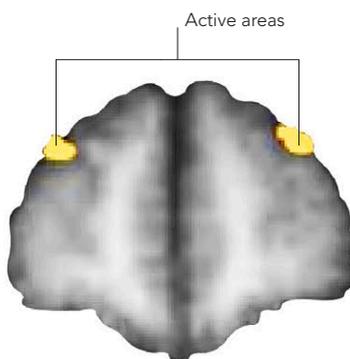


THE ROLE OF EMOTIONS

Decision-making and judgment are profoundly affected by emotions. This is because emotion “drives” action—without it, the brain is like a car with steering but no power. Moods may have a profound effect on the outcome of decision-making. Being in a pleasant, anxious, or neutral mood, or experiencing extreme emotion, can have a significant short-term influence on areas of the brain that are critical for reasoning, intelligence, and other types of higher cognition.

MOODS

The ventrolateral prefrontal cortex is shown in fMRI scans to work harder if a person is in the “wrong” mood for a task, perhaps by stifling emotions.



DECISION—OR PREDICTION?

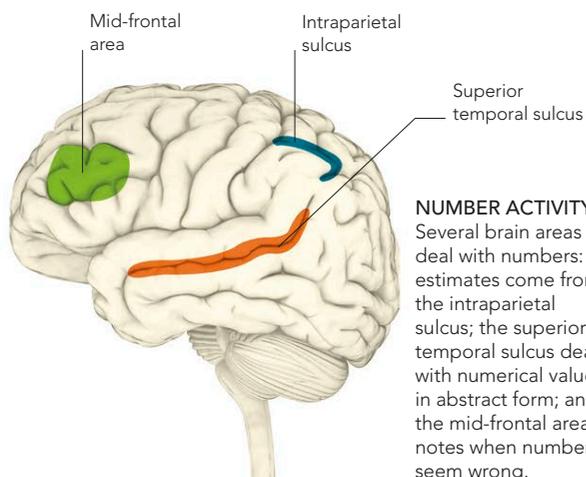
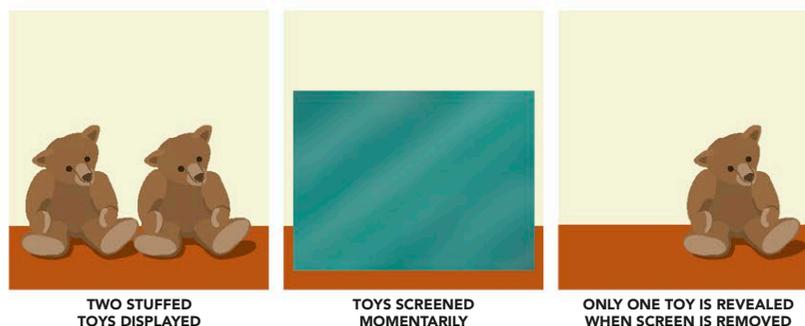
When we make a conscious “decision,” it feels as though we could have chosen something else instead, in other words, we seem to be exercising free will. Experiments show, however, that the conscious decision to do a voluntary act arises after the brain has unconsciously computed what to do and sent the appropriate instructions to the muscles (see p.193). This suggests that a “decision” marks the moment at which we know what we are about to do—a prediction rather than a choice.

THE NUMERICAL BRAIN

Number sense seems “hardwired” into the human brain. Babies as young as six months can spot the difference between one and two. One study recorded electrical activity from babies’ brains while they watched a pair of soft toys. The toys were then momentarily screened, and one was removed then the screen was lifted to reveal just one toy. The babies’ brains registered the “error” by activating the same circuit known to mark error detection in adults, suggesting that even very young babies are able to recognize such discrepancies.

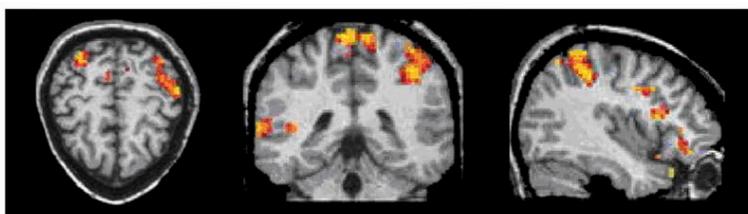
TESTING BABIES

When two toys in this test “become” one, the brains of babies register an error, showing they can discriminate between one and two.

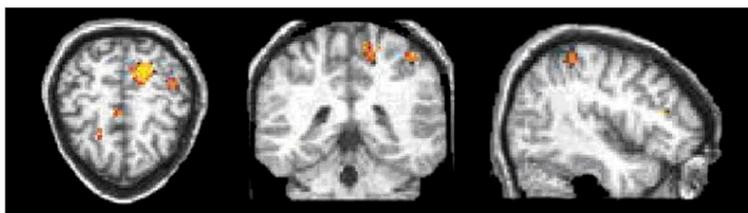


NUMBER ACTIVITY

Several brain areas deal with numbers: estimates come from the intraparietal sulcus; the superior temporal sulcus deals with numerical values in abstract form; and the mid-frontal area notes when numbers seem wrong.



FMRI SCANS OF ADULT BRAINS



FMRI SCANS OF CHILDREN'S BRAINS

NUMBER DEVIATION

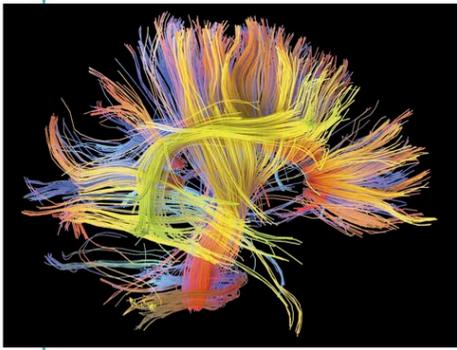
When confronted with a numerical “error,” such as the number of items on view unexpectedly changing, children’s brains register the change in an area that estimates quantities of what is seen. Adults engage both this area and one concerned with abstract numbers. This suggests that the ability to “guesstimate” develops earlier than the ability to think of numbers in the abstract and also that, as numeracy develops, our brains deal with numbers in different ways.

CREATIVITY AND HUMOR

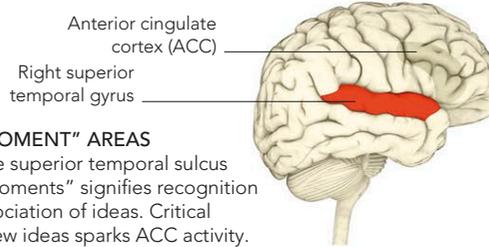
CREATIVITY IS THE ABILITY TO RECONFIGURE WHAT YOU KNOW, OFTEN IN THE LIGHT OF NEW INFORMATION, AND COME UP WITH AN ORIGINAL CONCEPT OR IDEA. IN ORDER TO BE CREATIVE, A PERSON MUST BE CRITICAL, SELECTIVE, AND GENERALLY INTELLIGENT.

THE CREATIVE PROCESS

Our brains are bombarded with stimuli, much of which is filtered out before it reaches consciousness. Focusing on immediate tasks is vital in day-to-day life, but to be creative it is necessary to open our minds to new inputs and memories that may not seem useful. This process allows us to connect things that otherwise stay apart. The brain state most conducive to kindling new ideas is relaxed attentiveness, or the resting state (see p.184), characterized by alpha waves (see p.181). Being creative involves connecting information and reconfiguring it to make something new. The resting state allows information to flow around the brain. The “eureka moment” that occurs when several thoughts combine into a new idea is marked by a change in brain activity involving a shift to the temporal lobe and anterior cingulate cortex. A period of critical assessment may follow, marked by a switch from the resting state to a task-oriented pattern centered on frontal lobe activity.



WHOLE BRAIN CONNECTIVITY
When the brain defocuses, information flows more freely around its highways of connecting fibers, as shown in this DTI scan.

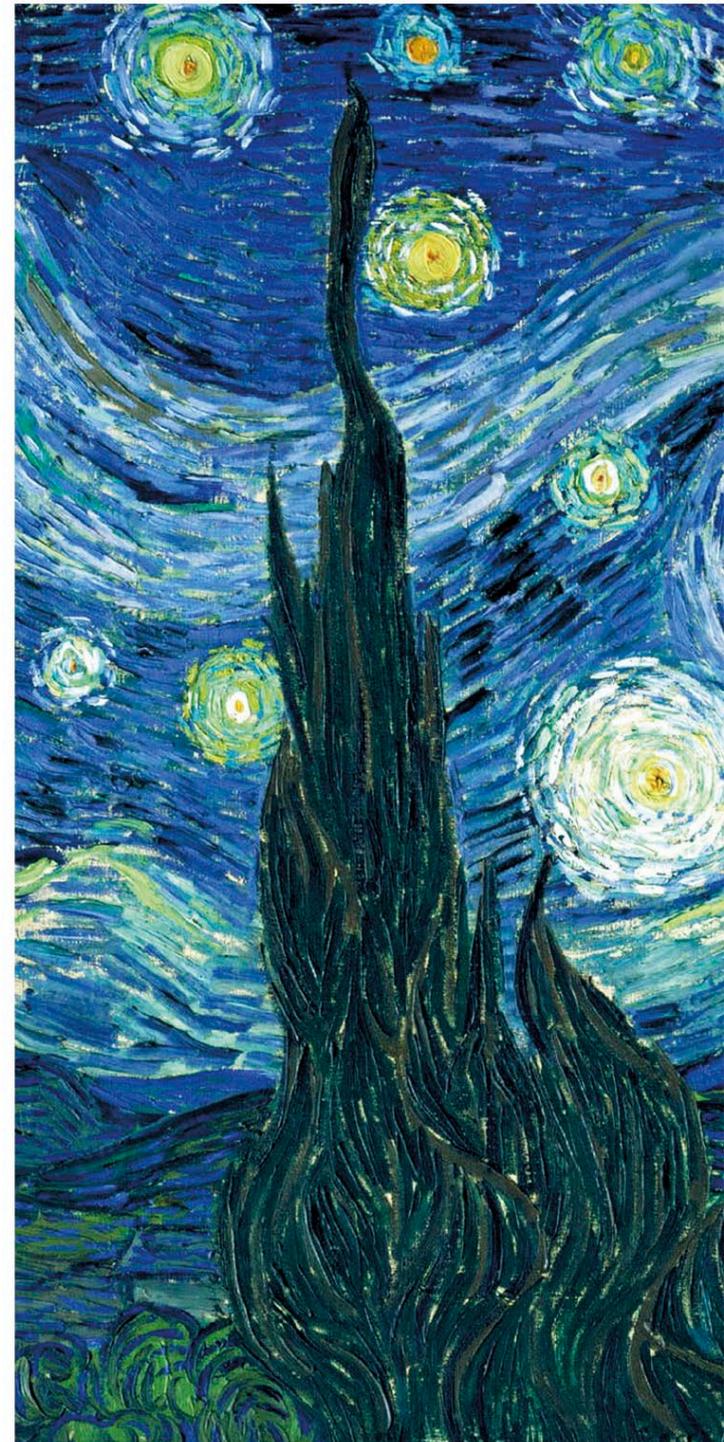


“EUREKA MOMENT” AREAS
Activity in the superior temporal sulcus in “eureka moments” signifies recognition of a new association of ideas. Critical analysis of new ideas sparks ACC activity.

CREATIVE INDIVIDUALS

Everyone is creative, but those who can put their brains into “idle” on demand are more likely to open up their minds to new possibilities and generate original ideas. This process only works, however, if the brain is already “primed” with knowledge that can be combined with the new material. Artists who have mastered the basics of their discipline, for instance, have a foundation of knowledge onto which improvements and changes can be fused. Their expertise allows this process to operate unconsciously, leaving greater resources available for processing new stimuli. Creative people also have relatively high IQs (see p.166), plus the ability to snap back to alertness when a new idea is hatched and to subject that idea to rigorous scrutiny and criticism. Ideas that survive this second creative thought process are likely to be valuable and therefore judged as genuinely new.

STARRY NIGHT
The artist Van Gogh worked on the painting *Starry Night* while in an asylum. He may have had temporal lobe epilepsy and/or bipolar disorder, both of which are associated with high levels of creativity.

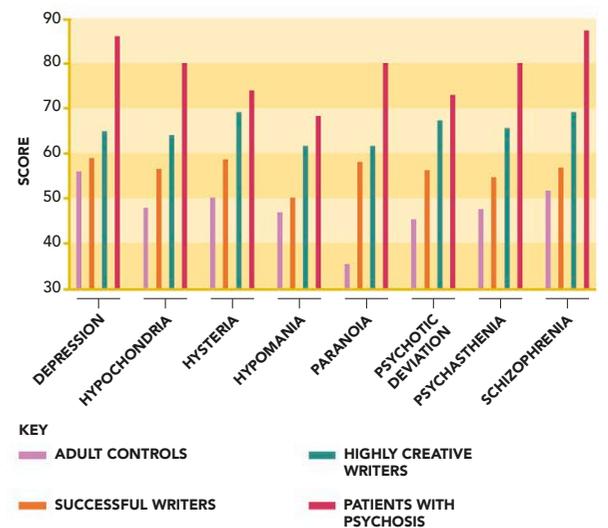


CREATIVITY AND MADNESS

Creativity and some types of insanity share certain features, such as intense imagination, a tendency to link things that may seem unconnected to others, and openness to ideas that others may swiftly discount. The difference between highly creative people and those who tip into madness is that creative people maintain insight. They recognize that their imaginings are not real and remain able to control any bizarre symptoms and channel them into their work.

MENTAL-DISORDER TESTING

Very creative people score highly on tests for mental disorders but rarely fulfill the diagnostic criteria for these conditions, so their mental states can be seen as being somewhere between normal and insane.



MUSICIANS
Brain-imaging studies of musicians at work show that frontal areas keep attention targeted when they play by rote, but turn off in improvisation so ideas can “float.”



HUMOR

A lot of humor arises from the juxtaposition of apparently unconnected ideas, which is similar to the process underlying creativity. Studies looking at how humorous interplay between coworkers affects workplace innovation suggest that keeping workers laughing may “jump-start” their creative faculties, perhaps because humor forces people to attend to “distractions,” making them more open to new information. Brain-imaging studies have shown that humor stimulates the brain’s “reward” circuit and elevates circulating levels of dopamine, which is linked to motivation and pleasurable anticipation.



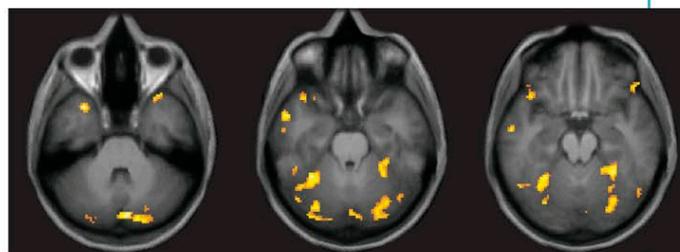
EXPECTATION OF INTENTION



INCONGRUITY

BRAIN AREAS LINKED TO HUMOR

The first frame sparks activity in brain areas linked to predicting intention—here, the cartoon character’s. The next frame activates areas linked to surprise and emotion, suggesting that such incongruity is central to humor.



EXPECTATION OF INTENTION



APPRECIATION

BRAIN IMAGING DURING CARTOON READING

The top row of fMRI scans show brain areas activated by the first frame of the cartoon above, including the temporal and parietal areas and the cerebellum. These become active when, by observing a person’s actions, we “know” what their intentions are. When the expectation is subverted, as in the second frame, it creates activity in the left amygdala (bottom row, circled). The amygdala is active in emotion, and the left side is particularly linked to pleasant feelings.

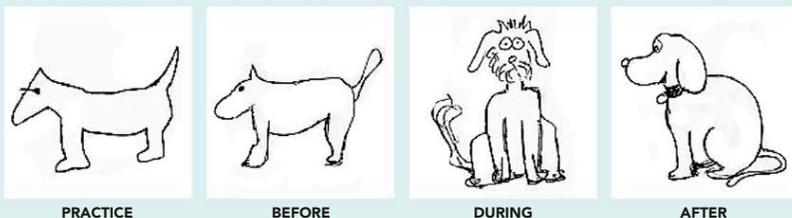
TURNING ON CREATIVITY

As soon as we can categorize a stimulus we tend not to scrutinize it further, but immediately edit it out. So, when we see a dog, we mentally label it as “dog” and do not stop to take in every detail. The frontal lobes manage this editing process,

and there is some evidence to suggest that if activity in this area is inhibited, people “take in” more. Tests using transcranial magnetic stimulation (TMS) to “turn off” the frontal lobes show that creative skills can emerge as frontal-lobe activity decreases.

TMS TEST

Volunteers subjected to TMS displayed new creative drawing skills when frontal-lobe activity was turned off.



BELIEF AND SUPERSTITION

OUR BRAINS ARE CONSTANTLY TRYING TO MAKE SENSE OF THE WORLD IN ORDER TO GUIDE OUR ACTIONS. ONE WAY OF DOING THIS IS BY CREATING EXPLANATORY STORIES OR IDEAS INTO WHICH WE FIT OUR EXPERIENCES. SUCH FRAMEWORKS ARE OFTEN USEFUL BUT MAY NOT ALWAYS BE CORRECT.

BELIEVING IS SEEING

Most people have some kind of belief system, which forms a framework for their experience. Some were taught their beliefs, while others arrived at them by examining their experience and working out their own interpretation. Once a belief system has been formed, it acts both as an explanation for what has happened in the past and also a “working hypothesis” that is projected onto the world. For example, if a person believes that the world is governed by a benign supernatural being, they will “see” events such as coincidences or strokes of good fortune as evidence of this, while a person with a materialist belief system would interpret them merely as chance happenings. People who are quick to see meaningful connections between, for example, random events are more inclined than others to have a magical or superstitious belief system.



HOLY TOAST

People with a tendency toward magical thinking are quicker to see patterns like the “face” in this piece of toast. They are also more likely to see such things as “meaningful”—perhaps even as signs from God.

PATTERN-MAKING

The ability to “see” patterns helps us make sense of the world and respond appropriately. But we can be both too good and too poor at it.

AUTISM

Autistic people do not see patterns that are obvious to most of us so get swamped by information, all of which seems equally important.

LITERAL-MINDEDNESS

Failure to recognize subtle patterns leads to concrete-mindedness, such as failure to understand metaphors (as seen in Asperger’s syndrome).

SUPERSTITION

Too much pattern-making may lead people to “see” things that are not there or make links between events that are not actually connected.



FLYING PIG

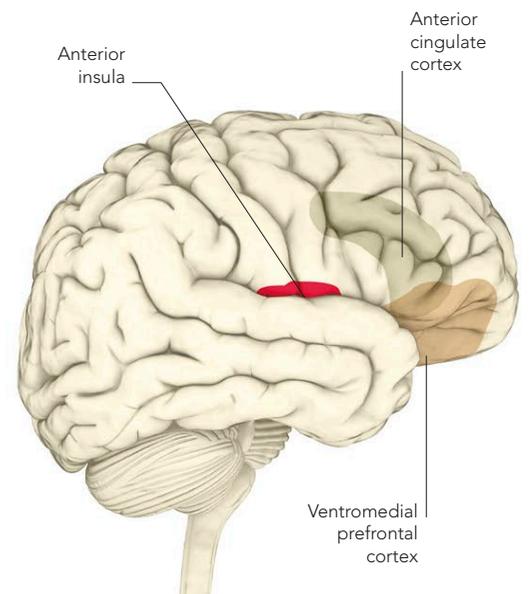
The human brain has evolved to pick up very quickly on visual stimuli that might signal danger or opportunity. Hence faces, human bodies, and animal forms are among the most likely things to be “seen” in clouds.

RELIGION IN THE BRAIN

Religious practice is largely determined by cultural factors. However, studies of identical twins who have been brought up separately suggest that the likelihood of a person experiencing a religious conversion or spiritual transcendence may be due more to genes than to upbringing. Spiritual transcendence shares some features with other “weird” experiences, such as out-of-body experiences, auras, and “the sensed presence” (see opposite page). These are associated with flurries of unusually high activity in the temporal lobes. The areas involved in intense religious experiences seem to be more widespread, however. For example, a study of nuns from a meditative order showed that, as they recalled an intense religious experience, many different areas were activated. So there does not seem to be a single “God-spot.”

SALEM WITCH TRIALS

Rigid belief systems can lead people to “see” things that do not exist. During the Salem witch trials of 1692, for example, religious bigots “saw” evidence of the devil in the behavior of entirely ordinary people.



THE BASIS OF BELIEF

Belief and disbelief are driven by parts of the brain to do with emotions, not reasoning. Belief activates the ventromedial prefrontal cortex, which processes reward, emotion, and taste, while disbelief is registered by the insula, which generates feelings of disgust.

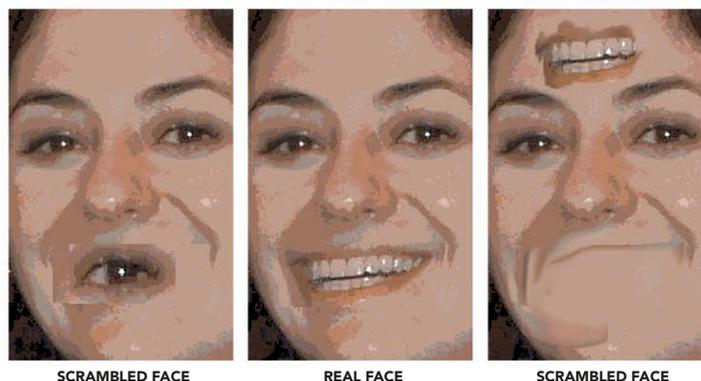


BRAIN CHEMISTRY

High natural levels of the neurotransmitter dopamine may explain why some people are unusually quick to pick out patterns. Believers are known to be more likely than skeptics to see a word or face in nonsense images, and skeptics more likely to miss real faces or words that are partly hidden by visual “noise.” One study found that skeptics’ tendency to see hidden patterns increased when they were given L-dopa, a drug that increases dopamine levels.

SCRAMBLED FACES STUDY

Believers are more likely than skeptics to see “real” faces when presented with a rapid sequence of “scrambled” faces. Skeptics, by contrast, are more likely to fail to spot “real” faces mixed in with the scrambled ones.



SEEING LITTLE PEOPLE

The content of supernatural “sightings” varies according to culture. Fairies were once commonly seen, while today it is more usual for people to report seeing alien beings. Claims of being abducted by aliens seem to be more common at times when the magnetic effects of solar radiation are high. One theory is that the radiation causes tiny temporal-lobe seizures in susceptible people, creating hallucinations.

THE COTTINGLEY FAIRIES

This faked photograph (part of a series) was made by two mischievous children in 1917. Many adults believed that the fairies were real.



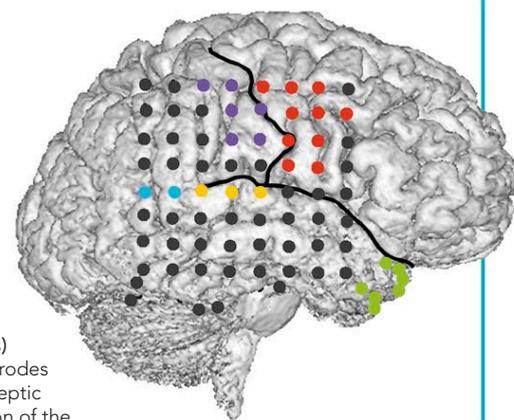
THE HAUNTED BRAIN

Apparently “supernatural” experiences may be due to disturbances in various parts of the brain. Tiny seizures in the temporal lobes are thought to be responsible for many of the emotional effects reported in such events, such as feelings of ecstasy or intense fear. Temporal-lobe disturbance is also associated with the sense of an invisible presence that often accompanies perceiving ghosts. Distortions of space and embodiment, such as the illusion of looking down at oneself, known as an “out-of-body” experience, are linked to a change in activity in the parietal lobes, which normally maintain a relatively stable sense of space and time. Hallucinations may

result from faulty visual or auditory processing or failure to interpret sights and sounds normally.

KEY

- TEMPOROPARIETAL JUNCTION (TPJ)
- MOTOR CORTEX
- SOMATOSENSORY CORTEX
- AUDITORY CORTEX
- FOCUS OF EPILEPTIC ACTIVITY IN TEMPORAL LOBE



OUT-OF-BODY EXPERIENCES (OBEs)

This diagram shows areas where electrodes were implanted in the brain of an epileptic person to evoke responses. Stimulation of the TPJ (blue dots) was found to induce OBEs.



WHITE LADY

Expectation has a strong effect on what a person sees. Many “hauntings” arise because people have been led to expect to see a ghost in a certain place. Any unusual sensory effect is then interpreted as a specter.

SO YOU THINK YOU’RE CLAIRVOYANT?

Our brains are continually making predictions about the near future, using knowledge of past and present to guess what will happen next. Sometimes things happen that the brain can’t predict because they are random. Usually, we are alerted to such events by snapping to attention, but if the change is very fast, we may become aware of it unconsciously, before we know consciously that it has happened, giving the impression that we have perceived the event in the future. This “out-of-sync” brain glitch occurs more often in people who hold superstitious beliefs.



FORESIGHT

Sometimes it feels as though we foresaw an event, because our emotional reaction to it occurs before we consciously see it happen.

ILLUSIONS

ILLUSIONS OCCUR WHEN SENSORY DATA CLASHES WITH OUR ASSUMPTIONS ABOUT THE WAY THINGS ARE. THE BRAIN ATTEMPTS TO MAKE THE INFORMATION "FIT." THE RESULTING CONFUSION GIVES US A GLIMPSE OF HOW THE BRAIN WORKS.

TYPES OF ILLUSION

The brain has certain rules that it applies to incoming information in order to make sense of it quickly. If we hear a voice and at the same time see a mouth moving, for example, we assume the voice comes from the mouth. Like all such rules, though, this is only a best guess and can be wrong. Hence it leaves us open to the illusion of ventriloquy. Low-level illusions—those created in the early stages of perception—are unavoidable, but those that arise due to higher-level cognition are less robust. It is impossible not to see the after-image that occurs when you have been looking at a bright light, for example, because this is created by low-level nerve activity, which



cannot be affected by conscious thought. However, once you know the voice comes from the ventriloquist rather than the dummy, a result of higher-level cognition, the illusion is less convincing. Illusions may be generated by both conscious and unconscious assumptions. A child's concept of how a horse looks, for example, includes four distinct legs (top left), which governs how the horse is visualized. An "expert" viewer of horses—such as the artist Leonardo Da Vinci (top right)—has a more realistic concept.

ARTIST'S EYE
The middle drawing is by a five-year-old autistic savant, who probably had no concept of a horse at all. Unlike the normal child, her concepts do not mislead her.

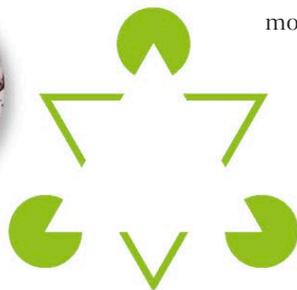
CANALS ON MARS

Until the early 20th century, some astronomers believed that Mars was crossed by canals. Maps were made, and for nearly a decade, the canals seemed to be visible to people with fairly strong telescopes. The canals did not "vanish" until analysis of the Martian atmosphere proved that life there was not possible. Acceptance that the canals could not exist stopped people from seeing them.



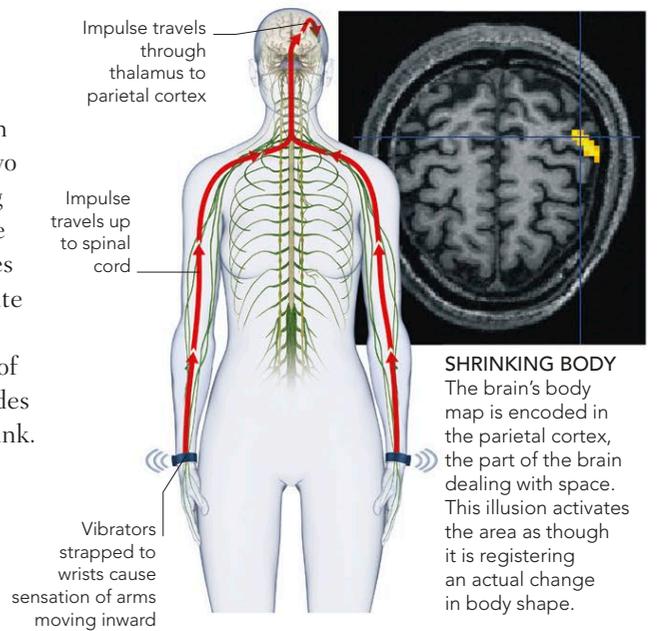
DISTORTING MIRRORS

Information from the outside world, including sensations from the rest of the body, is constantly compared to a "virtual" world within the brain, which includes a conceptual map of the body. When the two fail to match up, the brain assumes that something outside has changed. It can even be fooled that the body has shrunk. The shrinking-body illusion involves stimulating the arm muscles with vibrators, to create the feeling that the limbs are moving in, beyond the sides of the body. The brain decides that the body has shrunk.



IMPOSED TRIANGLE

The brain imposes things that are not there, like this white triangle, when it is the most likely explanation for what we see.



SHRINKING BODY
The brain's body map is encoded in the parietal cortex, the part of the brain dealing with space. This illusion activates the area as though it is registering an actual change in body shape.

AMBIGUOUS ILLUSIONS

Something strange happens when we look at ambiguous figures. The input to the brain stays the same, but what we see flips from one thing to another. This demonstrates that perception is an active process, driven by information that is already in our brains as well as information from the outside world. The switching occurs because the brain is searching for the most meaningful interpretation of the image. Normally, the brain settles quickly on a solution by using basic rules such as, "if one thing surrounds another, the surrounded shape is the object and the other thing is the ground." Ambiguous figures confound such rules. For instance, in the vase illusion (left), it is impossible to see which shape is on top, so the brain tries one way of seeing it then another. You see both images, but you can never see both of them simultaneously.



SHAPE SHIFTERS

In the vase-face illusion (top), the figures switch between two facing profiles and the outline of a vase. The bottom figure can be seen as either a rabbit or a duck.

MY WIFE AND MY MOTHER-IN-LAW

In this illusion, the figure of either a young woman or an old hag may dominate at first, but once you have "seen" the alternative, the brain finds it again easily.



DISTORTING ILLUSIONS

Distorting illusions are characterized by visual images that generate a false impression of an object's size, length, or curvature. They generally exploit the "allowances" the brain normally makes in order to make sense of what it sees. For example, the brain "allows" that objects of the same size will look smaller if they are farther away, and that larger objects in an array should command greater attention than small ones. Like other illusions, distortions may occur at low or high levels of perception (see opposite page). Those that happen in the earliest stages, before the brain "recognizes" what it is looking at, are the most robust because they cannot be influenced by conscious thought.

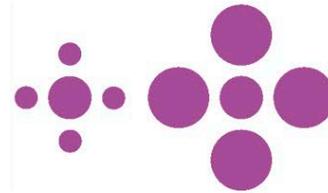


TOWER ILLUSION

These images of the Rockefeller Plaza in New York are identical, but the one on the right seems to lean to the right. This is because the brain treats them as a single scene. Usually, if two adjacent towers rise in parallel, their outlines converge due to perspective. When seeing two towers with parallel outlines, the brain assumes the towers are diverging.

PERSPECTIVE ILLUSION

Even though the figures walking along the road are the same height, the brain insists that the one farthest away looks taller. This is because the rule of perspective—things shrink with distance—is applied at an early stage of perception.



EBBINGHAUS ILLUSION

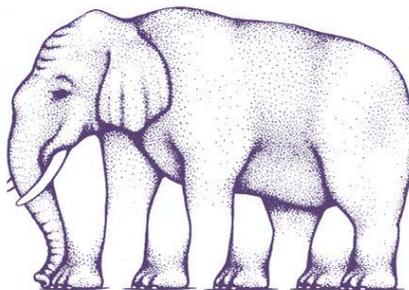
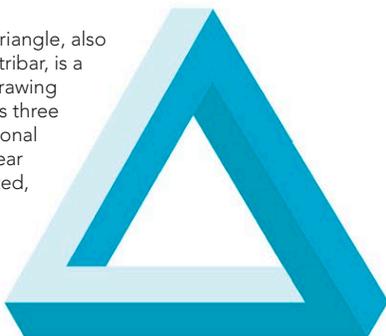
The central circle is the same size in both images, but we see it as bigger when compared to smaller circles rather than larger ones.

PARADOX ILLUSIONS

It is possible to represent objects in two dimensions that cannot actually exist in the real, three-dimensional world. Paradox illusions are generated by such images, which are often dependent on the brain's erroneous assumption that adjacent edges must join. Although impossible, the best examples are oddly convincing, and the conscious brain is teased and intrigued by them. As with ambiguous illusions, the brain tries first one interpretation and then another but is unable to settle because none of the available views make sense. Brain-imaging scans show that impossible images are recognized by the brain very early in the process of perception, well before conscious recognition. Unlike the conscious brain, the unconscious part is not very concerned with such images and spends less time trying to process them than it spends on "real" objects.

THE TRIBAR

The Penrose triangle, also known as the tribar, is a perspective drawing that comprises three three-dimensional bars that appear to be connected, but in reality could not be.



THE IMPOSSIBLE ELEPHANT

Although it is impossible to determine how many legs this elephant has, the brain keeps trying to match up the shaded areas of "legs" with the apparently detached feet.

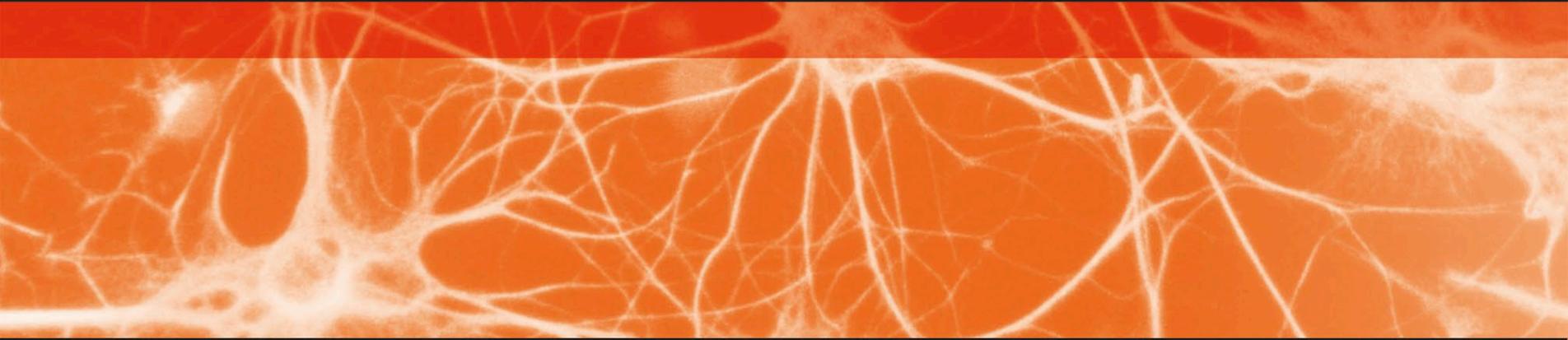
M.C. ESCHER

"Mauk" Escher, a Dutch graphic artist, started drawing elaborate impossible realities in the 1930s and produced a huge quantity of now famous illusions. He created the images from imagination rather than by reference to observation and incorporated many sophisticated mathematical concepts into his artworks. His images are both tantalizing and emotionally charged—some of his landscapes are witty, while others have a dark, surreal quality. Several of his works show buildings that could never actually be constructed.

RELATIVITY

The scene shown here is impossible in that it could exist only in a world in which gravity worked in three directions rather than one.





HOW DOES THE ELECTRICAL FIRING OF CELLS IN OUR BRAIN PRODUCE OUR CONSCIOUS EXPERIENCE OF THE WORLD, AND WITH IT SUCH THINGS AS OUR SENSE OF A PRIVATE SELF AND OUR ABILITY FOR ABSTRACT THOUGHT AND REFLECTION? THIS IS A FAMOUSLY DIFFICULT QUESTION. ANSWERING IT INVOLVES BUILDING A BRIDGE BETWEEN THE PHYSICAL AND MENTAL WORLDS. AS NEUROSCIENCE ADVANCES, WE ARE GETTING CLOSER TO UNDERSTANDING WHAT CONSCIOUSNESS IS AND HOW IT COMES ABOUT. FOR EXAMPLE, DIFFERENT CONSCIOUS STATES CAN NOW BE CORRELATED WITH ACTIVITY IN SPECIFIC BRAIN AREAS.

CONSCIOUSNESS



WHAT IS CONSCIOUSNESS?

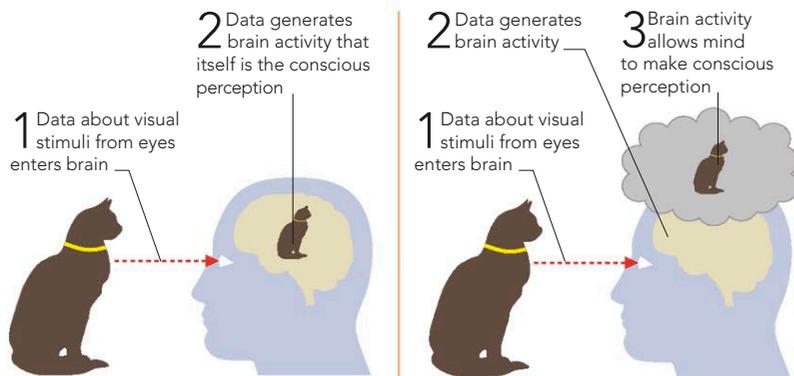
CONSCIOUSNESS IS ESSENTIAL—WITHOUT IT, LIFE WOULD HAVE NO MEANING. WE CAN IDENTIFY THE SORT OF BRAIN ACTIVITY THAT GENERATES CONSCIOUS AWARENESS, BUT HOW THIS APPARENTLY INTANGIBLE PHENOMENON ARISES FROM A PHYSICAL ORGAN REMAINS A MYSTERY.

SPANDRELS
This is the name given to the spaces between arches. Although we talk of them as objects, without the arch, they cease to exist. Consciousness may have appeared in the same way, as a result of other evolved features.



THE NATURE OF CONSCIOUSNESS

Consciousness is like nothing else. A thought, feeling, or idea seems to be a different kind of thing from the physical objects that make up the rest of the universe. The contents of our minds cannot be located in space or time. Although they appear to be produced by particular types of physical activity in the brain, it is not known if this activity itself forms consciousness (the Monist/materialist view) or if brain activity correlates with a different thing altogether that we call “the mind” or consciousness (the dualist view). If consciousness is not simply brain activity, this suggests that the material universe is just one aspect of reality and that consciousness is part of a parallel reality in which entirely different rules apply.

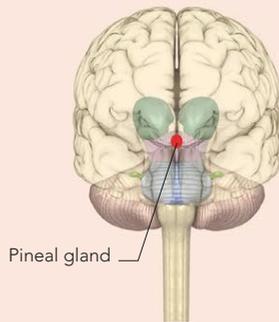


MONISM
According to this theory, consciousness is part of the material universe. It is identical to the brain activity that correlates with it. It developed when cognitive mechanisms evolved, but only as a result of them, rather than for any purpose of its own.

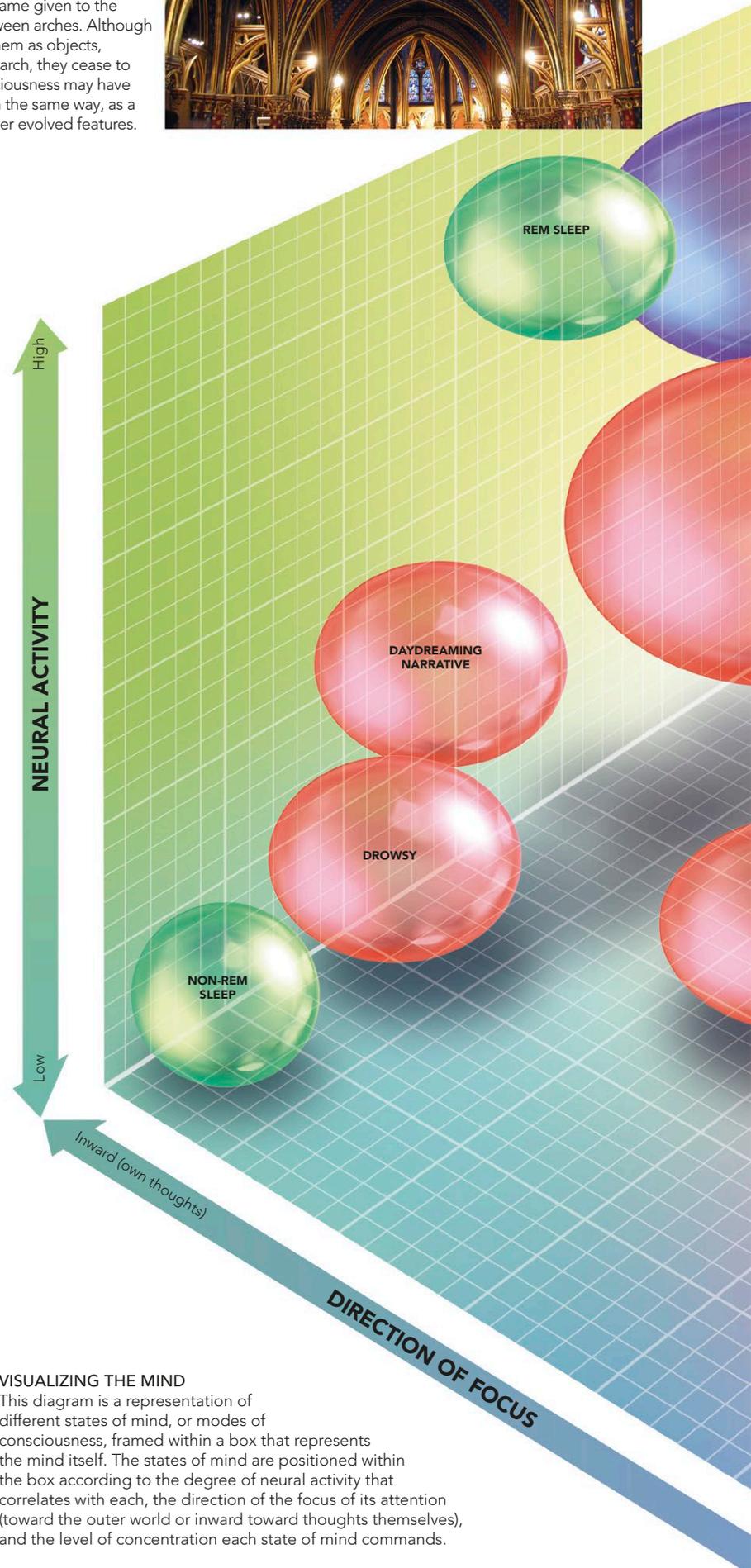
DUALISM
Consciousness is not physical but exists in another dimension to the material universe. Certain brain processes are associated with it but are not identical to it. Some dualists believe consciousness may exist without the brain processes associated with it.

DESCARTES AND THE MIND-BODY PROBLEM

The French philosopher René Descartes (1596–1650) is generally held to have founded modern dualism when he proposed that matter is separate and distinct from the mind (things like emotions, thoughts, and perceptions). This presented a problem: how can the two “kinds” of things interact? Descartes’ solution was that “mind-stuff” affected the body via the pineal gland—a small nucleus in the center of the brain. His solution to what has become known as the mind-body problem is now generally discounted, especially as the function of the pineal gland—hormone modulation—becomes clearer.



THE THIRD EYE
The pineal gland produces melatonin, a hormone that modulates sleep cycles. It is sometimes called the third eye—a reference to the mystical role attributed to it.



VISUALIZING THE MIND
This diagram is a representation of different states of mind, or modes of consciousness, framed within a box that represents the mind itself. The states of mind are positioned within the box according to the degree of neural activity that correlates with each, the direction of the focus of its attention (toward the outer world or inward toward thoughts themselves), and the level of concentration each state of mind commands.

TYPES AND LEVELS OF CONSCIOUSNESS

Consciousness has different modes, such as emotions, sensations, thoughts, and perceptions, which are all experienced at different levels of neural activity, focus, and concentration. The level of neural activity determines the intensity of consciousness. The direction of focus can be toward the outside world or the inner world (thinking about thoughts). Concentration can be loosely targeted, involving a range of objects or fixed, involving just one particular aspect. Consciousness also divides into three types of awareness: awareness in the moment—the brain registers and reacts to moment-by-moment events but does not encode them in memory; conscious awareness—events are registered and encoded in memory; and self-consciousness—events are registered and remembered, and the person is conscious of doing this.



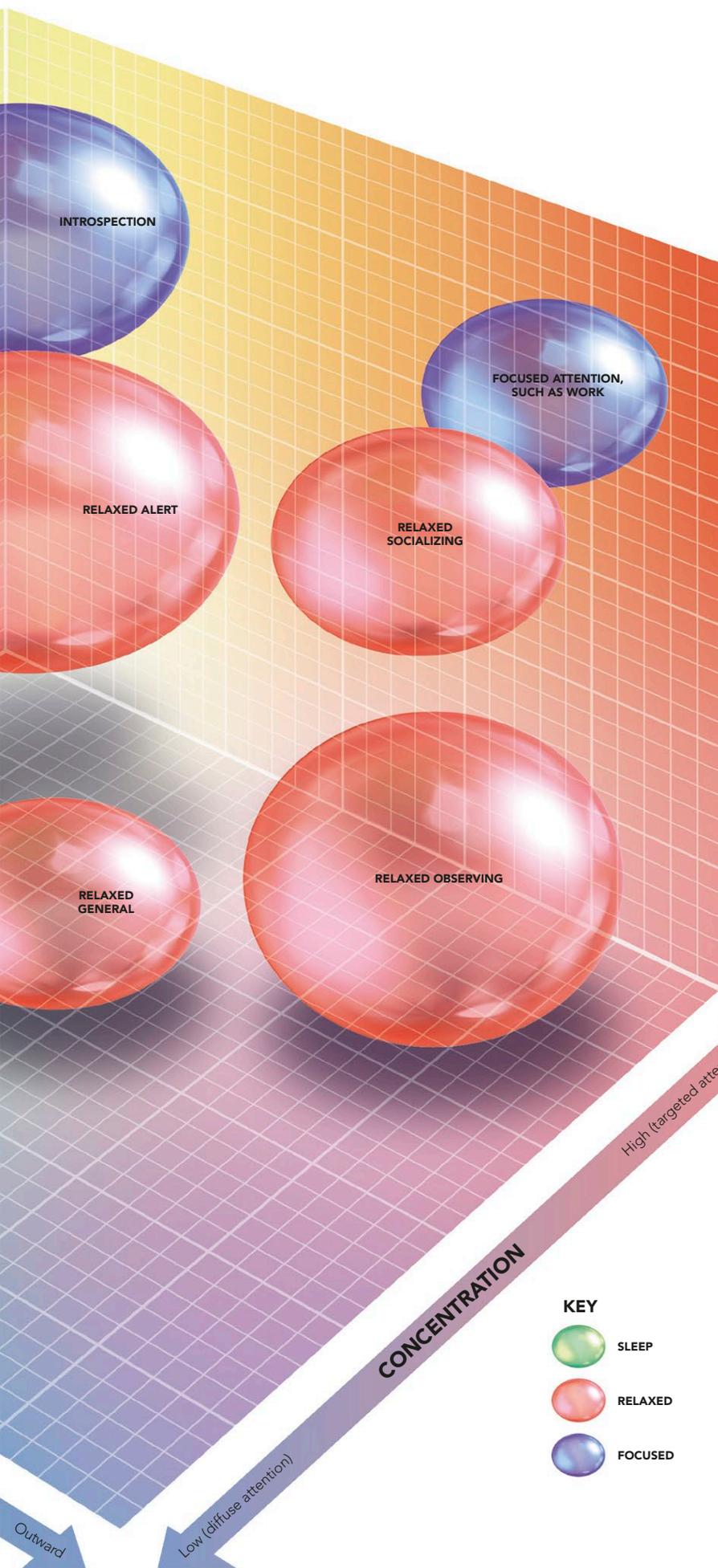
THE THINKER

Most conscious thinking is couched in language. Words function as symbolic “handles,” used to grasp the objects they represent. However, about 25 percent of thoughts are experienced as sensations or perceptions.



FIXED CONCENTRATION

When focusing on an object, attention narrows. Other potential focal points are neglected. This can be useful—this child notices less of a potentially traumatizing medical procedure when focused on a toy.



- KEY**
- SLEEP
 - RELAXED
 - FOCUSED

THE CHINESE ROOM

Is consciousness needed for “understanding”? Philosopher John Searle invented the idea of a room in which every dictionary and rule relating to the Chinese language was stored. Inside is a man who is able to translate and respond to questions written in Chinese by manipulating these resources, despite not being able to speak a word of Chinese. Hence, someone posting the words “How does your dog smell?” in Chinese may receive the reply, in Chinese, “Awful!” From outside, it looks as though the man inside must have “understood” the question, but Searle argues that merely behaving this way is not the same as understanding. In the same way, a computer could never be described as “having a mind” or “understanding.” Other philosophers argue that understanding—and perhaps every other type of consciousness—is merely the process of behaving as though one understands.

Labels in diagram: Book of Chinese symbols, Room, Non-Chinese speaker, Message in, Message out.

LOCATING CONSCIOUSNESS

HUMAN CONSCIOUSNESS ARISES FROM THE INTERACTION OF EVERY PART OF A PERSON WITH THEIR ENVIRONMENT. WE KNOW THAT THE BRAIN PLAYS THE MAJOR ROLE IN PRODUCING CONSCIOUS AWARENESS, BUT WE DO NOT KNOW HOW. CERTAIN PROCESSES WITHIN THE BRAIN, AND NEURONAL ACTIVITY IN PARTICULAR AREAS, CORRELATE RELIABLY WITH CONSCIOUS STATES, WHILE OTHERS DO NOT. THESE PROCESSES AND AREAS SEEM TO BE NECESSARY FOR CONSCIOUSNESS, ALTHOUGH THEY MAY NOT BE SUFFICIENT FOR IT.

SIGNIFICANT BRAIN ANATOMY

Different types of neuronal activity in the brain are associated with the emergence of conscious awareness. Neuronal activity in the cortex, and particularly in the frontal lobes, is associated with the arousal of conscious experience. It takes up to half a second for a stimulus to become conscious after it has first been registered in the brain. Initially, the neuronal activity triggered by the stimulus occurs in the “lower” areas of the brain, such as the amygdala and thalamus, and then in the “higher” brain, in the parts of the cortex that process sensations. The frontal cortex is activated usually only when an experience becomes conscious, suggesting that the involvement of this part of the brain may be an essential component of consciousness.



SELF AWARENESS
In order to be conscious, the brain needs to “own” its perceptions—that is, to recognize that those perceptions are occurring within itself. To do this, it has to generate a sense of self (as opposed to unconscious awareness). Without this, consciousness may not be possible.

CRUCIAL PARTS OF THE BRAIN

Various areas of the brain are involved in generating conscious experience, even though none of them alone is sufficient to sustain it. If any of these are severely damaged, consciousness is compromised, altered, or lost.

Supplementary motor cortex

Deliberate actions are “rehearsed” here, distinguishing them from unconscious reactions

Dorsolateral prefrontal cortex

Different ideas and perceptions are “bound” together here—a process thought to be necessary for conscious experience

Orbitofrontal cortex

Conscious emotion arises here; if inactive, reactions to stimuli are merely reflexive body actions with no emotion

Temporal lobe

Personal memories and language depend on these; without these faculties, consciousness is severely curtailed

Tempo-parietal junction

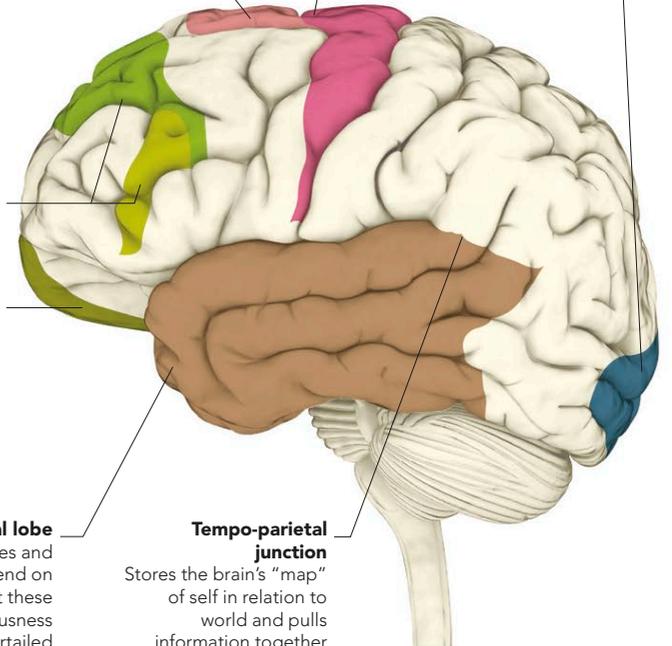
Stores the brain’s “map” of self in relation to world and pulls information together from many areas

Motor cortex

Body awareness (involving motor cortex) may be crucial to sense of self, which seems necessary for consciousness

Primary visual cortex

Without this, there is no conscious vision, even if other parts of visual cortex are functioning

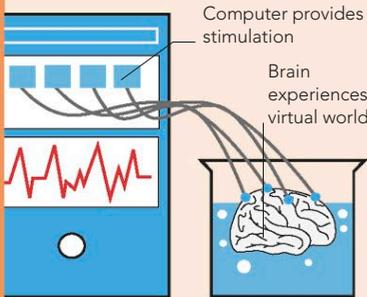


THE “BRAIN-IN-A-VAT”

The idea of a conscious but disembodied brain is central to many science fiction and horror films and is often used as a thought experiment in philosophical debates about the nature of reality. In recent years, the notion has ceased to be entirely theoretical as modern technology edges toward the possibility of inducing in the brain a virtual reality, indistinguishable from the reality experienced through the body. It is even possible that such a thing has been achieved already, and the external world, as we experience it, is not “real” at all.

VIRTUAL REALITY

The idea that we are simply disembodied brains hooked up to a supercomputer that simulates conscious experience is a famous thought experiment.



THE MATRIX

This 1999 film explores the idea of virtual reality being the only “reality” humans experience. People’s brains are “plugged” into the Matrix, a huge computer program simulating physical experience.

Thalamus

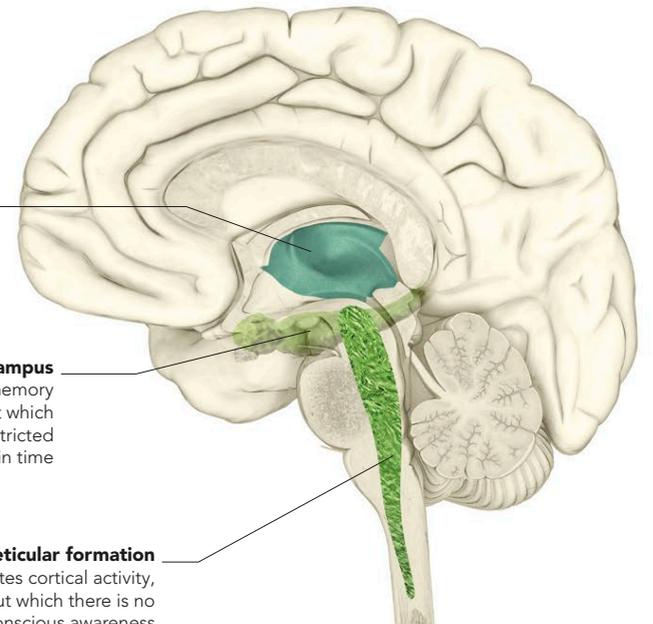
Directs attention and switches sensory input on and off

Hippocampus

Underlies memory encoding, without which consciousness is restricted to a single point in time

Reticular formation

Stimulates cortical activity, without which there is no conscious awareness

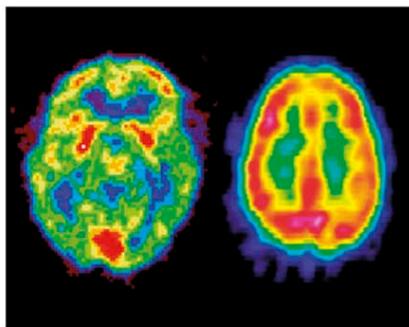
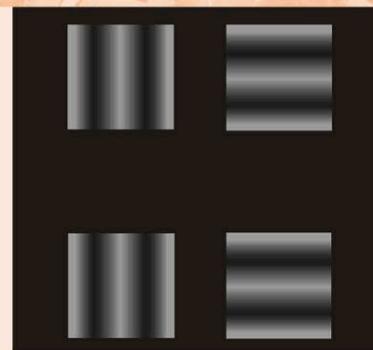


REQUIREMENTS OF CONSCIOUSNESS

Every state of conscious awareness has a specific pattern of brain activity associated with it. These are commonly referred to as the neural correlates of consciousness. For example, seeing a patch of yellow produces one pattern of brain activity; seeing grandmother, another. If the brain state changes from one pattern to another, so does the experience of consciousness. The processes relevant to consciousness are generally assumed to be found at the level of brain cells rather than at the level of individual molecules or atoms. It is likely that, for consciousness to arise, the factors listed below need to be present. Yet it is also possible that consciousness does arise at the far smaller atomic (quantum) level, and if so, it may be subject to very different laws.

VISUAL PHANTOMS

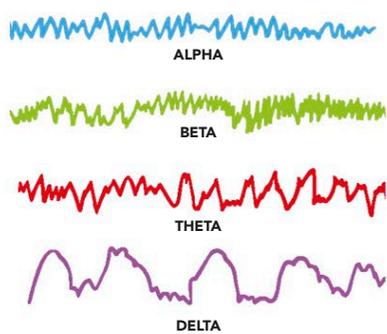
Conscious perception does not rely solely on external stimuli—it can also arise internally. Our brains constantly “fill in” missing data to make sense of the world. For example, you may see phantomlike vertical lines connecting the two blocks in the first column. This “imaginary” perception depends on similar neural-activity patterns as conscious perceptions of “real” stimuli.



NORMAL EPILEPTIC SEIZURE

LEVEL OF COMPLEXITY

Neural activity must be complex for consciousness to occur, but not too complex. If all the neurons are firing, such as in an epileptic seizure, consciousness is lost.



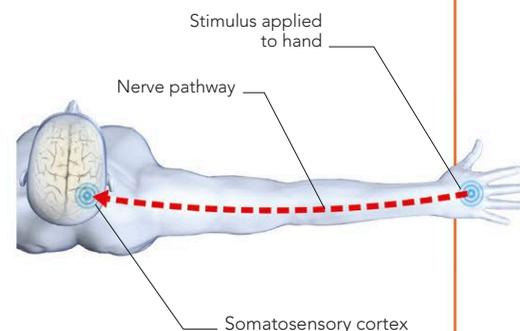
FIRING THRESHOLDS

Consciousness arises only when brain cells fire at fairly high rates. The high firing rate of Beta waves indicates alertness, while the low rate of Delta waves indicates deep sleep.



SYNCHRONOUS FIRING

Clusters of cells across the brain fire in unison. This seems to “bind” independent perceptions (say, the left and right visual fields) into one conscious perception.



TIMING

It takes half a second for the unconscious brain to process stimuli into conscious perceptions, but the brain fools us into thinking we experience things immediately.

MEASURING NEURAL ACTIVITY

Every conscious state correlates with a pattern of neural activity. These patterns of firing cells can be gauged by measuring the level of electrical activity in the brain through the skull, using a cap fitted with electrodes.



ATTENTION AND CONSCIOUSNESS

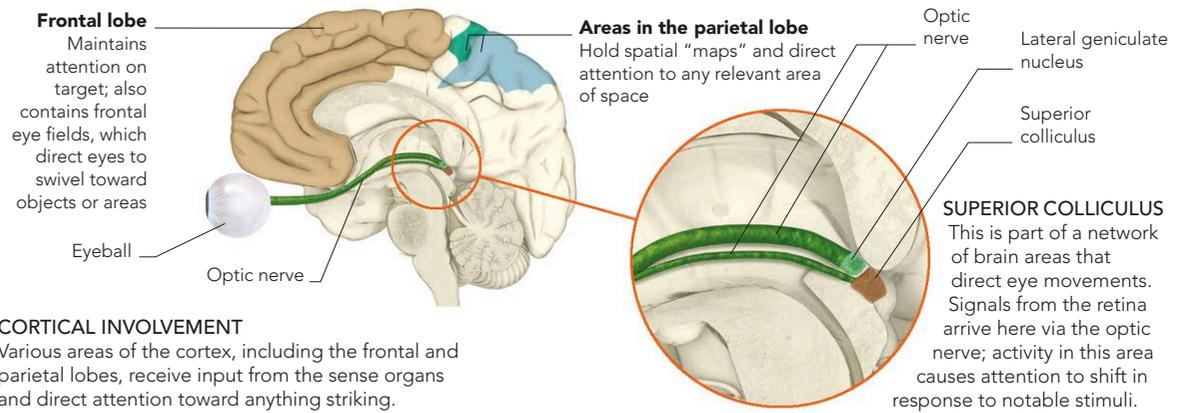
ATTENTION CONTROLS AND DIRECTS CONSCIOUSNESS. IT ACTS LIKE A HIGHLIGHTER THAT MAKES CERTAIN PARTS OF THE WORLD "JUMP OUT" AND CAUSES THE REST TO RECEDE. IT SELECTS THE FEATURE THAT IS CURRENTLY MOST IMPORTANT IN THE ENVIRONMENT AND AMPLIFIES THE BRAIN'S RESPONSE TO IT.

WHAT IS ATTENTION?

Attention causes you to select one item from the sensory inputs you are receiving and allows you to become more fully or sharply conscious of it. Consciousness and attention are so closely linked that it is almost impossible to attend to something and not be conscious of it. Overt attention involves consciously directing the eyes, ears, or other sense organs toward a stimulus and processing information from it. Covert attention involves switching attention to a stimulus without directing the sense organs toward it.

Attention may seem continuous, but maintaining focused attention is actually rare and difficult. It is also hard to switch attention from one object to another: the more attentive you are to one stimulus, the slower you are to turn your attention away from it. Hence an event that captures your attention will "blot out" anything else for a fraction of a second.

ATTENTION TYPES	
TYPE	DESCRIPTION
Focused attention	 This is the ability to single out one object in one's environment and respond to it. An example might be an athlete focusing on the starter's gun, while "tuning out" the noise from the crowd.
Sustained attention	 Attention naturally tends to wander. Sustained attention is the ability to maintain concentration on a particular object or activity, such as operating heavy machinery for a continuous period of time.
Selective attention	 This form is similar to sustained attention but involves the ability to resist shifting attention from the selected target, for example, when focusing on a putt despite other competing stimuli.
Alternating attention	 This involves shifting quickly from one stimulus to another, which requires a different sort of cognitive response—for example, when shifting attention from a model you are painting to the actual painting.
Divided attention	 Often known as "multitasking," this involves dividing attention between two or more competing tasks. Recent research suggests that apparently divided attention is actually very quick alternating attention.



INTENSE CONCENTRATION

When you concentrate hard, you filter out other possible objects of attention so that maximum cognitive resources are available for the task at hand.

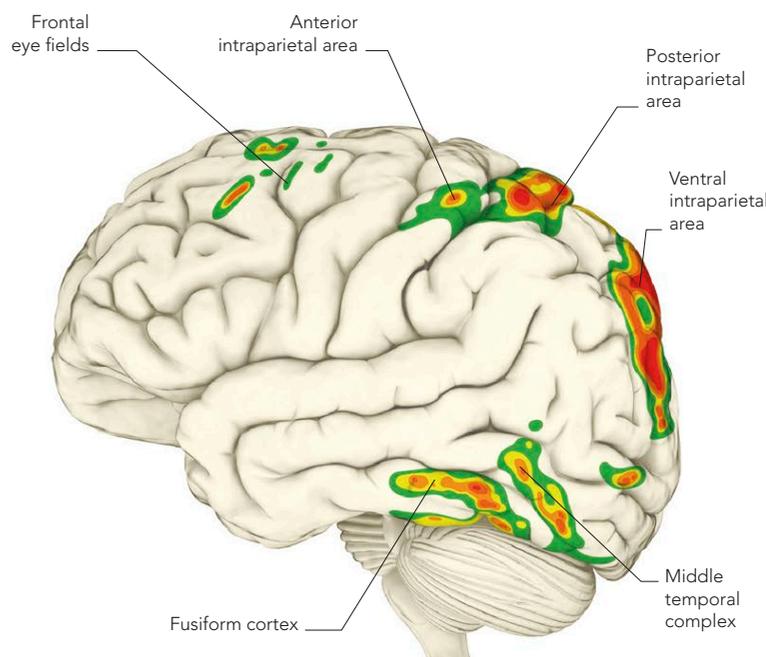
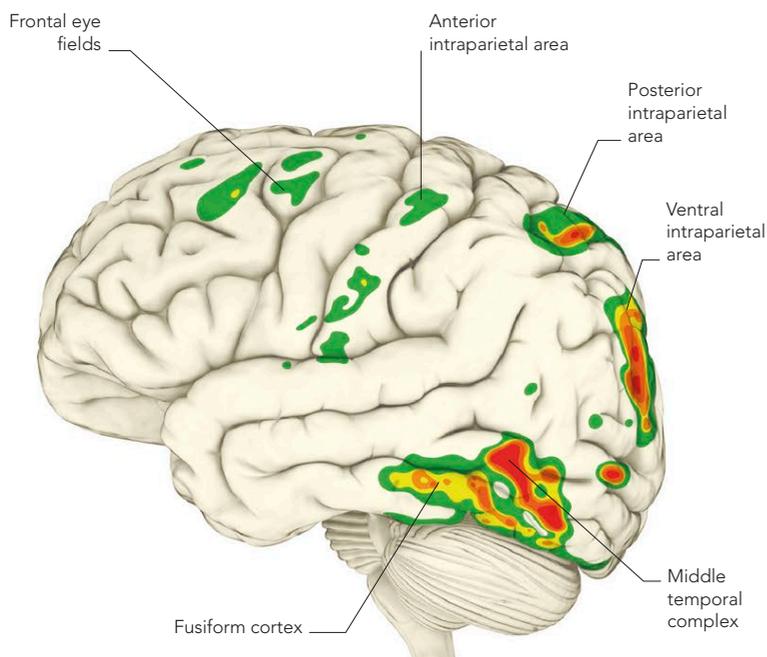
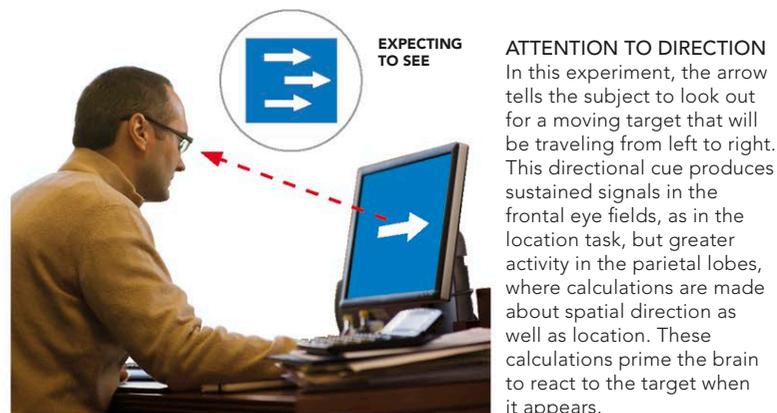
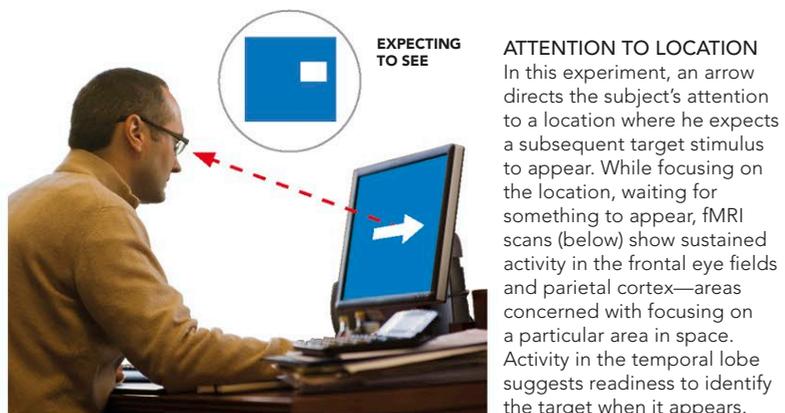
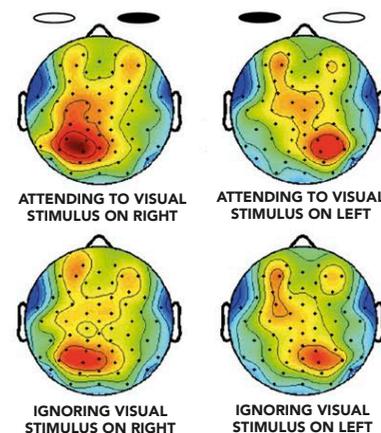


NEURAL MECHANISMS

If the brain registers an unexpected movement, a loud sound, or some other potentially significant stimulus, it directs the sense organs toward it—for example, by swiveling the eyes in the direction of a sudden movement. This happens automatically, in the lower regions of the brain, and it does not in itself create consciousness of the stimulus. However, attention also increases activity in the neurons that are concerned with the stimulus. If the stimulus is a person, for example, neural activity increases in the visual areas that monitor the place in space where the person is located; the face-recognition area; the amygdala; the temporal-parietal areas, which work out their intentions; and the supplementary motor area, which works out what to do about them. If the neurons are excited beyond a certain point, consciousness “kicks in.”

NEURONAL ACTIVITY

When you attend to a thought, emotion, or perception, the brain activity is amplified and becomes more synchronous. This EEG study shows activity while attending to a visual stimulus and ignoring it. Attending to stimuli on the left activates the right hemisphere and vice versa.



ABILITY TO FOCUS

The best-known attention disorder is ADHD (see p.246), but there are many others, affecting both adults and children. Any variation from a normal ability to focus or shift attention might be considered a disorder if it disrupts a person's ability to function in their environment. Someone who gets absorbed in things that interest them and fails to notice other people talking to them might do very well in a job that calls for intense focus (such as scrutinizing medical images for abnormalities) but could be considered strange, or even ill, in a highly sociable environment. One type of attention failure—so-called “inattention blindness”—occurs when a person is so intent on focusing on one aspect of a scenario that they completely miss some other, major component. It is so common that it is considered normal.

CARD TRICK

If the ace = 14, what number do the cards add up to? Focusing on this mathematical task might cause you to miss an unusual detail.

Never mind the total—did you spot that the 4 of hearts is black?

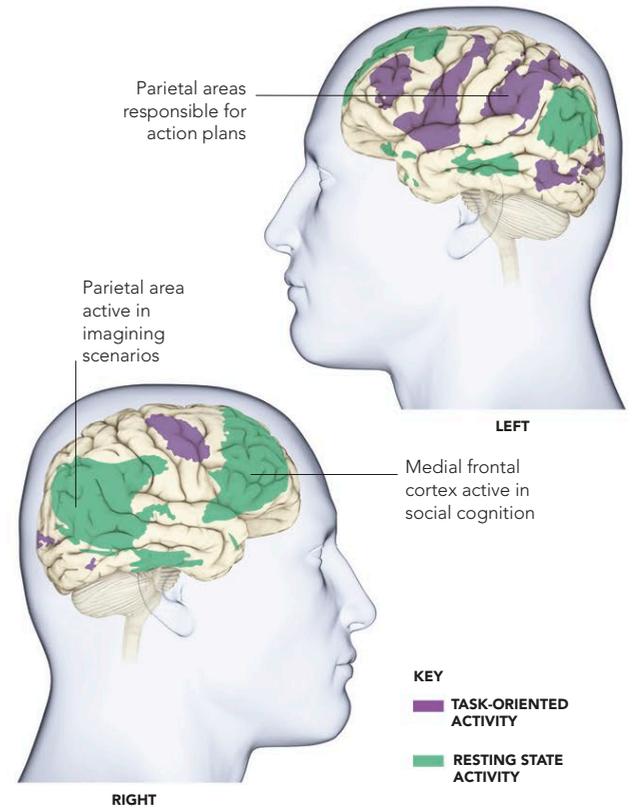


THE IDLING BRAIN

THE BRAIN HAS DISTINCT MODES, EACH OF WHICH USES A DIFFERENT NETWORK OF NEURONS. "RESTING-STATE NETWORKS" BECOME ACTIVATED WHEN WE CEASE TO BE FULLY ENGAGED WITH THE OUTSIDE WORLD.

THE RESTING STATE NETWORK

When the brain is not actively engaged in a task it falls into one of a number of resting states. The most common of these involves the default-mode network (DMN). When the brain is in task mode it responds to sensation by creating action plans, which are then turned into actual actions. In contrast, while in a resting state, the brain creates action plans but does not act them out: they are imagined scenarios. The medial frontal cortex is active in the resting state, indicating social rumination, while the lateral frontal cortex is active in task mode, indicating sequential thought patterns suited to handling objects.



READING THE DEFAULT-MODE NETWORK

Although default mode network activity is recognizably similar in everyone, there are small individual differences, which seem to tally with differences in personality. Researchers at several centers are charting individuals' DMN activity by EEG and correlating the information with those people's personalities. Drawing on this information, it may be possible to produce a brain-activity-related personality test.

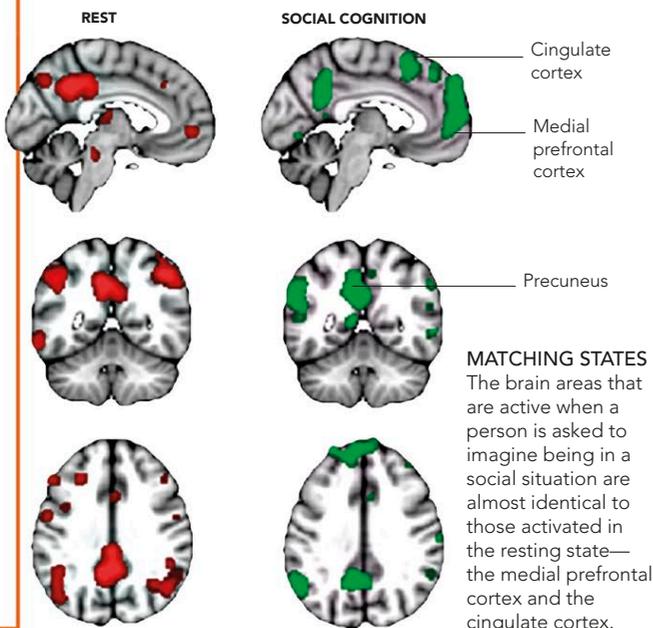


TASK AND RESTING STATES

These scans show the brain in two different states—at rest and while performing a task. The green areas show the areas of high activity in the resting state. When a person is actively engaged in a task, the purple areas become active and the green ones calm down.

THE DMN AND SOCIAL AWARENESS

The areas of brain activated in the default-mode network are very similar to those activated when a person is asked to interpret a social situation and, in particular, his or her own situation with regard to other people. This suggests that whenever we are free of immediate mental tasks, we fall back into a state of rumination about our relationships with others, and our place in the social world.

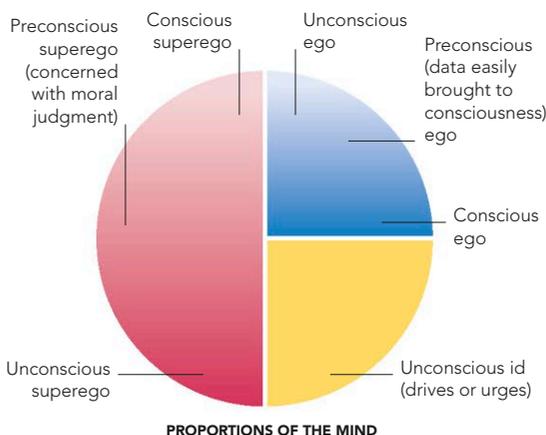


THE BRAIN'S EGO

The thoughts associated with the default-mode network are mainly self-centered and driven by one's own autobiography and place in the social hierarchy. They often draw on half-forgotten memories and are colored by emotion. These are the concerns that Sigmund Freud identified with the half-submerged mind-state that he called the ego. Some researchers have suggested the DMN is functionally the same as the Freudian ego.

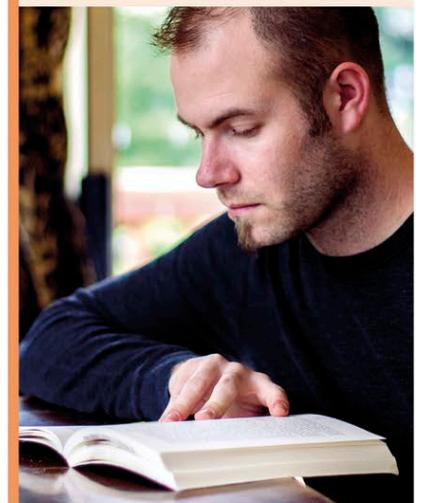
FREUD'S THEORY OF MIND

Freud thought that most brain processes were unconscious. The ego, part of the mind involved in self, was partly conscious. The rest of the conscious mind controlled thoughts and actions, and roughly matches the "task-oriented" mind state.



THE WANDERING MIND

People seem to spend about one-third of their waking hours in a resting state mode, and if they are doing something untaxing, such as driving down a straight, empty road, the proportion is even higher. In one experiment, people were invited to sit in a laboratory and do nothing except read a novel and report whenever their mind wandered. Over the course of half an hour, they typically reported one to three episodes of mind-wandering.



RESTING STATE AND CREATIVITY

Most people switch very cleanly from default mode to task mode if they are suddenly called upon to engage with the outside world. In some people, though, the two modes run concurrently. This may make them more creative, because the free-floating, discursive nature of thinking in the default mode may drift toward a solution to a problem that would escape the more targeted, constrained thinking associated with task-oriented cognition. However, overlap between the two modes is also associated with schizophrenia and depression.

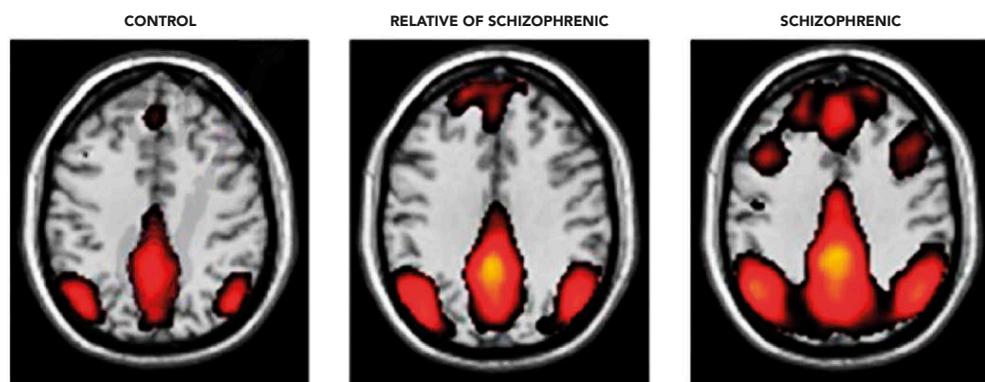
This may account for some of the unconventional thinking associated with schizophrenia and the lack of concentration displayed by many depressed people.

FIRING THE DMN

The connections between areas involved in the DMN are tighter in schizophrenics and their relatives. This means that if one of the areas is triggered, it is more likely to fire up the whole network.

DMN IN ANIMALS

The default-mode network has been observed in animals other than humans. In fact, researchers have found it in all animal species tested so far, including dogs and rats. The regions of the brain most active in the DMN appear to be more highly developed the more social the animal species is—with humans forming the largest social groups of any animal and having correspondingly large “social brain” areas. One theory is that the DMN may allow all social animals to keep themselves secure and “up to speed” in their societies.



ON AUTOPILOT

The brain flips into resting state when we are not actively engaged in a task. This includes carrying out actions that are second nature—the moment-by-moment activity is carried out on autopilot while the conscious brain ruminates.



ALTERING CONSCIOUSNESS

THE BRAIN IS CAPABLE OF GENERATING A WIDE RANGE OF CONSCIOUS EXPERIENCES, INCLUDING SOME STATES THAT ALTER OUR PERCEPTIONS AND EMOTIONS TO SUCH AN EXTENT THAT THE ENTIRE WORLD SEEMS DRAMATICALLY DIFFERENT. SUCH "ALTERED STATES" ARE NOW THE SUBJECT OF INTENSE NEUROSCIENTIFIC RESEARCH.

ALTERED BRAIN STATES

Our normal waking state varies from daydreaming, through relaxed awareness, to sharply focused. The brain is capable of generating a much wider range of conscious experiences than this, though. Sometimes we slip outside the normal range spontaneously, when feverish or exhausted, for instance, or during or after an emotionally overwhelming event. We may also deliberately seek to get out of our normal state by engaging in rituals such as prolonged dancing, through meditation, or by taking drugs.



TRANCE STATE

A trance is an altered state of consciousness that may be induced by hypnosis, drugs, or ritual. It can be pleasurable or frightening.

Frontal lobe

May go "offline" in altered states, reducing critical thinking; can be hyperactive during meditation, indicating increased attention

Parietal lobe
Altered activity here may create out-of-body feeling or distorted experience of space and time

Corpus callosum
Allows the two hemispheres to communicate; blissful states are linked with greater synchrony between hemispheres and sudden switches of activity from one hemisphere to another

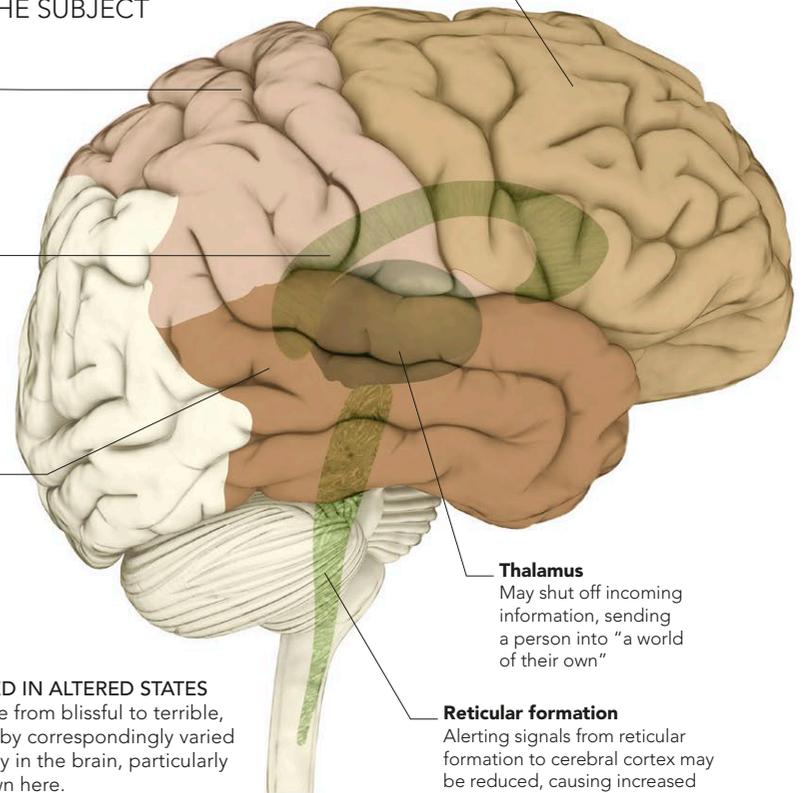
Temporal lobe
Flurries of activity here are associated with unexplainable experiences, including hallucinations and sensing auras or an invisible presence

Thalamus

May shut off incoming information, sending a person into "a world of their own"

Reticular formation

Alerting signals from reticular formation to cerebral cortex may be reduced, causing increased relaxation and sense of well-being



BRAIN AREAS INVOLVED IN ALTERED STATES

Altered states may range from blissful to terrible, and they are generated by correspondingly varied changes in neural activity in the brain, particularly involving the areas shown here.

DISSOCIATION

Dissociation refers to instances when elements of consciousness (the sensations, thoughts, and emotions of the moment) that are sometimes bound together as a whole are, instead, experienced separately or are cut out of conscious awareness. Many altered states fall into this category. Usually, dissociation is referred to as a mental or behavioral disorder, but some "normal" conscious states, such as daydreaming or concentrating, are dissociative. It is more accurate to look at these conscious states as a spectrum (see below), with highly unified or "bound" experience at one end and "fractured" consciousness at the other.

HYPNOSIS

Hypnosis is a form of dissociation in which a person's field of attention is narrowed to a single thought, feeling, or idea. When experiencing this state of mind, normal distractions and preoccupations may be kept out of mind. People undergoing hypnosis voluntarily may become very suggestible to the hypnotist's ideas, so it is often used therapeutically, for instance, to break a habit such as smoking.



BOUND TOGETHER

NORMAL

FRACTURED

FEELING OF ONENESS OR "MEANINGFULNESS"



STATE OF EXTREME RELAXATION WITH FEWER INTROSPECTIVE THOUGHTS



DAYDREAMING; CAN SPRING BACK TO ALERT IMMEDIATELY



HIGH LEVEL OF ALERTNESS AND AWARENESS

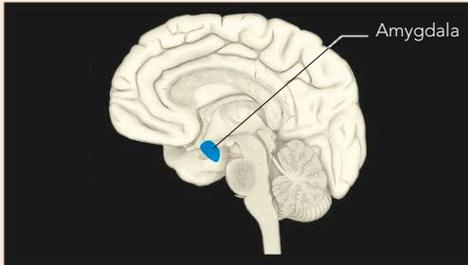


SEPARATION FROM SELF OR SENSE OF BEING DISTANCED FROM REALITY



MINDFULNESS

Brain-imaging studies suggest that mindfulness practices are associated with an amygdala that is smaller and has reduced connections with those parts of the brain associated with fear, anxiety, and panic. It thickens tissue in the prefrontal cortex—an area that produces thoughtful, calm responses.



CALMING EFFECT

The amygdala reacts to threats and surprises, generating emotion. Mindfulness practice appears to calm the area down.

MINDFULNESS TRAINING

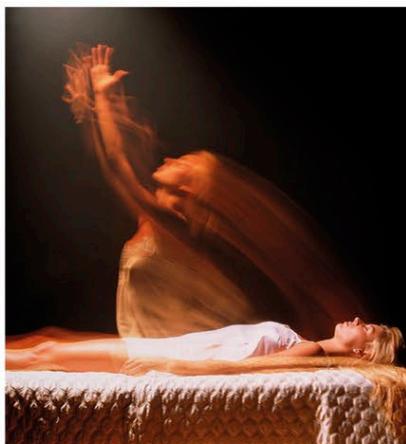
Meditation trains people to hold attention without overreacting to passing ideas and events. Mindfulness is currently the most fashionable method. Transcendental meditation, Zen, and other practices aim to achieve the same things: calmness and reduced anxiety.

OUT-OF-BODY EXPERIENCES

Out-of-body experiences (OBEs) occur when the internal representation of the body is out of kilter with the real body. This often happens in dreams, but when you are awake, it may be interpreted as a supernatural event. OBEs typically occur as you wake up, before the brain has properly reconnected with the external world (see p.173), and are associated with activity in the temporoparietal junction.

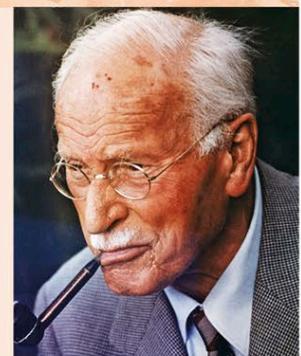
NEAR-DEATH EXPERIENCES

OBEs are often accompanied by feelings of ecstasy, and they are a central feature of many so-called "near-death experiences."



THE COLLECTIVE UNCONSCIOUS

Carl Jung (1875–1961) was a Swiss psychiatrist who developed the idea of the collective unconscious—a part of the unconscious mind shared by everyone as a product of ancestry, which can be accessed in certain states of mind. He thought it included "archetypes" (innate, universal concepts), such as the mother, God, and hero, and that we detect their influences in the form of myths, symbols, and instinct. Presumably, he saw the collective unconscious as a sort of "folk memory," embodied in the structure of the brain.

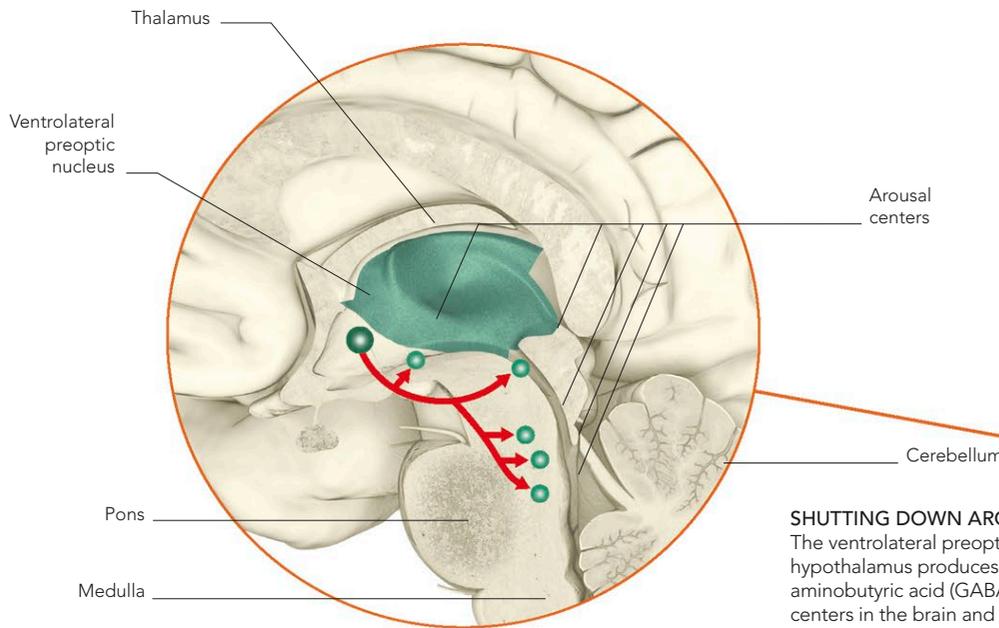


SLEEP AND DREAMS

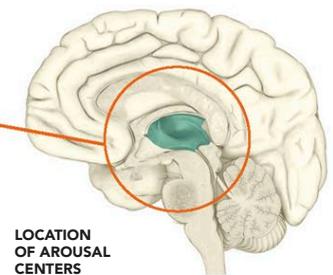
ABOUT A THIRD OF LIFE IS SPENT ASLEEP, DURING WHICH TIME THE BRAIN REMAINS ACTIVE, FULFILLING A RANGE OF IMPORTANT FUNCTIONS. DURING SLEEP, THE BRAIN GENERATES DREAMS, WHICH PROVIDE US WITH SOME OF THE MOST INTENSE AND STRANGE EXPERIENCES THAT WE HAVE.

THE SLEEPING BRAIN

No one is quite sure what it is about sleep that makes it so important. One theory is that it allows “down time” for the body to repair itself. One way it may do this is by draining away detritus—the broken-down molecules that accumulate in cerebrospinal fluid during cell activation. Another is that it simply keeps the person out of danger for a period of time during each day, by keeping him or her still. A third is that the brain needs to switch off from the outside world in order to sort, process, and memorize information. Certainly, important memory functions do occur during sleep, but whether this is the primary purpose of sleep remains unclear. Sleep-wake cycles are controlled by neurotransmitters that act on different parts of the brain to induce sleep or waking up. Research also suggests that a chemical called adenosine builds up in the blood while we are awake and causes drowsiness; while we sleep, the chemical is gradually broken down.



SHUTTING DOWN AROUSAL SIGNALS
The ventrolateral preoptic nucleus in the hypothalamus produces the neurotransmitter gamma aminobutyric acid (GABA), which travels to arousal centers in the brain and shuts them down for sleep.



LOCATION OF AROUSAL CENTERS

SLEEP PROBLEMS

About one in five people suffer problems with sleep. The most common complaint is insomnia—the inability to fall or stay asleep. Insomnia is treated with drugs that bind to receptors for GABA (the brain’s inhibitory neurotransmitter). Narcolepsy is a sleep disorder that causes people to fall asleep suddenly and inappropriately, or to feel exhausted throughout the day. Narcoleptics cannot experience the amount of restorative deep sleep that healthy people have and live in a state of sleep deprivation. When they fall asleep—which may occur with little warning—they enter REM sleep almost immediately and have vivid dreams. Sleepwalking occurs in deep sleep when a block that stops motor impulses is lifted but other sleep mechanisms remain. Sleepwalkers do complex things, such as driving cars, but perform actions robotically because they are following automatic action plans stored in the unconscious brain.

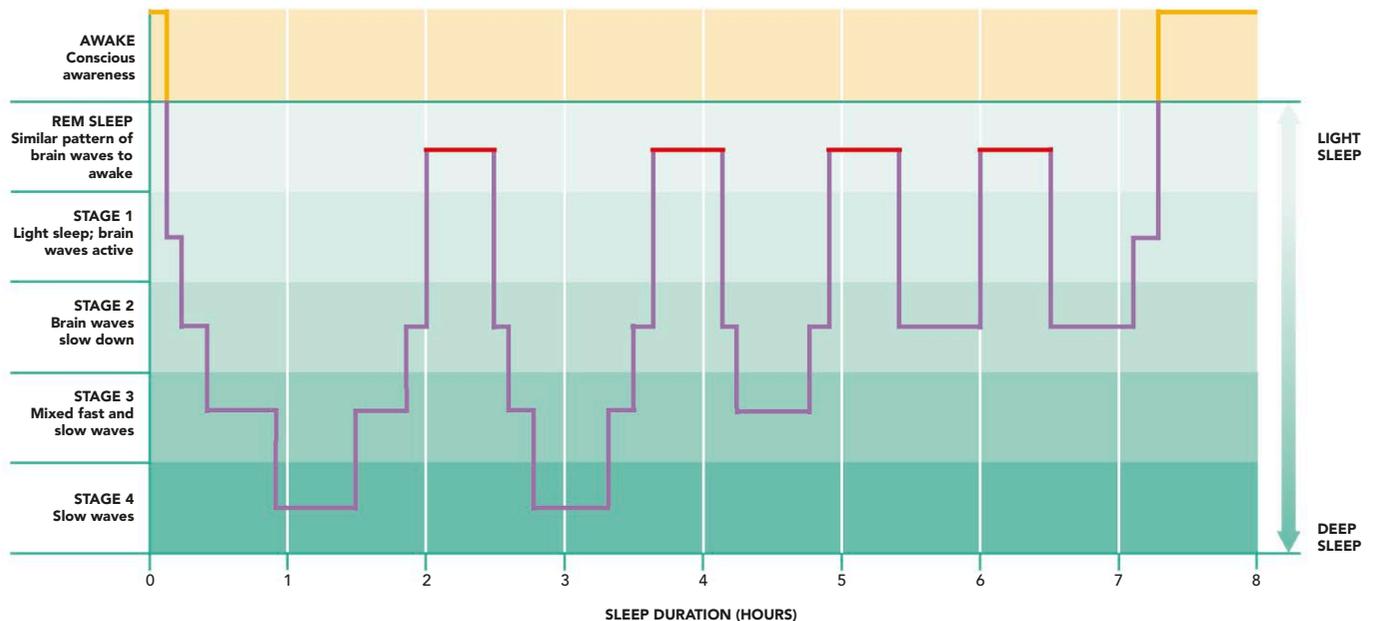


KEY

- AWAKE
- REM SLEEP
- NON-REM SLEEP

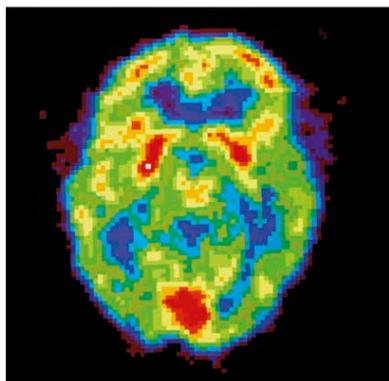
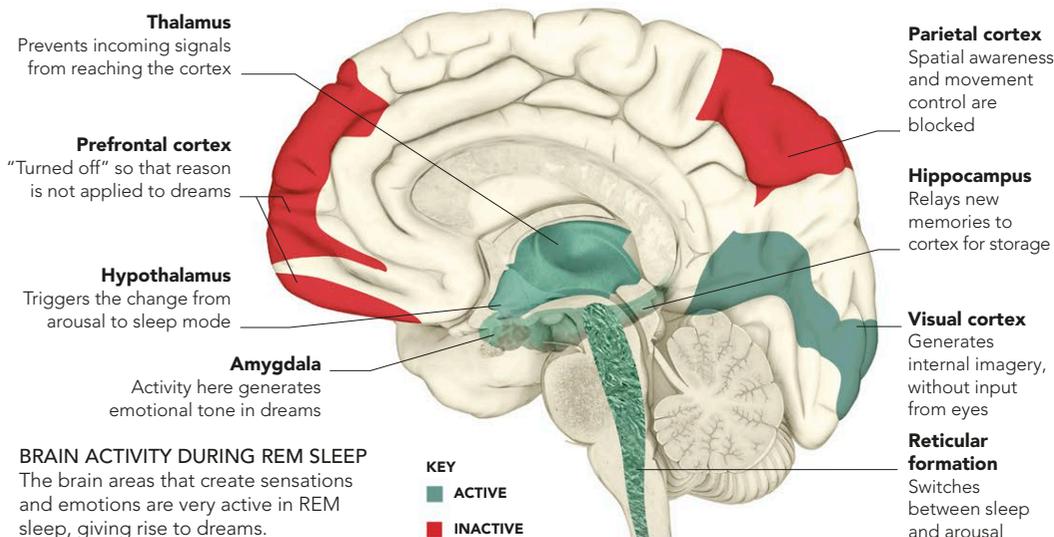
THE SLEEP CYCLE

Although sleep may seem like a constant state, it actually occurs in cycles. Brief, dreamlike fragments mark stage one, while stage two involves total loss of consciousness and muscle paralysis. Deep sleep occurs in stages three and four, where brain activity is low. Rapid eye movement (REM) sleep signals vivid dreaming.

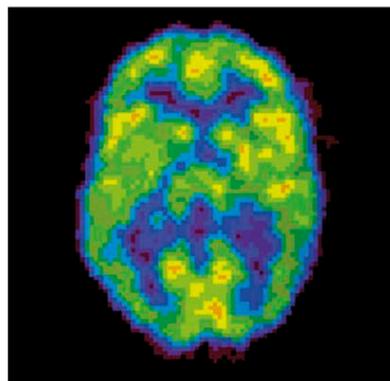


THE DREAMING BRAIN

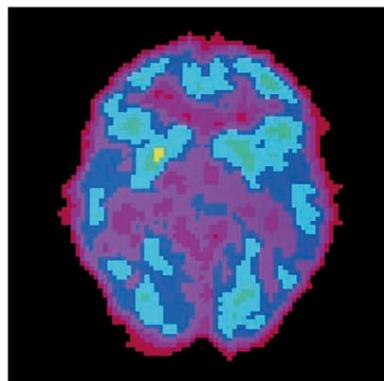
There are two types of dreaming. During deep sleep, we have vague, often emotionally charged and nonsensical dreams that are often forgotten immediately. The brain is not very active but seems to be gently processing information in order to lay it down in memory. In REM sleep, the brain becomes very active and produces vivid, intense “virtual realities,” typically with a narrative. The part of the brain that processes sensations is very active during REM dreaming. The frontal lobes, which include areas that apply critical analysis to our experience, are effectively turned off, so when crazy events happen in our dreams, we just accept them.



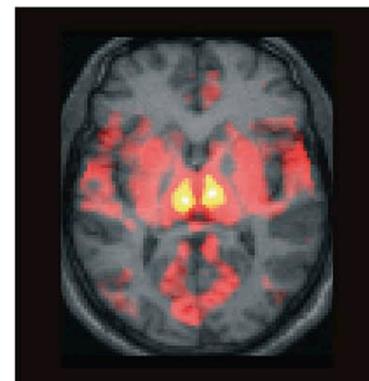
AWAKE
This PET scan shows the areas that are active when a person is awake (shown in red and yellow). The green and blue areas are less active.



DEEP SLEEP
This PET scan shows that activity quiets down in many areas of the brain during deep sleep. The purple areas are the least active.



DRUGGED SLEEP
Most sleeping drugs induce a deeper sleep than normal. The purple areas on this PET scan show that much of the brain is inactive.



REM SLEEP
This fMRI scan shows activity (yellow most active, then red) during REM sleep, spanning areas involved with generating sensations.

WAKING AND LUCID DREAMS

Usually, when shifting from dreaming to waking, several changes occur together in the brain. The block on incoming stimuli is lifted, so external sensory inputs enter the brain again, which overrides and turns off the internally generated sensations that comprise dreams. The block on outgoing signals from the motor cortex is also lifted so that it becomes possible to move again. Additionally, the frontal lobes are reactivated, shifting us back into a normal state of consciousness in which we know who and where we are and can tell the difference between fantasy and reality. Lucid dreams occur when the frontal lobes “wake up” during sleep, but the block on incoming and outgoing signals continues. Because the frontal lobes are active, the dreamer is able to deduce that he or she is actually dreaming and experience events in a normal state of mind.



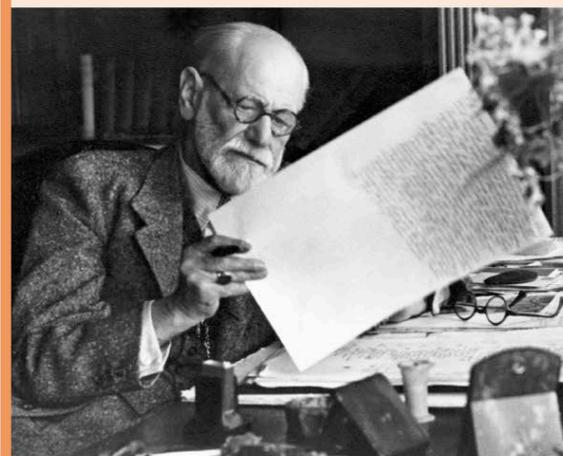
ANYTHING IS POSSIBLE
In lucid dreams, you can control the action just as in a waking daydream, but the experience is more intense and seems real.



SLEEP PARALYSIS
Waking up while the motor-impulse blockade is still operating is known as sleep paralysis. This frightening sensation feels like being weighed down, which may be the origin of the myth of the incubi and succubi, evil spirits that were thought to squat on sleepers.

FREUD AND PSYCHOANALYSIS

Sigmund Freud was an Austrian psychiatrist, who founded the study of psychoanalysis. He called dreaming the “royal route to the unconscious,” because he thought that dreams revealed the emotions and desires that we suppress when we are awake. He postulated that these suppressed desires are often too shocking to be consciously admitted, and even in dreams they have to be disguised in symbols. Freudian dream analysis aimed to decode the symbols to reveal the true nature of the desires of the dreamer.



TIME

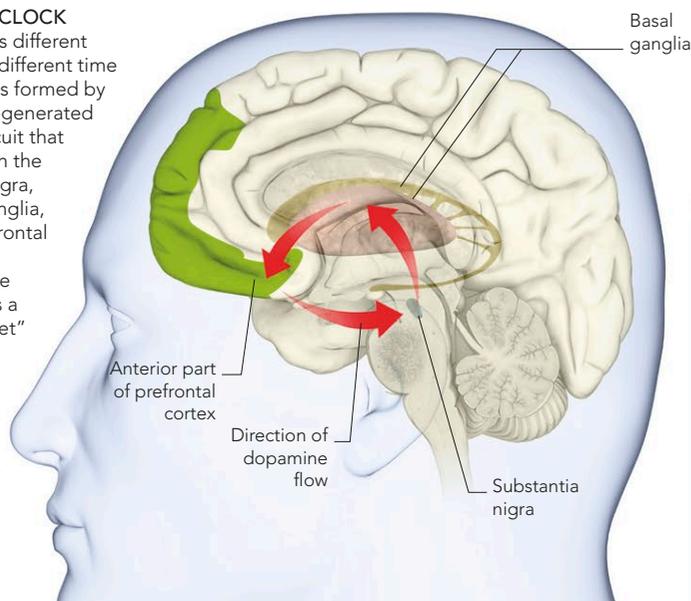
TIME IS NOT A CONSTANT IN THE BRAIN—IT SPEEDS UP AND SLOWS DOWN ACCORDING TO WHAT IS BEING EXPERIENCED. THE BRAIN HAS MANY DIFFERENT WAYS OF MEASURING TIME. LONGER DURATIONS, SUCH AS DAY LENGTH, ARE MEASURED BY THE EBB AND FLOW OF HORMONES, WHILE THE MILLISECOND INTERVALS INVOLVED IN MANY BRAIN PROCESSES ARE MARKED BY THE OSCILLATION OF NEURONS.

SUBJECTIVE TIME

The passage of time as we experience it (known as subjective time) is not the same as the regular passage of time as measured by our clocks (objective time). The crucial difference is that subjective time can speed up and slow down, according to what we are experiencing. On a moment-by-moment scale, the rate at which time seems to pass is dictated by the rate of firing, or oscillation, of clusters of neurons. The faster they fire, the more events we register in any given second, giving us the impression that time lasts longer. Neuronal firing is controlled by neurotransmitters—excitatory ones speed it up, and inhibitory ones slow it down. Young people have more excitatory neurotransmitters and, therefore, are able to cope with faster external events.

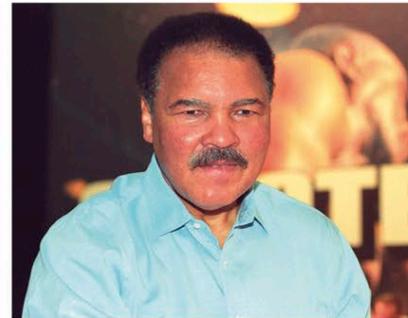
THE BRAIN CLOCK

The brain has different “clocks” for different time scales. One is formed by a dopamine-generated neuronal circuit that runs between the substantia nigra, the basal ganglia, and the prefrontal cortex. Each “cycle” of the clock creates a single “packet” of subjective time.



TIME PASSING SLOWLY

Stimulants like caffeine speed up the brain, allowing more external events to be registered. This produces a sense of time stretching out.



TIME RUSHING BY

Severe depletion of dopamine, as in Parkinson's disease, may slow the brain down so much that the external world seems to be rushing by.

CATATONIA

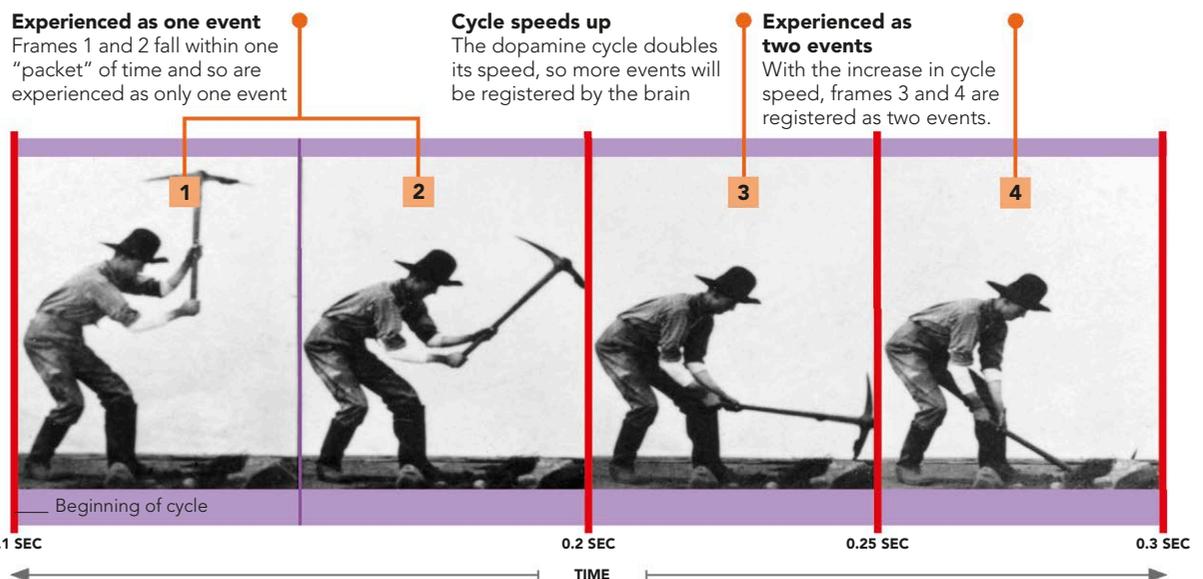
Catatonia is a state most commonly observed in people with certain types of schizophrenia. The sufferer becomes motionless and stops reacting to external stimuli. They may remain mute, or rigid, for days on end, sometimes striking bizarre poses, which would normally be impossible to maintain. The state seems to come about when the flow of dopamine slows down, and people who have experienced this condition report that they lose all sense of time.

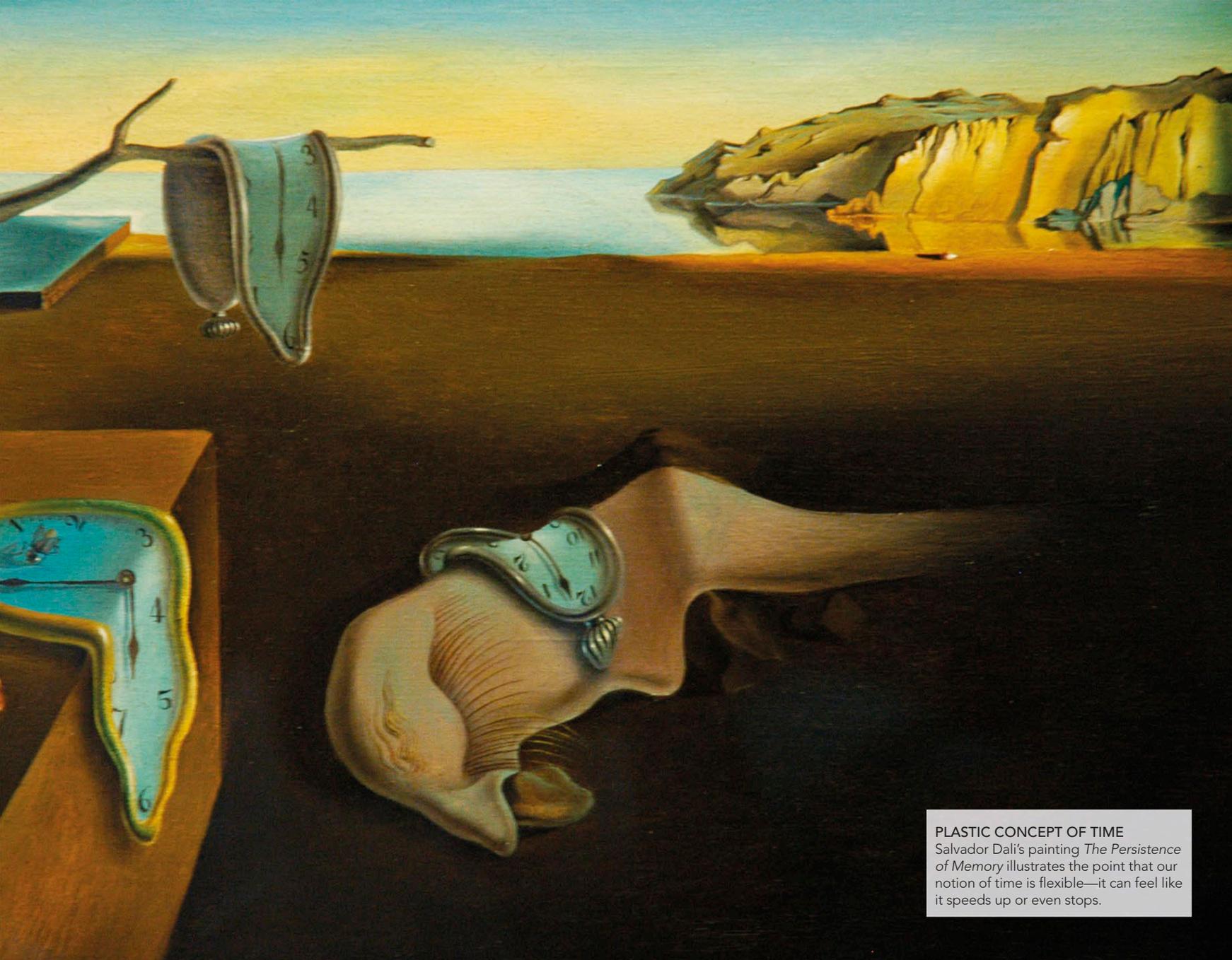
PACKETS OF TIME

The brain divides time into “packets” (a cycle of neural activity), each of which registers a single event. The size of the packet depends on how fast the relevant neurons are firing, but regardless of the size of the packet, the brain will only be able to take in one event from that packet. If two events happen, the brain will miss the second one. Some events will always appear blurred to us, such as the beating of a dragonfly's wings, because several flaps occur in each packet.

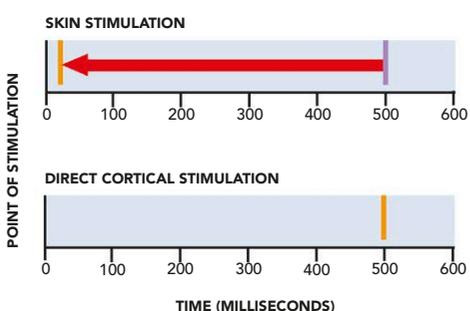
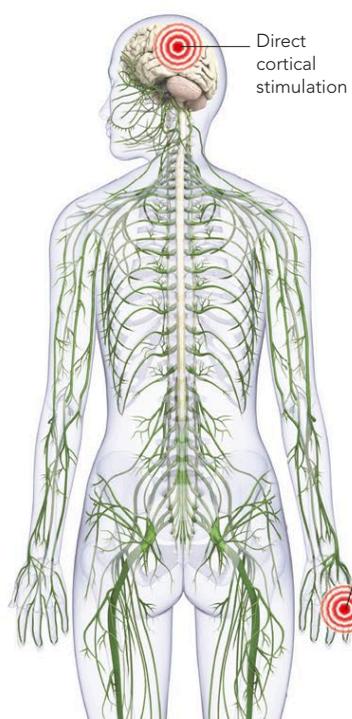
EXPERIENCING EVENTS

If the “clock” neurons fire only once in $\frac{1}{10}$ of a second, only one event will be registered in that time, although many more may actually occur. If the neural clock doubles its speed, both events will be registered because the neural clock will have created two “packets” of subjective time.





PLASTIC CONCEPT OF TIME
 Salvador Dalí's painting *The Persistence of Memory* illustrates the point that our notion of time is flexible—it can feel like it speeds up or even stops.



KEY
█ Stimulus registered in brain
← Start of conscious experience
← Backdating

LIBET'S EXPERIMENT
 The backdating effect does not kick in if the brain is stimulated directly. This has been shown by stimulating the "hand" area of the somatosensory cortex, which produces the same subjective feeling as touching the hand. But, as Benjamin Libet discovered, if you stimulate the brain and the hand at the same time, the feeling brought on by touching the hand is reported before the one produced by stimulating the brain.

BACKDATING TIME

It takes on average half a second for the unconscious mind to process incoming sensory stimuli into conscious perceptions. Yet we are not aware of this time lag—you think you see things move as they move, and when you stub your toe you get the impression of knowing about it right away. This illusion of immediacy is created by an ingenious mechanism, which backdates conscious perceptions to the time when the stimulus first entered the brain. On the face of it, this seems impossible because cortical signals take the same "real" time to process to consciousness, but somehow we are tricked into thinking we feel things earlier. One way it might be explained is that consciousness consists of many parallel streams and that the brain jumps from one to another, revising them and redrafting them.

HALF A SECOND LATE
 We become conscious of events around us nearly half a second after they occur, but we do not notice this time lag.



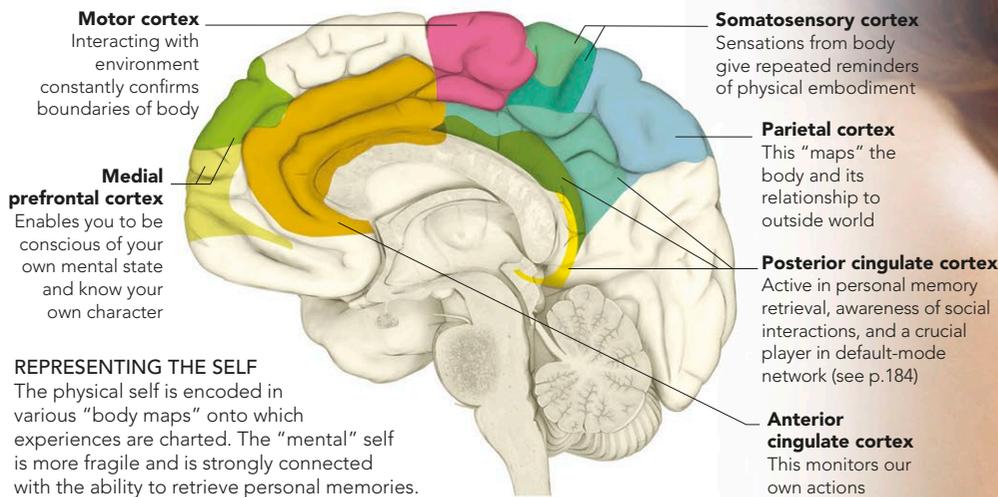
THE SELF AND CONSCIOUSNESS

THE HUMAN BRAIN GENERATES AN IDEA OF “SELF” THAT ALLOWS US TO “OWN” OUR EXPERIENCES AND FORGES A CONNECTION BETWEEN OUR THOUGHTS AND INTENTIONS, OUR BODIES, AND OUR ACTIONS. OUR SENSE OF SELF ALSO ALLOWS US TO EXAMINE OUR OWN MINDS AND TO USE WHAT WE SEE TO GUIDE OUR BEHAVIOR.

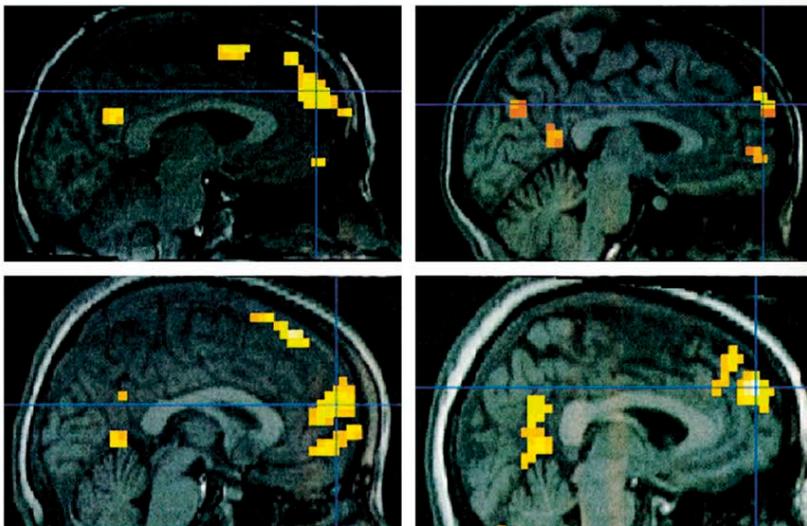
WHAT IS THE SELF?

We divide the world into that which is subjective and internal and that which is objective and external. The boundary between the two acts like a container, which holds the former and places the latter outside. This container is what we know as the “self.” Among other things, it includes our thoughts, intentions, and habits, as well as our actual bodies. Except in altered states (see p.186), all experiences we report include a sense of self, but most of the time the sense is unconscious. This “consciousness-with-self” is what we generally call “consciousness.” When the sense of self becomes conscious, we talk of being “self-conscious.”

LEVELS OF CONSCIOUSNESS	
The sense of self lies at the heart of our experiences. It takes various forms and operates at different levels of our consciousness.	
Introspection	You think about your own thoughts or actions; one form is being “self-conscious” about your performance of an act.
Normal Consciousness	You feel that your thoughts are your own and your actions are the result of your decisions; you can report experiences.
Knowledge	You react to the environment, perhaps by doing complex actions (such as driving), but if asked, you can’t recall doing it.
Unconsciousness	In deepest sleep, your brain does not perceive the outside world or generate a sense of self to experience anything.



REPRESENTING THE SELF
The physical self is encoded in various “body maps” onto which experiences are charted. The “mental” self is more fragile and is strongly connected with the ability to retrieve personal memories.

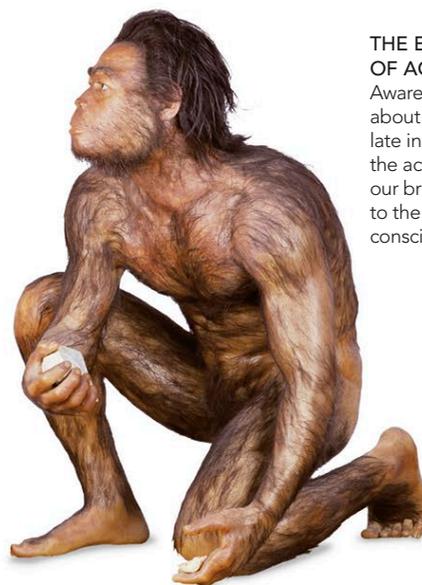


SELF-REFLECTIVE THOUGHT
This kind of thought creates activity in several areas of the brain. The areas toward the top and back are mainly concerned with body “maps,” while those at the front are concerned with the mental self.

EXAMINING THE “I”
Trying to examine the “I” is like trying to look at your own eye—it is impossible because you are trying to see the thing you are using to see with. In effect, a shadow self arises, observing the “I.”

AGENCY AND INTENTION

Agency is our sense of control over our actions. We feel that our conscious thoughts dictate what we do, but this appears to be incorrect. A famous experiment by Benjamin Libet (see below) revealed that a person's brain starts to plan and execute a movement unconsciously, before the person has consciously decided to do it. This is often interpreted to show that our sense of agency and of making "decisions" is illusory. The sense of agency we experience may actually have evolved primarily to give us early warning, not of our own actions, but of the actions of others. Because we feel ourselves to be agents, we also intuit agency in others and thus think we know their intentions and can predict what they will do.

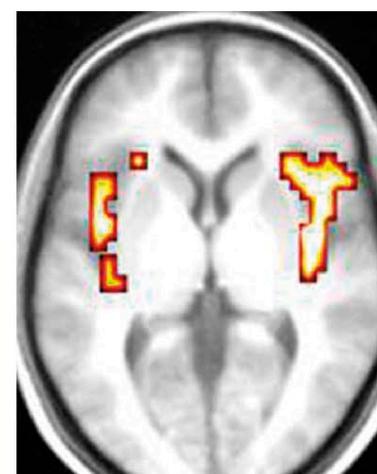


THE EVOLUTION OF AGENCY

Awareness of what we are about to do may have arisen late in our evolution, once the action-planning part of our brain had connected to the areas that support consciousness.

SCHIZOPHRENIA AND AGENCY

People with schizophrenia may have a disturbed sense of agency. Some attribute their own actions to the intentions of others, claiming they are being "controlled" by outside forces; others, that they "cause" events unconnected with their own actions, such as moving the sun. Studies have suggested that these disturbances of the sense of agency are the result of failure to predict the consequences of an action.

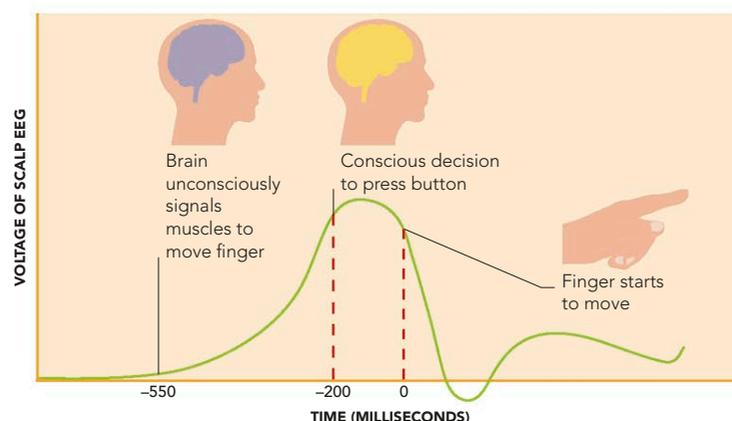


AUDITORY HALLUCINATION

This fMRI scan shows brain activity in a hallucinating schizophrenic. The lit-up right hemisphere speech areas may elicit sounds that could be imagined as an external voice, distorting the sense of agency.

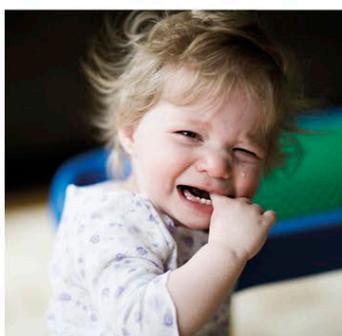
FREE WILL EXPERIMENT

Libet asked volunteers to make a finger movement when they wanted to and to report the exact moment they "decided" to move by noting the time on a huge clock face with a sweep hand. Meanwhile, their brain activity was monitored, and EEGs showed the unconscious activity that planned the movement and sent the message to move to the relevant muscles. The timing of this activity was also noted, along with the time that the movement became visible. The experiment revealed that the conscious decision to make the movement occurred about one-fifth of a second after the brain had instructed the muscles to move.



DISLOCATED SELF

The brain holds various "body maps"—internal representations of the physical self. The earliest, most basic map to emerge tells us where our body ends and the rest of the world begins. A more developed body "atlas" enables us to know our spatial location in the world. Normally, the internal maps and the body itself are closely matched, but it is possible for them to be askew. If a person loses a limb, for example, they may develop what is known as a phantom limb—a feeling that they have a limb that, in fact, no longer exists (see p.104). People can also be tricked into "owning" a limb or even a body that is not actually theirs.



INFANT BODY MAPS

Babies probably do not distinguish between their body and external objects until their body maps start to take in information from the world.

VIRTUAL BODY

People can be fooled into "losing" their real body and adopting another. In one experiment, volunteers wore virtual-reality headsets that substituted their view of their legs for those of an adjacent doll-sized mannequin. When the model was touched, the person reported feeling that the model's limbs were theirs. They also felt as though they had shrunk in relation to their surroundings.



LOSING THE SELF

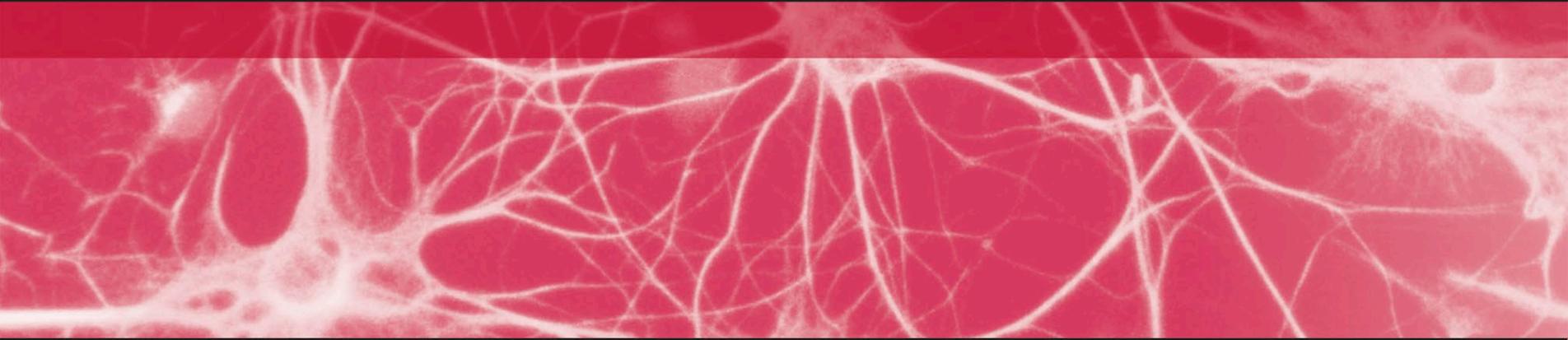
Normal conscious activity involves keeping our "self" in mind, at least unconsciously. This means we see the world from our own, embodied perspective and color our perceptions and behavior with the background notion of ourselves as agents. Sometimes, though, the self temporarily disappears—for example, when we enter mental states such as "flow" or "loss of control" (see below). These states can be both joyous and potentially perilous experiences.

Flow

In this pleasant state, we become so absorbed in something outside ourselves that self-consciousness vanishes and, with it, the self's tendency to inhibit and interfere with whatever else the brain is doing. This allows us to perceive things more intensely and may help us perform better.

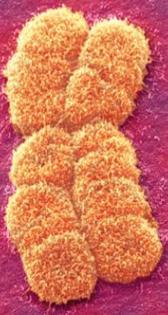
Loss of control

Failing to exercise control of our emotions is another instance of self-diminishment, but, unlike flow, it can be seriously disadvantageous. Brain-imaging studies suggest that people "lose it" when the prefrontal area of the brain fails to respond adequately to alerts sent by the anterior cingulate cortex (ACC), which monitors one's own actions. Under provocation, the ACC registers that the emotional brain is tending to produce impulsive behavior, and this usually triggers activity in the prefrontal cortex that inhibits the response. When someone is unusually stressed or tired, however, the prefrontal cortex may not respond, so the emotions are acted out. People in this state often report a sense of being "taken over," as though their agency has been hijacked.



NO TWO BRAINS ARE EXACTLY ALIKE. ALTHOUGH THEY ARE BUILT ACCORDING TO THE SAME BASIC PLAN, EACH ONE IS PRODUCED FROM INSTRUCTIONS ENCODED IN A UNIQUE SET OF GENES, WHICH ARE ENGAGED IN COMPLEX INTERACTION WITH THE ENVIRONMENT. WE OFTEN THINK THAT OUR INDIVIDUALITY IS EXPRESSED THROUGH OUR PERSONALITY, BUT RECENT STUDIES SUGGEST THAT PERSONALITY IS A MUTABLE PHENOMENON. WE ALL HAVE SUBTLY DIFFERENT PERSONALITIES THAT WE EXHIBIT IN DIFFERENT SITUATIONS.

THE INDIVIDUAL BRAIN



NATURE AND NURTURE

NATURE AND NURTURE ARE THE TWO FACTORS SHAPING THE WAY THE BRAIN FUNCTIONS. NATURE REFERS TO AN INDIVIDUAL'S GENOTYPE—THAT IS, THE PARTICULAR SET OF GENES INHERITED FROM THE PARENTS. THE BRAIN IS ALSO ALTERED BY NURTURE, WHICH IS ALL THE ENVIRONMENTAL FACTORS AN INDIVIDUAL IS EXPOSED TO THROUGHOUT LIFE.

GENES AND THE ENVIRONMENT

A gene is a unit of hereditary information linked to one or more physical traits (such as eye color). The full complement of genes—around 20,000 in all—existing in the nucleus of the cell is called the genome. Genes are arranged on chromosomes, of which a healthy person has 22 pairs, plus one sex pair. Genes are made of DNA (see panel, below), and some genes achieve their effects by the production of proteins. However, 99 percent of DNA is noncoding—some of it regulates gene expression, while other parts of it have no known function and are sometimes referred to as “junk” DNA. Genes are like dimmer switches—they can turn their activity (expression) on, off, up, or down. In the brain, gene expression affects the levels of neurotransmitters, which, in turn, influences complex functions like personality, memory, and intelligence. However, neurotransmitters also affect gene expression. Environmental influences affect patterns of gene expression so that brain function also depends upon factors

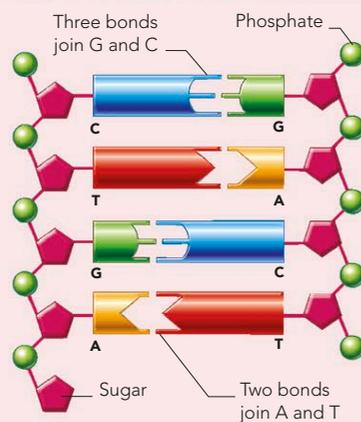
such as diet, geographical surroundings, social networks, and even stress levels. Chemical tags attach to DNA and alter gene expression, a process known as epigenetic alteration (see opposite).



MUSICAL BRAIN
Having a “musical brain” may be the result of being raised in a family that values music and/or genetic influences.

THE DNA MOLECULE

Found in the nuclei of all cells in the human body except red blood cells, the DNA molecule is shaped like a twisted ladder—the famous double helix. The two strands of the helix are held together by chemicals called bases, which are arranged in pairs. There are four bases, known by the letters A, C, G, and T, and they always pair in the same combinations (A pairs with T, and C pairs with G). The sequence of base pairs can be read by the cell as the instructions for making proteins.

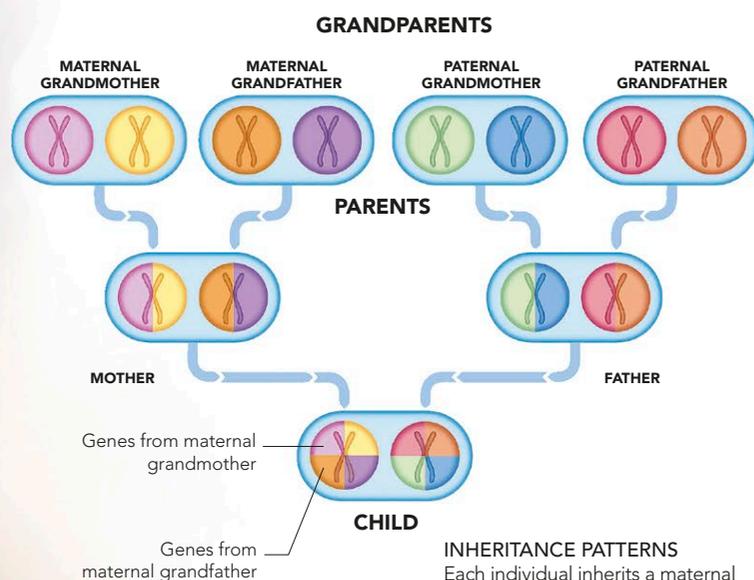


BUILT FOR SPEED

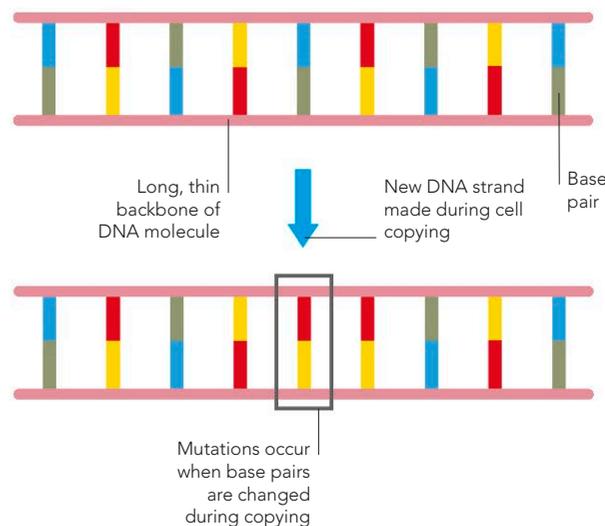
Like many aspects of physical performance, sprinting is genetically influenced. For example, a gene for insulin-like growth factor (IGF) influences an athlete's muscle mass. Although most successful sprinters share a genetic advantage, the right genes alone are not enough. Athletes have to train hard and have a desire to win if they are to become champions.

GENETICS AND THE BRAIN

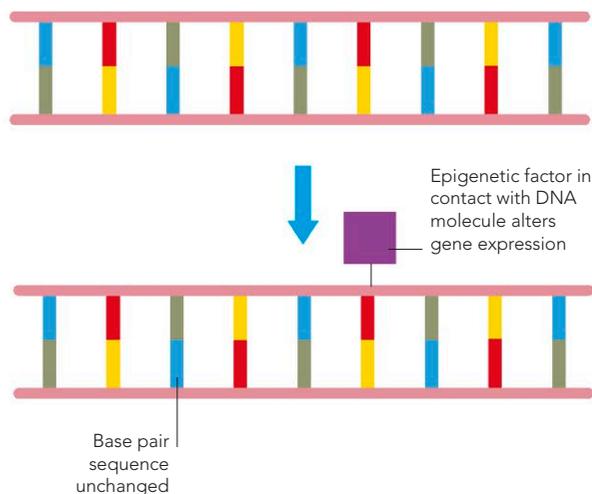
Genes make proteins, which have many roles in the body. Some form structures, such as hair, while others, such as enzymes, regulate processes. For example, several genes in the genome may code for the protein molecules that make serotonin, one of the neurotransmitters involved in mood. Each variant of this gene makes a slightly different protein molecule, which may carry out its job more, or maybe less, efficiently. Thus, gene variants may result in one person having more serotonin and another person less serotonin. Less serotonin may mean a predisposition to depression or a tendency to overeat. This is also true of other neurotransmitters, such as dopamine—a lack of dopamine has been linked to increased risk-taking behavior. Therefore, your genotype can affect the structure and functioning of your brain, which, in turn, will influence behavior. Another way that behavior may be altered by genes is through epigenetic changes. These occur when the pattern of gene activation—rather than the genes themselves—is altered by molecular changes in DNA near to the genes. The changes may be passed on through several generations. Trauma provokes epigenetic changes in brain cells, probably due to raised stress hormones. People who commit suicide following childhood abuse have been found to have more epigenetic changes affecting genes, which act in the brain. Their offspring also show such changes and are also more likely than others to commit suicide. Research is underway to find a method of reversing epigenetic alterations.



INHERITANCE PATTERNS
Each individual inherits a maternal allele and a paternal allele that make up a pair for each gene. Some alleles may be dominant, which affects how traits are inherited.



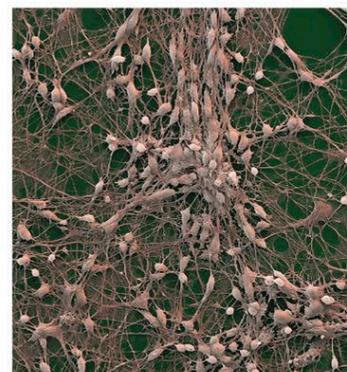
MUTATION
Genes are a series of base pairs, linked molecules that form rungs in a DNA ladder (see opposite). Molecules of guanine (G), cytosine (C), adenine (A), and thymine (T) join in G-C and A-T pairs. The pair sequence in a particular gene is similar in all of us, but variations in the sequence help to make us unique. These may be introduced as a result of errors, or mutations, during the process of cell copying.



EPIGENETIC CHANGES
Epigenetic changes alter how the gene works, without actually changing the base pairs. Molecules from outside the gene, known as epigenetic factors, attach to the DNA and make it difficult for one or more genes to act in the normal way in the body. Epigenetic factors can be passed through a number of generations, but, unlike mutations, they will finally disappear.

THE PLASTIC BRAIN

The brain was once believed to be immutable from birth, with a certain number of brain cells and fixed neuronal circuits. The only changes thought to occur were the loss of brain cells and a reduction in brain volume. But researchers have shown that experience and learning remodel brain circuits. Examples of such neuronal plasticity include long-term potentiation, where memory and learning generate new circuits (see p.158); the remodeling of the brain after a stroke or in drug addiction to strengthen pathways or create new ones; and the formation of new brain cells (neurogenesis). The brain, it seems, has a certain ability to repair itself and continue to grow and develop throughout life.



BIRTH OF NEURONS
This colored electron micrograph shows neural progenitor cells. These cells lie between stem cells and fully differentiated cells. They are capable of developing into neurons and other neural cells.

INFLUENCING THE BRAIN

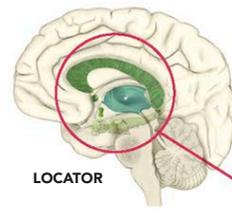
EVERYONE'S BRAIN IS DIFFERENT, AND SOME STUDIES SUGGEST THAT GENDER AND SEXUAL ORIENTATION ARE REFLECTED IN DIFFERENCES IN THE BRAIN'S ANATOMY AND FUNCTIONING. THE BRAINS OF RIGHT- AND LEFT-HANDED PEOPLE ARE ORGANIZED DIFFERENTLY, AND EVEN SOCIAL AND CULTURAL INFLUENCES CAN SHAPE THE WAY THE BRAIN CARRIES OUT CERTAIN TASKS.

MALE AND FEMALE BRAINS

Research into brain differences between the sexes is controversial. Some are convinced that differences are culturally, not biologically, determined. However, many studies have found anatomical differences between female and male brains. The corpus callosum and anterior commissure (linking the hemispheres) are larger in women. This may be why women are more emotionally aware—the emotional right is better connected to the analytical left. It may allow emotion to be built more readily into thought and speech. Imaging studies may reflect stereotypical differences between the sexes, showing different areas connected in each—though this could be culturally influenced.

ONE OF THE CROWD

Just as each individual's face in this crowd is different and unique, so too are their brains. Genetic differences at birth are just one factor—cultural and environmental influences during life can also have a profound effect.

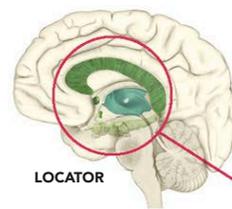
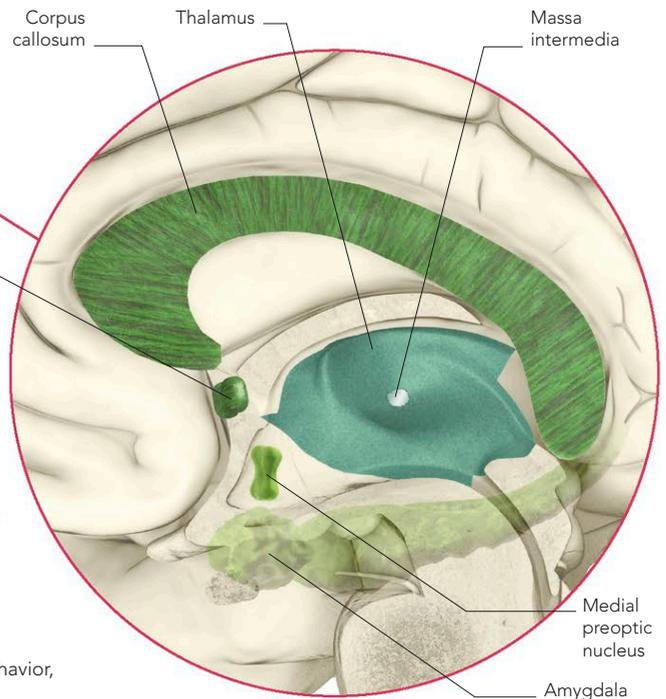


LOCATOR



THE MALE BRAIN

In men, the right side of the amygdala appears more likely to become active when stimulated. The medial preoptic nucleus of the hypothalamus, which is responsible for male-typical sexual behavior, is also larger in the male brain.

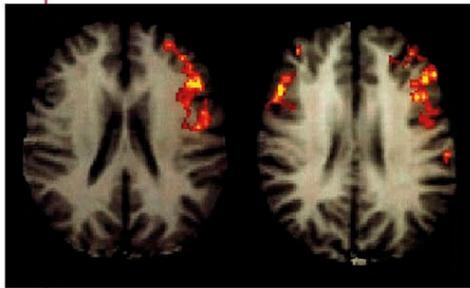
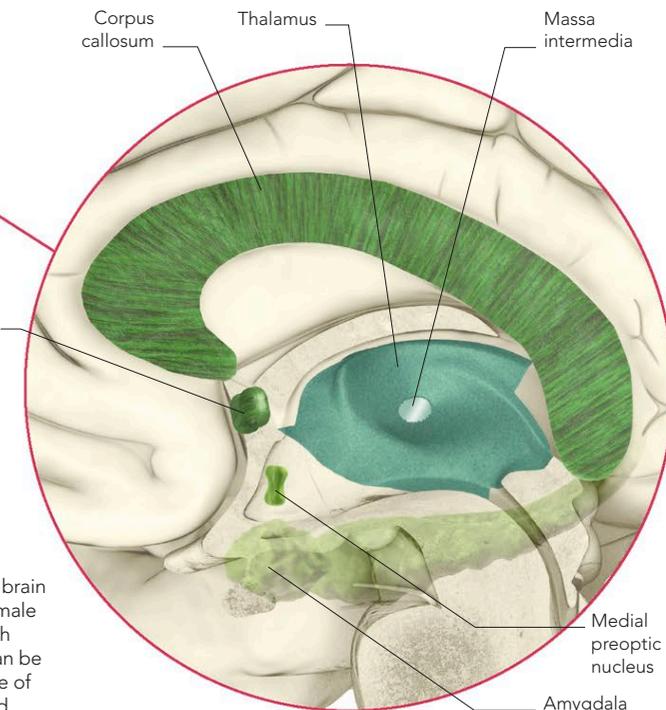


LOCATOR



THE FEMALE BRAIN

The anterior commissure of the female brain is about 10 percent bigger than in the male brain. Also, the massa intermedia, which connects the left and right thalamus, can be up to 50 percent larger. The significance of these differences is not fully understood.



MALE

FEMALE

RESPONDING TO LANGUAGE

These fMRI scans reveal that women show activity on both sides of the brain when responding to language. In men, however, the activity is restricted more to the left hemisphere (shown on the right side of the scan).

THE GAY BRAIN

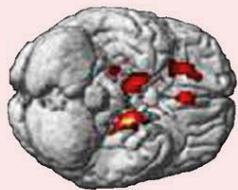
Brain-imaging studies suggest that in homosexual people, important brain structures involved in mood, emotion, anxiety, and aggression tend to resemble those of heterosexuals of the opposite sex. Heterosexual men tend to have asymmetric brains (the right hemisphere is slightly larger), a characteristic shared by gay women. Patterns of brain connectivity are similar between heterosexual women and gay men, particularly in areas involved with anxiety.



HETEROSEXUAL MEN



HETEROSEXUAL WOMEN



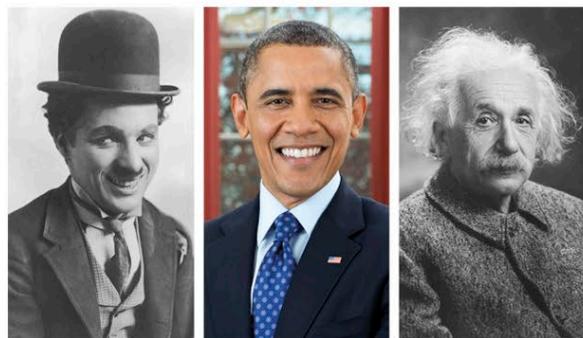
HOMOSEXUAL MEN



HOMOSEXUAL WOMEN

LEFT OR RIGHT HAND?

About 88 percent of people are right-handed—that is, they use their right hands rather than left for tasks requiring fine motor skills, such as signing their name. Archaeological evidence, such as tools, suggests this has been the case for several million years. About 70 percent of left-handers have language dominance in the left hemisphere, like right-handers, but 30 percent show language distributed between the hemispheres. This unusual arrangement may help those who have it to integrate ideas more easily than others, but there is little evidence to support this.



CHARLIE CHAPLIN

BARACK OBAMA

ALBERT EINSTEIN

LEFT-HANDED LUMINARIES

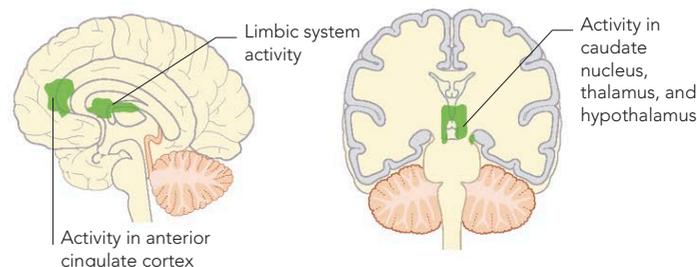
Many talented and brilliant people are or have been left-handed, including five of the last eight US presidents. This has led to a widespread notion that left-handed people are particularly gifted. Statistical analysis, however, suggests there are little or no consistent differences between left- and right-handers in IQ or other cognitive skills.

FAMILY EFFECTS

The way a person reacts to stress throughout their life is set, at least in part, by their very earliest experiences. In one study, fMRI scans were taken of sleeping babies' brains, which revealed activity stimulated by the sound of angry voices in two areas that react to emotional stimuli. Babies who came from homes where parents argued frequently showed greater activity than those from peaceful homes. The study suggests that the strength of a person's response to angry voices is primed in the cradle.

STRESSED BABY

Studies in which sleeping babies' brains were imaged as they were read to in an angry voice show activity in areas regulating emotion and stress.



Activity in anterior cingulate cortex

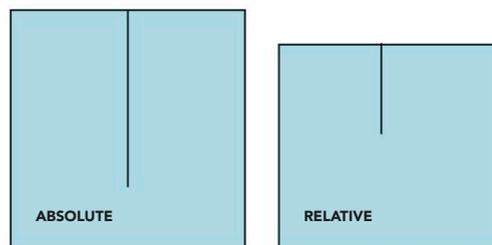
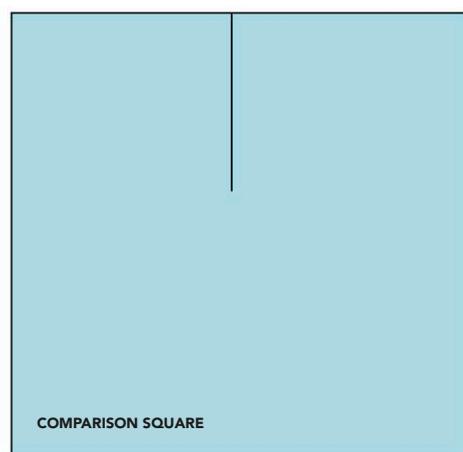
TWINS

Studies of identical twins who were separated at birth and brought up in different families show that, even as adults, they are very similar in terms of interests and personality, as well as looks. This demonstrates how genes continue to exert their effects throughout life and often override environmental influences. Twin fetuses, including fraternal twins, effectively compete for resources, and a baby's position in the womb can affect the hormones they receive. In the case of boy twins, for example, one may partially block the other's uptake of testosterone, reducing the degree of brain masculinization that happens to the other. Girls with a boy twin may receive a higher-than-normal dose of testosterone because the mother's release of the hormone is elevated if she is carrying a male fetus. Studies have shown such girls are more likely than those with girl twins to display "tomboyish" behavior.



CULTURAL INFLUENCES

Researchers have shown that culture influences the way the brain works. They carried out tests during fMRI scans on people raised in the US and people raised in East Asia, in which participants did puzzles involving lines in a square (see below). US culture is perceived to be focused upon the individual, while East Asian culture tends to be more focused on family and community. The brains of the US participants had to work harder when they were doing tasks involving context, while those of the East Asians worked harder when they had to judge individual lines. Brain activity lessened when participants undertook tasks related to their culture's comfort zone. Participants were also asked how closely they identified with their culture, and the brains of those who identified most strongly had to work the hardest when doing tasks related to the "opposite" culture.

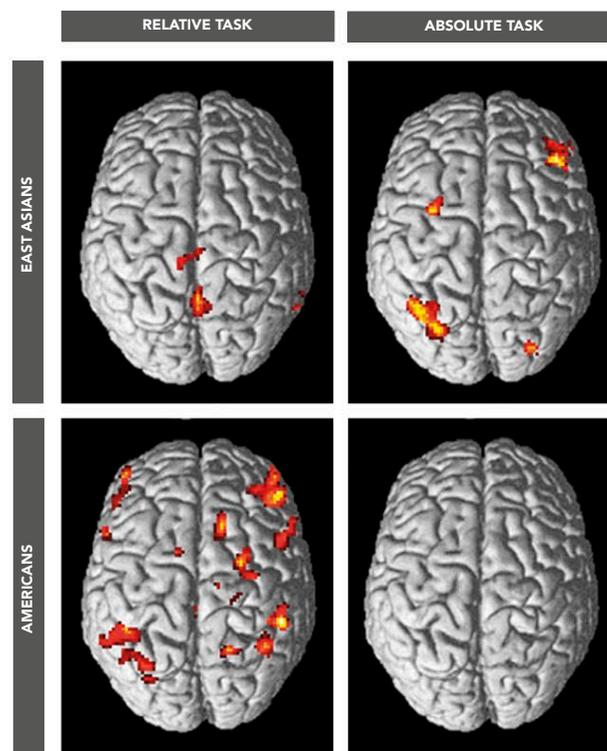


PERCEPTUAL TEST

The length of the line in this square may be perceived differently if it is compared to another line. Whether the brain is comfortable judging its length depends on the context of the test and cultural background.

ABSOLUTE AND RELATIVE TASKS

In an absolute task, the line's length is compared to that of the line in the comparison square. In the relative task, the length of the line and its relation to the size of the square is compared to the same relationship in the first square.

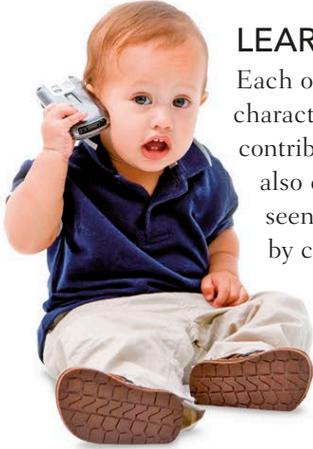


BRAIN ACTIVATION PATTERNS

East Asian brains have to work less at the relative line perception task, whereas Americans are the opposite, with the absolute task being less demanding of their brains. This is because these tests are "easier" when the tasks are more in line with cultural norms.

PERSONALITY

PERSONALITY IS GENERALLY AGREED TO BE A GROUP OF BEHAVIORAL CHARACTERISTICS TYPICALLY EXHIBITED BY AN INDIVIDUAL. SOME PEOPLE DISPLAY THE SAME BEHAVIOR IN DIFFERENT SITUATIONS AND AT DIFFERENT TIMES, WHILE OTHERS ARE MUCH MORE CHANGEABLE.



LEARNING TO BE YOU

Each one of us has a genetic blueprint that predisposes us to characteristics such as aggression or extroversion. Although genes contribute greatly to personality development, the way we turn out also depends on how we learn to behave. Personality can be seen as a bundle of habitual responses. These may be learned by copying behavior from caregivers or even from television.

MIMICKING BEHAVIOR

Many of the mind habits that make up personality are initially learned by mimicking the adults that care for us as infants.

If a response is repeated frequently, it is encoded as a memory. Thereafter, it is as much a “part” of the person as a genetic inclination.

PERSONALITY AND THE BRAIN

Many different personality traits have been linked to specific patterns of activity in the brain, some of which are linked to the expression of certain genes or particular genetic mutations. For example, a person who produces more excitatory neurotransmitters is less likely to feel the need to seek thrills than someone who needs a lot of stimulation to experience the same level of excitement.

PERSONALITY MARKERS IN THE BRAIN

Extroversion	Extroverts have reduced activity, in response to stimuli, in the neural circuit that keeps the brain aroused (shown here). As a result, they need more environmental stimuli to keep them feeling energized.	<p>Dorsolateral prefrontal cortex</p> <p>Anterior cingulate cortex</p> <p>Thalamus</p>
Aggression	People with a version of a gene previously linked to impulsive violence show abnormally reduced volume and unusually low activity in the cingulate cortex—an area concerned with monitoring and guiding behavior.	<p>Cingulate cortex</p>
Social behavior	Socially secure people have a stronger response to friendly looking people in the striatum—an area concerned with reward—than shy people. Avoidant types show a stronger reaction in the amygdala to unfriendly looking people.	<p>Striatum</p> <p>Amygdala</p>
Novelty seeking	People who like novelty may have better connections between areas shown here. The hippocampus sends signals to the striatum—which registers pleasure—when it identifies an experience as new.	<p>Striatum</p> <p>Hippocampus</p>
Cooperation	Cooperative people show increased activity in the insula if they think their treatment is unfair. Uncooperative people do not register unfairness to the same extent, suggesting an underdeveloped sense of trust.	<p>Insula</p>
Optimism	Optimism is linked to enhanced activation in the amygdala and in the anterior cingulate cortex when imagining positive future events relative to negative ones.	<p>Cingulate cortex</p> <p>Amygdala</p>



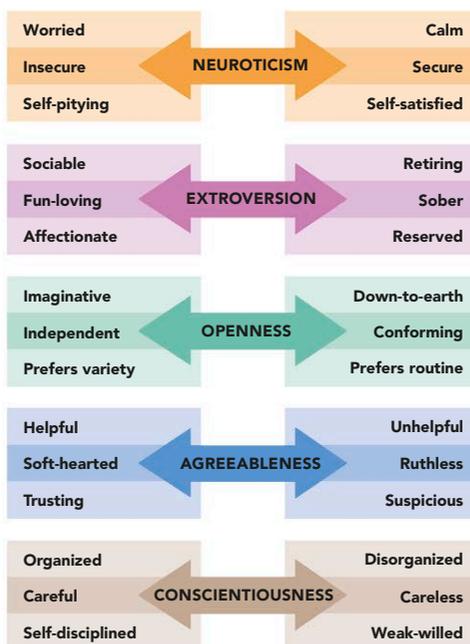
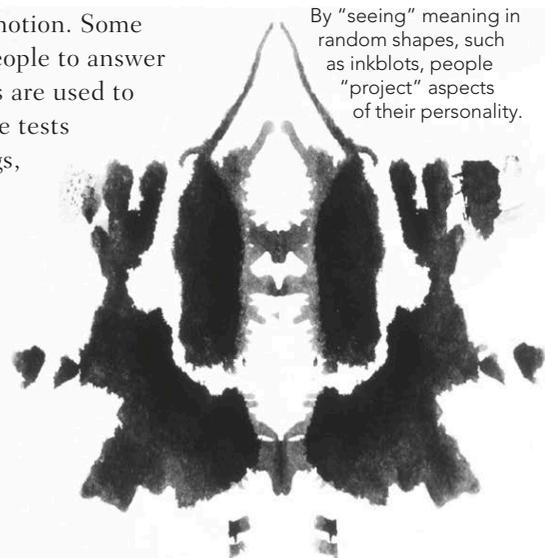


PERSONALITY ASSESSMENT

Personality testing is used for many reasons, such as for determining a person's suitability for a job or promotion. Some tests are standardized assessments that require people to answer questions about their typical behavior. The results are used to determine the individual's personality profile. Type tests place people in a particular category. Myers-Briggs, for example (below, right) sorts people into categories based on the predominance of certain attributes. Trait tests do not fit people into types, but draw up a profile based on where they lie along a number of dimensions. Projective tests, such as the Rorschach inkblot test, invite people to "reveal" aspects of their personality when responding to ambiguous stimuli.

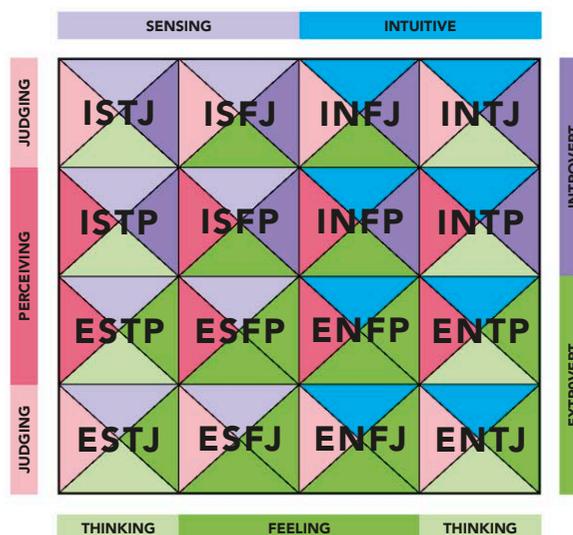
PROJECTIVE TESTS

By "seeing" meaning in random shapes, such as inkblots, people "project" aspects of their personality.



THE BIG FIVE

According to this trait test model, basic differences in personality can be "boiled down" to five dimensions. People may fall anywhere on each dimension.



MYERS-BRIGGS INDICATORS

The Myers-Briggs test asks a wide range of questions and places the person in one of 16 types. Despite criticisms of its lack of validity, it is the most widely used personality test used by businesses.

MANY PERSONALITIES?

Type tests like the Myers-Briggs (above) have been found to give different results according to the situation in which the person is tested. Trait tests allow for people to be different at different times, but still assume they have a "major" personality that is more real than others. Some evidence suggests, however, that practically everyone has more than one personality, and that many people have a large number of them. Memories that are available to a person in one situation may not be accessible in another. In extreme cases, this results in dissociative identity

DR. JEKYLL AND MR. HYDE

Dramatic personality changes, such as a "split personality," are a staple of horror films and ghost stories. They reflect a distrust of people who appear not to have stable personalities.

disorder (DID), but in normal people it merely shows up as mood changes, memory "glitches," and the coming and going of different skills, behaviors, and ways of seeing the world.

DISSOCIATIVE IDENTITY DISORDER

Extreme multiplicity, in which personalities are completely compartmentalized, results in people switching from one personality to another without retaining any memory of the previous state. They may behave differently according to which personality they are, and may even adopt a different name and history for each one. Because they have no memory of the others, each of them is likely to have memory gaps. Some people with DID find, for example, that they do things of which, in another personality, they disapprove.

BRAIN MONITORING AND STIMULATION

IT IS NOW POSSIBLE TO WATCH THE BRAIN'S ACTIVITY TAKING PLACE ON AN EXTERNAL DISPLAY AND DELIBERATELY CHANGE IT. THIS IS KNOWN AS NEUROFEEDBACK. MORE DIRECTLY, ACTIVITY CAN BE STIMULATED BY ELECTRICAL INPUT SENT THROUGH THE SKULL OR FROM ELECTRODES IMPLANTED INTO THE BRAIN ITSELF.

NEUROFEEDBACK

Brain activity is constantly altered by what an individual feels, thinks, or senses. The neurofeedback process works by turning the brain's activity itself into an external stimulus, which the person then responds to. For instance, EEG sensors may be used to pick up a person's brain waves. Different mind-states, such as relaxation or anxiety, have characteristic waveforms that are translated into a dynamic visual display. The activity registered by the EEG is then sent to a device that turns them into a form that the person can easily understand and manipulate. This may be as simple as a line that moves up or down, or a more complex game. The person tries to change the on-screen information by using the brain. The result of the effort is then displayed, so the person learns what to do in order to achieve the desired effect. Repeatedly doing this makes it increasingly easy to gain a desired state of mind, such as relaxation or focused attention.



MUSICAL MIND

Neurofeedback can help musicians attain a mind-state in which they play better. Students from London's Royal College of Music improved their performances by up to 15 percent after a course of treatment.

Step 1 EEG (or a similar brain "reading" device) charts the neural activity in the person's brain. The information is then transferred to a computer.

Step 2 The computer turns the neural patterns into a dynamic visual display, such as an interactive game with a clear goal, like making an on-screen object move.

Step 4 The player associates the "wins" in the game with certain brain states. The process then begins again, and through repetition the player learns to achieve them more easily.

Step 3 The person plays the game just by altering their brain-state. The machine registers neural changes, such as those marking relaxation, and "rewards" them with wins.



FEEDBACK LOOP

The neurofeedback process teaches people to change their brain-state. Once people have learned to do this with the equipment, they find it easier to do so at will.

MIND CONTROL

EEG is the usual brain "reading" process used for neurofeedback. Dozens of scalp-mounted electrodes pick up oscillations of underlying neurons and convert them into waves.

ECT

Electroconvulsive therapy (ECT) involves sending an electric current through the brain until the neurons are so stimulated that they produce a seizure (see p.226). It is used as a treatment of last resort for chronic depression, and it often works when drugs and psychotherapy have no effect. The way that it works is not fully understood, but it is thought that the seizure resets certain neurons' potential to fire, making them more or less sensitive. The seizure induced by ECT is short-lasting and harmless, and muscle relaxants are used to prevent convulsions. However, patients often complain of memory problems following the treatment.

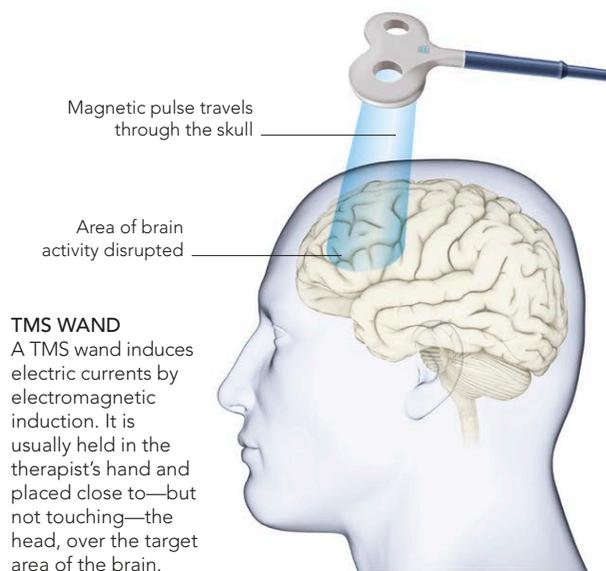
HISTORIC ECT

ECT was widely used in the 1950s in mental institutions. At that time, it was a crude technique, which involved creating a whole-brain seizure that caused the patient to thrash about.



TMS

Transcranial magnetic stimulation (TMS) sends a magnetic pulse through the skull and into the brain. The pulse temporarily disrupts normal activity in the part of the brain beneath it. Repeated stimulation of a particular area causes long-term changes in the way it functions. For example, it can increase activity in parts of the brain that are known to be underactive in people with depression or decrease it in areas known to be overactive in those with obsessive compulsive disorder. Repeated sessions of TMS are increasingly used as treatment for these and other conditions.



DEEP BRAIN STIMULATION

In deep brain stimulation, tiny electrodes are placed surgically in the brain. They radiate current to nearby target areas, activating otherwise sluggish neurons, which in turn create local changes in brain chemistry. The electrodes are attached to



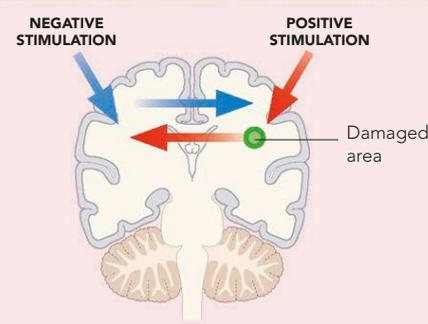
very fine wires, which are inserted deep into the brain through small holes in the skull. They are situated in different brain areas according to the condition being treated and may be sited quite differently in each patient.

BRAIN SURGERY
Patients are operated on while conscious so that they are able to communicate. Their reactions guide the surgical team to implant the electrodes in the right spot.

In some cases, the wires are connected to an external switch that allows the patient to turn the current on and off as required.

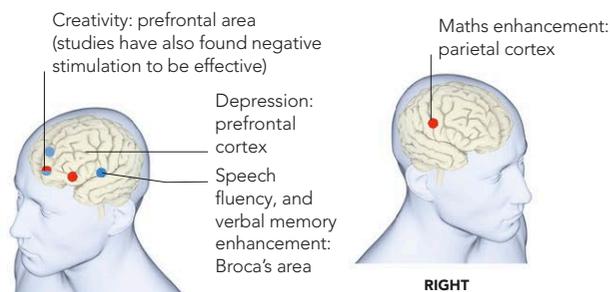
TREATING STROKE

Neurostimulation may be used to help people recover from the effects of stroke. The damaged area of the brain is stimulated in order to help neighboring neurons grow and take over the work of the cells that have been killed off. Conversely, inhibitory stimulation may be applied to the brain cells corresponding to the damaged area on the opposite side of the brain. This prevents the opposite side of the brain from compensating for the damaged area and interfering with its recovery.



TDCS

Transcranial direct current stimulation (tDCS) is a way of stimulating (or inhibiting) selected neurons by sending a minute charge of electricity through the cortex via scalp-mounted electrodes. The current used is less than 2 milliAmps—so small that most people can barely feel it. Thousands of studies show that it is safe and that it may reduce symptoms of mood disorders, chronic pain, tinnitus, motor and speech disorders (especially after stroke) and possibly schizophrenia and dementia. It also enhances brain function in healthy people; for example, it can be used to improve math skills and creativity, and lead to swifter learning.



KEY

● **ANODE STIMULATION**

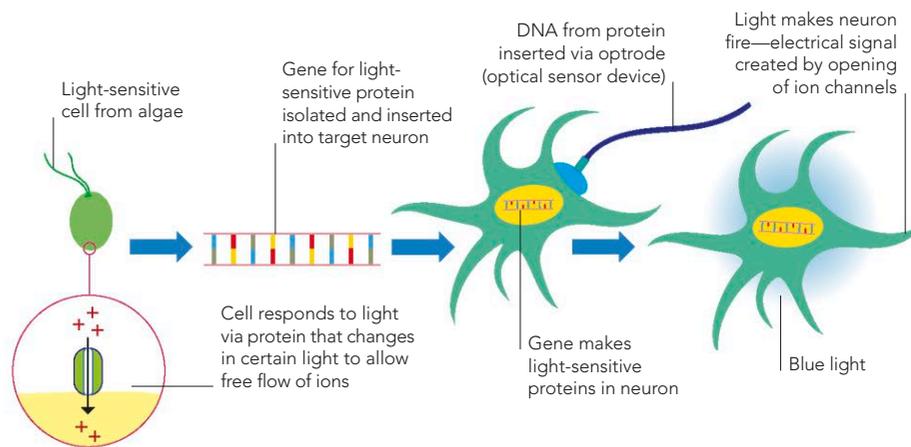
● **CATHODE INHIBITION**

BRAIN AREAS

Stimulating (or inhibiting) different brain areas with tDCS has different effects. These are some of the areas where anode stimulation (red) or cathode inhibition (blue) has been shown to alter experience or enhance a particular skill.

OPTOGENETICS

Optogenetics allows specific neural pathways in the brain to be turned on and off by light. At the moment, it is used only in research animals to map brain circuits, but eventually it is expected to have a number of medical uses. The first application is likely to be to repair retinal cells in the eye that have ceased to be sensitive to light. The technique involves taking light-sensitive molecules from algae then inserting them into specific brain cells. A fiber-optic light is then inserted into the brain, and, when the light is switched on, the cells containing the inserted molecules become active. Depending on where the neurons are, the stimulation can alter behavior and create new memories and habits.



INSERTING MOLECULES

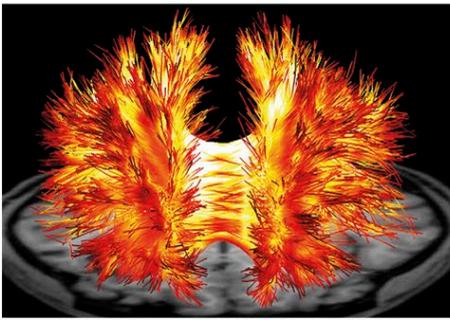
There are various methods of inserting light-sensitive molecules into brain cells. The most common involves using a virus that targets particular neurons as a carrier.

STRANGE BRAINS

ON THE WHOLE, ONE BRAIN LOOKS VERY MUCH LIKE ANOTHER, GIVE OR TAKE A SMALL VARIATION IN SIZE. SOME BRAINS, HOWEVER, ARE DRAMATICALLY DIFFERENT FROM NORMAL, AND IN MANY CASES PHYSICAL ECCENTRICITY PRODUCES UNUSUAL WAYS OF BEHAVING AND SEEING THE WORLD.

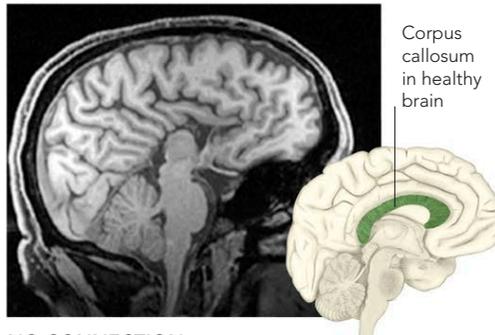
THE SPLIT BRAIN

The corpus callosum carries signals between the two hemispheres. Rarely, this tissue is surgically severed in people with epilepsy, in order to prevent the spread of seizures. Researchers projected images separately to each hemisphere (see split-brain experiment, below) of split-brain patients. Normally the two sides would share the information via the corpus callosum, but without it each side recognized only its own image. The patients could identify the picture known by the language-dominant left brain, but denied seeing anything else. Yet they were able to select the object seen by the right brain, using the left hand (which is controlled by the right hemisphere). Asked why they selected that object, however, they were unable to say. This suggests that the right hemisphere (in right-handers) is unconscious—even though the information it holds affects behavior.



CONNECTING THE HEMISPHERES

This diffusion tensor image clearly shows the wide band of fibers that forms the corpus callosum, which connects the left and right hemispheres of the brain.



Corpus callosum in healthy brain

NO CONNECTION

Occasionally the corpus callosum fails to develop, in a condition known as agenesis of the corpus callosum (shown here in an MRI scan). This leaves the two hemispheres of the brain unconnected.

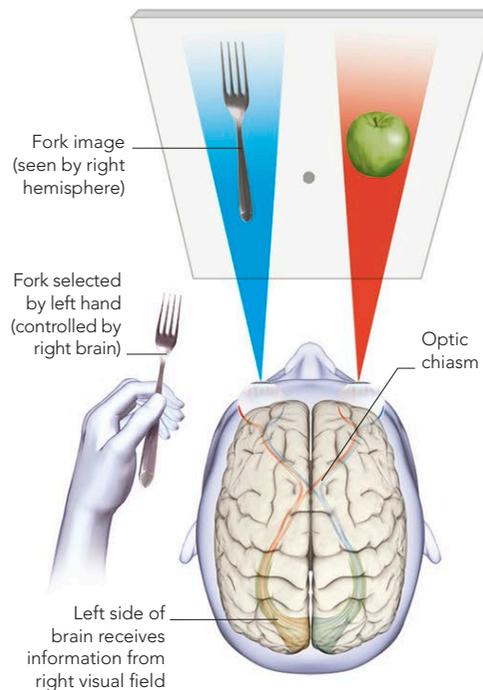
TESTING YOUR CORPUS CALLOSUM

Close your eyes and spread out your hands, palms facing upward. Get someone to touch one of your fingertips, and with your opposite hand try to touch the corresponding finger with thumb of the same hand (see below). If information is flowing properly between the hemispheres, you should be able to do this without opening your eyes.



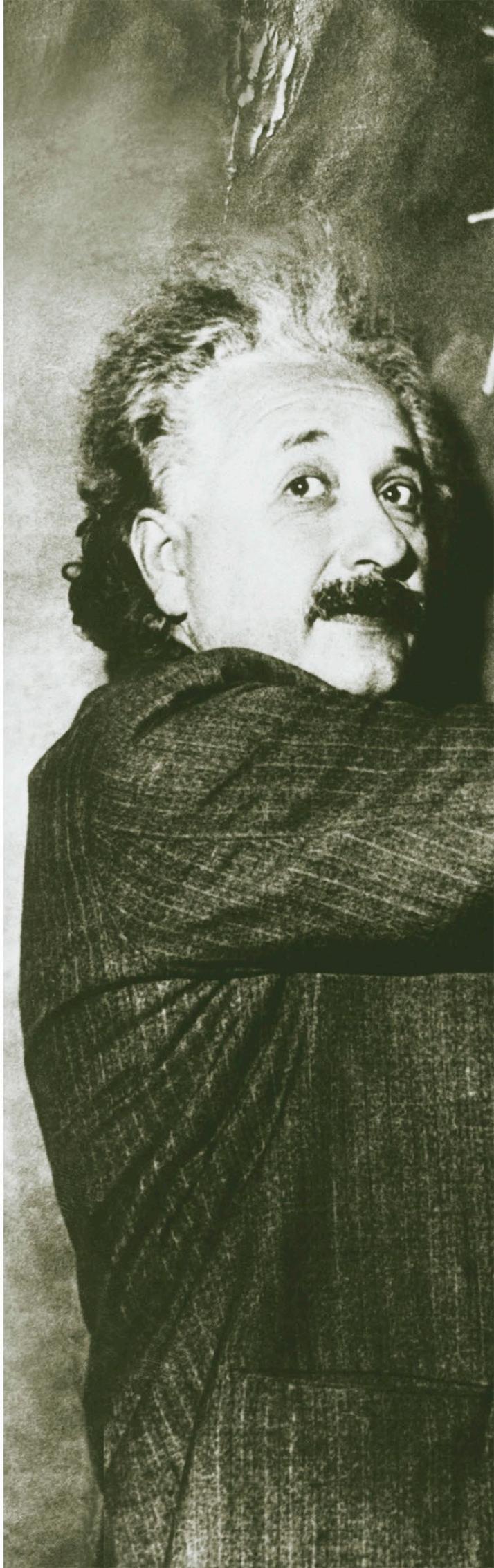
Touch thumb to finger on left hand

Sensation experienced on right hand



SPLIT-BRAIN EXPERIMENT

In a split-brain experiment, the image shown to the right side of the brain can guide the actions of the left hand to select an object, even though the person is not conscious of seeing the image and is only aware of seeing the apple.

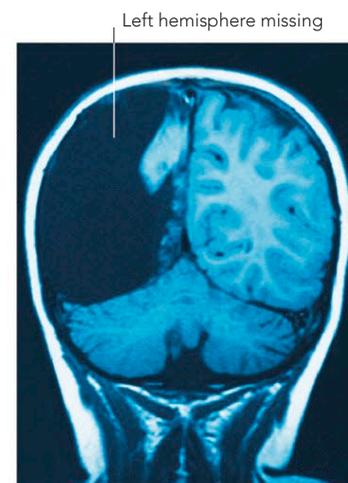




WEIRD BRAINS

Brain scans have revealed some astonishing physical abnormalities, such as brains that are missing an entire hemisphere. The effect of losing half a brain would be catastrophic if it happened in later life. However, several cases have come to light in which brain growth has been severely restricted in infancy and yet the person has gone on to live a near normal life with few, if any, adverse symptoms.

HALF A BRAIN
Despite having one side of her brain removed, this girl learned to be fluent in two languages.



SIZE DOESN'T MATTER

Brains do not, generally, vary greatly in size, and there is little evidence to suggest that bigger brains produce greater intelligence. At one extreme, Irish writer Jonathan Swift (1667–1754) had a brain that, at the time of his death, weighed a relatively enormous 70oz (2,000g). In 1928, the Moscow Brain Research Institute started collecting and mapping the brains of famous Russians, including that of the physiologist Ivan Pavlov (1849–1936). His brain was at the other end of the size scale, weighing a mere 53½oz (1,517g).



JONATHAN SWIFT

IVAN PAVLOV

VARYING SIZES

The brains of famous intellectuals vary greatly in size, so the connection between IQ and size is unclear.



THE TERRORIST'S BRAIN

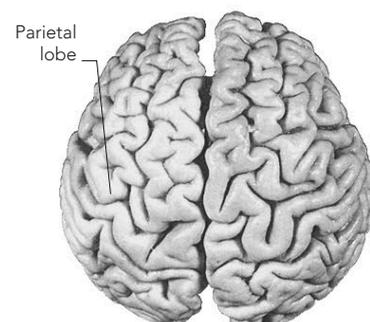
Ulrike Meinhof (1934–76) was a member of the infamous Baader–Meinhof Gang, responsible for a number of killings, bombings, and kidnappings in Germany during the 1970s. She was captured and committed suicide in prison. After her death, studies suggested that brain damage resulting from an operation on a swollen blood vessel might have accounted for her violent behavior.

FACE OF A KILLER

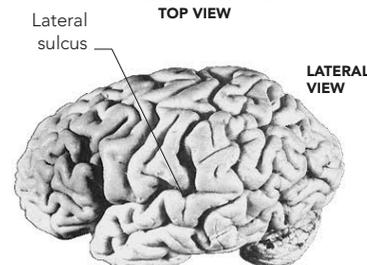
This rare image of Meinhof was taken when she was arrested in 1972. In 1962, she had a metal clip inserted in her brain during surgery, which helped police identify her.

EINSTEIN'S BRAIN

Albert Einstein's brain was removed after his death. Many years later, it was examined by Dr. Sandra Witelson and compared with other brains in a brain bank. It was found to be wider than normal, and part of a deep groove that normally runs through the parietal lobe was missing. The area affected is concerned with mathematics and spatial reasoning, and it is possible that the missing groove allowed neurons in that area to communicate more easily, giving him his extraordinary talent for describing the universe mathematically.



TOP VIEW



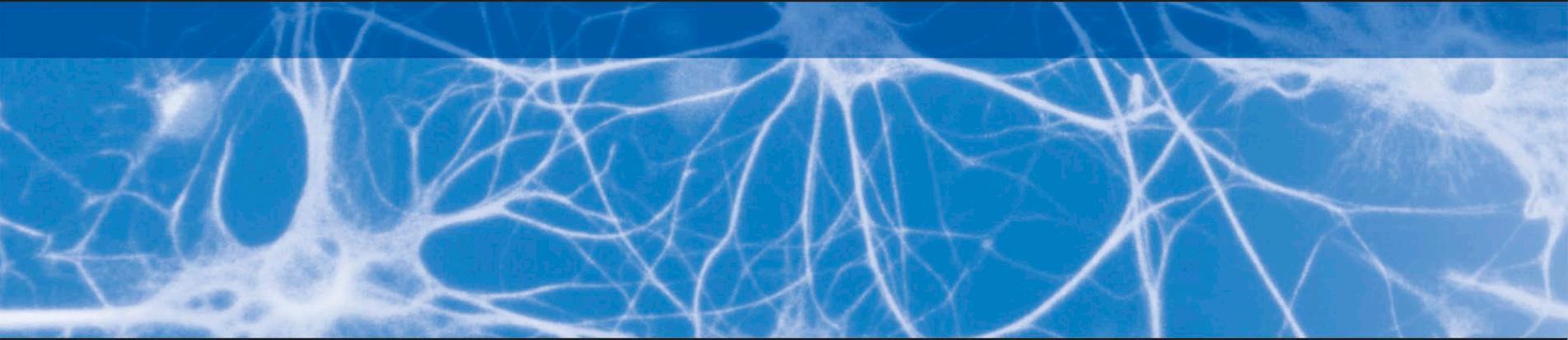
LATERAL VIEW

A MATHEMATICAL BRAIN?

Einstein's brain was wider than normal (top) and the part of the lateral sulcus normally found in the parietal cortex was apparently missing.

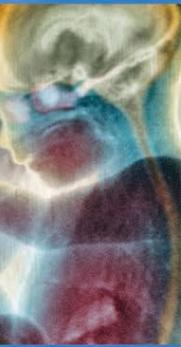
GREAT MIND AT WORK

Physicist Albert Einstein (1879–1955) claimed to “see” his mathematical theories as a whole rather than “working them out bit by bit.” The odd structure of his brain may explain how he was able to do this.



OUR BRAIN CHANGES OVER THE COURSE OF OUR LIFE, AND THIS HAS FAR-REACHING EFFECTS ON WHAT WE CAN DO AND HOW WE BEHAVE. DEVELOPMENT STARTS A FEW WEEKS AFTER CONCEPTION, AND TO BEGIN WITH IS INCREDIBLY RAPID, WITH HUNDREDS OF THOUSANDS OF NEURONS BEING ADDED EVERY MINUTE. THE PACE GRADUALLY SLOWS, AND WE ARE WELL INTO OUR 20S BEFORE OUR BRAINS ARE FULLY DEVELOPED. AS WE AGE FURTHER, NATURAL AND IRREVERSIBLE DEGENERATION SETS IN, BUT THE BRAIN HAS VARIOUS MECHANISMS TO COMPENSATE FOR THIS.

DEVELOPMENT AND AGING

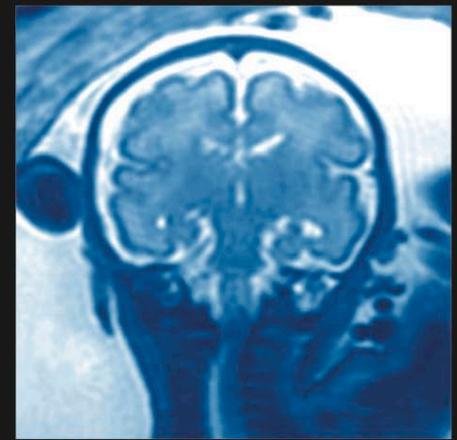


THE INFANT BRAIN

THE HUMAN BRAIN FORMS FROM THE OUTERMOST LAYER OF TISSUE IN A DEVELOPING EMBRYO, AND IT UNDERGOES SEVERAL TRANSFORMATIONS BEFORE EMERGING AS THE RECOGNIZABLE ORGAN. AFTER A PERIOD OF RAPID CELL GROWTH, NEWLY GENERATED NEURONS MOVE AROUND TO FORM THE VARIOUS PARTS OF THE BRAIN. IT TAKES MORE THAN 20 YEARS FOR THE BRAIN TO BECOME FULLY MATURE.

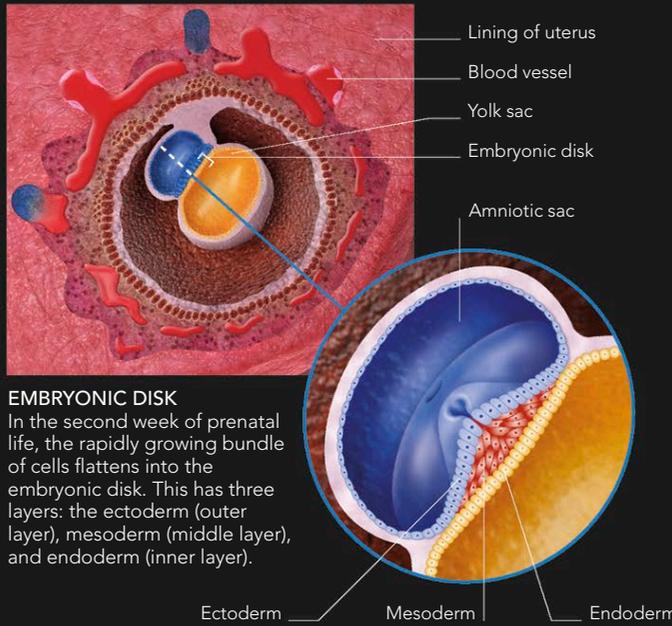
CONCEPTION TO BIRTH

In the days after conception, the embryo is just a minute ball of cells. Development of the brain and nervous system starts at about three weeks as the cells differentiate into layers, the outermost of which thickens and flattens to form a feature called the neural plate (see below) along the back of the embryo. This broadens and folds to form the liquid-filled neural tube, which will become the brain and spinal cord. The brain starts to develop at about four weeks as a bulb at the upper end of the neural tube, while the lower part begins to form the spinal cord. The main sections of the brain, including the cerebral cortex, are visible within seven weeks. Over the next weeks, the brain grows, develops, and becomes more complex.



DEVELOPMENT OF THE CORTEX

The cerebral cortex develops from the forebrain, one of three vesicles formed from the neural tube. The frontal lobes form first, followed by the parietal then temporal and occipital lobes.

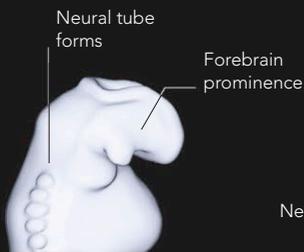


EMBRYONIC DISK

In the second week of prenatal life, the rapidly growing bundle of cells flattens into the embryonic disk. This has three layers: the ectoderm (outer layer), mesoderm (middle layer), and endoderm (inner layer).

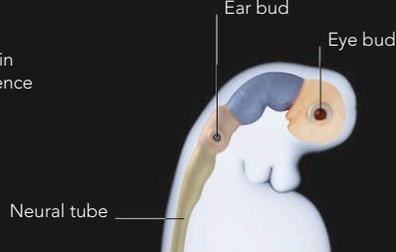
KEY FOR EMBRYO DEVELOPMENT

- FOREBRAIN (PROSENCEPHALON)
- MIDBRAIN (MESENCEPHALON)
- HINDBRAIN (RHOMBENCEPHALON)
- SPINAL CORD



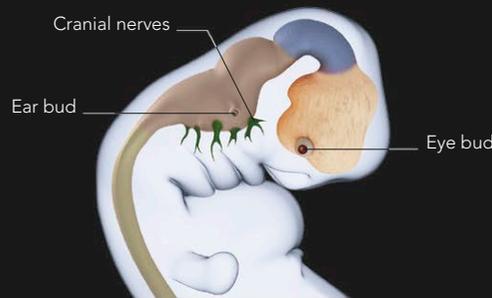
3 WEEKS

Within three weeks of conception, the neural tube is well developed along the back of the embryo, and the prominence that will develop into the forebrain is clearly defined.



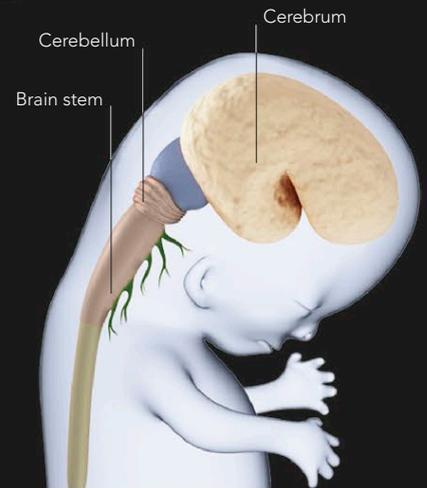
5 WEEKS

The future forebrain, midbrain, and hindbrain can be seen clearly by five weeks, and rudimentary eye and ear buds emerge. The optic nerve, retina, and iris start to form.



7 WEEKS

The embryo is around 3/4in (2cm) long, and the bulges that will become the brain stem, cerebellum, and cerebrum are now clearly visible. The cranial and sensory nerves also start to develop.

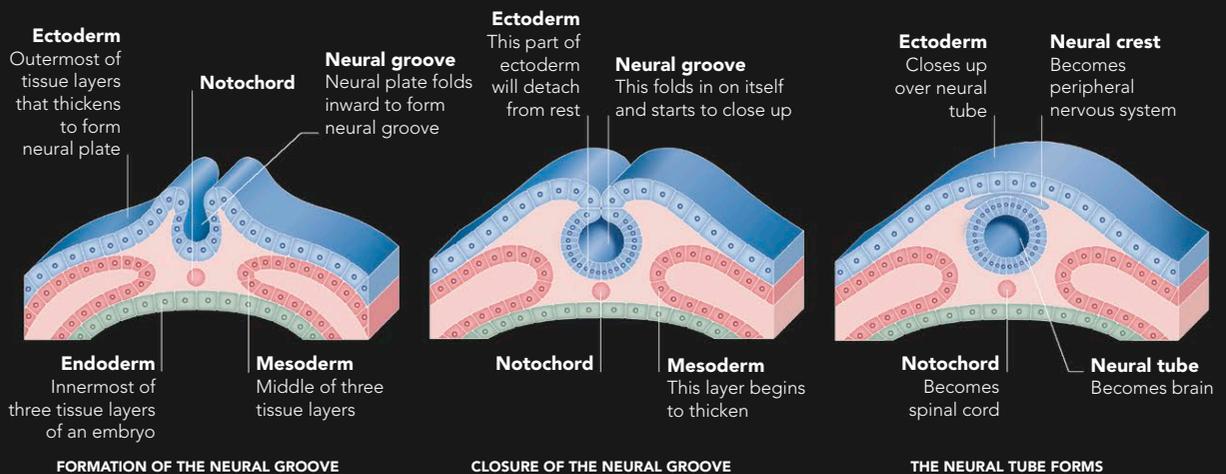


11 WEEKS

The cerebrum enlarges, and the eyes and ears mature, moving into position. The fetus's head is still large relative to the body. The hindbrain divides into the cerebellum and the brain stem.

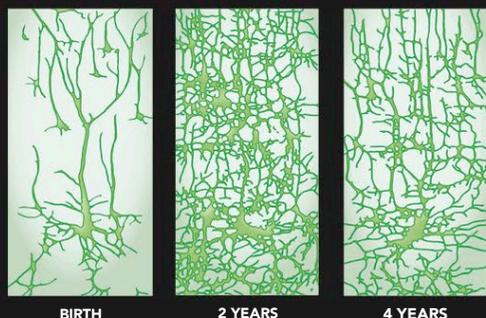
FORMATION OF THE NEURAL TUBE

The key event in the development of the nervous system is the formation of the neural tube. This process is known as neurulation and begins when the primitive spinal cord (notochord) sends a signal to the tissue above it to thicken, forming the neural plate. The neural plate turns inward and forms a depression, known as the neural groove. Folds within the groove fuse together and then close in on themselves to form the neural tube. Some neural-fold tissue is pinched off to form the neural crest, which will become the peripheral nervous system.



NERVE GROWTH AND PRUNING

Only one-sixth of the brain develops before birth, and the growth rate in the first three years of life is phenomenal. Most of the growth, however, is connective tissue, as pathways are forged between neurons. By the age of three, this dense network of fibers requires “pruning” back, a process known as apoptosis. The pruning allows the preserved connections to work more efficiently. It is similar to the way in which “noise” can be extracted from a radio signal to leave only the intended content without the interference.

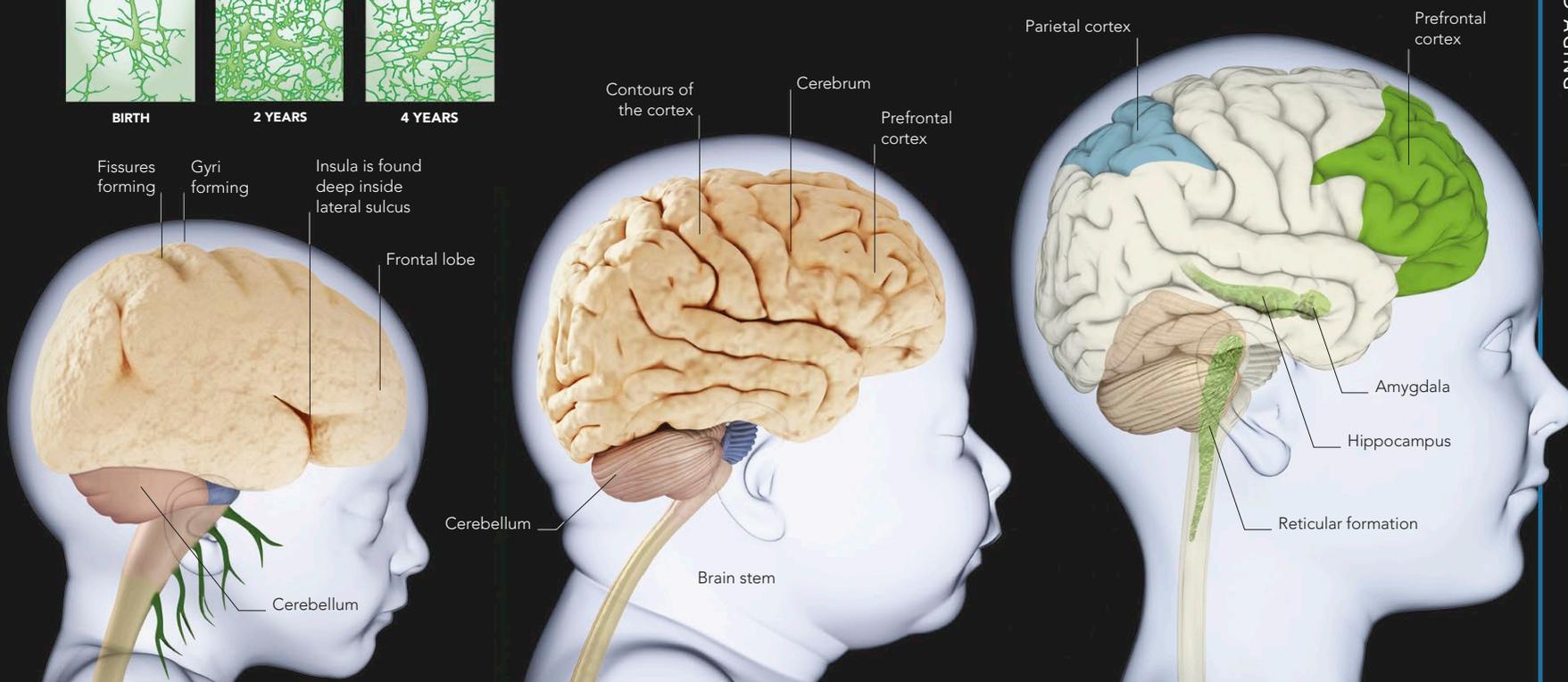


NEURAL NETWORKS

A dense network of connecting fibers forms between the brain's neurons during the first few years of life. By the age of four, these connections have been pruned back.

LANGUAGE DEVELOPMENT

Speech, and some other higher faculties, are wired into the human brain, but appropriate stimulation is needed to help it develop normally. Babies start babbling at about six months, with simple vowel-and-consonant combinations, is formed. “Motherese” is a universal adult reaction to babbling. It involves uttering repetitive sing-song noises, such as “goo-goo” and simple words. It aids speech development in the child and promotes bonding.



25 WEEKS

The hemispheres are now clearly dissected, and some of the deeper grooves that form the bulges and valleys—gyri and sulci—are becoming visible. The cerebellum is tucked under the cerebrum.

BIRTH

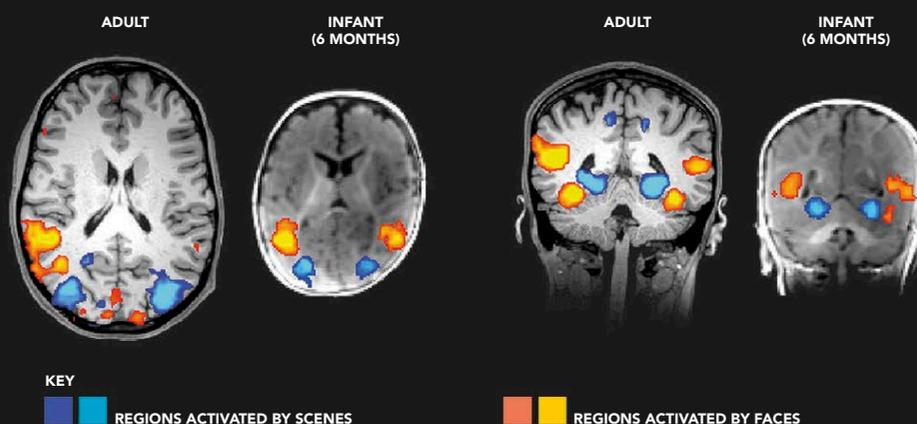
The cerebrum develops, and the ridges (gyri) and fissures (sulci) increase in complexity. At birth, a baby has as many neurons as an adult: 100 billion. Most are formed in the first six months of gestation but are not yet mature.

3 YEARS

Parts of the brain, like the prefrontal cortex, develop, but large areas are offline as the connections between areas are yet to form or are yet to be coated in myelin, so signals can't travel along them reliably. This limits the ability of the frontal brain to think and judge. Growth of the amygdala and hippocampus allows memories to be retained.

PLACES AND FACES

The basic functional blueprint of the brain is in place even at birth. The back of the brain is already wired to receive information from the eyes, for example, which it will start to turn into visual images, and the limbic areas, which register “good” and “bad” events, are already working. Even quite detailed areas are already determined.



PRIMED TO SEE
Brain scans of 6-month-old infants show they already process faces in a different area from other images, just like an adult.

CHILDHOOD AND ADOLESCENCE

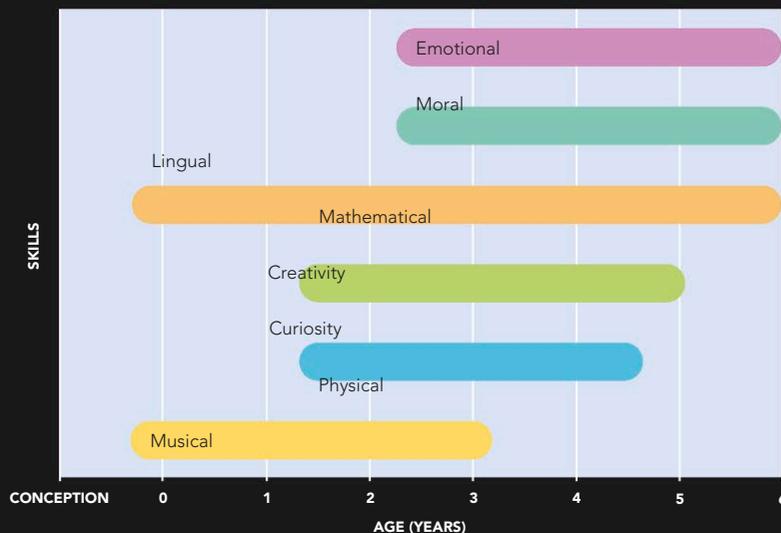
THE BRAIN DEVELOPS BY CREATING MORE AND MORE NEURAL PATHWAYS, WHICH CONNECT THE VARIOUS FUNCTIONAL AREAS. THE EARLIEST PARTS TO BECOME FULLY INTEGRATED ARE CONCERNED WITH PERCEPTION, CLOSELY FOLLOWED BY MOTOR AREAS.

THE BRAIN IN CHILDHOOD

The brain matures throughout childhood and young adulthood—the process is not complete until a person is in their late 20s. During that time, different areas of the brain connect, producing increasingly complex and controlled behavior. Connection occurs as the neurons grow axons—threads that reach out to other neurons—and the axons become covered in fatty sheaths (myelin), which allow electrical signals to move faster and more reliably along them.



MOTOR SKILLS
Physical dexterity develops fairly early as perception and motor areas of the brain become connected.

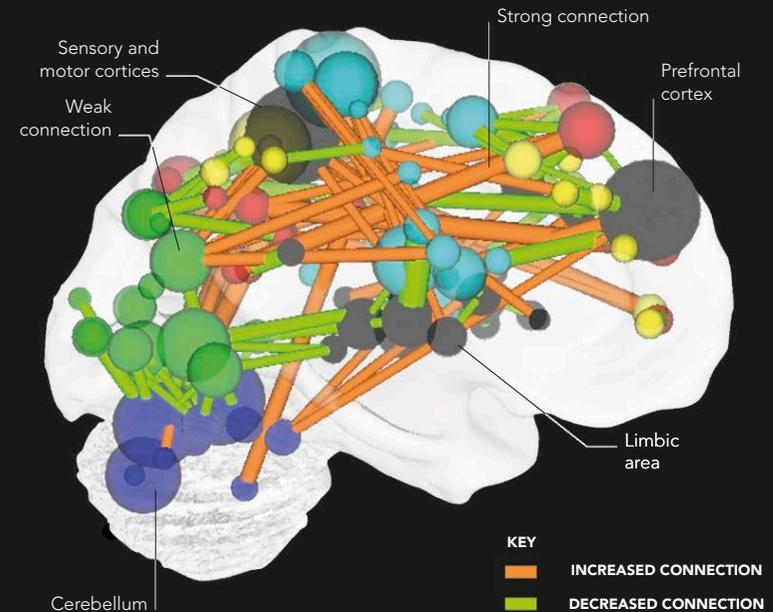


WINDOWS OF LEARNING

Human skills and faculties develop as the associated parts of the brain mature. The timetable is under genetic control, and no amount of teaching can instill in a child an ability that the brain is not ready to acquire. Until they are about three, for instance, infants cannot make moral judgments because their prefrontal cortex, where such decisions are made, is not fully “online.” When the area is maturing, however, a child will learn the skill associated with it easily and rapidly, given the right stimuli. If a window of learning is missed, the child will have difficulty acquiring the skill later.

CHANGING CONNECTIONS

Scientists have devised a typical growth chart of the human brain, formed by taking more than 200 fMRI scans of individuals with an age range of 7–10 years. They found that fibers connecting peripheral brain areas decreased, while increases occurred in those connecting the limbic areas with the frontal cortex as the brain matured.

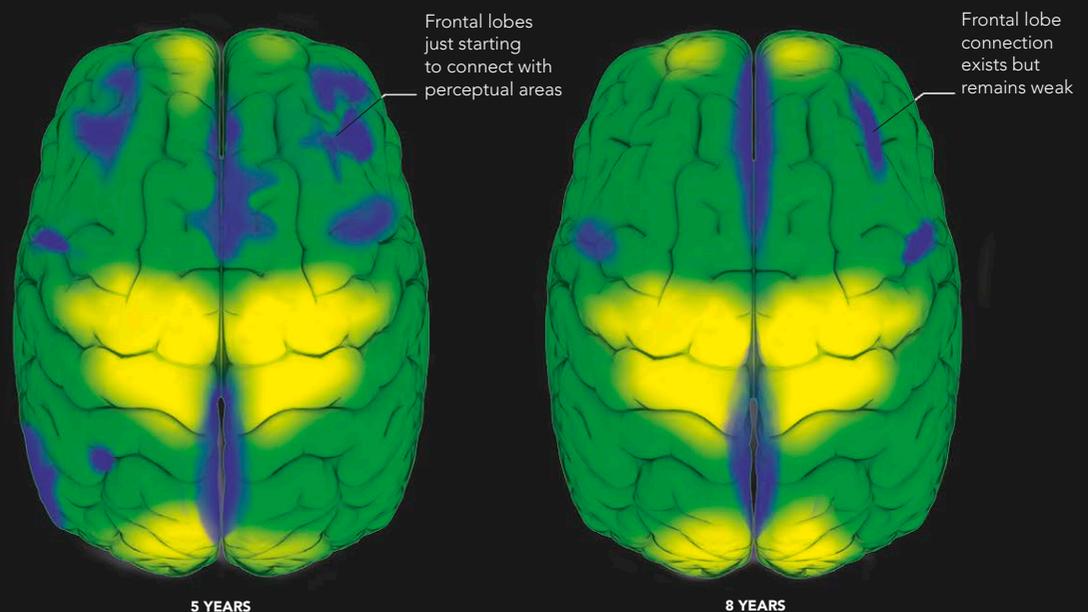


JOINING UP

In order to think and behave as an adult, a person’s brain needs to be “joined up.” This allows perceptions to be fully understood and actions to be considered. Connection depends on a process called myelination, during which neuronal pathways between areas are coated with fat to allow the transmission of electrical signals.

MYELINATION

These scans show, on average, the degree of connection that exists at various ages. Yellow shows full myelination, green is partial, and blue is none.



THE TEENAGE BRAIN

Between puberty and early adulthood, the human brain undergoes a dramatic restructuring. This process is often reflected in impulsive and rebellious behavior, and sudden personality changes. While all these changes take place, the teenage brain is particularly vulnerable. Personality traits such as risk-taking or pessimism may be amplified to the extent that they cause dysfunction, such as heavy drug-taking, reckless or criminal behavior, intense anxiety, or depression. In many cases, the issue passes as the brain becomes more mature, but sometimes it signals the start of a serious, long-term mental health problem.

BRAIN CHANGES

Teenage brain changes, in both sexes, are driven by testosterone release. The hormone makes neural pathways exceptionally plastic for a while so connections make and break easily. This allows teenagers to learn new things quickly and to adopt new habits and personality traits, which in turn will be changed again if they are not advantageous. The instability of the teenage brain results in baffling changeability and a tendency towards risk-taking and rebellious behavior. The prefrontal cortex is still developing, which is thought to be one reason for impulsiveness and rash decision-making. It is closely connected to the basal ganglia, which play an important role in motor skills. The fiber tract that links the two hemispheres—the corpus callosum—thickens, allowing for increased information-processing skills.

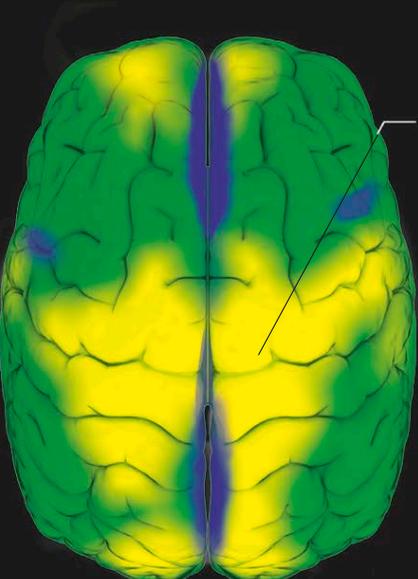
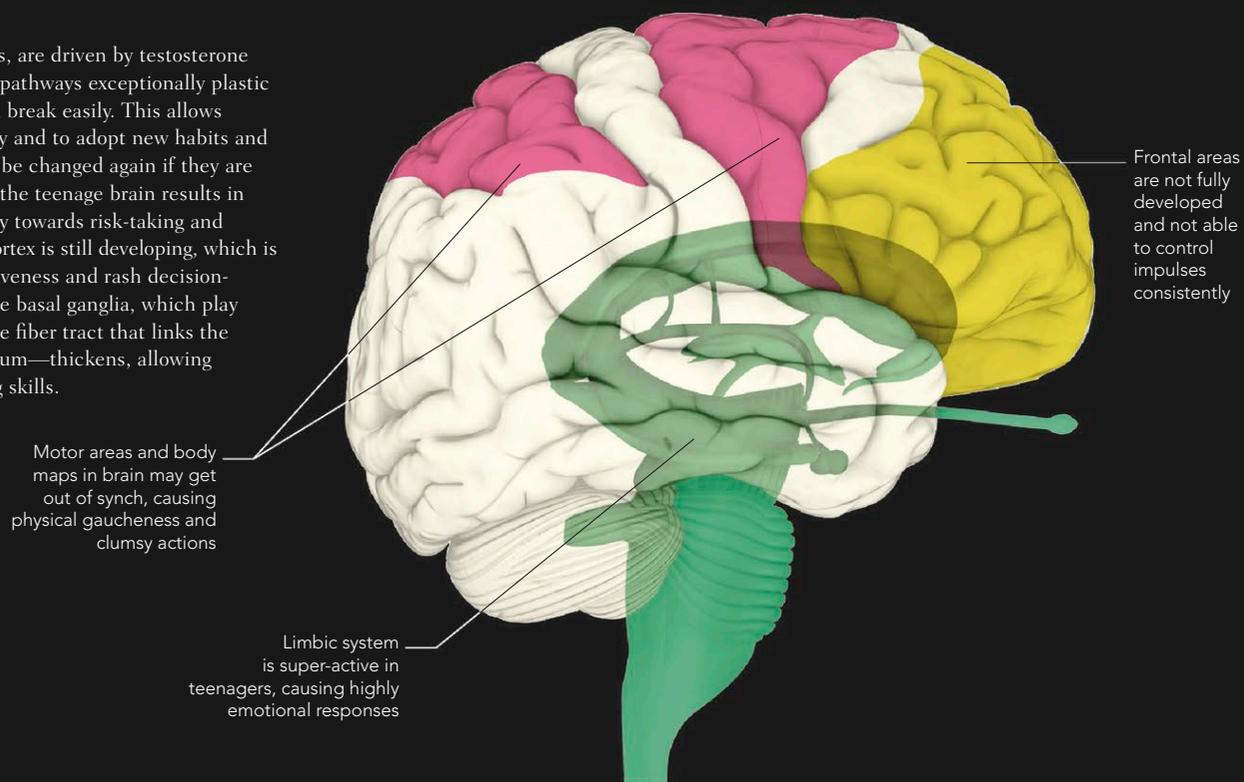
WORK IN PROGRESS

Many different areas of the brain undergo changes, each causing a particular, temporary characteristic of teenagers.



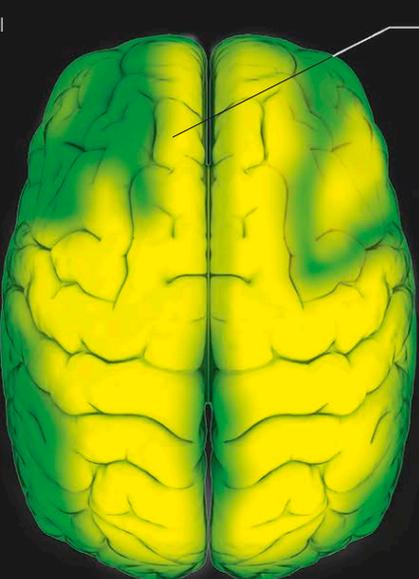
MENTAL HEALTH RISKS

The dramatic brain changes that occur during adolescence make teenagers particularly susceptible to mental ill-health. One in five adolescents has a mental illness that will persist into adulthood.



Connection is well forged in back of brain but remains weak in frontal areas

12 YEARS



Connectivity is established, but teen brain is undergoing seismic changes, which make links unreliable

18 YEARS



Whole brain is now connected, but new links continue to be forged for another 10 years or so

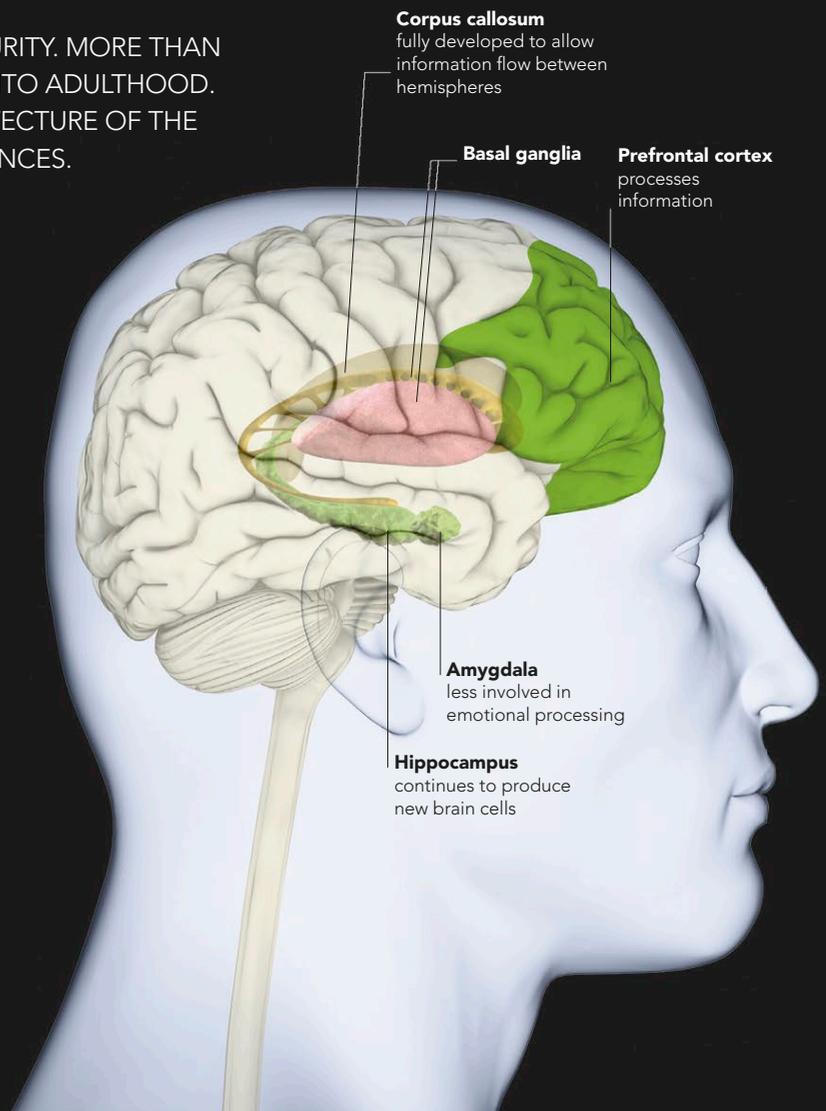
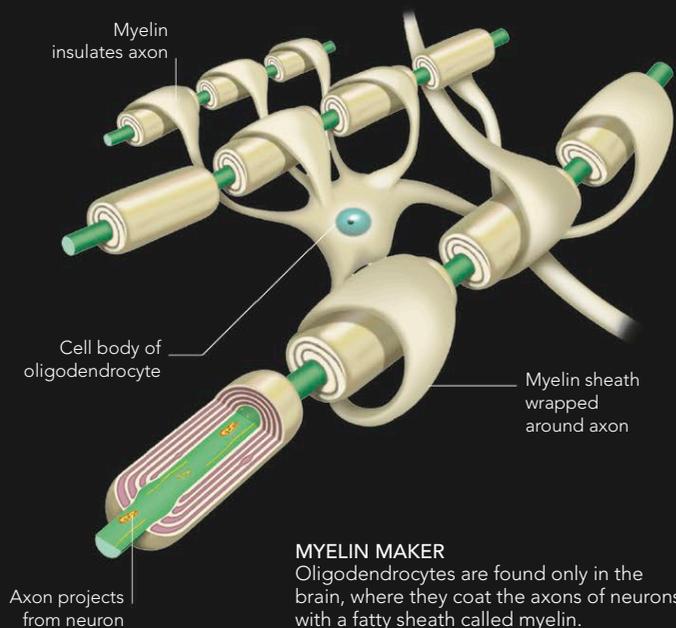
20 YEARS

THE ADULT BRAIN

THE BRAIN DOES NOT STOP GROWING WHEN IT REACHES MATURITY. MORE THAN ANY OTHER ORGAN, IT CONTINUES TO REFORM ITSELF LONG INTO ADULTHOOD. NEW BRAIN CELLS CONTINUE TO BE CREATED, AND THE ARCHITECTURE OF THE BRAIN IS CHANGED CONSTANTLY IN RESPONSE TO LIFE EXPERIENCES.

REACHING MATURITY

Human brains are slow to reach full maturity. The prefrontal cortex is the last part to become fully active, and full myelination, the sheathing of neuronal connections that allows information to flow freely along them, does not occur until a person is in their late 20s or early 30s. Once the prefrontal cortex is fully online, it becomes more active in situations that have emotional content. Whereas a teenager or child might be overwhelmed by emotion, the prefrontal cortex inhibits emotion when necessary, allowing a more thoughtful, deliberated response.

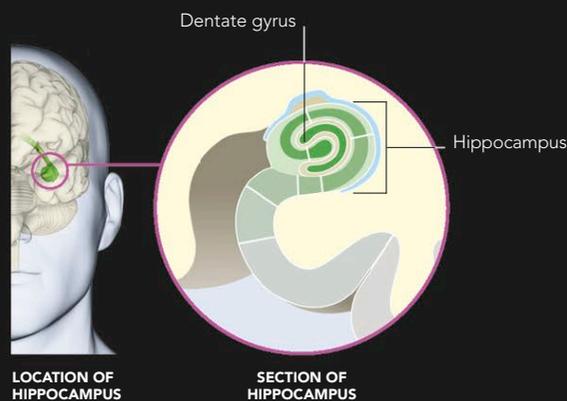


AT 30 YEARS OLD

The prefrontal cortex is now fully developed, allowing for improved executive functions. This also means that the brain is less reliant on the amygdala to process emotional information. The other areas of the brain that were still developing in adolescence have now reached maturity.

NEUROGENESIS

It used to be thought that the number of brain cells in the adult brain was fixed early in life and that laying down new memories and learning new things was achieved entirely by changes to existing neurons and their connections with one another. While this sort of rewiring is important for learning, it is now known that adults also benefit from the creation of new brain cells. Neurogenesis occurs mainly in the dentate gyrus of the hippocampus, the brain region that is centrally important for learning and memory. About one-third of the neurons in the adult hippocampus are replaced in a person's lifetime.



MEMORY MAKER

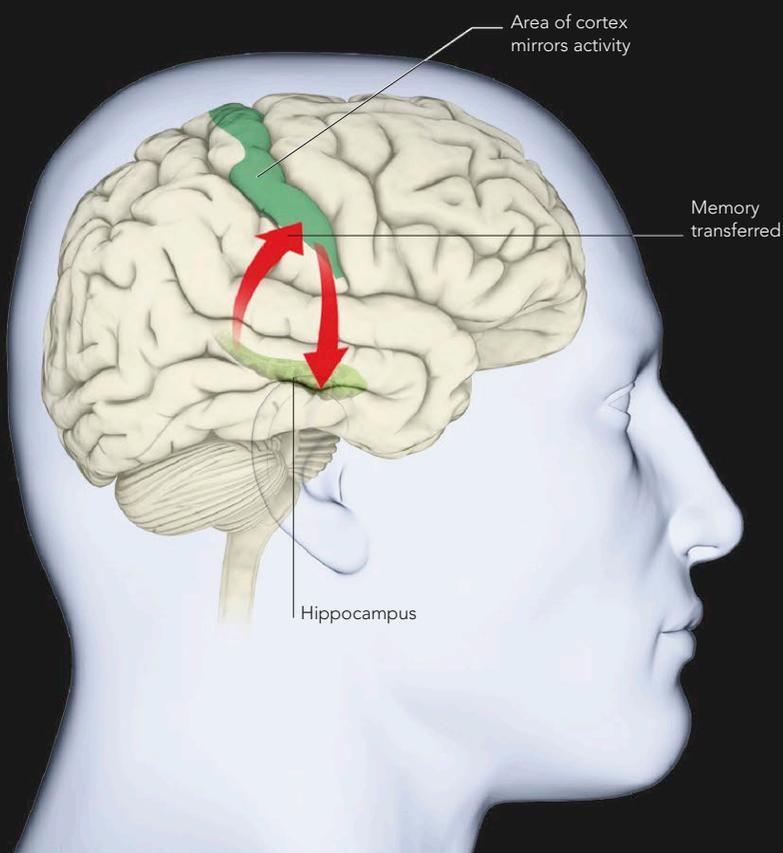
The hippocampus is a vital part of the brain, which is essential for laying down and recalling memories. Neurogenesis, which occurs in the dentate gyrus (see opposite), helps it to encode new information. Neurogenesis is measured in animals by injecting their brains with a radioactive marker that attaches to dividing cells. Counting the marked cells when the animals die shows how many cells have multiplied.

HIGHER FUNCTION

A person's brain continues to mature right up until their late 20s. The main changes take place inside the "higher" functional areas of the brain, such as the frontal cortex, which gradually becomes more active—pulling together information from the rest of the brain and forming a complex and holistic view of the world. Until then, the emotional parts of the brain are not fully connected with those areas concerned with thought, judgment, and behavioral inhibition. As the connections between the areas become more stable, people tend to react less emotionally and impulsively—instead becoming more cautious and considered, and exercising better judgment.

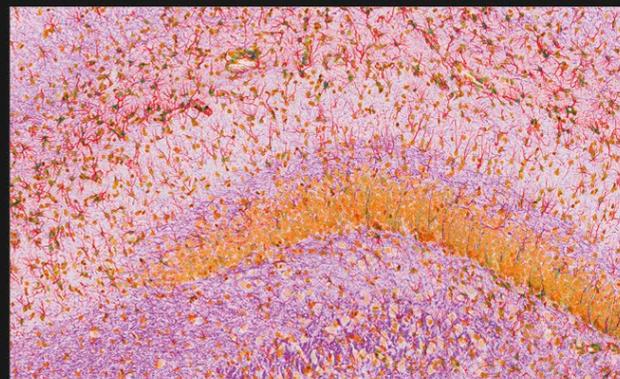
NEW MEMORIES FOR OLD

The creation of new brain cells allows new information to be stored, but their arrival disrupts existing memories because they change the wiring pattern. Most memories form in the hippocampus and are transferred to long-term storage in other brain areas. For a while, the memory resides both in the hippocampus and elsewhere. After a few years, the memory is cleared from the hippocampus. Until the memory is fully transferred, the arrival of new cells in the hippocampus may weaken the connections encoding memories stored there. This may be why we rarely retain memories from when we were very young.



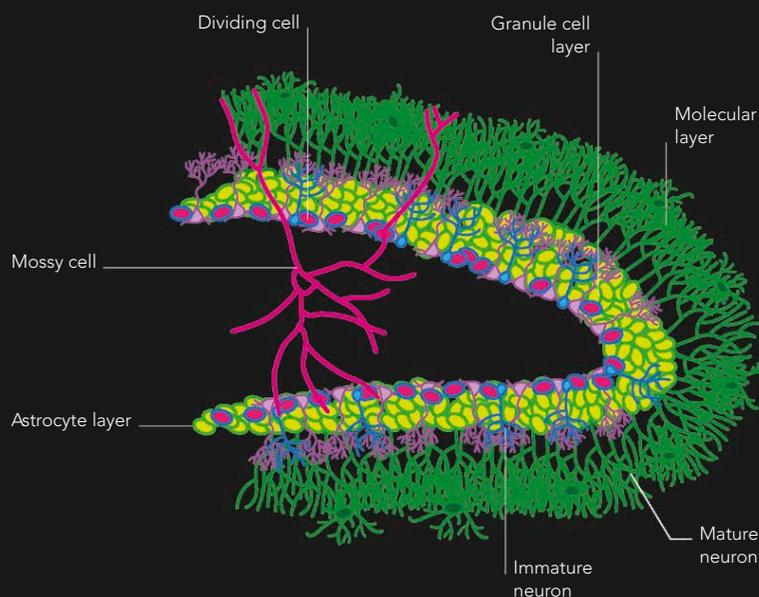
MEMORY TRANSFER

Memories are first formed as patterns of neural activity in the hippocampus, which are then echoed in areas of the cortex (see pp.160–161).



SITE FOR NEW CELLS

This light micrograph shows a section through the hippocampus, which has been magnified and stained to show nerve cells in the dentate gyrus, where new neurons are made.



DENTATE GYRUS CELLS

In adults, new neurons are made in just two areas of the brain—the olfactory cortex (the part of the frontal cortex that registers smells) and, more commonly, in part of the hippocampus called the dentate gyrus. Astrocyte cells in this area produce a protein that triggers the process. Cells divide and mature, moving up through the granular to the molecular layer of the dentate gyrus.

PARENTHOOD

Having a child is a major event in most adult lives and usually brings about profound changes in behavior. These are accompanied by changes in the brains of both mothers and fathers. In both parents, raised levels of hormones, particularly prolactin and oxytocin, sensitize areas of the brain concerned with alarm (such as the amygdala) and action, making them more sensitive to their babies' cues, such as cries and expressions. Men's testosterone level falls and prolactin level rises, making their brains temporarily more like that of a female.

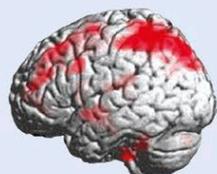


BRAIN CHANGE

Research shows that becoming a parent produces a flurry of neurogenesis. MRI studies reveal an increase in cortical thickness in new mothers' brains, shown red in the scans above.



RIGHT HEMISPHERE

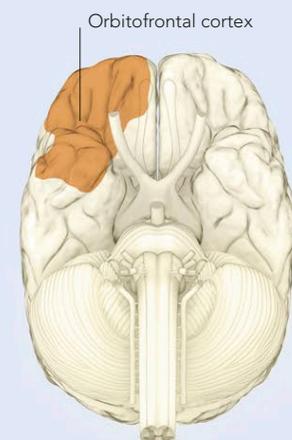


LEFT HEMISPHERE

SEEING BABIES

Parents' brains react more strongly to the sight of their own child's face than to that of others. In mothers, the strength of response, especially in the amygdala, is correlated with the extent to which the mother is bonded with the child. Mothers suffering postnatal depression show a reduced amygdala response compared to those strongly attached to their child.

Imaging studies reveal that all adults show a particular response to the sight of a baby's face. A spot in the orbitofrontal cortex – a brain area associated with emotion – becomes active when they see an infant, but not when they see an adult face. This "signature" response is the same in men and women, and in both parents and non-parents. It suggests that we are primed by evolution to feel an emotional bond with infants of our own species.



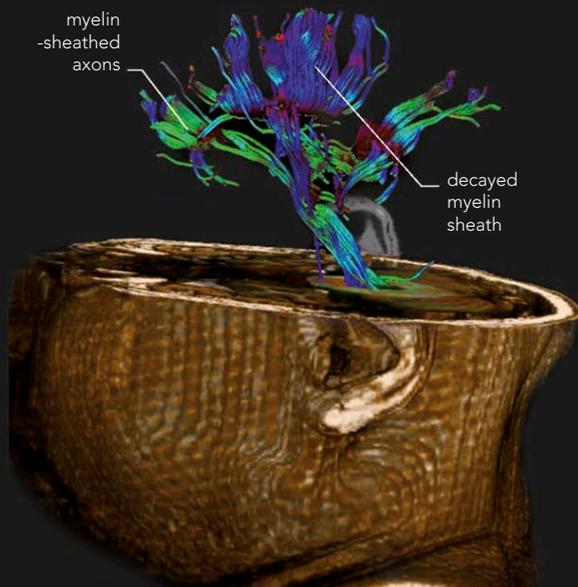
INFERIOR VIEW

THE AGING BRAIN

THE TRADITIONAL VIEW OF AGING IS THAT THE BRAIN AND THE BODY START TO DEGENERATE. THIS IS TRUE IN THAT NEURONS ARE LOST AND, FOR THOSE THAT REMAIN, IMPULSES ARE TRANSMITTED MORE SLOWLY. THIS CAN LEAD TO SLOWING THOUGHT PROCESSES, MEMORY PROBLEMS, AND DETERIORATING REFLEXES, WHICH CAN CAUSE PROBLEMS WITH BALANCE AND MOVEMENT.

NATURAL DEGENERATION

In the past, it was rare for people to live to the age of 50 and beyond, so we have not evolved to use the brain in such advanced years. This makes the aging brain a relatively new phenomenon in human history and evolution. The natural degeneration of the brain and nervous system is not caused by disease, so it should not be confused with the pathology of dementia, which is associated with a pattern of specific brain changes. Recent research shows that most neurons actually remain healthy until you die, but brain volume and size decrease 5-10 percent from the age



of 20-90. There are also changes in topography, with the grooves widening and tangles and plaques (small, disk-shaped growths) forming. However, the role of these deficits is not absolutely clear. They can occur in the brains of both healthy people and sufferers of Alzheimer's disease.

MYELIN DECAY

The myelin sheath that insulates the axons of neurons is vital for effective cell-to-cell communication. This protein-based structure decays with age, leaving brain circuits less efficient, leading to balance and memory problems. The decayed myelin sheaths traveling from the cortex to spine are shown as blue and purple on this image, while the healthy ones are shown in green.

AGE AND EXCITEMENT LEVELS

Dopamine is a neurotransmitter that triggers excitement and rapid decision-making. Brain-imaging studies suggest that, as people age, activity in their dopamine circuits decreases. This might be reflected in behavioral changes, because dopamine is linked with thrill-seeking and risk-taking. Perhaps older people prefer a quieter life than younger people because dopamine is less abundant.

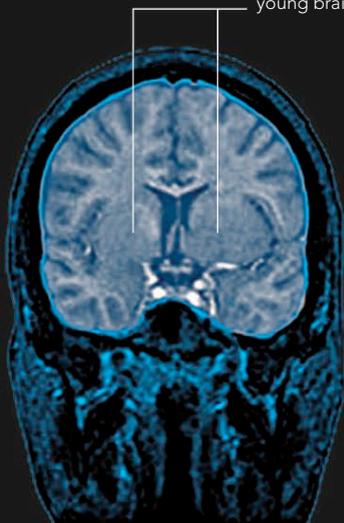


THE THRILL OF CHRISTMAS

Opening presents is highly exciting for children, but much less so for older people because dopamine, which is triggered by "rewards" (in this case, gifts), has less impact as you age.

Basal ganglia

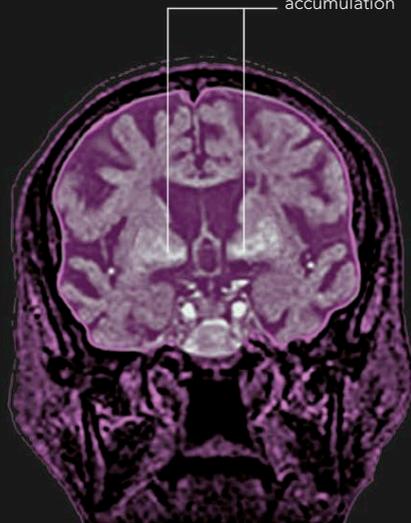
These clusters of nerve cells appear normal in the young brain



27-YEAR-OLD

Basal ganglia

The brighter areas are the product of iron accumulation



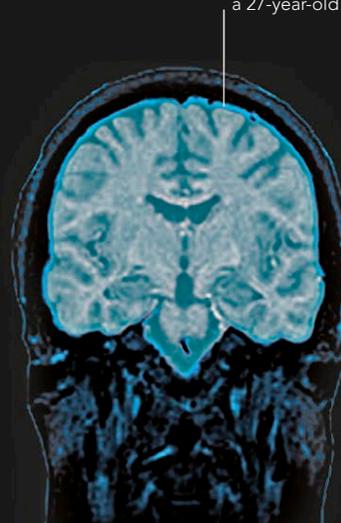
87-YEAR-OLD

BASAL GANGLIA

This series of MRI scans shows the differences between crucial areas of the brain of a young adult compared to an elderly adult. The scans above show the basal ganglia, which plays a vital role in coordinating movement.

Subarachnoid space

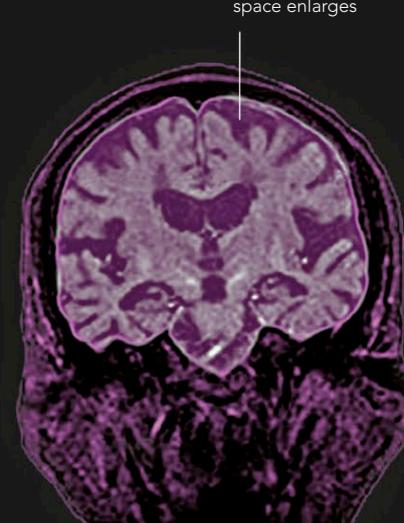
The size of this area as shown here is normal in a 27-year-old



27-YEAR-OLD

Subarachnoid space

As the brain becomes smaller due to lifelong loss of brain cells, this space enlarges



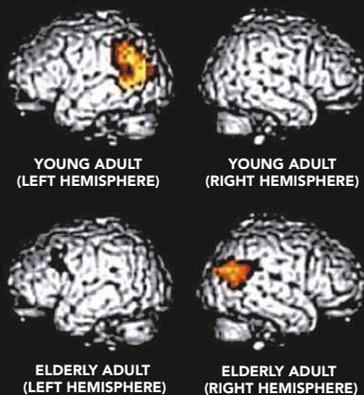
87-YEAR-OLD

SUBARACHNOID SPACE

The subarachnoid space is the area around the outside edge of the brain, and is known as a potential site for brain hemorrhage (see p.221). It becomes notably larger as the brain ages, reflecting a general reduction in brain volume.

POSITIVE AGING

The brain can compensate for the effects of aging, and mental function can even improve with age. Myelin increases in the temporal and frontal lobes in the 45–50 age group may enable people to manage their knowledge better. Also, comprehension studies have shown that high-functioning older adults use either both hemispheres together, or a different hemisphere than either young adults or lower-functioning older adults. This may be the brain's way of making up for declining functions, to keep thought and memory processes stronger.



BRAIN ACTIVATION CONTRASTS

One study compared fMRI scans of brain activity in young adults (top row) and older adults (bottom row) during sentence comprehension. The results suggest that older people with good comprehension compensate for the deficits in language areas of the brain by recruiting other areas.

KEEPING THE BRAIN YOUNG

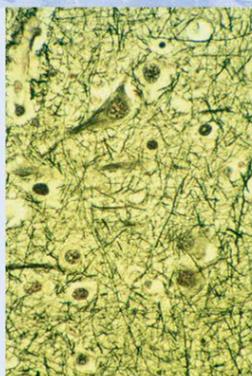
New research into brain aging indicates that the rate of decline may be slowed by lifestyle factors, such as regular exercise. Research has also found that reducing food intake, resulting in lower blood glucose levels, may slow the pace of change because blood glucose can cause damage to proteins. Certainly, people with elevated blood glucose levels, such as those with type 1 diabetes, show more signs of brain aging than nondiabetic individuals.



PROTEIN ACCUMULATION

A recent study examined the brains of five people in their eighties, who had performed very well in memory tests, and compared them to the brains of "normal," nondemented elderly people of a similar age. The ones who performed well in the memory tests had fewer tangles consisting of a protein called tau in their brains than the other group. These tangles grow inside brain cells and are thought to eventually kill them.

FIBERLIKE TANGLES
Microscopic tangles (shown as dark masses) are often found in large numbers in the brains of Alzheimer's patients.



BENEFITS OF A HEALTHY LIFESTYLE

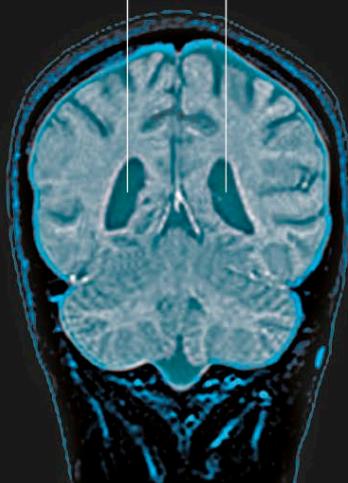
A number of lifestyle factors may help stimulate the growth of neural tissue. Gentle aerobic exercise, such as rapid walking, regular sleep, a good diet, and mental exercises help delay age-related mental decline and protect against age-related problems, such as memory loss.

Ventricles

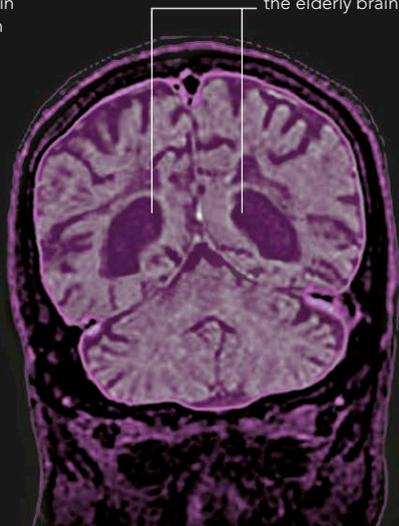
These hollow spaces filled with cerebrospinal fluid are a normal size in the younger brain

Ventricles

These hollow spaces are much larger in the elderly brain



27-YEAR-OLD



87-YEAR-OLD

VENTRICLES

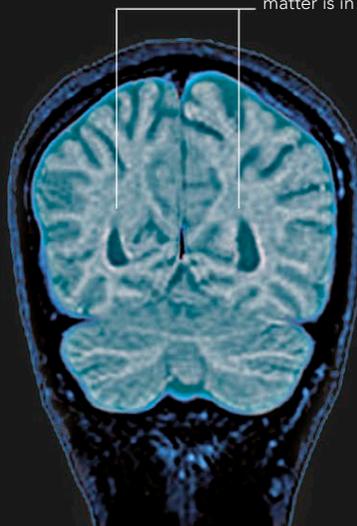
The ventricles contain cerebrospinal fluid, which performs several functions, including protecting the brain from injury and transporting hormones. These areas become larger as the brain ages, as a result of the general loss of gray matter.

White-matter tract

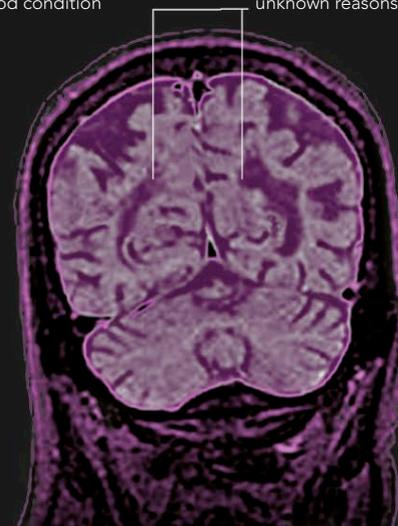
This communication channel for the brain's information-processing gray matter is in good condition

White-matter tract

This changes in appearance during aging for as yet unknown reasons



27-YEAR-OLD



87-YEAR-OLD

WHITE-MATTER TRACTS

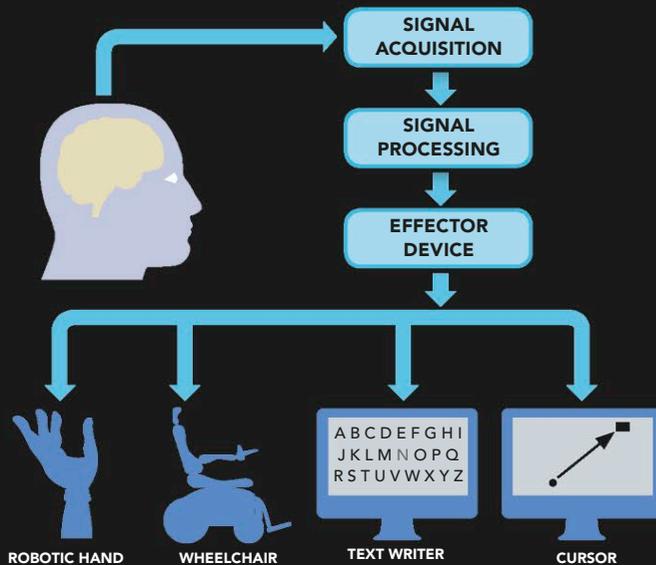
The white matter contains mainly supporting (glial) cells, which are needed to support neurons. Because there are less supporting cells as the brain gets older, neurons function less efficiently.

THE BRAIN OF THE FUTURE

AS WE DISCOVER HOW THE BRAIN WORKS, THE PROSPECT OF CHANGING IT, ENHANCING IT, AND DEVELOPING ARTIFICIAL BRAINS IS FAST BECOMING FACT RATHER THAN FICTION. TECHNOLOGIES FOR MIND READING, THOUGHT CONTROL, AND ARTIFICIAL INTELLIGENCE ARE ALREADY WITH US AND ARE BECOMING MORE SOPHISTICATED EVERY DAY.

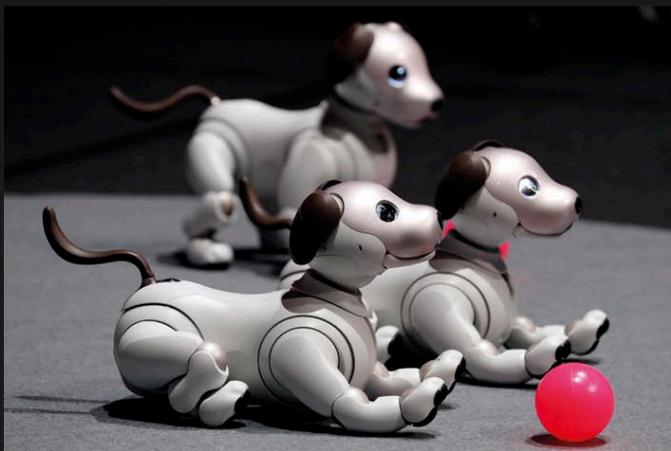
BRAIN-MACHINE INTERFACES

When a person is thinking, the brain produces electrical signals. Scientists have discovered ways in which the electrical signals can be picked up by sensors and sent wirelessly to other electrical devices, making it possible for a person to move or alter objects by thought alone. Most research in this field is directed toward developing devices to help people with nervous-system injuries regain the use of paralyzed limbs. The technology has also been picked up by some computer-game manufacturers, who have produced games that can be played using thought power.



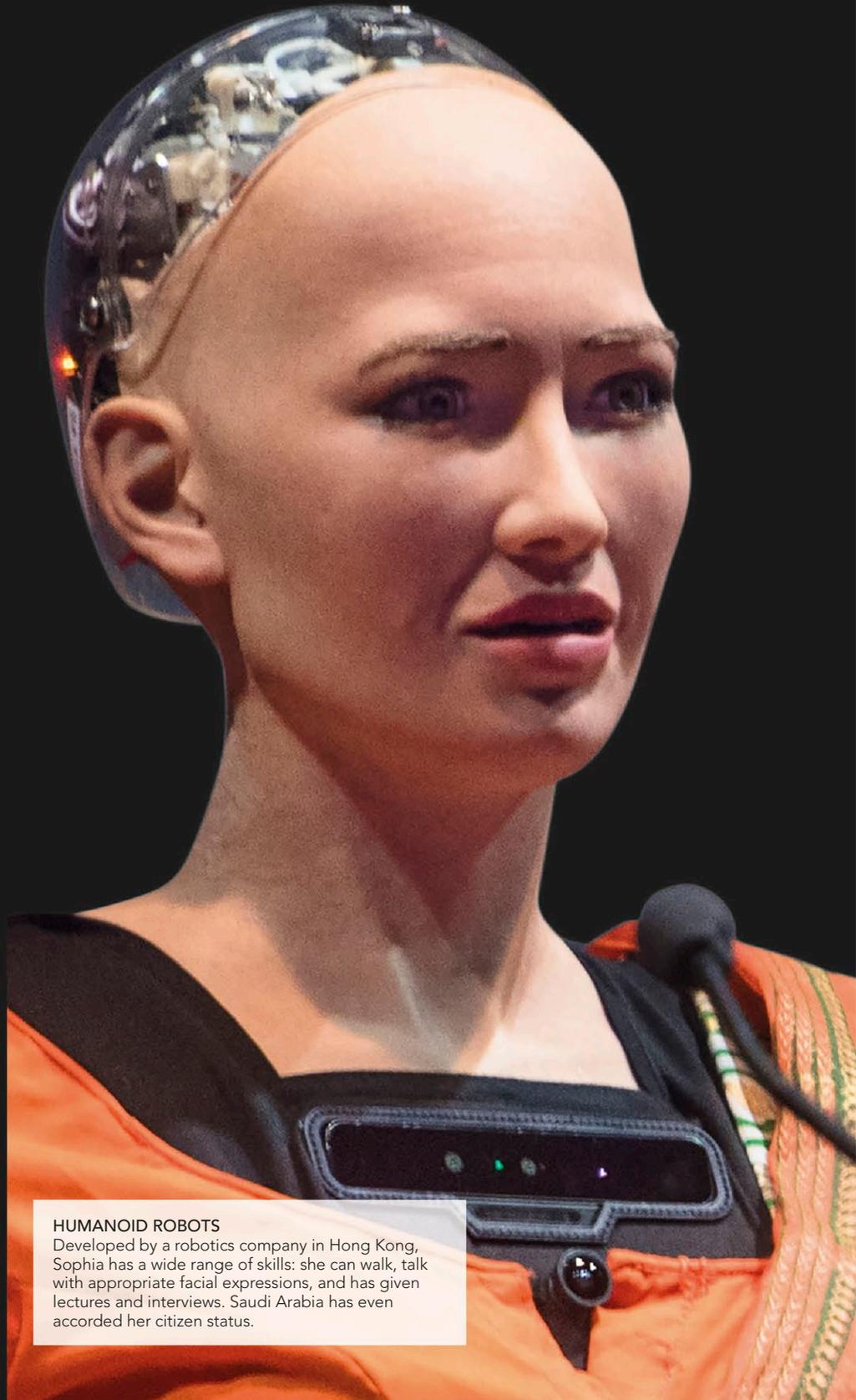
REGAINING CONTROL

Mind-control technology allows people to use devices such as artificial limbs, wheelchairs, and computers simply by directing their thoughts. Signals from the brain are received, then analyzed and recoded, before being transmitted to a device as instructions.



HELPFUL ROBOTS

Modern robots are designed to help people and fulfill a wide range of functions. The latest robots can serve food, do housework, help in hospitals, take risks on the battlefield, and even function as cute, playful pets.



HUMANOID ROBOTS

Developed by a robotics company in Hong Kong, Sophia has a wide range of skills: she can walk, talk with appropriate facial expressions, and has given lectures and interviews. Saudi Arabia has even accorded her citizen status.

MIND READING

The “picture” of neural activity created by fMRI scanning can be translated into a precise description of what a person is seeing and, to some extent, thinking. To achieve this, the output of a person’s fMRI scan, captured while he or she is looking at a particular image, is processed by sophisticated computer software that translates the pattern of activity into a visual “readout.” Such “mind reading” is made possible because neurons in the visual cortex are specialized for specific stimuli—horizontal or vertical lines, for example—so their firing patterns are indicative of the type of visual stimuli the neurons are registering.

MAKING FACES

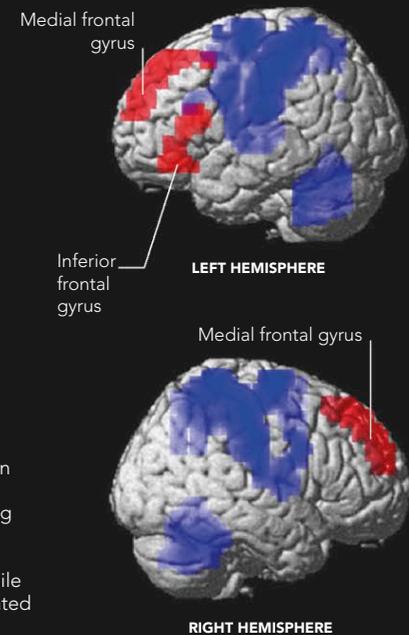
EEG brain scans of people looking at faces have been decoded by scientists in Canada and then fed into a computer, which reproduces what that person is seeing.



STIMULI RECONSTRUCTIONS

LIE DETECTION

Mind reading is not limited to revealing what a person is looking at. Brain-scanning studies have shown that, when a person is lying, the brain generates a different pattern of neural activity from when they are telling the truth. This has been used to develop a “lie detector” that analyzes brain activity captured by fMRI. Although still in development, the technology is claimed to have an accuracy rate of over 90 percent—significantly greater than the accuracy rate of polygraph tests.



REVEALING THE TRUTH

Different areas of the brain are activated according to when someone is telling the truth or lying. Here, the red areas show the telltale activity of a lie, while the blue areas are associated with telling the truth.

ARTIFICIAL INTELLIGENCE

Scientists have been working for decades on producing intelligent nonbiological systems, and have been very successful in developing computer programs that can equal, or sometimes outperform, the human brain. Chess programs, for instance, can now compete on even terms with the best players in the world. However, it has proved difficult to develop systems that are as flexible as the human brain, and thus able to operate in the constantly changing environments that constitute “real” life. To overcome this, the emphasis of artificial intelligence research has recently shifted from developing more advanced computers to creating “emotional” machines that are able to make crude but quick “holistic” or “intuitive” judgments that do not depend on enormous calculating capacity.

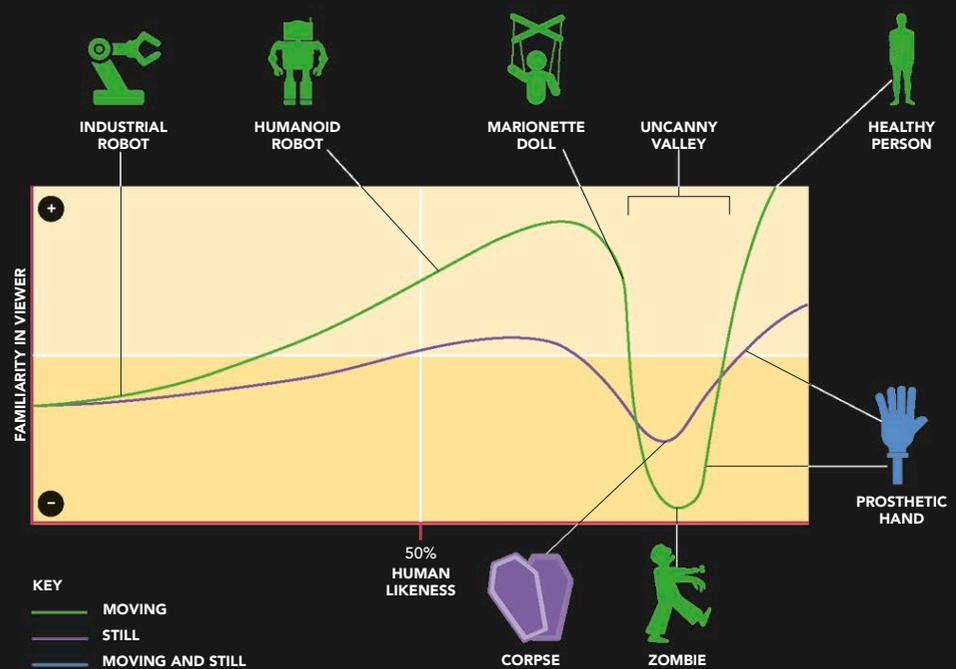


GO WINNER

AlphaGo—a program developed by Google DeepMind—beat Ke Jie, the world number-one Go player, at the 2017 Future of Go Summit. Go is an ancient game, even more complex than chess.

THE UNCANNY VALLEY

As robots are made to look more like humans, people find them increasingly uncomfortable. Robots such as Sophia (see opposite), fall into what is known as the “uncanny valley.” This is a dip in a graph relating to a machine, which has a vertical axis measuring how comfortable people feel with it and a horizontal axis measuring how closely the machine resembles a real person. While mechanical robots do not worry people, once a device looks human yet “not quite right,” a sense of uneasiness occurs.

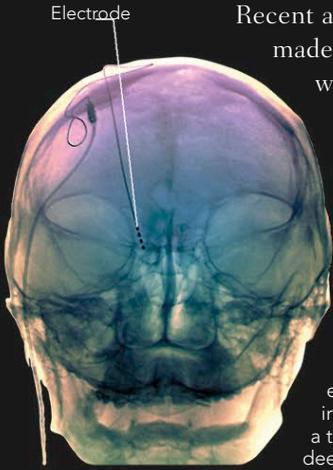


MONSTER OR MACHINE?

This graph illustrates that although humanoid robots are more familiar to people than more functional-looking industrial robots, there is a tipping point at which increased likeness to humans results in less familiarity. This is the “uncanny valley.”

THE STATE OF TECHNOLOGY

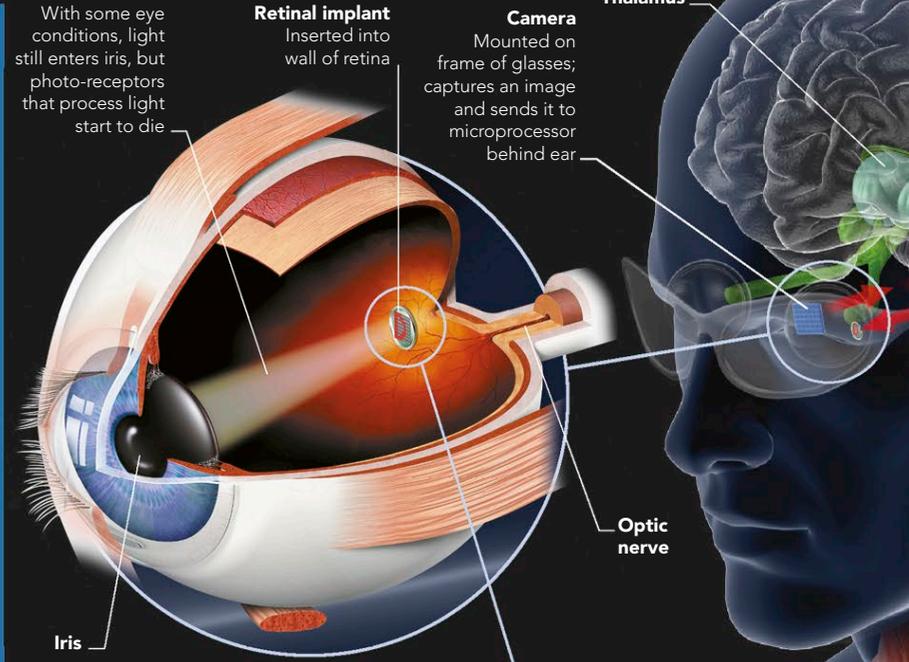
Recent advances in biotechnology have made it possible to replace damaged limbs with artificial ones that can be controlled by thought, operating in much the same way as the original. Another advance involves altering brain function by inserting electrical pacemakers. Artificial sense organs, such as the bionic eye, are already on trial, and artificial brain parts such as memory add-ons and hippocampus replacements are not far behind.



BRAIN PROBE
This X-ray shows an electrode inserted into the brain during a technique called deep-brain stimulation.

THE BIONIC EYE

People who have become blind as a consequence of eye conditions (as opposed to damage to areas of the brain associated with vision) may soon be able to see again thanks to the development of artificial eyes. A “bionic” eye prototype has been created, comprising a computer chip that sits in the back of the individual’s own eye socket, which is linked up to a tiny video camera built into a pair of glasses. Images captured by the camera are beamed to the chip, which translates them into electrical impulses and sends them on to the visual cortex via the optic nerve.



Retinal implant
Inserted into wall of retina

With some eye conditions, light still enters iris, but photo-receptors that process light start to die

Camera
Mounted on frame of glasses; captures an image and sends it to microprocessor behind ear

Iris

Thalamus

Optic nerve

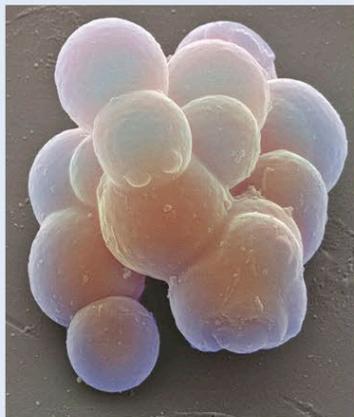
Retina cross section

Retinal implant
Receives signals from microprocessor and emits pulses, which travel via optic nerve to visual cortex in brain

Photoreceptors destroyed by disease

ETHICS AND TECHNOLOGY

As biotechnology advances, it generates ethical and moral dilemmas. Brain technologies are particularly sensitive because most of us consider the products of our brain—thoughts, feeling, desires—as the central part of our “selves.” Stem cells—immature body cells that have the potential to turn into many different types of cells—might one day be used to restore damaged neurons. Their use in other areas of medicine has already generated huge debate, because initially they had to be harvested from human fetuses, but they can now be obtained another way.



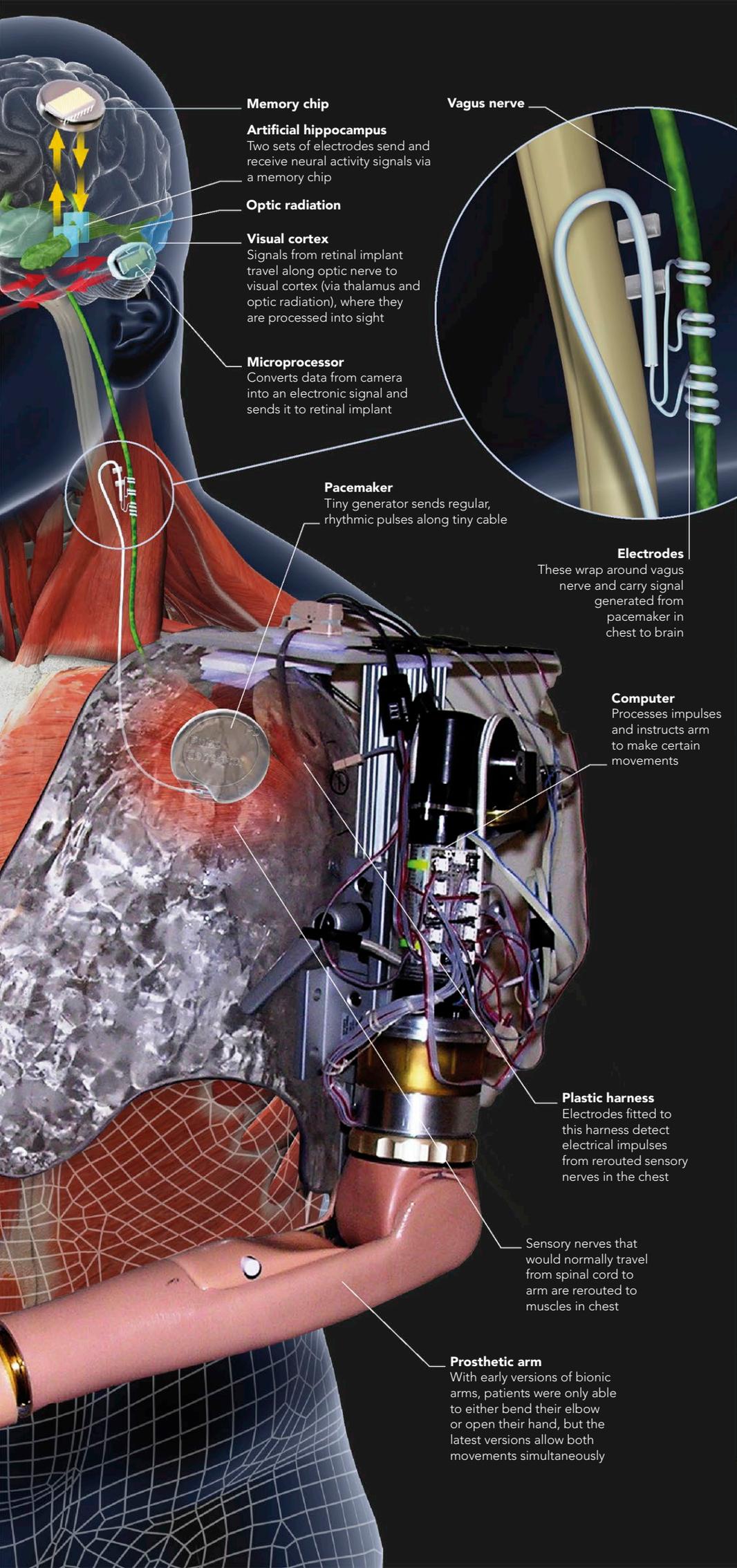
STEM CELLS
Stem cells like these can now be taken from blood flowing through the umbilical cord. Initially they came from fetuses, which caused much ethical debate.

NANOROBOTS
Microscopic robots could one day reengineer our bodies to be stronger, more intelligent, and resistant to disease, presenting complicated life choices.



BRAIN AND BODY ENHANCEMENTS

Practically every part of the body, including the sense organs, may soon have artificial counterparts. Some of these are already in development, although of those shown above only the vagus nerve stimulator is in widespread clinical use.



Memory chip

Artificial hippocampus

Two sets of electrodes send and receive neural activity signals via a memory chip

Optic radiation

Visual cortex

Signals from retinal implant travel along optic nerve to visual cortex (via thalamus and optic radiation), where they are processed into sight

Microprocessor

Converts data from camera into an electronic signal and sends it to retinal implant

Pacemaker

Tiny generator sends regular, rhythmic pulses along tiny cable

Electrodes

These wrap around vagus nerve and carry signal generated from pacemaker in chest to brain

Computer

Processes impulses and instructs arm to make certain movements

Plastic harness

Electrodes fitted to this harness detect electrical impulses from rerouted sensory nerves in the chest

Sensory nerves that would normally travel from spinal cord to arm are rerouted to muscles in chest

Prosthetic arm

With early versions of bionic arms, patients were only able to either bend their elbow or open their hand, but the latest versions allow both movements simultaneously

VAGUS-NERVE STIMULATION

The vagus nerve is a cranial nerve, traveling from the brainstem to various internal organs, that has an important role in mediating brain arousal. A number of different types of brain disorders, such as chronic epilepsy and severe depression, benefit from the effects of stimulating this nerve. A small disk with a tiny generator fueled by a lithium battery is surgically implanted in the chest, which sends regular, rhythmic pulses along a wire that is tethered to the left vagus nerve (the right vagus nerve runs directly to the heart). The frequency and intensity of the electrical pulses can be altered according to the severity of the condition.

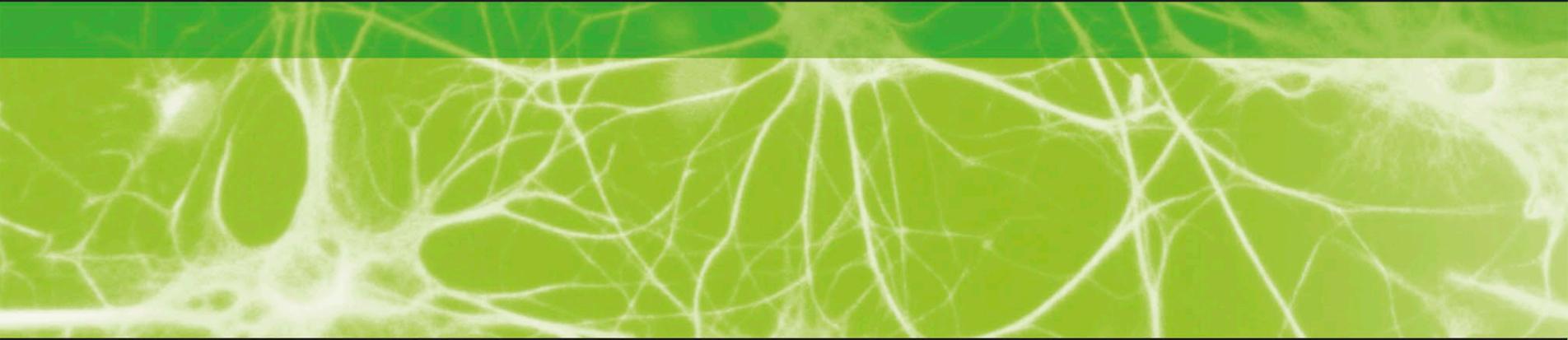
THE BIONIC ARM

A bionic arm that is operated by the power of thought alone is already in use, and future models, which are currently being developed, are likely to be more lifelike and increasingly dextrous. The current versions work by rerouting motor nerves from the brain that originally ran to the hand, and terminating them instead in electrodes, which communicate with computer-driven motors in the arm itself. Sensors feed a limited degree of sensory information back to the brain, so the user can determine both temperature and pressure.

THE FUTURE

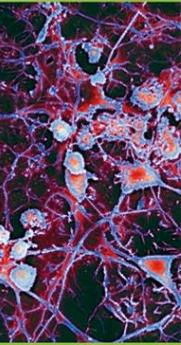
The rampant progress of biotechnology raises profound questions about what it is to be human. This is particularly true with technology that affects the human brain, because of all organs this is the one we identify with the most closely. Some of the most common questions raised include:

QUESTION	ANSWER
What changes in the way our brains function might we see if technology advances at its present rate?	"Thought" devices enabling us to control the world by mind power alone; synthetic brain "modules" to replace failing ones; conscious mood control by direct stimulation of the relevant brain areas.
Won't these things change what it means to be human? Will they even be acceptable?	Many of them, in crude form, are with us already and proving to be quite acceptable. We have "bionic" limbs, brain pacemakers, and even a prototype replacement hippocampus (see p.161).
What are the main technical problems still to be overcome?	The main problem is to do with mapping—despite the advances of the last ten years, the complex interconnections between different brain areas are still largely unknown.
Will machines ever be conscious?	There seems no reason why not. The ultimate challenge may not be technical at all, but rather the ethical implications of human consciousness being embodied in a nonhuman form.



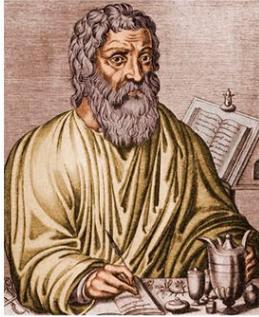
THE PERCEPTION OF BRAIN DISORDERS AND THEIR CAUSES HAS CHANGED PROFOUNDLY OVER THE COURSE OF HUMAN HISTORY. EVEN TODAY, DIFFERENT CULTURES HOLD MARKEDLY DIFFERENT VIEWS ABOUT THE DIVIDING LINE BETWEEN NORMAL AND DISORDERED STATES OF MIND. BUT, JUST AS OUR KNOWLEDGE OF HOW THE BRAIN WORKS IS CURRENTLY UNDERGOING A REVOLUTION, SO TOO IS OUR UNDERSTANDING OF WHAT CAN GO WRONG. NEVERTHELESS, THERE ARE MANY DISORDERS WITH CAUSES THAT REMAIN MYSTERIOUS.

DISEASES AND DISORDERS



THE DISORDERED BRAIN

EVERY MENTAL STATE HAS A CORRESPONDING BRAIN STATE, CONSISTING OF A PARTICULAR PATTERN AND SEQUENCE OF NEURAL PROCESSES. UNTIL RECENTLY, MOST OF THESE PROCESSES WERE UNDETECTABLE, BUT THE ADVENT OF HIGH-TECH IMAGING HAS MADE THEM VISIBLE, WITH THE RESULT THAT MENTAL DISORDERS ARE INCREASINGLY BEING RECOGNIZED AS NEUROLOGICAL BRAIN DISORDERS.



FOUR HUMORS

Hippocrates developed the idea that illness was the result of a lack of balance among four humors—blood, phlegm, and black and yellow bile.

HISTORICAL THEORIES OF MENTAL ILLNESS

Mental illness has commonly been regarded as disease of the spirit. In the Middle Ages it was assumed that devils (foul spirits) entered people and made them depressed (poor-spirited) or insane. Physical theories of mental illness include an imbalance of the “four humors,” which were thought to determine a person’s general mood and health, and fluctuations or blockages of various types of “forces.” The 19th-century physician Franz Mesmer, for example, thought he had discovered “animal magnetism,” which could cause ill health, including madness, if it was blocked. His treatment to control the magnetic flow was, effectively, hypnotism. Sigmund Freud (see p.189) popularized the concept of the unconscious, and believed that suppressed desires caused neurosis. He developed psychoanalysis, based on the idea of bringing hidden conflicts to consciousness.



EXORCISM Exorcism is a ritual designed to expel bad spirits from the living. It was widespread in the Middle Ages, when demonic possession was often thought to be the cause of mental illness.



HEALING ENERGY

“Mesmerists” healed anxious minds by hypnotism, although at the time they thought they were using animal magnetism (energy flow).

WHAT IS MENTAL DISORDER?

Mental illness is generally diagnosed when a person reports that they are experiencing the world in a way that is radically different from others or when their behaviour makes it difficult for them to function in society. The shifting nature of mental illness makes diagnosis notoriously difficult. Yet standard diagnosis is important because the presence or absence of mental illness may decide whether a person is criminally responsible, suitable for particular types of employment, or eligible for state aid. Medical practice also makes diagnosis essential before treatment can be given. The most commonly consulted guide to mental disorders is the US Diagnostic and Statistical Manual (DSM) published by the American Psychiatric Association (see panel below).

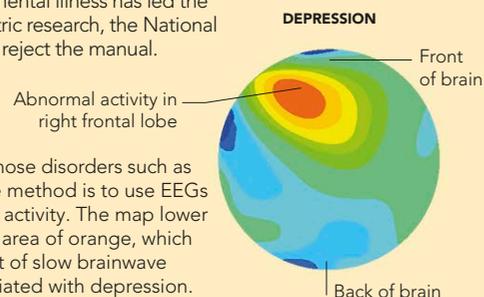
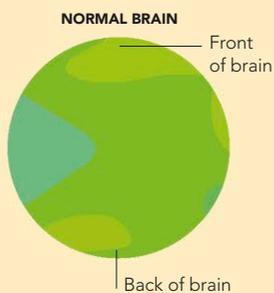


MODERN DIAGNOSTIC TOOLS

Some mental illnesses may be diagnosed by brain imaging—CT and MRI scans are good at showing tumors and areas of damage. Functional brain imaging may be used to explore abnormal brain patterns, such as those found in epilepsy.

DIAGNOSING MENTAL DISORDERS

The first edition of the DSM was published in 1952 following research by the US military during the Second World War. The current edition, DSM-5, was published in 2013 after 14 years of research. DSM-5 includes new diagnostic and classification criteria for some conditions—for example, Asperger’s is now part of the autistic spectrum rather than a condition in its own right. But controversy has arisen over whether the manual has changed sufficiently to reflect advances in brain research. Diagnosis is still based firmly on behavioral tests not brain imaging or biological markers. The failure of DSM-5 to take a neuroscientific approach to mental illness has led the largest US center for psychiatric research, the National Institute of Mental Health, to reject the manual.



IMAGING DEPRESSION

Brain imaging can help diagnose disorders such as anxiety and depression. One method is to use EEGs to reveal abnormal electrical activity. The map lower right, for example, shows an area of orange, which represents an excess amount of slow brainwave activity. This pattern is associated with depression.

PHYSICAL DISORDERS

All mental illness is physiological in that the behavior and experience associated with it is created by a pattern of neuronal activity, but only conditions that are clearly linked to damage are considered to be physical.

DEVELOPMENTAL Growing brains are very sensitive to environmental assault, such as oxygen deprivation. A problem before or during birth may cause permanent damage.

TRAUMATIC Brain trauma may arise from external events such as accidents that cause head injuries, and also from “cerebral” accidents, such as strokes and aneurysms.

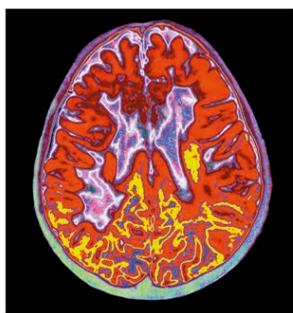
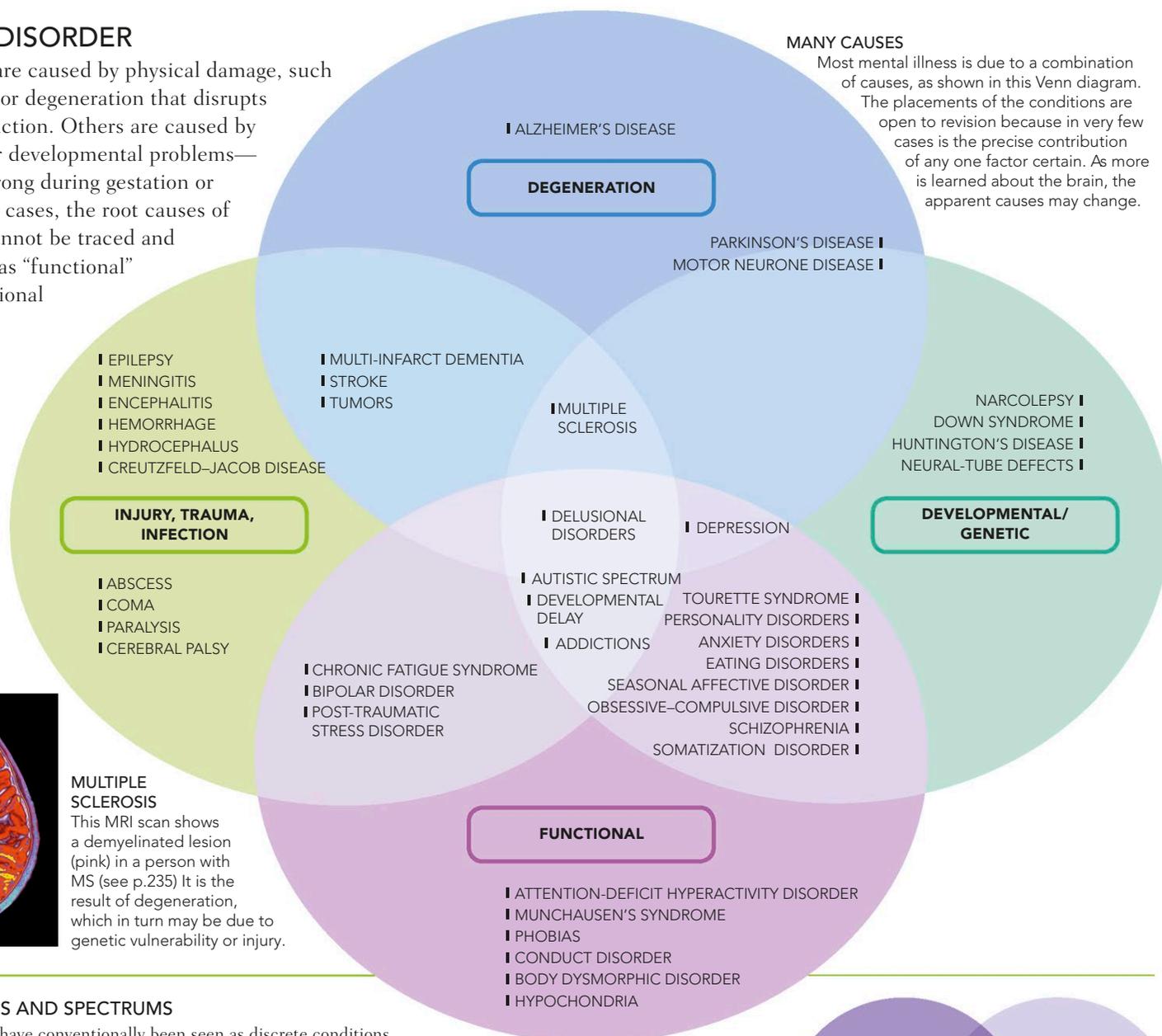
DEGENERATIVE Brains, like all the organs, degenerate, and this can result in mental conditions such as memory loss, cognitive impairment, and, in severe cases, dementia.

ROOTS OF DISORDER

Some disorders are caused by physical damage, such as a head injury, or degeneration that disrupts normal brain function. Others are caused by “faulty” genes, or developmental problems—things that go wrong during gestation or infancy. In many cases, the root causes of mental illness cannot be traced and simply manifest as “functional” problems. Functional disorders may be marked by abnormalities in brain function, but it is often unclear if these are the cause or effect of the condition.

MANY CAUSES

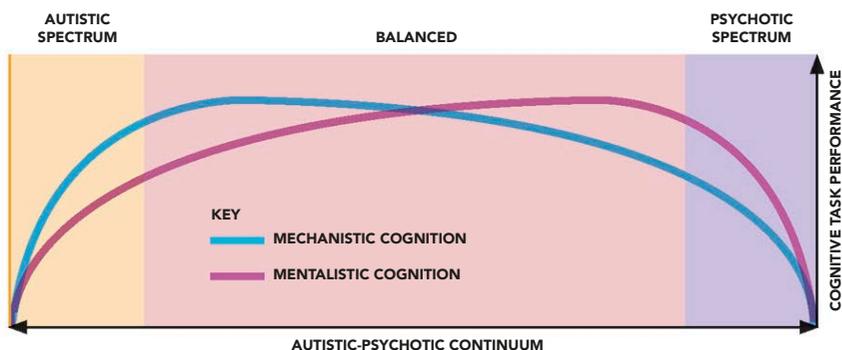
Most mental illness is due to a combination of causes, as shown in this Venn diagram. The placements of the conditions are open to revision because in very few cases is the precise contribution of any one factor certain. As more is learned about the brain, the apparent causes may change.



MULTIPLE SCLEROSIS
This MRI scan shows a demyelinated lesion (pink) in a person with MS (see p.235). It is the result of degeneration, which in turn may be due to genetic vulnerability or injury.

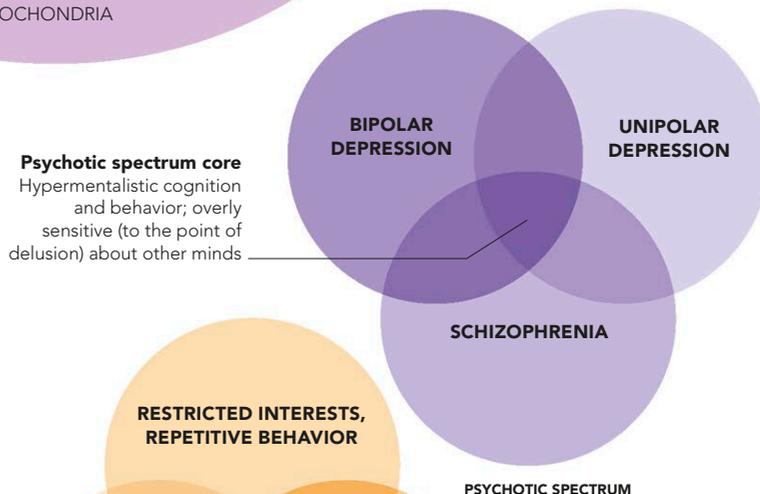
CONSTELLATIONS AND SPECTRUMS

Many disorders that have conventionally been seen as discrete conditions are now being recognized as related. People with autism, for example, have a core problem with understanding other people's mental processes. Around this core lies a constellation of symptoms grouped into three overlapping “suites” of behavior. These suites are conventionally seen as different types of problem, but their common relationship to the core deficit suggests that they share a genetic underpinning. Psychosis has a core that is characterized by over-interpretation of other minds. It too can be seen as the core of symptoms from overlapping suites.

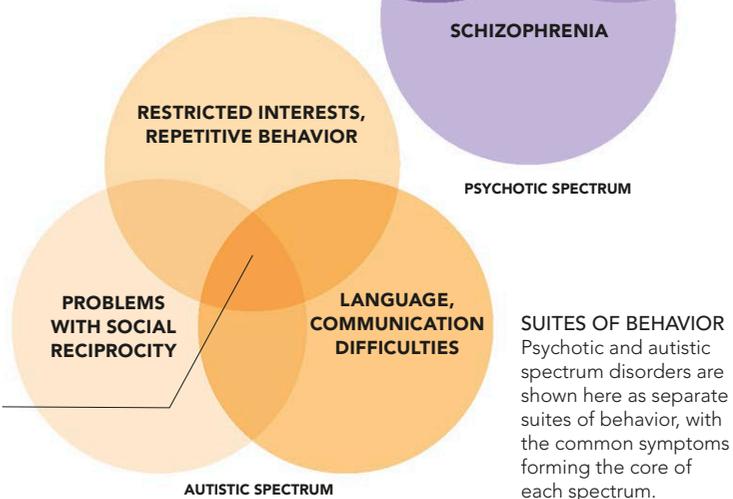


OPPOSITE PROBLEMS?

Although they appear entirely different, autistic constellation disorders and psychotic spectrum conditions may actually be related. The two clusters of symptoms may be envisaged as existing on a single spectrum (above) with normal behavior in the middle.



Autistic spectrum core
Hypomentalistic—problems with understanding other people's mental processes or reflecting on one's own



HEADACHE AND MIGRAINE

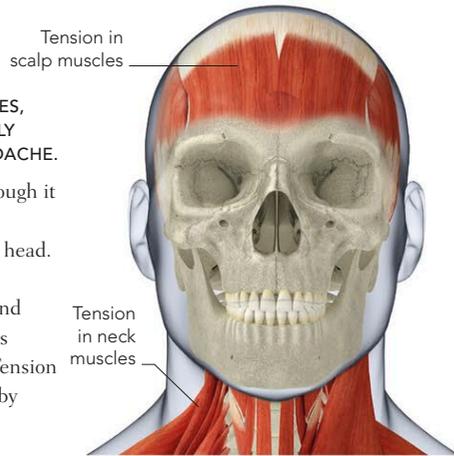
Headache is a common symptom but the mechanism underlying it is not known for certain. The brain itself has no pain-sensitive nerve receptors. In many cases, it is thought that tension in the meninges or in blood vessels or muscles of the head and/or neck

stimulates pain receptors, which send impulses to the sensory cortex of the brain, resulting in a headache. However, in some types of headache, such as migraine, the pain is thought to be due to overactivity of neurons that affects the brain's sensory cortex.

TENSION HEADACHE

ALSO KNOWN AS STRESS HEADACHES, TENSION HEADACHES ARE PROBABLY THE MOST COMMON TYPE OF HEADACHE.

The pain tends to be constant, although it may throb, and it may occur in the forehead or more generally over the head. The pain may be accompanied by tightening of the neck muscles and a feeling of pressure behind the eyes and/or tightness around the head. Tension headaches are typically brought on by stress, which causes tension in the muscles of the neck and scalp. This, in turn, is thought to stimulate pain receptors in these areas, which send "pain impulses" to the sensory cortex.



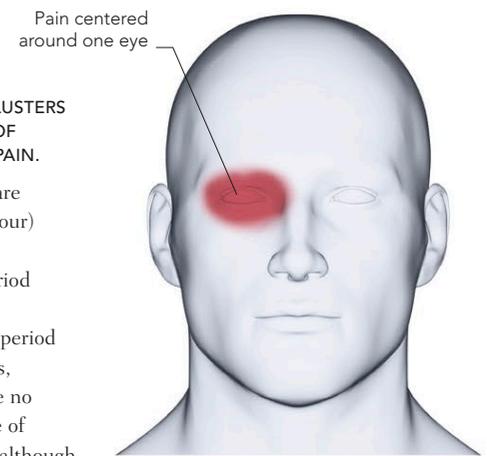
MUSCULAR TENSION

Pain receptors in the muscles of the scalp and neck are stimulated by muscular tension, leading to the pain of a tension headache.

CLUSTER HEADACHE

THESE HEADACHES OCCUR IN CLUSTERS OF RELATIVELY SHORT ATTACKS OF SEVERE, OFTEN EXCRUCIATING, PAIN.

During cluster headaches there are several attacks (typically one to four) a day, followed by an attack-free remission period. The cluster period usually lasts from a few weeks to a couple of months. A remission period may last for months or even years, although some people experience no significant remissions. The cause of cluster headaches is not known, although there is some evidence that abnormal nerve cell activity in the hypothalamus may be involved.



AREA OF PAIN

A cluster headache typically affects one side of the head and is centered around the eye, which may also water and become inflamed.

MIGRAINE

A MIGRAINE IS AN INTENSE, OFTEN THROBBING HEADACHE, MADE WORSE BY MOVEMENT AND OFTEN ACCOMPANIED BY SENSORY DISTURBANCES AND NAUSEA.

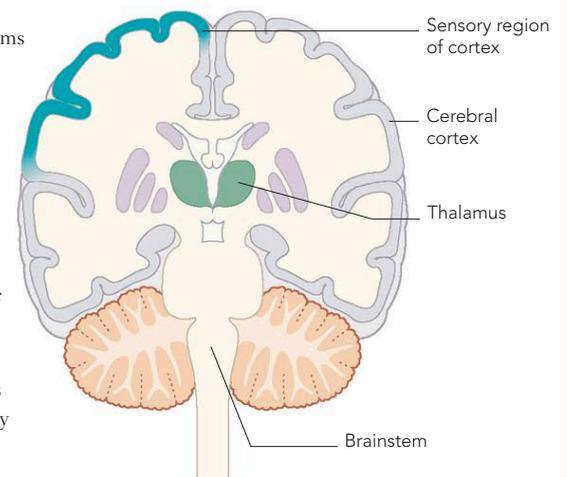
A migraine headache usually occurs at the front or one side of the head, although the area of pain can move during an attack.

Migraine is classified into two types: classical migraine and common migraine. In classical migraine, the headache is preceded by aura, a group of warning symptoms that includes: visual disturbances, such as flashing lights and other distortions; stiffness, tingling, or numbness; difficulty speaking; and poor coordination. In common migraine there is no aura. In both types there may be an early stage, known as prodrome, with features such as difficulty concentrating, mood changes, and fatigue or excessive energy. In common migraine, the prodrome is followed by the headache; in classical migraine, the prodrome is followed by aura, which

is then succeeded by the headache. The headache gets worse with movement, and it is accompanied by symptoms including nausea and/or vomiting, and increased sensitivity to sound, light, and sometimes smells. It is often followed by a postdrome stage, in which there may be fatigue, difficulty focusing, poor concentration, and persistence of increased sensitivity.

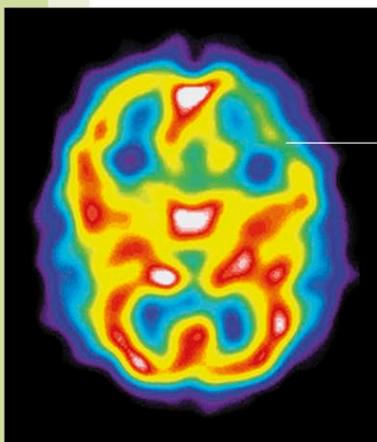
Causes and triggers

The underlying cause of migraine is not known, but recent research suggests that it may be due to a surge of neuronal activity that sweeps through parts of the brain, eventually stimulating the sensory cortex, which results in the sensation of pain. However, many external factors that trigger migraine attacks have been identified: dietary factors, such as irregular meals, specific foods, and dehydration; physical factors, such as fatigue and hormonal changes; emotional factors, such as stress or shock; and environmental conditions, including changes in the weather or a stuffy atmosphere.

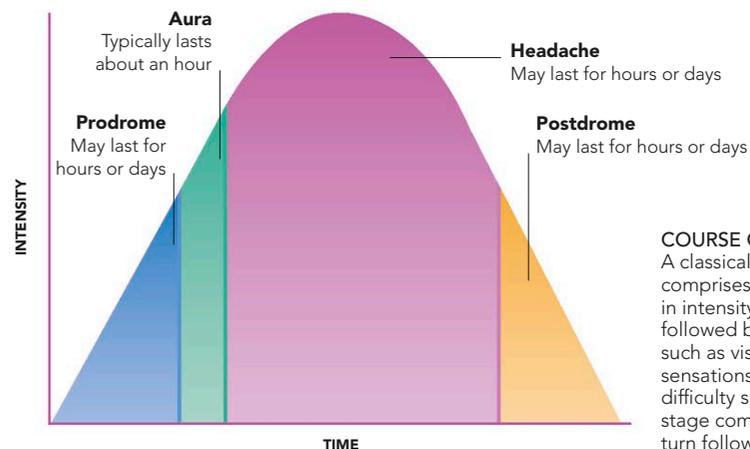


MECHANISM OF MIGRAINE

The neurological pathways that cause migraine are unknown, but may involve intense neuronal activity in the brainstem, thalamus, and sensory cortex.



DURING AN ATTACK
This SPECT scan shows different levels of brain activity during a migraine: red and yellow indicate high activity; areas of low activity are shown in green and blue.



COURSE OF MIGRAINE ATTACK
A classical migraine attack typically comprises four stages, which can vary in intensity and duration. Prodrome is followed by aura, with warning signs such as visual disturbances, abnormal sensations, poor coordination, and difficulty speaking. After the aura stage comes the headache, which is in turn followed by the postdrome stage.

CHRONIC FATIGUE SYNDROME

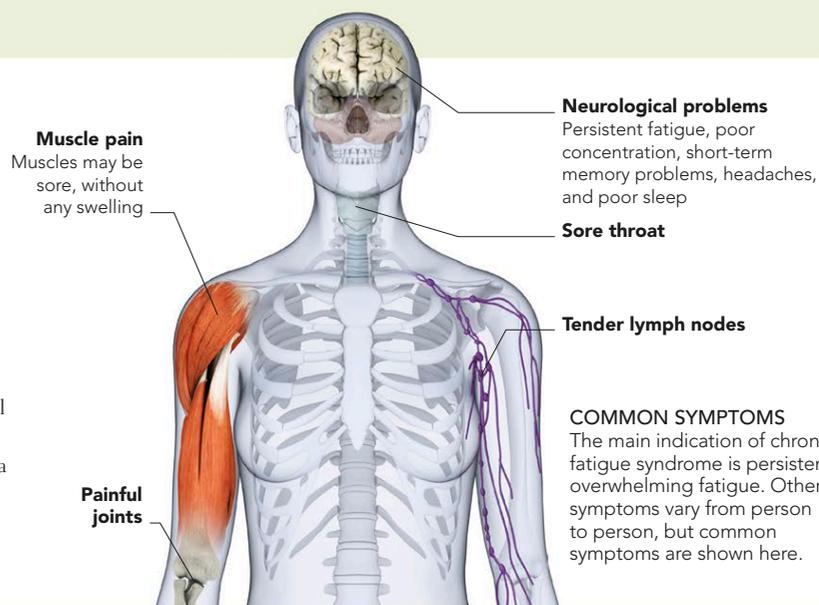
ALSO KNOWN AS MYALGIC ENCEPHALOMYELITIS (ME), CHRONIC FATIGUE SYNDROME IS A COMPLEX CONDITION THAT CAUSES EXTREME FATIGUE THAT LASTS FOR A PROLONGED PERIOD OF TIME.

The cause of chronic fatigue syndrome is not known. It can develop after a viral infection or a period of emotional stress, but in many cases there is no specific preceding factor. The principle symptom is persistent, overwhelming fatigue that lasts for at least several months.

Other symptoms vary, but commonly include poor concentration, impaired short-term memory, muscle and joint pain, and feeling ill and/or extremely tired after even mild exertion. The

disorder is also often associated with depression or anxiety, but it is unclear whether these are a cause or a result of the condition.

Chronic fatigue syndrome is usually diagnosed from the symptoms, although various tests and psychological assessments can be carried out to exclude other possible conditions. It is a long-term disorder, although there may be periods of remission and sometimes the disorder clears up spontaneously.



HEAD INJURIES

HEAD INJURIES RANGE FROM MINOR BUMPS WITH NO LONG-TERM EFFECTS TO BRAIN DAMAGE THAT CAN BE FATAL.

Injuries to the head are often classified as closed, in which the skull is not broken, or open, in which the skull is fractured, leaving the brain exposed. Closed head injuries may cause indirect damage to the brain. For example, a hard blow to the head that does not fracture the skull may cause brain injury at the site of impact as the inside of the skull hits the brain. Such a trauma may also cause brain injury at the opposite side of the head (a contrecoup injury). Open head injuries are caused by a strong impact from a sharp object that fractures the skull and may penetrate the brain, for example, a stab wound.

Effects

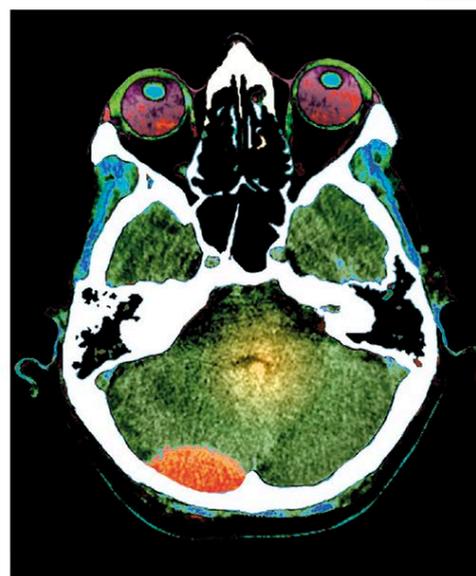
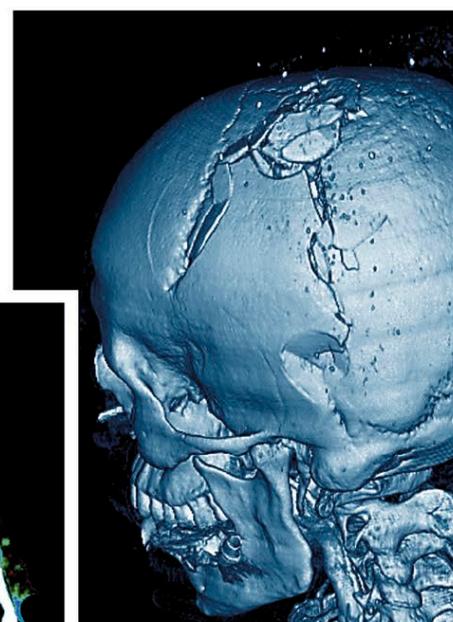
Head injuries can rupture blood vessels, causing a brain hemorrhage (see p.229). Minor head injuries typically produce only mild, short-lived symptoms, such as a bruise on the head. In some cases, a temporary disturbance of brain function (concussion) may follow even relatively

minor injuries, particularly if the injury has caused unconsciousness, and this may cause confusion, dizziness, and blurred vision, which may last for several days. Postconcussive amnesia can also occur. Repeated concussions eventually cause detectable brain damage, which may result in punchdrunk syndrome, symptoms of which may include impaired cognitive abilities, progressive dementia, parkinsonism (see p.234), tremors, and epilepsy.

Severe head injury may produce unconsciousness or coma, and usually brain damage, which in very severe cases may be fatal. In nonfatal cases, the effects of brain damage vary widely according to the severity and location of damage. The effects may include weakness, paralysis, problems with memory and/or concentration, intellectual impairment, and even personality changes. Such effects can be long-term or permanent.

FRACTURED SKULL

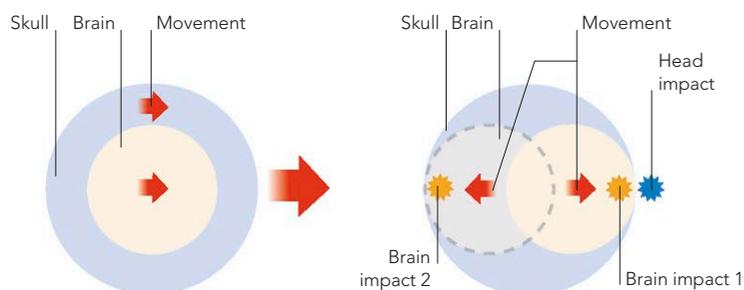
This three-dimensional CT scan of the skull reveals multiple fractures, including two large depressed fractures in which the skull has been pushed inward and fragmented. Such injuries are usually the result of a powerful blow from a blunt object and, in severe cases, may cause brain damage or even death.



HEMATOMA

This color-enhanced CT scan shows a large extradural hematoma (orange)—a mass of clotted blood caused by a hemorrhage that occurred due to a head injury. If not treated, it may press on the brain, causing brain damage or death.

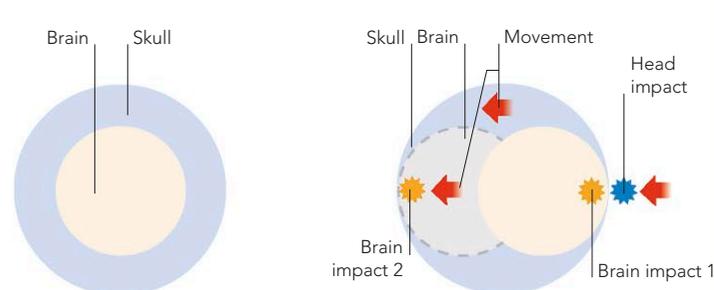
MOVING PERSON



1 In a person who is moving rapidly—for example, when traveling in a car—the skull and brain enclosed within it are moving at the same speed.

2 If movement is suddenly stopped due to an impact, the brain hits the front of the skull, and a contrecoup injury occurs when it rebounds and hits the back of the skull.

STATIONARY PERSON



1 In a situation in which a person is stationary, both the skull and the brain within it are motionless at the time that they are struck.

2 If the head is struck suddenly, the front of the skull is pushed against the brain, and the brain then rebounds and hits the back of the skull, causing a contrecoup injury.

EPILEPSY

EPILEPSY IS A BRAIN FUNCTION DISORDER IN WHICH THERE ARE RECURRENT SEIZURES OR PERIODS OF ALTERED CONSCIOUSNESS.

Normally, neuronal activity in the brain occurs in a regulated way. However, during an epileptic seizure neurons start firing in an abnormal way, disrupting normal brain function. Although seizures are a defining symptom of epilepsy, they can occur without epilepsy being the cause.

The mechanism underlying epileptic seizures is not known for certain, but it is thought to involve a chemical imbalance in the brain. Normally, the neurotransmitter gamma-aminobutyric acid (GABA) helps regulate brain activity by inhibiting neurons in the brain. When the level of GABA falls too low—which itself may be due to abnormal amounts of enzymes that regulate GABA levels—neurons are not inhibited and they send a flood of impulses through the brain, resulting in a seizure. Epilepsy can have a number of causes, although in many cases the cause is unclear.



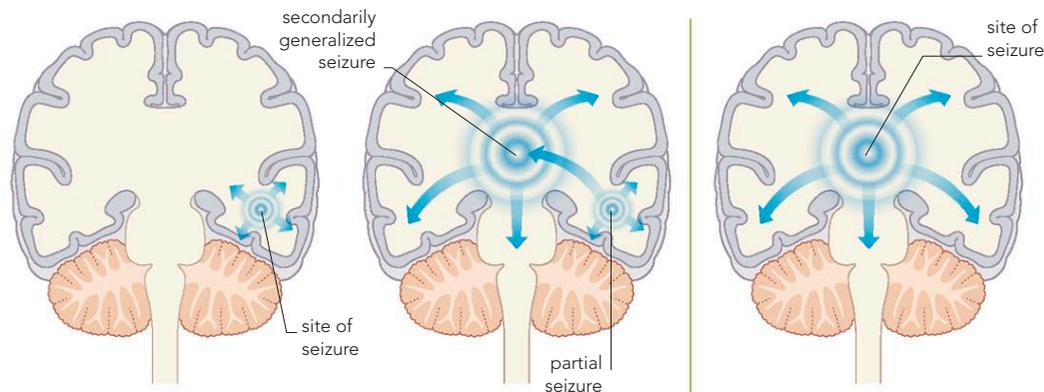
SEIZURE

This color-enhanced brain scan of a person with epilepsy reveals that the focus of seizure activity is in the right frontal lobe, as shown by the large orange cluster at the top right of the image.

A genetic factor may be involved in some cases. Other causes include head injury; birth trauma; an infection such as meningitis or encephalitis; a stroke; a brain tumor; and abuse of drugs or alcohol.

Many people find that specific factors can trigger a seizure. These triggers include stress; lack of sleep; fever; flashing lights; and drugs such as cocaine, amphetamines, Ecstasy, and opiates. Some women who suffer from epilepsy are more likely to have a seizure before the start of a menstrual period.

Broadly, epileptic seizures fall into two main types:



PARTIAL EPILEPTIC SEIZURES

In a partial seizure, the seizure starts in and affects only part of the brain (above left). Sometimes, a seizure may start as a partial seizure and then become generalized and spread (above right).

GENERALIZED EPILEPTIC SEIZURE

In generalized seizures, most or all of the brain is affected by abnormal neuron activity.

generalized seizures and partial seizures (see table below). Seizures often start in one area of the brain, which might contain scar tissue or some structural abnormality, and then spread throughout the rest of the brain.

Some people experience a warning sign (called an aura) before an epileptic seizure. These warning signs may include a strange smell or taste; a feeling of foreboding; déjà vu; and a sense of unreality. In most cases, seizures stop by themselves. Sometimes a seizure can persist or seizures follow on from each other without the person recovering in between.

This is known as status epilepticus and is a medical emergency.

STATUS EPILEPTICUS

Status epilepticus is the term used to refer to a potentially life-threatening condition in which there is a prolonged epileptic seizure or a series of repeated seizures that occur one after the other without recovery of consciousness between attacks. Precise definitions of status epilepticus vary, but generally it is defined as a single seizure that lasts for longer than 30 minutes, or a series of repeated seizures that lasts for longer than this time. In people who are known to have epilepsy, the most common cause of status epilepticus is failure to take antiepileptic medication. In other cases, the causes include a brain tumor, brain abscess, brain injury, cerebrovascular disease (such as a stroke), metabolic disorders, and drug abuse. Status epilepticus is a serious condition that may result in long-term disability or even death without prompt treatment with intravenous medications to control the seizures.

TYPES OF SEIZURES

Epileptic seizures can be categorized into two broad types, partial seizures and generalized seizures, depending on how much of the brain is affected by the abnormal neuron activity.

Partial seizures

In these types of seizures, abnormal neuron activity is restricted to a relatively small region of the brain. There are two main subtypes: simple partial seizures and complex partial seizures.

Simple partial seizures During these seizures there may be twitching on one side of the body; numbness or tingling; stiffness of the muscles in the arms, legs, and face; hallucinations of vision, taste, or smell; and sudden intense emotions. The person remains conscious throughout.

Complex partial seizures In these seizures the person is confused and unresponsive; may make peculiar, repetitive, apparently purposeless movements; and may scream or cry out, although there is no pain. The person remains conscious but usually has no memory of the seizure.

Generalized seizures

In these types of seizures, abnormal neuron activity affects most or all of the brain. There are six main subtypes, described below.

Tonic seizures In these seizures the muscles suddenly become stiff, which often causes the person to lose balance and fall over, usually backward. Tonic seizures tend to happen without warning, are usually short-lived, and the person recovers quickly.

Clonic seizures These seizures are very similar to myoclonic ones, causing jerking or twitching of the limbs or body, although they last longer, typically up to about two minutes. In addition, a person suffering a clonic seizure may lose consciousness.

Myoclonic seizures These generally happen shortly after waking up. During such seizures the arms, legs, or body twitch or jerk. A seizure usually lasts only a fraction of a second, but sometimes several seizures may occur in quick succession. Myoclonic seizures may occur on their own, but usually happen in association with other types, such as tonic-clonic seizures.

Atonic seizures These seizures are also sometimes called drop attacks. During the seizures the muscles suddenly relax and the person becomes floppy, which often causes them to lose balance and fall over, usually forward. Like tonic seizures, atonic seizures happen without warning, are short-lived, and the person recovers quickly after the seizure.

Tonic-clonic seizures Also sometimes known as grand mal, this type of seizure first causes the body to become rigid, this is followed by uncontrollable jerking or twitching. The person becomes unconscious and often loses bladder control. Typically, the seizure ends spontaneously after a few minutes, and afterward the person may be drowsy and confused.

Absence seizures Sometimes also known as petit mal, this type of epileptic seizure mainly affects children. During an absence seizure, the person loses awareness of his or her surroundings and appears to be staring vacantly into space. A seizure typically lasts for less than about 30 seconds, and in some cases seizures occur several times a day.

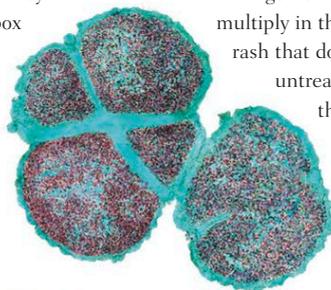
MENINGITIS

MENINGITIS IS INFLAMMATION OF THE MENINGES, THE MEMBRANES COVERING THE BRAIN AND SPINAL CORD, OFTEN AS A RESULT OF A VIRAL OR BACTERIAL INFECTION.

Typically, the infection reaches the meninges through the bloodstream from elsewhere in the body, although it may occasionally result from direct infection of the meninges after an open head injury. It may occur as a complication of various other diseases, including Lyme disease, encephalitis, tuberculosis, and leptospirosis. Viral meningitis may be caused by viruses such as herpes simplex or chickenpox virus. It tends to be relatively mild and causes symptoms similar to those of flu.

MENINGITIS BACTERIA

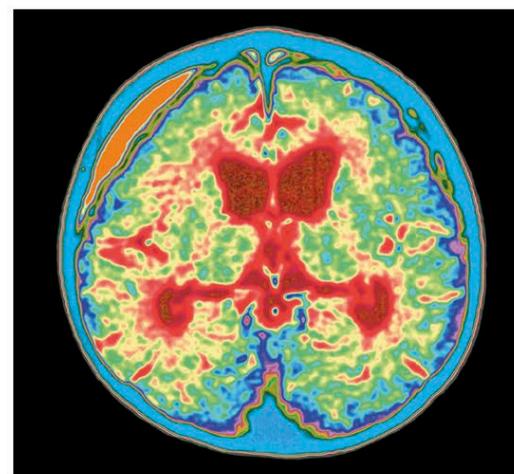
The five bacterial cells in the micrograph (right) are *Neisseria meningitidis* (also known as meningococcus), which is one of the most common causes of bacterial meningitis.



Rarely, it may cause serious symptoms, such as weakness or paralysis, speech problems, visual impairment, seizures, and coma.

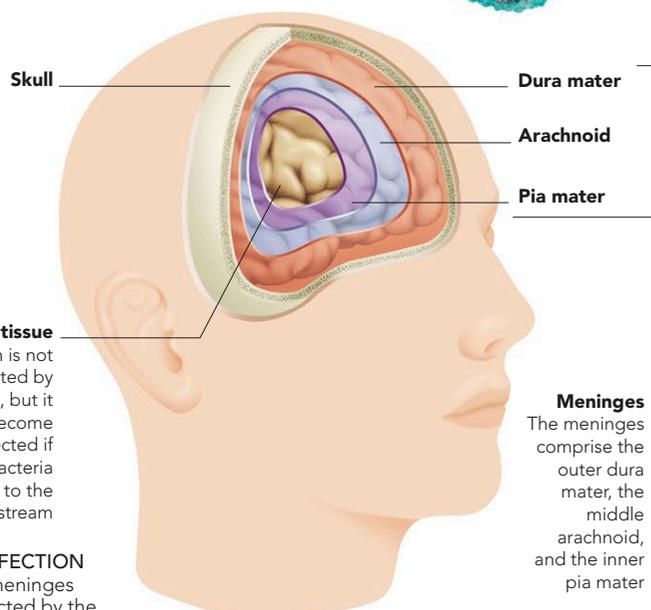
Bacterial meningitis is less common than the viral form, but is more serious and can be fatal. It may be caused by various bacteria but is usually due to infection with meningococcal or pneumococcal bacteria. Symptoms may develop rapidly, over only a few hours, and include fever, stiff neck, severe headache, nausea, vomiting, abnormal sensitivity to light, confusion, and drowsiness, and sometimes seizures and loss of consciousness.

In meningo-coccal meningitis, the bacteria may multiply in the blood, leading to a reddish purple rash that does not fade when pressed. If left untreated, bacterial meningitis can enter the cerebrospinal fluid, triggering an immune response that causes increased intracranial pressure, which in turn can cause brain damage.



ABSCESS DUE TO MENINGITIS

This color-enhanced MRI scan of a baby's brain shows a large abscess (pale orange at the upper left of the image) between the dura mater and arachnoid that has formed as a result of infection of the meninges.



Skull

Dura mater

Arachnoid

Pia mater

Brain tissue

The brain is not directly affected by meningitis, but it may become infected if meningitis bacteria spread to the bloodstream

Meninges

The meninges comprise the outer dura mater, the middle arachnoid, and the inner pia mater

SITES OF INFECTION

Usually the meninges become infected by the spread of bacteria or viruses (or rarely fungi) from elsewhere in the body. In some cases, infective bacteria may cause septicemia, which may affect the brain and other organs and may be fatal.

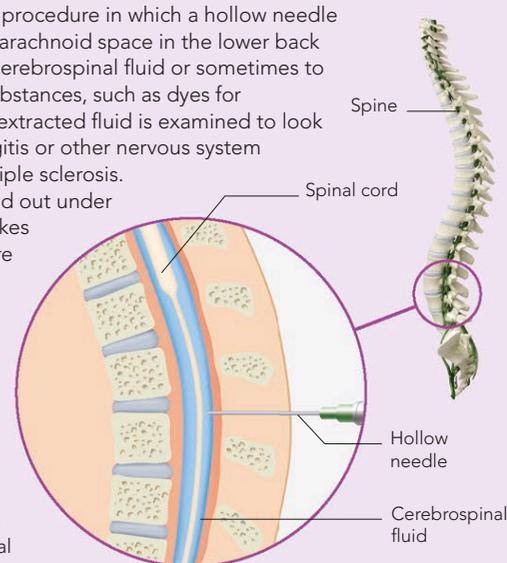
LUMBAR PUNCTURE

A lumbar puncture is a procedure in which a hollow needle is inserted into the subarachnoid space in the lower back to obtain a sample of cerebrospinal fluid or sometimes to inject drugs or other substances, such as dyes for specialized scans. The extracted fluid is examined to look for evidence of meningitis or other nervous system disorders, such as multiple sclerosis.

The procedure is carried out under local anesthesia and takes about 15 minutes. There are usually no after-effects except occasionally a headache.

THE PROCEDURE

A hollow needle is inserted between vertebrae in the lower spine into the subarachnoid space and a sample of cerebrospinal fluid is withdrawn.



ENCEPHALITIS

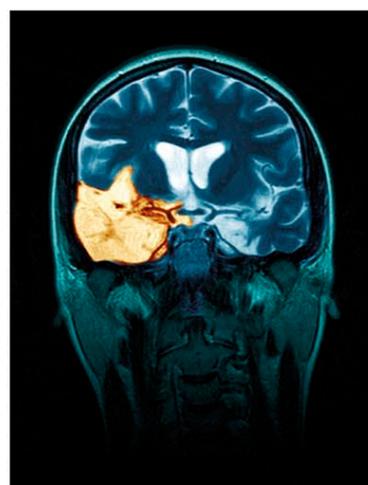
ENCEPHALITIS IS INFLAMMATION OF THE BRAIN. IT IS USUALLY DUE TO INFECTION BY A VIRUS OR MAY OCCUR AS A RESULT OF AN AUTOIMMUNE REACTION.

A rare condition, encephalitis varies in severity from a mild, barely noticeable illness to one that can be life-threatening.

Only certain viruses are able to gain access to the central nervous system and affect nerves, and therefore potentially cause encephalitis. These viruses include the herpes simplex virus (which also causes cold sores), chickenpox virus, and measles virus. Occasionally, the infection may also affect the meninges, causing meningitis. In most cases, the immune system deals with the viral infection before it can affect the brain. However, if the

immune system is compromised, there is a greater risk of developing encephalitis. When encephalitis develops, the infection causes swelling, and parts of the brain may be damaged when it is compressed against the skull. Rarely, encephalitis is due to an autoimmune reaction, in which the immune system attacks the brain, leading to inflammation and brain damage.

Mild encephalitis usually causes only a slight fever and headache. In more severe cases, there may also be nausea and vomiting; weakness, loss of coordination, or paralysis; abnormal sensitivity to light; loss or impairment of speech; memory loss; uncharacteristic behavior; stiff neck and back; drowsiness; confusion; seizures; and coma. In very severe cases, encephalitis can cause permanent brain damage and may even be fatal.



VIRAL ENCEPHALITIS

This color-enhanced MRI scan of a brain reveals a large area of abnormal tissue in the temporal lobe (the pale orange area) that is due to infection with the herpes simplex virus, one of the most common causes of viral encephalitis.

BRAIN ABSCESS

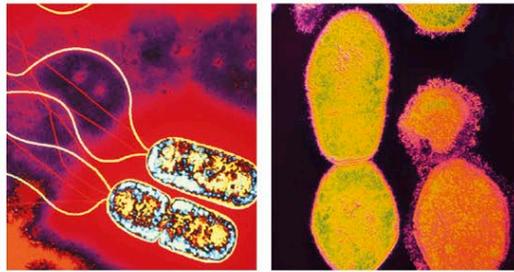
AN ABSCESS IS A COLLECTION OF PUS, SURROUNDED BY INFLAMED TISSUE, THAT CAN FORM IN THE BRAIN OR ON ITS SURFACES. THERE MAY BE SEVERAL AT ONCE.

A brain abscess can result from a bacterial or, more rarely, a fungal or parasitic infection. Fungal and parasitic infections are usually restricted to people whose immune systems have been impaired—for example, those with HIV/AIDS, people undergoing chemotherapy, or those taking immunosuppressants.

A brain abscess can occur as a result of a penetrating head injury or an infection spreading from elsewhere in the body, such as from a dental abscess, middle-ear infection, sinusitis, or pneumonia. It can also result from injecting drugs using a nonsterile needle.

Symptoms and effects

Once an abscess has formed, the tissue around it becomes inflamed, which may cause brain swelling and increased pressure in the skull. Symptoms may develop over a few days or weeks and depend on the area of the brain affected. Common general symptoms include: headache; fever; nausea and vomiting; stiff neck; drowsiness; confusion; and seizures. A person

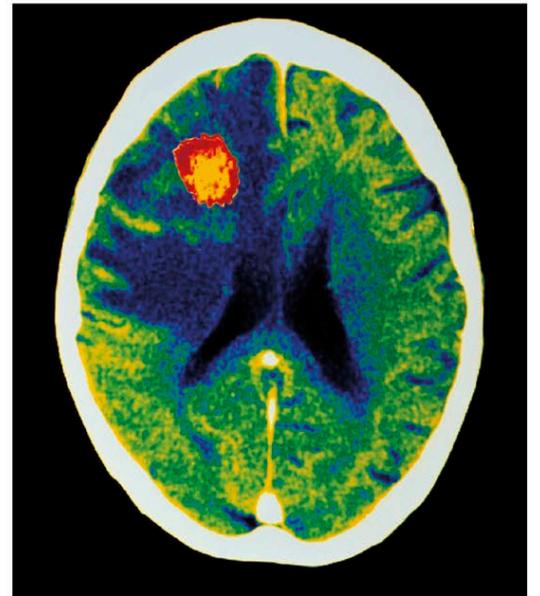


INFECTIOUS BACTERIA

A brain abscess may be caused by a wide variety of bacteria, including *Pseudomonas* (above left) and *Streptococcus* (above right), the most common cause.

may also experience speech difficulties, vision problems, and weakness of the limbs.

A brain abscess can be diagnosed by a scan and tests to identify the infecting organisms. Without treatment, an abscess can cause unconsciousness, and a coma (see p.238) may develop. It may also lead to permanent damage, and in some cases can be fatal. Drug treatment can eliminate the infection and reduce the swelling in the brain, but a craniotomy (a procedure to make a small opening in the skull) may be needed to drain pus from a large abscess.



ABSCESS IN BRAIN TISSUE

This color-enhanced CT scan shows a large abscess in the brain (orange area) of a person with AIDS. People who are immunocompromised, such as those with HIV/AIDS, are particularly vulnerable to abscesses.



TRANSIENT ISCHEMIC ATTACK

THIS IS AN EPISODE OF TEMPORARY LOSS OF BRAIN FUNCTION DUE TO AN INTERRUPTION OF THE BLOOD SUPPLY TO PART OF THE BRAIN.

Also called a “mini-stroke,” a transient ischemic attack (TIA) is most commonly caused by a blood clot that temporarily blocks an artery supplying blood to the brain. It can also occur due to excessive narrowing of an artery as a result of atherosclerosis (buildup of fatty deposits on the artery wall). There are numerous risk factors that

NARROWED CAROTID ARTERY

This X-ray shows an area of narrowing (circled) in the carotid artery in the neck. If an embolus temporarily lodges here, it may cause a TIA.

contribute to the likelihood of a TIA, such as diabetes mellitus, previous heart attacks, high blood-fat levels, high blood pressure, and smoking.

Symptoms usually develop suddenly and vary according to the part of the brain affected by the restricted blood flow, but they include visual disturbances or loss of vision in one eye, problems speaking or understanding speech, confusion, numbness, weakness or paralysis on one side of the body, loss of coordination, dizziness, and possibly brief unconsciousness. If symptoms last for more than 24 hours, the attack is classed as a stroke. Having had a TIA indicates increased risk of stroke.

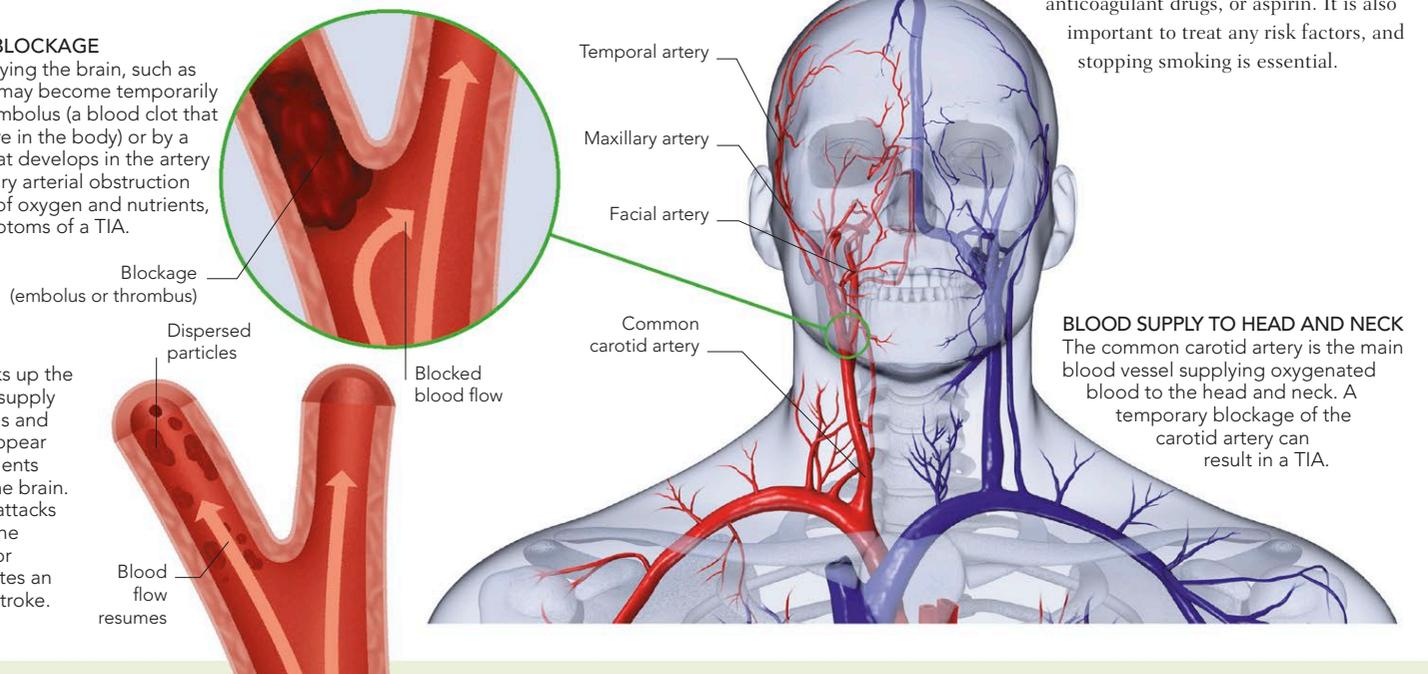
Treatment for TIA is aimed at preventing a stroke and includes endarterectomy (a procedure to remove the lining of an artery affected by atherosclerosis), anticoagulant drugs, or aspirin. It is also important to treat any risk factors, and stopping smoking is essential.

1 TEMPORARY BLOCKAGE

An artery supplying the brain, such as the carotid artery, may become temporarily obstructed by an embolus (a blood clot that originates elsewhere in the body) or by a thrombus (a clot that develops in the artery itself). The temporary arterial obstruction deprives the brain of oxygen and nutrients, producing the symptoms of a TIA.

2 DISPERSAL OF BLOCKAGE

As blood flow breaks up the obstruction, blood supply to the brain resumes and the symptoms disappear as oxygen and nutrients once again reach the brain. Transient ischemic attacks tend to recur, and the occurrence of one or more attacks indicates an increased risk of a stroke.

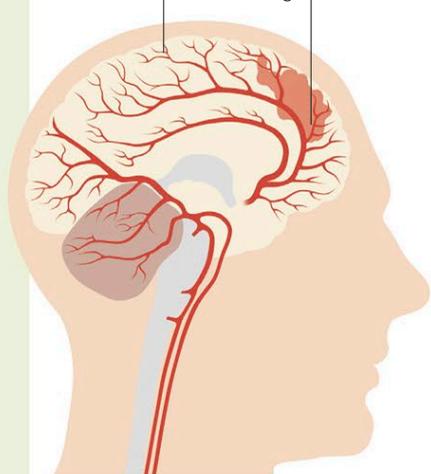


STROKE

DAMAGE TO PARTS OF THE BRAIN CAN OCCUR WHEN BLOOD SUPPLY TO THE BRAIN IS INTERRUPTED.

Interruption to the blood supply to the brain can occur as a result of a blockage of an artery in the brain (ischemic stroke), bleeding into the brain from a ruptured artery (hemorrhagic stroke), bleeding from a blood vessel in the brain (possibly from a ruptured aneurysm), or a subarachnoid hemorrhage (see below right). Risk factors

Blood vessel Hemorrhage



include age, high blood pressure, atherosclerosis, smoking, diabetes mellitus, heart-valve damage, previous or recent heart attack, high blood-fat levels, certain heart-rhythm disorders, and sickle cell disease.

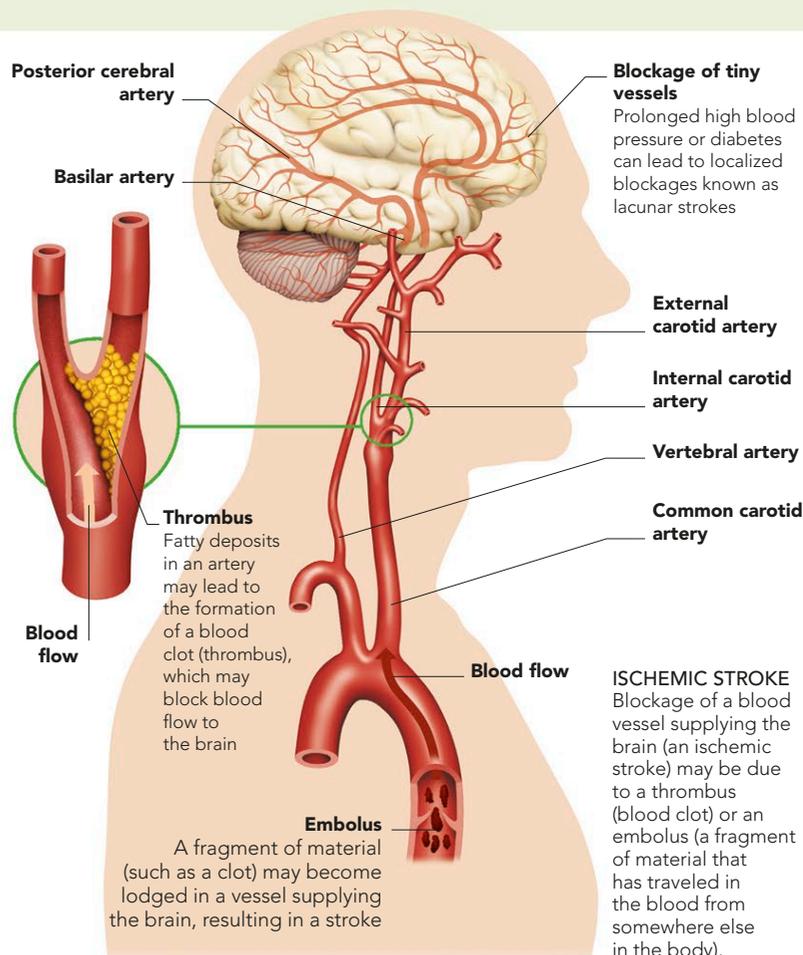
Symptoms and effects

Symptoms develop suddenly and vary depending on the brain areas affected, but can include sudden headache, numbness, weakness or paralysis, visual disturbances, problems speaking or understanding speech, confusion, loss of coordination, and dizziness. If severe, a stroke can cause loss of consciousness, coma, and death.

Treatment depends on the cause—strokes due to a clot require drugs and hemorrhagic strokes may require surgery. Nonfatal strokes can cause long-term disability or impairment of function, for which rehabilitative therapies (such as physical therapy and speech therapy) may be required.

HEMORRHAGIC STROKE

A hemorrhagic stroke is caused by bleeding into the brain from a ruptured blood vessel. High blood pressure is a significant risk factor because the increased pressure makes the vessels more likely to rupture.

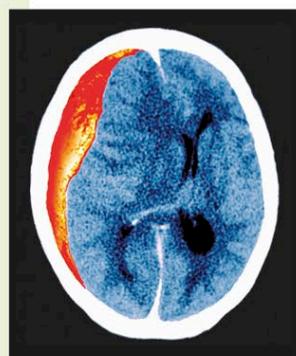


SUBDURAL HEMORRHAGE

A RUPTURED BLOOD VESSEL CAN CAUSE BLEEDING BETWEEN THE TWO OUTER MENINGES THAT SURROUND THE BRAIN.

The most common cause of a subdural hemorrhage is a head injury—it can occur from minor injuries, especially in the elderly.

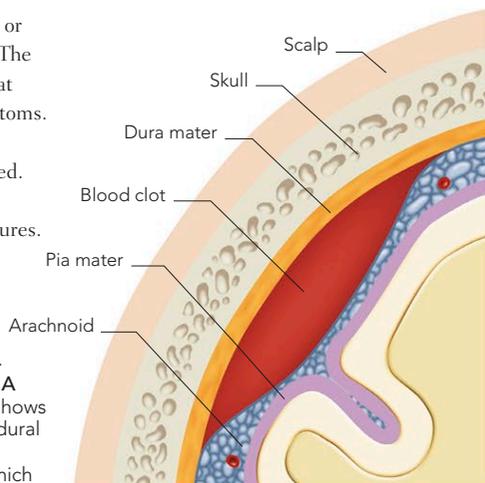
After the injury, bleeding may occur rapidly (within minutes) in the case of an acute subdural hemorrhage, or slowly over days or weeks for a chronic subdural hemorrhage. The trapped blood forms a clot in the skull that compresses brain tissue and causes symptoms. These are variable and may fluctuate depending on the area of the brain affected. They may include headache, one-sided paralysis, confusion, drowsiness, and seizures.



SUBDURAL HEMATOMA
A CT scan shows a large subdural hematoma (orange), which occurs when blood from a subdural hemorrhage forms a solid mass.

In severe cases, there may be unconsciousness and coma. The long-term outcome depends on the size and location of the hemorrhage. A severe subdural hemorrhage may be fatal.

A subdural hemorrhage is usually diagnosed with a brain scan (CT or MRI). An X-ray may be taken if skull fracture is suspected. A small hemorrhage may not need treatment and can clear up on its own, but usually surgery is needed.



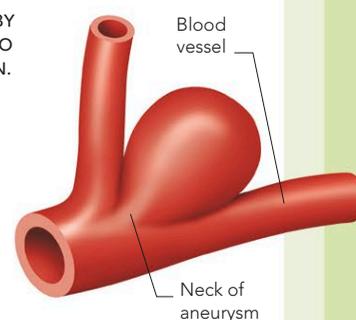
SITE OF SUBDURAL HEMORRHAGE

A subdural hemorrhage is bleeding into the space between the dura mater (outermost of the three meninges) and the arachnoid (the middle meninx).

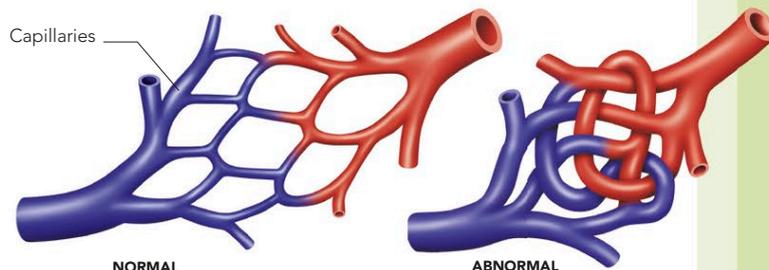
SUBARACHNOID HEMORRHAGE

A SUBARACHNOID HEMORRHAGE IS CAUSED BY BLEEDING INTO THE SPACE BETWEEN THE TWO INNER MEMBRANES SURROUNDING THE BRAIN.

This type of hemorrhage is most commonly caused by rupture of a berry aneurysm or, rarely, is due to the rupture of an arteriovenous malformation. High blood pressure is a significant risk factor. Symptoms occur suddenly, without warning, and often develop rapidly (over minutes). Some people recover completely, some are left with residual disability, and some die. Arteries in the brain may constrict to reduce blood loss, which can reduce blood supply to part of the brain and cause a stroke.



BERRY ANEURYSM
A berry aneurysm is a swelling that develops at a weak point in a blood vessel. It is usually present from birth.



ARTERIOVENOUS MALFORMATION

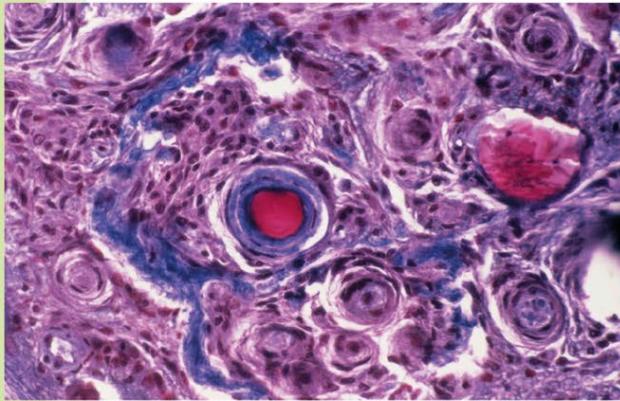
An abnormal knot of blood vessels on the brain's surface that is present from birth, an arteriovenous malformation is susceptible to rupture, causing a subarachnoid hemorrhage.

BRAIN TUMORS

BENIGN OR MALIGNANT GROWTHS CAN FORM IN THE BRAIN OR IN THE MEMBRANES AROUND THE BRAIN AND SPINAL CORD.

Primary brain tumors first develop in the brain itself and can be malignant or benign. They can arise in various types of brain cells and in any part of the brain, but primary tumors in adults are most common in the front two-thirds of the cerebral hemispheres.

Secondary tumors result from the spread of malignant cancer (metastasis) from elsewhere in the body, most commonly the lungs, skin, kidney, breast, or colon.



MENINGIOMA

This micrograph shows a section through a meningioma, a type of benign tumor that develops in the meninges, the membranes that cover the brain and spinal cord.

Several secondary tumors can develop simultaneously and the cause of most tumors is not known. Rarely, some tumors may be associated with certain genetic conditions.

A tumor compresses the surrounding brain tissue and raises pressure inside the skull. Symptoms therefore depend on the size and location of the tumor, but may include severe, persistent headaches; blurred vision or other sensory disturbances; speech

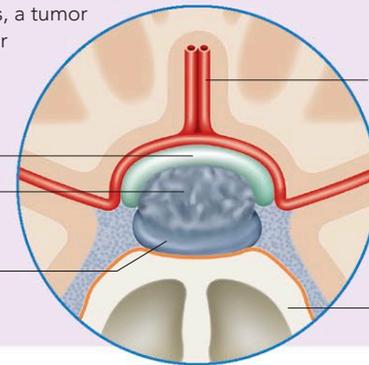
problems; dizziness; muscle weakness; poor coordination; impaired mental functioning; behavioral or personality changes; and seizures. If left untreated, a brain tumor may be fatal.

Brain tumors are diagnosed through brain scans and neurological tests. Treatment may involve a surgical removal (if possible), radiation therapy, and/or chemotherapy. Drugs to reduce the brain swelling may also sometimes be given.

PITUITARY TUMORS

The pituitary gland is a pea-sized structure that hangs from the base of the brain, connected by a stalk of nerve fibers to the hypothalamus just above it. Pituitary tumors are comparatively rare and usually benign. However, they can have a wide range of effects. A tumor may press on nearby nerves, particularly the optic nerve that passes directly above it, causing symptoms such as visual disturbances and headaches. In other cases, a tumor may cause underproduction or overproduction of hormones.

Compressed optic nerve
Pituitary tumor
Tumor presses on optic nerve above
Pituitary gland
May overproduce or underproduce hormones



Anterior cerebral artery

Pituitary gland

Skull

DEMENTIA

THIS DISORDER IS CHARACTERIZED BY A GENERALIZED DECLINE IN BRAIN FUNCTION, PRODUCING MEMORY PROBLEMS, CONFUSION, AND BEHAVIORAL CHANGES.

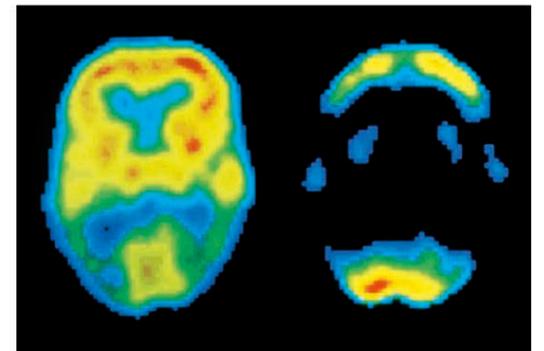
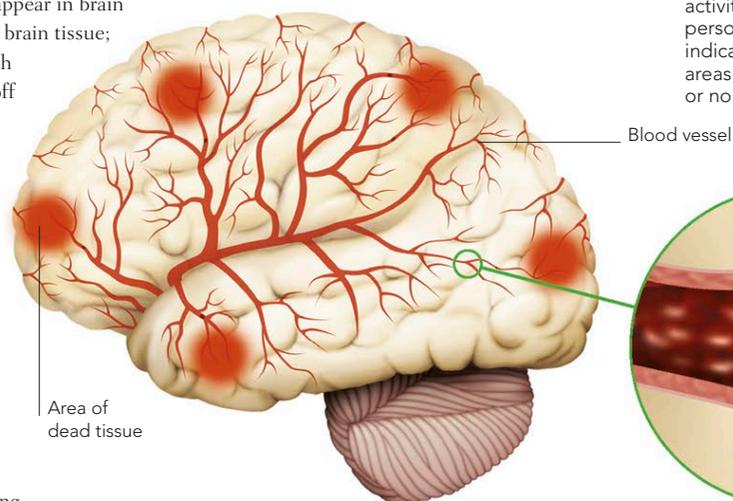
Dementia is caused by microscopic damage to brain tissue that leads to atrophy. It can be caused by various disorders, some covered on the following pages. Most commonly, it is due to Alzheimer's disease (see opposite page). Another common cause is vascular dementia, in which reduced or blocked blood supply causes death of brain cells. This can occur suddenly due to a stroke or gradually through a series of small strokes. Other causes include frontotemporal degeneration; Lewy body dementia, in which small, round structures appear in brain cells, leading to the degeneration of affected brain tissue; and neurological deterioration associated with conditions such as AIDS, Wernicke–Korsakoff syndrome, Creutzfeldt–Jakob disease (see opposite page), Parkinson's disease (see p.234), Huntington's disease (see p.234), head injury, brain tumors (see above), and encephalitis (see p.227). In rare cases, it may occur due to vitamin or hormone deficiency, or as a side effect of certain medications. Rarely, dementia may be caused by inherited genetic mutations.

Symptoms and effects

Dementia is characterized by progressive memory loss, confusion, and disorientation. It can also give rise to atypical or embarrassing

behavior, personality changes, paranoia, depression, delusions, unusual irritability, and anxiety. The affected person may make up explanations to account for memory gaps or strange behavior. As the condition progresses, a person with dementia may become indifferent toward other people and external events, as well as his or her own personal hygiene.

In rare cases, dementia may be due to a treatable cause, such as a side effect of medication or a vitamin deficiency, but usually there is no cure. Most forms are progressive, and a person may need total nursing care. Treatment with drugs may slow the deterioration of mental function and improve behavioral symptoms.



BRAIN ACTIVITY IN DEMENTIA

These two PET scans show the level of metabolic activity in a normal brain (left) and in the brain of a person with dementia (right), with yellow and red indicating areas of high activity, blue and purple areas of low activity, and black indicating minimal or no activity.

MULTI-INFARCT DEMENTIA

Vascular dementia can occur due to a series of blockages of blood vessels that supply the brain, usually due to clots. Each clot prevents oxygenated blood from reaching a small area of the brain, causing tissue death (infarct) in the affected area.

ALZHEIMER'S DISEASE

THE MOST COMMON CAUSE OF DEMENTIA, THIS IS A PROGRESSIVE DEGENERATIVE CONDITION IN WHICH PLAQUES CAUSE DAMAGE TO THE BRAIN.

Alzheimer's disease is rare before the age of 60, but increasingly common thereafter. Most cases occur without an identifiable cause. Mutations in several genes are associated with this disorder, however, and the genetic component is especially strong in the relatively rare cases of early onset disease (symptoms occurring before 60). In late-onset Alzheimer's disease, mutations in genes responsible

for the production of a blood protein called apolipoprotein E are implicated. These genes result in a protein (beta amyloid) being deposited in the brain as plaques, which leads to the death of neurons. Alzheimer's disease is also associated with reduced levels in the brain of the neurotransmitter acetylcholine. Additionally, it is thought that the disruption of the mechanism that controls the inflow of calcium ions into neurons may be involved, leading to excessive calcium in the neurons, which prevents them from receiving impulses from other brain neurons.

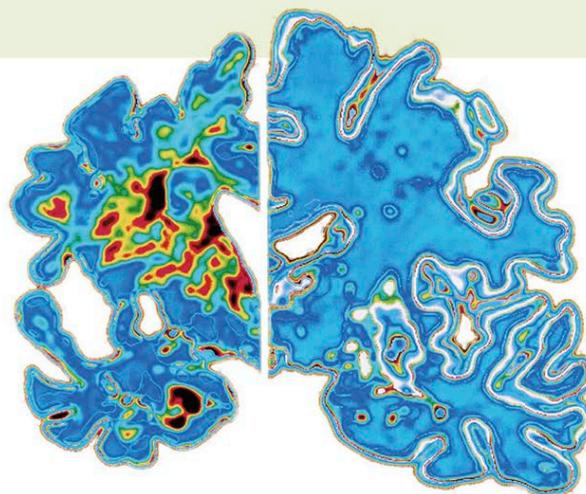
Symptoms may vary from one person to another, but typically Alzheimer's progresses through three stages (see panel, left). Alzheimer's disease is usually diagnosed from the symptoms, although brain scans, blood tests, and neuropsychological tests are also carried out.

Treatment

Treatment for this disorder is aimed at slowing down the degeneration, but it does not completely halt decline, and eventually complete nursing care is needed.

Acetylcholinesterase inhibitors may slow progress of Alzheimer's disease in the early and middle stages, and memantine in the later stages.

PROTEIN FILAMENTS Alzheimer's disease is often associated with the formation of tangled masses of protein filaments (shown in purified form in this micrograph), which may develop to form plaques.

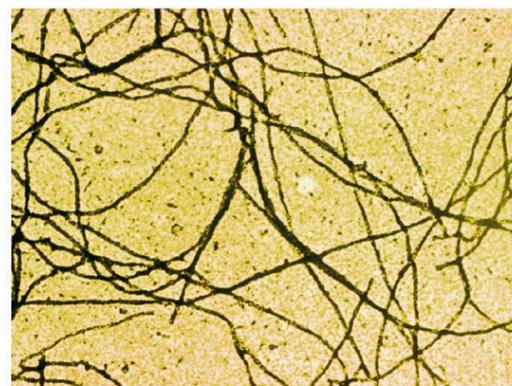


ALZHEIMER'S

HEALTHY BRAIN

ANATOMICAL CHANGES

These two vertical sections through the brain show the loss of brain tissue and increased surface folding in Alzheimer's disease (left) compared to a healthy brain (right).



STAGES OF ALZHEIMER'S DISEASE

The symptoms and progression of Alzheimer's disease vary from person to person. However, the symptoms become increasingly severe as the disease progresses and larger areas of the brain are damaged, although in some cases there may be periods in which the person seems to improve. Generally, there are three broad stages in the development of Alzheimer's disease.

STAGE	SYMPTOMS
Stage 1	The person becomes increasingly forgetful, and these memory problems may cause anxiety and depression. However, memory deterioration is a normal feature of aging and is not in itself evidence of Alzheimer's.
Stage 2	Severe memory loss, particularly for recent events, along with confusion about time and/or place; diminished concentration; aphasia (inability to find the right word); and anxiety, unstable moods, and personality changes.
Stage 3	In the third stage, confusion becomes very severe and there may be psychotic symptoms, such as delusions or hallucinations. There may also be abnormal reflexes and incontinence.

CREUTZFELDT-JAKOB DISEASE

DEMENTIA CAN BE CAUSED BY AN ABNORMAL PRION PROTEIN THAT ACCUMULATES IN THE BRAIN, CAUSING WIDESPREAD DESTRUCTION OF BRAIN TISSUE.

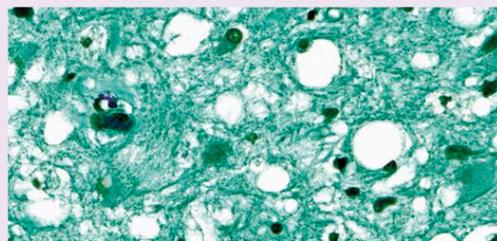
Prions are proteins that occur naturally in the brain, but their function is unknown. These proteins may become abnormally distorted, forming clusters in the brain and destroying brain tissue. This tissue destruction leaves holes in the brain, giving it a spongelike appearance, and results in various neurological dysfunctions, dementia,

and finally death. There are four main types of Creutzfeldt-Jakob disease: sporadic CJD; familial CJD; iatrogenic CJD; and variant CJD, which is caused by infection with bovine spongiform encephalopathy (BSE).

Initial symptoms include memory lapses, mood changes, and apathy. These may be followed by clumsiness, confusion, unsteadiness, and speech problems. Toward the final stages there may be uncontrollable muscle spasms, stiffness of the limbs, impaired vision, incontinence, progressive dementia, seizures, and paralysis. Eventually, CJD is fatal.

VARIANT CJD AND BSE

Creutzfeldt-Jakob disease, previously an obscure illness, came to public prominence in the 1990s when a few people in the UK developed a form of the disease—known as variant CJD (vCJD)—after eating meat from cattle infected with bovine spongiform encephalopathy (BSE), commonly known as “mad cow disease.” Initially it was thought that BSE was not transmissible to humans but this proved to be wrong, and stringent measures were introduced to prevent infected meat from entering the human food supply. As a result, the number of deaths in the UK from vCJD declined from a peak of 28 in 2000 to 1 in 2008.



BRAIN TISSUE IN CJD

This micrograph of brain cortex tissue from a person with variant CJD shows the characteristic spongelike appearance that is caused by the loss of neurons.

TYPES OF CJD

There are four main types of Creutzfeldt-Jakob disease (CJD). They are differentiated principally by the cause of the disease, although there are also other differences between them, such as the typical age of onset and the general length of illness.

TYPE OF CJD	CHARACTERISTICS
Sporadic CJD	Also known as classic or spontaneous CJD, this is the most common form of the disease. It mainly affects people over 50, and usually progresses rapidly (over a period of months).
Familial CJD	This is an inherited form of CJD, caused by a genetic mutation. It first appears between the ages of 20 and 60 and typically has a long course, generally between 2 and 10 years.
Iatrogenic CJD	This rare form of CJD is due to contamination with blood, tissue, or other substances from an infected person as a result of a medical procedure, such as brain surgery or certain hormone treatments.
Variant CJD (vCJD)	This type of CJD is acquired by eating meat contaminated with BSE. Typically, the disease lasts about a year before causing death. This type is rare, as there are measures to prevent infected meat from entering the food supply.

BRAIN SURGERY

SURGERY ON THE BRAIN IS A SPECIALIZED FIELD OF NEUROSURGERY IN WHICH OPERATIONS ON THE BRAIN OR MENINGES ARE CARRIED OUT THROUGH AN OPENING MADE IN THE SKULL (A CRANIOTOMY) OR, MORE RARELY, VIA THE NOSE AND NASAL CAVITY.

USES OF BRAIN SURGERY

Surgery may be used to treat various disorders. These include tumors of the brain or the meninges; raised pressure inside the skull due to a hemorrhage, hematoma, or hydrocephalus; traumatic brain injury, for example, due to a head wound; blood vessel abnormalities, such as aneurysms; and brain abscesses. Less commonly, surgery may be used to treat severe cases of epilepsy that have not responded to medication and to obtain biopsy samples. A highly experimental form of brain surgery known as deep-brain stimulation, which involves placing electrodes inside the brain, has been used to treat a few patients with movement disorders such as Parkinson's disease (see p.234) and Tourette's syndrome (see p.243).



STEREOTACTIC BRAIN SURGERY

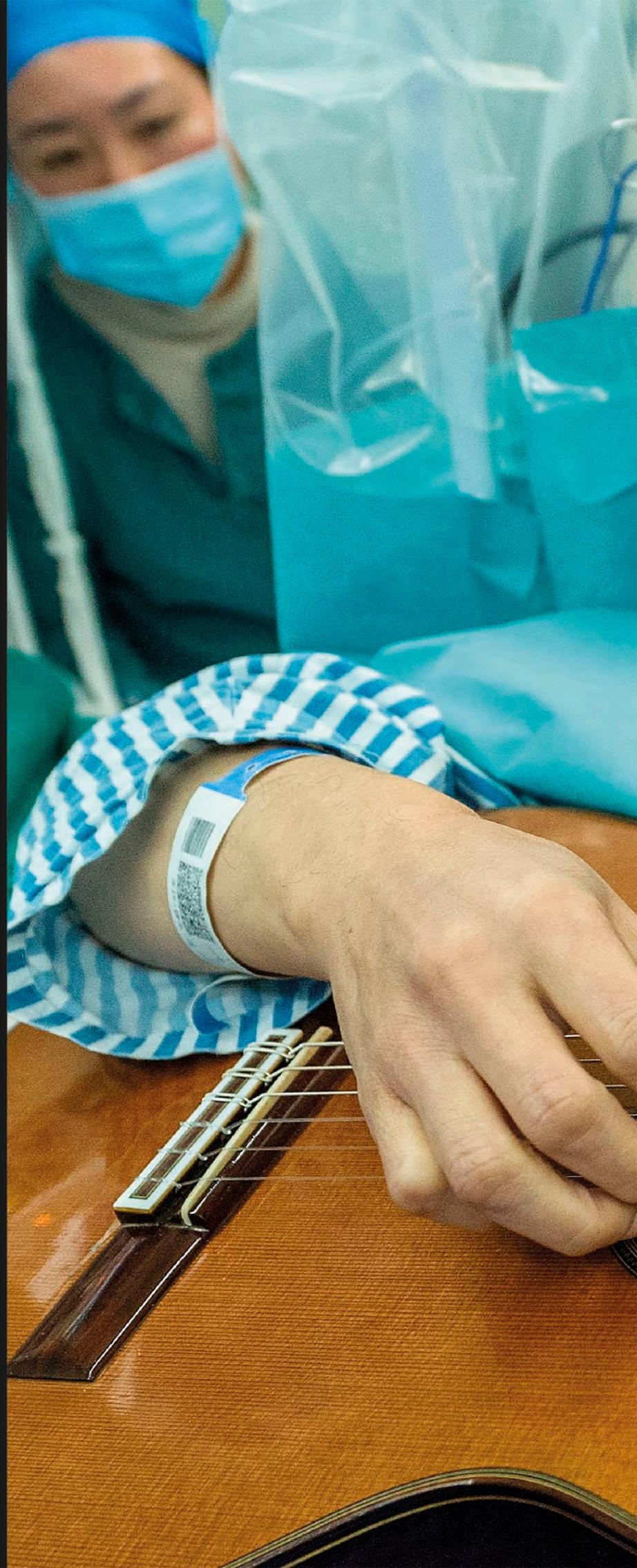
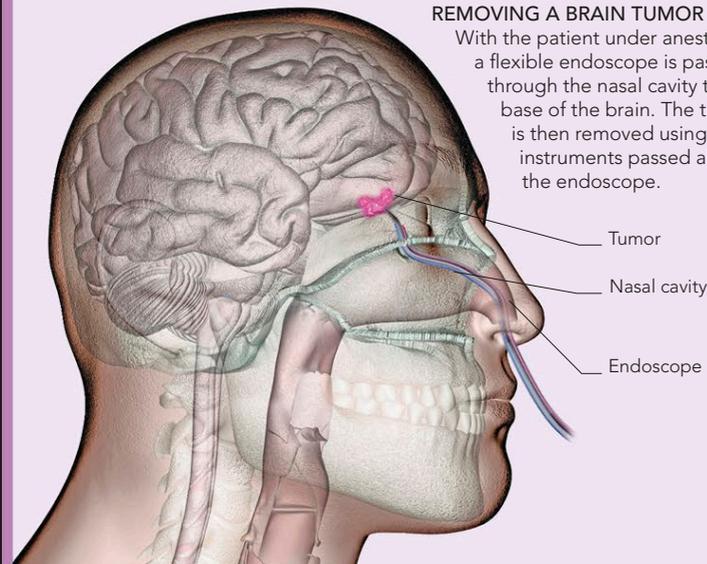
A patient about to undergo deep-brain stimulation first has a frame fixed to the scalp. The frame helps the surgeon navigate to the precise site in the brain where electrodes are to be implanted.

TRANSNASAL SURGERY

A minimally invasive procedure, transnasal surgery involves inserting an endoscope (viewing tube) through the nose to reach the base of the brain. The endoscope enables the surgeon to view the operation site, and instruments can be passed along it to perform surgical procedures. The main use of this type of brain surgery is to remove tumors of the pituitary gland or of the meninges at the base of the brain. It leaves no external scar, usually requires only a short hospital stay, and tends to cause less pain afterward than traditional surgery.

REMOVING A BRAIN TUMOR

With the patient under anesthesia, a flexible endoscope is passed through the nasal cavity to the base of the brain. The tumor is then removed using instruments passed along the endoscope.



DELICATE BRAIN SURGERY

This patient is playing the guitar while undergoing brain surgery. He is conscious so that his responses can be monitored, thereby ensuring that brain damage is avoided. This surgery involves deep-brain stimulation in which two thin, insulated electrodes are inserted into the brain.



PARKINSON'S DISEASE

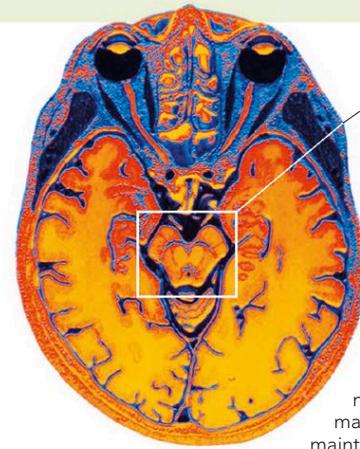
THIS IS A PROGRESSIVE BRAIN DISORDER THAT CAUSES TREMORS, MUSCLE RIGIDITY, PROBLEMS WITH MOVEMENT, AND DIFFICULTY KEEPING BALANCE.

Parkinson's disease is caused by degeneration of cells in the substantia nigra nuclei of the midbrain. These cells produce dopamine, a neurotransmitter that helps control muscles and movement. Damage to the cells reduces dopamine production, leading to the characteristic motor symptoms of Parkinson's disease.

In most cases, the underlying cause is not known, although in a very few cases, specific genetic mutations have been linked to Parkinson's disease.

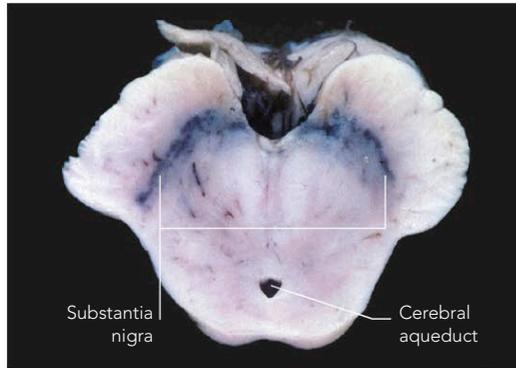
Symptoms usually develop gradually (over months or years), typically beginning with a tremor in a hand, arm, or leg that is worse when at rest. As the disease progresses, it becomes difficult to initiate voluntary movements; walking becomes a shuffling motion—it may be difficult to take the first step, and the normal arm swing when walking may be reduced or lost; muscles become rigid; handwriting becomes small and illegible; posture becomes stooped; and there may be loss of facial expression.

In the late stages, there may be problems speaking, swallowing may be difficult, and depression may occur. The intellect is usually unaffected, although dopamine depletion may cause symptoms of dementia.



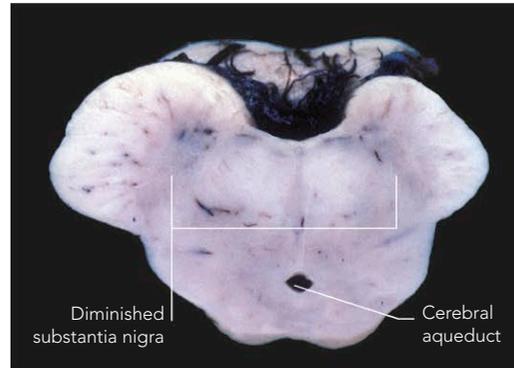
Location of substantia nigra

DEEP IN THE BRAIN
This color-enhanced MRI scan of a horizontal section through the head shows the location of the substantia nigra. A tiny electrode may be inserted here to maintain neuronal activity.



HEALTHY BRAIN

This section of brain tissue shows the substantia nigra in a healthy brain, with the dark pigmented areas of the substantia nigra clearly visible.



DISEASED BRAIN

In this section of brain tissue of a person with Parkinson's disease, the pigmented neurons in the substantia nigra are significantly reduced.

PARKINSONISM

The term "parkinsonism" refers to any condition that causes the movement abnormalities that occur in Parkinson's disease resulting from the reduced production of dopamine (for example, tremors, muscle stiffness, and slow movements). Parkinson's disease is the most common cause of parkinsonism, but not everybody with parkinsonism has Parkinson's disease. Other causes include stroke, encephalitis, meningitis, head injury, prolonged exposure to herbicides and pesticides, other degenerative nerve diseases, and certain drugs, such as some antipsychotic drugs.

HUNTINGTON'S DISEASE

HUNTINGTON'S IS A RARE, INHERITED DISEASE IN WHICH NEURONS IN THE BRAIN DEGENERATE, LEADING TO JERKY, UNCONTROLLED MOVEMENTS AND DEMENTIA.

The underlying cause of Huntington's disease is a single abnormal gene that occurs when a group of DNA base pairs is repeated many times. The faulty gene generates an abnormal version of Huntingtin protein, which then builds up in nerve cells and leads to the degeneration of neurons in the basal ganglia and cerebral cortex.

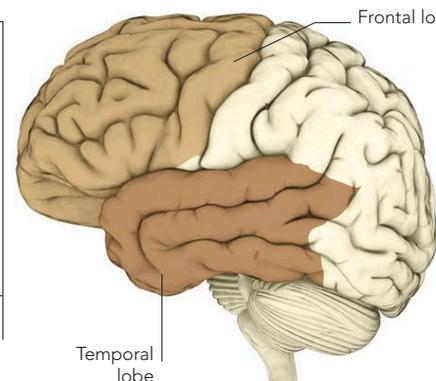
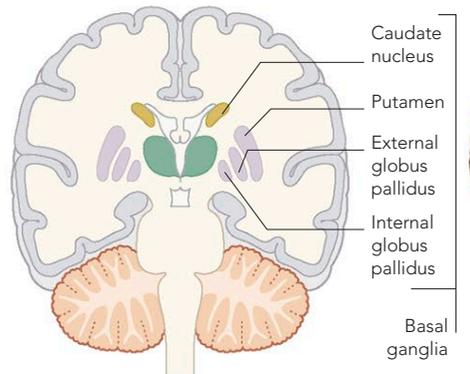
Effects

Symptoms usually start to appear between the ages of 35 and 50, although they may sometimes start in childhood. Early symptoms include chorea (jerky, rapid, uncontrollable movements), clumsiness, and involuntary facial grimaces and twitches. Other symptoms then develop, including speech problems; difficulty swallowing;

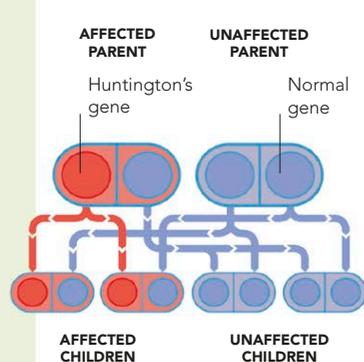
depression; apathy; and dementia, which usually takes the form of lack of concentration, memory problems, and personality and mood changes (including aggressive or antisocial behavior). The disease usually progresses slowly, eventually causing death some 10–30 years after symptoms first appear.

A diagnosis of Huntington's disease is made from the symptoms, with brain scans, and also genetic (to test for the abnormal gene) and neuropsychological testing.

There is no cure for Huntington's disease, and drug treatment is aimed at reducing the symptoms. Keeping physically and mentally active is also advised.

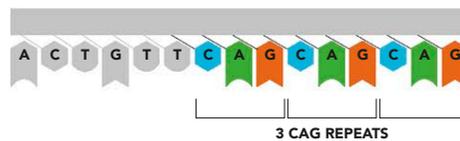


Affected areas
Huntington's disease causes degeneration of neurons in the basal ganglia (primarily in the caudate nuclei, putamen, and globus pallidus). It is also associated with degeneration in the frontal and temporal lobes.



INHERITANCE PATTERN

Huntington's disease is inherited in an autosomal dominant fashion, which means that if one parent has a copy of the gene, each child has a 1 in 2 chance of inheriting the faulty gene and therefore of developing the disease in adulthood.

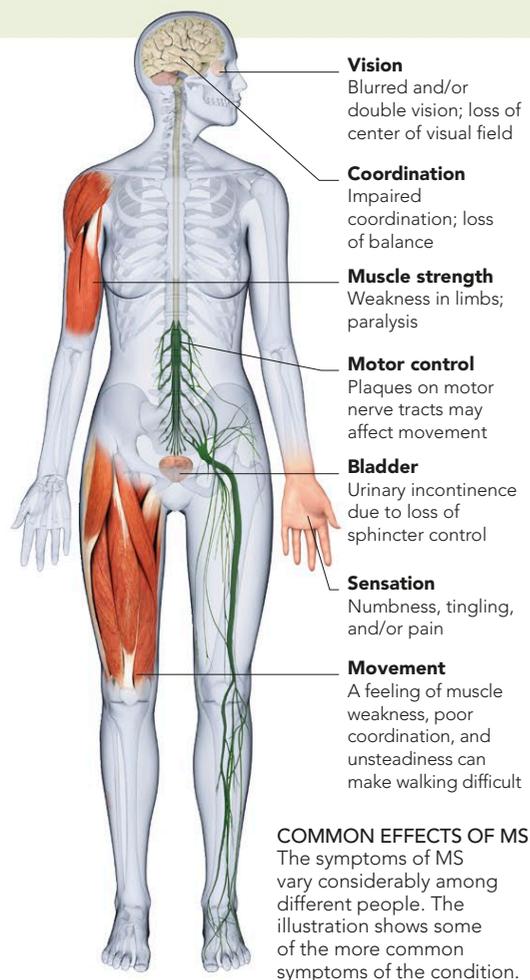


GENETIC DEFECT

The genetic abnormality that causes Huntington's disease is a sequence of DNA on chromosome 4 in which a group of base pairs (CAG) is repeated numerous times. Whether or not a person develops the disease depends on the number of CAG repeats (see table, right).

HUNTINGTON'S DISEASE AND CAG REPEATS

NUMBER OF REPEATS	EFFECTS
0–15	No adverse effect; Huntingtin protein functions normally.
16–39	Huntington's disease may or may not develop.
40–59	Huntingtin abnormal; Huntington's disease will eventually develop.
60 or more	Huntingtin abnormal; Huntington's disease will develop early.



COMMON EFFECTS OF MS
The symptoms of MS vary considerably among different people. The illustration shows some of the more common symptoms of the condition.

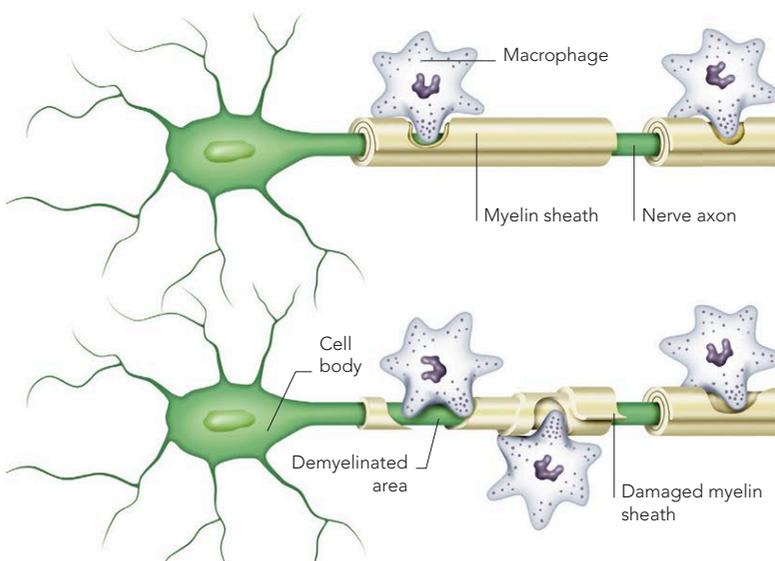
MULTIPLE SCLEROSIS

A PROGRESSIVE DISEASE, MULTIPLE SCLEROSIS CAUSES THE DESTRUCTION OF THE MYELIN SHEATHS THAT SURROUND NEURONS IN THE BRAIN AND SPINAL CORD.

Multiple sclerosis (MS) is thought to be an autoimmune disease in which the body's immune system destroys the cells that produce the myelin sheaths that surround and insulate neurons. Eventually hardened (sclerosed) plaques of scar tissue form over the demyelinated areas and the neurons themselves degenerate. The effect of these changes is to impair or block nerve impulses. The reason

for this autoimmune reaction is not known, although there may be genetic, environmental, or infectious factors involved.

The course and symptoms of MS vary among individuals. In addition to common symptoms (see illustration, left), there may also be mental changes, such as poor memory, anxiety, and depression. The most common type is relapsing-remitting MS, in which attacks (relapses) of gradually worsening symptoms are followed by periods of remission. In progressive MS, symptoms worsen without remission. In most cases, relapsing-remitting MS may develop into progressive MS.



EARLY STAGE

In the early stages of MS, the fatty myelin sheaths that surround the nerve axons are damaged. Macrophages, a type of white blood cell, remove the damaged areas, leading to demyelinated patches along the axons and impairing nerve conduction.

LATE STAGE

As the disorder progresses, there is an increasing amount of damage to the myelin sheaths and more nerves become affected, leading to a worsening of symptoms. Hardened (sclerosed) patches form over the demyelinated areas and eventually the nerve degenerates.

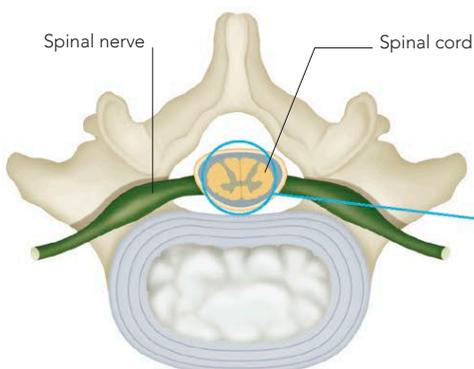
MOTOR NEURON DISEASE

IN THIS GROUP OF DISORDERS, PROGRESSIVE DEGENERATION OF MOTOR NEURONS LEADS TO INCREASING WEAKNESS AND WASTING OF MUSCLES.

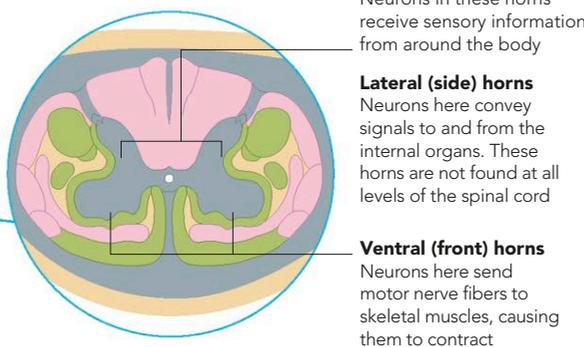
In most cases, the cause of motor neuron disease (MND) is not known. However, genetic factors are thought to be important in affecting a person's susceptibility to the condition. Some rare types of MND are inherited. Motor neuron disease can affect the upper motor neurons (those originating in the motor cortex or brain stem) and/or the lower motor neurons (those in the spinal cord

and brain stem that connect the central nervous system to the muscles). Damage to the upper motor neurons is indicated by spasticity, muscle weakness, and exaggerated reflexes. Damage to the lower motor neurons produces a weakening of muscles, paralysis, and atrophy of the skeletal muscles.

In addition to muscular symptoms, some people also experience personality changes and depression, but intellect, vision, and hearing remain unaffected. There are many types of motor neuron disease, the most common of which are amyotrophic lateral sclerosis (ALS, or Lou Gehrig's disease) and progressive bulbar atrophy. Both of these types affect the upper and lower motor neurons.

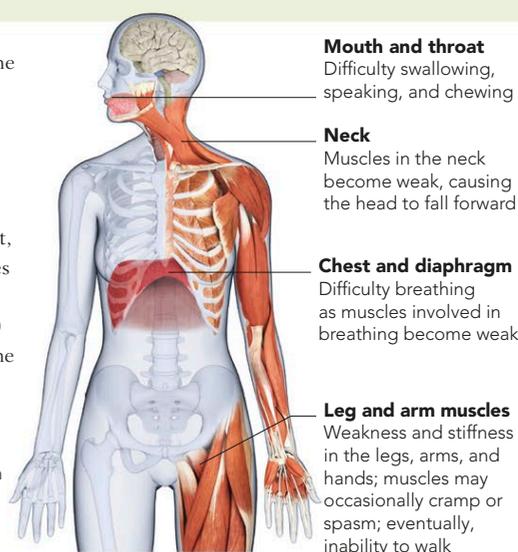


NERVE TRACTS OF THE SPINAL CORD
Nerve fibers in the spinal cord are grouped into bundles, or tracts, depending on the type and direction of nerve impulses they convey. MND may affect the lower motor neurons, in the ventral horns of the spinal cord.



ASCENDING TRACTS
These nerve fibers convey sensory signals from the body to the brain.

DESCENDING TRACTS
These convey motor signals from the brain to the skeletal muscles of the torso and limbs.



AFFECTED AREAS

The effects of MND depend on the specific form of the disease, and there is also some variation among different individuals. In almost all cases, the disease is progressive and ultimately fatal. The principal effects of the main types of the condition are shown here.



STEPHEN HAWKING

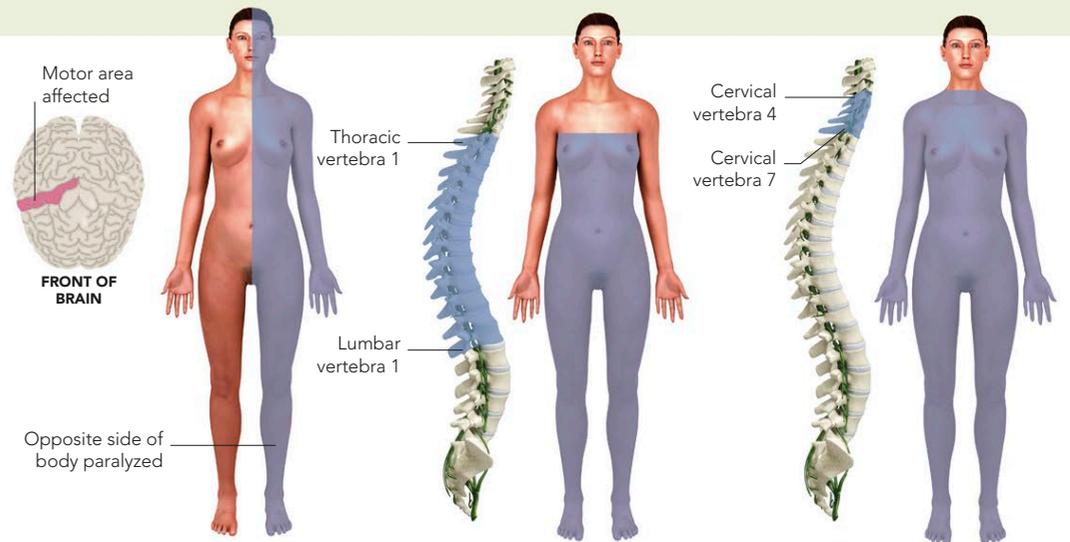
Renowned theoretical physicist and cosmologist Stephen Hawking died in 2018, aged 76. Few people with motor neuron disease have survived to such an age. Hawking retained his brilliant mind and worked practically until the end of his life.

PARALYSIS

PARTIAL OR COMPLETE LOSS OF CONTROLLED MOVEMENT DUE TO IMPAIRED MUSCLE FUNCTION MAY BE THE RESULT OF A NERVE OR MUSCLE DISORDER.

Paralysis can affect areas ranging from a single small muscle to most of the major muscles of the body. It is classified by the areas of body affected. Hemiplegia is paralysis of one-half of the body. Paraplegia is the paralysis of both legs and sometimes part of the trunk. Quadriplegia is paralysis of all four limbs and the trunk. Paralysis may also be classified as “flaccid” (causing floppiness) or “spastic” (causing rigidity).

Paralysis can be caused by any injury or disorder that affects the motor cortex or the motor nerve pathways that run from the motor cortex via the spinal cord and peripheral nerves to the muscles. It may also result from a muscle disorder or myasthenia gravis (a disorder affecting the junction between nerves and muscles). The affected area sometimes feels numb.



HEMIPLEGIA

Paralysis of one-half of the body may be caused by damage to the motor area of the brain on the opposite side.

PARAPLEGIA

Both legs and possibly part of the trunk may be paralyzed as a result of damage to the middle or lower spinal cord.

QUADRIPLEGIA

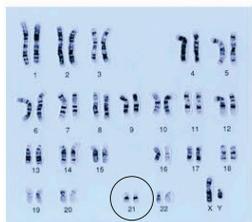
Damage to motor nerves in the lower neck causes quadriplegia. Damage higher in the neck is usually fatal.

DOWN SYNDROME

ALSO KNOWN AS TRISOMY 21, DOWN SYNDROME IS A CHROMOSOMAL ABNORMALITY THAT AFFECTS BOTH MENTAL AND PHYSICAL DEVELOPMENT.

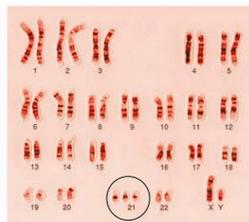
One of the most common chromosomal abnormalities, Down syndrome is usually the result of an extra copy of chromosome 21; affected people therefore have 47 chromosomes in all of their body cells, rather than 46. It may also result when part of chromosome 21 breaks off and attaches to another chromosome, a process called translocation, so that cells have the normal number of chromosomes but chromosome 21 is abnormally sized. Very rarely, Down syndrome may be the result of mosaicism, in which some body cells have 47 chromosomes and some have 46. Exactly how these abnormalities produce the characteristic mental and physical features of Down is not known.

In most cases, there is no identifiable reason for the chromosomal abnormality, although maternal age is a risk factor—after the early 30s, the risk of having a child with Down increases significantly. Paternal age can also be a risk factor, if the father is over 50. Parents who already have a child with Down or who have abnormalities of their own chromosome 21 have a higher risk of having a baby with Down syndrome.



NORMAL CHROMOSOME COMPLEMENT

This karyotype (a photograph of the full set of chromosomes) shows the chromosome complement of a normal male, comprising a total of 46 chromosomes: 22 pairs of autosomes plus one pair of sex chromosomes (X and Y).



TRISOMY 21
This karyotype shows the chromosome complement of a male with Down syndrome. There are three chromosome 21s (hence the term “trisomy 21”) instead of the normal two, resulting in the characteristic symptoms of Down syndrome.

Symptoms

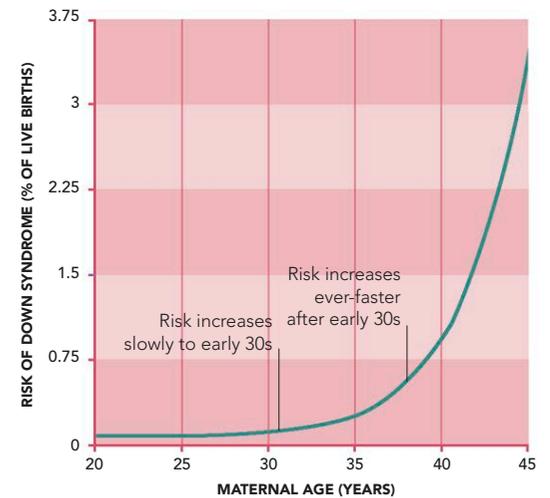
There is considerable variation in the severity of symptoms, but typically they include slow motor and language development and learning difficulties. Physical symptoms may include a small face with upward-sloping eyes; a flattened back of the head; a short neck; a large tongue; small hands with a single horizontal crease on the palm; and short stature.

There is also increased risk of various disorders, such as heart disease (often associated with congenital heart problems), hearing problems, underactivity of the thyroid gland, narrowing of the intestines, leukemia, and respiratory-tract and ear infections. Adults are at increased risk of eye problems such as cataracts. In older people, there is a heightened risk of Alzheimer’s disease. People with Down syndrome have lower than normal life expectancy, but some survive into old age.

Tests

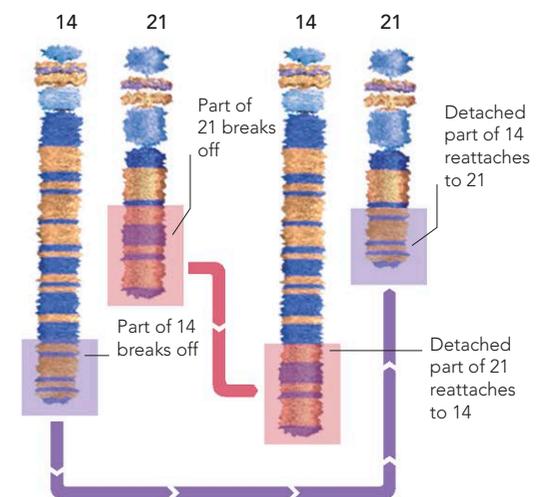
Pregnant women with a higher than normal risk of producing a child with Down syndrome may be offered amniocentesis—a diagnostic test that can detect chromosome abnormalities and other genetic disorders. It involves inserting a needle through the mother’s abdomen into the uterus and withdrawing a small amount of amniotic fluid. The fluid is then sent to a laboratory for analysis; reports usually take a few days. Amniocentesis is usually performed between 14 and 20 weeks’ gestation.

Although it is thought a safe procedure, it carries a small risk of miscarriage—roughly 1 in 300. Miscarriages can occur because of infection in the uterus, water breaking, or labor being induced prematurely. In very rare cases, the needle may come into contact with the baby. Great precautions are taken by using ultrasound to guide the needle away from the baby. The mother may experience a sharp pain when the needle enters the skin and again when it enters the uterus. She may also experience cramps after the procedure, or minor fluid leakage from the site. Having amniocentesis provides parents with the chance to pursue interventions—such as fetal surgery for spina bifida—and to plan, if necessary, for having a child with special needs. It also gives women the opportunity to opt for an abortion if they do not want to carry the child to term.



MATERNAL AGE AND DOWN SYNDROME

The risk of having a child with Down syndrome is related to the mother’s age—increasing slowly up to the early 30s, and then at an ever-faster rate with increasing maternal age.



BALANCED TRANSLOCATION

Down syndrome may be caused by a translocation, in which part of chromosome 21 breaks off and reattaches to another chromosome. A balanced translocation occurs when part of the other chromosome in turn moves to chromosome 21.

TYPES OF CEREBRAL PALSY

Cerebral palsy can be classified into four main types, primarily on the basis of the type of movement abnormality, although there may also be other symptoms.

TYPE	CHARACTERISTICS
Spastic cerebral palsy	Exaggerated reflexes; stiff, difficult movement due to tight, stiff, and weak muscles.
Athetoid cerebral palsy	Involuntary writhing movements, especially in the face, arms, and trunk; difficulty maintaining posture.
Ataxic cerebral palsy	Problems maintaining balance; shaky movements of the hands and feet; and speech difficulties.
Mixed cerebral palsy	A combination of symptoms from the other types; often tight muscle tone and involuntary movements.

CEREBRAL PALSY

CEREBRAL PALSY REFERS TO A GROUP OF DISORDERS THAT AFFECT MOVEMENT AND POSTURE DUE TO BRAIN DAMAGE OR THE FAILURE OF THE BRAIN TO DEVELOP PROPERLY.

There are many possible causes of cerebral palsy, and often the cause is not identified. Usually, the brain damage occurs before or around birth. Possible causes include extreme prematurity; lack of oxygen to the fetus before or during birth (hypoxia); hydrocephalus (see below); infections transmitted from the mother to the fetus; or hemolytic disease, which is caused by a blood incompatibility between the mother and the fetus. After birth, infections such as encephalitis and meningitis, head injury, or a brain hemorrhage may cause cerebral palsy.

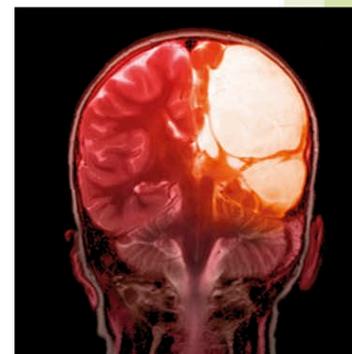
In addition to movement and posture abnormalities and the difficulties that these can cause (such as difficulty walking, talking, and eating), cerebral palsy may also give rise to various other problems, such as vision and hearing impairment and epilepsy. It may also sometimes cause

learning difficulties. The severity of symptoms varies widely among different people, from slight clumsiness to severe disability.

There is no cure for cerebral palsy, but treatment includes physical therapy, occupational therapy, and speech therapy. Drugs may be used to control muscle spasms and increase joint mobility. Surgery may help correct any deformities that have developed as a result of abnormal muscle development. Cerebral palsy is not progressive.

BRAIN DAMAGE

This MRI scan shows the head of a child with cerebral palsy. The abnormal brain tissue (in the left side of the brain, but seen on the right of this image) has resulted in paralysis of the right side of the body.



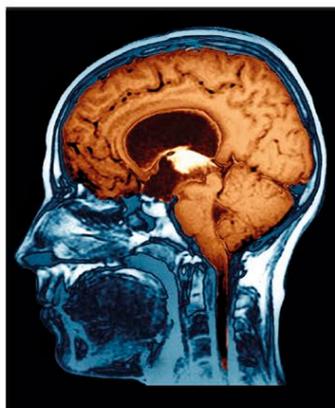
HYDROCEPHALUS

COMMONLY KNOWN AS WATER ON THE BRAIN, HYDROCEPHALUS IS AN EXCESSIVE BUILDUP OF CEREBROSPINAL FLUID WITHIN THE SKULL.

Hydrocephalus occurs either because excess cerebrospinal fluid is produced or because the fluid does not drain away normally. The fluid accumulates in the skull and compresses the brain, which may lead to brain damage.

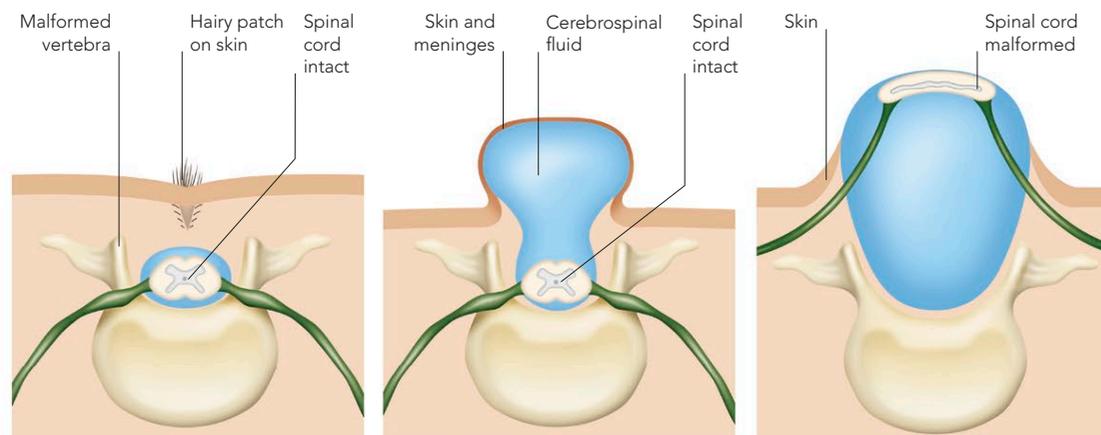
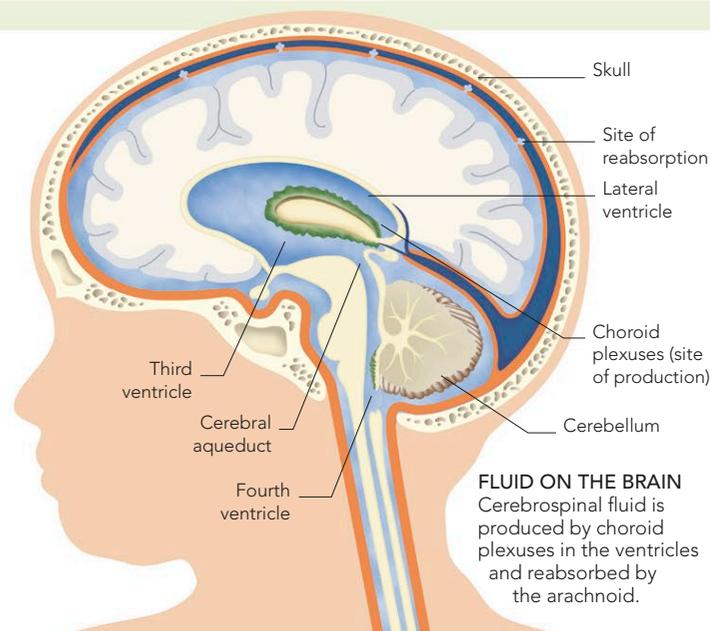
This condition can be present at birth, often in association with other abnormalities, such as a neural-tube defect. The main symptom is an abnormally large head that continues to grow rapidly. Without treatment, severe brain damage may occur, which may lead to cerebral palsy or other physical or mental disabilities, or may even be fatal.

Hydrocephalus may occur later in life, as a result of a head injury, brain hemorrhage, infection, or a brain tumor. It usually clears up once the cause is treated.



ENLARGED VENTRICLES

In this MRI scan through the center of the head, the ventricles (black areas in the middle of the brain) are enlarged due to hydrocephalus. This abnormal accumulation of cerebrospinal fluid has compressed the brain.



SPINA BIFIDA OCCULTA

In spina bifida occulta, the only defect is malformation of one or more vertebrae; the spinal cord is undamaged. There may be a hair tuft, dimpling, or a fatty lump at the base of the spine.

MENINGOCELE

In meningocele, the meninges protrude through the malformed vertebra, forming a sac filled with cerebrospinal fluid, which is called a meningocele. With this type of defect the spinal cord is not damaged.

MYELOMENINGOCELE

This is the most severe form of spina bifida, in which the spinal cord is malformed and, contained within a sac of cerebrospinal fluid, protrudes through a defect in the skin.

NEURAL-TUBE DEFECTS

A NUMBER OF DEVELOPMENTAL ABNORMALITIES OF THE BRAIN OR SPINAL CORD CAN OCCUR WHEN THE NEURAL TUBE DOES NOT FORM PROPERLY.

The neural tube is the region along the back of an embryo that develops into the brain, spinal cord, and meninges. The cause of neural-tube defects is unknown, but they tend to run in families and have been associated with certain anticonvulsant drugs during pregnancy. A lack of folic acid during early pregnancy is also associated with the defects.

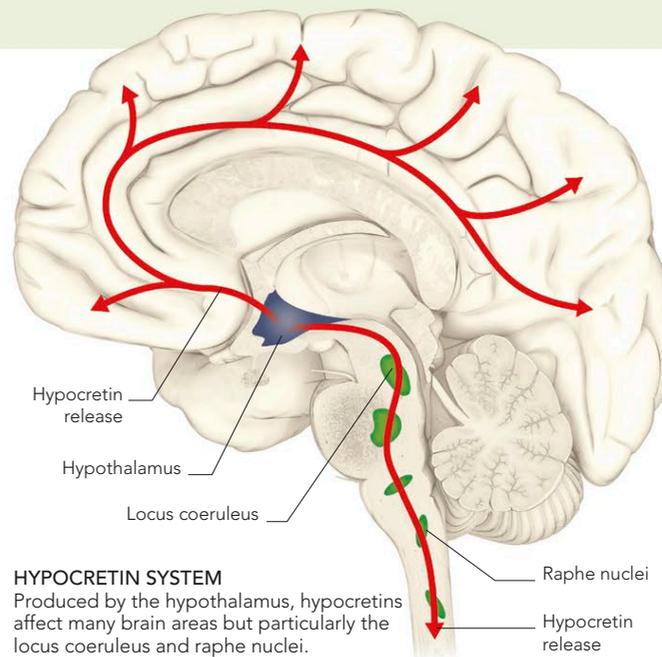
The most common types are anencephaly and spina bifida. In anencephaly there is a complete lack of a brain, which is always fatal. In spina bifida the vertebrae do not close completely around the spinal cord. In the most severe form of spina bifida, called myelomeningocele, the spinal cord is malformed and there may be paralysis of the legs and loss of bladder control.

NARCOLEPSY

THIS IS A NEUROLOGICAL DISORDER CHARACTERIZED BY CHRONIC DROWSINESS AND RECURRENT, SUDDEN EPISODES OF SLEEP THROUGHOUT THE DAYTIME.

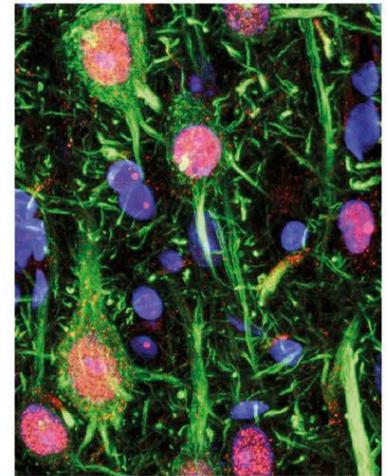
This condition is thought to be due to abnormally low levels of proteins called hypocretins (also known as orexins) in the brain. Hypocretins are produced by cells in the hypothalamus and help regulate sleep and wakefulness. In people with narcolepsy, these cells are damaged. The underlying cause of the damage is not known, but it may be due to an autoimmune response, possibly triggered by an infection. A genetic factor may be involved, as the condition tends to run in families.

The main symptoms are overwhelming drowsiness and an uncontrollable urge to sleep—people with narcolepsy may fall asleep without warning at any time and place. Other common symptoms include a sudden loss of muscle tone (cataplexy) while awake and hallucinations at the start or end of sleep.



HYPOCRETIN SYSTEM

Produced by the hypothalamus, hypocretins affect many brain areas but particularly the locus coeruleus and raphe nuclei.



HYPOCRETIN RECEPTORS

This light micrograph of brain tissue shows a large number of neurons with hypocretin receptors (colored red).

COMA

A STATE OF UNCONSCIOUSNESS IN WHICH THERE IS A LACK OF RESPONSIVENESS TO INTERNAL AND EXTERNAL STIMULI IS CALLED A COMA.

Coma results from damage or disturbance to parts of the brain involved in maintaining consciousness or conscious activity, especially the limbic system and the brainstem. A wide range of problems can cause a coma, including head injury; lack of blood supply to the brain, as may occur after a heart attack or stroke; infections, such as encephalitis and meningitis; toxins, such as carbon monoxide or drug overdoses; and prolonged high or low blood-sugar levels, as can occur in diabetes mellitus.

Symptoms

There are varying degrees of coma. In less severe forms, the person may respond to certain stimuli and spontaneously make small movements. In the condition known as a persistent vegetative state there may be sleep-wake cycles, movements of the eyes and limbs,

and even speech, although the person does not appear to respond to any stimuli. In a deep coma, the person does not respond to any stimuli nor make any movements, although automatic responses such as blinking and breathing may be maintained. In severe cases, in which the lower brainstem is damaged, vital functions such as

breathing are impaired or lost and life support is necessary. Total and irreversible loss of brainstem function is classed as brain death.

Coma is diagnosed when a person remains persistently unconscious and unresponsive to stimuli. It is an emergency and requires immediate treatment.

1 Conscious Normal responses to stimuli such as sound, light, pain, and orientation (prompt response to questions about name, date, time, and/or location).

2 Confused The person is aware but bewildered and disoriented (does not respond promptly to questions about name, date, time, and/or location).

3 Delirious The person is disoriented, restless, or agitated, and shows a marked impairment of attention; there may be hallucinations or delusions.

4 Obtunded The person is sleepy, shows a marked lack of interest in the surroundings, and responds very slowly to stimuli.

5 Stuporous A sleeplike state with little or no spontaneous activity; typically, a person responds only to painful stimuli, by moving away or grimacing.

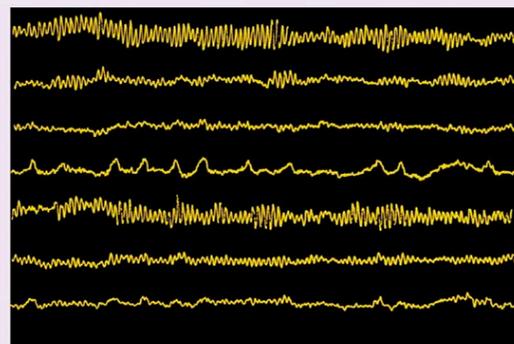
6 Comatose The person cannot be woken and does not respond to any stimuli, even painful ones; there is no gag reflex, and the pupils may not respond to light.

LEVELS OF CONSCIOUSNESS

There are various systems used to classify levels of consciousness, one of which is outlined here. The depth of a coma may also be assessed using a scale, most commonly the Glasgow Coma Scale.

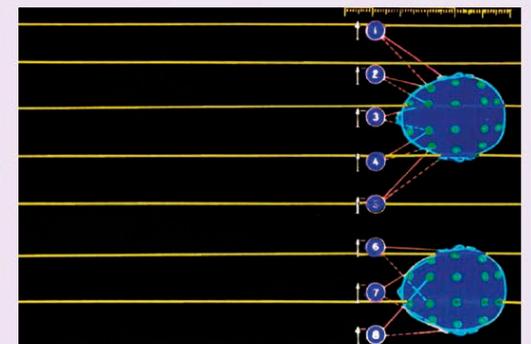
BRAIN DEATH

Brain death is the irreversible cessation of functions of the brain and particularly the brainstem. The brainstem is responsible for maintaining vital functions such as breathing and heartbeat. If there is no activity in the brainstem and it is damaged so severely and irreversibly that these vital functions cannot be carried out independently without a life-support machine, a person may be diagnosed as brain dead. To confirm the diagnosis, a series of tests are carried out by two experienced physicians. These tests include checking responses to stimuli, checking functions controlled by the brainstem, and testing the ability to breathe without life support. Only if the two physicians are in agreement that brainstem and brain functions have been irreversibly lost is the diagnosis of brain death confirmed.



NORMAL EEG

Brain activity can be assessed by electroencephalography (EEG), in which electrodes are attached to the scalp and connected to a machine that records the levels of electrical activity in the brain.



NO ACTIVITY

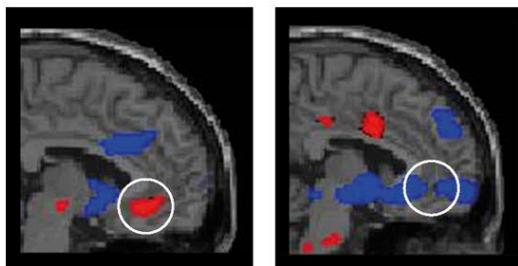
Electroencephalography can be used to help diagnose brain death. If the EEG lines are flat, as in the recording above, it indicates that there is no activity in the brain, which is one of the criteria used to diagnose brain death.

DEPRESSION

DEPRESSION IS CHARACTERIZED BY PERSISTENT FEELINGS OF INTENSE SADNESS, HOPELESSNESS, AND LOSS OF INTEREST IN LIFE THAT INTERFERE WITH EVERYDAY LIFE.

In many cases, depression occurs without an obvious cause. A number of factors may trigger it, such as a physical illness; hormonal disorders or the hormonal changes during pregnancy (prenatal depression) or after childbirth (postpartum depression); or distressing life events, such as a bereavement. It may also occur as a side effect of certain drugs, such as oral contraceptives. Depression is more common in women, it tends to run in families, and various genetic mutations are associated with this disorder.

Various biological abnormalities have been found in the brains of depressed people, such as decreased levels of the neurotransmitter serotonin, raised levels of the enzyme monoamine oxidase, loss of cells from the hippocampus (an area of the brain involved in mood and memory), and abnormal patterns of neural activity in the amygdala and parts of the prefrontal cortex. However, the mechanisms by which such biological abnormalities may lead to depression are not known.



DEEP BRAIN STIMULATION

In the PET scan on the left, a patient suffering from depression shows overactivity in the cingulate cortex (circled). After six months of deep brain stimulation, activity in this area (shown in the scan to the right) decreased and symptoms had improved.

SEASONAL AFFECTIVE DISORDER

Commonly known as SAD, seasonal affective disorder is a type of depression in which mood changes occur according to the season. The cause is not known, although it is thought that changes in daylight levels may cause alterations in brain chemistry that affect mood. Typically, the onset of winter brings depression, fatigue, lack of energy, cravings for sugary and starchy food, weight gain, anxiety and irritability, and avoidance of social activities. The symptoms then spontaneously clear up with the coming of spring. SAD can usually be treated with daily light therapy (sitting in front of a special light box that produces bright light similar to daylight) or antidepressants.



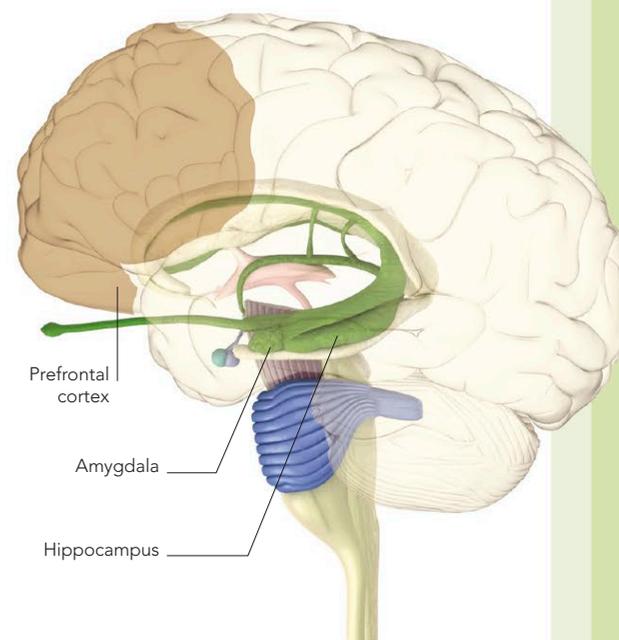
Symptoms and treatment

There is considerable variation among different people in the symptoms and in their severity. Most people experience several of the following: feeling unhappy most of the time; loss of interest and enjoyment in life; difficulty coping and making decisions; impaired concentration; persistent fatigue; agitation; changes in appetite and weight; disrupted sleeping patterns; loss of interest in sex; loss of self-confidence; irritability; and thoughts of, or attempts at, suicide. In some people, episodes of depression alternate with periods of extreme highs (manic episodes); this is known as bipolar disorder (see below).

Usually depression is treated with a talking therapy, antidepressant drugs, or both. Experimental treatment using deep brain stimulation (where implanted electrodes stimulate areas of the brain) is also being studied.

BRAIN AREAS

The biological basis of depression is not fully understood but several areas of the brain are thought to be involved, including the prefrontal cortex, hippocampus, and amygdala.



BIPOLAR DISORDER

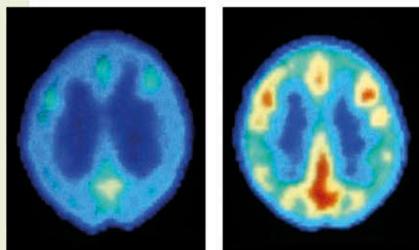
BIPOLAR DISORDER IS A MOOD DISORDER CHARACTERIZED BY MOOD SWINGS BETWEEN DEPRESSION AND MANIA.

The exact cause of bipolar disorder (sometimes called manic-depressive illness) is not known, although it is believed that it results from a combination of biochemical, genetic, and environmental factors. The levels

of certain neurotransmitters in the brain, such as norepinephrine, serotonin, and dopamine, may play a role. Bipolar disorder tends to run in families and has a strong genetic component. However, environmental factors, such as a major life event, may act as triggers.

Symptoms

Typically, symptoms of depression and mania alternate, with each episode lasting for an unpredictable period. Between mood swings, a person's mood and behavior are often normal. Symptoms of a depressive episode may include feelings of hopelessness, disturbed sleep, changes in appetite and weight, fatigue, a loss of interest in life, and a loss of self-confidence; there may also be suicide attempts. Symptoms of a manic episode may include extreme optimism, increased energy levels, drive and activity, inflated self-esteem, racing thoughts, and risk-taking behavior.

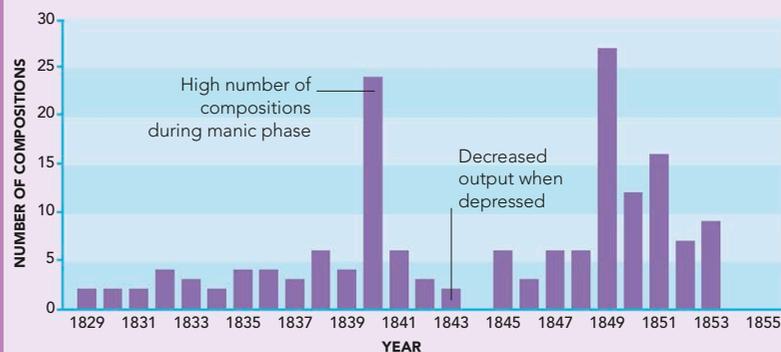


BRAIN ACTIVITY IN BIPOLAR DISORDER

These PET scans show brain activity during normal periods (left) and increased levels of activity during a manic phase (right).

CREATIVITY AND BIPOLAR DISORDER

Biographical studies suggest that bipolar disorder may be more common among accomplished artists than in the general population, and some artists seem to be able to utilize periods of mania as a spur to creativity. For example, the musical output of the German composer Robert Schumann (1810–56)—illustrated on the graph below—shows a link between his bouts of mania and the number of compositions he produced. He was most productive during manic phases and least productive when depressed. However, the quality of his work was not affected by his moods.



ANXIETY DISORDERS

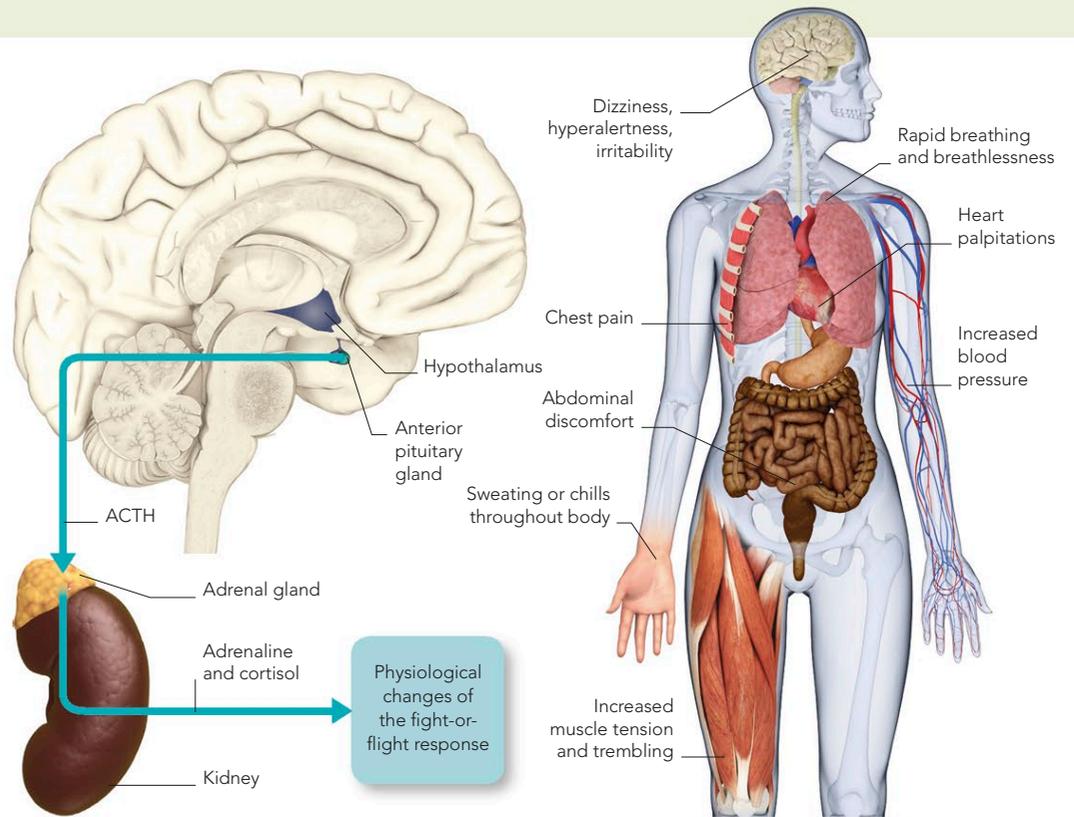
THIS IS A GROUP OF DISORDERS IN WHICH FEELINGS OF ANXIETY AND/OR PANIC OCCUR FREQUENTLY ENOUGH TO CAUSE PROBLEMS IN COPING WITH EVERYDAY LIFE.

Temporary feelings of nervousness, apprehension, and even panic in stressful situations are normal and appropriate. However, when these anxiety reactions occur frequently in ordinary situations and disrupt normal activities, it is considered to be a disorder.

In a few cases there may be an identifiable physical cause for persistent anxiety, such as a thyroid disorder or substance abuse, and sometimes generalized anxiety may develop after a stressful life event, such as a bereavement. In most cases the cause is not known, although a family history of an anxiety disorder increases the risk of developing one. The brain mechanisms underlying anxiety disorders are also unknown, although disruption of neurotransmitters in the frontal lobes or limbic system may be involved.

Whatever the underlying cause, the effect is to disrupt the body's normal control of its stress response—the “fight or flight” response. With anxiety disorders either the stress response fails to turn off or the stress response becomes activated at inappropriate times.

There are several forms of anxiety disorder. The most common is generalized anxiety disorder, which is characterized by excessive, inappropriate worrying that lasts for at least six months. Another form of anxiety disorder is panic disorder, in which there are sudden, unexpected attacks of intense anxiety or fear.



STRESS RESPONSE

In response to stress, the hypothalamus stimulates the pituitary gland to produce adrenocorticotropic hormone (ACTH). ACTH stimulates production of epinephrine and cortisol by the adrenal glands, and these hormones produce the fight-or-flight response.

PHYSICAL EFFECTS OF ANXIETY

Activation of the body's flight-or-flight stress response produces widespread effects on the body. Normally, this response turns off when the stress disappears, but in anxiety disorders the stress response may be oversensitive or may fail to turn off.



FEAR OF SPIDERS

Arachnophobia is one of the most common phobias. Sufferers may experience anxiety about encountering a spider even when it is extremely unlikely.

AVIOPHOBIA

Fear of flying may occur by itself or as a manifestation of other phobias, such as acrophobia (fear of heights) or claustrophobia.



FEAR OF CROWDS

Enochlophobia may be associated with other fears, such as fear of catching a disease or being trampled.

ACROPHOBIA

Fear of heights is a generalized fear of being in a high place, even an enclosed space such as a high floor in a building.



PHOBIAS

A PHOBIA IS CONSIDERED TO BE A DISORDER WHEN PERSISTENT, IRRATIONAL FEARS OF PARTICULAR THINGS, ACTIVITIES, OR SITUATIONS DISRUPT EVERYDAY LIFE.

There are many different forms of phobia, but they can be categorized into two broad types: simple and complex. Simple phobias are fears of specific objects or situations, for example, spiders (arachnophobia) or enclosed spaces (claustrophobia). Complex phobias are more pervasive and involve several anxieties. For example, agoraphobia may involve fear of crowds and public places or of traveling in planes, buses, or other forms of public transportation; it also includes anxiety about being unable to escape to a safe place, usually home. Social phobia (also known as social anxiety disorder) is another complex phobia in which there is intense anxiety in social or performance situations (such as public speaking) because of fear of public embarrassment or humiliation.

Causes and effects

The causes of phobias are not known for certain. Some phobias tend to run in families, which may be a result of children learning a specific fear from their parents. In other cases, a phobia may develop in response to a traumatic event or situation.

The main symptom of a phobia is an intense, uncontrollable anxiety when confronted by the feared object or situation. Merely anticipating an encounter with the feared object or situation can cause anxiety. In severe cases there may be symptoms of a panic attack, such as

sweating, palpitations, breathing difficulty, and trembling, when the object or situation is actually encountered. There is also usually a strong desire to avoid the feared object or situation, often to the extent of taking extreme measures. These effects can severely limit normal everyday activities and sometimes a person with a phobia may try using drugs or alcohol in an attempt to reduce the anxiety.

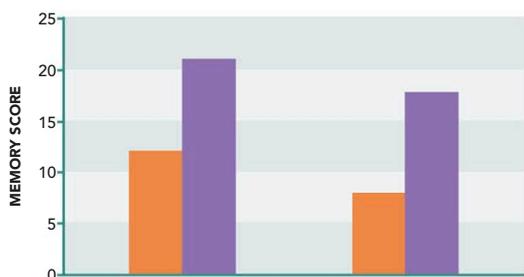
COMMON PHOBIAS

NAME	DESCRIPTION
Astraphobia	Fear of thunder and lightning
Carcinophobia	Fear of cancer
Claustrophobia	Fear of enclosed spaces
Cynophobia	Fear of dogs
Mysophobia	Fear of contamination by germs
Necrophobia	Fear of death or dead things
Nosophobia	Fear of developing a specific disease
Nyctophobia	Fear of the dark
Ophidiophobia	Fear of snakes
Trypanophobia	Fear of injections or medical needles

POST-TRAUMATIC STRESS DISORDER

A SEVERE ANXIETY RESPONSE CAN DEVELOP AFTER A PERSON IS INVOLVED IN OR WITNESSES A DISTRESSING OR LIFE-THREATENING EVENT, SUCH AS A TERRORIST ATROCITY, NATURAL DISASTER, RAPE OR PHYSICAL VIOLENCE, SERIOUS PHYSICAL INJURY, OR MILITARY COMBAT.

The external cause of post-traumatic stress disorder (PTSD) is the experience of trauma. In the brain itself, various abnormalities in areas involved in memory, the stress response, and the processing of emotions have been



KEY
PTSD PATIENTS (orange bars)
CONTROLS (purple bars)

IMPAIRED MEMORY FUNCTION
 Patients with PTSD and normal individuals (controls) were read a paragraph and asked to recall it immediately and after a delay. PTSD patients scored lower on both tests.

identified. The amygdala (involved in memory and emotion processing) is overactivated in response to memories of traumatic events whereas the prefrontal cortex is under-responsive to fearful stimuli, which may result in its failure to inhibit the amygdala and thereby inhibit traumatic memories. The thalamus may also be involved; some people have a genetic constitution that is associated with an enlarged thalamus, which may in turn lead to an exaggerated response to fearful memories and an increased susceptibility to PTSD.

Symptoms and treatment

The symptoms of PTSD may develop immediately after a traumatic event or may not appear for months. They may include flashbacks or nightmares that trigger the same intense fear originally felt; emotional numbness; loss of enjoyment in usually pleasurable activities; memory problems; hypervigilance and an exaggerated startle response; sleeping problems; and irritability.

SHELL SHOCK

Stress reaction to the trauma of combat—shell shock—came to be widely recognized during World War I. Today, the term “shell shock” is categorized as “combat stress reaction” and refers to a collection of short-lived physical and mental symptoms, such as exhaustion and hypervigilance. If symptoms persist long-term, the condition is usually categorized as PTSD.



OBSESSIVE–COMPULSIVE DISORDER

COMMONLY KNOWN AS OCD, OBSESSIVE–COMPULSIVE DISORDER IS CHARACTERIZED BY RECURRENT THOUGHTS THAT CAUSE ANXIETY AND/OR OVERWHELMING URGES TO PERFORM REPETITIVE ACTS OR RITUALS IN AN ATTEMPT TO RELIEVE ANXIETY.

The exact cause of OCD is not known, but it is generally thought to be due to a combination of factors and may have different causes in different people. OCD tends to run in families, so there may be a genetic link in some cases. It has also been associated with childhood infection with *Streptococcus* bacteria. Brain imaging studies have found evidence of abnormal physiological connections in the communication loop between the orbitofrontal cortex, caudate nucleus, and thalamus involving

the neurotransmitter serotonin. In addition, personality type may be a factor—perfectionists appear to be more susceptible to developing OCD.

Symptoms

Symptoms typically appear during the teenage or early adult years and may consist of obsessions, compulsions, or both. Obsessions are thoughts, feelings, or images that recur involuntarily and provoke anxiety. For example, there may be an excessive fear of dirt that may be

so powerful that the person fears leaving home in case he or she becomes contaminated. Compulsions are actions that a person feels compelled to carry out repeatedly in an effort to ward off anxiety, such as repeatedly checking things such as locks or doors. The person may recognize that the obsessions and/or compulsions are unreasonable but cannot control them.

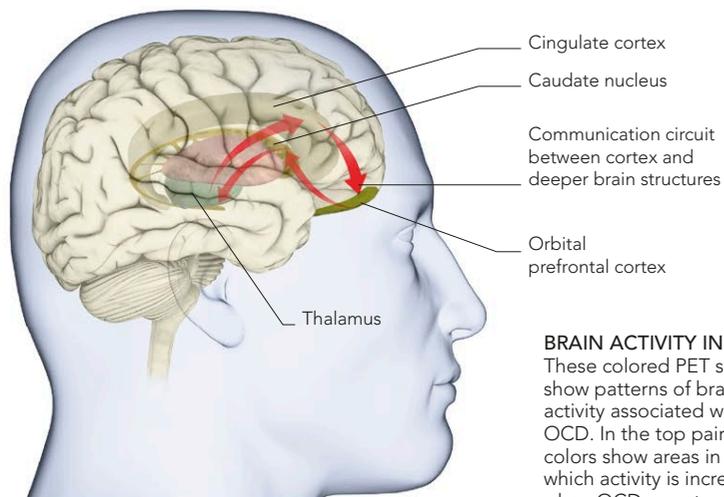
Diagnosis and outlook

To be diagnosed with OCD, the symptoms must cause anxiety, must be present on most days for at least two weeks, and must interfere significantly with everyday life. With treatment most people recover, although symptoms may recur under stress.

Deep brain stimulation, in which tiny electrodes are inserted into the brain to modulate the activity, is a promising new treatment for this condition.



COMPULSIVE BEHAVIOR
 Compulsions, such as constant handwashing, are actions that a person feels compelled to carry out repeatedly.

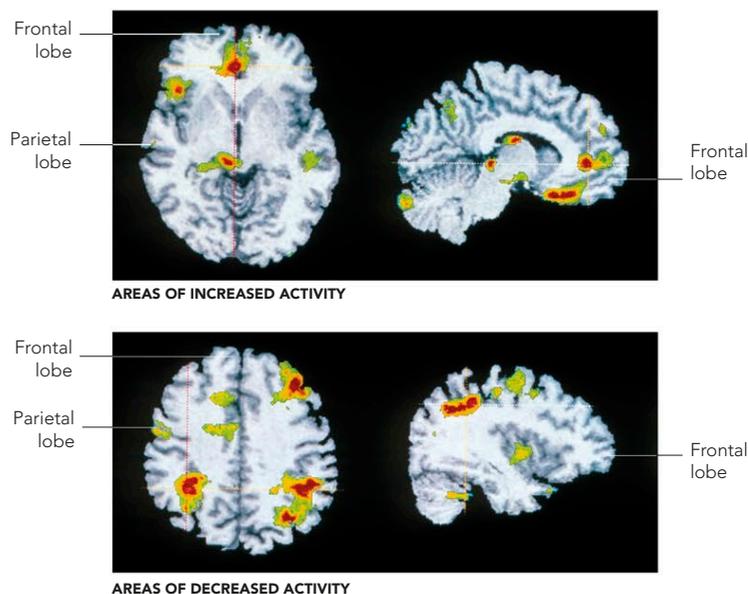


BRAIN CIRCUIT IN OCD

This disorder may be associated with abnormalities in the communication circuit between the orbital prefrontal cortex and deeper brain structures.

BRAIN ACTIVITY IN OCD

These colored PET scans show patterns of brain activity associated with OCD. In the top pair, the colors show areas in which activity is increased when OCD symptoms get stronger. The bottom scans show areas of decreased activity when symptoms strengthen.



BODY DYSMORPHIC DISORDER

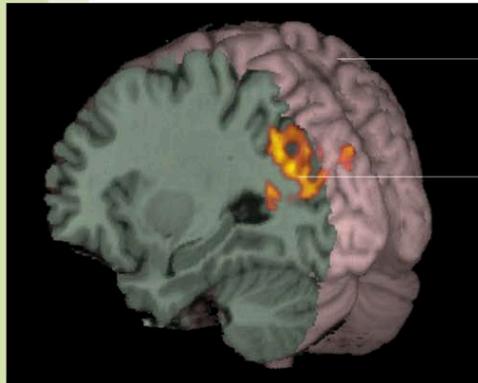
BODY DYSMORPHIC DISORDER (BDD) IS A MENTAL HEALTH PROBLEM IN WHICH A PERSON IS EXCESSIVELY CONCERNED ABOUT A PERCEIVED DEFECT IN HIS OR HER APPEARANCE AND THIS PREOCCUPATION WITH BODY IMAGE CAUSES SIGNIFICANT DISTRESS.

The cause of body dysmorphic disorder is unclear, although it is thought to be due to a combination of several factors, possibly including low levels of serotonin. It may occur in combination with other disorders, such as eating disorders,

obsessive-compulsive disorder, and generalized anxiety disorder, although it is not clear whether there is a causative relationship with such disorders.

Many people are dissatisfied with some aspect of their appearance, but people with BDD are obsessed with one or more

perceived flaws. Typical signs of BDD include refusing to be in photographs; trying to hide the “flaw” with clothing or makeup;



Right hemisphere

Active area in left hemisphere

PROCESSING FACES IN BDD

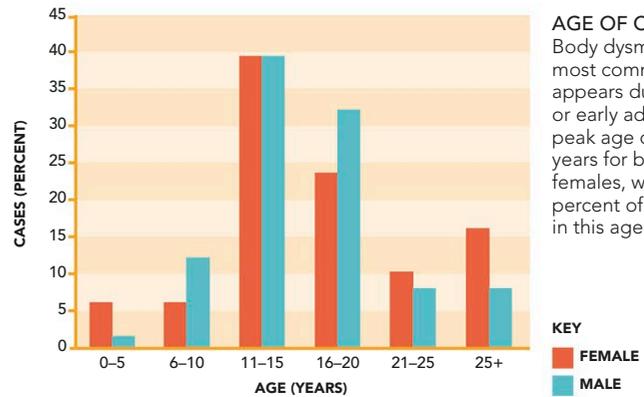
Studies of BDD patients have revealed that they tend to use the left side of the brain, which normally processes complex detail, for processing pictures of faces. Normal people usually use their right hemisphere, unless they are examining a face closely.

constantly checking one's appearance in mirrors; frequently comparing one's appearance with that of others; often seeking reassurance about one's appearance; frequently touching the perceived flaw; and picking the skin to make it smooth. In addition, a person may feel anxious and self-conscious around other people because of the perceived flaw and may avoid social situations in which it might be

noticed. In some cases, medical and surgical treatment may be sought to correct the perceived flaw.

Diagnosis

Body dysmorphic disorder is diagnosed by psychiatric evaluation. To be diagnosed with this disorder, preoccupations with appearance must cause considerable distress and interfere with everyday life.



AGE OF ONSET

Body dysmorphic disorder most commonly first appears during puberty or early adulthood. The peak age of onset is 11–15 years for both males and females, with about 40 percent of cases starting in this age group.

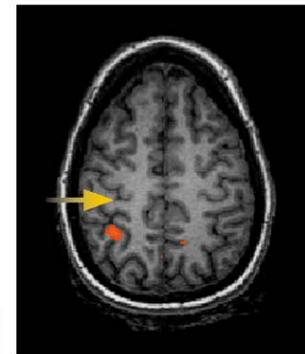
SOMATIZATION DISORDER

IN THIS CHRONIC PSYCHOLOGICAL PROBLEM, A PERSON COMPLAINS OF PHYSICAL SYMPTOMS FOR WHICH NO UNDERLYING PHYSICAL CAUSE IS FOUND.

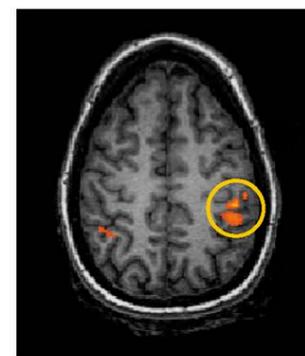
A person with this disorder typically experiences several physical symptoms that persist for years. The symptoms are not generated intentionally and are often severe enough to interfere with everyday life, but no physical cause for them can be identified.

The symptoms may affect any part of the body, but complaints involving the digestive, nervous, and reproductive systems are the most common. If symptoms involve the voluntary central nervous system, such as paralysis, the condition is sometimes classed as conversion disorder (formerly known as hysteria).

The cause of somatization disorder is not known. In some cases it may be associated with other disorders such as anxiety and depression, but it is not clear whether these are causes or effects of the disorder.



LEFT HAND STIMULATED



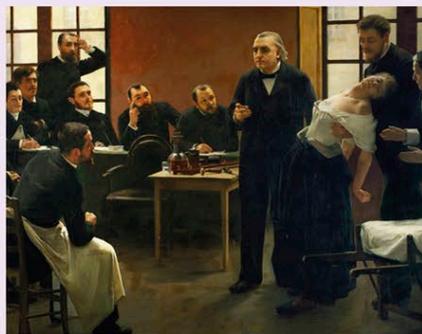
RIGHT HAND STIMULATED

BRAIN ACTIVITY

Unusual patterns of brain activity may be detected in some cases of somatization disorder. These MRI scans show the brain of a person who experiences a loss of sensation in the left hand (the right side of the brain appears on the left in the images). The scans reveal an absence of brain activity (shown by the arrow) in the right somatosensory cortex when the left hand is stimulated. There is normal brain activity (circle) when the unaffected right hand is stimulated.

HYSTERIA

The term “hysteria” originates from the Greek word *hysterikos*, which referred to a medical disorder caused by disturbances of the uterus. The Austrian psychoanalyst Sigmund Freud (see p.189) suggested that hysteria was an attempt by the subconscious to protect the patient from stress. The term is no longer generally used in psychiatry, although it is still in everyday use to refer to a state of uncontrollable emotional excess.



DEMONSTRATION OF HYSTERIA

Hysteria was believed to be an inherited neurological disorder by the French neurologist Jean-Martin Charcot (1825–93), who used hypnosis to induce hysteria in patients and then studied the results.

HYPOCHONDRIA

THIS DISORDER IS CHARACTERIZED BY EXCESSIVE AND UNREALISTIC ANXIETY ABOUT HAVING A SERIOUS ILLNESS.

In hypochondria (also known as hypochondriases) trivial symptoms assume unrealistic significance.

The symptoms are real, such as a cough or headache, but people with hypochondria are genuinely worried that they indicate a serious disease, such as lung cancer or a brain tumor. In mild forms, the person may simply worry constantly. In more severe cases, hypochondria can seriously disrupt everyday life, with the person making

frequent visits to the doctor to have tests. Even when the test results prove negative, people may remain convinced that they have a serious illness and often seek other medical opinions. In addition, the person may believe they have a particular disease after hearing about it; for example, after

hearing about Alzheimer's disease, an instance of momentary forgetfulness might lead the person to believe they have that disease. Many people with hypochondria also have other mental health disorders, such as depression, obsessive-compulsive disorder, phobia, or generalized anxiety disorder.

MUNCHAUSEN'S SYNDROME

SOMETIMES ALSO KNOWN AS HOSPITAL ADDICTION SYNDROME, MUNCHAUSEN'S SYNDROME IS A RARE PSYCHIATRIC CONDITION IN WHICH A PERSON REPEATEDLY SEEKS MEDICAL ATTENTION FOR FAKED OR SELF-INDUCED SYMPTOMS OF ILLNESS.

People with Munchausen's syndrome are aware that they are fabricating symptoms, unlike those with hypochondria, who truly believe they are ill. They do not fake illness in order to receive tangible benefits (such as financial gain). Instead,

the motive seems to be to obtain investigation, treatment, and attention from medical personnel. People with the syndrome often have a good medical knowledge and create plausible symptoms and explanations for their

faked illness, which makes diagnosis of Munchausen's syndrome very difficult. In addition to lying about symptoms, they may try to manipulate test results—for example, by adding blood to a urine sample—and may even inflict symptoms on themselves; they may injure themselves or ingest poisons, for instance. Typically, they attend many different hospitals, often repeatedly presenting the same symptoms. In a related condition,

known as Munchausen's by proxy or fabricated and induced illness (FII), people may invent or induce symptoms in somebody else. This usually involves parents faking or inducing symptoms in their child.

Diagnosis is difficult and involves carrying out various tests to exclude an underlying illness. If a genuine underlying cause is not found, a diagnosis is made from a psychiatric assessment.

FEIGNING DISEASE

Many people feign illness at some point in their lives, but in the majority of cases it is simply an occasional occurrence—to avoid going to work or school, for example. However, in some people fabricating illness is a pathological problem. This chart summarizes the ways in which feigning illness can be classified.

Nonpathological

This form of feigning typically involves using minor symptoms as a means of avoidance or of getting attention. The feigning tends to occur only sporadically and for no tangible gain.

Pathological

Pathological disease feigning, unlike the nonpathological form, tends to occur repeatedly and usually involves the feigner obtaining a significant tangible gain, such as a financial reward.

Malingering

This is the intentional use of false or exaggerated symptoms to obtain a significant gain, such as financial compensation or sympathy. It is not a disorder itself, but it may indicate a mental problem.

Factitious disorders

These involve intentional disease forgery to obtain emotional gain, such as sympathy, attention, and nurturing. Extreme forms of factitious disorders include Munchausen's syndrome.

TOURETTE'S SYNDROME

TOURETTE'S SYNDROME IS A NEUROLOGICAL DISORDER THAT IS CHARACTERIZED BY SUDDEN, REPETITIVE, INVOLUNTARY MOVEMENTS (CALLED MOTOR TICS) AND NOISES OR WORDS (CALLED VOCAL TICS).

In most cases, Tourette's syndrome runs in families and genetic factors may be involved, although the relevant genes and the mode of inheritance have not been identified. In some cases, known as sporadic Tourette's syndrome, there is no apparent inherited link. Various brain abnormalities have been implicated, including malfunctioning of the basal ganglia, thalamus, and frontal cortex, and abnormalities in the neurotransmitters serotonin, dopamine, and norepinephrine, although their causative relationship

to Tourette's has not been established. Environmental factors may also play a role in the development of Tourette's syndrome.

Symptoms and effects

The characteristic symptoms of Tourette's syndrome are motor tics, such as blinking, facial twitches, shoulder shrugging, and head jerking, and vocal tics, such as grunting or repeating words. The involuntary utterances of swear words (coprolalia) is a well-known feature, but

is comparatively rare. Other mental health problems, such as depression or anxiety disorders, may also develop. Typically, the symptoms first appear during childhood and get worse during the teenage years but then improve. However, in some cases the condition gets progressively worse and lasts throughout adulthood.

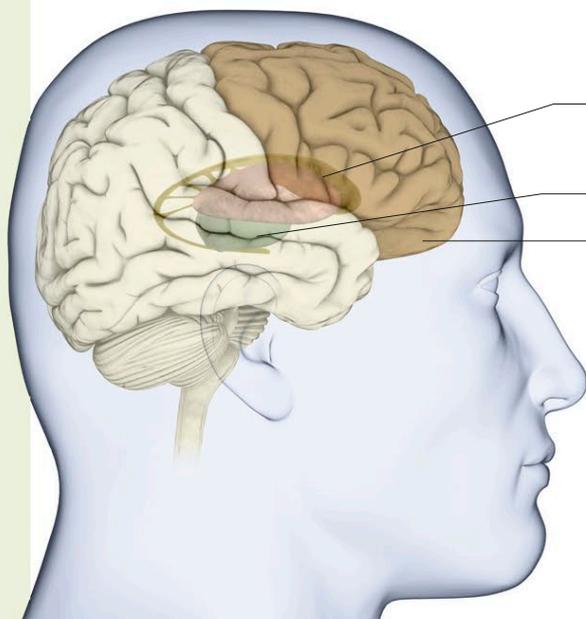
Diagnosis

For a positive diagnosis of Tourette's, both motor and vocal tics must be present and they must not be due to another medical condition, medications, or other substances. They must occur several times a day on most days or intermittently for more than a year.



TOURETTE'S MOTOR TICS

This long-exposure photograph illustrates the repetitive movements characteristic of Tourette's syndrome. A Tourette's sufferer, on the left, has had lights attached to his fingers to show his hand movements.



Basal ganglia

Responsible for implementing movement routines

Thalamus

Filters and relays nerve impulses to the cortex

Frontal cortex

Plays a key role in sequencing actions

IMPLICATED BRAIN AREAS

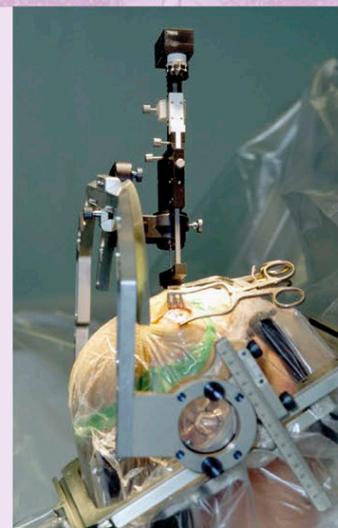
Brain studies of people with Tourette's have found abnormalities in certain areas of the brain, including the basal ganglia, thalamus, and frontal cortex, but it is not clear if these are a cause or effect of the disorder.

EXPERIMENTAL TREATMENT

Most people with Tourette's learn to live with it and do not require treatment. In severe cases, it is usually treated primarily with medication to help control the tics, although talking therapy may also be useful, particularly if there are other problems such as anxiety or obsessions. In a few, very severe, debilitating cases that have not responded to other treatments, deep-brain stimulation has been used. However, this procedure is still highly experimental and it is not yet clear whether the benefits outweigh the risks.

DEEP-BRAIN STIMULATION

This procedure involves surgically implanting a device known as a brain pacemaker into the brain (as shown here). The pacemaker sends electrical impulses to specific areas of the brain, thereby controlling their activity.



SCHIZOPHRENIA

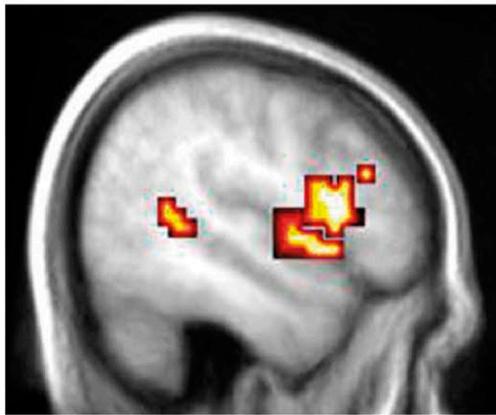
A SERIOUS MENTAL HEALTH DISORDER, SCHIZOPHRENIA IS CHARACTERIZED BY DISTORTIONS IN THINKING, PERCEPTIONS OF REALITY, EXPRESSION OF EMOTIONS, SOCIAL RELATIONSHIPS, AND BEHAVIOR.

Contrary to popular belief, schizophrenia is not a “split personality,” but rather a form of psychosis in which a person is not able to distinguish what is real from what is imagined.

The cause of schizophrenia is not known, although it is believed to result from a combination of genetic and environmental factors. Schizophrenia runs in families, and a person who has a close family member with the disorder is at increased risk of developing it. However, it is believed that

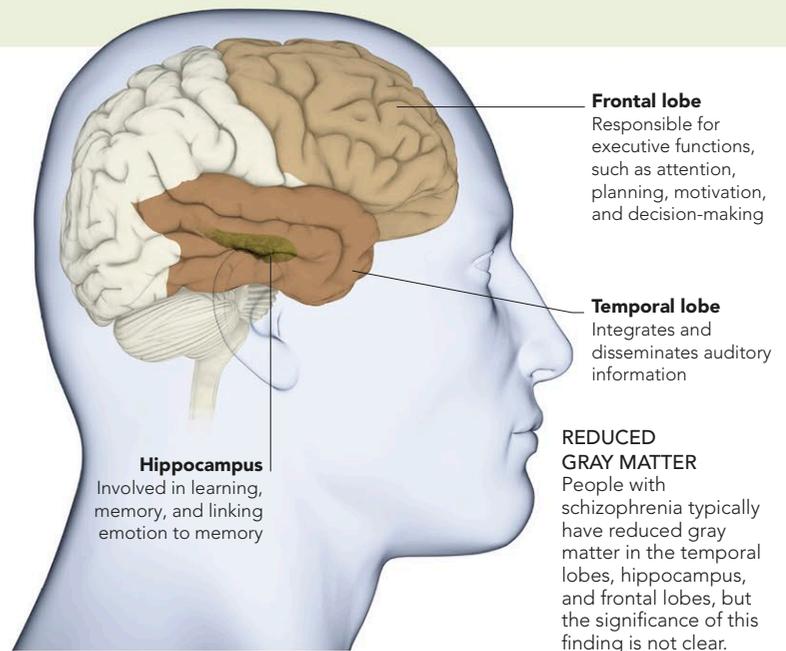
genetic susceptibility alone is insufficient to cause schizophrenia and environmental factors are also necessary. Among the environmental factors that may be involved are exposure to infection or malnutrition before birth, stressful life events, and the use of marijuana. Excess dopamine levels may also be involved since all antipsychotic drugs block dopamine, and drugs that release dopamine can trigger schizophrenia.

Various brain abnormalities have been identified in people with schizophrenia, including unusually low levels of



HEARING VOICES

During auditory hallucinations, fMRI scans show activity mainly in right-hemisphere language areas, rather than in the left-hemisphere areas typically active in speech production. This may explain why the speech produced by the “voices” is simple and derogatory and why the patient mistakenly attributes them to an external source.



Frontal lobe

Responsible for executive functions, such as attention, planning, motivation, and decision-making

Temporal lobe

Integrates and disseminates auditory information

Hippocampus

Involved in learning, memory, and linking emotion to memory

REDUCED GRAY MATTER

People with schizophrenia typically have reduced gray matter in the temporal lobes, hippocampus, and frontal lobes, but the significance of this finding is not clear.

glutamate receptors and a reduction of gray matter in certain brain regions, notably the hippocampus, frontal lobes, and temporal lobes. However, the significance of these abnormalities in schizophrenia has not been established.

Symptoms and treatment

Schizophrenia can take various forms (see panel, left). The symptoms typically develop during late adolescence or early adulthood in men, and some 4–5 years later in women. Different individuals may have different patterns of symptoms, and with varying degrees of severity. However, in general they may include delusions; hallucinations, especially auditory ones; jumbled,

incoherent speech (so-called “word salad”); lack of emotions or inappropriate emotions, such as amusement at bad news; disorganized thoughts; clumsiness; involuntary or repetitive movements; social isolation; neglect of personal health and hygiene; and unresponsive (catatonic) behavior.

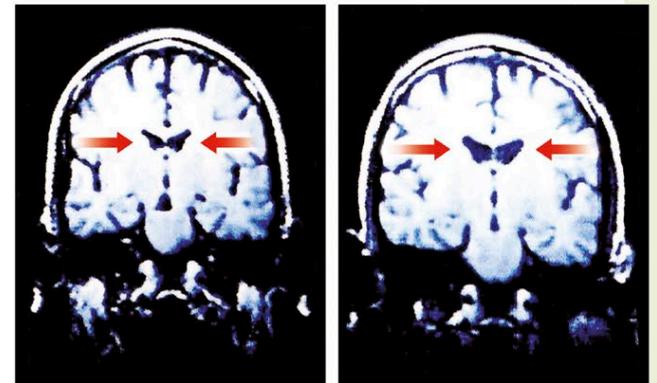
Schizophrenia is diagnosed from the symptoms, but various tests are also usually performed to exclude other possible causes of abnormal behavior. Treatment is with medication, such as antipsychotic drugs, and talking therapy. About 1 in 5 people make a full recovery, but for the remainder schizophrenia is lifelong.

TYPES OF SCHIZOPHRENIA

TYPE	DESCRIPTION
Paranoid schizophrenia	Delusions (particularly about being persecuted) and hallucinations are present, but thinking, speech, and emotions are often relatively normal.
Disorganized schizophrenia	Thinking and speech are confused and disordered, and emotions may be flat or inappropriate; behavior is disorganized and often disrupts everyday activities, such as cooking or washing.
Catatonic schizophrenia	Lack of responsiveness to the surroundings and immobility are typical features; in some cases, the person may exhibit strange postures or purposeless movements, or repeat overheard words.
Undifferentiated schizophrenia	Some of the symptoms of paranoid, disorganized, or catatonic schizophrenia are present, but the pattern of symptoms does not clearly fall into any of the types above.
Residual schizophrenia	Symptoms of schizophrenia are present, but these are now significantly less severe than when the schizophrenia was originally diagnosed.

LOSS OF TISSUE

These MRI scans of a pair of twins show that the ventricles (indicated by arrows) are enlarged—suggesting loss of brain tissue—in the twin on the right, who is schizophrenic. The twin on the left is not affected.



DELUSIONAL DISORDER

THIS DISORDER IS A TYPE OF PSYCHOSIS CHARACTERIZED BY THE PRESENCE OF PERSISTENT, IRRATIONAL BELIEFS THAT ARE NOT CAUSED BY ANOTHER MENTAL DISORDER.

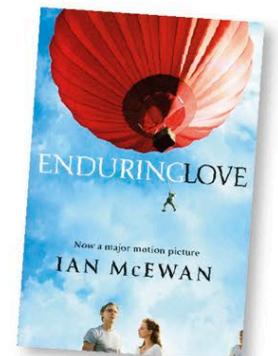
In delusional disorder, the delusions are “non-bizarre” (involving things that are within the realms of possibility). Apart from the delusion and behavior related to it, someone with the disorder often functions normally, although preoccupation with the delusion can disrupt everyday life. The cause of delusional disorder is not known, but it is more common in people with family

members who have this disorder or schizophrenia. Socially isolated people tend to be more susceptible, and in some cases it may also be triggered by stress.

There are several types of delusional disorder: jealous (the delusion that their partner is unfaithful); persecutory (a belief that somebody is hounding or trying to harm them); erotomanic (somebody—often a celebrity—is in love with them); grandiose (an inflated sense of worth, power, talent, or knowledge); somatic (the delusion that they have a physical defect or medical problem); and mixed (two or more of the other delusional types).

DE CLERAMBAULT'S SYNDROME

Also called erotomania, de Clerambault's syndrome is a rare delusional disorder in which the sufferer believes that another person is in love with him or her. This disorder is a central theme in British novelist Ian McEwan's *Enduring Love*.



ADDICTIONS

AN ADDICTION IS A STATE OF BEING SO DEPENDENT ON SOMETHING THAT IT BECOMES DIFFICULT OR IMPOSSIBLE TO DO WITHOUT IT FOR ANY SIGNIFICANT PERIOD.

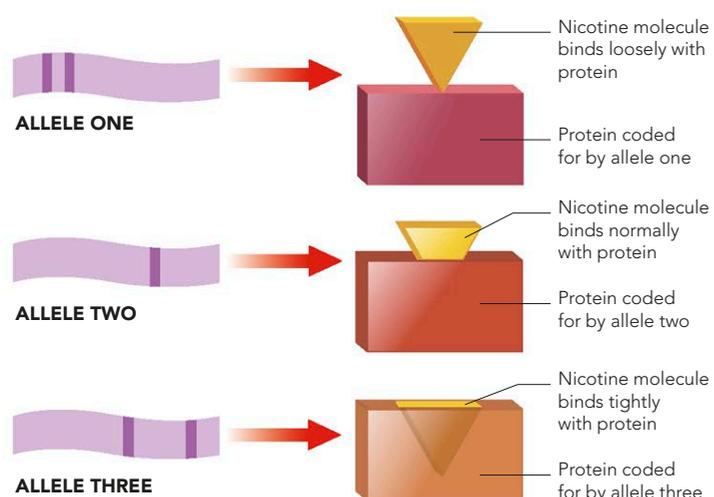
It is possible to become addicted to anything, but whatever the addiction is, the person cannot control it. An addiction may be to a substance or an activity.

It is believed that addictive substances or activities affect the brain so that it reacts in the same way that it responds to pleasurable experiences, by increasing the release of the neurotransmitter dopamine. It is not known why some

people seem to be more likely to become addicted than others, although it is thought that genetic susceptibility and environmental factors probably play a role. For example, children who grow up in a family where there is drug or alcohol abuse are more likely to become addicted.

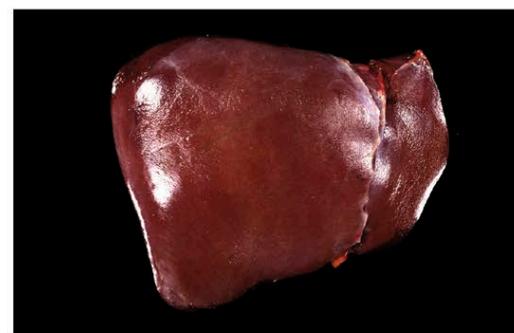
Although some symptoms are specific to the addictive substance or activity, there are several general symptoms that occur in all addictions. These include the development of tolerance—the need for increasing amounts to produce the desired effect; unpleasant physical and/or psychological withdrawal symptoms when the substance or activity is stopped; and continuing

to use the substance or engage in the activity even though it may be detrimental to physical or mental health, or relationships.



GENES AND NICOTINE ADDICTION

Research indicates that there may be a genetic factor involved in some addictions. In people who carry one version (allele) of a particular gene, the allele may code for a protein that binds only loosely with nicotine. In people who carry other alleles, the proteins they code for may bind normally or tightly to nicotine. The tightness of binding alters the effects nicotine has on the body, which may, in turn, affect the susceptibility to nicotine addiction.



HEALTHY LIVER

A normal, healthy liver is dark red in color, has a smooth outer surface without lumps or scar tissue, and is free of areas of discoloration.



CIRRHOTIC LIVER

This liver shows advanced cirrhosis, with large areas of scar tissue, a lumpy surface, and general discoloration. Cirrhosis is one of the possible complications of alcohol addiction.

PERSONALITY DISORDERS

THIS IS A GROUP OF DISORDERS IN WHICH A PERSON'S HABITUAL BEHAVIOR AND THOUGHT PATTERNS CAUSE RECURRENT PROBLEMS IN EVERYDAY LIFE.

The cause of personality disorders is not known but they are thought to be due to a combination of genetic and environmental influences. Factors that may increase

the risk of developing a personality disorder include a family history of such a disorder or another mental illness; abuse during childhood; a dysfunctional family life during childhood; and having conduct disorder (see p.248) in childhood.

There are many types of personality disorders (see panel, below), but in general they are all characterized by an inflexible way of thinking and behaving, irrespective of

the situation. Symptoms tend to develop in adolescence or early adulthood and may vary in severity. Often a person with a personality disorder is not aware that the behavior and thought patterns are inappropriate, but may be aware of problems with personal, social, or work relationships, and these problems may cause distress. Specific symptoms depend on the type of personality disorder a person has.

TYPES OF PERSONALITY DISORDERS

Personality disorders are classified into three broad groups, known as clusters, according to the behavioral symptoms and types of thinking exhibited.

Cluster A The disorders that comprise this group are characterized by odd or eccentric behavior and/or thinking.

Paranoid People with paranoid personality disorder are suspicious and distrustful of others, may believe others are trying to harm them, and tend to be hostile and emotionally detached.

Schizoid Those with this disorder are uninterested in social relationships, introverted and solitary, and have a limited range of emotional expression; often they seem unable to recognize normal social cues.

Schizotypal People with this type are socially and emotionally detached and exhibit peculiarities of behavior and thinking, such as "magical" thinking (believing their thoughts can influence others).

Cluster B These are characterized by dramatic, erratic, or overemotional thinking and behavior.

Antisocial Previously called sociopaths, people with this personality disorder persistently disregard the feelings, rights, and safety of others; they may also persistently lie, steal, or behave aggressively.

Borderline Borderline types have problems with self-identity and fear being alone, yet often have volatile relationships; they engage in impulsive or risky behavior; and tend to have unstable moods.

Histrionic Histrionic types are highly emotional and constantly seek attention; they tend to be very sensitive to the opinions of others and overly concerned with their physical appearance.

Narcissistic Narcissistic types believe that they are superior to others, but still constantly seek approval; they tend to exaggerate their achievements and exhibit marked lack of empathy.

Cluster C The personality disorders that comprise this group are distinguished by habitual patterns of anxious, fearful, or inhibited thinking or behavior.

Avoidant People with avoidant personality disorder feel inadequate and are oversensitive to criticism or rejection; they are timid and extremely shy in social situations, which may lead to social isolation.

Dependent People with this type of personality disorder are extremely dependent on, and submissive toward, others; they feel unable to cope with everyday life alone and often feel an urgent need to be in a relationship.

Obsessive-compulsive Those with this personality disorder conform rigidly to rules and moral codes, are inflexible and often want to be in control; also tend to be perfectionists. This is not the same as OCD (see p.233), which is an anxiety disorder.

EATING DISORDERS

AN EATING DISORDER IS A CONDITION IN WHICH THERE ARE EXTREME PREOCCUPATIONS WITH FOOD AND/OR WEIGHT AND DISTURBANCES IN EATING BEHAVIOR.

The causes of eating disorders are not clear, although a combination of biological, genetic, psychological, and social factors are thought to be involved. The effects of social and peer pressure to be thin may be a contributory factor. Anxiety about body image, low self-esteem, and depression may also be involved.

Types of eating disorders

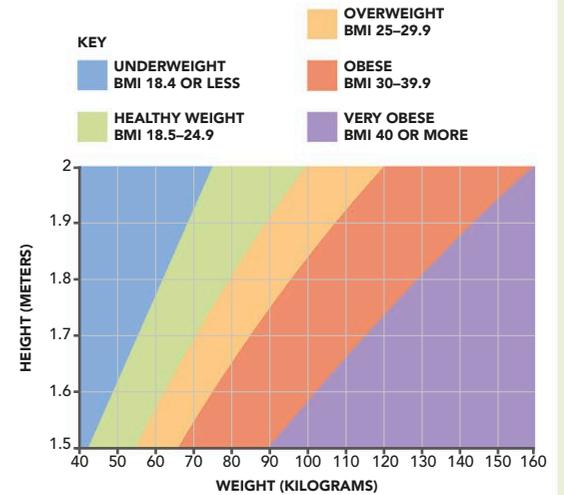
Eating disorders are most common in adolescent girls and young women, but also affect older women and men. The most common types are anorexia nervosa, bulimia nervosa, and binge-eating disorder.

Anorexia nervosa is characterized by self-starvation and excessive weight loss. Its main features are an intense fear of being fat or gaining weight; a resistance to maintaining normal weight; and the denial of the seriousness of low body weight. It can be fatal.

Bulimia nervosa is characterized by binge eating and then repeated compensatory actions to prevent weight gain, such as self-induced vomiting, laxative or diuretic use, excessive exercise, or fasting. It can result in life-threatening heart abnormalities due to an imbalance of electrolytes.

Binge-eating disorder is similar to bulimia nervosa but without the compensatory actions to counter the binges, which can lead to obesity.

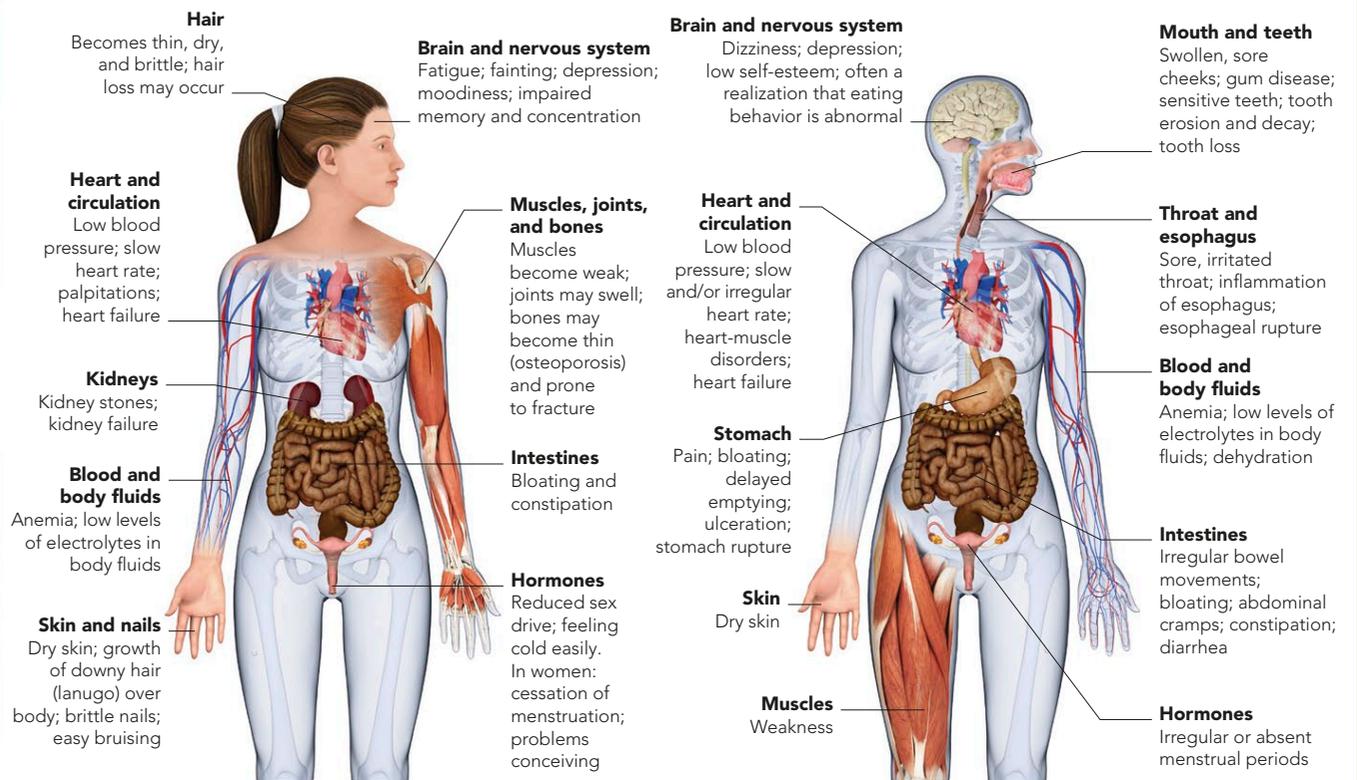
BODY MASS INDEX
Body mass index (BMI) is a figure that indicates whether a person is within a healthy weight range. Adults with anorexia nervosa have a BMI of 17.5 or less.



WASTING AWAY
The extreme weight loss associated with anorexia nervosa leads to wasting of body tissues, as is evident in this photograph.



DENTAL EROSION
Repeated self-induced vomiting in bulimia nervosa can lead to erosion of tooth enamel by stomach acid, and this may lead to loss of teeth.



EFFECTS OF ANOREXIA NERVOSA ON THE BODY
The most obvious effect of anorexia nervosa is extreme weight loss. However, it can also have a number of other effects on the body and may even be fatal.

EFFECTS OF BULIMIA NERVOSA ON THE BODY
Bulimia nervosa tends to have less obvious outward effects than anorexia nervosa as the person is often of normal weight. However, repeated bingeing and purging can have widespread physical effects.

ATTENTION DEFICIT HYPERACTIVITY DISORDER

COMMONLY KNOWN AS ADHD, ATTENTION DEFICIT HYPERACTIVITY DISORDER IS ONE OF THE MOST COMMON BEHAVIORAL DISORDERS OF CHILDHOOD.

ADHD is characterized by persistent difficulty paying attention and/or hyperactivity. It is most common in children, but it may persist into adulthood. ADHD tends to run in families and in most cases genetic inheritance, probably involving many genes, is thought to be the most probable underlying cause. However, this genetic predisposition interacts with various other factors, such as exposure to certain toxins (such as nicotine and alcohol) before birth, brain damage

before birth or in the early years of life, and food allergies. There is no evidence that parenting problems cause ADHD, but they may influence its severity and a child's coping strategies. Some brain abnormalities have been found in children with ADHD, including low dopamine levels. Drugs that increase dopamine

levels in the brain, such as Ritalin, may lessen symptoms. Symptoms usually appear during early childhood and may become worse when the child starts school. Due to the various ADHD-related problems, there may also be difficulty making friends, low self-esteem, anxiety, or depression.

TYPES OF ADHD

Attention deficit hyperactivity disorder can be categorized into three broad types, according to the predominant type of behavior exhibited.

Inattentive Symptoms include a short attention span; poor concentration; difficulty carrying out instructions; and changing activities often.

Hyperactive/impulsive Characterized by fidgeting; excessive activity; acting without thinking; excessive talking; and repeatedly interrupting a speaker.

Combined Symptoms include those of both other types, such as a short attention span, overactivity, and acting without thinking.

DEVELOPMENTAL DELAY

DEVELOPMENTAL DELAY IS A TERM USED WHEN A BABY OR YOUNG CHILD HAS NOT ACQUIRED THE SKILLS AND ABILITIES NORMALLY ACHIEVED BY A PARTICULAR AGE.

In the few first years of life there are important stages—developmental milestones—when a child is normally expected to have acquired certain basic physical, mental, social, and language skills. Child development is assessed in several areas, including physical and motor development; vision, hearing, speech, and mental development; and social development.

Generalized or specific delay

Delays can vary in severity and may affect one or more areas of development. Generalized delay affects most areas

WALKING UNAIDED

Being able to walk without help is one of the key developmental milestones. Typically, children manage this when between about 10 and 19 months old.

of development and may be due to various factors, such as severe visual or hearing impairment; brain damage; learning difficulties; Down syndrome; severe, prolonged disease, such as heart disease, muscle disease, or a nutritional disorder; or a lack of physical, emotional, or mental stimulation.

Developmental delay may also occur in specific areas only. Delay in movement and walking is quite common, and often a child catches up. However, there may be a serious underlying cause such as muscular dystrophy, cerebral palsy, or a neural-tube defect (see p.237). Delay in speech and language development may have various causes, including lack of stimulation, hearing problems, or more rarely, autism. Generalized difficulty with muscle control that affects speaking, which may be due to cerebral palsy, for example, can also cause delay in this area.

Diagnosis and treatment

Often delays are first noticed by parents, but a delay may also be detected during routine developmental checks. If a problem is suspected, a full developmental assessment is done, and the child may be referred to a specialist. Treatment depends on the severity and type of delay. It may include physical aids, such as glasses or hearing aids, therapies such as speech therapy, and possibly special educational help.



SCRIBBLING AND DRAWING

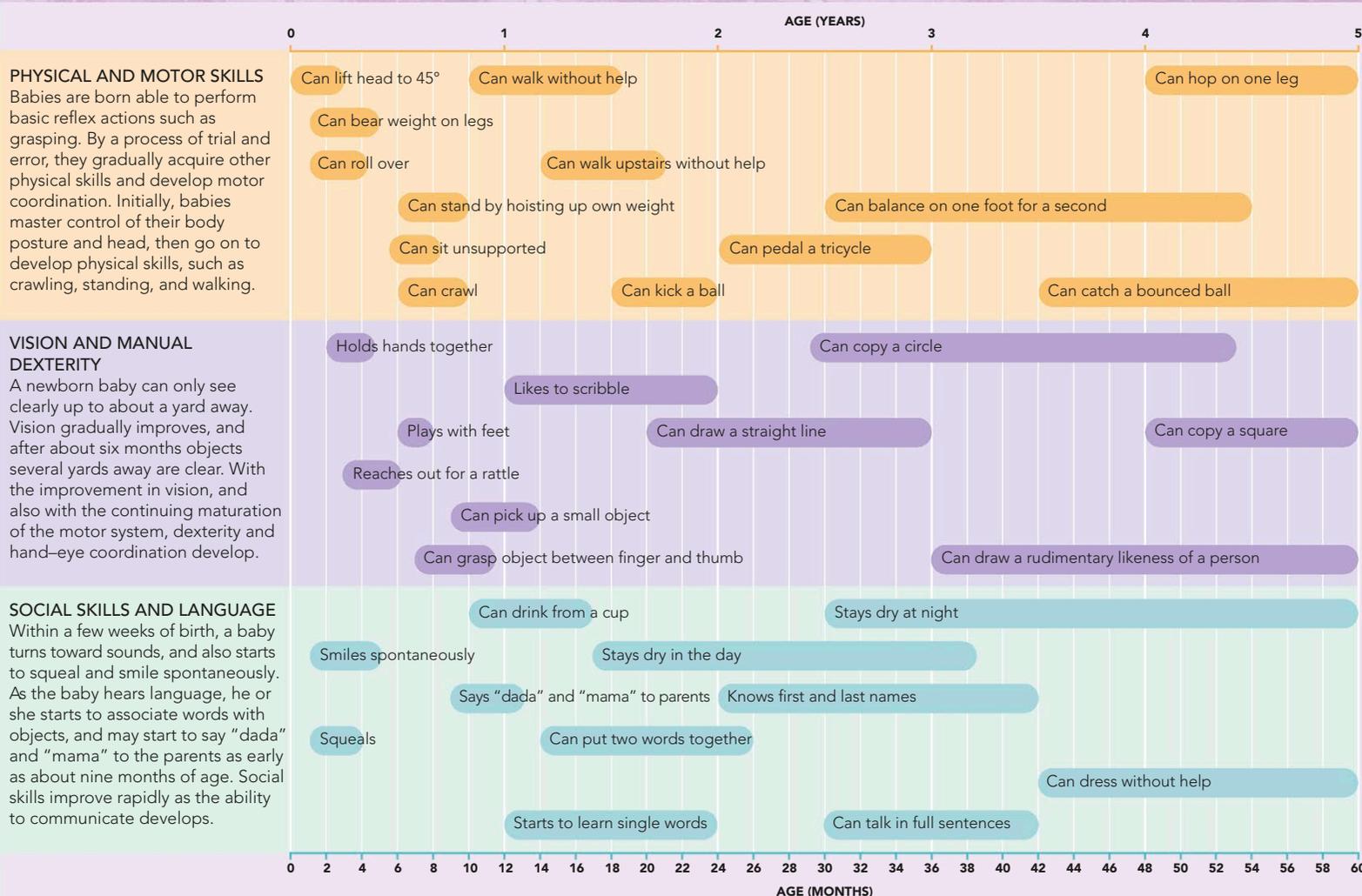
Normally, a child likes to scribble from about one year old, and by the age of about three most children are able to draw a reasonably straight line.



RIDING A TRICYCLE

The ability to pedal a tricycle is an indicator of motor-skill and physical development. Normally, this ability develops between about two and three years of age.

DEVELOPMENTAL MILESTONES



LEARNING DISABILITY

LEARNING DISABILITY REFERS TO PROBLEMS IN UNDERSTANDING, REMEMBERING, USING, OR RESPONDING TO INFORMATION.

There are differences in opinion about what the term “learning disability” encompasses, but, in general, it applies to conditions in which there is developmental delay. However, learning difficulty may also refer to a specific difficulty, for example, in reading or writing.

Types

Learning disabilities are commonly categorized as generalized or specific. Generalized learning disability affects all or almost all intellectual functions, leading to developmental delay. In addition to below-average intelligence, there may also be behavioral problems

and, in severe cases, physical developmental problems as well, impairing motor skills and coordination.

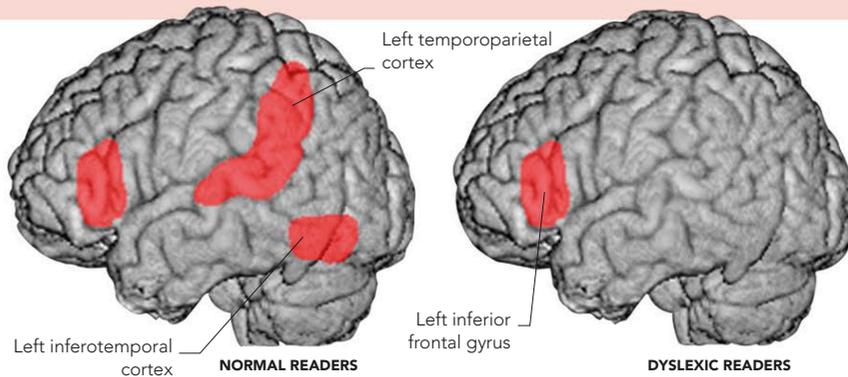


DYSCALCULIA

Difficulty with mathematics—dyscalculia—is the numerical counterpart of dyslexia. It usually first becomes apparent in the early school years when a child has problems with learning number facts and calculations such as addition and subtraction.

FRAGILE X SYNDROME

This syndrome is a major cause of severe learning disability in boys. It is caused by a constriction near the end of an X chromosome (circled), making it prone to break.



DYSLEXIC BRAIN

These two images show the areas of the brain that are active while reading in normal people (far left) and those with dyslexia (left). Only the left inferior frontal gyrus is active in those with dyslexia, whereas in normal readers other areas are also active.

Specific learning disabilities (see table, below) affect only one or a few areas of mental functioning, and, in many cases, intelligence is not impaired.

People with learning disability may also have various associated conditions, such as ADHD (see p.246), autistic disorder (see opposite page), or epilepsy (see p.226).

Causes

Learning disability can have a wide range of causes, including genetic abnormalities, such as Williams syndrome, or chromosomal abnormalities, such as Down

syndrome (see p.236) and fragile X syndrome (see below). Other factors include problems with brain development before or during birth, possibly due to exposure to toxins such as alcohol or drugs in the uterus, lack of oxygen, or premature or prolonged labor; and a head injury, malnutrition, or exposure to environmental toxins (such as lead) at a young age.

If a learning disability is suspected, a developmental assessment will be carried out. Hearing, vision, and other medical and genetic tests will also be done to check for underlying physical causes of the learning difficulties.

COMMON SPECIFIC LEARNING DISABILITIES

TYPE	DESCRIPTION
Dyslexia	Impaired ability to learn to read and/or write. In addition to poor reading and spelling, there may also be difficulty with sequences, such as date order, and problems with organizing thoughts.
Dyscalculia	Difficulty performing mathematical calculations and trouble learning mathematical concepts, such as quantity and place value, and with organizing numbers.
Amusia	Commonly called tone deafness, the inability in a person with normal hearing to recognize musical notes, rhythms, or tunes or to reproduce them.
Dyspraxia	The inability to make skilled movements with accuracy. It can cause difficulty with establishing spatial relationships, such as positioning objects accurately.
Specific language impairment	Difficulties with understanding and/or expressing oral language in a child with no physical impediment to hearing or speaking and no generalized developmental delay.

CONDUCT DISORDER

CONDUCT DISORDER IS A BEHAVIORAL DISORDER IN WHICH A CHILD OR ADOLESCENT REPEATEDLY AND PERSISTENTLY BEHAVES IN A WAY THAT IS ANTISOCIAL.

Various factors put a child at increased risk of conduct disorder, including genetic factors, an unstable and/or violent family life, lack of supervision, abuse, and bullying. Learning disabilities (see above), attention deficit hyperactivity disorder (see p.246), and mental health problems such as depression also increase the risk. Children with conduct disorder also tend to have abnormal responses to reward and punishment.

Symptoms and effects

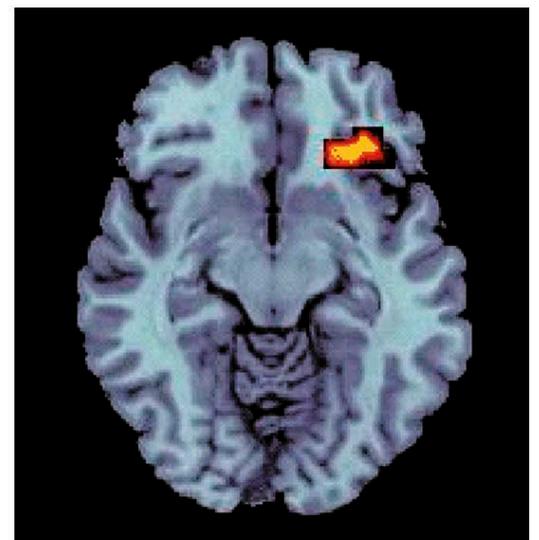
Symptoms vary from individual to individual, but they include aggressive behavior, physical cruelty, theft or persistent lying, deliberate destruction of property, and violations of rules, such as playing truant from school. In

some cases, a child may also engage in alcohol or drug abuse. Many children act in an antisocial or disruptive way from time to time, but in a child with conduct disorder, the behavior occurs repeatedly over a period of several months or longer. As a result of such behavior, a child may find it difficult to make friends, have low self-esteem, and do poorly at school.

A diagnosis is usually based on a psychiatric assessment of the child's behavior patterns. Treatment of conduct disorder, through talking therapies such as cognitive-behavioral therapy, can be difficult, but early treatment is more likely to be effective. It is important that parents are involved in the treatment.

REDUCED BRAIN ACTIVITY

Children with conduct disorder tend to show reduced activity in the right orbitofrontal cortex (orange in this fMRI scan) when rewarded for a task. This supports the idea that this disorder arises from abnormal responses to the rewards and punishments that normally shape behavior.



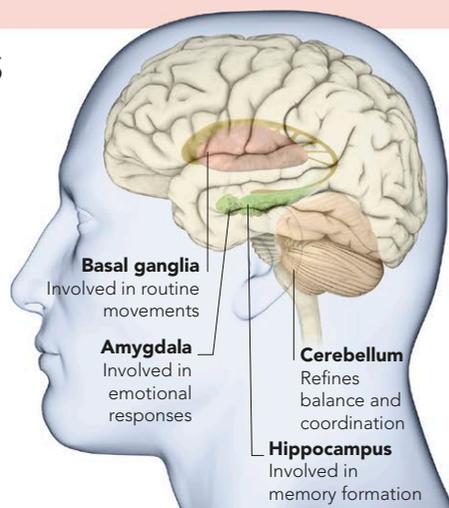
AUTISM SPECTRUM DISORDERS

THIS IS A GROUP OF DEVELOPMENTAL DISORDERS CHARACTERIZED BY PROBLEMS WITH COMMUNICATION, SOCIAL RELATIONSHIPS, AND REPETITIVE BEHAVIOR.

There are several types of autism spectrum disorders, but the main ones are autistic disorder (sometimes referred to as “classic” autism) and high-functioning autism.

Autistic disorder usually appears in early childhood, before the age of about three years. It produces problems in three main developmental areas: impaired social skills, impaired communication, and restricted behavior. Typically, such children fail to respond to their name or to other speech directed at them; avoid eye contact; resist physical contact; start talking late and speak with an abnormal tone or rhythm; show abnormal response to social cues, such as faces and voices; perform repetitive movements, such as rocking; develop specific routines and become disturbed when they are changed; and may be unusually sensitive to sound, light, and touch but sometimes ignore sensory signals. About half of all children with autistic disorder have learning difficulties, and some children develop seizures.

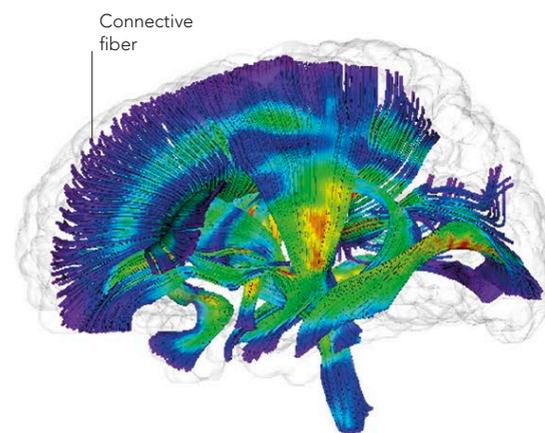
However, some children with autism have a high ability in one area, such as rote memory or precocious reading,



AFFECTED AREAS OF THE BRAIN

Autism has been associated with abnormalities in many brain regions (including those shown here), but their causal connection to autism is not yet clear.

and, rarely, a child may have an exceptional ability in a specific area (called savant syndrome), such as mathematics. Children with high-functioning autism tend to have similar symptoms but in a less severe form. Many children are of average or above average intelligence and develop speech and language skills at the normal time.



ORGANIZED CONNECTIONS

This diffusion tensor scan shows the clear, organized tracts of connective tissue in a healthy infant brain. These fibers are disorganized in a person that goes on to develop autism.

However, they have very narrow interests, find it difficult to interact socially with their peers, and are usually inflexible in their behavior and routines.

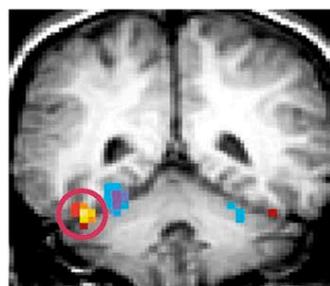
There is no cure for autism spectrum disorders, and treatment is based on supportive education to help a child reach his or her potential.

RARE AUTISM SPECTRUM DISORDERS

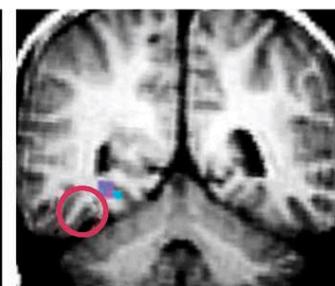
TYPE	DESCRIPTION
Rett syndrome	This autism spectrum disorder affects females almost exclusively and is caused by a mutation in a single gene. Typically, there is a period of normal development, but then autism-like symptoms begin to appear, usually between about six and 18 months of age. The child's development then regresses: she shies away from social contact and no longer responds to her parents. The child stops talking, if she had been talking before, loses coordination of her feet, has repeated writhing movements of her hands, and has inappropriate outbursts of crying or laughter.
Childhood disintegrative disorder	This very rare form of autism spectrum disorder primarily affects males. As with Rett syndrome, there is a period of normal development followed by the onset of autism-like symptoms and regression. Symptoms typically appear between the ages of three and four years, although they may sometimes appear as early as two years. There are extensive and severe losses of previously acquired social, language, and motor skills, and there may also be loss of bladder and bowel control, repetitive, stereotyped behavior patterns, seizures, and severe intellectual impairment.

RESPONSE TO FACES

In these two MRI scans the yellow and red colors show areas of brain activity when looking at faces. In a normal person, there is activity in the fusiform gyrus of the temporal lobe (circled) but no corresponding activity in the brain of a person with autism.



NORMAL BRAIN



AUTISTIC BRAIN

RESPONSE TO VOICES

These two scans show brain activity when normal people and those with autism listened to human voices. In the normal brain, the superior temporal sulcus was active (the yellow and red area), whereas there was no activity in that area in those with autism.



NORMAL BRAIN



AUTISTIC BRAIN

TEMPLE GRANDIN

One of the best-known writers on autism, Temple Grandin is herself a high-functioning autistic who has graphically described what it is like to have autism. Born in 1947 in the US, she was diagnosed with autism at the age of three. After a supportive early education, she attended ordinary schools, where she was often teased and picked on for being different. Nevertheless, she graduated from college and became a prominent researcher in animal science and welfare as well as an advocate for people with autism. In the field of animal welfare,

she considers her autism, hypersensitivity to stimuli, and unusual visual thought processes to be a positive advantage, giving her a unique insight into the stresses to which livestock are vulnerable. As a result of her early childhood experiences, Grandin is an advocate of early intervention and a supportive educational regime in autism, to help direct children with autism in productive directions. Even though autism affects every aspect of her life, Temple Grandin has said that she would not support a cure for all autism spectrum disorders.



UNIQUE INSIGHT

Temple Grandin became famous for her ability to understand animals' minds and use her insights to improve their lives. Today, she helps people on the autism spectrum to be more comfortable in the world.

GLOSSARY

A

acalculia The inability to perform numerical calculations due to neurological injury; see also *dyscalculia*.

acetylcholine A neurotransmitter that plays an important role not only in learning and memory but also in sending messages from the motor nerves to the visceral muscles.

action potential A brief pulse of electrical current that is generated by a neuron, and may be transmitted to neighboring cells.

adrenaline See *epinephrine* and *norepinephrine*.

afferent Traveling toward or entering; see also *efferent*.

agonist A molecule that binds to a receptor and stimulates the cell to fire; see also *antagonist*. An agonist is often a chemical that mimics the effect of a naturally occurring neurotransmitter.

agraphia The inability to write due to neurological injury.

alexia The inability to read due to neurological injury; also known as word blindness.

amnesia A general term for memory deficit.

amygdala A nucleus located in the limbic area of the temporal lobe that is crucial to emotion.

androgens The sex steroid hormones (including testosterone), which are responsible for male sexual maturation and associated with stereotypically masculine behavioral traits.

angular gyrus A ridge of the neocortex in the parietal lobe, next to the temporal and occipital lobe. It is concerned with the position of the body in space and linking sound and meaning.

anomia The inability to name objects.

anosmia The inability to smell.

anosognosia The failure, due to neurological injury, to be aware of a deficit in oneself, such as paralysis or blindness.

ANS See *autonomic nervous system*.

antagonist A molecule that blocks or prevents activation of a receptor.

anterior The front, or toward the front.

anterograde amnesia The loss of memory of things that occur after a brain injury, especially after concussion.

apraxia A partial or total inability to perform coordinated movements, including speech.

arachnoid membrane The middle of the three meninges (layers of tissue that cover the brain).

arcuate fasciculus The nerve-fiber tract that connects Broca's and Wernicke's areas.

ascending reticular formation A part of the reticular formation, responsible for the arousal and sleep-wake cycle.

association areas The regions of the brain that combine different types of information to produce a "whole" experience.

astrocyte A type of support cell that provides brain cells with nutrients and insulation.

ataxia A symptom of neurological disorder in which the sufferer experiences difficulty with balance and coordinated movement.

athetosis A condition in which muscles make slow, involuntary, writhing movements, seen in some forms of epilepsy.

attention deficit hyperactivity disorder (ADHD) A syndrome of learning and behavioral problems characterized by a short attention span and often by inappropriately energetic or frenzied activity. It usually occurs first in early childhood.

auditory cortex The region of the brain responsible for receiving and processing information relating to sound.

autonomic nervous system (ANS)

A component of the peripheral nervous system, responsible for regulating the activity of internal organs. It includes both the sympathetic and parasympathetic nervous systems.

axon The fiberlike extension of a neuron that carries electrical signals to other cells. Most neurons have only one axon.

B

basal ganglia A bundle of nuclei in the base of the forebrain, including the striatum and globus pallidus. It is primarily concerned with selecting and mediating movements.

bilateral On both sides of the body; for example, both brain hemispheres.

bipolar disorder An illness that is characterized by dramatic mood swings.

blindsight The ability to respond to visual stimuli in spite of being blind due to damage to the visual cortex.

blood-brain barrier A network of tightly packed cells surrounding the brain, which prevents toxic molecules from entering.

bottom-up Usually refers to relatively "raw" information flowing from the primary sensory areas of the brain rather than from areas involved in thinking, imagining, or creating expectations.

brainstem The lower part of the brain that becomes the spinal cord.

brainwaves The regular oscillations (firings) of neurons. Different rates of firing indicates different mental states; see also *electroencephalograph (EEG)*.

Broca's area A frontal-lobe brain region, concerned with articulating speech.

Brodman areas The microscopically distinct cortical areas that were mapped out by neurologist Korbinian Brodmann (1868–1918).

C

Capgras' delusion A rare syndrome in which people believe that a close friend or spouse has been replaced by a double. It is thought to be caused by damage to nerve pathways concerned with emotional recognition.

caudal Toward the tail end; see also *posterior*.

caudate nucleus A part of the striatum.

cell body The central structure of a neuron; also referred to as the soma.

central fissure Also called the central sulcus. A long, deep fissure that runs across the brain, dividing the parietal and frontal lobes.

central nervous system (CNS) The brain and spinal cord.

cerebellum The “small brain” behind the cerebrum that helps regulate posture, balance, and coordination.

cerebral cortex The outer, wrinkled “gray” part of the cerebral hemispheres.

cerebral hemispheres The two halves of the brain.

cerebrospinal fluid (CSF) The fluid found in the brain's ventricles, which brings nutrients to, and removes waste from, the brain.

cerebrum The major part of the brain, excluding the cerebellum and brainstem.

cerebellar peduncles The short, stalklike extensions of the cerebellum, which connect it to the brainstem.

cholinergic system The nerve pathways that are activated by the neurotransmitter acetylcholine.

cingulate cortex The area of cortex that makes up the sides of the longitudinal fissure. It is closely connected to the underlying limbic system as well as to cortical areas of the brain, and is important in combining “top-down” and “bottom-up” information to guide actions.

circadian rhythm A cycle of behavior or physiological change lasting about 24 hours.

cochlea The spiral-shaped bony canal in the inner ear, containing the hair cells that transduce sound.

cognition Conscious and unconscious brain processes, such as perceiving, thinking, learning, and remembering information.

commissurectomy The surgical severing of the corpus callosum.

computed tomography (CT) A scanning technique that uses weak levels of X-ray to produce images of the brain and body.

concussion A brain trauma, usually caused by a blow to the head and resulting in temporary loss of consciousness.

cone A color-sensitive receptor cell in the retina, used primarily for daytime vision.

contralateral On the other side of the body or brain. Damage to the brain often leads to problems on the contralateral side of the body; see also *ipsilateral*.

coronal A vertical “slice” through the brain, running parallel to the shoulders.

corpus callosum The thick band of nerve tissue that connects the left and right hemispheres of the brain and carries information between them.

cortex See *cerebral cortex*.

Cotard syndrome A rare disorder in which patients assert that they are dead, often claiming to smell rotting flesh or feel worms crawling over their skin.

cranial fossa The various bowl-shaped cavities in the skull. The posterior cranial fossa houses the brainstem and cerebellum.

cranial nerves The 12 pairs of nerves that arise from the brainstem. These include the olfactory nerve, which conveys information about smell to the brain, and the optic nerve, which carries data about vision.

cranium The skull.

D

decussation The crossing of nerve fibers, as in the optic chiasm.

delusion A false belief that is not easily eradicated by exposure to evidence that reveals its falsity.

dementia A loss of brain function due to degeneration through age or cumulative damage to the brain.

dendrite A branch that extends from a neuron's cell body and receives signals from other neurons.

dentate gyrus The part of the hippocampus containing nerve cells that receive input from the entorhinal cortex.

depression A common illness characterized by intense and chronically low mood and energy levels.

diencephalon A part of the brain that includes the thalamus and the area that surrounds it.

dopamine A neurotransmitter that produces motivation and strong feelings of pleasurable anticipation.

dorsal At or toward the (upper) back.

dorsal horn The back part (in cross section) of the spinal cord, where nerve fibers, especially pain-carrying fibers, merge with the spinal cord to travel upward toward the brain.

dorsal route The pathway in the visual system that connects the visual cortex to the parietal lobe, also referred to as the “where” or “how” pathway; see also *ventral route*.

dorsolateral prefrontal cortex The area of the frontal lobe concerned with planning, organization, and various other executive functions of cognition.

dura mater The top of the three layers of tissue separating the brain from the skull; see also *meninges*.

dyscalculia A condition associated with difficulty in learning simple arithmetical operations in the absence of any other intellectual problems.

dyslexia A condition associated with difficulty in learning to read and write in the absence of any other intellectual problems.

E

EEG See *electroencephalograph*.

efferent Leading away from; see also *afferent*.

electroencephalograph (EEG) A graphic record of the electrical activity of the brain, made by attaching electrodes to the scalp that pick up the underlying brainwaves.

encephalin A type of endorphin.

encephalitis Inflammation of the brain.

endorphins A group of chemicals produced by the brain, which produce effects similar to those of opium.

entorhinal cortex The main route for information entering the hippocampus.

epilepsy An illness characterized by repeated seizures.

epinephrine and norepinephrine Hormones and neurotransmitters secreted by the adrenal gland; also referred to as adrenaline and noradrenaline.

event-related potential (ERP) The neural activity generated in response to a given stimulus recorded by EEG.

excitatory neurotransmitter A type of neurotransmitter that encourages neurons to fire; see also *inhibitory neurotransmitter*.

explicit memory The memories that can be consciously retrieved and reported.

F

fissure A deep cleft, or sulcus, on the surface of the brain.

fMRI See *functional magnetic resonance imaging*.

forebrain A major part of the brain, including the cerebrum, thalamus, and hypothalamus.

fornix An arching band of nerve tissue that carries signals around the limbic system from the hippocampus at one end, to the mammillary bodies at the other.

fovea The central part of the retina, composed of densely packed cones. It is the area of the retina that has the highest visual acuity.

frontal lobe The area at the front of the brain, responsible for thinking, making judgments, planning, decision-making, and conscious emotion.

functional imaging A range of techniques that allow neural activity to be measured and shown as visual images.

functional magnetic resonance imaging (fMRI) A brain-imaging technique in which magnetic resonance imaging is used to measure the changes in blood properties associated with neural activity; see also *magnetic resonance imaging*.

fusiform gyrus A long cortical bulge on the underside of the temporal lobe, important for object and face recognition; see also *ventral route*.

G

gamma-aminobutyric acid (GABA) The major inhibitory neurotransmitter in the brain.

ganglion A cluster of interactive nuclei. The term also refers to light-sensitive cells in the retina.

Geschwind's territory A region of the brain concerned with language.

glial cells Also referred to as glia, the brain cells that support neurons by performing a variety of "housekeeping" functions in the brain. They may also mediate signals between neurons.

globus pallidus A part of the basal ganglia involved in movement control; see also *basal ganglia*.

glutamate The most common excitatory neurotransmitter in the brain.

grand mal See *seizure*.

gray matter The darker tissues of the brain, made up of densely packed cell bodies, as seen in the cortex.

gustatory cortex The area of the brain responsible for processing taste.

gyrus (pl. gyri) The bulges of tissue on the surface of the brain.

H

hallucination A false perception that occurs in the absence of any sensory stimuli.

hemiplegia A condition in which there is paralysis of one half of the body.

hemisphere One half of the brain.

hindbrain The back part of brain, adjoining the spine, which includes the cerebellum, pons, and medulla.

hippocampus A part of the limbic system lying on the inside of each temporal lobe. It is crucial for spatial navigation and encoding and retrieving long-term memories.

hormones The chemical messengers secreted by endocrine glands to regulate the activity of target cells. They play a role in sexual development, metabolism, growth, and many other physiological processes.

hypothalamus A cluster of nuclei that controls many body functions, including feeding, drinking, and the release of many hormones.

I

illusion A false perception or distortion of the senses often caused by unconscious brain processes.

implicit memory The memories that cannot be retrieved consciously, but are activated as part of particular skills or actions, or in the form of an emotion linked to an event that cannot be made conscious. Implicit memories underlie the learning of physical skills such as playing a ball game or tying a shoelace; see also *procedural memory*.

inferior Below or underneath.

inferior colliculi The principal midbrain nuclei of the auditory pathway.

inhibitory neurotransmitter A type of neurotransmitter that stops neurons from firing; see also *excitatory neurotransmitter*.

insula Also referred to as the insular cortex, the brain region that lies in a deep recess between the temporal and frontal lobes.

intelligence quotient (IQ) A score based on a range of tests that represents the relative intelligence of a person.

interneuron A “bridging” neuron connecting afferent and efferent neurons.

ipsilateral On the same side of the body as that in which a condition occurs; see also *contralateral*.

IQ See *intelligence quotient*.

K

Korsakoff syndrome A brain disease that is associated with chronic alcoholism. The symptoms include delirium, insomnia, hallucinations, and a lasting amnesia.

L

lateral On or to the side.

lateral geniculate nucleus (LGN) A nucleus in the thalamus that acts as a relay in the visual pathway.

lesion An area of injury or cell death.

limbic system A set of brain structures lying along the inner border of the cortex, crucial for emotion, memory, and mediating consciousness.

lobe One of four main areas of the brain that are delineated by function (occipital, temporal, parietal, and frontal).

longitudinal fissure Also called the longitudinal sulcus, the deep groove that marks the division of the two cerebral hemispheres.

long-term memory The final phase of memory, in which information storage may last anywhere from hours up to a lifetime.

long-term potentiation (LTP) A change in a neuron that increases the likelihood of it firing in unison with one that it has fired with before.

M

magnetic resonance imaging (MRI) A brain-imaging technique that provides high-resolution pictures of brain structures.

magnetoencephalography (MEG)

A non-invasive functional brain-imaging technique that is sensitive to rapid changes in brain activity. Recording devices (SQUIDS) measure small magnetic fluctuations associated with neural activity in the cortex and present these in visual form.

magnocellular The pathways from large retinal ganglion cells to cortical visual areas. They are sensitive to movement.

mamillary bodies The small limbic-system nuclei that are concerned with emotion and memory.

medial In the middle.

medulla Also known as the medulla oblongata or myelencephalon. A part of the brainstem situated between the pons and the spinal cord. It is responsible for maintaining vital body processes, such as breathing and heart rate.

melatonin A hormone that helps regulate the sleep–wake cycle. It is produced by the pineal gland.

meninges The three layers of protective tissue between the brain and the skull.

mesencephalon Also referred to as the “midbrain,” the area of the brain between the forebrain and the brainstem, involved in eye movement, body movement, and hearing. It includes the basal ganglia.

midbrain See *mesencephalon*.

mind The thoughts, feelings, beliefs, intentions, and so on, that arise from the processes of the brain.

motor cortex The region of the brain containing neurons that send signals, directly or indirectly, to the muscles. It stretches around the brain like a horseshoe.

motor neuron A neuron that infiltrates muscle and causes it to contract or stretch.

MRI See *magnetic resonance imaging*.

myelencephalon See *medulla*.

myelin The fatty material that surrounds and insulates the axons of some neurons.

N

narcolepsy An illness characterized by uncontrolled bouts of sleeping.

near-infrared spectroscopy (NIRS) A functional imaging technique that shows varying levels of oxygen use in the brain (a marker of neural activity) by measuring the reflection of near-infrared light from cerebral tissues.

neocortex The wrinkled outer layer of the brain; also referred to as the cerebral cortex.

nervous system The nerve cells that connect to the brain and extend throughout the entire body. They are grouped into the central nervous system (CNS) and the peripheral nervous system (PNS).

neurogenesis The generation of new neurons in the brain.

neuron Also referred to as a nerve cell, a brain cell that signals to others by generating and passing on electrical signals.

neurotransmitter A chemical secreted by neurons that carries signals between them across synapses.

nociceptive Responding to painful or noxious stimuli.

norepinephrine An excitatory neurotransmitter, also known as noradrenaline; see also *epinephrine*.

nucleus A bound cluster or group of nerve cells with specialist functions.

nucleus accumbens A limbic-system nucleus that processes information related to motivation and reward.

O

occipital lobe The back part of the cerebrum, mainly dedicated to visual processing.

olfactory nerve/system The nerve/body system that responds to smell molecules.

opium A drug derived from poppy seeds that produces intense euphoria, pain relief, and relaxation.

optic chiasm The point of decussation (crossing) of the optic nerves from each eye; see also *decussation*.

optic nerve A bundle of nerve fibers carrying signals from retinal ganglion cells into the main part of the brain for processing.

oscillations The rhythmic firings of neurons.

oxytocin A neurotransmitter involved in social bonding.

P

parasympathetic nervous system A branch of the autonomic nervous system, concerned with the conservation of the body's energy. It inhibits the sympathetic nervous system.

parietal lobe The top-back subdivision of the cerebral cortex, mainly concerned with spatial computation, body orientation, and attention.

Parkinson's disease An illness characterized by tremors and slowness of action; it is thought to be caused by degeneration of dopamine-producing cells.

parvocellular The nerve pathways from small areas of the retina to cortical visual areas. They are sensitive to color and form.

peptides The chains of amino acids that can function as neurotransmitters or hormones.

peripheral nervous system (PNS) The part of the nervous system that includes all nerves and neurons outside the brain and spinal cord.

PET See *positron emission tomography*.

phantom limb An absent limb (usually amputated) that the person continues to experience as part of the body.

pia matter The innermost layer of the meninges; a thin, elastic tissue that covers the surface of the brain.

pineal gland A pea-sized gland located near the thalamus that produces melatonin, which regulates the sleep-wake cycle.

pituitary gland A hypothalamic nucleus

that produces hormones, including oxytocin.

plasticity The capacity of the brain to change its structure and function.

pons A part of the hindbrain lying in front of the cerebellum.

positron emission tomography (PET) A functional imaging technique for measuring brain function in living subjects by detecting the location and concentration of small amounts of radioactive chemicals associated with specific neural activity.

posterior Toward the back or tail end. Also referred to as "caudal."

postsynaptic neuron A neuron that receives messages from another; see also *presynaptic neuron*.

prefrontal cortex The region of the brain in the forward-most part of the frontal cortex, involved in planning and other higher-level cognition.

premotor cortex A part of the frontal cortex concerned with planning movements.

presynaptic neuron A neuron that releases a neurotransmitter to carry signals across a synapse to another neuron; see also *postsynaptic neuron*.

primary cortex A region of the brain that first receives sensory information from organs, such as the primary visual cortex.

procedural memory A form of implicit memory relating to learned movements, for example, riding a bicycle.

proprioception Sensory information relating to balance and the position of the body in space.

prosopagnosia Inability to recognize faces.

psychasthenia A condition in which the sufferer experiences heightened sensitivity to negative stimuli, resulting in chronic anxiety.

psychedelic A drug that distorts perception, thought, and feeling.

psychoactive Changing brain function, usually referring to drugs.

psychosis A condition in which a person loses touch with reality.

psychotherapy The treatment of a mental disorder using psychological rather than medical methods.

putamen A part of the striatum, which itself is part of the basal ganglia, that is mainly concerned with regulating movement and procedural learning.

pyramidal neuron An excitatory neuron with a distinctive triangular body, found in the cortex, hippocampus, and amygdala.

Q

qualia The conscious, subjective sensations that arise from stimulation of sense organs, for example, pain, warmth, or seeing a color.

R

raphe nuclei The brainstem nuclei that mainly release serotonin and have wide-ranging effects on mental function.

rapid eye movement (REM) A phase of sleep characterized by rapid eye movements and vivid dreams.

reflex An involuntary movement, controlled by neurons in the spinal cord.

reticular formation A complex area in the brainstem containing various nuclei that affect arousal, sensation, motor function, and vegetative functions such as heartbeat and breathing.

retina The part of the eye containing light-sensitive cells, which send electrical signals to the visual area of the brain for processing into visual imagery.

reuptake The process by which excess neurotransmitters are removed from the synapse by being carried by transporter cells back into the axon terminals that first released them.

rhombencephalon See *hindbrain*.

rod A sensory neuron in the outer edge of the retina. It is sensitive to low-intensity light and is specialized for night vision.

rostral Toward or at the front side of the body; see also *anterior*.

S

sagittal A vertical plane passing through the brain from front to back. The midsagittal, or median, plane splits the brain into left and right hemispheres.

schizophrenia An illness characterized by intermittent psychosis.

seizure A disruption of normal neural activity. Grand mal seizures involve widespread synchronous neural firing, which produces unconsciousness.

serotonin A neurotransmitter that regulates many functions, including mood, appetite, and sensory perception.

short-term memory A phase of memory in which a limited amount of information may be held for several seconds to minutes; see also *working memory*.

single photon emission computed tomography (SPECT) An imaging process that measures the emission of single photons of a given energy from radioactive tracers in the brain, giving a measure of neural activity.

somatosensory cortex An area of the brain concerned with receiving and processing information about body sensations, such as pain and touch.

SPECT See *single photon emission computed tomography*.

SQUIDS See *magnetoencephalography*.

striate cortex An area of the visual cortex characterized (in cross section) by visually distinct strips of cells.

striatum A structure in the basal ganglia composed of the caudate and the putamen.

sulcus (pl. sulci) A valley or groove in the brain surface (the opposite of gyrus).

superior Toward or at the top.

superior colliculi Paired structures of nuclei of the midbrain that play a part in relaying visual information.

supplementary motor cortex An area in the front of the motor cortex involved in planning actions that are under internal control, such as actions done from memory rather than guided by current sensations.

survival value The benefit of a physical or behavioral characteristic to an individual's chances of surviving and reproducing.

sympathetic nervous system A part of the autonomic nervous system that speeds up heart rate, among other things, in response to stimulation; see also *parasympathetic nervous system*.

synesthesia The experience of having two or more senses “blended” in response to a stimulus—for example, a shape might be tasted as well as seen, or a sound may be seen as well as heard.

synapse A gap between two neurons that is bridged by neurotransmitters.

T

tegmentum The lower-back part of the midbrain.

telencephalon The largest part of the brain; see also *cerebrum* and *forebrain*.

temporal lobe A division of the cerebral cortex at the side of the head, concerned with hearing, language, and memory.

thalamus Large paired masses of gray matter lying between the brainstem and the cerebrum, the key relay station for sensory information flowing into the brain.

TMS see *transcranial magnetic stimulation*.

top-down A phrase used to distinguish “processed” information or knowledge that is used to interpret “raw” sensory data.

transcranial magnetic stimulation (TMS)

A method by which electrical activity in the brain is influenced by a magnetic field, usually generated by a wand held on the scalp.

U

unilateral On one side of the body; see also *bilateral*.

V

V1 The primary visual cortex—other visual areas are often referred to as V2, V3, V4, and so on.

ventral Toward the lower, front surface (such as the abdomen of an animal).

ventral route The pathway in the visual system that connects the visual cortex to the temporal lobe, concerned with the recognition of objects and faces.

ventral tegmental area (VTA) A group of dopamine-containing neurons that make up a key part of the brain's reward system.

ventricle A cavity within the brain containing cerebrospinal fluid.

ventromedial prefrontal cortex A part of the prefrontal cortex, associated with emotions and judgment.

visual cortex The surface of the occipital lobe in which visual information is processed.

W

Wernicke's area The major language area, in the temporal lobe, concerned with comprehension. In most people, it is situated in the left hemisphere, near the junction with the parietal lobe.

white matter A type of brain tissue that is made up of densely packed axons that carry signals to other neurons. It is distinguished from cell bodies by the lighter color. White matter generally lies beneath the gray matter that forms the cortex.

working memory A process by which information is held “in mind” as active neural traffic until it is forgotten, or encoded in long-term memory.

INDEX

Page numbers in **bold** indicate extended treatments of a topic.

A

- A-delta fibers, pain signals 107
 abducens nerve 43
 abortion 236
 abscesses 227, **228**
 absence seizures 226
 abuse, childhood 197
 acetylcholine 73
 Alzheimer's disease 231
 executing a movement 119
 acrophobia 240
 ACTH (adrenocorticotropic hormone) 61, 114, 240
 action potentials 72
 adaptive behaviors, emotions 129
 addictions **245**
 cocaine 130
 oxytocin 137
 pleasure-seeking **130**
 to sugar **115**
 adenosine, sleep-wake cycles 188
 ADH (antidiuretic hormone) 61, 114, 115
 adipose tissue 102
 adolescence
 brain development **210–211**
 emotions 39
 adrenal glands 114, 240
 adrenaline (epinephrine) 114
 and heat production 113
 stress response 240
 thrill-seeking 131
 adrenocorticotropic hormone (ACTH) 61, 114, 240
 adults, brain development **212–213**
 aerobic exercise, effect on brain 44
 after-images 174
 aging 44, **214–215**
 agency, self-consciousness **193**
 aggression
 amygdala and 127
 body language 144
 personality markers 200
 agnosia, visual 85
 agouti-related peptide (AgRP) 115
 alcohol
 addiction to 44, 245
 and brain shrinkage 44
 intoxication test 162
 and proprioception 104
 alertness 186
 alien beings 173
 Alpha waves 181
 and creativity 170
 alphabet 153
 AlphaGo 217
 altering consciousness **186–187**
 alternating attention 182
 altruism **141**
 Alzheimer, Alois 9
 Alzheimer's disease 230, **231**
 tangles and plaques 214, 215
 ambiguous illusions **174**
 American Psychiatric Association 222
 amino acids 73
 amnesia, head injuries 225
 amniocentesis 236
 amphibians, brains 48
 amputation, phantom limbs **104**, 193
 amusia 248
 amygdala 53, 74, **127**, 209
 anticipation 130
 and body language 144
 and depression 239
 and dreams 189
 emotions 39, 126–129, 139, 212, 213
 functions 58
 gender differences 198
 and humor 171
 imaging **24**
 limbic system 64, 65
 memory 156, 160, 161
 mindfulness 187
 morality 140
 and optimism 200
 pain signals 109
 phobias 127
 post traumatic stress disorder 241
 recognizing faces 84
 self-awareness 139
 sense of smell and 97
 stria terminalis 126
 amyotrophic lateral sclerosis (ALS) 235
 anesthesia, general **112**
 analgesia, congenital 109
 anarchic hand syndrome (AHS) 57
 anatomy **50–73**
 brain cells **70–71**
 brain structures **52–55**
 brain zones and partitions **56–57**
 brain stem and cerebellum **62–63**
 cerebral cortex **66–69**
 early study of 8
 limbic system **64–65**
 nerve impulses **72–73**
 nuclei **58–59**
 scanning the brain 12
 speech 147
 thalamus, hypothalamus, and pituitary gland **60–61**
 androstadienone 99
 androstenedione 99
 anencephaly 237
 aneurysm
 ruptured 229
 subarachnoid hemorrhage 229
 anger
 adaptive behaviors 129
 facial expressions 136
 angiotensin II 115
 "animal magnetism" 222
 animals
 evolution of brain **48–49**
 sense of smell **98**
 social behavior 138
 understanding 249
 anorexia nervosa 246
 ANS see autonomic nervous system
 anterior cingulate cortex (ACC)
 creativity 170
 emotions 126, 128
 imaging **20**
 and optimism 200
 anterior cingulate cortex (ACC)
 continued
 and pain 108, 109
 self-awareness 138, 139
 self-consciousness 192, 193
 and stress 199
 anterior commissure, gender differences 198
 anterior fissure, spinal cord 42
 anterior intraparietal area (AIP) 83
 anterior nucleus, hypothalamus 113
 anticipation 127, **130**
 antidiuretic hormone (ADH) 61, 114, 115
 antisocial personality disorder 245
 anxiety
 anxiety disorders 168, **240–241**
 eating disorders 246
 and experience of pain 109
 and meditation 187
 obsessive-compulsive disorder (OCD) **241**
 phobias **240**
 post traumatic stress disorder (PTSD) **164**, **241**
 stria terminalis 126
 aphasia 149, 151
 apolipoprotein E 231
 apoptosis 209
 appearance, body dysmorphic disorder **242**
 appeasement 129
 apperceptive agnosia 85
 appetite control 115
 arachnoid
 arachnoid granulations 45
 skull 56
 spinal cord 42
 arachnophobia 240
 arbor vitae 63
 archetypes, collective unconsciousness 187
 arcuate fasciculus 148, 151
 Aristotle 6, 8
 arms
 bionic arms **219**
 executing a movement 118
 motor neuron disease 235
 phantom limbs **104**, 193
 arteries 46–47
 arteriovenous malformation 229
 carotid arteries 146
 Circle of Willis 45
 stroke **229**
 transient ischemic attacks 228
 artificial intelligence **217**
 artists, creativity 170
 aspartate 73
 aspartic acid 73
 Asperger's syndrome 139, 172
 association, accessing memories 159
 association areas **68**
 emotions 129
 perceptions 39
 associative agnosia 85
 astraphobia 240
 astrocytes 71, 213
 asymmetry **57**
 ataxic cerebral palsy 237
 atherosclerosis 228
 athetoid cerebral palsy 237
 athletes 196
 atonic seizures 226
 ATP 106
 atriopeptin 114
 atrioventricular node 112
 attention
 and consciousness **182–183**
 disorders 168, 183, **246**
 laying down a memory 160
 attention deficit hyperactivity disorder (ADHD) 183, **246**
 attraction, sexual 134
 auditory canal, hearing 94
 auditory cortex **91**
 and conversation 151
 hearing 94, 95
 laying down a memory 161
 music 93
 perception of sound 92
 reading 152
 auditory frequency ranges 91
 auditory nerve 90
 aura 172
 epileptic seizures 226
 migraine 224
 auricular cartilage 90
 autism **139**, 223
 autism spectrum disorders **249**
 autistic savants 164–165, 174, 249
 high-functioning autism 249
 hyperlexia 153
 patternmaking 172
 autoimmune diseases
 multiple sclerosis **235**
 narcolepsy **238**
 automata **75**
 automatic movements 116
 autonomic nervous system (ANS) 40
 heart rate regulation **112**
 hypothalamus and 113
 phobias 127
 reticular formation 112
 autopilot 185
 aversion
 aversion behavior 129
 flavor-aversion learning 101
 aviophobia 240
 avoidant personality disorder 245
 awareness 186
 agency and intention 193
 and consciousness 180
 types of 179
 axodendritic synapses 71
 aging 214
 in brain stem 62
 cerebellum 63
 myelin sheaths 70, 71, 212
 nerve impulses 72, 73
 spinal cord 42
 synapses 71
 types of neurons 71
 axospinodendritic synapses 71
 Baader-Meinhof Gang 205
 babies
 body maps 193
 bonding with 135, 213
 development of hearing **93**
 developmental delay **247**
 babies *continued*
 infant brain **208–209**
 language development **209**
 maternal love 134
 number sense 169
 personality development 200
 sense of smell 99
 stress 199
 backdating time **191**
 bacteria
 brain abscesses 228
 meningitis 227
 balance 118
 postural instability 119
 proprioception 104, 105
 vestibulocochlear nerve 43
 ballism 119
 basal nuclei (ganglia) 53, **58**, 211, 212
 aging 214
 and brain "clocks" 190
 imaging **23**
 planning movement 117
 Tourette's syndrome 243
 bases, DNA 196, 197
 basilar membrane 90
 behavior and personality **194–205**
 adaptive behaviors 129
 autism spectrum disorders **249**
 aversion behavior 129
 conduct disorder **248**
 dementia **230**
 influencing the brain **198–199**
 morality **140–141**
 nature and nurture **196–197**
 obsessive-compulsive disorder (OCD) **241**
 personality **200–201**
 personality disorders **245**
 state-dependent memory 162
 strange brains **204–205**
 belief systems **172–173**
 belladonna 144
 bereavement 239
 Berger, Hans 9
 berry aneurysm 229
 beta amyloid 231
 Beta waves, and consciousness 181
 biceps muscle,
 proprioceptors 104
 bilingualism 149
 binge-eating disorder 246
 binocular rivalry 87
 biogenic amines 73
 biometric sensors 129
 bionic arms **219**
 bionic eyes **218**
 biotechnology **218–219**
 bipolar disorder 223, **239**
 bipolar neurons 71
 birds, brains 49
 bladder
 multiple sclerosis 235
 panic responses 127
 bleeding see hemorrhage
 "blind" smell **98**
 blind spot, retina 81
 blindness 78
 bionic eyes **218**
 blindsight **79**
 seeing with sound **89**

- Bliss, Timothy 9
 blood clots
 stroke 229
 subdural hemorrhage **229**
 transient ischemic attacks 228
 blood supply
 functional magnetic resonance
 imaging 13
 glucose 45
 oxygen in 45
 blood vessels
 arteries 46–47
 arteriovenous malformation 229
 Circle of Willis 45
 hemorrhage 225
 panic responses 127
 stroke **229**
 and temperature control 113
 transient ischemic attacks **228**
 vascular dementia 230
 Blue Brain Project 75
 body clock 63
 body dysmorphic disorder (BDD)
242
 body language **144–145**
 and conversation 150
 tennis players 120
 “body maps,” self-consciousness
 192, 193
 body mass index (BMI) 246
 body memories 157, 160
 body weight, and brain
 weight 44
 bonding
 love and 134
 oxytocin **135**
 bones
 in ear 90, 94
 skull 53, 54
 borderline personality disorder
 245
 bottom-up processing **79, 87**
 bovine spongiform
 encephalopathy (BSE) 231
 bowels, panic responses 127
 bradykinesia 119
 bradykinin 106
 brain death **238**
 Brain Research through Advancing
 Innovative Neurotechnologies
 (BRAIN) 75
 brain waves
 and consciousness 181
 and creativity 170
 electroencephalographs 12
 “brain-in-a-vat” **180**
 brain-machine interfaces **216**
 brain stem 52, **62–63**, 208
 anatomy **62**
 brain death 238
 coma 238
 emotions 128, 129
 executing a movement 118
 functions 38, **63**
 hearing 94
 imaging **27**
 locked-in syndrome **63**
 reticular formation **112**
 breastfeeding 135
 breathing
 panic responses 127
 regulation of 112
 Broca, Paul 9, **10**
 Broca’s area 10, **148**
 aphasia 149
 imaging **25**
 and memory 157, 203
 reading 152
 speech 151, 203
 Brodmann areas **67, 68**
 Brodmann, Korbinian 9, **67**
 brown fat 113
 BSE (bovine spongiform
 encephalopathy) 231
 bulbous corpuscles 102
 bulimia nervosa 246
C
 C-fibers, pain signals 107
 calcium
 Alzheimer’s disease 231
 executing a movement 119
 ion channels 65
 camphor 96
 cancer
 brain tumors 230
 fear of 240
 carbohydrates 45
 carbon dioxide, regulation
 of breathing 112
 carcinophobia 240
 carotid arteries
 stroke 229
 transient ischemic attacks 228
 Wada test 146
 cars, driving 116
 cartoons, humor 171
 cataplexy 238
 catatonia **190**
 catatonic schizophrenia 244
 cats
 brains 49
 expressions 136
 caudate nucleus 52, 54, 58
 and memory 156, 157
 and obsessive-compulsive
 disorder 241
 and stress 199
 cells
 cell membrane 70
 cerebral cortex 68–69
 and consciousness 181
 retina 81
 stem cells 218
 support cells 68, 71
 see also glial cells; neurons
 central executive, memory 157
 central nervous system (CNS)
40–41
 evolution of 48
 central sulcus, imaging **29**
 cerebellar cortex 63, 117
 cerebellar hemispheres see
 cerebellum
 cerebellar peduncles 62
 cerebellum 52, 53, 55, 56, 62,
63, 69, 208
 anatomy 63
 and conversation 151
 disorders 119
 functions 63
 imaging **32**
 internal structure 63
 and memory 156, 157
 planning movement 117
 cerebral cortex 55, **66–69**
 association areas **68**
 and consciousness 180
 cortical folding **69**
 cortical functioning **69**
 cortical layers 68, 69
 development 208
 evolution 49
 functional areas **67**
 functions 57
 hippocampus 65
 imaging **18**
 landmarks **66**
 limbic lobe 65
 memory 157
 pain signals 106, 108
 sense of taste 101
 structure **68–69**
 cerebral hemispheres see
 hemispheres; left
 hemisphere; right hemisphere
 cerebral palsy **237**
 cerebrospinal fluid (CSF) 44, 57
 aging 215
 detritus 188
 flow of **45**
 hydrocephalus **237**
 lumbar puncture 227
 protection of brain 45
 in spinal cord 42, 45
 cerebrum 53, 208, 209
 see also hemispheres; left
 hemisphere; right hemisphere
 cervical region, spinal nerves 42
 cervical spinal cord 53
 cervical vertebra 53
 Chaplin, Charlie 199
 Charcot, Jean-Martin 242
 chess 217
 chest, motor neuron disease 235
 chicken pox virus 227
 childbirth 135
 postpartum depression 239
 children
 attention deficit hyperactivity
 disorder (ADHD) **246**
 autism spectrum disorders **249**
 brain development **210–211**
 childhood disintegrative
 disorder 249
 conduct disorder **248**
 developmental delay **247**
 dyslexia 153
 maternal love 134
 personality development 200
 reading and writing 152
 see also babies
 chimpanzees
 brains 49
 language areas 147
 Chinese room, theory of
 consciousness **179**
 chloride ions, nerve impulses 72
 chloroform 112
 cholinergic circuits 130
 chorea 58, 119, 234
 choroid, eye 81
 choroid plexuses, cerebrospinal
 fluid 45
 chromosomes 196
 Down syndrome **236**
 chronic fatigue syndrome **225**
 cilia, hair cells 94
 cingulate cortex
 and aggression 200
 and depression 239
 emotions 126
 self-consciousness 192
 social cognition 184
 see also anterior cingulate
 cortex; posterior cingulate
 cortex
 cingulate gyrus 64, 65
 cingulate sulcus 65
 circadian rhythm 63
 Circle of Willis 45, 46
 circuits
 emotion circuits 128
 forming memories 158
 CJD (Creutzfeldt-Jakob
 disease) **231**
 clairvoyance **173**
 claustrophobia 240
 “clocks” 190
 clonic seizures 226
 clots see blood clots
 cluster headaches **224**
 CNS see central nervous system
 cooperation, personality
 markers 200
 coordination, multiple
 sclerosis 235
 cocaine addiction 130
 coccyx 42
 cochlea 90, 92, 94
 cochlear duct 90
 cochlear implants **91, 94**
 cochlear nerve 90, 92, 94, 95
 cochlear nucleus 95
 cocktail-party effect 92
 cognitive illusions **174–175**
 cold, thermoreceptors 102, 113
 collateral fissure 65
 collective unconscious **187**
 color
 color vision **83**
 illusions 87
 language and 147
 visual perception 87, 89
 column of fornix 64
 coma 225, **238**
 commitment 134
 communication **142–153**
 autism 223
 body language and gestures
144–145
 conversation **150–151**
 expressions **136–137**
 gestures and body limbic
 system **144–145**
 limbic system areas **148–149**
 origins of language **146–147**
 reading and writing **152–153**
 smell and **99**
 complex partial seizures 226
 complicated grief **130**
 comprehension, and aging 215
 compulsions 241
 computed tomography (CT) scans
 12, 222
 computers
 artificial intelligence **217**
 brain-machine interfaces 216
 consciousness 219
 concentration 179, 186
 concha, ear 94
 concrete-mindedness 172
 concussion 225
 conduct disorder 245, **248**
 conduction aphasia 149
 cone cells, retina 81, 88
 congenital analgesia 109
 congruence 139
 conjunctiva, eye 80
 connective tissue
 support cells 68, 71
 white matter 69
 connectivity 39
 Connectome 9, **74**
 conscious awareness 179, 180
 conscious movement 116, 117
 conscious vision 84
 consciousness 6, **176–193**
 altering **186–187**
 attention and **182–183**
 dissociation **186**
 emotions 126, **128–129**
 investigating the brain 8
 and language 146
 levels of 192
 locating **180–181**
 in machines 219
 proprioception 104
 requirements of **181**
 self **192–193**
 sensory information **79**
 sleep and dreams **188–189**
 time **190–191**
 types and levels of **179**
 what is consciousness? **178–179**
 constellations, mental
 disorders **223**
 contrecoup injury 225
 control see movement and
 control
 conversation **150–151**
 conversion disorder 242
 coprolalia, Tourette’s syndrome 243
 cornea, eye 80
 corpus callosum 52, 53, 56, 74,
 204, 212
 altered brain-states 186
 and emotions 126
 gender differences 198
 split brain 204
 corpus striatum 58
 cortex see cerebral cortex and
 individual cortices
 cortisol, stress response 240
 Cottingley fairies 173
 covert attention 182
 cranial nerves 42, **43**, 53, 55, 208
 brain stem 62
 peripheral nervous system 40
 taste and smell 101
 craniotomy 11, 232
 creativity **170–179**
 and bipolar disorder **239**
 Creutzfeldt-Jakob disease (CJD)
231
 criminal behavior 211
 CSF see cerebrospinal fluid
 CT (computed tomography) scans
 12, 222
 cuddling babies 135
 cultural influences **199**
 cynophobia 240
 cytoplasm 70, 71
D
 Dale, Henry H. 9
 Dali, Salvador 191
 danger, thrill-seeking 131
 Darwin, Charles 9
 day length 190
 daydreaming 186
 De Clerambault’s syndrome 244
 deafness 78, 91
 death **238**
 decision-making 11, **169, 211**
 and aging 214
 agency and intention 193
 morality 140
 “declarative” memories 158, 160
 deep brain stimulation 218
 for depression 239
 for movement disorders 232
 for Tourette’s syndrome 243
 default-mode network (DMN) 184
 defenses, physical **45**
 degenerative disorders 222, 223
 déjà vu **163**
 Delgado, José **10**
 Delta waves, sleep 181
 delusions
 delusional disorder **244**
 schizophrenia 244
 dementia 203, 214, **230**
 Alzheimer’s disease 230, **231**
 artificial hippocampus 161
 Creutzfeldt-Jakob disease **231**
 Huntington’s disease **234**
 demons, exorcism 222
 dendrites 69, 70, 71
 requirements of **181**
 synapses 71
 dendritic spines 71
 dentate gyrus 65, 212, 213
 dentate nucleus, cerebellum 117
 dependent personality disorder 245
 depolarization, nerve impulses 72
 depression 185, 203, 222, 223, **239**
 and creativity 170
 genetics and 197

- depression *continued*
 grief and 129
 pacemakers and 219
 depth, vision **83**, 89
 dermatomes **42**
 dermis 102
 Descartes, René 6, **8**, **178**
 desire **130**
 and dreams 189
 detritus 188
 development and aging **206–219**
 developmental delay **247**
 learning disability **248**
 developmental disorders 222, 223
 diabetes, history of mental illness 222
 diabetes, and aging 215
 diagnosis
 Down syndrome tests 236
 mental disorders **222**
 Diagnostic and Statistical Manual
 (DSM) of Mental Disorders 222
 diaphragm, motor neuron disease
 235
 diencephalon 53, 60
 diet *see* food
 diffusion tensor imaging 13
 digestive system, panic
 responses 127
 digital modeling **75**
 digital simulation **75**
 direction, attention to 183
 disbelief 172
 diseases and disorders **220–249**
 causes **223**
 constellations and spectrums
223
 diagnosis **222**
 historical theories of **222**
see also individual disorders
 disgust 128
 adaptive behaviors 129
 bad odors 98
 facial expressions 136
 dislocated self **193**
 disorganized schizophrenia 244
 dissociation **186**
 dissociative identity (DID) **201**
 distorting illusions **175**
 distraction, and experience of
 pain 109
 divided attention 182
 DNA **196**, 197
 Huntington's disease 234
 in neurons 70
 dogs
 brains 49
 expressions 136
 sense of smell 97, 98
 dolphins, brains 49
 dopamine 73
 and addiction 130, 245
 and aging 214
 anticipation 130
 and attention deficit
 hyperactivity disorder 246
 bipolar disorder 239
 and brain "clocks" 190
 and desire 130
 and emotions 127, 128
 "falling in love" 134
 genetic influences 197
 and humor 171
 Parkinson's disease 234
 and patternmaking 173
 reward system 115, 130
 and schizophrenia 244
 substantia nigra 58
 thrill-seeking 131
 dorsal horn, pain signals 106
 dorsal pathway, vision **84–85**, 89
 dorsal root, spinal nerves 42
 dorsal striatum 58
 dorsolateral prefrontal cortex
 and consciousness 180
 and morality 140
 Down syndrome **236**, 248
 drawings, autistic savants
 164–165, 174
 dreams 188, **189**
 conscious perception 181
 lucid dreams **189**
 driving, movements 116
 drop attacks 226
 drowsiness 188
 narcolepsy **238**
 drugs
 addiction to 130, 211
 altering consciousness 186
 opiates 107
 placebo effect 109
 and proprioception 104
 sleeping drugs 189
 dualism 178
 Duchenne, Guillaume 137
 "Duchenne" smile 137
 dura mater
 skull 56
 spinal cord 42
 dyscalculia 248
 dysgraphia **153**
 dyslexia 149, **153**, 248
 treating 153
 dysphasia 149
 dyspraxia 248
- E**
 eardrum 90, 91, 94
 ears **90–95**
 anatomy **90**
 balance 105
 deafness 78, 91
 hearing **94–95**
 nerves 43
 sending information to
 thalamus 60
 earthworms 48
 East Asia, cultural influences 199
 eating disorders 242, **246**
 Ebbinghaus illusion 175
 echolocation 91
 ecstasy
 out-of-body experiences 187
 supernatural experiences 173
 ECT *see* electroconvulsive therapy
 EEG *see* electroencephalography
 Egypt, ancient 6, 8
 Einstein, Albert 199, 204–205
 electrical impulses 38
 brain-machine interfaces **216**
 executing a movement 119
 imaging techniques 9
 nerve impulses **72–73**
 scanning the brain 12, 13
 electroconvulsive therapy (ECT) 202
 electrodes 9
 electroencephalography
 (EEG) 6, 12, 202
 brain death 238
 elephants
 brains 49
 impossible drawing 175
 embodied cognition 138
 embolus
 stroke 229
 transient ischemic attacks 228
 embryonic disk 208
 emotions 39, **124–131**
 amygdala 187
 artificial intelligence 217
 body language 144
 and brain maturity 212
 cerebral cortex 67
 conflicting emotions 137
 emotions *continued*
 conscious emotions **128–129**
 and consciousness 179
 and conversation 150
 and decision-making 169
 desire and reward **130–131**
 and dreams 189
 empathy and sympathy 140
 and experience of pain 109
 expressing **136**
 feeling **128**
 gender differences 198
 gestures 145
 laying down a memory 160, 161
 limbic system 126
 loss of control 193
 mirroring **123**
 morality 140, 141
 post traumatic stress disorder
164, **241**
 reading **137**
 and recognition 163
 recognition of faces 84
 responding to 139
 schizophrenia 244
 and sense of smell 96, 99
 and the supernatural 173
 timing 129
 empathy **140**, 141
 facial expressions and 139
 and literacy 152
 mirror neurons 11
 encephalins 107
 encephalitis **227**
 endocrine system **114–115**
 pituitary gland 61
 endoplasmic reticulum 70
 endorphins 107
 endoscopes, transnasal surgery 232
 energy, sources of **45**
 English language, dyslexia 153
 enochlophobia 240
 entorhinal cortex, memory 161
 environment
 influence of 196
 and intelligence 168
 and schizophrenia 244
 enzymes 197
 epidermis, skin 102
 epigenetic changes 196, **197**
 epilepsy **226**
 pacemakers and 219
 seizures 181
 split brain 204
 epinephrine (adrenaline) 114
 and heat production 113
 stress response 240
 thrill-seeking 131
 episodic memory 157, 160
 epithelium, olfactory 97
 erotomania 244
 erythropoietin 114
 Escher, M. C. 175
 estrogen 114
 ether 112
 ethics, biotechnology 218
 ethmoid bone 54
 euphoria 130, 134
 eustachian tube 90
 evolution **48–49**
 and aging 214
 of language **147**
 sense of smell 98
 sense of taste 100
 excitatory neurotransmitters 73
 and personality 200
 and subjective time 190
 excitement, and aging 214
 exercise
 and aging 215
 effect on brain 38, 44
 exorcism 222
 expressions 127, **136–137**, 139
 microexpressions **136**
 exterior globus pallidus 53, 54
 extradural hematoma 225
 extroversion, personality markers
 200
 eyes **80–89**
 attention 182, 183
 bionic eyes **218**
 blindness 78
 blindsight **79**
 brain stem functions 63
 communication **144**
 eye contact 144
 eye-tracking studies 86–87
 facial expressions 136–137
 iris 208
 macular degeneration 218
 nerves 43
 neurons 71
 panic responses 127
 retina **81**, 208
 seeing **88–89**
 sending information to
 thalamus 60
 structure **80–81**
 visual pathways **80**, **84–85**
 visual perception **86–87**
- F**
 fabricated and induced illness (FII)
 243
 face
 autism spectrum disorders 249
 babies 213
 bones 54
 expressions 127, **136–137**, 139
 pain 107
 patternmaking 173
 recognition 83, 84, **85**, 139, 163,
 209
 sending information to
 thalamus 60
 sexual attraction 134
 smiling 129, **137**
 symmetry 134
 facial nerve 43
 facial nucleus 58
 factitious disorders 243
 factual recognition, faces 84
 fairies 173
 "falling in love" 134
 Fallon, James 141
 false memory **164**
 fasciculus cuneatus 62
 fasciculus gracilis 62
 fat tissues
 brown fat cells 113
 regulation of hunger 115
 fatigue, chronic fatigue syndrome
225
 fatty acids 100
 fatuous love 134
 fear
 adaptive behaviors 129
 body language 144
 facial expressions 127, 136
 hemispheres and 128
 hypothalamus and 126
 and moods 129
 phobias 127, **240**
 feedback mechanisms
 neuroendocrine system **114**
 proprioception 104
 feelings *see* emotions
 feigning disease **243**
 female brain 198
 femininity
 female brain 198
 and sexual attraction 134
 fetus, development of hearing 93
 field sobriety tests 104
 "fight or flight" response 127,
 129, 240
 filum terminale 42
 fish, brains 48
 fissures
 cerebellum 63
 cerebral cortex 66
 flashbacks, post traumatic stress
 disorder 164, 241
 flavor-aversion learning 101
 flight response 127, 129, 240
 flocculonodular lobe 63
 flow 193
 fMRI (functional magnetic
 resonance imaging) 7, 12, 13
 focus, ability to **183**
 focused attention 182
 folia, cerebellum 63
 folic acid 237
 "folk memory" 187
 follicle-stimulating hormone
 (FSH) 61, 114
 food
 and aging 215
 appetite control 115
 eating disorders **246**
 taste **100–101**
 foramen magnum 53, 62
 foramina, facial bones 54
 forebrain 53
 foresight 173
 forgetting 156, **164**
 Alzheimer's disease 231
 blocking memory 164
 form, visual perception 89
 fornicate gyrus 65
 fornix 53, 55, 65
 fovea **81**
 fractures, skull 225
 fragile X syndrome 248
 free will 11, 169, 193
 Freeman, Walter 11
 "freezing," motor disorders 119
 Freud, Sigmund 9, 184, 222
 and dreams **189**
 on hysteria 242
 friendship 134
 frogs, brains 48
 frontal bone 54, 66
 frontal cortex
 bilingualism 149
 brain development 210, 212
 and consciousness 180
 emotions 126, 128, 129
 false memory 164
 functions 138
 and limbic system 64
 pain signals 108
 planning movement 117
 Tourette's syndrome 243
 frontal lobe 66, 69
 altered brain states 186
 attention 182
 and consciousness 180
 and creativity 171
 decision-making 169
 dreams 189
 emotions 128
 imaging **17**
 intelligence 168
 memory 156, 157, 160, 161
 visual pathways 84, 85
 visual perception 88
 frontal-polar cortex, imaging **16**
 functional brain imaging 12
 functional disorders 223
 functional magnetic resonance
 imaging (fMRI) 7, 12, 13
 fungi, brain abscesses 228
 fusiform gyrus
 emotions 129

- fusiform gyrus *continued*
 face recognition 139
 imaging **31**
 future prospects **216–217**
- G**
- GABA *see* gamma-aminobutyric acid
- Gage, Phineas **8, 10, 141**
- Galen **8**
- Gall, Franz Joseph **9, 10**
- Galvani, Luigi **8**
- Gamma waves, and creativity 170
- gamma-aminobutyric acid (GABA) **73**
 and emotions 127
 epilepsy 226
 and sleep 188
- ganglia *see* nuclei
- ganglions, sensory nerves **42**
- gender differences
 emotions 126, 127
 and facial symmetry 134
 influencing the brain 198
 stria terminalis 126
- general anesthetics **112**
- generalized anxiety disorder 240, 242
- generalized seizures 226
- genes and genetics **196–197**
 and addictions 245
 Alzheimer's disease 231
 attention deficit hyperactivity disorder 246
 autism spectrum disorders 249
 and individuality 38
 and intelligence 168
 and language **147**
 and personality 200
 personality disorders 245
 tests in pregnancy 236
- genetic diseases
 Huntington's disease **234**
 motor neuron disease (MND) **235**
 schizophrenia **244**
 Tourette's syndrome **243**
- geniculate nuclei 53, 60
- genome 196, 197
- Geschwind's territory 148
- gestures **145**
- ghosts 173
- ghrelin 115
- glands
 neuroendocrine system 114
see also adrenal glands;
 pituitary gland
- Glasgow Coma Scale 238
- glial cells 44, 68, 70, **71**
 cerebellum 63
 cerebral cortex 67
- global aphasia 149
- globus pallidus **58, 117**
- glossopharyngeal nerve 43, 101
- glucagon 114
- glucose **45**
 and aging 215
 PET scans 12
- glutamate 73
- glutamic acid 73
- glycine 73
- Golgi, Camille **9**
- Golgi cells 63
- Golgi complex 70
- "goose bumps" 113
- gorillas, brains 49
- grammar 146
 of gestures 145
- grand mal epilepsy 226
- Grandin, Temple **249**
- grandiose delusional disorder 244
- granule cells, cerebellum 63
- greebles 83
- Greece 8
- gray matter 44
 bilingualism 149
 cerebral cortex 68
 spinal cord 42
 in thalamus 60
- grief 129
 complicated grief **130**
- growth of brain 39
- growth hormone (GH) 61, 114
- gustatory areas 101
- gymnasts 105
- gyri (gyrus) 54, 209
 cerebral cortex 66, 67, 69
- H**
- hematoma 225
 subdural 229
- hemorrhage 225
 hemorrhagic stroke 229
 subarachnoid hemorrhage **229**
 subdural hemorrhage **229**
- hair cells, in ear 90, 91, 94
- hairs
 "goose bumps" 113
 touch receptors 102
- hallucinations
 schizophrenia 193, 244
 supernatural experiences 173
- halothane 112
- hands
 anarchic hand syndrome (AHS) 57
 bionic arms **219**
 gestures **145**
 handedness 57, **199**
 manual dexterity 247
 phantom limbs 104
 touch receptors 103
- happiness, facial expressions 137
- "hard" problems, vision 88
- hatred 128
- Hawking, Stephen 235
- head
 cranial nerves **43**
 injuries 6, **225**
see also face; skull
- headaches **224**
- hearing **90–95**
 cerebral cortex 67
 conversation **150**
 development of **93**
 hearing loss **91**
 perception of sound **92–93**
- heart
 hormones 114
 panic responses 127
 regulation of heart rate **112**
- heart attack, referred pain 107
- heat, thermoreceptors 102, 113
- Helmholtz, Hermann von 8
- hemiplegia 236
- hemispheres
 and aging 215
 basal nuclei 58
 missing 205
 split brain **11, 204**
 tumors 230
see also left hemisphere; right hemisphere
- Hensen's cells 90
- herbivores, sense of taste 100
- heroin
 addiction 130
 pain relief 107
- of gestures 145
- Herpes simplex virus 227
- heterosexuality 198
- hierarchy of brain **53**
- hindbrain 53, 62
- hippocampal fold 69
- hippocampus 53, **65, 209**
 artificial hippocampus **161, 219**
 cellular organization 59
 and consciousness 180
 and depression 239
 dreams 189
 emotions 126, 127
 general anesthetics 112
 imaging **24**
 limbic system 64, 65
 memory 65, **156–163, 164, 213**
 and novelty-seeking 200
 reading 152
 smell and 99
 spatial memory 162
- Hippocrates 222
- histamine 73, 106
- history
 investigating the brain **8–9**
 neuroscience **10–11**
- histrionic personality disorder 245
- HM (Henry G. Molaison) **11, 159**
- Holmes, Gordon Morgan 9
- homeostasis 114
- Homo erectus* 49
- Homo habilis* 49
- Homo neanderthalensis* 49
- homosexuality **198**
- hormones
 and brain changes 211, 213
 and day length 190
 and emotions 126, 127
 feedback mechanisms **114**
 neuroendocrine system **114–115**
 oxytocin **135**
 pituitary gland 61
 pituitary tumors 230
 twins 199
- hospital addiction syndrome 243
- Human Brain Project (HBP) 75
- human genome 196, 197
- humor **171**
 "humors," history of mental illness 8, 222
- hunger 115
- Huntington's disease **234**
- hydra 48
- hydrocephalus **237**
- hydrogen atoms, magnetic resonance imaging 13
- hyperactive ADHD 246
- hyperkinesia 119
- hyperlexia **153**
- hyperthymesia 164
- hyperventilation 127
- hypnosis **186, 222**
- hypochondria **242**
 and creativity 170
- hypocretins 238
- hypodermis 102
- hypoglossal nerve 43
- hypokinesia 119
- hypomania, and creativity 170
- hypothalamus 53, 55, 56, **61**
 anticipation 130
 appetite control 115
 dreams 189
 emotions 126, 127, 128
 functions **113**
 gender differences 198
 hormones 114
 limbic system 64
 narcolepsy 238
 neuroendocrine system 114
 oxytocin 135
 sleep-wake cycles **115**
 and stress 199
 stress response 240
 thermoreceptors 113
- hysteria **242**
 and creativity 170
- "I," self-consciousness 192
- "ice-pick" lobotomy 11
- illness *see* diseases and disorders
 and individual disorders
- illusions
 cognitive illusions **174–175**
 Mona Lisa illusion 85
 motion 85
 visual perception 87
- imagination
 and creativity 170
 and literacy 152
- imaging techniques 6–7, **9, 12–13, 222**
- immune system
 autoimmune diseases 235, 238
 brain abscesses 228
 encephalitis 227
- implants
 artificial hippocampus **161, 219**
 bionic eyes 218
 cochlear **91, 94**
 early experiments **10**
- implicit memory 157, 160
- impulses *see* nerve impulses
- impulsive ADHD 246
- inattentive ADHD 246
- incubi 189
- incus 90
- individuality 38, 198
- infatuation 134
- infections
 encephalitis **227**
 meningitis **227**
- inferior colliculus 62, 95, 118
- inferior temporal lobe, visual pathways 85
- inflammation 106
 brain abscesses 228
 encephalitis **227**
 meningitis **227**
- influencing the brain **198–199**
- information
 creativity 170
 dreams 189
 illusions 174
 processing 38
 sensations 39
 signaling speed 39
- infrared light, hearing **94**
- inheritance patterns 197
- inhibitory neurotransmitters 73, 190
- injuries
 head injuries **225**
 and language problems 149
 pain 106–107
- inkblot test 201
- "inner ear" 157
- "inner speech" 39
- insomnia 188
- instability, postural 119
- instincts
 collective unconsciousness 187
 empathy and sympathy 140
 language 146
 morality 140–141
- insula 66
 and cooperative behavior 200
 conflicting emotions 137
 and disbelief 172
 emotions 128, 129
 imaging **22**
 self-awareness 138, 139
 sense of taste 101
- insular cortex 108
- insulin 114, 115
- insulin-like growth factor (IGF) 196
- intelligence **168–169**
 artificial intelligence **217**
 decision-making **169**
 IQ ("intelligence quotient") 168, 170
 and size of brain 44, 168, 205
- intention
 mirroring **123**
 self-consciousness **193**
- interior globus pallidus 53, 54
- intestines, hormones 114
- intimacy 134
- intoxication test 162
- intracranial cavity 44
- intraparietal sulcus 169
- introspection 192
- invertebrates, evolution of brain **48**
- investigating the brain **8–9**
- ion channels, in hippocampus 65
- ions, nerve impulses 72, 73
- IQ ("intelligence quotient") 168, 170
- iris, eye 80
- iron, in basal ganglia 214
- ischemic stroke 229
- Italian language, dyslexia 153
- itching 102
- J**
- jamais vu **163**
- jaw bones 53
- jealous delusional disorder 244
- joints, proprioceptors 104
- judgments, morality **140–141**
- Jung, Carl 187
- "junk" DNA 196
- K**
- Ke Jie 217
- kidneys
 hormones 114
 thirst 115
- knee, patellar spinal reflex 116
- knowledge
 and aging 215
 self-consciousness 192
 taxi drivers 162
- L**
- L-dopa 173
- language 38
 autism 223
 bilingualism **149**
 brain imaging 13
 Broca's area **10, 148**
 conversation **150–151**
 definition 146
 development **209**
 developmental milestones 247
 dyslexia **153**
 evolution of **147**
 gender differences 198
 genes **147**
 and handedness 199
 language areas **148–149**
 language problems **149**
 and memory 157
 origins of **146–147**
 and perception **147**
 reading and writing **152–153**
 specific language impairment 248
 and thinking 179
 Wernicke's area **10, 148**
- larynx, and evolution of language 147
- lateral Brodmann areas 67
- lateral corticospinal tract 118
- lateral geniculate nucleus 80, 81

- lateral hypothalamic area 113
 lateral hypothalamic nucleus 115
 lateral lobes, cerebellum 63
 lateral sulcus 66
 asymmetry 57
 Einstein's brain 205
 laughter 171
 and visual illusions 87
 learning **154–165**
 anticipation 130
 flavor-aversion learning 101
 intelligence 168
 long-term potentiation 197
 memory 156, 157
 reading and writing 152
 learning disability **248**
 Down syndrome 236
 language problems 149
 left hemisphere 53
 bilingualism 149
 and conversation 151
 functions **57**
 and language 146
 split brain 204
 left-handedness 57, 198, **199**
 legs
 executing a movement 118
 motor neuron disease 235
 phantom limbs **104**, 193
 lemurs 138
 lens, eye 80
 lentiform (lenticular) nucleus 58
 Leonardo da Vinci 174
 leptin 115
 letters
 reading and writing 152–153
 visual processing 79
 Lewy body dementia 230
 Libet, Benjamin 9, **11**, 191, 193
 lie detection **217**
 life, quality of 134
 life-support machines 238
 lifestyle, and aging 215
 ligaments, proprioceptors 104
 light
 after-images 174
 hearing light **94**
 optogenetics **203**
 seasonal affective disorder 239
 sleep-wake cycles 115
 vision 80, 82, 88
 liking 130
 limbic lobe 64, **65**, 66
 limbic system **64–65**, 74, 211
 and desire 130
 emotions 39, 126
 “falling in love” 134
 functions 57
 hypothalamus and 113
 and recognition 163
 smells and 64, 96, 98, 99
 and stress 199
 limbs
 executing a movement 118
 phantom **104**, 193
 prosthetic 104, **219**
 lipids 44
 listening
 Broca's area 148
 conversation **150**
 literacy 152
 literal-mindedness 172
 liver, alcohol addiction 245
 lobes, cerebral 66
 see also *individual lobes*
 lobotomy **11**
 location, attention to 183
 locked-in syndrome **63**
 Lomo, Terje 9
 long-term memory **156**, 158,
 160–161
 long-term potentiation 158, 197
 longitudinal fissure 66
 loss of control 193
 loss, emotions 128
 Lou Gehrig's disease 235
 love **134–135**
 lucid dreams **189**
 lumbar puncture **227**
 lumbar region, spinal nerves 42
 lumbar vertebrae 42
 lungs
 panic responses 127
 regulation of breathing 112
 luteinizing hormone (LH) 61, 114
- ## M
- macaques, language areas 147
 McEwan, Ian 244
 McGurk effect 78
 machines
 artificial intelligence **217**
 brain-machine interfaces 216
 consciousness 219
 macular degeneration, eyes 218
 “mad cow disease” 231
 madeleine effect, sense of smell
 99
 madness, and creativity 170
 magnetic encephalography (MEG)
 7, 12, 13
 magnetic resonance imaging
 (MRI) 7, 9, **13**, **14–35**, 74, 222
 male brain 198
 malingerer 243
 malleus 90
 mamillary body 55
 and emotions 126
 functions 58, 64
 memory 156
 mammals, brains **49**
 manic-depressive illness **239**
 manual dexterity, developmental
 milestones 247
 mapping the brain **10**, **74–75**
 maps
 “body maps” 192, 193
 spatial memory 162
 marijuana, and schizophrenia 244
 Mars, canals on 174
 masculinity
 male brain 198
 and sexual attraction 134
 massa intermedia 198
 material universe, and
 consciousness 178
 maternal love 134
 mathematics 205
 maxilla 53
 mazes, spatial memory 162
 ME (myalgic encephalomyelitis)
 225
 “meaningfulness” 186
 measles virus 227
 medial Brodmann areas 67
 medial frontal cortex 184
 medial frontal gyrus 140, 217
 medial preoptic nucleus 113, 198
 median sulcus 62
 medicine
 biotechnology **218–219**
 see also *drugs*
 meditation **187**
 medulla 53, 56, 62
 functions 57, 63
 pain signals 106
 sense of taste 101
 MEG (magnetic encephalography)
 7, 12, 13
 Meinhof, Ulrike 205
 Meissner's corpuscles 102
 melanin 58
 melanocortin 115
 melanocyte-stimulating hormone
 (MSH) 61, 114
 melatonin 114, 178
 circadian rhythm 63
 sleep-wake cycles 115
 membranes
 cell membrane 70
 see also *meninges*
 memory 38, **156–165**
 accessing memories **159**
 Alzheimer's disease 231
 anticipation 130
 artificial hippocampus **161**, 219
 association 159
 autism 249
 cerebral cortex 67
 collective unconsciousness 187
 and conversation 150, 151
 d  ja vu and jamais vu **163**
 dementia **230**
 distribution in brain **158**
 and emotions 126
 forming memories 158
 head injuries 225
 hippocampus and 65, **156–163**,
 164, 213
 HM (Henry G. Molaison) **11**, 159
 inability to store **159**
 laying down a memory **160–161**
 location of **161**
 long-term potentiation 158, 197
 movement memory 120
 multiple personalities 201
 oxytocin and 135
 and personality 200
 phobias 127
 post traumatic stress disorder
 164, 241
 principles of **156–157**
 reading 152
 recall and recognition 84,
 162–163
 short- and long-term memory
 156
 sleep and 188
 smell and **99**
 spatial memory **162**
 storage **158–159**
 super memory **164**
 suppressing 164
 types of **157**
 unusual memory **164–165**
 men
 emotions 126, 127
 influences on the brain 198
 male brain 198
 and sexual attraction 134
 meninges
 skull 45, 54
 spinal cord 42
 meningitis **227**
 meningocele 237
 MENSA 168
 menstrual cycle 99
 mental disorders 211, **222–223**
 and intelligence 168
 Merkel's disks 102
 mesencephalon 53
 Mesmer, Franz Anton 8, 222
 metabolic rate, and heat
 production 113
 metastases, brain tumors 230
 microexpressions **136**
 microglia 71
 microtubules 70
 mid-frontal area, and
 numbers 169
 midbrain 53, 55, 62
 disorders 119
 limbic system 64
 migraine **224**
 Milner, Brenda 9
- mimicry
 mirror neurons 11
 personality development 200
 mind
 consciousness **178–179**
 dualism 178
 mind-control technology 216
 mirror neurons 11
 theory of **139**
 mind reading **217**
 mind/body problem **178**
 mindfulness **187**
 “ministrokes” **228**
 mirror illusion, phantom limbs 104
 mirror neurons **11**, **122–123**, 139
 mirror writing 153
 mitochondria 70
 mixed cerebral palsy 237
 mixed delusional disorder 244
 modeling, digital **75**
 modiolus 90
 modules 38
 Molaison, Henry G. (HM) **11**, 159
 molecules 41
 neurotransmitters 73
 smell 96, 97
 Mona Lisa illusion 85
 monism 178
 Moniz, Egas 9, 11
 monkeys
 and facial symmetry 134
 language areas 147
 monoamine oxidase, and
 depression 239
 monoamines 73
 moods **129**
 bipolar disorder **239**
 and decision-making 169
 mood disorders 168
 neurotransmitters and 197
 morality **140–141**, 210
 morphine
 addiction 130
 pain relief 107
 mosaicism 236
 Moscow Brain Research Institute
 205
 motion see *movement and control*
 motor control
 brain development 210
 cerebral cortex 67
 developmental milestones 247
 executing a movement 119
 multiple sclerosis 235
 nuclei 58
 paralysis 236
 Parkinson's disease 234
 motor cortex
 and consciousness 180
 disorders 119
 executing a movement 118
 pain signals 108
 planning movement 117
 self-awareness 139, 192
 speech 151
 motor nerves
 bionic arms 219
 disorders **119**
 executing a movement 119
 reflex actions 116
 spinal cord 42
 motor neuron disease (MND) **235**
 motor tics, Tourette's syndrome
 243
 mouth
 facial expressions 136–137
 motor neuron disease 235
 sending information to
 thalamus 60
 sense of taste 100, 101
 speech 151
- movement and control **110–123**
 attention 183
 blindsight 79
 cerebral palsy **237**
 executing a movement **118–119**
 illusions 85
 mirroring **122**
 motor disorders **119**
 multiple sclerosis 235
 neuroendocrine system **114–115**
 nuclei 58
 parkinsonism 234
 planning **116–117**
 proprioception 104
 reflex actions **116**
 regulation **112–113**
 unconscious action **120–121**
 vision 85, 89
 “Mozart effect,” listening to music
 93
 MRI (magnetic resonance
 imaging) 7, 9, **13**, **14–35**, 74,
 222
 multi-infarct dementia 230
 multi-sensory perceptions 39
 multitasking 182
 multiple sclerosis (MS) 71, 223,
 235
 multipolar neurons 71
 Munchausen's by proxy 243
 Munchausen's syndrome **243**
 muscles
 cataplexy 238
 executing a movement **118–119**
 facial expressions 136–137
 motor disorders **119**
 motor neuron disease 235
 moving 117
 multiple sclerosis 235
 paralysis **236**
 proprioceptors 104, 105
 sleep paralysis 189
 music 196
 amusia 248
 creativity 170
 hearing **93**
 mutation 197
 myalgic encephalomyelitis (ME)
 225
 myasthenia gravis 236
 myelin sheaths 68, 70, 210, 212
 and aging 214, 215
 manufacture of 71
 multiple sclerosis 71, 235
 myelinated axons, nerve impulses
 72
 myelination 210–211, 212
 myelomeningocele 237
 Myers-Briggs personality test 201
 myoclonic seizures 119, 226
 mysophobia 240
- ## N
- nanorobots 218
 narcissistic personality disorder
 245
 narcolepsy 188, **238**
 nasal cavity 53
 nature and nurture **196–197**
 navigation, spatial memory 162
 Neanderthals 49
 “Near Death Experiences” 187
 neck, motor neuron disease 235
 Necker cubes 87
 necrophobia 240
 neglect 85
Neisseria meningitidis 227
 neocortex 74, 138
 nerve fibers 68
 nuclei **58**
 pain fibers **107**

- neocortex 74, 138
 nerve fibers 68
 nuclei **58**
 pain fibers **107**
 spinal cord 42
see also axons; dendrites
 nerve impulses 42, **72–73**
 nerve tracts, in brain stem 62
 nerves
 cranial nerves **43**
 paralysis **236**
 spinal nerves **42**
 nervous system **40–43**
 aging 214
 damage to 107
 evolution of 48
 nervousness 129, 240
 networks, neural 41
 NeuraBASE 75
 neural correlates of consciousness 181
 neural crest 208
 neural groove 208
 neural plate 208
 neural progenitor cells 71, 197
 neural tube 208
 defects **237**
 neurites 70
 neurocranium 54, 66
 neuroendocrine system **114–115**
 neurofeedback 202
 neurogenesis 71, 197, **212**, 213, 215
 neuroglia 68
 neuromuscular junction 119
 neurons 41, **70–71**, 210
 aging 44, 214–215
 Alzheimer's disease 231
 attention 183
 in cerebral cortex 68, 69
 connectivity 39, 168
 epilepsy 226
 general anesthetics 112
 hippocampus 65, 212
 learning 156, 157
 memory 156, 158, 160, 161
 mind reading 217
 mirror neurons **11**, **122–123**, 139
 motor neuron disease **235**
 multiple sclerosis 235
 nerve impulses 42, 72
 neural networks 41
 neurogenesis 71, 197, **212**, 213, 215
 nuclei **58–59**
 plasticity **197**
 regeneration 39, **71**
 signal transmission 38
 spinal cord 42
 spindle cells 126
 synapses **71**
 in thalamus 60
 and time 190
 types of **71**
 neuropathic pain 107
 neuropeptide (NPY) 115
 neuroscience, history of **10–11**
 neurostimulation 202–203
 neurosurgery **232–233**
 neurotransmitters 38, 71, **73**
 and aging 214
 Alzheimer's disease 231
 anxiety disorders 240
 bipolar disorder 239
 and depression 239
 and desire 130
 and emotions 128
 executing a movement 119
 “falling in love” 134
 gene expression and 196
 and mood 197
 nerve impulses 72, 73
 Parkinson's disease 234
 neurotransmitters *continued*
 and personality 200
 sleep-wake cycles 188
 and time 190
 neurotubules 73
 neurulation **208**
 nicotine addiction 130, 245
 nightmares, post traumatic
 stress disorder 241
 Nixon, Richard 136
 nocebo effect 109
 noise
 hearing **93**
 Tourette's syndrome 243
 nonpathological disease
 feigning 243
 noradrenaline (norepinephrine)
 73, 239
 noradrenergic receptors 130
 nose
 facial expressions 136–137
 sense of smell **96–97**, 101
 transnasal surgery **232**
 nosophobia 240
 novelty-seeking, personality
 markers 200
 nuclei 53, **58–59**
 in brain stem 62
 in hypothalamus 61, 113
 in thalamus 60
 nucleus accumbens 115, 130
 numbers **169**
 dyscalculia 248
 language and 147
 visual processing 79
 nurture *see* nature and nurture
 nutrition *see* food
 nyctophobia 240
- O**
 Obama, Barack 199
 objective time 190
 objects, recognition **83**
 obsessive-compulsive disorder
 (OCD) 164, **241**, 242
 obsessive-compulsive personality
 disorder 245
 occipital bone 53, 54, 66
 occipital cortex 121
 occipital lobe 66
 imaging **33**
 octanoic acid 96
 octanol 96
 oculomotor nerve 43
 odors, sense of smell 96
 olfaction *see* smell
 olfactory bulb 64, 96, 101
 olfactory cortex
 emotions 126
 functions 97, 101, 213
 memory 162
 olfactory epithelium **97**
 olfactory nerve 43, 55, 101
 olfactory receptors 96
 olfactory tract 97
 oligodendrocytes 69, 70, 71,
 212, 213
 olivary nuclei 53
 olives 53
 omnivores, sense of taste 100
 ophidiophobia 240
 bipolar disorder 107, 130
 opiate system
 pain relief 107, 130
 sugar addiction 115
 optic chiasm 53, 80, 88
 optic disk 80, 81
 optic nerve 43, 80, 88, 208
 optic radiation 80, 81, 88
 optic tracts 80
- optical illusions **174**
 optimism, personality markers 200
 optogenetics **203**
 orbital frontal gyri **19**
 orbitofrontal cortex 68, 213
 anticipation 130
 and body language 144
 conduct disorder 248
 conflicting emotions 137
 and consciousness 180
 decision-making 169
 emotions 128
 morality 140
 and obsessive-compulsive
 disorder 241
 sense of smell 97
 orexins 238
 organ of Corti 90, 91, 94, 95
 organelles 70
 orgasm 135
 orphans 135
 orthonasal smell 96, 97, 101
 ossicles, ear 90, 94
 otitis media 91
 otosclerosis 91
 out-of-body experiences (OBEs)
187
 parietal lobe and 186
 and spiritual transcendence 172
 supernatural experiences 173
 ovaries, hormones 114
 overt attention 182
 oxygen **45**
 regulation of breathing 112
 oxytocin 61, **135**
 and fear 127
 functions 114, 213
- P**
 pacemakers 218, **219**, 243
 Pacinian corpuscles 102
 pain **106–109**
 brain surgery 39, 109
 experiencing **108–109**
 headache and migraine **224**
 inability to feel **109**
 morality 140, 141
 pain relief **107**
 phantom limbs 104
 receptors 39, 102
 reflex actions 116
 social rejection 139
 types of **107**
 pair bonding 134
 pancreas, hormones 114
 panic
 panic attacks 240
 phobias 127, 240
 papillae, taste buds 100
 paradox illusions **175**
 parahippocampal gyrus 64, 65
 paralysis **236**
 motor cortex damage 119
 sleep paralysis 189
 paranoia
 and creativity 170
 paranoid personality
 disorder 245
 paranoid schizophrenia 244
 paraplegia 236
 parasites, brain abscesses 228
 parenthood **213**
 parietal bones 54
 parietal cortex
 attention 182
 disorders 119
 and dreams 189
 executing a movement 118
 false memory 164
 maths enhancement 203
 pain signals 108
- parietal cortex *continued*
 self-consciousness 192
 unconscious action 120
 parietal lobe 66, 69
 altered brain states 186
 emotions 128
 imaging **28**
 intelligence 168
 memory 156
 morality 140
 out-of-body experiences 173, 187
 unconscious movement 117
 visual pathways 84
 parietal-temporal junction, and
 consciousness 180
 parkinsonism **234**
 Parkinson's disease **234**
 nuclei problems 58
 pacemakers and 219
 and time 190
 partial seizures 226
 partitions and zones **56–57**
 passion 134
 passivity, sadness 129
 patellar spinal reflex 116
 pathological disease feigning 243
 patternmaking **172–173**
 Pavlov, Ivan 205
 Penfield, Wilder 9, **10**, 103
 Penrose triangle 175
 perception
 backdating time **191**
 brain development 210
 and consciousness 179
 illusions 174–75
 language and **147**
 multisensory perceptions 39
 self-awareness 180
 visual perception 84, **86–87**
 peripheral nervous system (PNS)
40–41, 74
 evolution of 48
 persecutory delusional disorder
 244
 persistent vegetative state 238
 personality 38, **200–215**
 learning **200**
 multiple personalities **201**
 and obsessive-compulsive
 disorder 241
 personality assessment **201**
 personality disorders **245**
 persuasive illusions 175
 pessimism 211
 PET (positron emission
 tomography) 7, 12, 13
 petit mal epilepsy 226
 phantoms
 phantom limbs **104**, 193
 visual phantoms **181**
 phenylethylamine, “falling in
 love” 134
 phenylthiocarbamide (PTC) 100
 pheromones 99
 philosophy, investigating the
 brain 8
 phobias **127**, **240**
 phonological deficit hypothesis,
 dyslexia 153
 photoreceptors 80, 81
 phrenology **10**
 physical skills
 brain development 210
 developmental milestones 247
 disorders 222
 pia mater
 skull 56
 spinal cord 42
 pictures, visual perception **86–87**
 piloerection 113
 pineal gland 55, 62, 114, 178
 sleep-wake cycles 115
- pinna 90, 94
 Piraha Indians 147
 pituitary gland 53, 56, **61**
 emotions 126
 hormones 114
 hypothalamus and 113
 neuroendocrine system 114
 oxytocin 135
 pain relief 107
 stress response 240
 thirst 115
 tumors **230**, 232
 placebo effect 109
 plaques, aging brain 214, 215
 plasticity 38, **197**
 Plato 8
 pleasure 127, **130–131**, 135
 pons 53, 56, 62
 pontine nucleus,
 cerebellum 117
 position, proprioception 104
 positron emission tomography
 (PET) 7, 12, 13
 post traumatic stress disorder
 (PTSD) **164**, **241**
 posterior cingulate cortex
 imaging **34**
 morality 140
 pain signals 108
 posterior cranial fossa 55
 posterior nucleus, hypothalamus
 113
 posterior parietal complex 108
 posterior temporal sulcus
 morality 140
 self-awareness 139
 postnatal depression 213, 239
 postural instability 119
 potassium ions, nerve impulses 72
 potentiation, forming memories
 158, 161, 197
 precuneus, imaging **34**
 predictions **169**, 173
 prefrontal cortex 66, 74, 209, 210,
 211, 212
 and brain “clocks” 190
 conscious movement 117
 and consciousness 138, 180,
 192, 193
 decision-making 169
 and depression 203, 239
 and doing two things at once
 168
 and dreams 189
 emotions 128
 morality 140
 post traumatic stress disorder
 241
 reward system 130
 social rejection 139
 thalamus and 60
 pregnancy
 prenatal depression 239
 diagnostic tests during 236
 premotor cortex
 conscious movement 117, 118
 decision-making 169
 thalamus and 60
 prenatal depression 239
 primary auditory cortex **30**, 95
 primary geniculate nucleus 95
 primary motor cortex 117, 118
 primary visual cortex
 and consciousness 180
 imaging **35**
 visual perception **88–89**
 primates
 and language 146, 147
 sexual attraction 134
 social behavior 138
 prions, Creutzfeldt-Jakob
 disease 231

- procedural memory 157, 160
 “processing gap,” and doing two things at once 168
 production aphasia 149
 progesterone 114
 progressive bulbar atrophy 235
 projective tests, personality 201
 prolactin 61, 114, 213
 proprioception 102, **104–105**
 propylthiouracil (PROP) 100
 prosencephalon 53
 prosopagnosia **85**
 prostheses 104, **218–219**
 proteins 197
 accumulation in brain 215
 Alzheimer’s disease 231
 amino acids 73
 Creutzfeldt-Jakob disease 231
 narcolepsy 238
 Proust, Marcel 99
Pseudomonas 228
 psychasthenia, and creativity 170
 psychoanalysis 222
 and dreams **189**
 psychopaths **141**
 psychosis 223
 psychotic deviation, and creativity 170
 puberty 211
 pulvinar 158
 punch-drunk syndrome 225
 pupil, eye 80, 88, 144
 Purkinje cells 63
 pus, brain abscesses **228**
 putamen 52, 54, 58
 body memory 120
 memory 156, 157
 planning movement 117
 pyramid 53
 pyramidal decussation 118
- Q**
 quadriplegia 236
 quality of life 134
- R**
 Ramón y Cajal, Santiago 9
 rapid eye movement (REM) sleep 188, 189
 Rasmussen, T. 9
 rats, investigating the brain 8
 reaction pathways **120–121**
 reading **152–153**
 dyslexia **153**, 248
 hyperlexia **153**
 reality, and consciousness 178
 recall and recognition **162–163**
 receptor cells
 sense of smell **96–97**
 thermoreceptors 102, 113
 touch 102
 recognition **163**
 faces 83, 84, **85**, 139, 163
 objects **83**
 oxytocin and 135
 visual perception 89
 recollection, memory 156
 red nucleus, cerebellum 117, 118
 referred pain **107**
 reflex actions **116**
 and body language 144
 regeneration, neurons 39, **71**
 regulation, movement and control **112–113**
 Reissner’s membrane 90
 rejection, social 139
 relaxation 186
 religion **172**
 renin 115
 repetitive behavior 223
 repolarization, nerve impulses 72
 reptiles, brains 48
 “reptilian brain” 48
 residual schizophrenia 244
 resting-state networks 184
 reticular activating system (RAS) 112
 reticular formation **112**
 altered brain-states 186
 and consciousness 180
 and dreams 189
 reticular laminar 90
 reticulospinal tract 118
 retina 80, **81**
 bionic eyes 218
 neurons 71
 optic nerve 43
 optogenetics 203
 seeing 88
 sending information to thalamus 60
 retronasal smell 96, 101
 Rett syndrome 242
 reward **130–31**
 and aging 214
 altruism 141
 emotions 127
 and humor 171
 sex, love, and survival **134–137**
 rhinal sulcus 65
 rhombencephalon 53
 ribosomes 70
 right hemisphere 53, **55**
 emotions **128**
 functions **57**
 and language 146, 151
 split brain 204
 right and wrong **140–141**
 right-handedness 57, 198, **199**
 rigidity, motor disorders 119
 risk-taking 141, 197, 211
 Ritalin 246
 rituals
 altering consciousness 186
 collective unconsciousness 187
 Rizzolatti, Giacomo 9, **11**
 robots 216–217
 and mirror neuron system 139
 nanorobots 218
 Rockefeller Plaza (New York) 175
 rods, retina 81, 88
 romantic love 134
 root hair plexus 102
 rootlets, spinal nerves 42
 Rorschach inkblot test 201
 rough endoplasmic reticulum 70
 rubrospinal tract 118
 Ruffini corpuscles 102
- S**
 sacral region, spinal nerves 42
 SAD (seasonal affective disorder) **239**
 sadness 128, 129
 adaptive behaviors 129
 facial expressions 137
 Salem witch trials 172
 Sally-Ann test 139
 salt, thirst 115
 satisfaction 130
 savants, autistic 164–165, 174, 249
 scalp
 nerves 54
 skin 54
 scanning the brain **12–13**, **14–35**
 skepticism, and patternmaking 173
 schizoid personality disorder 245
 schizophrenia 185, 203, 223, **244**
 and agency 193
 catatonia 190
 and creativity 170
 schizotypal personality disorder 245
 Schumann, Richard 239
 Schwann cells 71
 sclera 80, 81
 Searle, John 179
 seasonal affective disorder (SAD) **239**
 sebaceous glands 102
 seeing **88–89**
 seizures
 epilepsy 181, **226**
 status epilepticus **226**
 selective attention 182
 self
 agency and intention 193
 consciousness and 179, 180, **192–193**
 losing the **193**
 social behavior **138–139**
 split brain experiments 11
 selfishness 141
 semantic memory 157, 160
 semicircular canals, ear 90
 sensations 39
 cerebral cortex 67
 and consciousness 179
 laying down a memory 160
 multiple sclerosis 235
 “sensed presence” 172
 senses **76–109**
 attention 182–183
 dermatomes **42**
 ears **90–95**
 eyes **80–89**
 information sent to brain 38
 mixed senses 78
 pain **106–109**
 proprioception **104–105**
 smell **96–99**, **101**
 spinal cord 42
 synesthesia **78**
 taste **100–101**
 touch **102–103**
 sensory aphasia 149
 sensory cortex
 emotions 127
 laying down a memory 160, 161
 phantom limbs 104
 sensory nerves 208
 reflex actions 116
 spinal cord 42
 serotonin 73
 bipolar disorder 239
 and body dysmorphic disorder 242
 and depression 239
 genetics and 197
 and obsessive-compulsive disorder 241
 sex **134–135**
 sex hormones 114, 127
 sexual orientation 198
 shape-shifting illusions 174
 shell shock **241**
 shivering 113
 short-term memory **156**, 161
 shrinking-body illusion 174
 sign language, primates 146
 signal transmission 38, 39
 Silbo language 146
 simple partial seizures 226
 simulation **74–75**
 singing 151
 sinoatrial node 112
 sixth sense **104–105**
 size of brain **44**
 and aging 214
 and evolution of brain 49
 and intelligence 44, 168, 205
 skills
 brain development 210
 plasticity of brain 38
 skin
 dermatomes **42**
 injuries 106
 itching 102
 proprioceptors 105
 scalp 54
 structure 102
 touch receptors **102**
 skull 53, 54
 and cerebral cortex 66
 craniotomy 11, 232
 fractures 225
 head injuries 225
 phrenology **10**
 protection of brain 45
 and size of brain 44
 trepanation 8
 sleep **188–189**
 and aging 215
 brain waves 181
 circadian rhythm 63
 consolidation of memories 161
 dreams **189**
 narcolepsy **238**
 sleep paralysis 189
 sleep-wake cycles **115**, **188**
 sleeping drugs 189
 sleepwalking 188
 smell, sense of **96–99**
 brain areas **101**
 chemistry of **96**
 and emotions 126
 limbic system 64
 perception of smell **98–99**
 smiling 129, **137**
 smoking, nicotine
 addiction 245
 smooth endoplasmic reticulum 70
 sniffing, sense of smell 96
 social brain **134–143**
 autism spectrum disorders 223, 249
 developmental milestones 247
 expressions **136–139**
 personality markers 200
 the self and others **138–139**
 social awareness **138–139**, 184
 social groups 134
 and size of neocortex 138
 social phobia 240
 sodium ions 65, 72
 soldiers, bonding 135
 soma, neurons 70, 71
 somatic delusional disorder 244
 somatic sensory system, proprioception **104–105**
 somatization disorder **242**
 somatosensory cortex **103**
 executing a movement 118
 pain 108
 phantom limbs 104
 self-consciousness 192
 sense of taste 101
 Sophie (robot) 216
 sound
 attention 183
 dyslexia 153
 hearing 90, 91, **92–95**
 language 146
 localization 92
 seeing with (soundscapes) **89**
 Tourette’s syndrome 243
 spastic cerebral palsy 237
 spasticity 119
 spatial awareness 65
 spatial memory **162**
 spatial relationships, visual pathways 85
 spatial skills 157
 specific language impairment 149
 spectrums, mental disorders **223**
 speech
 anatomy of 147
 conversation **150–151**
 development of 93
 dyslexia **153**
 hearing 93
 hyperlexia **153**
 language areas 148
 origins of language **146–147**
 problems **149**
 stuttering **149**
 Wernicke’s area 92
 Sperry, Roger 9, **11**
 sphenoid bone 53, 54
 spina bifida 236
 spina bifida occulta 237
 spinal accessory nerve 43
 spinal cord **42**, 53, 208
 cerebrospinal fluid 45
 disorders 119
 executing a movement 118–119
 lumbar puncture 227
 motor neuron disease 235
 neural-tube defects **237**
 pain signals 106
 reflex actions 116
 spinal tracts 118–119
 spinal nerves 40, **42**
 patellar spinal reflex 116
 spindle cells 126
 spiritual experiences 172, 173
 split brain **11**, **204**
 stammering see stuttering
 stapes 90, 91, 94
 state-dependent memory **162**
 status epilepticus 226
 stem cells 197, 218
 stereocilia 90
 stereograms 83
 stereoscopic smell **98**
 stereotactic brain surgery 232
 stimulus and reward 130
 stomach
 hormones 114, 115
 panic responses 127
 storage, memory **158–159**
 strange brains **204–205**
Streptococcus 228, 241
 stress
 anxiety disorders 240
 family experiences 199
 and intelligence 168
 post traumatic stress disorder (PTSD) **164**, **241**
 shell shock **241**
 stress response 240
 stria terminalis 126
 stress headaches 224
 stria terminalis 126
 striatum, and social behavior 200
 stroke 203, 228, **229**
 brain remodeling 197
 and language problems 149
 motor disorders 119
 neurostimulation 203
 speech problems 151
 vascular dementia 230
 structures of the brain **52–55**
 stuttering 147, **149**
 subarachnoid hemorrhage **229**
 subarachnoid space 56
 aging 214
 lumbar puncture 227
 spinal cord 42
 subcutaneous tissue,
 pain signals 107
 subdural hematoma 229
 subdural hemorrhage **229**
 subfornical organ 115

- subjective time 190
 substantia nigra **58**
 and brain “clocks” 190
 Parkinson’s disease 58, 234
 planning movement 117
 subthalamic nuclei 53, 55, **58**
 planning movement 117
 succubi 189
 sugar addiction **115**
 suicide 197
 sulci (sulcus) 54, 209
 cerebral cortex 66, 67, 69
 Sumerians 8
 “superhighway,” intelligence **168**
 superior colliculus 53, 62
 attention 182
 emotions 129
 superior olive, hearing 95
 superior sagittal sinus 56
 superior temporal sulcus
 and body language 144
 conflicting emotions 137
 and numbers 169
 supernatural **173**, 187
 superstition **172–173**
 “supertasters” 100
 supplementary motor cortex
 conflicting emotions 137
 conscious movement 117, 118
 and consciousness 180
 disorders 119
 pain signals 108
 support cells 68, 71
 suprachiasmatic nucleus (SCN)
 113, 115
 surgery
 brain **109**, **232–233**
 deep brain stimulation 218, 233
 fetal 236
 general anesthetics **112**
 lobotomy **11**
 surprise
 adaptive behaviors 129
 facial expressions 136
 survival, sex, love and **134–135**
 sustained attention 182
 swearing, Tourette’s syndrome
 243
 sweat glands 99, 102
 Swift, Jonathan 205
 Sylvian fissure 57, 66
 symbols
 collective unconsciousness 187
 communication 146
 dreams and 189
 reading 152
 symmetry of brain, and language
 146
 symmetry of face, and sexual
 attraction 134
 sympathy **140**
 synapses 70, **71**
 memory 158
 nerve impulses 72, **73**
 neural networks 41
 synaptic cleft 71, 73, 119
 synaptic vesicles 73
 synesthesia **78**, 164
- T**
 tangles, aging brain 214, 215
 taste **100–101**
 laying down a memory 160
 taste associations **101**
 tau 215
 taxi drivers, spatial memory 162
 technology **218–219**
 tectorial membrane 90
 teenagers
 emotions 39
 teenage brain **211**
- telencephalon 53
 temperature, thermoreceptors
 102, **113**
 temporal bones 54
 temporal lobe 66
 altered brain states 186
 and consciousness 180
 and conversation 151
 imaging **21**
 memory 156, 157
 morality 140
 olfactory cortex 101
 reading 152
 and the supernatural 173
 visual pathways 84
 weird experiences 172
 temporoparietal junction 69
 self-awareness 139
 tendons, proprioceptors 104, 105
 tennis players 120–121
 tension headaches **224**
 testosterone 199, 211, 213
 thalamic bodies 60
 thalamus 53–56, **60**
 altered brain states 186
 and consciousness 180
 and dreams 189
 emotions 126, 127, 128
 functions 57, 58
 gender differences 198
 general anesthetics 112
 hearing 94, 95
 imaging **26**
 laying down a memory 160
 limbic system 64
 memory 156
 and obsessive-compulsive
 disorder 241
 pain relief 107
 pain signals 108
 planning movement 117
 post traumatic stress disorder
 241
 sense of taste 101
 and stress 199
 Tourette’s syndrome 243
 visual perception 88
The Matrix (film) 180
 theory of mind (ToM) **139**
 empathy and sympathy 140
 thermoreceptors 102, **113**
 thinking 39, **166–175**
 belief and superstition **172–173**
 brain-machine interfaces 216
 cognitive illusions **174–175**
 and consciousness 179
 controlling prostheses 218, 219
 creativity **170–171**
 decision-making **169**
 emotions 126
 humor **171**
 intelligence **168–169**
 language and 179
 number sense **169**
 self-consciousness **192–193**
 Third Eye 178
 thirst **115**
 thoracic region, spinal nerves 42
 thoracic spinal cord 53
 thought see thinking
 threat, emotions 128
 three-dimensional brain
 imaging 12
 three-dimensional vision **83**
 paradox illusions **175**
 thrill-seeking 131, 200, 214
 throat
 and evolution of language 147
 motor neuron disease 235
 speech 151
 thrombus
 stroke 229
- transient ischemic attacks 228
 thymus gland 114
 thyroid gland 114
 thyroid-stimulating hormone
 (TSH) 61, 114
 tics 119
 Tourette’s syndrome 243
 timbre, music 93
 time **190–191**
 tiredness 115
 chronic fatigue syndrome **225**
 tobacco, addiction 130
 Tomatis, Alfred 93
 tone deafness 248
 tongue
 sense of taste **100**
 speech 151
 tonic seizures 226
 tonic-clonic seizures 226
 top-down processing **79**, **87**
 touch, sense of **102–103**
 mirroring **122**
 Tourette’s syndrome **243**
 tower illusion 175
 trait tests, personality 201
 trances 186
 transcortical motor aphasia 149
 transcortical sensory aphasia 149
 transcranial direct current
 stimulation (tDCS) 203
 transcranial magnetic stimulation
 (TMS) 171, 203
 transient ischemic attacks
 (TIA) **228**
 translocation, chromosomes 236
 transnasal surgery **232**
 transsexuals, stria terminalis 126
 trauma
 and epigenetic changes 197
 laying down a memory 160
 post traumatic stress disorder
 (PTSD) **164**, **241**
 traumatic disorders 222, 223
 traumatic memory **164**
 trepanation 8, 11
 triangle
 imposed 174
 Penrose 175
 tribar 175
 trigeminal nerve 43, 53
 facial pain 107
 sense of taste 101
 “trigger points,” facial pain 107
 trisomy 21 (Down
 syndrome) **236**
 trochlear nerve 43
 trust, oxytocin 135
 truth telling 217
 trypanophobia 240
 tumors **230**
 surgery 232
 tunnel of Corti 90
 turtles, brains 48
 twins
 genetics **199**
 religion 172
 “two minds” 82
 tympanic canal 90
 tympanum 90, 94
- U**
 “uncanny valley” **217**
 unconscious mind
 decision-making **11**
 emotions **127**
 memories 160
 proprioception 104
 psychoanalysis 222
 sensory information **79**
 unconscious action 116, 117,
 120–121
- unconscious vision 84
 unconsciousness 192
 collective unconsciousness **187**
 coma **238**
 epileptic seizures 226
 general anesthetics 112
 head injuries 225
 sleep 188
 understanding 179
 undifferentiated schizophrenia
 244
 unipolar depression 223
 unipolar neurons 71
 United States of America, cultural
 influences 199
- V**
 vacuoles 70
 vagus nerve 43
 pacemakers **219**
 values, morality 140
 Van Gogh, Vincent 170–173
 variant CJD **231**
 vascular dementia 230
 vase-face illusion 174
 vasopressin 135
 “vegetative” centers 57
 veins, arteriovenous
 malformation 229
 ventral pathway, vision **84–85**, 89
 ventral pontine syndrome 63
 ventral striatum 128
 ventral tegmental area (VTA) 115,
 130
 ventricles 57
 aging 215
 cerebrospinal fluid 45, 57
 hydrocephalus 237
 ventriquoists 174
 ventrolateral prefrontal cortex 169
 ventrolateral preoptic nucleus 188
 ventromedial nucleus,
 hypothalamus 113
 ventromedial prefrontal cortex
 (VMPC)
 and belief 172
 morality 140
 vermiform 63
 vertebrae 53
 spina bifida 237
 spinal nerves 42
 vertebrates, evolution of brain **48**
 Vesalius, Andreas 8
 vestibular canal 90, 94
 vestibulocochlear nerve 43, 90
 vestibulospinal tract 118
 vibrations
 hearing 90, 91, 94, 95
 touch receptors 102
 virtual reality
 “brain-in-a-vat” 180
 pain relief 109
 virtual body 193
 viruses
 encephalitis 227
 meningitis 227
 visceral pain 107
 vision **80–89**
 bionic eyes **218**
 bottom-up/top-down
 processing 79
 cerebral cortex 67, 68
 developmental milestones 247
 infant brain 209
 multiple sclerosis 235
 reactions to visual stimuli
 120–121
 seeing **88–89**
 split brain 204
 visual agnosia 85
 visual attention defects 85
- vision *continued*
 visual neurons 78
 visual pathways **84–85**
 visual perception **86–87**, **88–89**
 visual phantoms **181**
 visual cortex 81, **82–83**
 and consciousness 180
 and dreams 189
 imaging **35**
 and memory 157, 158, 161
 mind reading 217
 reading 152
 and recognition 163
 thalamus and 60
 visual perception **88–89**
 visual word-recognition area 152
 vocal tics, Tourette’s syndrome
 243
 voices, autism spectrum
 disorders 249
 volume of brain **44**
 vomeronasal region (VMO) 99
- W**
 Wada test 146
 wanting 130
 water
 in brain 44
 thirst **115**
 water on the brain
 (hydrocephalus) **237**
 weight of brain **44**
 Wernicke, Karl 9, **10**
 Wernicke’s area 10, 92, **148**
 aphasia 149
 conversation 150
 whistles, Silbo language 146
 white blood cells 45, 106
 white matter 44, 69
 aging 215
 cerebellum 62, 63
 spinal cord 42
 in thalamus 60
 Williams syndrome 248
 Willis, Thomas 8
 Wiltshire, Stephen 164–65
 wine tasters 100
 women
 emotions 126, 127
 female brain 198
 and sexual attraction 134
 words 146
 “inner ear” 157
 Tourette’s syndrome 243
 see also language
 working memory 156, 157, 161
 worms 48
 worrying 240
 hypochondria 242
 writers, creativity 170
 writing **152–153**
 dysgraphia **153**
 dyslexia 153, 248
 hyperlexia 153
 mirror writing 153
- XYZ**
 X-rays, computed tomography
 (CT) 12
 Yakovlevian torque 57
 yawning 138
 zones and partitions **56–57**

ACKNOWLEDGMENTS

For the third edition, DK would like to thank Dharini Ganesh for editorial assistance, Pooja Pipil and Garima Agarwal for design assistance, Helen Peters for compiling the index, and Jamie Ambrose for proofreading.

The publisher would like to thank the following for their kind permission to reproduce their photographs:

(Key: a-above; b-below/bottom; c-center; f-far; l-left; r-right; t-top)

Edward H. Adelson: 87cr; **Alamy Images:** Alan Dawson Photography 146bl, Alan Graf / Image Source Salsa 173br, allOver photography 45tr, Bubbles Photolibrary 186cr, Mary Evans Picture Library 174br, Photo by M. Flynn / © Salvador Dali, Gala-Salvador Dali Foundation, DACS, London 2009 191t, Paul Hakima 2001t, Barrie Harwood 202cr, Hipix 10bc, Kirsty McLaren 130c, Mira 44bc, 115cr, Robin Nelson 179c, Old Visuals 92cra, Photogenix 122tl, Pictorial Press 200–201, Stephanie Plick / dpa picture alliance archive 181b, Simon Reddy 114t, Supapix 153tr, Tetra Images 123tl, vario images GmbH & Co. KG 190cr; ZUMA Press, Inc. 135br; **Arionaurto Cartuns:** 171cr; **Helen Dr. Jason J.S. Barton:** 85cr; **George Bartzokis, MD, UCLA Neuropsychiatric Hospital and Semel Institute:** 214cl; **Dr. Theodore W. Berger, University of Southern California:** 161tl; **Blackwell Publishing:** European Journal of Neuroscience Vol. 25, Issue 3, pp.863–871, Renate Wehrle et al., Functional microstates within human REM sleep: first evidence from fMRI of a thalamocortical network specific for phasic REM periods. © 2007 John Wiley & Sons, Inc. / Image courtesy Renate Wehrle 189fcr; **© EPFL / Blue Brain Project:** 74cb, 75c, Thierry Parel 75cr; **The Bridgeman Art Library:** Archives Charmet 8ftl, 10cl, Bibliothèque de l'Institut de France 7tl, The Detroit Institute of Arts / Founders Society purchase with Mr. & Mrs. Bert L. Smokler & Mr. & Mrs. Lawrence A. Fleischman funds 189bc, Maas Gallery, London 134c, Peabody Essex Museum, Salem, Massachusetts 172bl, Royal Library, Windsor 174tr; **Vergleichenle Lokalisationslehre der Grosshirnrinde, Dr. K. Brodmann:** 1909, publ: Verlag von Johann Ambrosius Barth, Leipzig 67bc; **Dr. Peter Brugger:** 173tr; **Caltchec Brain Imaging Center:** J. Michael Tyszka & Lynn K. Paul 204ca; **Center for Brain Training (www.centerforbrain.com):** 222bl; **Copyright Clearance Center – Rightslink:** Brain 2008 131(12):3169–3177; doi:10.1093/brain/awn251, Iris E. C. Sommer et al., Auditory verbal hallucinations predominantly activate the right inferior frontal area. Reprinted by permission of Oxford University Press 193cra, Brain Lang 80: 296–313, 2002, Murray Grossman et al., Sentence processing strategies in healthy seniors with poor comprehension: an fMRI study (c) 2002 with permission from Elsevier 215tl, Brain Vol. 125, No. 8, 1808–1814, Aug. 2002, Sterling C. Johnson et al., Neural correlates of self-reflection (c) 2002. Reprinted with permission of Oxford University Press 192bl, Brain, Vol. 122, No. 2, 209–217, Feb. 1999, Noam Sobel et al., Blind smell: brain activation induced by an undetected air-borne chemical © 1999 by permission of Oxford University Press 98bl, Current Biology, Vol. 13, Dec. 16, 2003, Nouchine Hadjikhani and Beatrice de Gelder, Seeing Fearful Body Expressions Activates the Fusiform Cortex and Amygdala, 2201–2205, Fig. 1, © 2003, with permission from Elsevier Science Ltd. 144br, Int J Dev Neurosci. 2005 Apr–May;23(2–3):125–141, Robert Schultz, Developmental deficits in social perception in autism: the role of the amygdala and fusiform face area © 2005, with permission from Elsevier 249cr, International Journal of Psychophysiology, V63, No. 2, Feb. 2007, p.214–220, Michael J. Wright & Robin C. Jackson, Brain regions concerned with perceptual skills in tennis, An fMRI study (c) 2007 with permission from Elsevier 121, Journal of Neurophysiology 96: 2830–2839, 2006; doi:10.1152/jn.00628.2006, Arthur Wingfield & Murray Grossman, Language and the Aging Brain: Patterns of Neural Compensation Revealed by Functional Brain Imaging © 2006 The American Physiological Society 215, Journal of Neurophysiology Vol. 82 No. 3 Sept. 1999 1610–1614, 128cl, Journal of Neuroscience, Aug. 27, 2008 Vol. 28 p.8655–8657, Duerden & Laverdure-Dupont, Practice makes cortex. (c) The Society of Neuroscience 1573r, Journal of Neuroscience, May 28, 2008, 28(22):5623–5630, Todd A. Hare et al., Dissociating the Role of the Orbitofrontal Cortex and the Striatum in the Computation of Goal Values and Prediction Errors © 2008. Printed with permission from The Society for Neuroscience 169r, Journal of Neuroscience, Nov. 7, 2007, 12190–12197, Hongkeun Kim, Trusting our memories: Dissociating the Neural Correlates of Confidence in Veridical versus Illusory Memories. © 2007, Society for Neuroscience 164c, Michael S. Beauchamp & Tony Ro; Adapted with permission from Figure 1, Neural Substrates of Sound-Touch Synesthesia after a Thalamic Lesion; Journal of Neuroscience 2008 28:13696–13702 78bl, The Journal of Neuroscience, Dec. 7, 2005 • 25(49):11489–11493, Peter Kirsch et al., Oxytocin Modulates Neural Circuitry for Social Cognition and Fear in Humans 127c, Reprinted from The Lancet, Vol. 359, Issue 9305, Page 473, Feb. 9, 2002, Half a Brain, Johannes Borgstein & Caroline Grootendorst, © 2002, with permission from Elsevier 205tr, Nature 373, 607–609 (Feb. 16, 1995), Bennett A. Shaywitz et al., Yale, Sex differences in the functional organization of the brain for language. Reprinted by permission from Macmillan Publishers Ltd. 198cl, Nature 415, 1026–1029 (Feb. 28, 2002), Antoni Rodriguez-Fornells et al., Brain potential and functional MRI evidence for how to handle two languages with one brain © 2002. Reprinted by permission of Macmillan Publishers Ltd. 149tr, Nature 419, 269–270 (Sept. 19, 2002), Olaf Blanke et al., Neuropsychology: Stimulating illusory own-body perceptions (c) 2002. Reprinted by permission from Macmillan Publishers Ltd. 173cr, Nature Neuroscience 7, 801–802 (July 18, 2004) | doi:10.1038/nrn1291, Hélène Gervais et al., Abnormal cortical voice processing in autism © 2004 Reprinted by permission from Macmillan Publishers Ltd. / image courtesy Mónica Zilbovicius 249cbr, Nature Neuroscience Vol. 10, Jan. 1, 2007, p.119 Figure 3, Yee Jon Kim et al., Attention induces synchronization-based response in steady-state visual evoked potentials © 2007. Reprinted by permission from Macmillan Publishers Ltd. 183tr, Nature Reviews

Neuroscience 4, 37–48, Jan. 2003 | doi:10.1038/nrn1009; Arthur W. Toga & Paul M. Thompson, Mapping brain asymmetry © 2003. Reprinted by permission from Macmillan Publishers Ltd. / image courtesy Dr. Arthur W. Toga, Laboratory of Neuro Imaging at UCLA 57cr, Nature Reviews Neuroscience 7, 406–413 (May 2006) | doi:10.1038/nrn1907, Usha Goswami, Neuroscience and education: from research to practice? © 2006. Reprinted by permission from Macmillan Publishers Ltd. / courtesy Dr. Guinevere Eden, Georgetown University, Washington, DC 248r, redrawn by DK courtesy Nature Reviews Neuroscience 3, 201–215 (March 2002), Maurizio Corbetta & Gordon L. Shulman, Control of goal-directed and stimulus-driven attention in the brain © 2002 Reprinted by permission from Macmillan Publishers Ltd. 183cb, NeuroImage 15: 302–317, 2002, Murray Grossman et al., Age-related changes in working memory during sentence comprehension: an fMRI study (c) 2002 with permission from Elsevier 215ftl, Neuron, March 6, 2013, 77(5): 980–991, Fig 6; Charles E. Schroeder et al., “Mechanisms Underlying Selective Neuronal Tracking of Attended Speech at a Cocktail Party” © 2013 with permission from Elsevier (http://dx.doi.org/10.1016/j.jneurosci.2012.12.037) 92tr, Neuron Vol. 42 Issue 4, May 16, 2004, p.687–695, Jay A. Gottfried et al., Remembrance of Odors Past: Human Olfactory Cortex in Cross-Modal Recognition Memory; with permission from Elsevier 162tr, Neuron, Vol. 42, Issue 2, 335–346, Apr. 22, 2004, Christian Keysers et al., A Touching Sight (c) 2004 with permission from Elsevier 122bl, Neuron, Vol. 45 Issue 5, 651–660, March 3, 2005, Helen S. Mayberg et al., Deep Brain Stimulation for Treatment-Resistant Depression (c) 2005 with permission from Elsevier Science & Technology Journals 239cl, Neuron, Vol. 49, Issue 6, Mar 16, 2006, p.917–927, Nicholas B. Turke-Browne, Do-Joon Yi & Marvin M. Chun, Linking Implicit and Explicit Memory: Common Encoding Factors and Shared Representations © 2006 with permission from Elsevier 159cbr, Psychiatric Times Vol. XXII No. 7, May 31, 2005, Dean Keith Simonton, PhD, Are Genius and Madness Related: Contemporary Answers to an Ancient Question, (c) 2005 CMPMedica, reproduced with permission of CMPMedica 170br, Science 2010: 329 (5997): 1358–1361 “Prediction of Individual Brain Maturity Using fMRI.” Fig. 2, Nico U.F. Dosenbach et al. (c) 2010 The American Association for the Advancement of Science, Reprinted with permission from AAAS 210cr, Science Feb. 20, 2004; © 2004 The American Association for the Advancement of Science, T. Singer, B. Seymour, J. O’Doherty, H. Kauber, R.J. Dolan, C. De. Frith, Empathy for Pain involves the affective but not sensory components of pain 138br, Science, July 13, 2007, Vol. 317, No. 5835, pp.215–219, Fig. 2, Brendan E. Depue et al., Prefrontal regions orchestrate suppression of emotional memories via a two-phase process. Reprinted with permission from AAAS 158cl, Science, Oct. 10, 2003, Vol. 302, No. 5643 p.290–292, Naomi I. Eisenberger et al., Does Rejection Hurt? An fMRI Study of Social Exclusion © 2003 The American Association for the Advancement of Science 139tl, Science, Vol. 264, Issue 5162, 1102–1105 (c) 1994 by American Association for the Advancement of Science 174c, A.R. Damasio, T. Grabowski, R. Frank, A.M. Galaburda & A.R. Damasio, “The return of Phineas Gage: clues about the brain from the skull of a famous patient” / Dept of Image Analysis Facility, University of Iowa 141cr, Trends in Cognitive Sciences, Vol. 11, Issue 4, Apr. 2007 p.158–167 Naotsugu Tsuchiya & Ralph Adolphs, Emotion & Consciousness © 2007 Elsevier Ltd. / image: Ralph Adolphs 128tr; **Corbis:** Alinari Archives 6tl, Steve Allen 39bc, The Art Archive 8tl, 8cb, Bettmann 6tc, 6tr, 7c, 8c, 8bl, 8bc, 9ca, 9br, 11tr, 75br, 136cl, 136c, 136cl, 137cra, 187br, 204–205, 205cra, Blend Images 215c, Bloomimage 186bl, Keith Brofsky 144tr, Fabio Cardoso 157c, Peter Carlsson / Etsa 96br, Christophe Boissieuvi 118bl, Gianni Dagli Ortì 85bl, Kevin Dodge 140l, Ecoscene / Angela Hampton 39cr, EPA 186tl, 190t, 248cla, ER Productions 222cr, Fancy / Vncr 159tc, Peter M. Fisher 179tr, Robert Garvey 134tl, Rune Hellestad 196cl, Hulton Collection 99cr, Hutchings Stock Photography 104c, Image 100 157bl, Tracy Kahn 168c, Ed Kashi 151tr, Helen King 183cr, 183cr (Man using computer), Elisa Lazo de Valdez 180tl, Walter Lockwood 182cra, Tim McGuire 39t, MedicalRF.com 9tr, Mediscan 199cr, Moodboard 38br, 123tr, 157br, 182cr, Greg Newton 186fbr, Tim Pannell 186br, PoodlesRock 7tr, Premium Stock 159cr, Louie Psihoyos 99bl, Radius Images 185b, Redlink 182tr, Reuters 196–197, Lynda Richardson 159cl, Chuck Savage 138bc, 198tr, Ken Saeed 135t, Sunset Boulevard 57t, Sygma 84br, 180bc, Tim Tadder 38tr, 39bl, William Taufic 172tl, 184c, 189br, TempSport 118–119, Thinkstock 38c, Visuals Unlimited 213tr, Franco Vogh 13c, Zefa 101br, 182ftr, 186bc, 192r, 214cr; **Luc De Nil, PhD:** & Kroll, R. (2000). Nieuwe inzichten in de rol van de hersenen tijdens het stotteren van volwassenen aan de hand van recent onderzoek met Positron Emission Tomography (PET). Signaal, 32, 13–20. 149cr; **Dr. Jean Decety:** Neuropsychologia, Vol. 46, Issue 11, Sept. 2008, 2607–2614, Jean Decety, Kalina J. Michalska & Yoko Akitsuki, Who caused the pain? An fMRI investigation of empathy and intentionality in children. © 2008 with permission from Elsevier. 140tr; **Dr. José Delgado:** 10bl; **Brendan E. Depue:** 164b; **DACS (Design and Artists Copyright Society):** 191; **Dorling Kindersley:** Bethany Dawn 138clb, Colin Keates / Courtesy of the Natural History Museum, London 49cr; **Dreamstime.com:** Sean Pavone 175cl; PhotoEuphoria 152tl; **Henrik Ehrsson et al.:** Neural substrate of body size: illusory feeling of shrinking of the waist; PLoS Biol 3(12): e412, 2005 174cr; **© 2012 The M.C. Escher Company – Holland, All rights reserved.** www.mcescher.com: 175br; **Henrik Ehrsson et al.:** Staffan Larsson 193bl; **Explore-At-Bristol:** 87c; **eyevine:** 11cl; **Dr. Anthony Feinstein, Professor of Psychiatry, University of Toronto:** 242cbr; **Professor John Gabrieli:** Stanford Report, Tuesday, Feb. 25, 2003, Remediation training improves reading ability of dyslexic children 153cl; **Getty Images:** AFP 145t, 202bl, The Asahi Shimbun 216bl, Assembly 187t, John W. Banagan 240bl, Blend Images 247t, The Bridgeman Art Library / National Portrait Gallery, London 205cla, Maren Curuso 100cr, Praktik Chorge / Hindustan Times 216r, Comstock Images 134tr, Digital Vision 144tc, ElementImages 116–117, 153cr, 170bl, 185cr, Gazimal 182cbr, Tim Graham 162br, Louis Grandadam 153cr, Hulton Archive 11bl, 93cr, 129b, 160–161 (girls ice cream), 162–163t, 190b, 201tr, 202br, 205c, 222tl, 222tr, 242bl, International Rescue 105, Lifestock 144cb, Tanya Little 184br,

Don Mason 135cb, Victoria Pearson 215tr, Peter Ginter 243t, Hulton Archive /Stringer 199cl, Photo and Co. 127cra, Photodisc 215cr, 241cr, Popperfoto 241tr, Louis Psihoyos 239tr, Purestock 215tc, Juergen Richter 175tr, Charlie Schuck 162bl, Chad Slattery 131t, Henrik Sorensen 189bl, Sozajitten / Datacraft 247cr, Tom Stoddart 119br, David Sutherland 191b, Time & Life Pictures 8cbr, VCG 217bl, Bruno Vincent 235bc; WireImage 240clb, Elis Years 240bl; **Jordan Grafman PhD:** 141t; **Dr. Hunter Hoffman, U.V.:** 109t, 109c, 109cr; **Courtesy of the Laboratory of Neuro Imaging at UCLA and Martinos Center for Biomedical Imaging at MGH, Consortium of the Human Connectome Project – www.humanconnectomeproject.org; Courtesy of the Laboratory of Neuro Imaging at UCLA and Martinos Center for Biomedical Imaging at MGH, Consortium of the Human Connectome Project – www.humanconnectomeproject.org; Courtesy of the Laboratory of Neuro Imaging at UCLA and Martinos Center for Biomedical Imaging at MGH, Consortium of the Human Connectome Project – www.humanconnectomeproject.org; 74r; **Imprint Academic:** The Volitional Brain: Towards a neuroscience of free will, Ed Benjamin Libet, Anthony Freeman & Keith Sutherland © 1999 / Cover illustration by Nicholas Gilbert Scott, Cover design by J.K.B. Sutherland 11cr; **Photographic Unit, The Institute of Psychiatry, London:** 247cl; **iStockphoto.com:** 175c, Jens Carsten Rosemann 85t, Kiyoshi Takahase Segundo 181cr; **Frances Kelly:** Lorna Selife 174tc; **Pilyoung Kim et al.:** Fig. 1 from “The Plasticity of Human Maternal Brain: Longitudinal Changes in Brain Anatomy During the Early Postpartum Period,” Behavioural Neuroscience 2010, Vol. 124, No. 5 695–700 (c) 2010 American Psychological Association DOI: 10.1037/a0020884 213bl; **© 2008 Little et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited (see http://creativecommons.org/licenses/by/2.5/);** Little A.C., Jones B.C., Waitt C., Tiddeman B.P., Feinberg D.R., et al. (2008) Symmetry Is Related to Sexual Dimorphism in Faces: Data Across Culture and Species. PLoS ONE 3(5): e2106. doi:10.1371/journal.pone.0002106 134bl; **Ian Loxley / TORRO / The Cloud Appreciation Society:** 172–173t; **Library of Congress, Washington, DC:** Official White House photo by Pete Souza. 199cl, Orren Jack Turner, Princeton, NJ 199c; **Mairéad MacSwiney:** Brain. 2002. Jnl.125(Pt 7):1583–1593, B. Woll, R. Campbell, P.K. McGuire, A.S. David, S.C. Williams, J. Suckling, G.A. Calvert, M.J. Brammer, Neural systems underlying British Sign Language & audio-visual English processing in native users © 2002. Reprinted by permission of Oxford University Press 78cl; **Rogier B. Mars:** Rogier B. Mars, Franz-Xaver Neubert, MaryAnn P. Noonan, Jerome Sallet, Ivan Toni, and Matthew F. S. Rushworth, On the relationship between the “default mode network” and the “social brain”; Front. Hum. Neurosci., June 21, 2012 | doi: 10.3389/fnhum.2012.00189 184bl; **Mediscan:** 246tl; **Pierre Metivier:** 178tc; **Massachusetts Institute of Technology (MIT):** Ben Deen / Rebecca Saxe / Department of Brain and Cognitive Sciences and the McGovern Institute, MIT / Nat Comm 8, Article number: 13995 (2017) 209bc; **MIT Press Journals:** Journal of Cognitive Neuroscience Nov. 2006, Vol. 18, No. 11, p.1789–1798, Angela Bartolo et al., Humor Comprehension and Appreciation: A fMRI study, © 2006 Massachusetts Institute of Technology 171cbr, Journal of Cognitive Neuroscience, Fall 1997, V9, No. 5 p.664–686, D. Bavelier et al., Sentence reading: a functional MRI study at 4 Tesla, © 1997 Massachusetts Institute of Technology 146br; **The National Gallery, London:** Applied Vision Research Unit / Muller Alastair Gale, Dr. David Wooding, Dr. Mark Muggleston & Kevin Purdy with support of Derby University / Telling Time exhibition at National Gallery 86–87; **The Natural History Museum, London:** 103cr; **Neuramatrix (www.neuramatrix.com):** 75bl; **Oregon Brain Aging Study, Portland VAMC and Oregon Health & Science University:** 214–215b; **Oxford University Press:** 78; **Professor Eraldo Paules:** 153cla; **Pearson Asset Library:** Pearson Education Ltd. / Jules Selmes 122br; **Pearson Group:** © 1991 Pearson Assessment. Reproduced with permission. 85br; **Jack Pettigrew, FRs:** 87br; (c) **Phillips:** Philips Design concept dress “Bubble” 129cl, 129c; **Photolibrary:** David M. Dennis 8t; **PLoS Biology:** Cantlon J.F., Brannon E.M., Carter E.J., Pelphrey K.A. (2006) Functional Imaging of Numerical Processing in Adults and 4-y-Old Children. PLoS Biol 4(5): e125 doi:10.1371/journal.pbio.0040125 169b, Gross L (2006) Evolution of Neonatal Imitation. PLoS Biol 4(9): e311, Sept. 5, 2006 doi:10.1371/journal.pbio.0040311. © 2006 Public Library of Science 11br; **PNAS, Proceedings of the National Academy of Sciences:** Based on Fig. 4 from https://doi.org/10.1073/pnas.0903627106 147cb, Based on Fig. 3 from https://doi.org/10.1073/pnas.0402680101 Copyright (2004) National Academy of Sciences, US 210–211b, 103, 15623–15628, Oct. 17, 2006, Jordan Grafman et al., Human fronto-mesolimbic network guide decisions about charitable donation © 2006 National Academy of Sciences, US 141tc, June 16, 2008 (DOI: 10.1073/pnas.0801566105) Ivanka Savic & Per Lindström, PET and MRI show differences in cerebral asymmetry and functional connectivity between homo- and heterosexual subjects © 2008 National Academy of Sciences, US 198bl, March 19, 2002, V99, No. 6, 4115–4120, Jeremy R. Gray et al., Integration of emotion & cognition in the lateral prefrontal cortex © 2002 National Academy of Sciences, US 169c, Vol. 105, No. 39 15106–15111, Sept. 30, 2008, Jean-Claude Dreher et al., Age-related changes in midbrain dopaminergic regulation of the human reward system, © 2008 National Academy of Sciences, US 130bl; **Press Association Images:** 182b, **Public Health Image Library:** Sherif Zaki, MD, PhD; Wun-Ju Shieh, MD, PhD, MPH 231b; **Marcus E. Raichle, Department of Radiology, Washington University School of Medicine, St. Louis, Missouri:** 148bl; **The Random House Group Ltd.:** Vintage Books, Ian McEwan, Enduring Love, 2004 244br; **Courtesy of the Rehabilitation Institute of Chicago:** 218–219b; **M. Reisert:** University Medical Center Freiburg; based on the algorithm in M. Reisert et al., Global fiber reconstruction becomes practical, NeuroImage Vol. 54, Issue 2, Jan. 15, 2011 pages 955–962 (www.ncbi.nlm.nih.gov**

/pubmed/20854913) 204cl; **Courtesy of Professor Katya Rubia:** based on data published in the American Journal of Psychiatry, 2009; 166: 83–94 248b; **Kosha Rupareil & Daniel Langbehn, University of Pennsylvania:** 217cra; Rex by Shutterstock: Imaginetchina 232–233; **Science Photo Library:** 12c, 14, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 51r, 113cl, 125r, 126cl, 174cl, 215cl, 228tr, 238bc, AJ Photo / Hop American 193cla, Anatomical Travelogue 177r, Tom Barrick, Chris Clark, SGHMS 13tr, 75cla, Dr. Lewis Baxter 239bl, David Becker 81t, Tim Beddow 244cl, Juergen Berger 218bl, Biophoto Associates 68bc, Dr. Goran Bredberg 90br, BSIP VEM 238br, BSIP, Astier-Chruq, Lille 232cl, BSIP, Ducloux 96cl, BSIP, SEENME 12br, Oscar Burriel 188cr, 187bc, Scott Camazine 12bc, CNRI 230t, 245tr, 245cr, Custom Medical Stock Photo 248cl, Thomas Deerinck, Ncmir 59, 68fbl, 126bc, 155r, Steven Needell 141cbr, 141br, Department of Nuclear Medicine, Charing Cross Hospital 224bl, Eye of Science 71c, 197bc, 218bc, Don Fawcett 111r, 119tl, Simon Fraser 146tr, 237t, Simon Fraser / Royal Victoria Infirmary, Newcastle Upon Tyne 9tc, 207r, Dr. David Furness, Keele University 69bl, GJLP 7bl, Pascal Goetgheluck 104br, Steve Gschmeissner 58l, 61cr, 68bl, 96t, 107bl, C.J. Guerin, PhD, MRC Toxicology Unit 57br, 60l, 63cr, 65cr, 238tr, Dr. M. O. Habert, Pitie-Salpêtrière, ISM 181cl, Prof. J. J. Hauw 234cl, Innerspace Imaging 9bl, 9–241 (sidebar), ISM 46, Nancy Kedersha 4–5, 8–256 (sidebar), 36–37, 50–51, 76–77, 110–111, 124–125, 132–133, 142–143, 154–155, 166–167, 176–177, 194–195, 206–207, 220–221, Nancy Kedersha / UCLA 68cl, James King-Holmes 91c, 109b, Mehau Kulyk 223cl, 227tr, Living Art Enterprises, LCC 12bl, 44b, 126br, Dr. Kari Lounatmaa 221t, 228tr, Dr. John Mazziotta et al. / Neurology 12tr, 93cl, Duncan Shaw 100tl, Medi-mation 232b, MIT AI Lab / Surgical Planning Lab / Brigham & Women’s Hospital 10br, Hank Morgan 12cr, 181fcl, 189cl, 189cr, 189fcl, John Greim 112cr, Paul Parker 81br, Prof. P. Motta / Dept. of Anatomy, University “La Sapienza”, Rome 81bl, 91tr, National Institutes of Health 230r, National Library of Medicine 9cr, Susumu Nishinaga 94br, David Parker 7tr, Alfred Pasiaska 61cl, 80t, 133r, 135bc, 137t, 234t, Pasiaska 170cla, Alain Pol, ISM 47, Dr. Huntington Potter 231cr, C. Poudras 58cr, Philippe Psaila 7tr, 107tl, John Reed 100tr, Jean-Claude Revy ISM 12cl, Sovereign, ISM 6bl, 6dc, 6br, 13cra, 13c, 37r, 62l, 64t, 208t, Dr. Linda Stannard 228tl, Andrew Syred 195r, Sheila Terry 120t, 153bc, Alexander Tsiaras 7tr, 13br, US National Library of Medicine 10tr, Wellcome Dept. of Cognitive Neurology 57bl, 127cr, 143r, 241br, Prof. Tony Wright 91br, Dr. John Zajicek 71c, 221r, Zephyr 13cr, 57bc, 119crb, 218tl, 228ca, 225cb, 227br, 228cl, 229bl, 237c; **seeingwithsound.com:** Peter B. L. Meijer 89br; **Roger Shepard:** Adapted from Legs-istential Quarterly, 1974, pen and ink; Published in artist’s book, Mind Sights, 1990 W.H. Freeman 175bc; **Society for Neuroscience:** Fig. 8 / Nemrodov et al., “The Neural Dynamics of Facial Identity Processing: Insights from EEG-Based Pattern Analysis and Image Reconstruction” 217tc; **Stephen Wilshire Gallery, London:** Stephen Wilshire, Aerial view of Houses of Parliament and Westminster Abbey, June 23, 2008, 164–165; **© 2009 Michael J. Tarr:** 83cra; **Taylor & Francis Books (UK):** Riddoch M.J., Humphreys G.W. Birmingham Object Recognition Battery (BORB). Lawrence Erlbaum Associates, 1993 85cbr; **The Art Archive:** Musée Corda Chantilly / Gianni Dagli Orti AA 11tl; **Thanks to Flickr user Leigh LeBlanc for the use of this image:** 69bc; **TopFoto.com:** 173bl, Imageworks 803b; **Peter Turkeltaub, MD, PhD:** 152cr; **UCLA Health:** 203t; **Dept. of Neurology, University Hospital Geneva:** paper, ref: Seek et al. (1998) Electroenceph 226v; **University of California, Los Angeles:** 242tl; **Dr. Katy Vincent, University of Oxford:** 108c; **Image: Tor Wager:** from H. Kobet et al., Neuroimage 2008 Aug. 15;42(2): 998–1031, Functional grouping and cortical-subcortical interactions in emotion: a meta-analysis of neuroimaging studies, Fig. 7 (www.ncbi.nlm.nih.gov/pubmed/18579414) 12clca; **Wellcome Images:** 222cra, Wellcome Photo Library 91br, Wessex Reg. Genetics Centre 236bl, 236bc; **Susan Whitefield-Gabriel, McGovern Institute for Brain Research at MIT:** 185cl; **Wikimedia Commons:** Thomasbg 243br, Van Gogh, Starry Night, MoMA, New York 170–171t; **Wikipeidia:** 10c, Histologie du Systeme Nerveux de l’Homme et des Vertébrètes, Vols. 1 & 2, A. Maloine. Paris 1911 9c, Sternberg, Robert J. (1986). “A triangular theory of love,” Psychological Review 93 (2): 119–135, doi:10.1037/0033-295X.93.2.119 134ca; **John Wiley & Sons Ltd.:** Chris Frith, Making up the Mind – How the brain creates our mental world, 2007 Blackwell Publishing © 2007 John Wiley & Sons Ltd. / image courtesy Chiara Portas 13bc, Psychological Science, Vol. 19, Issue 1, p.12–17, Trey Hedden et al., Cultural Influences on Neural Substrates of Attentional Control, © 2009 Association of Psychological Science 199br, **David Williams, University of Rochester:** 81tr; **Dr. Daniel R. Winberger:** 244bc; **Adapted with permission of S.F. Witelson:** Reprinted from The Lancet, Vol. 353, Issue 9170, p.2150, (June 19, 1999), Sandra F. Witelson et al., The exceptional brain of Albert Einstein, (c) 1999 with permission from Elsevier & S.F. Witelson 205br; **Rosalie Winard / Temple Grandin:** 249br, **Jason Woff, PhD, UNC:** 249tr; **Professor Michael J. Wright:** International Journal of Psychophysiology, V63, No. 2, Feb. 2007, p.214–220, Michael J. Wright & Robin C. Jackson, Brain regions concerned with perceptual skills in tennis, An fMRI study © 2007 with permission from Elsevier 121t; **Professor Semir Zeki:** 128br

Front & Back Endpapers: **Science Photo Library:** Innerspace Imaging

All other images © Dorling Kindersley

For further information, see: www.dkimages.com