



PSIKONEUROFISIOLOGI

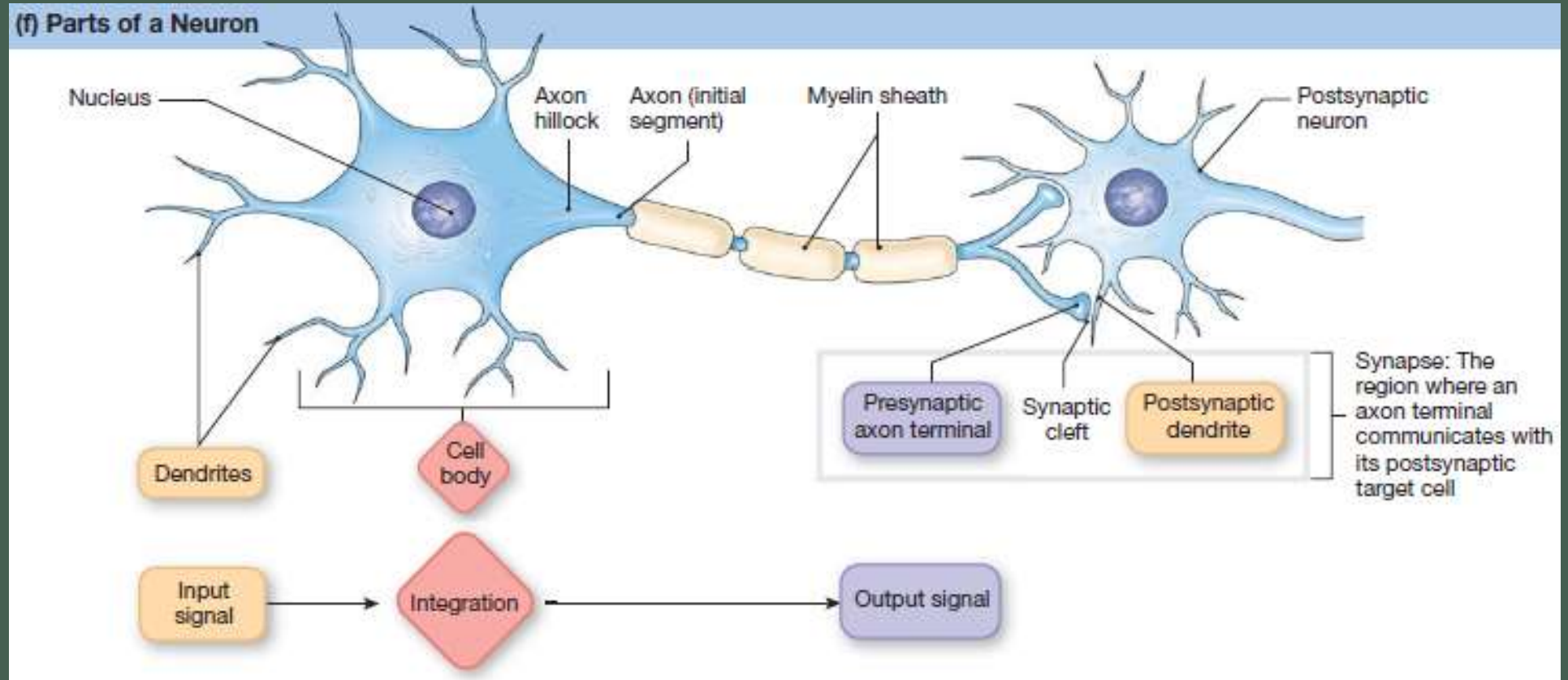
Astri Handayani

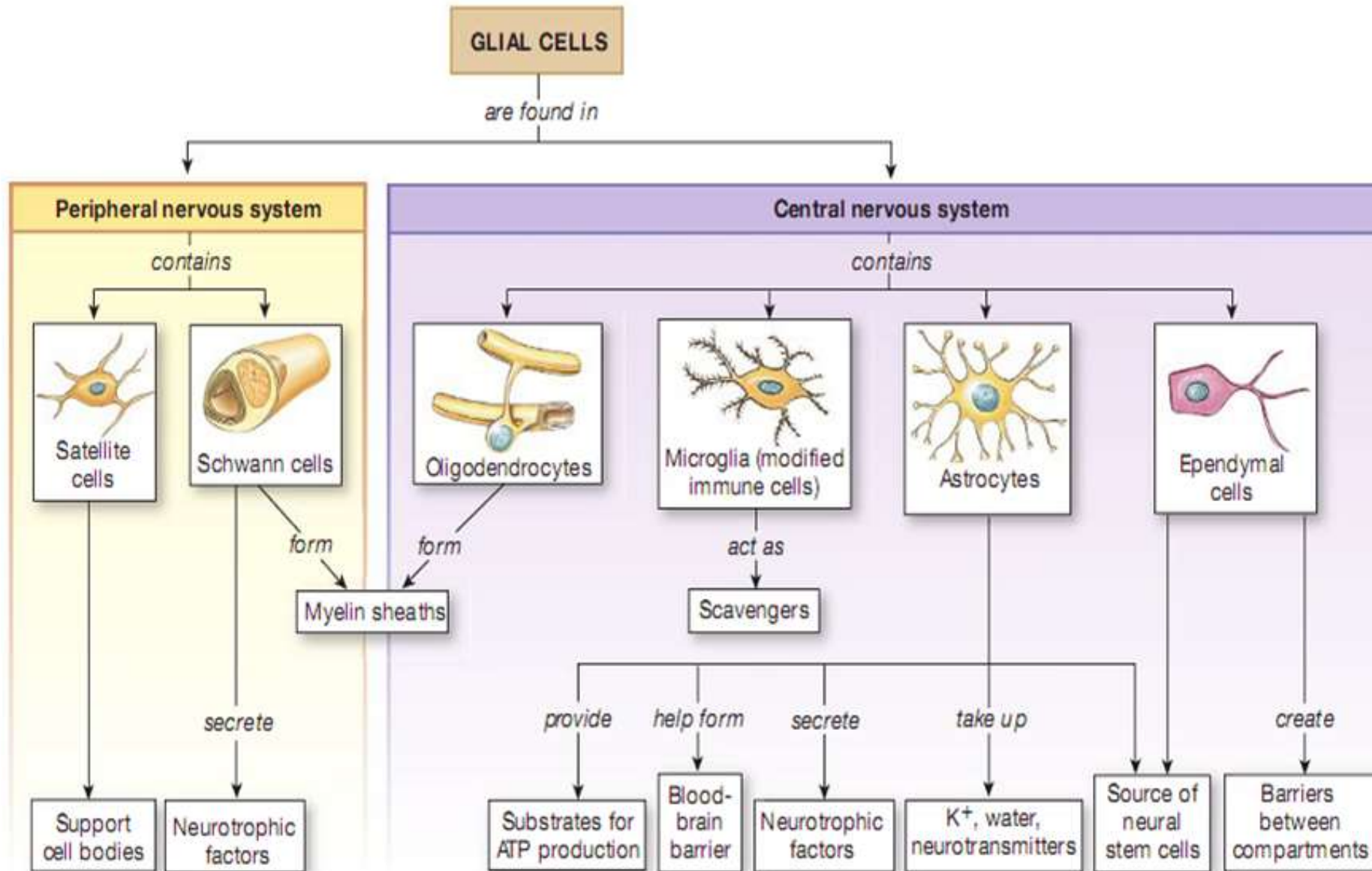
Tujuan Pembelajaran

Mahasiswa dapat menganalisis keterkaitan fisiologi otak dengan status mental emosional, proses biokimiawi otak terhadap gangguan jiwa.

(BPM MODUL PSIKIATRI, Sub-CPMK 4)

Neuron



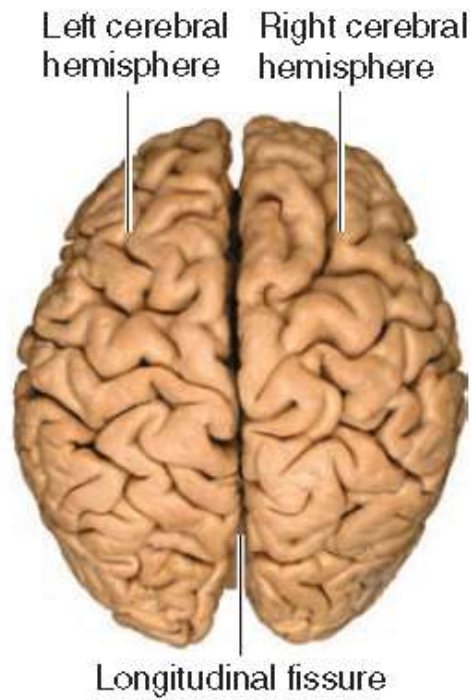


(b) Glial cells and their functions

● **FIGURE 8-5** *Glial cells provide physical and biochemical support for neurons.*



STRUKTUR OTAK SECARA ANATOMI



(a) Brain, superior (top) view

Figure 5-7 Brain of a human cadaver.

(a) Superior view looking down on the top of the brain. Note that the deep longitudinal fissure divides the cerebrum into the right and left cerebral hemispheres. (b) Sagittal view of the right half of the brain. All major brain regions are visible from this midline interior view. The corpus callosum serves as a neural bridge between the two cerebral hemispheres.

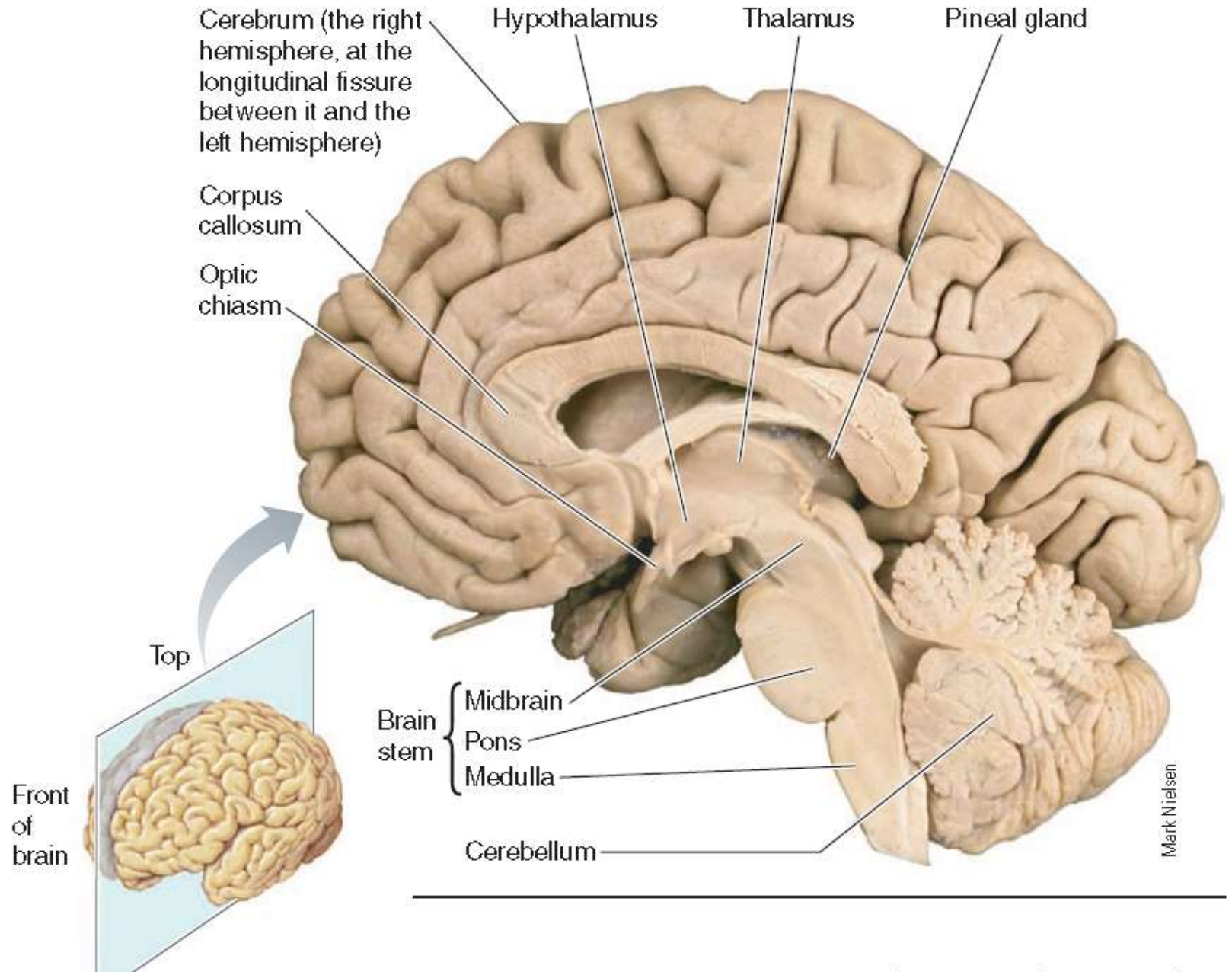
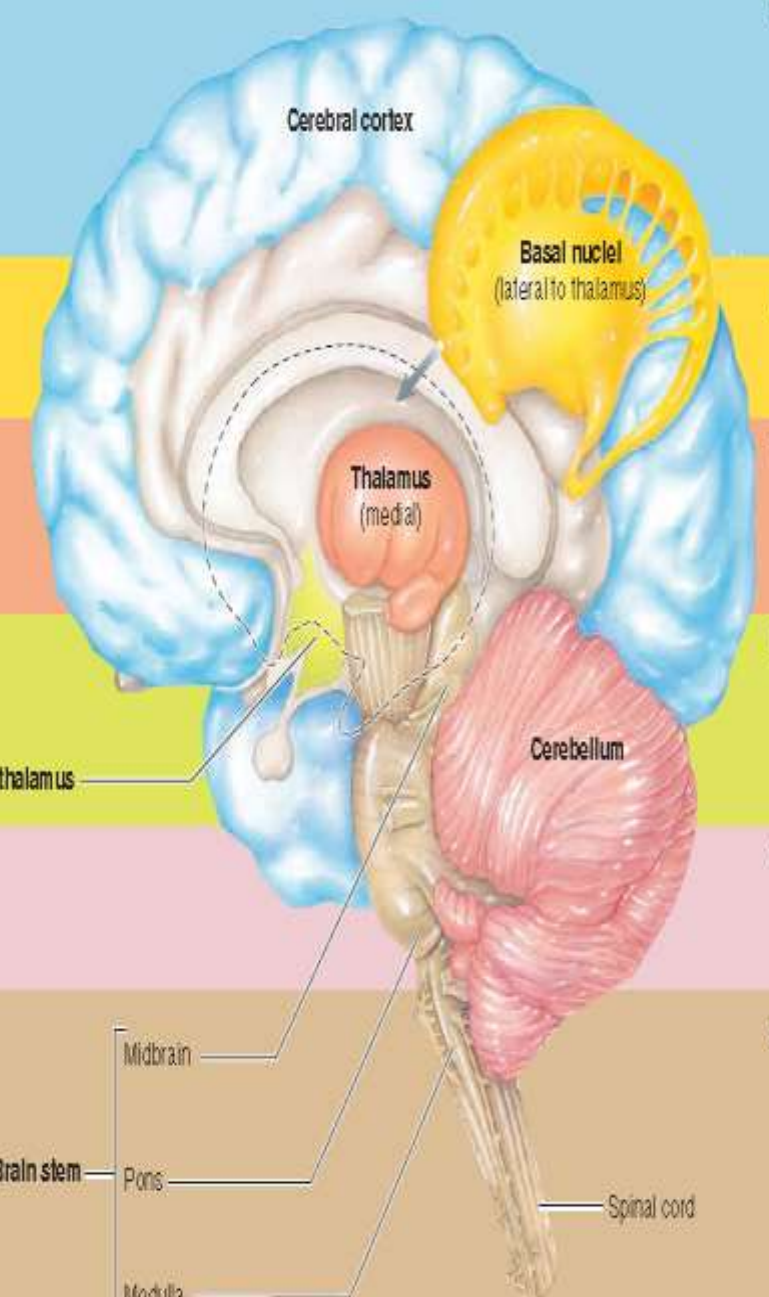
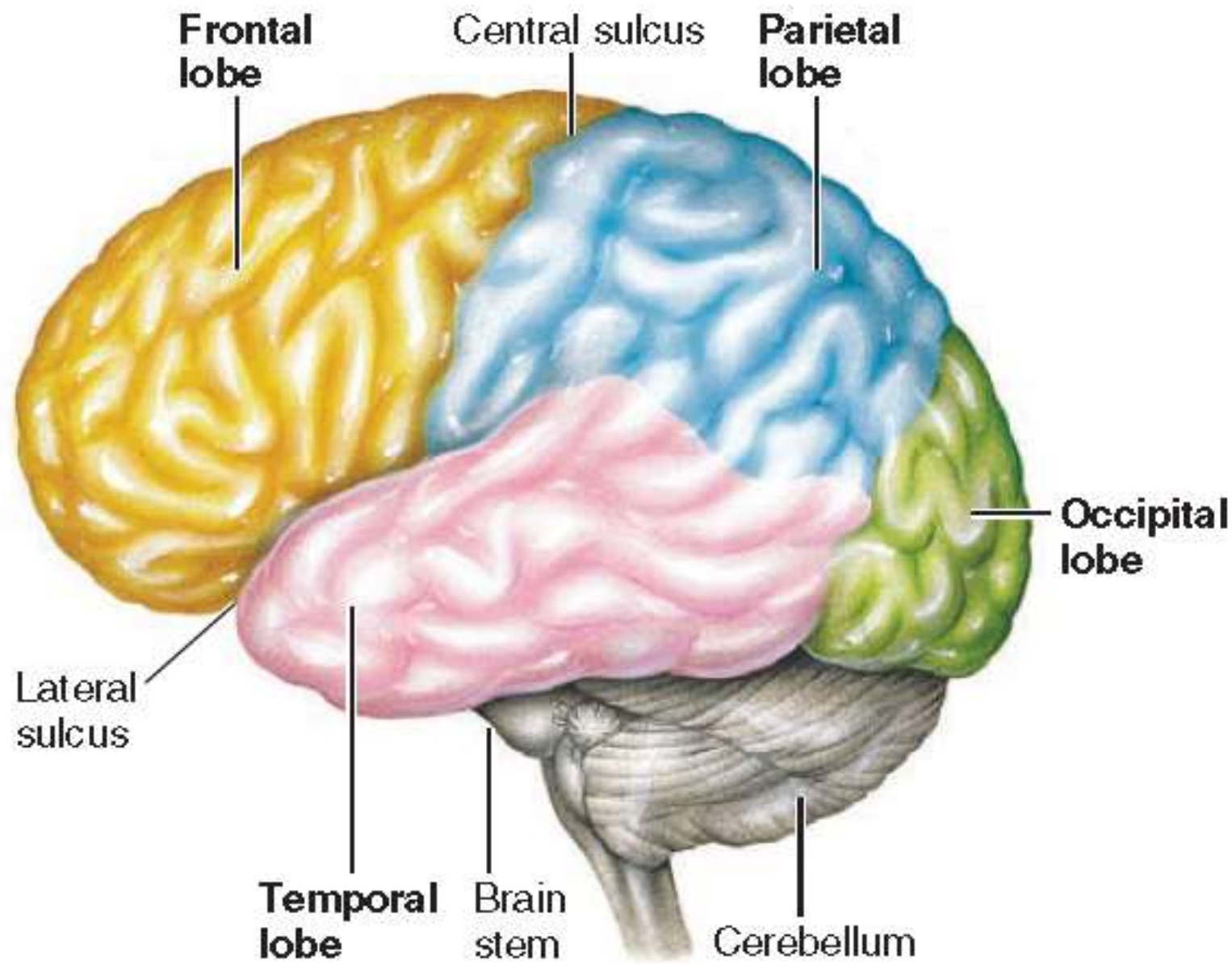


TABLE 5-1 Overview of Structures and Functions of the Major Components of the Brain

 Cerebral cortex Basal nuclei (lateral to thalamus) Thalamus (medial) Hypothalamus Cerebellum Brain stem Midbrain Pons Medulla Spinal cord	Cerebral cortex 1. Sensory perception 2. Voluntary control of movement 3. Language 4. Personality traits 5. Sophisticated mental events, such as thinking, memory, decision making, creativity, and self-consciousness
Basal nuclei	1. Inhibition of muscle tone 2. Coordination of slow, sustained movements 3. Suppression of useless patterns of movement
Thalamus	1. Relay station for all synaptic input 2. Crude awareness of sensation 3. Some degree of consciousness 4. Role in motor control
Hypothalamus	1. Regulation of many homeostatic functions, such as temperature control, thirst, urine output, and food intake 2. Important link between nervous and endocrine systems 3. Extensive involvement with emotion and basic behavioral patterns 4. Role in sleep–wake cycle
Cerebellum	1. Maintenance of balance 2. Enhancement of muscle tone 3. Coordination and planning of skilled voluntary muscle activity
Brain stem Midbrain Pons Medulla Spinal cord	1. Origin of majority of peripheral cranial nerves 2. Cardiovascular, respiratory, and digestive control centers 3. Regulation of muscle reflexes involved with equilibrium and posture 4. Reception and integration of all synaptic input from spinal cord; arousal and activation of cerebral cortex 5. Role in sleep–wake cycle



IFigure 5-10 Cortical lobes. Each half of the cerebral cortex is divided into the occipital, temporal, parietal, and frontal lobes, as depicted in this lateral view of the left side of the brain.

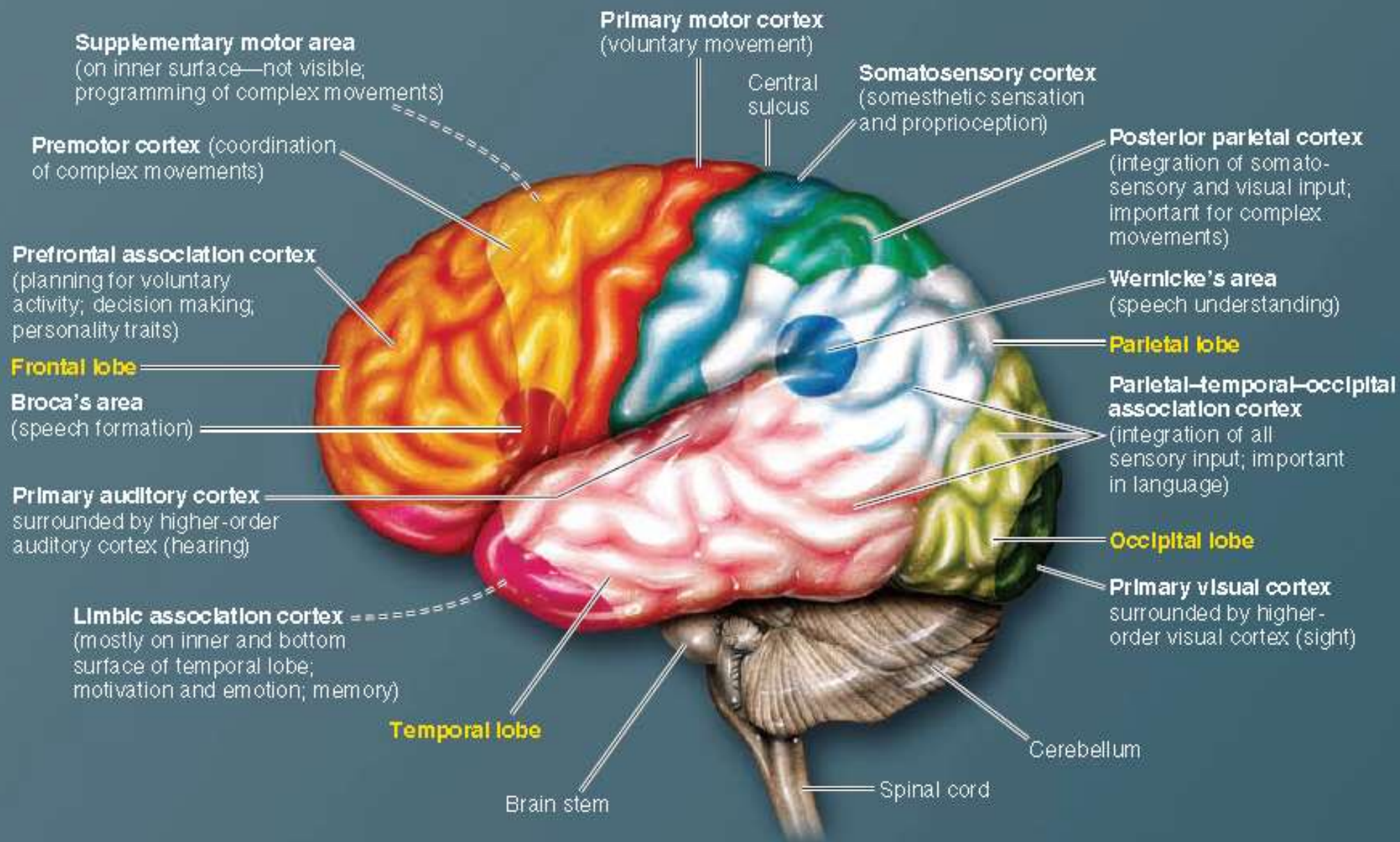


Figure 5-11 Functional areas of the cerebral cortex. Various regions of the cerebral cortex are primarily responsible for various aspects of neural processing, as indicated in this lateral view of the left side of the brain.

LIMBIC SYSTEM

HYPOTHALAMUS

regulates body temperature, circadian rhythms, and hunger; helps govern the endocrine system

PITUITARY GLAND

secretes many different hormones, some of which affect other glands

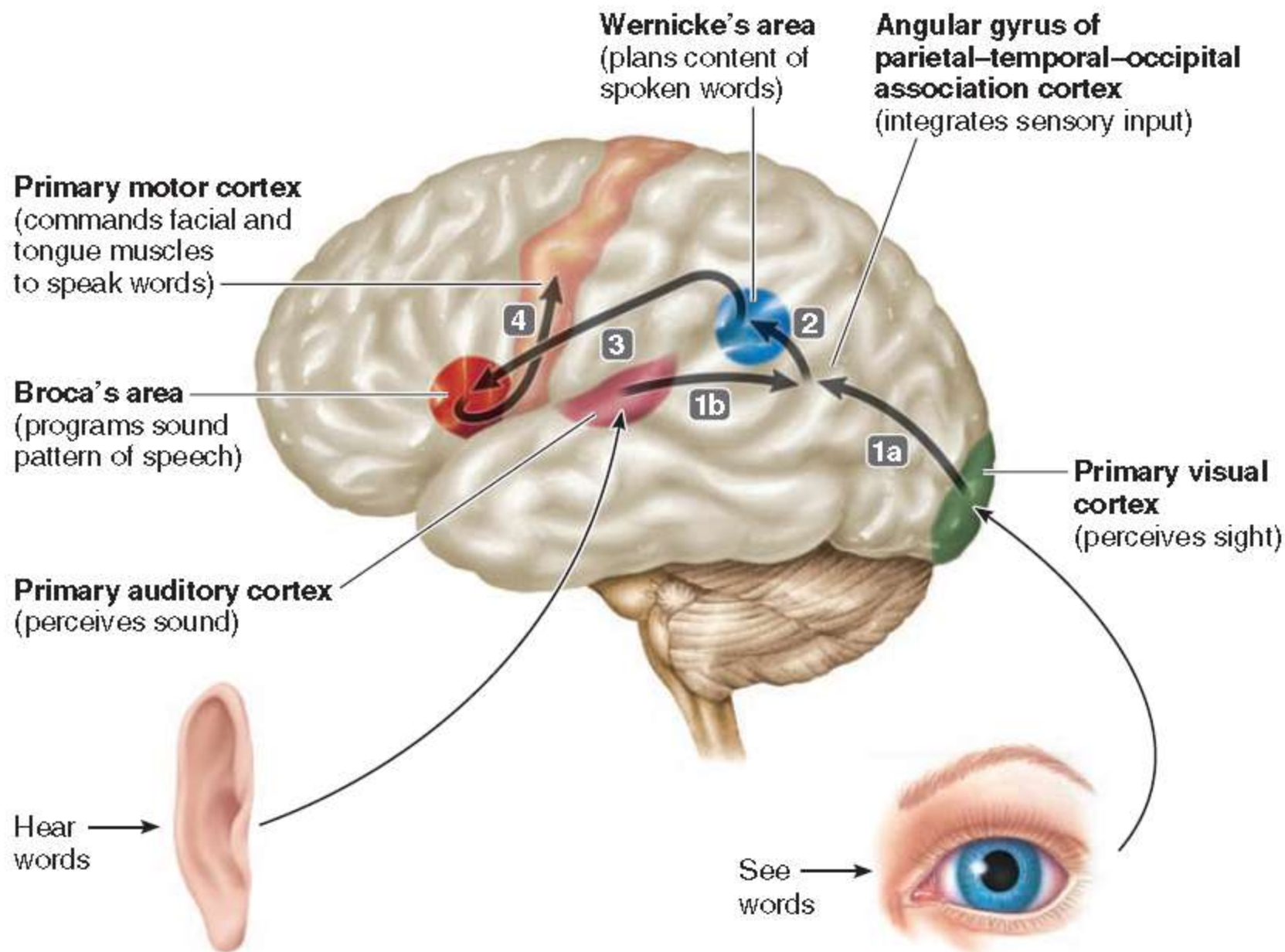
AMYGDALA

two lima-bean-sized clusters of neurons, involved in memory consolidation and emotion

HIPPOCAMPUS

central to learning and memory





1a To speak about something seen, the brain transfers the visual information from the primary visual cortex to the angular gyrus of the parietal-temporal-occipital association cortex, which integrates inputs such as sight, sound, and touch.

1b To speak about something heard, the brain transfers the auditory information from the primary auditory cortex to the angular gyrus.

2 The information is transferred to Wernicke's area, where the choice and sequence of words to be spoken are formulated.

3 This language command is then transmitted to Broca's area, which translates the message into a programmed sound pattern.

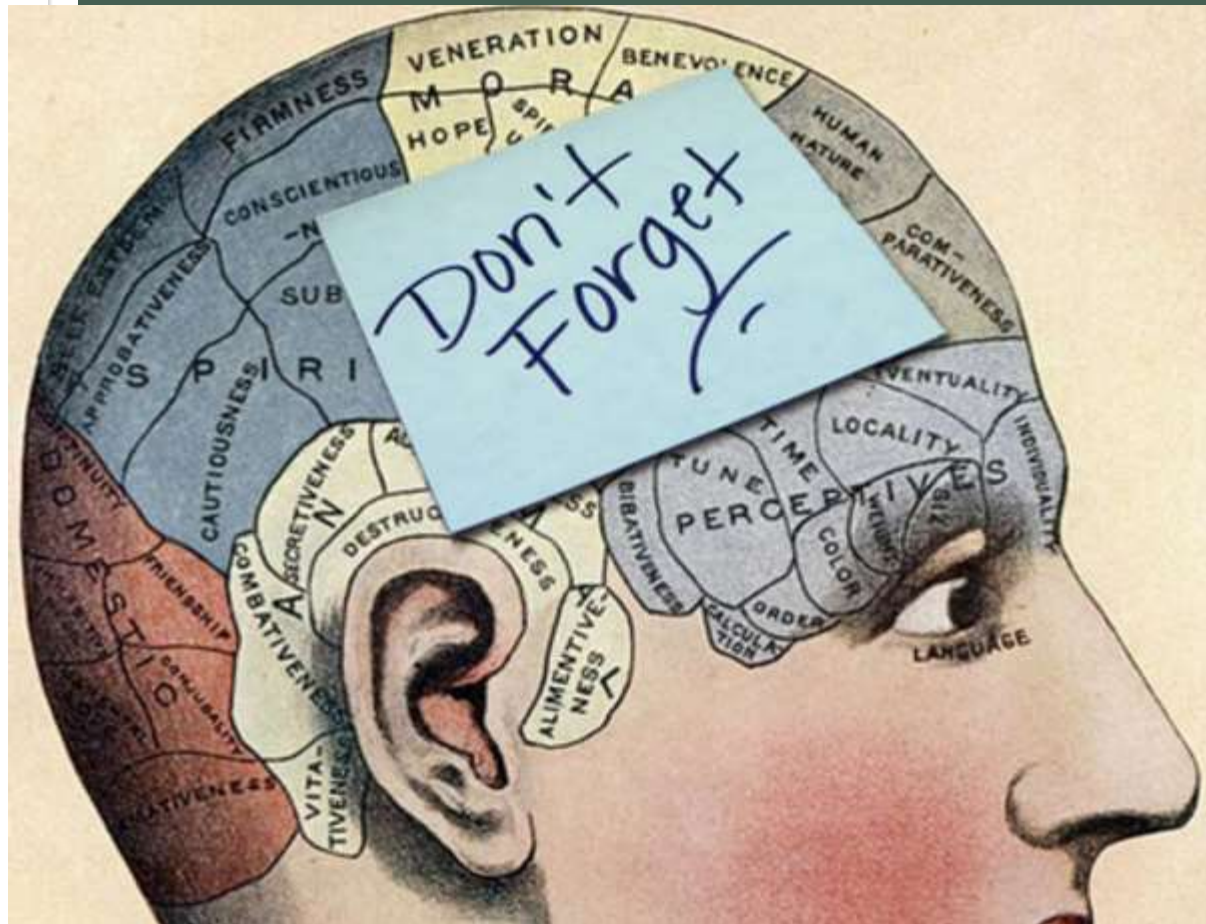
4 This sound program is conveyed to the precise areas of the primary motor cortex that activate the appropriate facial and tongue muscles for causing the desired words to be spoken.

Figure 5-13 Cortical pathway for speaking a word seen or heard. The arrows and numbered steps describe the pathway used to speak about something seen or heard. Similarly, appropriate muscles of the hand can be commanded to write the desired words.



DEFINISI MEMORI & PEMBELAJARAN

DEFINISI Memori



Kandell, 2015

Proses saat pengetahuan mengenai dunia dikode, disimpan dan diambil kembali

Bear, 2015

Retensi atau pengulangan dari informasi yang telah dipelajari

Ganong, 2015

Retensi dan penyimpanan dari informasi yang diperoleh

Penyimpanan pengetahuan → jejak memori

DEFINISI PEMBELAJARAN



Bear, 2015

Akuisisi dari pengetahuan atau keterampilan yang baru

Sherwood, 2013

Akuisisi pengetahuan atau keterampilan sebagai konsekuensi pengalaman, instruksi, atau keduanya

Belajar : Stimulus → Respon

Stimulus hadiah → Merespon dengan cara yang sama terhadap stimulus yang sama

Stimulus hukuman → Kemungkinan mengulang respon yang sama pada stimulus yang sama sangat kecil



Belajar : Perubahan perilaku yang terjadi akibat pengalaman

Belajar & Memori



Dasar untuk
mengadaptasikan perilaku
ke lingkungan tertentu

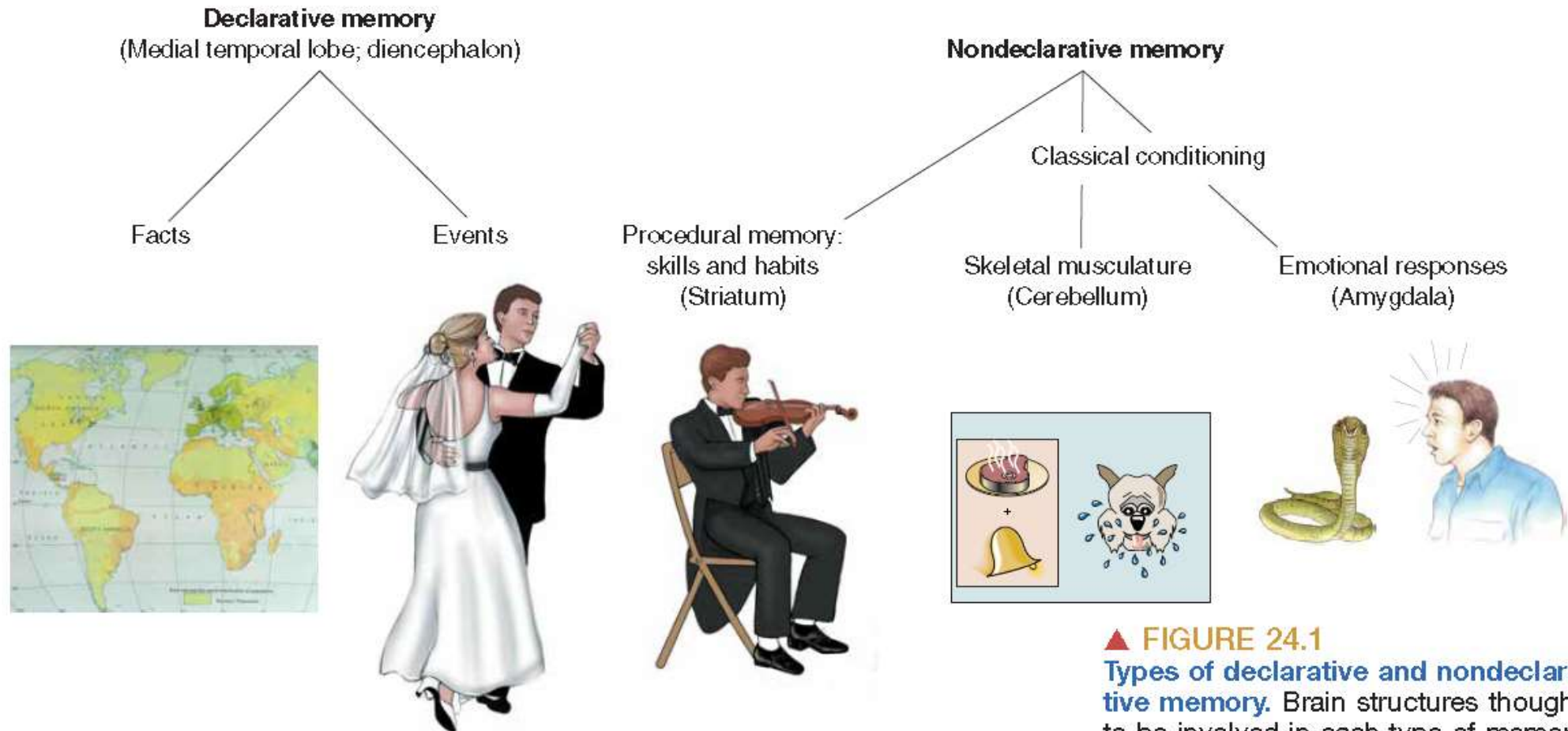


Survival



JENIS MEMORI DAN PEMBELAJARAN

Jenis memori



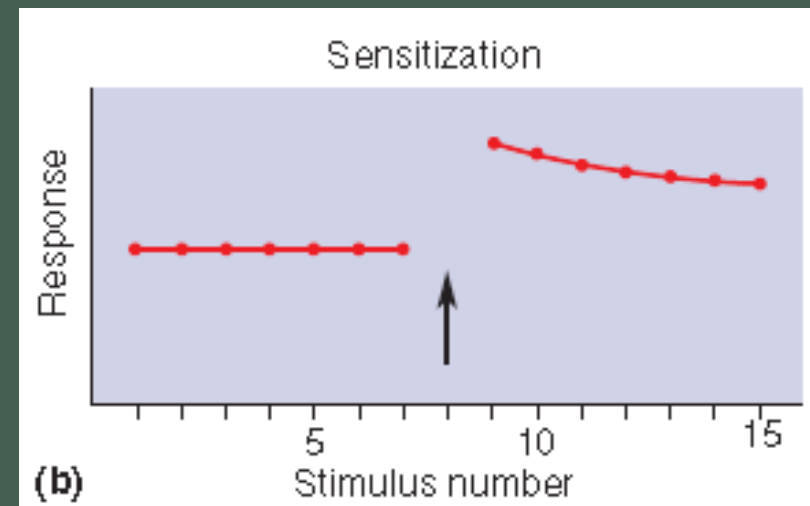
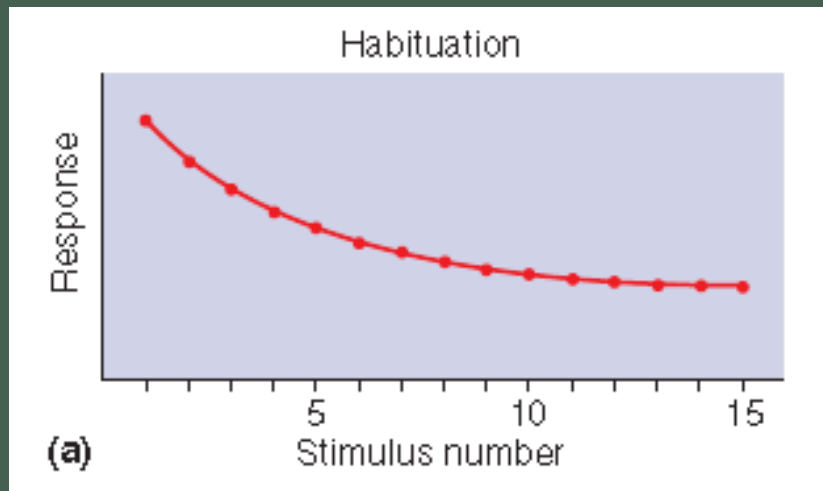
▲ **FIGURE 24.1**
Types of declarative and nondeclarative memory. Brain structures thought to be involved in each type of memory are indicated. (Note that this is not a complete representation of all types of memory.)

Types of procedural memories

- Involves learning a motor response (procedure) in reaction to a sensory input.
- Formation of procedural memories occurs through 2 categories of learning :
 1. Non associative learning
 2. Associative learning

Nonassociative learning

- Describe a change in behavioral response that occurs over time in response to a single type of stimulus.
- Two types :
 1. Habituation
 2. sensitization



Associative learning

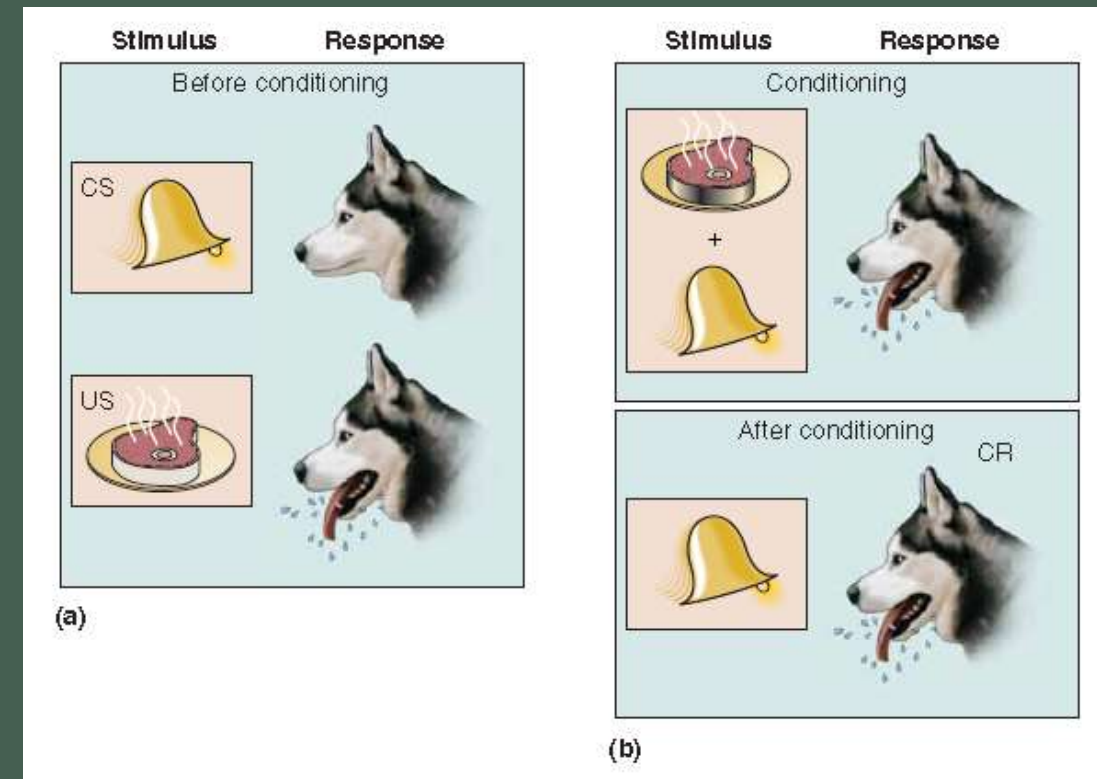
- Behavior is altered by the formation of association between events
- Two types :
 - Classical conditioning
 - Instrumental conditioning

Classical conditioning (pavlov)

Involves associating stimulus that evokes a measurable response with a second stimulus that normally doesn't evoke this response.

Unconditional stimulus → normally evoke the response
→ in pavlov experiments : sign of a piece of meat → salivation

Conditional stimulus → doesn't evoke the response, require training
→ in pavlov experiments : auditory stimulus



Instrumental conditioning

- Individual learn to associate a response, a motor act, with a meaningful stimulus, typically a rewards such as food
- Example :
Hungry rats placed in a box with a lever that dispenses food → when exploring the box, the rats bump the lever and pop out a piece of food → after happen few times, rat learns that pressing the lever lead to a food rewards
- Subject learns that a particular behavior is associate with a particular consequences.
- Motivation plays a large part in instrumental conditioning → neural circuits are more complex than those involved in classical condition



CRAIG SWANSON © WWW.PERSPICUITY.COM

▲ **TABLE 5-3**

Comparison of Short-Term and Long-Term Memory

Characteristic	Short-Term Memory	Long-Term Memory
Time of Storage after Acquisition of New Information	Immediate	Later; must be transferred from short-term to long-term memory through consolidation; enhanced by practice or recycling of information through short-term mode
Duration	Lasts for seconds to hours	Retained for days to years
Capacity of Storage	Limited	Very large
Retrieval Time (remembering)	Rapid retrieval	Slower retrieval, except for thoroughly ingrained memories, which are rapidly retrieved
Inability to Retrieve (forgetting)	Permanently forgotten; memory fades quickly unless consolidated into long-term memory	Usually only transiently unable to access; relatively stable memory trace
Mechanism of Storage	Involves transient modifications in functions of preexisting synapses, such as altering amount of neurotransmitter released	Involves relatively permanent functional or structural changes between existing neurons, such as formation of new synapses; synthesis of new proteins plays a key role

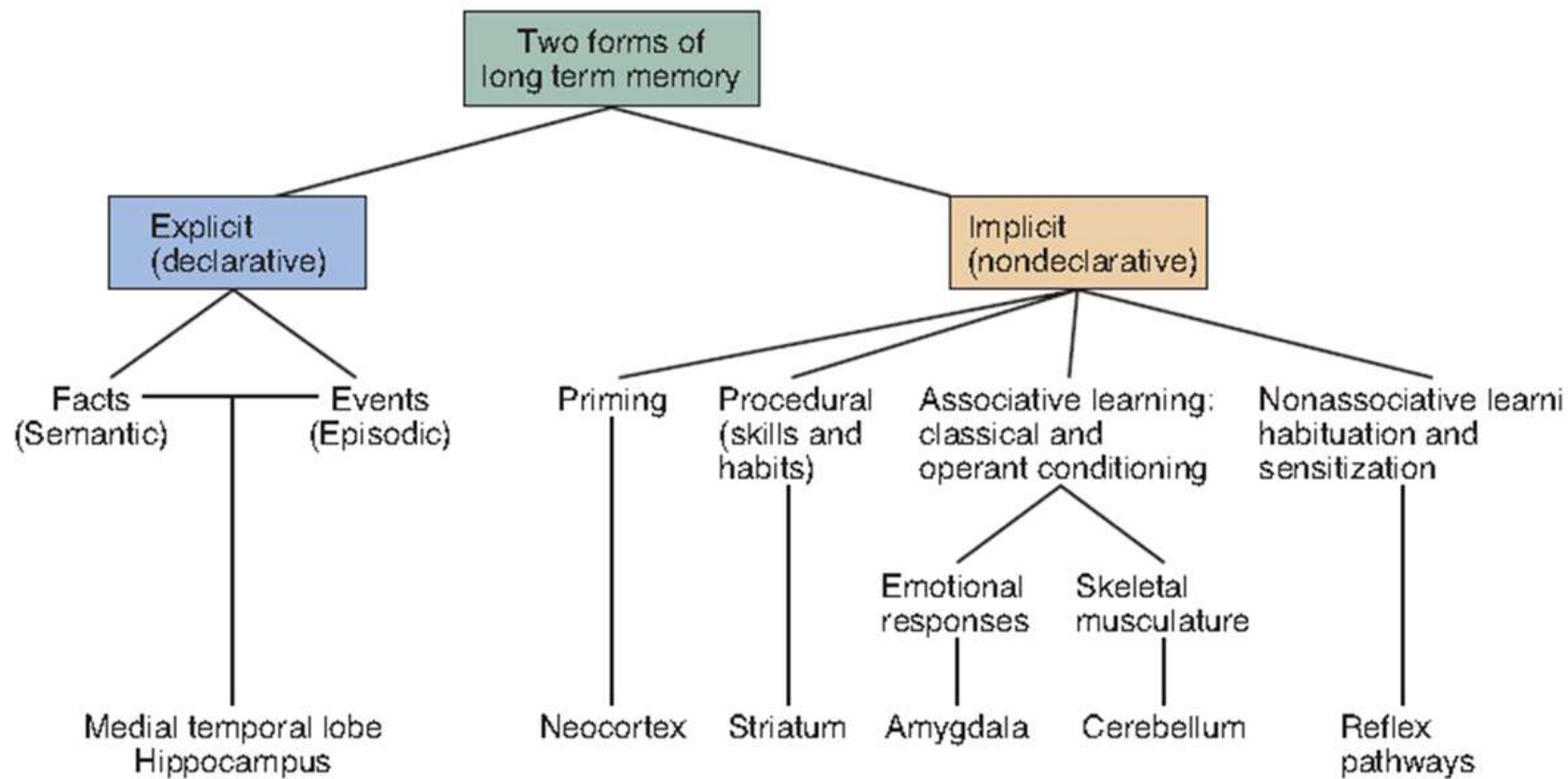
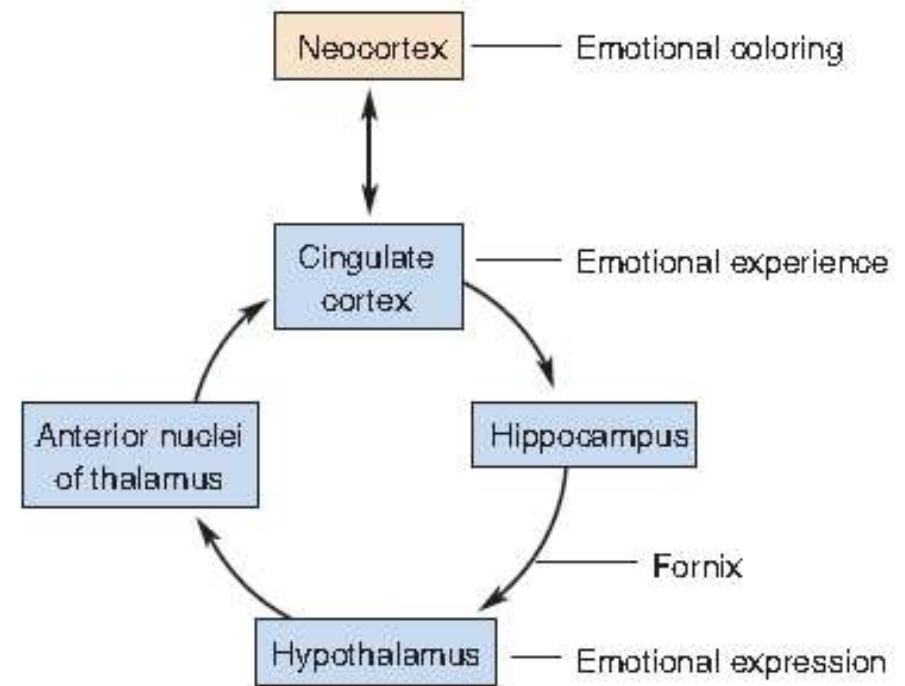
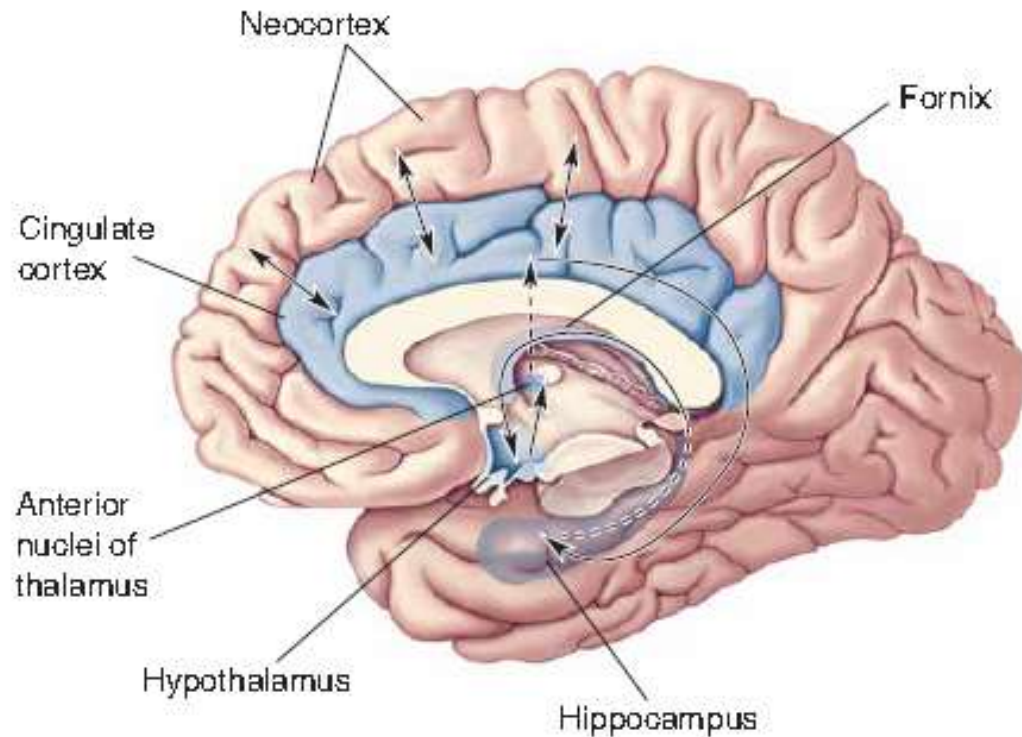


FIGURE 19-2 Forms of long-term memory. (Modified from Kandel ER, Schwartz JH, Jessell TM [editors]: *Principles of Neural Science*, 4th ed. McGraw-Hill, 2000.)

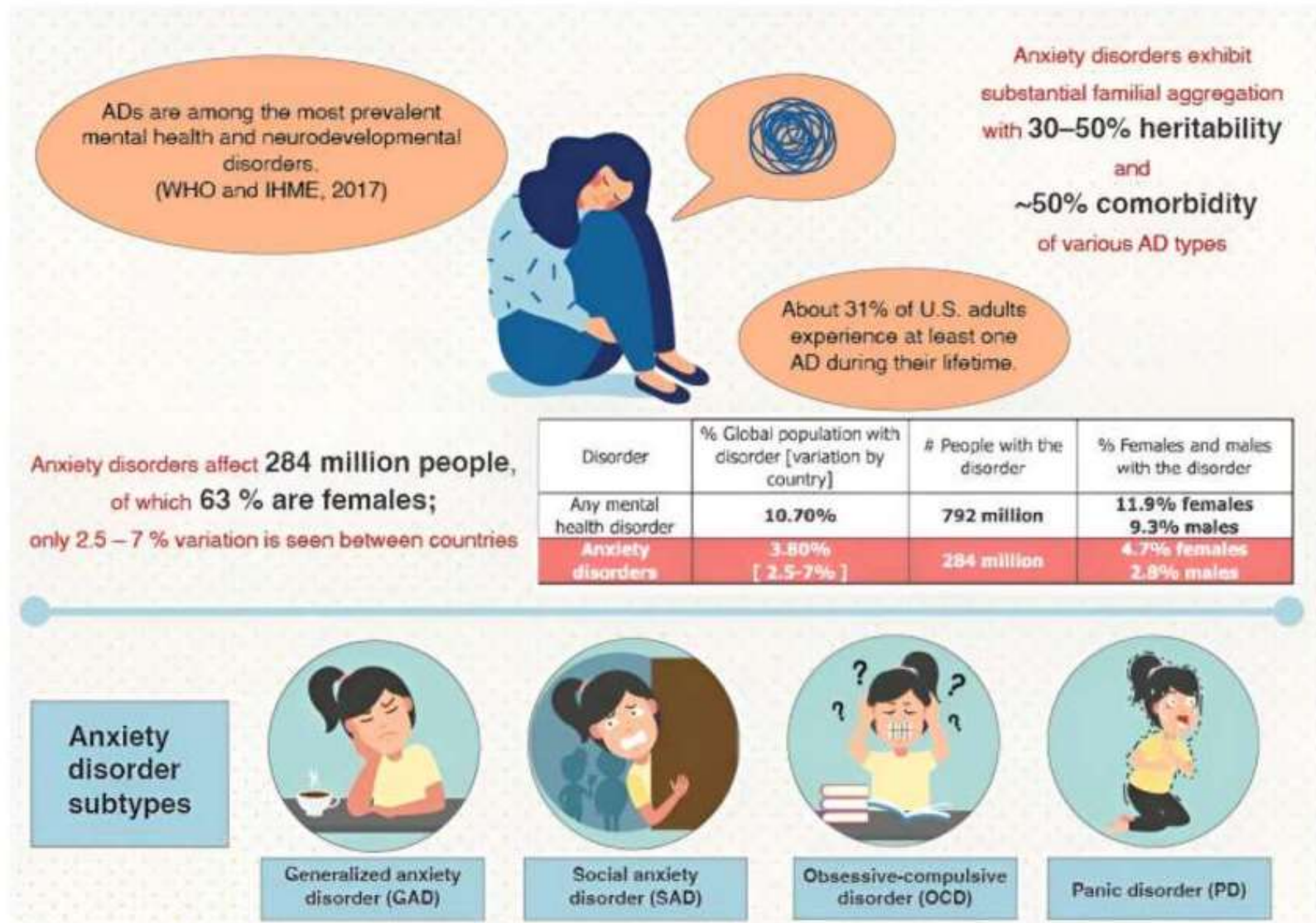
PAPEZ CIRCUIT



The background of the slide is a repeating pattern of stylized green leaves and branches on a light green background. A dark green rectangular box is centered on the slide, containing the text "MENTAL ILLNESS". Above the box, there is a small green rectangular tab.

MENTAL ILLNESS

ANXIETY DISORDERS



- Kalyani B. Karunakaran et al, Spatiotemporal expression patterns of anxiety disorder-associated genes, *Translational Psychiatry* (2023). DOI: [10.1038/s41398-023-02693-y](https://doi.org/10.1038/s41398-023-02693-y)

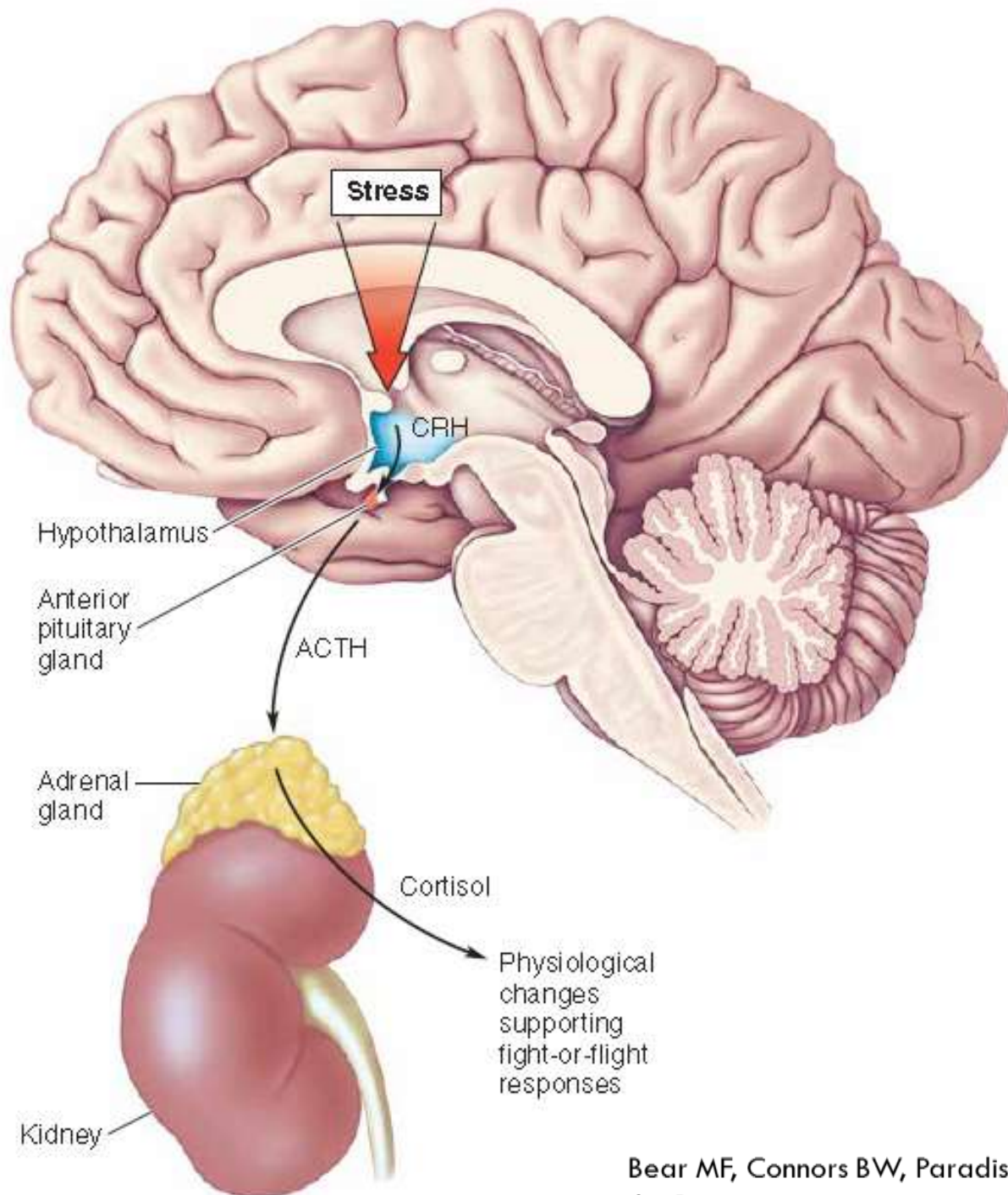
Biological bases of anxiety disorders

- Genetic predisposition
- the occurrence of stressful life events



Anxiety disorders are caused by a complex interplay of biological and environmental factors, including:

- Brain chemistry:** Anomalies in brain chemistry, such as differences in neurotransmitters like serotonin and dopamine
- Brain structure:** Structural differences in areas like the amygdala, hippocampus, and prefrontal cortex
- Genes:** Specific genes associated with anxiety, such as estrogen receptor-alpha and opiate-related nociceptin receptor 1 (OPRL1)
- Stress:** High levels of stress can cause blunted reactions in certain brain regions
- Personality and development:** Differences in the way threats are perceived, and personality and development
- Heredity:** Anxiety is more likely to be hereditary if it develops in younger people

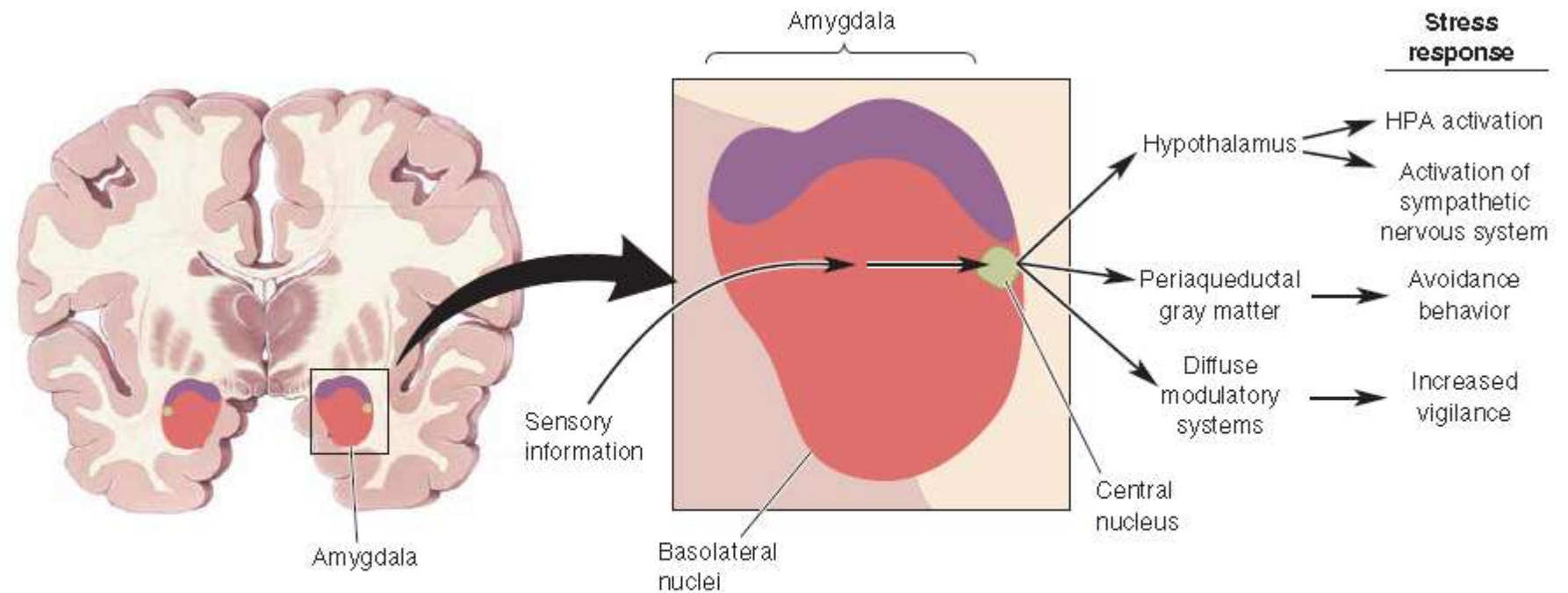


The stress reponses

The *stress response* is the coordinated reaction to threatening stimuli. It is characterized by the following:

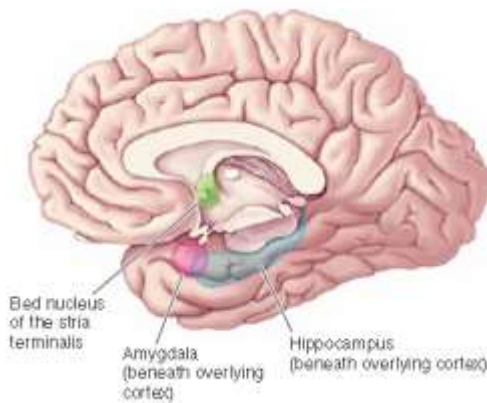
- Avoidance behavior
- Increased vigilance and arousal
- Activation of the sympathetic division of the ANS
- Release of cortisol from the adrenal glands

Regulation of HPA axis by Amygdala and Hippocampus



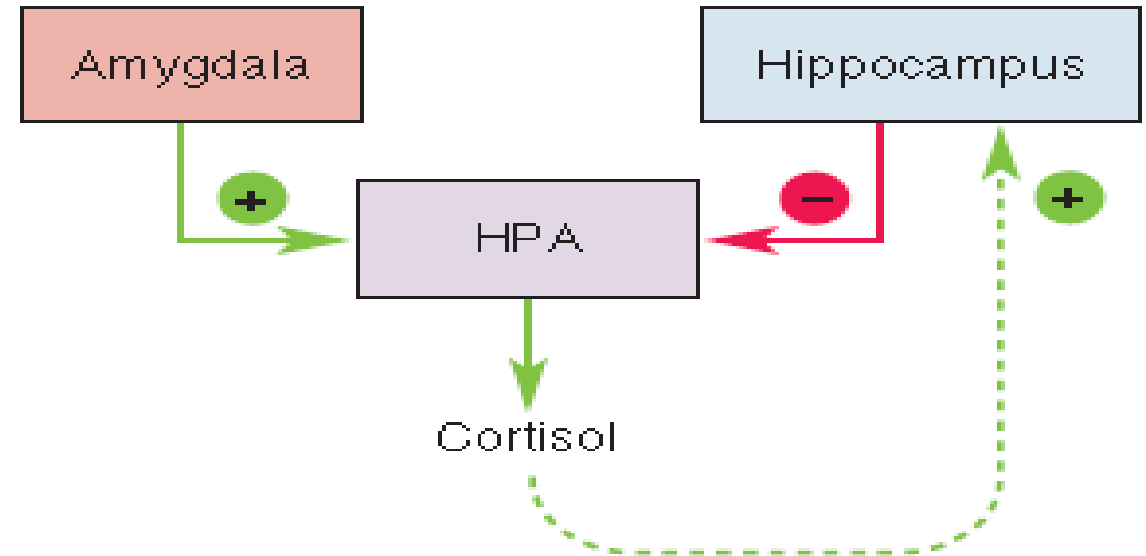
▲ **FIGURE 22.5**

Control of the stress response by the amygdala. The amygdala receives ascending sensory information from the thalamus as well as descending inputs from the neocortex. This information is integrated by the basolateral nuclei and is relayed to the central nucleus. Activation of the central nucleus leads to the stress response.



Regulation of HPA axis by Amygdala and Hippocampus

Bear MF, Connors BW, Paradiso, MA. Neurotransmitter system. 4th ed. Neuroscience: Exploring the Brain.



▲ **FIGURE 22.6**

Push–pull regulation of the HPA axis by the amygdala and hippocampus.

Amygdala activation stimulates the HPA system and the stress response (green lines). Hippocampal activation, on the other hand, suppresses the HPA system (red line). Because the hippocampus has glucocorticoid receptors that are sensitive to circulating cortisol, it is important in the feedback regulation of the HPA axis in preventing excessive cortisol release.



BIOLOGICAL BASES OF AFFECTIVE DISORDERS

1. MONOAMINE HYPOTHESIS OF MOOD DISORDERS
2. THE DIATHESIS-STRESS HYPOTHESIS
3. ANTERIOR CINGULATE CORTEX DYSFUNCTION

Biological bases of affective disorders

- *Affect* is the medical term for emotional state or mood; **affective disorders** are disorders of mood. In a given year, over 9% of the population will suffer from one of the mood disorders.
- **Major Depression.** The mental illness known as **major depression** is the most common mood disorder, affecting 6% of the population every year. The cardinal symptoms are lowered mood and decreased interest or pleasure in all activities. For a diagnosis of major depression, these symptoms must be present every day for a period of at least 2 weeks and not be obviously related to bereavement. Other symptoms also occur, including:
 - • Loss of appetite (or increased appetite)
 - • Insomnia (or hypersomnia)
 - • Fatigue
 - • Feelings of worthlessness and guilt
 - • A diminished ability to concentrate
 - • Recurrent thoughts of death

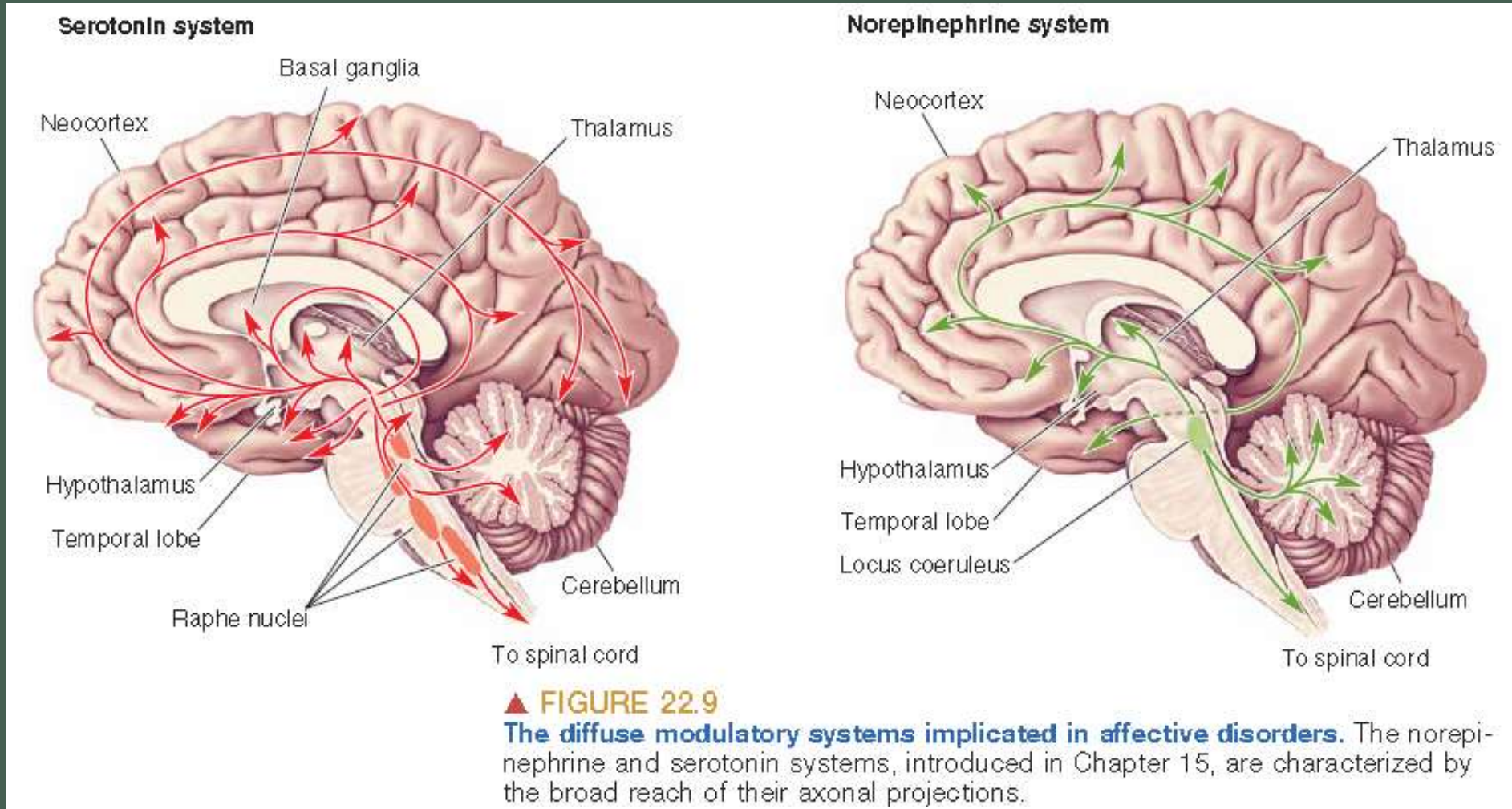
Biological bases of affective disorders

- **Bipolar Disorder.** Like major depression, **bipolar disorder** is a recurrent mood disorder. It consists of repeated episodes of mania, or mixed episodes of mania and depression, and therefore is also called *manic depressive disorder*. **Mania** (derived from a French word meaning “crazed” or “frenzied”) is a distinct period of abnormally and persistently elevated, expansive, or irritable mood.

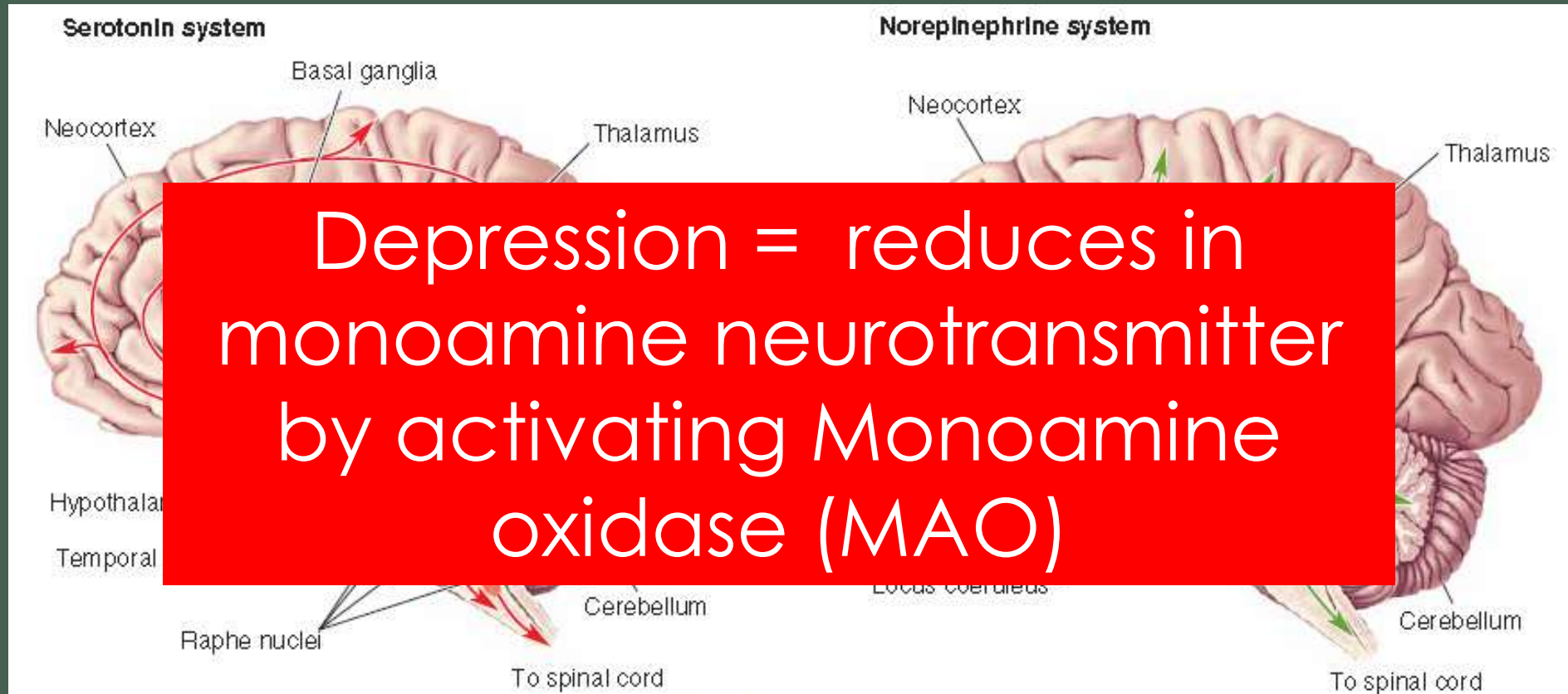
During the manic phase, other common symptoms include:

- • Inflated self-esteem or grandiosity
- • A decreased need for sleep
- • Increased talkativeness or feelings of pressure to keep talking
- • Flight of ideas, or a subjective experience that thoughts are racing
- • Distractibility
- • Increased goal-directed activity

Monoamine hypothesis of affective disorders



Monoamine hypothesis of affective disorders



Depression = reduces in monoamine neurotransmitter by activating Monoamine oxidase (MAO)

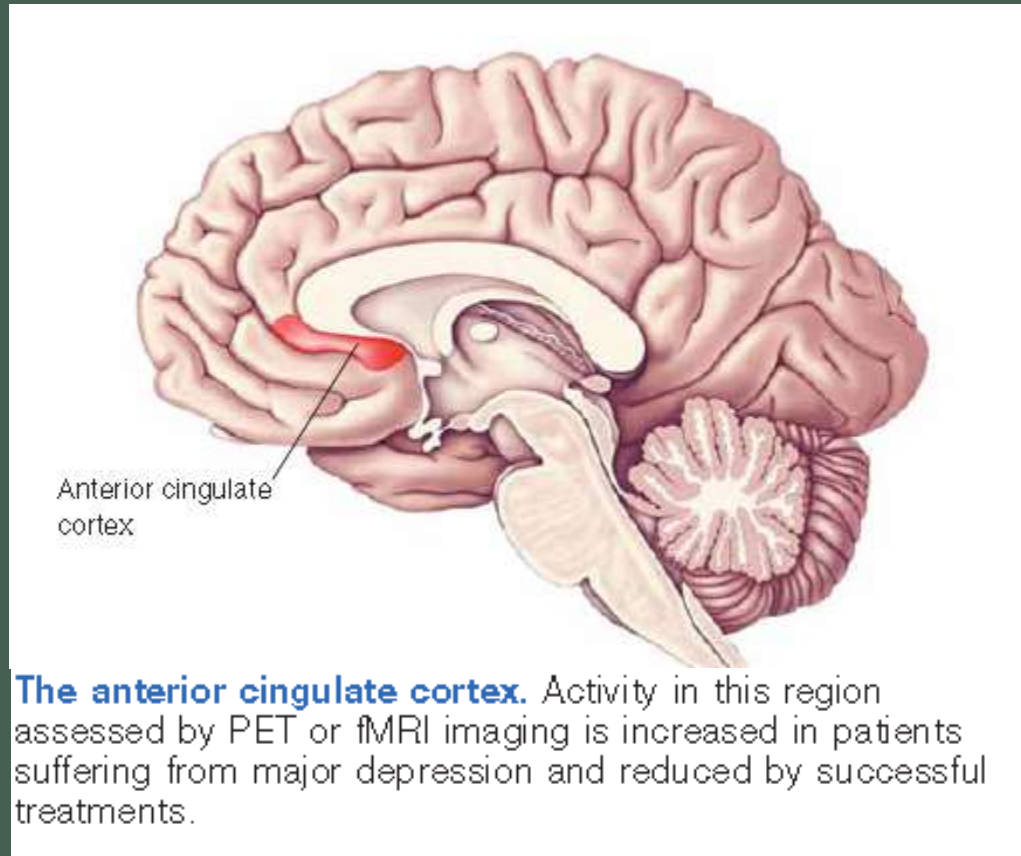
▲ **FIGURE 22.9**

The diffuse modulatory systems implicated in affective disorders. The norepinephrine and serotonin systems, introduced in Chapter 15, are characterized by

The Diathesis-Stress Hypothesis

- Evidence clearly indicates that mood disorders run in families and that our genes predispose us to this type of mental illness. The medical term for a predisposition for a certain disease is *diathesis*. However, researchers have also established that early childhood abuse or neglect and other stresses of life are important risk factors in the development of mood disorders in adults.
- According to the **diathesis-stress hypothesis of affective disorders**, the HPA axis is the main site where genetic and environmental influences converge to cause mood disorders.

ANTERIOR CINGULATE CORTEX DYSFUNCTION



This region of the brain is considered to be a “node” in an extensive network of interconnected structures that include other regions of the frontal cortex, hippocampus, amygdala, hypothalamus, and brain stem.

The Diathesis–Stress Model

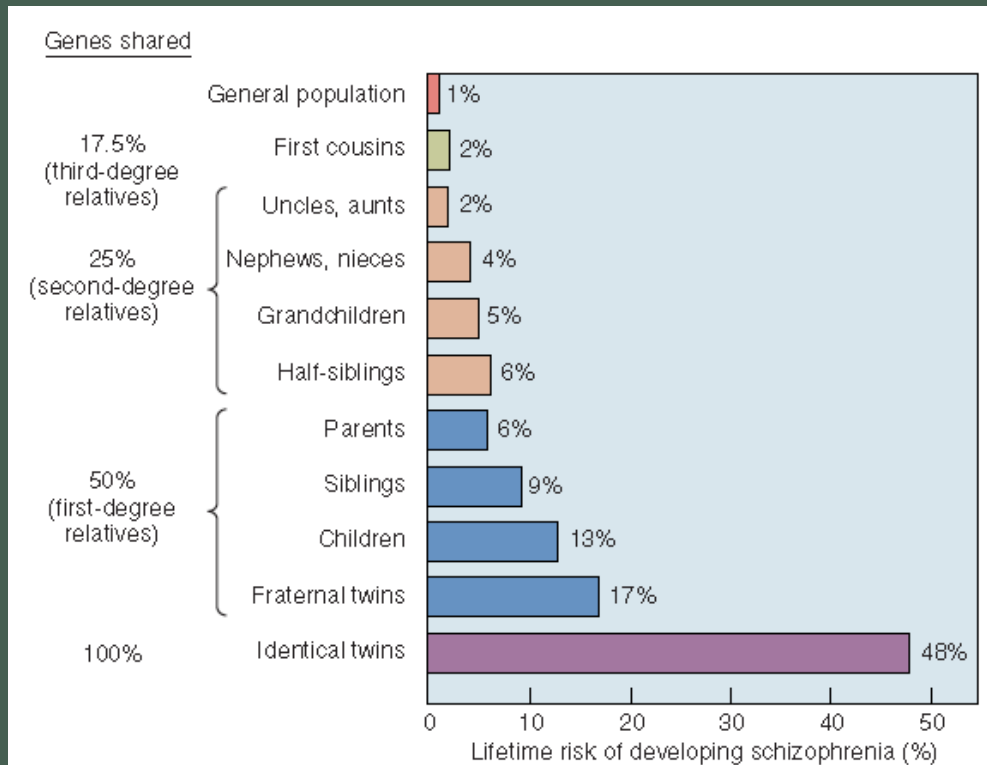


FIGURE 2.9 The diathesis–stress model.

SCHIZOPHRENIA

- characterized by a loss of contact with reality and a disruption of thought, perception, mood, and movement. The disorder typically becomes apparent during adolescence or early adulthood and usually persists for life.
- *Positive symptoms* reflect the presence of abnormal thoughts and behaviors, such as:
 - • Delusions
 - • Hallucinations
 - • Disorganized speech
 - • Grossly disorganized or catatonic behavior
- *Negative symptoms* reflect the absence of responses that are normally present. These symptoms include:
 - • Reduced expression of emotion
 - • Poverty of speech
 - • Difficulty in initiating goal-directed behavior
 - • Memory impairment

BIOLOGICAL BASES OF SCHIZOPHRENIA



▲ **FIGURE 22.13**

The familial nature of schizophrenia. The risk of developing schizophrenia increases with the number of shared genes, suggesting a genetic basis for the disease. (Source: Adapted from Gottesman, 1991, p. 96.)

GENES AND ENVIRONMENT

THANK YOU