

Central Nervous System

The Receives and processes sensory

Brain information, initiates responses,
stores memories, and generates
thoughts

Spinal Conducts signals to and from the Cord brain, controls reflex activities.

The Brain Structure

Cerebrum The largest part of the brain, made up of the *cerebral cortex* and other structures.

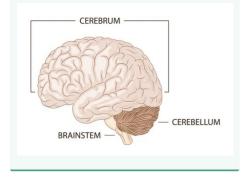
The cerebrum is divided into *two hemispheres*.

Cerebellum Coordinates movement by combining information from the eyes, ears, and muscles.

Brainstem Connects the *cerebrum* and *cerebellum* to the *spinal cord*.

The brainstem controls unconscious processes like

The Brain Structure Visual



Divisions of the Brain

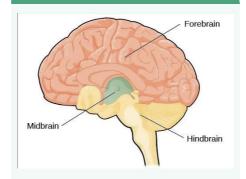
Forebrain Processes sensory information, helps with reasoning and problem-solving, and regulate autonomic, endocrine, and motor functions

Midbrain Helps to regulate movement and process auditory and

Hindbrain Helps regulate automatic functions, relay sensory information, and maintain balance and equilibrium

visual information

Divisions of the Brain Visual



The Limbic System

Limbic (Primitive brain), regulates

System emotions (basic survival instincts), influences memories/learning, and motivation (basic drives).

Thalamus Sensory relay center, receives

Sensory relay center, receives input from all our senses except olfaction, critical in the perception of pain

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Last updated 9th March, 2025.

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The Limbic System (cont)

Hippoc- Stores memories, consolampus idation of conscious memories,
stores new information and
events as lasting memories

Amygdala Processing fearful and threatening stimuli. Includes threat detection and activation of fear-related behaviors.

Hypoth- Maintains the body's homeosalamus tasis, (regulates temperature, hunger, thirst, sleep-wake cycle). Produces hormones that stimulate or inhibit the release of hormones from the pituitary gland.

Corpus Connects the brain's left and
Callosum right hemispheres, allowing
them to communicate

Basal Motor learning, executive

Ganglia functions and coordination of
movement, posture, inhibitory

(allows us to be still)

Olfactory is a part of the brain that Bulb processes smell



The Limbic System (cont)

Cingulate Involved in regulating

gyrus emotions, processing pain, and
regulating autonomic motor

function.

Pineal gland Helps control the circadian cycle of sleep and wakefulness

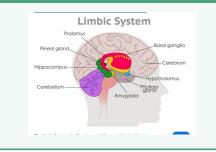
by secreting melatonin.

Suprachiasmatic Controls the body's circadian

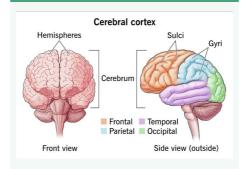
smatic rhythms

Nucleus (SCN)

The Limbic System Visual



The Cerebral Cortex Visual



The Cerebral Cortex

Cerebral Cortex Involved in many high-

level functions, such as reasoning, emotion, thought, memory, language and conscious-

ness.

Frontal Lobe: the largest portion of the

brain (about ½ of the entire brain) divided into *prefrontal cortex*,

premotor area, and motor area

Parietal Lobe: Primary sensory areas

that process somatosensory information, sensations of touch, pain, heat, and propri-

oception.

Temporal Lobe: Auditory processing,

memory information retrieval, and involved in emotional behavior. Connected to limbic system (hippocampus, amygdala, etc).

Occipital Lobe: Visual perd

Visual perception, visual interpretation, and

reading

Frontal Lobe

Left language, speech, and Frontal cognitive tasks. Includes

Lobe: broca's area

Right non-verbal communication
Frontal (facial recognition) and enviro-

Lobe: nmental awareness

Prefrontal Cortex

(PFC)

Integration center for all sensory information and executive functions (decision making, planning, working memory, personality expression, social behavior, speech

center

Broca's area

Controls the muscles that produce speech and language

and language). Personality

comprehension

Parietal Lobe

Left Directing attention, visual &

Parietal spatial skills

Lobe:

Right Motor routines and linguistic

Parietal skills (reading, writing)

Lobe:

Temporal Lobe

Left Verbal memory and language
Temporal comprehension. Includes

Lobe: wernicke's area.

Right Visual memory

Temporal Lobe:

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Last updated 9th March, 2025.
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Temporal Lobe (cont)

Wernicke's Area Language comprehension = receives auditory signals from the ear and processes them to understand the meaning of spoken words

?

How does the SCN interact with the Pineal Gland?

The SCN sends messages to the pineal gland, which triggers the release of melatonin at night and triggers the release of cortisol and other hormones to help you wake up in the morning.

Peripheral Nervous System (PNS)

Somatic	Sends and receives sensory
Nervous	messages that control
System:	voluntary motor movement of the skeletal muscles
Automatic	Controls automatic or involu-
Nervous	ntary bodily functions of the
System:	smooth muscles and glands

Automatic Nervous System (ANS)

Primary Function:	Maintain homeostasis (e.g., digestion, heart rate, & breathing)
Sympat-	The Body's mobilizing system. Prepares the body for stressful
Nervous	or energetic activity (e.g., fight
System (SNS)	or flight)

Automatic Nervous System (ANS) (cont)

Parasympathetic Dominates during rest
Nervous System state, directs mainte(PNS) nance activities (e.g.,
rest and rumination)

Sympathetic & Parasympathetic Effects



Myelination of the Vagus Nerve

Vagus	A major nerve in the parasy-
Nerve	mpathetic nervous system
	that helps regulate heart rate,
	digestion, and emotional
	responses.
Myelination	The process of covering
	nerve fibers with myelin,
	which improves the speed

and efficiency of nerve

Social Connection & Nervous System Development:

signaling

Safe Connection with Another Mammalian Nervous System:

This suggests that **social interactions**, particularly safe and supportive relationships, help stimulate and develop the vagus nerve.

Neuro Disorders

Brain Locus	of Neurologica	al Disorders
Туре	Disorders/ Description	Locus of the Brain
Movement disorders	tics/OCD/- tourettes	Basal Ganglia
Speech deficits		left frontal broca's; left temporal Wernicke's
Sleep disorders		hypothalamus; pineal gland; SCN
Memory		
Wernicke's aphasia	problems unders- tanding speech	left temporal
Broca's aphasia	inability to express language	left frontal lobe

Brain Areas for Neurological Disorders

Delirium

Alzheimer's Disease (AD)

Parkinson's Disease

Huntington's Disease (Chorea)

Pseudodementia

Major Frontotemporal Neurocognitive

Disorder

Major Neurocognitive Disorder Due to HIV

nfection

Neurocognitive Disorder due to Traumatic

Brain Injury



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Wernicke's Encephalopathy (WE)

Most often arises in people with AUD

Symptoms: Paralysis of the eye muscles,

ataxia, confusion

Caused by thiamine deficiency (vitamin B1)

Other severe malnutrition,

causes: prolonged vomiting, extended

IV nutrition, cancer, immunodeficiency, liver disease, hyperthyroidism, and severe

anorexia.

Risk: If left untreated, **WE** can lead

to Korsakoff syndrome (KS)

Wernicke-Korsakoff Syndrome

The disorder is associated with patients fabricating stories in the setting of clear consciousness. Confabulations can be spontaneous or provoked

Cause: Severe brain disorder caused

by a deficiency of thiamine

(vitamin B1)

Symptoms: Memory disturbances in

which there are significant deficits in anterograde and retrograde memory

Immediate memory is maintained, but shortterm memory is diminished with intact sensorium

Parkinson's

Features: Movement disorder with

tremors, rigidity, bradykinesia, shuffling gait, and neuropsychiatric symptoms (e.g., depression, neurocognitive disorder).

Parkinson's (cont)

Brain Caused by the gradual loss Locus: of dopaminergic neurons in

the **Substantia Nigra** (part of the **Basal Ganglia**, which regulates voluntary movement).

Treatment & Psychopharmacology

Levodopa Dopamine precursor to (L-Dopa) - improve movement

Carbidopa - Reduces Levodopa side effects (e.g., nausea,

hypotension) and enhances its effectiveness.

Dopamine Stimulate dopamine

Agonists - receptors

Enzyme MAO-B and COMT inhibitors
Inhibitors - slow dopamine breakdown.

Amantadine Helps reduce involuntary

movements

Anticholi- Reduce tremors and muscle

nergics - rigidity

Deep Brain Surgical treatment for severe

Stimulation cases

(DBS) -

Alzheimer's Disease (AD)

Defini The most common major neuroction: ognitive disorder (NCD).

ognitive disorder (NCD), accounting for up to 80% of cases.

Alzheimer's Disease (AD) (cont)

Preval- Affects 1 in 8 people over 65, ence: more common in women due to

longer life expectancy.

Neurop- Acetylcholine deficiency,

ath- affecting learning and memory; ology: Amyloid plaques & neurofibrillary

tangles; Damage to the hippoc-

ampus and amygdala

Disease Begins up to 20 years before

Progre- symptoms appear. 1: pre-clinical ssion: (no symptoms), 2: MCI, 3: dementia due to AD

Symptoms

Early: Memory loss, apathy,

depression

Progre- Disorientation, confusion,

impaired judgment, behavioral

changes, motor and gait issues

Late: Loss of communication, failure to

recognize loved ones, bedridden

Types of Dementia

ssive:

Alzhei- The most common type, characmer's terized by the accumulation of disease: **amyloid plaques** and **tau tangles**

in the brain

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Newborns: 50%; 5-year-olds:

Types of Dementia (cont)

Vascular dementia:	Caused by damage to blood vessels in the brain, which reduces blood flow and oxygen to the brain
Lewy body dementia:	Characterized by the presence of abnormal protein deposits called Lewy bodies in the brain.
Fronto- temporal dementia:	Affects the frontal and temporal lobes of the brain, leading to changes in <i>behavior</i> , personality , and language
Mixed dementia:	A combination of two or more types of dementia

Dementia VS. Pseudo-dementia

Pseudo-	Cognitive impairment in older
dementia	adults due to depression,
	mimicking a neurocognitive
	disorder (NCD).
	Key Differences

Dementia (NCDs)	Pseudodementia
Progressive cognitive decline	Slower processing speed, difficulty with concentration and attention, psychomotor retardation
Patients often deny memory issues	Patients acknowledge memory loss
Irreversible	Cognitive function improves

Sleep Patterns Over the Lifespan

sleep	20-25%; Older adults: 18%
decreases	
with age:	
Functions	Psychological restoration;
of REM	Memory consolidation &
Sleep:	emotional processing; Brain
	development; Dreaming (often
	bizarre and illogical)

Movement Disorders

Originates in the:	Basal Ganglia
Definition:	Abnormal repetitive movements
Basal Ganglia:	also the reservoir of our over-learned motor patterns, like riding a bike, automatic daily habits, backing out of the driveway, etc.
Hyperk- inetic	Excess or involuntary movements (e.g., huntington's disease/chorea, tremors, tics/ tourette's syndrome)
Hypoki- netic	Slow or reduced movements (e.g., parkinson's disease, dementia with lewy bodies)

Huntington's Chorea	
Cause:	Genetic disorder causing degeneration of basal ganglia neurons
Symptoms:	Choreiform (jerky, involuntary movements); Speech outbursts; Progressive cognitive decline

Huntington's Chorea (cont)

Onset:	Typically 40–50 years; often
	passed down before
	symptoms appear
Treatment:	No cure available

Parkinson's Disease

Cause:	Damage to the Substantia Nigra, caudate nucleus, and putamen, dopamine-rich brain areas essential for movement and mood regulation
Symptoms:	Movement difficulties (tremors, rigidity, slowed initiation); Depression, psychosis in severe cases
Possible Cause:	Bacterial infections (e.g., from foodborne pathogens) may travel via the Vagus nerve , leading to inflammation and degeneration
Preval- ence:	Increasing significantly (e.g., Michael J. Fox as a well- known case)
	Treatment
L-Dopa (dopamine precursor)	helps temporarily replenish dopamine and slow symptom progression
Music	may aid movement and mood



deteri-

oration

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once depression is treated

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regulation

Therapy



Tourette's Syndrome

Brain basal ganglia, frontal lobes

Area: and cortex

Comorbidi- OCD; ADHD; Anxiety

ties:

Five A's of Neurologic Symptoms

Ataxia Impaired coordination, balance, and speech

Common Damage to the cerebellum or

Cause: motor areas

Apraxia Inability to perform skilled

movements or gestures (e.g.,

difficulty winking)

Common Parietal lobe damage

Cause:

Aphasia Impaired speech or language

comprehension

Types & Receptive aphasia;

Causes: Expressive aphasia;

Conduction aphasia

Receptive Damage to Wernicke's area

Aphasia (left temporal lobe); speech is

not understood

Expressive Damage to Broca's area

Aphasia (posterior frontal lobe);

Conduction Damage to neural pathways

Aphasia connecting these areas,

affecting verbal repetition

difficulty producing speech

Anomia Difficulty naming objects,

people, or terms

Common Likely damage to the hippoc-

Cause: ampus, thalamus, or other

memory retrieval areas

Five A's of Neurologic Symptoms (cont)

Agnosia Inability to recognize objects,

people, or sensory stimuli

Common Brain damage from **strokes**, Cause: **injuries**, **dementia**, or *neurol*-

ogical disorders

Subtype Prosopagnosia ("Face Blindn-

of ess"

Agnosia:

Prosop- Difficulty recognizing faces

agnosia

Brain injuries

Traumatic Brain Injuries & Concussions

External Direct blow to the head, often

Trauma: blood vessels are torn so blood flow is blocked, tissue dies

Internal stroke (either clot or brain bleed)

Trauma: ,aneurysm, or brain tumor

(internal trauma)

Aftere- Can cause memory impairments ffects of (post-traumatic amnesia,

Head persistent memory deficits),
Trauma: executive functioning distur-

bances, and personality

changes

Traumatic Brain Injuries & Concussions (cont)

Phineas The most well-known case of
Gage frontal lobe dysfunction. His
injury led to drastic personality changes, later
associated with "frontotemporal dementia."

Aftere- May result in a short-term flects of loss of consciousness,

Concus- anterograde amnesia (diffisions: culty forming new memories),

and retrograde amnesia (loss of past memories)

Common Dizziness, headache, fatigue; symptoms: Difficulty concentrating,

memory deficits; Irritability, anxiety, insomnia; Heightened sensitivity to noise and light; Hypochondriacal concerns

Location of Brain Trauma & it's Impact

Aphasia: Loss of Speech or Language Comprehension

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Location of Brain Trauma & it's Impact (cont)

Receptive Aphasia (Wernicke's Aphasia):

Damage to the left temporal lobe (Wernicke's area) impairs language comprehension. The person may speak in gibberish but remain unaware of their incoherence. Temporal lobe damage can also affect semantic and longterm memory.

Expressive Aphasia (Broca's Aphasia):

Damage to the posterior frontal lobe (Broca's area) affects speech production. The person understands language and knows what they want to say but struggles to verbalize it, causing frustration.

Conduction Aphasia:

Damage to the neural pathways connecting Wernicke's & Broca's areas disrupts communication between comprehension and speech production. The person cannot effectively repeat verbal phrases.

Location of Brain Trauma & it's Impact (cont)

Global Aphasia:

Widespread damage affecting both comprehension and speech production, severely impairing communication.

Current Neuro-Imaging Options

Scan:

Quick, cost-effective images of basic structures, very useful as first-line assessment in emergencies to identify brain issues that need emergent care (brain bleeds, etc). Uses radio

MRI:

More expensive, detailed images possible with enhanced soft-tissue resolution to pick up more subtle structural issues. Uses magnetic resonance

CTScan:

Detailed metabolic picture of brain function. Can give info about low (Alzheimer's, stroke/blood vessel damaging affecting function) or high (brain tumor or other inflammatory or cancer-related process). Uses radioactive dye

Memory/Types of Amnesia

Retrograde Amnesia:

Occurs after head trauma, such as a blow to the head. The person cannot recall events leading up to the injury, sometimes spanning weeks or months before the event. However, the ability to form new memories remains intact.

Memory/Types of Amnesia (cont)

Anterograde Amnesia Seen in conditions like Alzheimer's disease, where working memory fails, preventing the formation of new memories. The person struggles to learn new information, making daily functioning difficult. Remote (long-term) memory, particularly music memory, is often preserved. Linked to damage in memory-related brain structures, such as the hippocampus.

Psychogenic Amnesia Caused by psychological factors rather than physical brain damage. Follows different patterns from neurological disorders. Short-term memory and the ability to form new memories remain intact. Longterm autobiographical memory is affected, leading to loss of personal details (e.g., name, birthdate). Unlike neurological amnesia, where long-term memory tends to be retained.

Psychogenic Amnesia /(Psych) = Only one that long-term is affected

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Differentiate	e Types of Seizures	Differentia	te Types of Seizures (cont)	Differentiate	Types of Seizures (cont)
Types of Seizures: Genera- lized Seizures (Gran & Petit Mal):	Affect both hemispheres of the brain from the start and often cause loss of consciousness. (grand mal & petit mal)	Simple Partial:	Person remains conscious and aware. Characterized by abnormal movements that begin in one group of muscles and progress to adjacent groups of muscles in a slow wave of seizure activity per location in the back of the frontal lobe motor area. Person has impaired consciousness or awareness. May involve staring, repetitive movements (lip-smacking, hand rubbing), or confusion. The person may not remember the seizure after it ends. Often a	Jacksonian Seizure:	A type of focal seizure that originates in the primary motor cortex of the brain (part of the frontal lobe) and progresses in a characteristic pattern. Begins with localized muscle twitching (jerking movements) in a small part of the body, such as a finger, toe, or corner of the mouth. Spreads gradually to larger areas of the body (e.g., from hand \rightarrow arm \rightarrow face).
Gran Mal:	Sudden loss of consciousness and stiffening (tonic activity), followed by clonic activity (rhythmic jerking). Most well-known type.	Complex Person Partial: ousness involve s moveme rubbing) person r seizure			
Petit Mal:	Brief episodes of staring, often mistaken for daydreaming. Occur most frequently in children. Last 0-30 sec			Temporal Lobe Epilepsy (TLE)	A type of focal seizure. Originate in the temporal lobes of the brain. Common Symptoms Before: Déjà vu .
Focal Seizures (Partial Seizures):	Originate in a specific area of one hemisphere of the brain. They may or may not cause loss of awareness. (simple partial & complex partial)	Ne	ext Two Are Focal Seizures		Can turn into tonic-clonic (grand mal) seizure.

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Neurotransmitter Functions & Effects			
Neurotran- smitter	Behaviors or Diseases Related		
Acetyl- choline (ACh)	Learning and memory; Alzheimer's Disease's muscle movement in the peripheral nervous system. + ACh = spasms ACh = paralysis		
Dopamine (DA)	Reward circuits; Motor circuits involved in Parkin- son's disease; Schizo- phrenia		
Norepinep- hrine (NE)	Arousal; Depression		
Serotonin (5HT)	Depression, Aggression; Schizophrenia behavior.		
GABA	Anxiety disorders, Epilepsy; Major inhibitory neurotran- smitter in the brain		
Glutamate	Learning; Major excitatory neurotransmitter in the brain		
Endogenous Opioids	Pain; Analgesia (inability to feel pain); Reward		
KEY TERMS:			

Mania: arousal, aggression ADHD: learning, memory Addiction: reward

Specific Disorders & Neurotransmitters

Alzhei-	Acetylcholine, due to it's role
mer's	in the development of
disease	memory of the hippocampus
(Repetitive)	dopamine, due to it's role in
Movement	movement
disorders	
Depression	Low serotonin and low
	norepinephrine

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Specific Disorders & Neurotransmitters (cont) Mania Low serotonin and high norepinephrine Too little GABA. Anxiety Schizo-Excess dopamine; GABA & phrenia Glutamate imbalance Autism Too much serotonin; GABA & Glutamate imbalance Substance use disorder (Repetitive) Movement disorders: Parkinsons, tics, OCD Glutamate and GABA Maintain a homeostatic balance Glutamate & GABA have a seesaw relationship When glutamate is high, GABA is low Children with autism and related disorders tend to lean towards excess glutamate and low GABA Balance must be maintained for their bodies

and nervous system to function properly

Excessive Levels of Glutamate: Can lead to excitotoxicity (overstimulation of neurons), contributing to neurodegenerative diseases (e.g., Alzheimer's, epilepsy, anxiety, stroke).

Deficiency in GABA: Can lead to excessive excitability, associated with anxiety, seizures, and insomnia.

Excessive Levels of GABA: Sedation, cognitive slowing, motor impairments. Disorders Related to this Imbalance: Epilepsy (excess excitation, insufficient inhibition). Schizophrenia (dysfunction in

both systems). Anxiety disorders (GABA

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deficiency).

Glutamate and GABA

Maintain a homeostatic balance

Glutamate & GABA have a seesaw relationship

When glutamate is high, GABA is low

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Balance must be maintained for their bodies and nervous system to function properly

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Disorders Related to this Imbalance: Epilepsy (excess excitation, insufficient inhibition). Schizophrenia (dysfunction in both systems). Anxiety disorders (GABA deficiency).

Disorders

Depression		
Impact on the Brain:	Can lead to brain shrinkage	
Neurotran-	Serotonin, dopamine, and	
smitter	norepinephrine. All of which	
Imbala-	are interconnected	
nces:		
Glutamate:	Profoundly out of balance	

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Depression (cont)

Treatment: Regular exercise can be as

Mild to effective as antidepressants;

Moderate Psychotherapy (CBT) to help

Depression address negative thought
patterns

Treatment:
Moderate
to Severe
Depression

Antidepressant medications may be necessary, though it can take time to find the right one. Side effects (e.g., sexual dysfunction, weight gain, emotional blunting) can be a challenge

Treatment: Electroconvulsive Therapy (ECT): Used for severe or treatment-resistant depression, particularly when rapid symptom relief is needed (e.g., in cases of suicidality or catatonia).

Emerging & Alternative Options

Ketamine therapy:

Research suggests it may rapidly reduce depressive symptoms, particularly in treatment-resistant cases

Depression (cont)

Transc-
ranialA non-invasive treatment
using magnetic pulses,Magneticeffective for depression,Stimul-
ationOCD, and other conditions.(TMS):

ADHD

Bipolar Disorder: Tx & Considerations

Mood Used to manage mood swings
Stabilin bipolar disorder, preventing
both manic and depressive
episodes

Common Lithium; anticonvulsants (e.g., Meds: valproate & lamotrigine); atypical antipsychotics

ElectroconvuIsive
Therapy
(ECT)

Typically used for severe cases of bipolar disorder or treatment-resistant depression; Involves brief electrical stimulation of the brain while the patient is under anesthesia. Effective for acute mood episodes but usually considered after medication and therapy have failed.

Psychotic Disorders

Medications that **block dopamine** can help reduce psychotic symptoms, particularly hallucinations and disorganized thinking, known as **"positive" symptoms** of *psychotic disorders*

However, "negative" symptoms (e.g., apathy, low motivation, and reduced activity) may be less responsive to treatment

Due to side effects, **medication adherence** is often a challenge for individuals with psychotic disorders

Panic Disorder

ECT Treatment

ECT involves a brief **electrical stimulation** of the brain while the patient is under anesthesia

Used in patients with **severe major depression** or **bipolar disorder** that has *not* responded to other treatments

Memory loss (retrograde

Side effects:

amnesia) is a significant concern; Patients may experience gaps in memory from weeks to months before treatment; If ECT is repeated over time, cumulative memory loss can become disruptive

retrograde amnesia - an individual is unable to recall events that occurred before the onset of brain injury/ trauma, but ability to form new memories is intact.

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Psychopharmacology

Antidepressants Classes			
Drug Class	Function		
SSRIs	Blocks reuptake of serotonin from space between neurons		
SNRI	Block serotonin & norepinephrine reuptake		
NDRI	Block reuptake of norepinephrine & dopamine		
TCAs	Tricyclic antidepressants affects serotonin & norepinephrine		
MAOIs	Block recycling of serotonin, norepinephrine, & dopamine		
Others	Affect serotonin/ norepinephrine in		

SSRI Danger Warnings

Increased Youth Suicide Risk Serotonin Syndrom

other ways

Neuroleptic Malignant Syndrome

Risk with Anticholinergic Agents

Seniors:	Anticholinergic agents interact
	with many common medica-
	tions taken by older people
Risk of	Increased risk of dementia in
dementia:	people who used them for
	longer than a few months

Antipsychotics

Movement	/A A		4:
	/////	/ IV/Ie/office	HIGHES
IVICTORIUM	/ IVICITION	IVICUIOU	UOIIO

Sleep/Mood Medications

Neuroleptic Medications Side Effects

Tardive Dyskinesia

Serotonin Syndrome			
Mild	Moderate	Life Threat- ening	
Mydriasis (dilated pupils)	Altered Mental Status (e.g., agitation, disori- entation, excite- ment)	Delirium	
Shiver- ing/Sw- eating	Autonomic Hyperactivity (e.g., rigidity, tachyc- ardia, hypert- hermia)	Hypert- ension/ Hypert- hermia	
Tachyc- ardia (mild)	Neuromuscular Abnormalities (e.g., tremor, clonus, hyperreflexia)	Muscle rigidity/ Tachyc- ardia	
	Management Stages:		
Observe for at least 6 hrs	Admit to hospital; Cardiac monitoring	Intensive Care Unit; cooling meaures	
Benzod- iazepines	Cyproheptadine	Sedation; SkM paralysis; ventil- ation	



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