

Neuroscience

Divisions of Nervous System

Peripheral nervous system

Nerves branching out from spinal cord

1. Motor: Efferent; carrying out actions- signals sent from CNS to muscles
 2. Sensory; Afferent; feeling from senses – signals sent from senses to CNS
- Autonomic nervous system: Involuntary, automatic regulation of body via internal organs e.g. breathing, heart rate, digestion
 - o Uses neurotransmitters e.g. digestion, breathing, heart rate
 - o Parasympathetic:
 - Relaxed state; increases digestion
 - o Sympathetic:
 - Fight/flight situation; increases HR, vigilance, decreases digestion
 - Enteric nervous system: 'Second brain' – many neurons in the gut – esophagus downwards
 - o Independent of ANS but interacts with ANS and the brain
 - o Controls digestion, peristalsis
 - o Also releases neurotransmitters

Central Nervous System

- Brain and spinal cord

Spinal cord

- Cable of fibers extending from brain stem down back
- Below head, sensory/motor nerves feed into spinal cord → brain
- In head, nerves feed directly from brain to muscles, senses to brain, etc.

Protection of brain

1. Bone: Skull
2. Meninges:
 - a. Dura mater: Tough, thick
 - b. Arachnoid mater: Spongy, has blood vessels, filled with CSF
 - c. Pia mater: Thin
3. Blood brain barrier
 - a. Layer of blood vessels with very small pores; prevents harmful substances in the blood from entering brain

Subdivisions of brain and functions

- Brain stem:
 - o Autonomous functions essential for life e.g. breathing, heart rate, walking
 - o Damage → coma and death
- Cerebellum: Precision movements and coordination, learned movements
 - o E.g. playing instrument, writing, eye movement and speech
 - o Damage → inability to coordinate precision movements, but not paralysis

- Hypothalamus and thalamus:
 - Hypothalamus: Hormone regulation via pituitary gland, motivational control (hunger)
 - Thalamus: Sleep cycle regulation, relaying sensory messages to parts of cortex
- Neocortex: Convoluted sheet of neural tissue on top of brain; largest volume
 - Convoluted to allow large surface area within constraints of brain to process a lot
 - Lobes:
 - Frontal lobe: Higher order processing, thinking and imagination, planning of actions
 - Contains motor cortex
 - Parietal lobe: Touch and recognizing space around us
 - Contains sensory cortex
 - Temporal lobe: Emotion, language and memory
 - Contains auditory cortex
 - Occipital lobe: Vision
 - Contains visual cortex
- Limbic system: Emotion and memory
 - Hippocampus: In charge of consolidation STM → LTM
 - Olfactory bulb: Large area; sensory input from nose feeds in directly rather than to thalamus and relayed; smell is very important, and important for memory
 - Amygdala: Fear system
- Basal ganglia: Thought and actions; choosing movements
 - Parkinson's disease → disjointed movements and thinking
 - Shaking, rigidity, slowness

Ventricles

- Space in brain filled with CSF
- 'Drainage system' – removes harmful substances from brain
- Enlargement of ventricles due to blockage → squashing of brain as pressure increases (hydrocephalus)

Neurons, action potentials and myelin

Interactions of neurons: Neural or electrical

Electrical

- Anatomy: Signal travels from dendrites → cell body → axons → synaptic terminal
- Myelination increases signal travel speed
- Interaction through action potentials (travelling nervous signal)
 - Normally in polarized state; more –ve inside than outside
 - Neurons open ion channel → +ve ion rush in
 - If depolarization enough, action potential generated; depolarization continues to occur along membrane and AP travels along
 - If not enough, neuron polarizes again; no AP generated
- Digital signal; on/off

Neural

- Neurotransmitters released from vesicles into synapse when AP reaches synaptic terminals
- NT bind to specific receptors in dendrites
- Lock and key concept
- Ion channels of next neuron open in response to NT → new AP
- NT removed from synapse by reuptake, or destroyed in synapse → increase signal rate

Neuropharmacology

- Drugs work to mimic NT/influence reuptake/destruction of NT
- If mimic NT can strengthen effect, or occupy receptors to block NT effect
- Recreational drugs:
 - o Opiates; mimic opioids → inhibitory
 - o Cocaine, amphetamine → promote dopamine, noradrenaline, serotonin
 - o Nicotine → acetylcholine → wakefulness
 - o Caffeine → blocks adenosine receptors (which normally → inhibitory)
- Therapeutic drugs:
 - o Antidepressant → blocks reuptake of serotonin and noradrenaline
 - o Antischizophrenic → blocks dopamine (which → excitement) → inhibits

Mapping functional organization of brain

Methods

1. Animal lesions: Remove part → observe; Lobotomies
2. Electrode monitoring/electrical stimulation: Insert electrode to monitor signals
3. Neuroinjection of drug: Observe effect of drug (not necessarily destroy)
4. TMS: Stimulate part of brain through magnetic pulse → depolarizing AP

Imaging methods and limitations

1. EEG: Measure magnetic field around brain
 - a. Poor spatial resolution
 - b. Good temporal resolution
2. PET: Inject isotopic glucose/oxygen which emits positrons; depends on increase in blood concentration in area with activity
 - a. Poor temporal resolution due to time delay
 - b. Good spatial resolution
3. MRI: Person placed in MRI scanner with electrical field ; radio waves change orientation of Hydrogen, measure reorientation via emission of waves
 - a. Maps water concentration for map of soft tissue; not function
4. fMRI: FUNCTIONAL MRI- Determines activity via changes in oxygen concentration in blood
 - a. Good spatial resolution; 3D image available
 - b. Poor temporal resolution due to BOLD response
5. MEG: Measures magnetic field
 - a. Good spatial resolution – 3D images

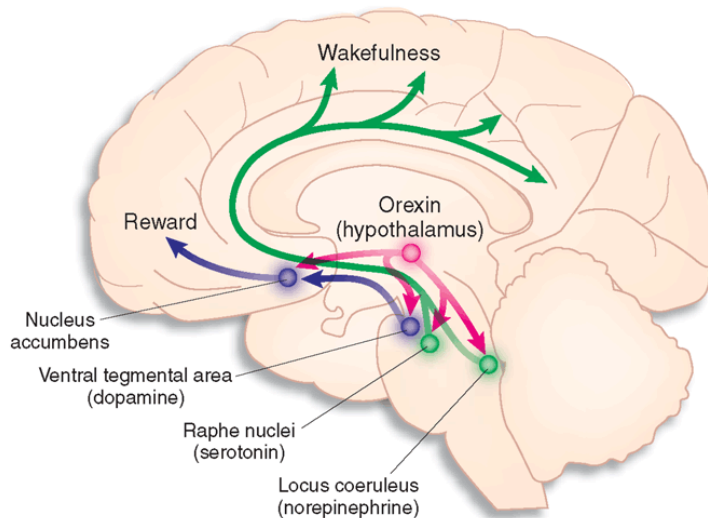
- b. Good temporal resolution
- c. Difficult to set up; have to be very controlled, very expensive
- 6. CT: Shows tissue only; not activity

Sleep and wakefulness (Brain stem and hypothalamus)

Wakefulness controlled by neurons in brain stem which release certain NT to rest of brain:

Stimulation → wakefulness

- Acetylcholine in Pons
- Serotonin in raphe nuclei
- Noradrenaline in locus coeruleus
- Stimulant drugs enhance Ach, SHT, NA



Sleep controlled by preoptic area of hypothalamus (stimulation → sleep) which release NT which inhibit other neurons

- GABA → inhibitory NT
- Inhibits acetylcholine, serotonin, noradrenaline in brainstem (responsible for arousal)
- Depressant drugs enhance GABA

Sleep Cycles

- When awake, noisy in brain
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- When asleep, neural activity synchronizes → 'slow wave' sleep
- Slow wave sleep is conducted by thalamus which relays activity of different parts of brain to diff parts so they synchronize
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- REM sleep; neural activity is noisy – our 'dream' stage- desynchronized
- Rapid eye movement
- Pons sends Ach to thalamus which relays neural signal to the visual cortex which interprets neural signals as a story/dream
- Movement is inhibited by Ach in pons which stimulates medulla neurons in brain stem that inhibit motor neurons in spinal cord

Feeding

Controlled by hypothalamus

- Lateral hypothalamus: Destruction → less eating
 - o Controls insulin release; usually causes insulin release (used to store glucose as glycogen); destruction → lower insulin release → glucose in blood high → lower appetite
 - o Regulates attention; destruction → Lower interest in food
 - o Influences taste; destruction → less liking of food
- Paraventricular nucleus of hypothalamus
 - o Destruction → more eating, larger portions i.e. not satiated
 - o Processes cholecystokinin released as food passes through stomach
 - o Destruction → unaware of food passing through → bigger portions
- Medial hypothalamus
 - o Destruction → obesity, not larger portions, but more often eating → not a problem with satiety
 - o Control of insulin release; usually inhibits insulin release to maintain high glucose level; If destroyed → excessive insulin → low blood sugar → higher appetite
 - o Controls gut motility – destruction → excessive gut motility

Reward system

- MFB w/ bundles of dopamine and noradrenaline fibres from brainstem to forebrain
- Primary reward pathway: Dopamine axons from VTA to nucleus accumbens
Release of dopamine into nucleus accumbens

Lateralisation of brain

- Brain although 'symmetrical' is not completely symmetrical in functions i.e. some functions more lateralized i.e. processed better in particular hemisphere e.g. language
- Language better processed by left side of brain although other factors e.g. emotion in language (e.g. satire) is better processed by right side of brain
 - o Right side processes emotion better → left side of face can express emotion better, left visual field can perceive emotion better
- Speech controlled by left side of brain: (even though comprehension processed by both)
 - o Broca's area: Expressive aphasia; cannot make sense, but can comprehend
 - o Wernicke's area: Receptive aphasia; can talk, but doesn't make sense; cannot comprehend
- Evidence of lateralisation of language:
 - o Dichotic listening task; better comprehension when hearing from right ear (processed by left side)
 - o Brain imaging; left side lights up more w/ comprehension and speech
 - o Stroke in left side → aphasia
- Information is shared between both sides of brain;
 - o If corpus callosum cut, (split-brain) then perfectly functional, but have difficulty with coordinating decisions and sides of brain work separately e.g. choosing with hands

- Can name object in right hand, but not left because speech is controlled by left side of brain

Memory

Anterograde amnesia

- Hippocampus → consolidating STM to LTM → anterograde amnesia
- HM had lobotomy of hippocampus
- Anterograde amnesia (has past info but cannot remember any new info)
- Retrograde amnesia (lost past memory but remembers new)
- Procedural memory not affected; can still learn things without remembering having learnt it or done it e.g. drawing in mirror

Wernicke Korsakoff's Syndrome

- Due to excessive alcoholism → deficiency in B12 → Wernicke Encephalopathy (disorientation, confusion) → Korsakoff's psychosis w/ anterograde amnesia, some retrograde amnesia
- Due to damage to mammillary bodies

Alzheimer's disease

- Enlargement of ventricles and sulci (gaps)
- Starts in temporal lobe (emotion and memory) → hippocampus
- Strange brain tissue e.g. senile plaques
- Loss of recent memory → older memory → loss of procedural learning; learned actions like feeding and talking