CH 1

History of Research on Brain and Behaviour

Egyptians aware of symptoms to brain damage. They viewed the heart as the organ that helps memories. It was seen as the centre of all intelligence and consciousness. There was minimal importance on the brain. Organs were preserved by mummification. All the organs were removed first. The brain was removed through the nostrils and discarded.

Aristotle also believed the heart to be the centre of all consciousness. He believed the brain was a cooling mechanism for the body.

Herophilus= father of anatomy. He obtained his advanced knowledge by dissecting animals and humans. Discovered that muscles and nerves were connected to the spinal cord.

Galen =father of medicine. He treated gladiators and their brain injuries. Discovered that brain damage leads to changes in behaviour. BRAIN= CONTROLLER OF BEHAVIOUR. Fluid (animal spirits) pass through the nerves.

Da Vinci studied inner workings of body introduced anatomical cross sectional drawings of brain. Recognized that the brain is complex and theres more to it than just whats on the outside. He made a wax figure of the brain and cut away ventricles to see how it looked inside.

Michelangelo believed the brain to be gods greatest gift to mankind.

1600s

Descartes said the brain functions like machinery. Fluid goes through ventricles and facilitates movement. He discovered this through observation. Thought it applied to animals only. Believed animals didn't have intellect or a god-given soul.

He differentiated between brain and mind. Mind and spiritual entity receives sensations which controls movement. Pineal gland. If you touch fire nervous activity foes up to the brain and reflects back to the muscle causing the person to contract said muscle (reflexes)

1800s

Willis: Brain coordinates and controls behaviour. Phrenology: Brain has different functional areas responsible for different behaviours such as love, happiness, fear, etc. Others thought that there was no localization but that everything functioned as a unit observed from the bumps in the skull. Brain functions as a whole.

Paul Broca

- Functions localized in brain regions
- Focused on language abilities

- Post mortem research

whole brain is active always not matter what you're doing. Localization refers to the peak action in an area of the brain when someone performs a task.

1900s

Ebbinghaus measured learning and memory in humans. Thorndike did this on animals.

Pavlov: conditioning

Franz removed different brain regions in animals In search for the learning and memory site of the brain

Hebb: complex cognitive behaviour could be accomplished by networks of active neurons. Brain cell connections may be organized by sensory input and stimulation into cell assemblies.

CONSCIOUSNESS

Zeman

- Consciousness matters
- Allows us to plan
- Brain activity
- Some brain activity is unconscious
- Deepest parts of brain are responsible for arousal
- Top part responsible for experiences from moment to moment.

2000s

86 billion neurons that contact each other at an area called the synapse

100 000 miles of axons. Electrical impulses travel 100 miles/hr.

What is behavioural Neuroscience? The study of the nervous system

5 viewpoints

1. Describing behaviour
2. Evolution
3. Development
4. Biological
5. Applications

1) Describing behaviour

Theres different criteria to describe (acts, processes, functions)

2) Evolution

Compare species. Continuity of behaviour and processes due to common ancestors. Differences in behaviour and biology that have evolved. We are all alike but

also different. We all have dna, vertebrates have a backbone/spinal cord, mammals suckle their young, etc

3) Development

Ontogeny= change through a lifespan. Learning happens within first three years

4) Biological mechanisms

Must understand present function of body. Understanding mechanisms. Electrical and biochemical processes.

5) Research Applications

Using behavioural neuroscience to improve understanding of human conditions. Helps understand diseases of the brain (alzheimers, Parkinson's, MS, etc. these diseases are caused by dopamine cells dying off. Treatment eldopa)

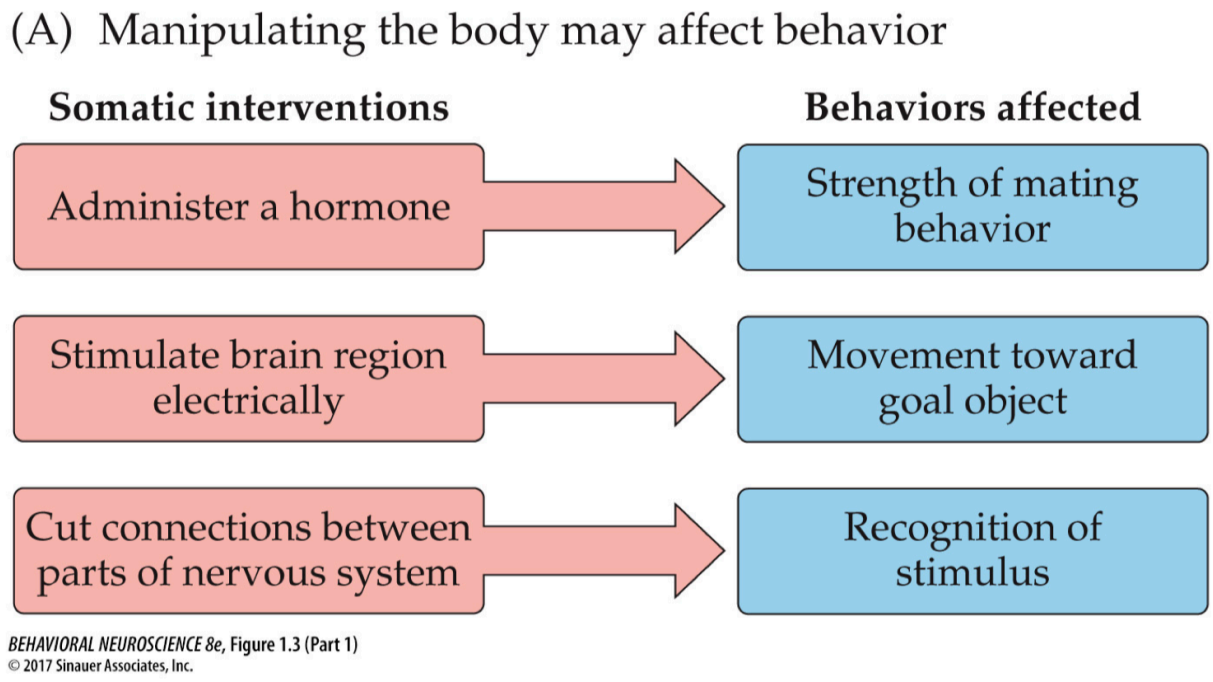
Three Approaches to Understand the Relationship Between Brain and Behaviour

1) Somatic Intervention

Body and behaviour variables. Manipulate brain region to then see a behavioural response.

I.V: Factor being manipulated: brain region

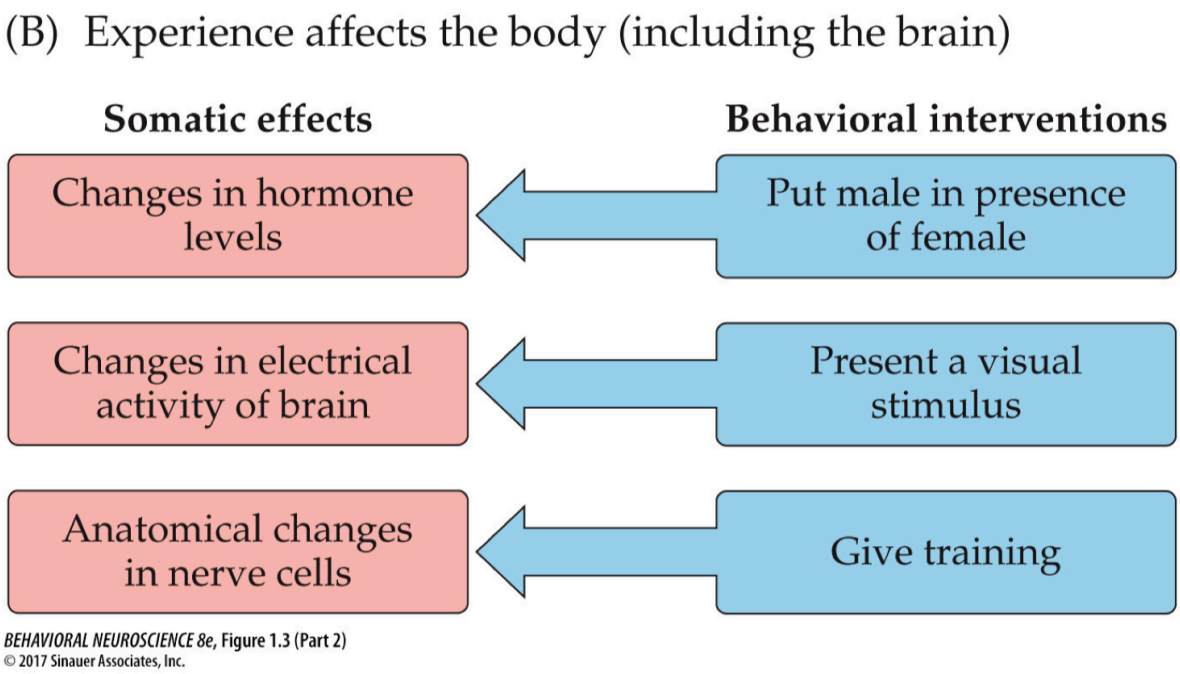
D.V: what is being measured: Behavioural response



BEHAVIORAL NEUROSCIENCE 8e, Figure 1.3 (Part 1)

2) Behavioural Intervention

Change in behaviour and measure change in function.



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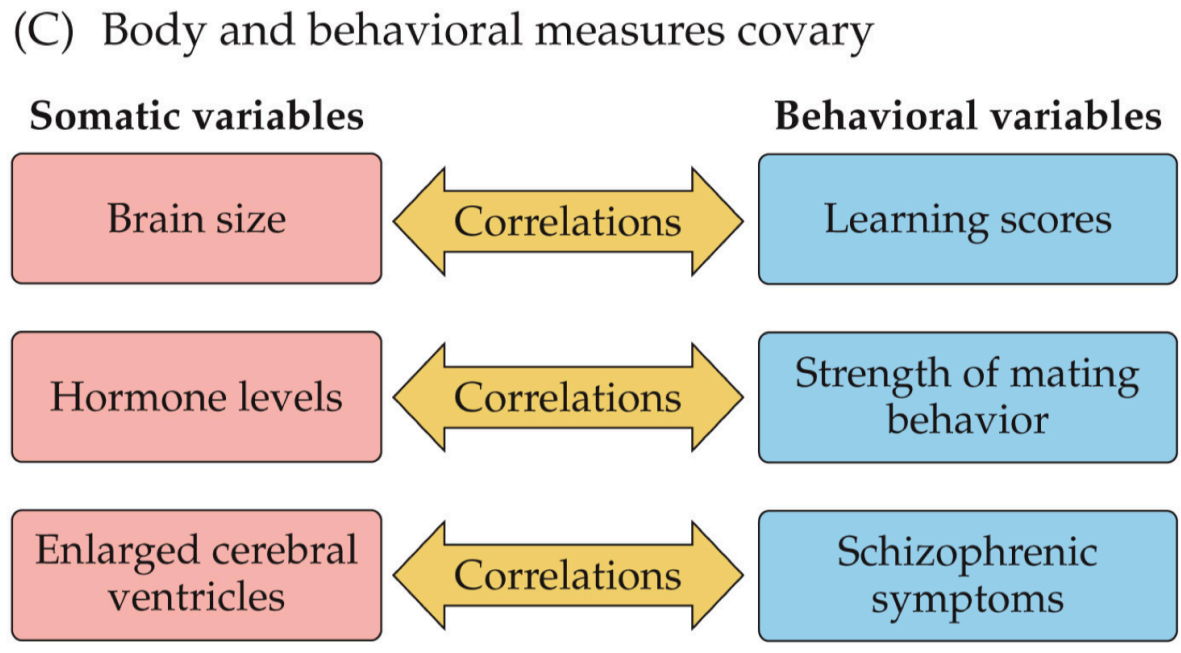
3) Correlation

Covariation. How much body variable varies with behavioural variable.



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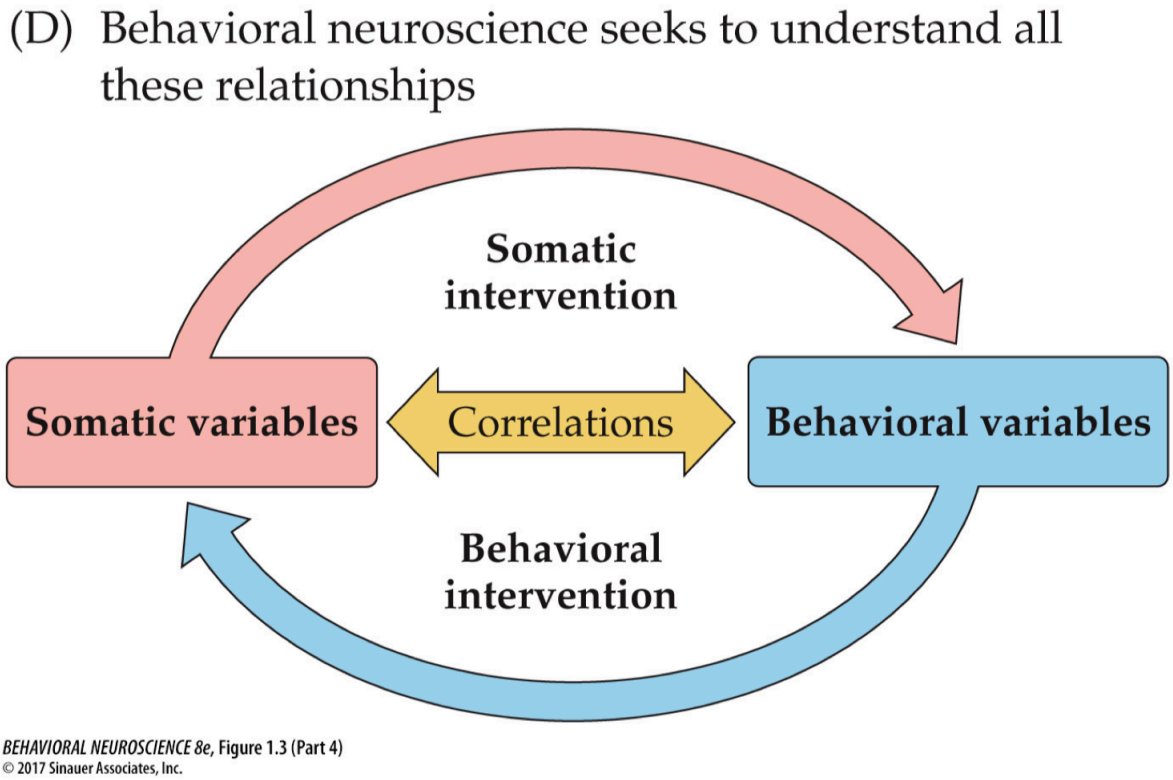


Neuroplasticity

Brain changes based on the environment and based on personal experience. Effects the number and the size of neurons.

Socially endured psychological expectations affect the magnitude of brain response even if the stimulus is the same.

Experience physically alters the brain and therefore effects behaviour.

Levels of Analysis

Reduction: scientific strategy of breaking a system down into increasingly smaller parts in order to understand.

Social interactions to the brain

The scope of experimental approaches.

Disorders

it is estimated that 38% of the population would suffer from a mental disorder at some point in a typical year. 1/5 people suffer from a neurological disorder. Cost of these disorders ! 400billion/year in USA

CHAPTER TWO

In response to electrical activity in the axon, the vesicles fuse with the presynaptic membrane and rupture. This process releases the neurotransmitters into the cleft. After defusing across the cleft they release neurotransmitters and these interact with the postsynaptic receptors. This causes electrical changes

Neurotransmitters bind to receptors and then dissociate. Postsynaptic membrane has a high density of receptors.

Histology changed when derivatives of fabric dyes were found to vividly stain cells to allow visualization of previously hidden microscopic structures stain selectively parts of neurons and glial cells

Cell stains: nissl stains outline all cell bodies because the dyes are attracted to ran distributed within the cells. These stains allow us to measure the cell body size and density of cells.

Examining the Forms mom Individual Neurons

Golgi stains label a minority of neurons in a sample. The affected cells are stained very deeply and completely and reveals fine details of the cell like dendrite branches. It helps quantify types and precise shapes of neurons in a region. Fluorescent molecule injections produce a similar result.

Mapping the Expression of Cellular Products

Autoradiography allows you to see the distribution of radioactive chemicals in tissues. Animals are treated with radioactive versions mod experimental drugs and then thin slices of the brain are placed alongside a photographic film.

Immunohistochemistry creates antibodies against a protein of interest. They detect proteins of interest. Antibody binds to the protein chemical treatments make the antibody visible and reveals cells with a common protein.

In-Situ hybridization: uses radioactively labeled lengths of nucleic acids and labels only neurones in which a gene of interest has been turned on

Tracing inter connections between neurons

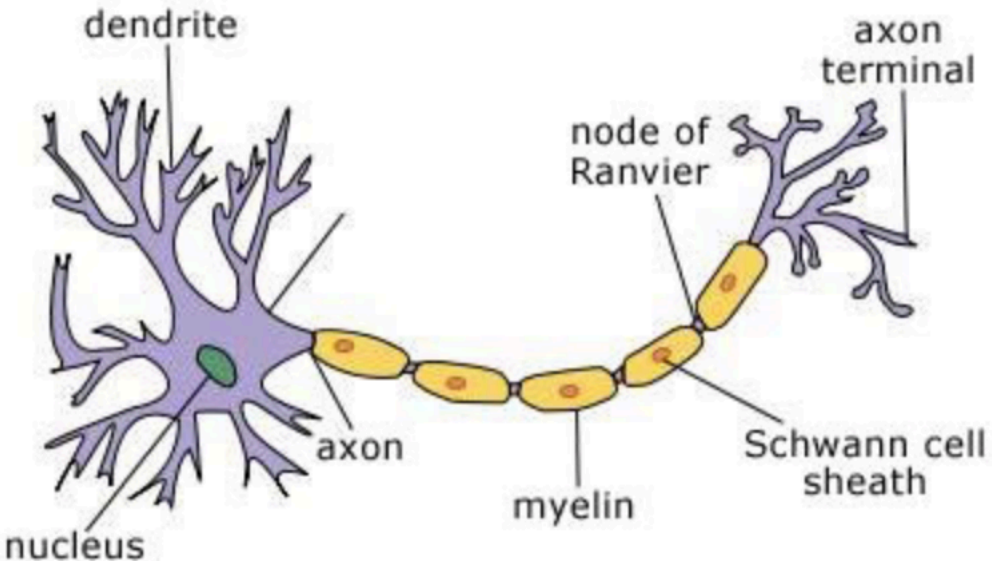
Tract tracers: substances taken up by neurons and transported through their axon.

Anterograde labelling reveals the axonal targets of cell bodies in a particular region.

Retrograde labelling reveals the cell bodies of axons termination in a region.

Some tract tracers can work trans synaptically meaning they can jump backwards across synapses and work their way upstream.

Axon hillock is the extension between the cell body that leads into the axon. Its the neurons integration zone. It gathers and integrates information and sends it down the axon. It manufactures important proteins and enzymes needed at the axon terminals



Axonal transport: movement of materials within an axon via motor proteins.

Loaded into vesicles with motor proteins. Guides to axon terminal.

2 directions:

Anterograde: down towards terminals

Retrograde: both directions. Recycled.

1.materials are produced in the cell body

2.they pass from cell body through axon hillock and then down the axon going towards the axon terminals

3.happens by way of motor proteins.

TABLE 2.1 Distinctions between Axons and Dendrites

PROPERTY	AXONS	DENDRITES
Number	Usually one per neuron, with many terminal branches	Usually many per neuron
Diameter	Uniform until start of terminal branching	Tapering progressively toward ending
Axon hillock	Present	No hillock-like region
Sheathing	Usually covered with myelin	No myelin sheath
Length	Ranging from practically nonexistent to several meters long	Often much shorter than axons

Glial Cells

Believed to hold the nervous system together. Communicate with each other and with neurons. Provide neurons with chemical signals that alter.

4 TYPES OF GLIAL CELLS

1. Astrocytes: star shaped numerous extensions. Receive neuronal input and monitor activity. They attach to cell and capillary
2. Microglial: they're the "cleanup crew" They're tiny and remove cellular debris from injured and dead cells. Towards area affected by doses and brain damage. DAMAGE CONTROL
3. Oligodendrocytes: Myelination. Myelin insulates the axon and improves speed production of action potential
4. Schwann: myelination

There is a section of the axon without myelin. This has to do with impulse transport. It increases the speed of conduction of impulses and allows it to jump from node to node.
Glial cells divide throughout a lifespan. Tumours (brain) usually a result of glial cells

-edema. Swell in result of injury. Responsible for symptoms of brain injury

-astrocytes: brain chemistry. Implicated in epilepsy.

-microglia: neurodegenerative diseases (alzheimers)

Multiple sclerosis is a demyelinating disease.

The Nervous System

Gross neuroanatomy: features visible to the eye.
Peripheral Nervous System is all parts aside form the skull and spinal column
Central Nervous System includes brain and spinal cord

PNS: bundle of neurons: ganglia. Nerves are a bundle of axons. Motor nerves transmit info from cns to muscles, organs and glands. Sensory nerves send info from body to cns.
Nerves

Somatic nervous system. Nerves interconnect the brain and major muscles and sensory systems. Autonomic.

Cranial nerves are part of the somatic nervous system. There are 12 pairs.

3 sensory pathways

- Olfactory (smell)
- Optic (vision)
- Vestibulocochlear (inner ear)

5 motor pathways

- Occulomotor (eyes)
- Troclear (eyes)
- Abducens (eyes)
- Spinal Accessory (neck)

- Hypoglossal (tongue)

4 sensory and motor pathways

- Trigeminal (faces)
- Facial (salivary)
- Glossopharyngeal (throat)
- Vagus (internal organs

Spinal nerves (somatic) 31 pairs

emerge through opening in back bone.

Dorsal root: carries sensory info from body to spinal cord

Ventral root: carries motor to muscles from spinal cord.

Named after where they're connected to

Cervical (8): neck

Thoracic (12): trunk

Lumbar (5): lower back

Sacral (5): pelvic

Coccygeal (1): bottom

Gray matter interneurons sends axons to muscles. White matter is myelinated axon.

Automatic nervous system spans across CNS and PNS and controls the organs within the body.

Autonomic ganglia, groups of neurons located outside the CNS

Preganglionic neurons run from the CNS to the autonomic ganglia. Whereas Postganglionic neurons run from the autonomic ganglia to targets in the body.

Autonomic nervous system has three major divisions:

- Sympathetic nervous system: prepares body for action. Increase blood pressure, heart rate, pupil size, etc. Fight or flight mode
- Parasympathetic nervous system: rest and digest. Prepares for potential action
- Enteric nervous system: network of sensory and motor. Regulate function in gut. Controlled by CNS. Regulates nutrient balance and fluid balance.

Central Nervous System

Consists of the brain and the spinal cord. The spinal cord tunnels sensory information from the body to the brain and uses brains motor commands to control the body.

Within the CNS, nuclei refer to to a collection of neurons and a tract is a bundle of axons.

The Brain

Two bilaterally symmetrical cerebral hemispheres.

Corpus collosum is a bundle of axons that connects these two hemispheres.

generally speaking, each side of the brain controls opposite sides of the body. It also receives information from the opposite (contralateral) side

The cerebral cortex is the this outermost sheet of the cerebral hemispheres which is made up of cell bodies, dendrites and axons

There are 2 lobes

1. Gyri / gyrus: ridges or raised portions of the convoluted brain surface
2. Sulci / sulcus: furrows of the convoluted brain surface.

Viewing the Brain

3 ORIENTATIONS

- Sagital plane: bisects the body into right and left halves
- Coronal plane: divides the body into front (anterior) and back (posterior)
- Horizontal plane: divides the brain into upper and lower parts
- Medial: towards middle
- lateral: towards side
- ipsilateral: same side
- Contralateral: opposite side
- Anterior or rostral: head end
- Posterior or caudal: tail end
- Proximal: near the centre
- Distal: towards periphery

The Flow of Information

Dorsal: towards the back

Ventral: towards the belly or front

Describing the flow:

- Afferent: carrying info into a region of interest
- Efferent: carrying info away from a region of interest

The Cortex

The cortex is divided into four lobes

1. Frontal: most anterior region. Control pannel. Cognitive function (memory, language)
2. Parietal: region between the frontal and occipital lobe. Sensory info integration. Knowledge of numbers, tactile snese, manipulation of objects.
3. Occipital: posterior region, visual processing. Receives and processes info from eyes

4. Temporal: lateral region, auditory processing. Sensory info (words/speech) from ears. Sense of smell. Learning and memory aspects.

Physical Boundaries

Boundaries between the lobes may be functional or physical

- Sylvian fissure (lateral sulcus)- boundary of the temporal lobe
- Central sulcus: divides frontal lobe from parietal lobe

Functional boundaries

Via corpus collosum

- Postcentral Gyrus: sense of touch. Behind central cortex
- Precentral Gyrus: in frontal lobe. Important in motor control

Brain Tissue

- Gray matter: Contains more cell bodies and dendrites. Lack myelin. Receives and processes.
- White matter: consists mostly axons with white myelin sheaths. Transmit info within brain and CNS

Developmental Subdivisions

Neural Tube: develops three subdivisions

1) forebrain (prosencephalon)
2) midbrain (mesencephalon)
3) hindbrain (rhombencephalon)

Forebrain later develops into telencephalon (later becomes frontal lobe) and diencephalon (thalamus/hypothalamus)

Hindbrain develops into two subdivisions

Metencephalon: becomes cerebellum, pons

Myelencephalon: becomes medulla

Brainstem: cerebellum, pons, medulla

Forebrain (telencephalon)

Brains bilaterally symmetrical with the exception of the corpus collosum and pituitary

Brain is contrallaterla

Neurons of the cerebral cortex are arranged in 6 layers. Each layer has a distinct appearance. They are unique and differentiated based on the type of neurons and the pattern of dendrites or axons.

Some structures have allocortex instead this has either three layers or is unlayered.

Pyramidal cells are the most prominent neurons in cerebral cortex. They are pyramid shaped cell bodies in layer 3 or 5. The **apical** dendrite extends to outermost cortex and the **basal** dendrite spreads horizontally from the cell body.

Neurons in the cortex are organized into cortical columns. Each column is perpendicular to the cortical layer and serves as a unit to process information. They allow each other to communicate via tracts of axons.

The **basal ganglia** is important in motor control. Gray matter structure

- Caudate nucleus, putamen and globus pallidus- under cerebral cortex.
- Substantia nigra: in the midbrain

The **limbic system** includes structures important for emotion, learning and memory

- Amygdala: emotional regulation (fear, anxiety, aggression) and odour perception
- Mammillary bodies, hippocampus and fornix: contribute to learning and memory
- Septal nuclei: reward and reinforcement in learning

Brain Shows Regional Specialization of Functions

- Cingulate gyrus (on top of corpus collosum): attention, behaviour regulation
- Olfactory bulb: sense of smell
- Stria terminalis-fibre pathway: connects amygdala to other structures. Motivated behaviours: sex/threat responses. Hormonal signals.

Forebrain (Diencephalon)

- Thalamus: cluster of nuclei. Switch box. Sends info to regions for processing. recieves info from cortex to know which info is to be transmitter
- Hypothalamus: contains nuclei with many vital functions. hunger, thirst, sex, temperature regulation. Controls the pituitary which secretes hormones and controls processes like growth, blood pressure, breast mile production, pain relief, etc

Midbrain

Small

top: tectum 2 bumps

1. Superior colliculi: processes vital information
2. Inferior colliculi (back of head): process auditory information

Midbrain has two motor sensors

1. Substantia nigra: contains neurons that release dopamine
2. Red nucleus: communicates with motor neurons in spinal cord. Baby crawling, arm movement when walking

Reticular formation:

Midbrain to medulla

Involved with sleep, arousal, temperature control and motor control.

Hindbrain (Myelencephalon)

Cerebellum: elaborately convoluted. Involved in motor coordination and learning. Attached to brain stem. Receives info from spinal cord. Coordinates voluntary movement.

The cerebellum consists of three layers:

1. Purkinje cell: middle layer. Large cells form single row. Enormous neurons. Elaborate fan shah dendrite patterns
2. Granule cell: axons rise to surface to form the parallels fibres. Composed of small neurons whose axons form third layer
3. Parallel fibers: molecular/outermost layer

The **pons** is part of the brainstem. It is attached to the cerebellum and contains motor control and sensory nuclei. It gives rise to cranial nerves. Info from ear first goes through the pons.

The **medulla** contains cranial nerve nuclei and marks the transition from brain to spinal cord. Most towards back. Controls neck and tongue. Regulates breathing and heart rate. Damage to this is fatal. All axons from the brain to the spinal cord pass through the medulla.

Specialized Support Systems Protect and Nourish the Brain

The brain and the spinal cord are surrounded by three protective membranes. The MENINGES:

1. Dura mater: tough outermost sheet
2. Pia mater: delicate innermost layer. Sticks lightly to brain surface.
3. Arachnoid: substance between dura mater and Pia mater that cushions the brain in cerebrospinal fluid (CSF). Web like

CSF has two main functions: (1)Acts as a shock absorber. (2)Provides an exchange medium between blood and brain. Fills ventricular system.

Meningitis is when the meninges are inflamed

The ventricular system is a series of champers filled with CSF

The choroid plexus produces CSF through blood. flows from later to third towards fourth. Below cerebellum there are three openings which allow CSF to leave. It circulates outside and the spine. Goes through the circulatory system through the veins at the top of the skull

Vascular system

brain makes up 2% of body weight but needs 20% of our energy when it is at rest. Large blood vessels fuel it.

The carotid arteries are the major arteries to the brain. There are located on the sides of the neck. The internal carotid enter the skill anteriorly and through the middle and supply blood to 2/4 of the cortex. The external (posterior) artery supplies the rest of the blood from the spinal column to the base ofd the skull.

The Circle of Willis is a structure formed by the major cerebral arteries which provides an alternate route for blood flow. In the event of arterial blockage or disease.

Stroke is caused by the rupture or blockage of blood vessels leading to insufficient blood supply to the brain. It isn't always fatal. Symptoms are: sudden weakness, numbness, double vision, dizziness, confusion, difficulty speaking.

The Blood Brain Barrier

WATCH VIDEO

Ch 3 NEUROPHYSIOLOGY

How do neurons in the nervous system communicate?

Fn as info processing device. Take in info, analyze, pass on results of analysis to other neurons.

They do this using electrical signals in neurons and them signals between them.

Stimulation of vagus nerve causes stimulation into fluid. This won a Nobel prize in 1936

Electrical signals (movement of ions)

All cells have electrical charge

more negative inside vs outside

these electrical signals underlie the whole range of thought and action.

Neuron at rest. Not influenced by others. Not producing signal

A Balance of Electrochemical Forces Produces the Resting Membrane Potential of Neurons

Ions: electrically charged molecules

-Anions: negatively charged

-cations: positively charged

These ions are dissolved in In cytoplasm (intercellular fluid) separated from extracellular fluid by cell membrane made of lipid bilayer (Two layers of linked fatty molecules).

More negatively charged ions inside neuron rather than outside.

This can be measured by inserting a fine microelectromechanical inside a neurone and using a voltmeter to compare the cells interior with the extracellular fluid surrounding it. This shows that a neuron at rest exhibits a resting membrane potential of -50 to -80 thousandths of a volt (mV) neurons have -65mV (negative bc negative polarity of cell interior).

Electrical signals are the vocabulary of the nervous system

Neurons have ion channels which are tubelike pores that allow specific ions to pass through the membrane. Some of these channels always remain open. An example of this channel is the one that uses selective permeability to allow potassium (k^+) to pass in and out of the cell while others cannot.

The resting potential of the neuron reflects a balancing act between two opposing forces that drive K^+ in and out of neurons:

Diffusion: causing ions to flow from areas of high to low concentration along their concentration gradient. They move until evenly distributed. When a selective permeability membrane divides the fluid, particles that can pass through the membrane, such as k^+, will diffuse across until they are equally concentrated on both sides. Its bc of this membrane preventing some molecules through that the inside of our cells are negative

Electrostatic pressure: causing ions to flow towards oppositely charged areas.

Neurons use the sodium-potassium pump to maintain resting potential. Neurons use this mechanism that pumps three sodium ions out of the cell for every two potassium ions pumped in. This consumes energy. A big portion of energy consumed by the brain is used to maintain these ionic differences across neuronal membranes. The sodium-potassium pump causes a build up of potassium ions in the cell. This causes potassium to leave the interior down the concentration gradient which causes a net buildup of anions in the cell. As this negative charge builds up it exerts electrostatic pressure. This pulls cations back into the neuron. eventually, the pushing out of K^+ ions out and the electrostatic pressure pulling them in reaches equilibrium. Any further moving of k+ into cell (electrostatic pressure) is then matched by its flow out of the cell in terms of diffusion (moving down the concentration gradient). When enough k+ have left the cell to bring the potential to -65mV, the electrostatic pressure is perfectly balanced by the gradient. Potassium equilibrium potential.

The **Nernst equation** predicts the voltage needed to counterbalance the diffusion force pushing an ion across a membrane.(the equilibrium potential) Ion moving from one side of ion to other

WILL NEVER BE ASKED EXACT EQUATION

Predicts the equilibrium potential of an ion, usually K+

The equation predicts a potential of -80mV

The measured equilibrium potential of neurons is typically closer to -65mV. This discrepancy is that the equation assumes its only permeable to potassium but its also somewhat permeable to some other ions

The **Goldman equation** predicts voltage potentials that are quite close to observed resting potentials. It takes inter and extracellular concentrations of K+ and Na+ into account as well as the degree of permeability. Goldman equation is better estimate of voltage potential needed for eq.

Ions inside and outside neuron: more intracellular concentration of k+ inside cell, more Na+ outside cell. This is thanks to pump. Cl vs Ca, mostly in extracellular fluid. These ions are exchanged through specialized channels in membrane. Also have own pump . Neurons keep levels of intracellular calcium ions low by using a pump to eject them and by using specialized proteins that store Ca^{2+} to use for intracellular singling like synaptic release of neurotransmitters.

Resting potential of neuron provides baseline level of polarization found in cells
unlike all cells, a neuron routinely undergoes a brief but large change in polarization. This is action potential

Action potentials are brief but large changes in membrane potential.
The originate in the axon hillock and are propagated at high speed along entire axon. Info sent to postsynaptic target it coded in its patterns of action potentials.

Hyperpolarization: increase in membrane potential. Increase negativity. Neuron becomes more negative on the inside compared to the outside. Could go from -65mV to -70mV.

Depolarization: decrease in membrane potential. Decreased polarization of cell membrane. Bring from -65mV closer to zero. Inside more like outside.

Action potential

The changes in membrane potential can send electrical signals through axon.

It originates in the axon hillock

Action potentials are produced by the movement of sodium ions into the cel. This is the peak of an action potential. The concentration gradient pushing Na+ into the cell equals the positive charge driving them out. The membrane shifts briefly from a resting state to an active state and back. The channel that this goes through is the voltage-gated Na+ channel. They open in response to initial depolarization. Tubular membrane spanning protein. More voltage-gated channels open and more Na+ ions enter. The channel is usually closed. The shape of the channel changes in order to open. This happens until the membrane potential reaches the Na+

equilibrium potential at +40mV. At this point the concentration gradient pushing Na+ ions into the cell is exactly balanced by the positive charge pushing them out. Goes from a potassium dependant resting state to a sodium dependant action state and then swiftly returning to the resting state. This occurs due to the voltage gated sodium channel. This pore is usually closed. When the cell membrane is depolarized to the threshold levels the channel changes shape and opens to let sodium pass through. Below threshold: closed pore. These channels only stay open for about a millisecond. At this point the membrane potential is near the sodium equilibrium of 40mV. Positive Charge inside nerve cell pushes K+ out the channels that remain open plus voltage gated K+ channels also open. This makes the membrane more permeable to K+ to restore resting potential.
Axons refractory to second stimulus. There are two phases to refractoriness.

1) Absolute refractory phase: brief period right after the production of an action potential because the voltage gated Na+ channels are unresponsive.

2) Relative refractory phase: only a very string stimulation can produce any other action potential because the flow of K+ ions out has temporarily hyperpolarized the neuron.
Action potentials limited to axons. Action potential is regenerated alone the length of the axon. Action potential is a spike of depolarizing electrical activity so it strongly depolarizes the next adjacent axon segment t. Because this segment is covered with voltage-gated Na+ channels, the depolarization immediately creates a new action potential, depolarizing the next patch of membrane, etc.

Large axons and the presence of myelin sheaths allow for higher conduction velocities.
Saltatory conduction: Action potential jumps from one node to the next.

Synapses

The action potential is converted into a chemical signal as the axon terminal releases a neurotransmitter. Neurotransmitters, aka synaptic transmitters is a chemical released from a presynaptic terminal that serves to communicated with a postsynaptic cell.
Neurotransmitters released into synapses briefly alters the membrane potential (postsynaptic potentials). Some of these generate excitatory post synaptic potentials while others produce inhibitory ones. It also depends what the receptor is present in the postsynaptic cell.

Synaptic inputs p75

September 25
Axonal membrane is refractory.

Conduction velocity is the speed of propagation of action potentials. It varies with diameter
Conduction velocities vary with:

-
-

Saltatory conduction: action potential jumps from node to node. At the nodes the depolarized sodium channels open which recreates the action potential at each nodes. Continues from node to node making it up to 15 times faster than unmyelinated axons. Myelinated axons, jumps so it goes faster.

What is the point of the neuron generated action potentials

the action potential is propagated down the axons and is recreated down each segment until it reaches the axon terminal. It is then. Converted to a chemical signal (neurotransmitters)

the transmitters releasing into synapses briefly change the membrane potential of the postsynaptic cell. This is postsynaptic potential. They spread passively across ____ decreasing in size and strength. Action potential has the all or none property. It either fires or does not. The amplitude of each action potential remains the same.

EPSP potential with the receiving neuron. Caused with excitatory connections. Increasing probability that the postsynaptic neuron will fire its own action potential.

when it fires it shows a normal potential. This causes a depolarization (EPSP) in postsynaptic neuron. Less negative

IPSP is hyperpolarizing (inside is becoming more negative) in postsynaptic neuron. Caused by inhibitory connections. Decreasing probability that postsynaptic neuron will fire and action potential. Because chloride ions enter the cell making it more negative on the inside.

when this fires it also shows normal action potential but it causes hyperpolarization in postsynaptic neuron. More negative. CHLORIDE

Excitatory and inhibitory neurons work in the same way but have opposite effects on postsynaptic neurons. why?

specific neurotransmitter being released by presynaptic neuron.
Some neurotransmitters generate EPSP and some generate IPSP. Balance between number of excitatory and inhibitory signals received decides this.
Postsynaptic neuron will fire action potential if a depolarization generating axon potential reaches the axon hillock. Integration of local potentials determine if the neuron will reach threshold and generate an action potential.

EXCITATORY: hyper polarizing. Neuron becomes more negative
INHIBITORY: depolarizing:
From the excitatory terminals. They're causing a depolarization over the cell body. This dissipates over the cell body and only a small amount reaches axon hillock. Need more than one synaptic input to reach the hillock to cause the action potential. If they both arrive at about the same time the sum of two depolarizations are enough to push the membrane potential to threshold to activate the action potential.
inhibitory terminals also active…postsynaptic hyperpolarization. Still have ex citatory terminals while others are inhibiting you see a spread of both in the cell body. They partially cancel each pother out. The effect is the difference between the two EPSP-IPSP. Hyperpolarization cancelled by depolarization. No action potential triggered bc threshold not reached. The resulting sum can be influenced by distance. Excitatory closer to axon hillock will produce a larger sum there compared to two terminals further from hillock. If the overall sum is sufficient to depolarize at axon hillock then the action potential will be triggered.

Spacial summation
Temporal summation is the summing of potentials that arrive at the hillock at different times
The closer they are in time the greater the summation and the

How does an electrical signal arriving a presynaptic terminal send chemical signal to post synaptic cell? **Synaptic Transmission**
Transfer of info across a synapse

1. Action potential travelling down axon reaches axon terminal
2. Depolarization opens voltage-gated calcium channels in membrane of the axis terminal allowing calcium to enter the terminal
3. The calcium causes synaptic vesicles filled with neurotransmitters to fuse with he presynaptic membrane and rupture causing the release of transmitter molecules into the synaptic cleft.
4. Transmitters cross cleft to bind to special receptor molecules in the postsynaptic membrane which opens ion channels in this membrane.
5. Creates local EPSP or IPSP in postsynaptic neuron.
6. Synaptic transmitter is:
    1. Inactivated (degraded) by enzymes
    2. Or removed from the synaptic cleft by transporters for reuptake and recycling so the transmission is brief and accurately reflects activity of presynaptic cell.

7. Synaptic transmitter may also activate presynaptic auto receptors which regulates/ decreases future transmitter release.

STEP THREE

When an action potential reaches presynaptic terminal sending out info it opens voltage gated calcium channels allowing for the influx of calcium. These calcium ions activate enzymes that cause the vesicles near the membrane to fuse with it and discard contents into cleft. This fusion with the presynaptic terminal membrane to release neurotransmitter into cleft is called exocytosis. The higher the frequency of action potential at terminal the greater the calcium influx and the more…..

Several specialized proteins mediate exocytosis. They can mediate ability for synaptic vesicles to fuse with membrane which then releases neurotransmitter into cleft.

One family is SNAREs they serve as tethers

v-SNAREs is the specialized protein that attaches to vesicles

t-SNAREs is a specialized protein that attach to presynaptic membrane

v attaches to t… vesicle now docked and ready for release

Other protein : synaptotagmin. Calcium sensor. When action potential arrives at terminal. Calcium incoming bind to and activate this protein. This then triggers final fusion of vesicle with presynaptic membrane which allows neurotransmitters to be released into cleft.

STEP SIX

The cessation of neurotransmitter effects depends on degradation or reuptake.

degration..neurotransmitter broke down and inactivated by special enzyme (deactivate acetylcholine is acetylcholinesterase)

Reuptake…clears neurotransmitters by taking them into terminal

transporters special receptors are located on terminal and bring transmitter back inside. Once taken back into terminal, neurotransmitters can be repackaged into presynaptic vesicles.

STEP SEVEN

Neurotransmitters might also activate presynaptic auto receptors which can regulate future synaptic release. Transmitters may bind do autoreceptors on presynaptic cell. Presynaptic membrane informed of concentration of release…

What about the chemical signal?

ch 4

transmitter in receptor. Key in lock. A molecule of right shape like ligand can fit into receptor and either activate it or block it. Ligands are any substance binding to receptor. They

can be endogenous (produced in body. Bound to receptor. Neurotransmitters and hormones) or exogenous (anything fitting into receptor that resides outside body. Pharmaceutical drug that fits into receptor to block it or activate it) Some neurotoxins can induce paralysis.
Receptors control ion channels in two ways:

inotropic receptors directly control ion channel. Can directly open it. when bound by transmitter it opens and ions flow across membrane. Neurotransmitter binds directly to protein. Channel opens immediately. ions flow across membrane for brief period of time

control in two ways

metabotropic doesn't directly control it. They activate g protein molecules. Neurotransmitter bings to g protein coupled receptor. G protein then activates. G protein subunit moves to adjacent ion channel cousin delay which allows it to open and allows for the ions to flow.

The number of receptors in cells can vary during development, daily in adulthood, with drugs

up-regulation: increase in number of receptors

down-regulation: decrease in number of receptors

Different ways of communicating.

1. Axon stimulating dendrite ()
2. Axon stimulating cell body ()
3. Form on axons near axon terminal. Axon synapsing on other axons. Allows presynaptic neuron to regulate how much transmitter will release from terminal ()
4. Some specialized dendrites synapse onto other dendrites. Allows for better coordination ()

Neural circuit: A neural chain is a simple series of neurons
Knee jerk reflex. Sensory neuron, motor neuron, single synapse that allows our sensory to communicate with out motor. A tap on patellar tendon stimulates stretch receptor in quads which starts events. Action potential speeds alone sensory neurons at about 100m/s. The a action potential in terminal causes release of transmitter glutimate. EPSP appears min motor neuron. Epsp spreads passively to hillock triggering more action potentials. This then speeds down large motor axon at ~100m/s. It then reaches neural muscular junction where acetylcholine Is released. This reflex happens ~40ms after initial hammer tap
Three factors

1)Sensory and motor axons myelinated an d large diameter…conduction velocity

2) sensory cells……

3) central synapse and junction both fast and inotropic. Directly controlling ion channels.