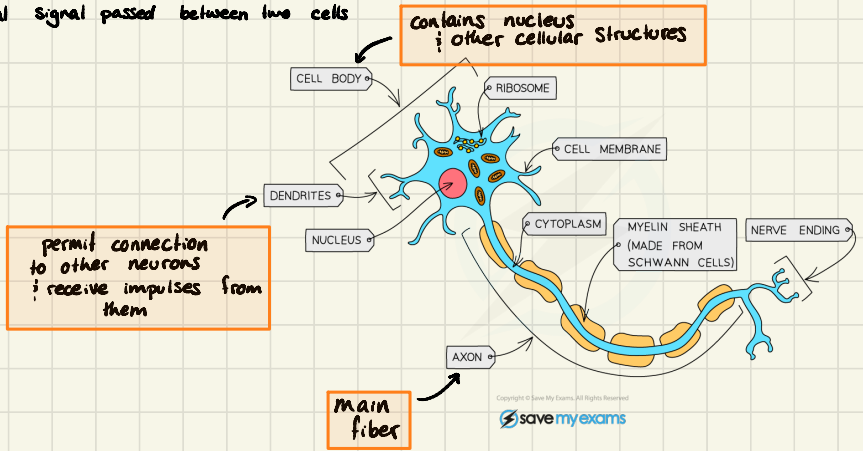


Neuron Structure

C2.2.1 - Neurons - cells within nervous system that carry electrical impulses

Nerve impulse → electrical signal passed between two cells

- Neuron - neuron
- Neuron - muscle
- Neuron - gland

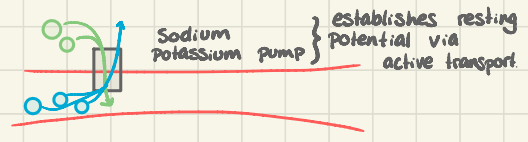


Movement of ions

C2.2.2 — Resting potential by pumping to create $[Na^+]_i$ $[K^+]_i$ gradients

Membrane potential => voltage created by an imbalance of charges (ions) on either side of the membrane

- Relatively (+/-) charge
- At rest:
 - a) Inside neurons = relatively negative
 - b) -70 mV
 - c) Sodium ions are outside cell

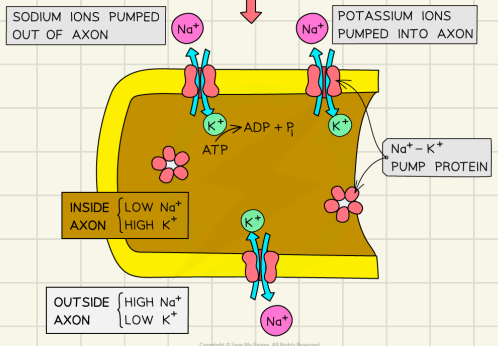
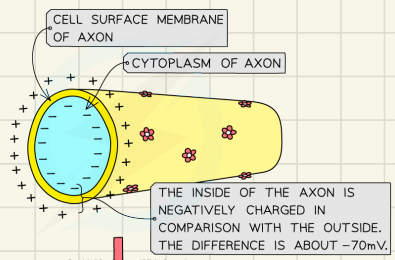
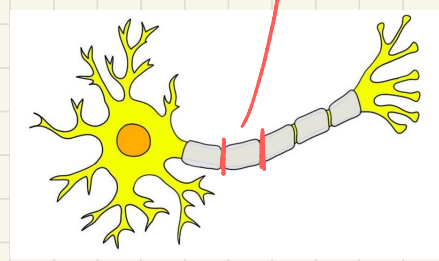
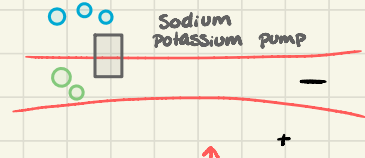


3 Na^+ out

2 K^+ in

↳ bc of generated gradient
 1. Na^+ will flow back in
 2. K^+ will flow out by facilitated diffusion
 ↳ K^+ can flow out w/ + ease, so more (+) ions will be outside neuron
 ↳ inside @ -70mV

At Rest:



Combine:

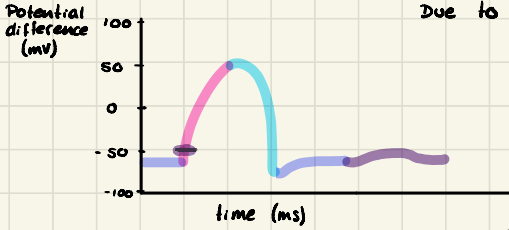
- 1. 3 + charges leave and only 2 enter
 - 2. $[]$ gradients established and now K^+ ions diffuse back out w/ more ease than Na^+ can come in
- Causes resting potential of -70mV

→ C2.2.8

C2.2.3 — Nerve impulses = action potentials that are propagated along nerve fibers

1. Depolarization ⇒ membrane potential goes from negative to positive

2. Repolarization ⇒ membrane potential goes from positive back to negative



Due to movement of ions during an action potential

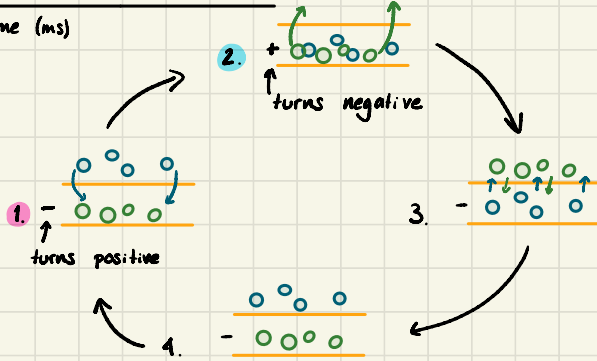


Threshold potential →

the membrane potential that must be reached in order for the voltage-gated ion channels to open [-50 mV]

↳ if this is not reached voltage-gated Na⁺ channels do not open.

↳ no action potential, resting potential continues

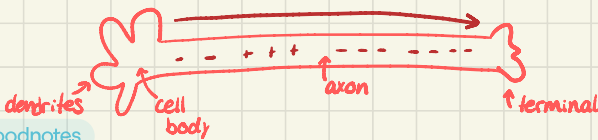


This is what ends up happening. But the ions should be the other way around. ↳ that is the job of the sodium-potassium pump

Steps:

1. Voltage-gated sodium ion channels open [when stimulus is strong enough]
2. Sodium ions diffuse into cell (facilitated diffusion) - There is a higher [] of Na⁺ out
3. Depolarization (-) to (+)
4. Voltage-gated sodium channels close ; voltage-gated potassium ion channels open - There is a higher [] of K⁺ in
5. Potassium ions diffuse out of the cell (facilitated diffusion)
6. Repolarization (+) to (-)
7. Sodium-potassium pump re-establishes resting potential by actively pumping K⁺ in ; Na⁺ out
ATP

Self-propagating → depolarization in one part triggers depolarization in the next part, due to the opening of voltage-gated channels

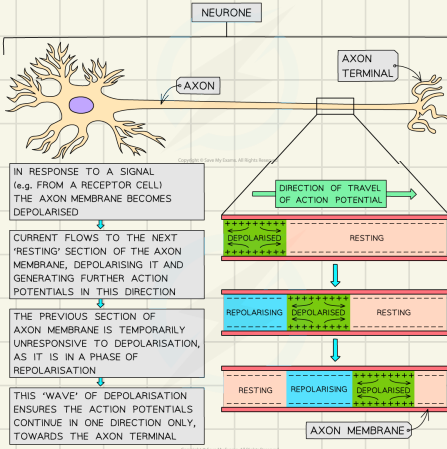


C2.2.9 — Propagation of action potential along nerve fiber → result of local currents

Local Current → the movement of Na^+ between polarised and depolarised regions

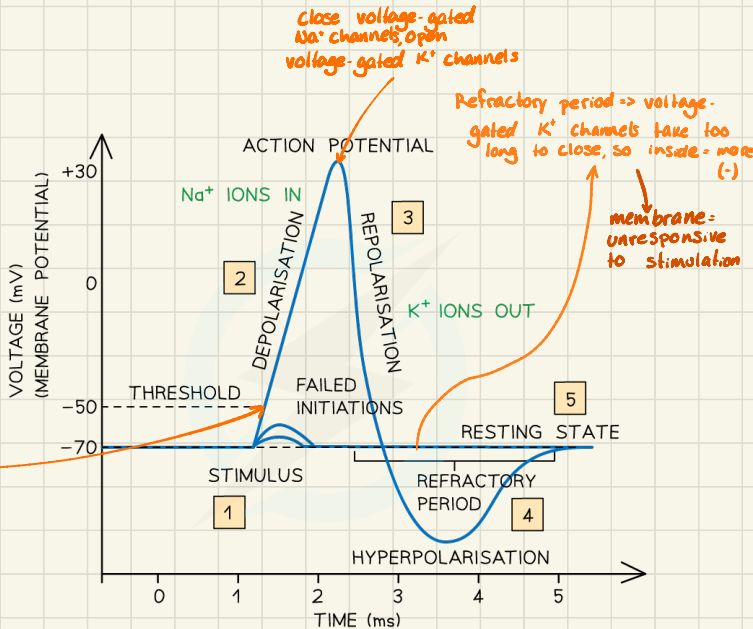
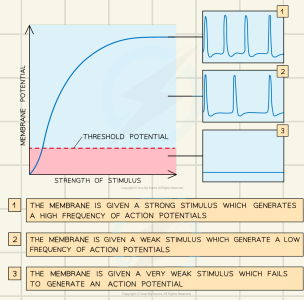
- Movement of sodium ions into cell in one part causes depolarisation (i.e. movement of Na^+) in the next part of the cell

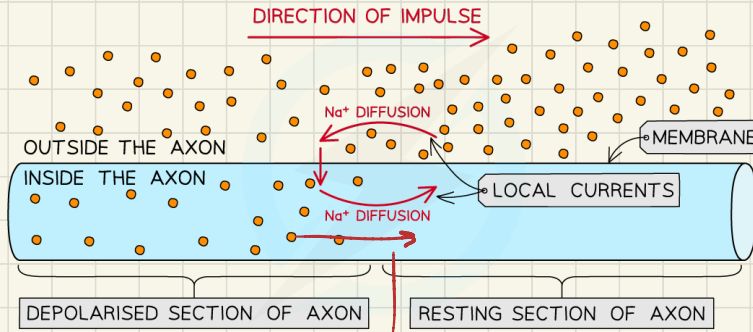
o:
local currents cause each successive part of the axon to reach the threshold potential o:



All-or-nothing principle:
if threshold potential = reached, action potential will ensue.
↳ no such thing as a small/large action potential

→ "strong" stimulus may cause + frequency of action potentials





The diffusion within the axon causes the next area to reach threshold voltage (from -70 to -50) and open the voltage-gated Na⁺ channels which causes further depolarization

C2.2.10 — Oscilloscopes

Oscilloscope → measures membrane potentials using electrodes
differences in charges

C2.2.11

Neuron's structure effects impulse speed

C2.2.4 - Variation in speed of nerve impulses

- Average speed for nerve impulses = 1 m/s

↳ Ways to speed this up:

1. Increase diameter

- i) Decrease resistance to flow
- ii) Signal may travel more quickly

2. Myelination

- i) Myelinated neurons have a myelin sheath
- ii) composed of Schwann cells acts as electrical insulator, impulses cannot pass through sheath
- iii) Allows for saltatory conduction

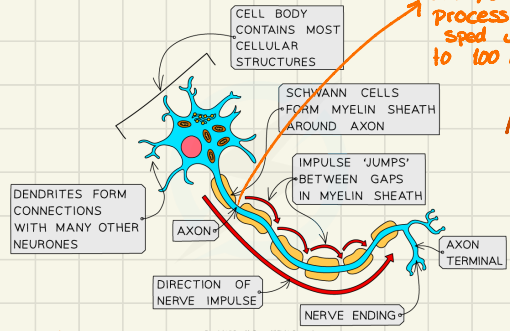
Saltatory conduction => jumping of the nerve impulse from node to node in myelinated neurons

Fat layers in Schwann cells act like insulators, so local currents are carried from node to node and the exchange of ions only needs to occur at the nodes instead of along the whole axon.

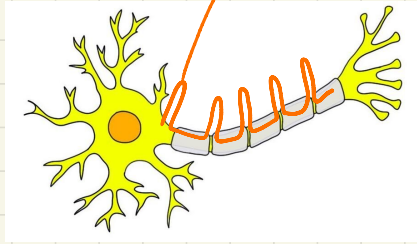
↳ Lowers ATP consumption

↳ Increases speed to 100 m/s

Na⁺ ; K⁺ exchange only @ nodes, not along the whole axon, so process-spiced up to 100 m/s



only get depolarization ; repolarization @ ranvier nodes



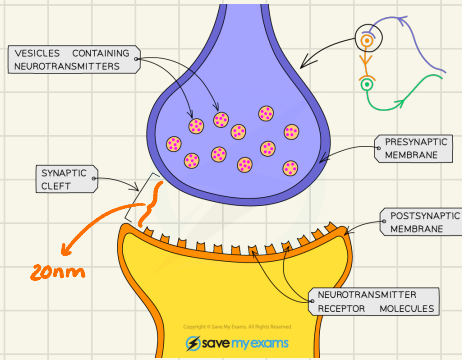
Neuron passing signal to next cell...

C2.2.5 — Synapses as junctions between neurons ; between neurons ; effector cells

Synapse → gap between cells through which signals are passed by neurotransmitters

- Can be:

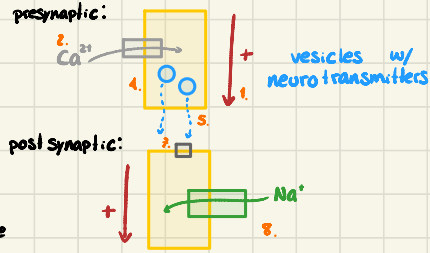
- neuron → sensory organ
- neuron → neuron
- neuron → effector (muscle)



C2.2.6 — Release of neurotransmitter from pre-synaptic membrane

- Steps:

- Action potential reaches the end of presynaptic neuron (+)
- Voltage-gated calcium ion channels open (Ca^{2+})
- Ca^{2+} enter presynaptic neurons (facilitated diffusion)
- Ca^{2+} force vesicles with neurotransmitters to fuse w/ membrane
- Neurotransmitters = released into synapse (exocytosis)
- Neurotransmitters diffuse across the synapse
- Neurotransmitters bind to receptors on postsynaptic membrane [Depending on type of cell, this receptor might also act as ion channel]
- Ion channels open
- If enough ions enter postsynaptic cell, that generates action potential
- Neurotransmitter = removed from synapse



↳ we do not want a continued message

1. Re-pumped
back into presynaptic
neuron

2. Enzyme that moves
into synapse ;
destroys neurotransmitters

Ca^{2+} functions as chemical signal triggering exocytosis of neurotransmitter from presynaptic cell

C.2.2.7 — Generation of an excitatory postsynaptic potential

- Acetylcholine

↳ Neurotransmitter between motor neurons & muscles

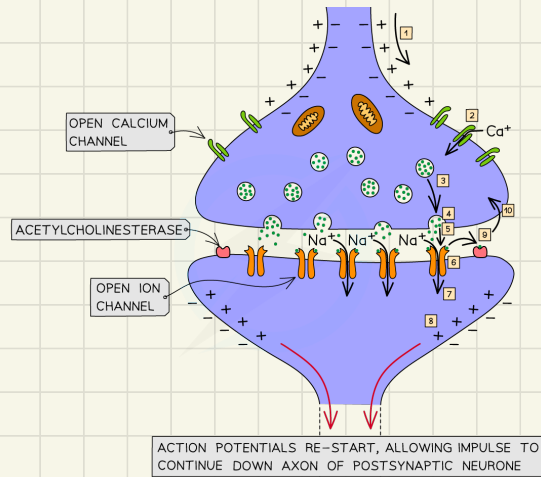
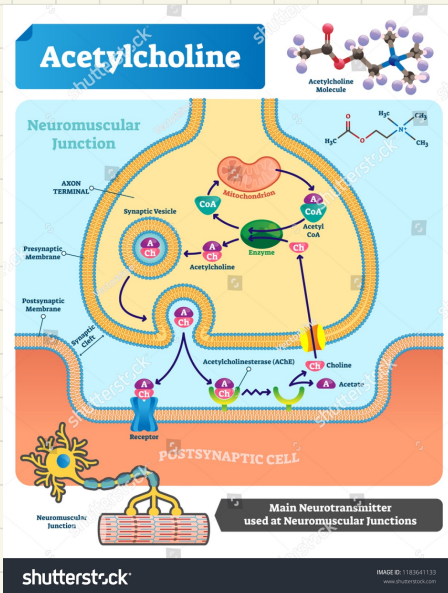
- Responsible for carrying messages between the 2
- Muscle-contraction neurotransmitter

1. When it binds to receptors on muscles, they contract

- We don't want muscle to contract permanently so an enzyme is required to break acetylcholine down

2. Enzyme acetylcholinesterase break acetylcholine into acetyl & choline

3. Choline is reabsorbed back into presynaptic neuron to make more acetylcholine



- | | | | |
|---|---|----|--|
| 1 | ACTION POTENTIAL ARRIVES, DEPolarISING PRESYNAPTIC MEMBRANE | 6 | ACh BINDS TO RECEPTOR PROTEINS |
| 2 | CALCIUM ION CHANNEL PROTEINS OPEN. CALCIUM IONS DIFFUSE IN | 7 | RECEPTOR PROTEINS OPEN. SODIUM IONS DIFFUSE THROUGH. |
| 3 | PRESYNAPTIC VESICLES FUSE WITH MEMBRANE | 8 | POSTSYNAPTIC MEMBRANE IS DEPolarISED |
| 4 | ACh RELEASED | 9 | ACh BROKEN DOWN INTO ACETATE AND CHOLINE BY ACETYLCHOLINESTERASE |
| 5 | ACh DIFFUSES ACROSS SYNAPTIC CLEFT | 10 | CHOLINE RECYCLED INTO ACh |

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Inhibitory & excitatory neurotransmitters + their effects

C2.2.13 - Inhibitory neurotransmitters & generation of inhibitory postsynaptic potentials

Excitatory neurotransmitters => those which result in the generation of action potential

Inhibit => prevent

- Some neurotransmitters make the membrane even more negative [harder for nerve impulses to be sent out]

↳ for ex.:

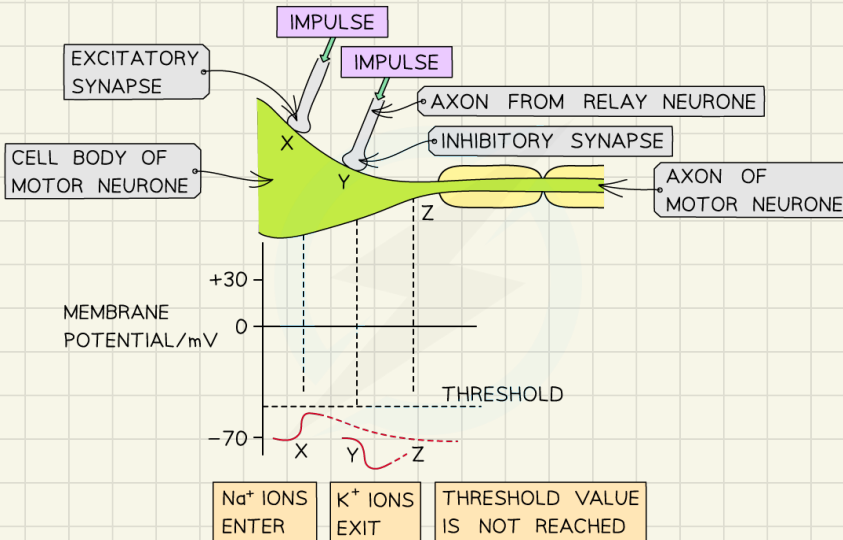
by opening voltage-gated K^+ channels & allowing them to diffuse out of cell \therefore making it even more negative!

Ex:

excitatory neurotransmitter	inhibitory neurotransmitter
Acetylcholine	Gaba

↳ This causes:

1. hyperpolarization
2. Threshold not to be reached even w/ stimulus

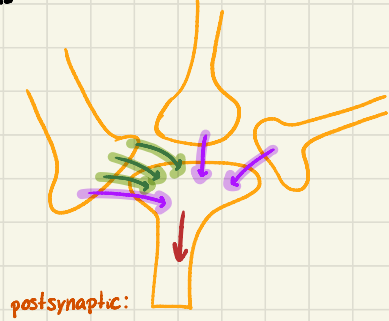


This does not usually happen if action potential is not reached } effects = canceled out

C2.2.14 - Summation of ex/inhibitory neurotransmitter's effects

presynaptic:

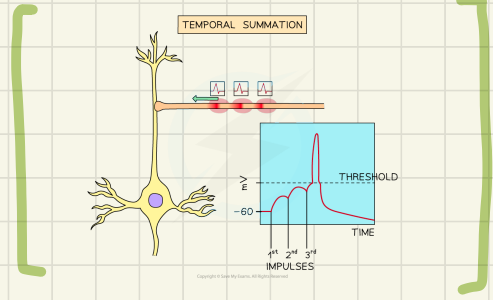
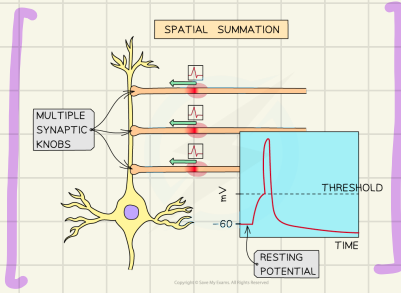
- Some synapses have many presynaptic neurons
- An **excitatory** neurotransmitter from one presynaptic neuron is usually not enough to reach threshold potential in the postsynaptic neuron



Summation => when multiple releases of an excitatory neurotransmitter are needed to cause an **action potential** in postsynaptic neuron will only ensue under 1 of 2 conditions

Excitatory neurotransmitters from several neurons → **Spatial summation**

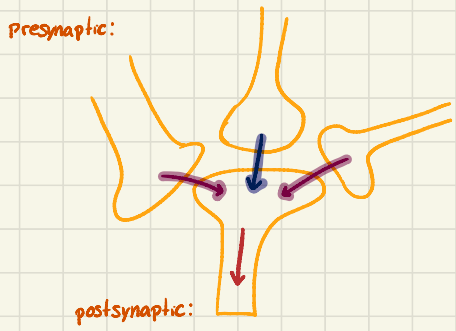
Several excitatory neurotransmitters from one neuron → **Temporal summation**



What if: conflicting messages...
 some inhibit → some excitatory

Excitatory neurotransmitter must outnumber inhibitory neurotransmitters in order to reach threshold potential

This is how central nervous system makes decisions } Summation is interpreting this sort of conflicting information



Pressure/ Pain triggering neurons

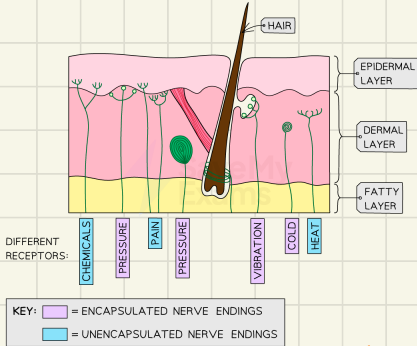
C2.2.15 - Perception of pain by neurons w/ free nerve endings in skin

- **sensory neurons** have endings on skin (**unencapsulated**)

1. An action potential is initiated in response to pain
2. Carries the impulse to the spinal column, then to the brain

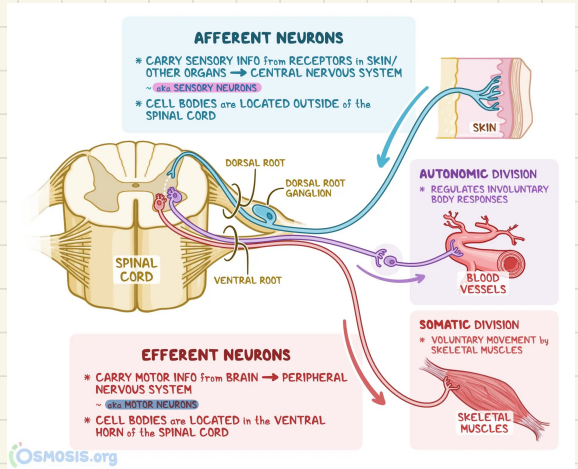
i) If enough **excitatory neurotransmitters** are being passed by **sensory neurons** to the **central nervous system**:

↳ Brain sends an impulse along **motor neurons** to affect behaviour



stimuli that trigger pain response:

1. Heat (high temp)
2. Chemicals
3. Acid



New properties on cells that did not possess that attribute

C2.2.16 - Consciousness = property that emerges from interaction of individual neurons in brain

Consciousness => simultaneous awareness of many things

↓
reduced consciousness
(sleep)

↓
unconsciousness
(anesthesia)

This is an **emergent property** (arises from the interaction between dif. neurons)

↳ Characteristic that arises from the interaction of individual components within a system