Information is transmitted from one end of a neuron to the other via electrical signals.

Each individual body cell is electrically neutral. This means that the number of positively charged ions (**cations**) is equal to the number of negatively charged ions (**anions**).

However, there can be certain areas within or around a cell where one type of ion predominates.
This occurs at a neuron's plasma membrane.
There is a slight local imbalance of charge between the inside and the outside of the plasma membrane.
The inside of the plasma membrane is slightly negative to the outside of the plasma membrane, i.e. the inside has more negative charges or the outside has more positive charges.
Such a separation of charge is a source of potential energy – remember that opposite charges are attracted to one another.
The potential energy created by the separation of charge is known as a **voltage**.
The greater the difference in charge, the greater the voltage.
The voltage created by the separation of charge at the plasma membrane is the **membrane potential**.

The membrane potential exists because the ions that are inside or outside the cell cannot flow towards one another through the plasma membrane and redistribute themselves in a balanced way.
In other words, the plasma membrane is a source of **resistance** to the flow of ions.
Such a flow of ions (were it to happen) would be an example of an **electrical current**.

Neurons have a membrane potential.
When the neuron is not signaling, its membrane potential is the **resting membrane potential**.
The voltage of the neuron's RMP is usually -70mV.
B/c of the RMP, neurons are said to be **polarized**.

This RMP exists because of a few main factors:
Sodium-potassium pumps are integral proteins abundant in the neuronal plasma membrane. Each is constantly breaking down a molecule of ATP and using the energy to move 3 Na+ from the inside of the cell to the outside and 2 K+ from the outside of the cell to the inside. Notice how this sets up an imbalance of charge which results in the cell interior being more negative than the cell exterior. The NA/K pump maintains high extracellular levels of sodium and high intracellular levels of potassium.
Potassium leak channels are integral proteins that allow potassium to flow from the cell interior to the exterior (i.e., down the K+ concentration gradient). This efflux of K+ continues until the tendency for K+ to move outward is balanced by the tendency of K+ to stay within in the cell due to the buildup of a negative voltage in the cell interior.

The neuron RMP is also affected by the presence of **sodium leak channels** which allow a small amount of sodium to flow down its concentration gradient and enter the cell.

Electrical signals travel from one end of a neuron to the other and are based upon altering the RMP.
A change in the RMP that decreases the charge difference between the inside and the outside of the cell is a **depolarization**. A change in the RMP that increases the charge difference between the inside and the outside of the cell is known as a **hyperpolarization**.

There are 2 basic means of electrical signaling within a neuron.
They are **graded potentials** and **action potentials**. Both rely on altering the RMP.

A graded potential is a local change in the RMP.
Suppose a sensory neuron releases a neurotransmitter adjacent to the soma of an interneuron.
The NT will bind to an integral protein in the soma's plasma membrane known as a **receptor**.
The receptor is actually a **chemically-gated ion channel**.
The binding of the NT causes the channel to open.
Ions then move through the channel. The primary ion is Na+, which is entering the cell.
Na+ entry decreases the local RMP and that particular region of the soma is depolarized.
The entry of Na+ will "push" potassium ions at this locale away from the site of Na+ influx.
Thus, neighboring regions of the soma membrane will become more positive and will have depolarized.
This will continue; however the spread of depolarization will diminish with distance as most of the ions spreading away from the initial site will leave the cell via the abundant K+ leak channels in the PM.
Note that the distance traveled by a graded potential depends on the initial amount of Na+ influx.

An action potential on the other hand does not decay with distance.
Suppose a graded potential reaches the axon hillock and depolarizes the membrane there.
If the graded potential is of enough magnitude, it will depolarize the axon hillock membrane to a point known as **threshold**.
Threshold is the membrane potential at which **voltage-gated sodium channels** open.
V-gated Na+ channels are integral proteins in the axon plasma membrane that are normally "closed", i.e. they bar Na+ from passing.
However, at the threshold voltage, the channels change their shape and open. Na+ then rushes in and depolarizes that area of membrane.
This depolarization will cause adjacent areas to depolarize and their v-gated Na+ channels will open.
Sodium will then rush in at this point and depolarize this new area of membrane which will cause more adjacent v-gated Na+ channels to open.
This continues down the length of the axon. The sequential opening of v-gated Na+ channels allows the wave of depolarization to move from the axon hillock to the axon terminals.

However the v-gated Na+ channels only remain open for 1ms. Then they will close again.
Thus the depolarization phase is brief. But it is long enough to allow for the influx of enough sodium so that adjacent v-gated Na channels will be stimulated.
By the time a v-gated sodium channel has closed, a neighboring voltage-gated potassium channel will have opened. This results in the efflux of K+ from the cell.
The movement of K+ from the cell will make the interior of the cell more negative and thus move the membrane potential back towards its initial resting value.
The egress of K+ is thus responsible for the **repolarization** of the membrane.
The repolarization wave will travel down the axon from the hillock to the terminals shortly after the depolarization wave.
This allows the axon membrane to return to the resting membrane potential so that it may conduct another electrical signal.

The wave of depolarization and repolarization travelling down the axon is an action potential.

Note that the v-gated K+ channels stay open a bit too long. This causes the membrane potential to become more negative than the normal RMP). This is known as **hyperpolarization**.
The RMP is restored by the action of the Na/K pump.

The action potential is self-propagating (since it causes the continual opening of adjacent v-gated sodium channels)
It is also unidirectional. It travels from the axon hillock towards the axon terminals.
This is because there exists a brief period following the closure of a v-gated sodium channel where it cannot reopen. Thus the depolarization wave can only open v-gated channels once and cannot reopen them and travel back towards the hillock. This period of v-gated sodium channel inactivity is known as the **refractory period**.

The action potential is an **"all or none event"**. It either happens completely and totally or not at all.
If threshold is reached, the action potential will occur. If threshold is not reached, no action potential will take place.
Thus no information can be encoded by the "size of an action potential."
Instead information is encoded by the frequency of action potentials.

The speed of action potential propagation depends on whether the axon is myelinated or unmyelinated.
In unmyelinated axons, the wave of depolarization travels sequentially down the axon as every bit of membrane is depolarized in turn. This type of propagation is known as **continuous conduction**.
In myelinated axons, the depolarization only occurs at those areas of the axolemma that are not covered by the myelin sheath (nodes of Ranvier). This type of propagation is known as **saltatory conduction**.

Information is sent from one end of a neuron to the other via electrical signals – primarily action potentials. But how is a signal sent from one neuron to another or from one neuron to an effector cell?

Recall that an axon terminal will abut either the dendrites or soma of another neuron or an effector cell.
This junction is known as a **synapse**. The space between the 2 cells is the **synaptic cleft**.
The cell that is the "sender of information" is the **presynaptic cell**.
The cell receiving the signal is the **postsynaptic cell**.
Imagine an action potential traveling down an axon.
The wave of depolarization begins at the axon hillock and travels to the axon terminals.
When the membrane of the axon terminal is depolarized, v-gated calcium channels will open.
This results in the influx of calcium into the interior of the axon terminal.
Recall that axon terminals are full of **synaptic vesicles** carrying **neurotransmitters**.
The influx of calcium causes the exocytosis of synaptic vesicles – releasing NTs into the synaptic cleft.
The NTs will then diffuse to the postsynaptic cell membrane and bind to receptors there.
The resulting action will depend on the nature of the postsynaptic cell: If it is a muscle cell it will contract; A gland cell will secrete something; And another neuron will generate a graded potential.

If the postsynaptic cell is another neuron, then the binding of the neurotransmitter will result in the opening of a chemically-gated ion channel and the initiation of a graded potential.
In this case, the graded potential is referred to a **postsynaptic potential**.
If the PSP is a local depolarization it is referred to as an **excitatory postsynaptic potential** (**EPSP**).
It's excitatory since a depolarization will move a membrane closer to the threshold potential.
If the PSP is a local hyperpolarization (e.g., if chemically-gated Cl- channels were opened and Cl- entered the cell) it is referred to as an **inhibitory postsynaptic potential** (**IPSP**).
It's inhibitory since a hyperpolarization will move a membrane farther from the threshold potential.
Normally a neuron will receive countless numbers of EPSP's and IPSP's.
The net effect of them on the cell's membrane potential will determine whether the cell reaches threshold and if an action potential is fired. Such integration is known as **summation**.

Summation comes in 2 flavors.
Spatial summation occurs when the postsynaptic neuron integrates PSP's received at the same time from different presynaptic cells.
Temporal summation occurs when the postsynaptic neuron integrates sequential PSP's from the same group of presynaptic neurons.
Usually a combination of these summation events is occurring.

The postsynaptic cell is influenced by the presynaptic cell for as long as the presynaptic cell releases NTs.
Neurotransmitters do not remain in the synaptic cleft for long.
They may be degraded by enzymes in the synaptic cleft.
They may be taken up by the presynaptic axon terminal or by glial cells.
Or they may simply diffuse away from the synapse into the surrounding extracellular fluid.