NOTE FROM SLIDE

Membrane potential and action potential

اخواني هاي ملاحظات موجود تحت بعض السلايدات من المحاضرة طبعا موجودة بالسلايد الالكتروني فقط ، فجمعنالكم اياها بملف pdf .

<u>Slide 4 :</u>

This voltage has been found to exist on every living cell plant, bacteria, skin;;;;; and this called resting membrane potential. It is called cell membrane because this voltage is found on the cell membrane.

In some cells this voltage can change in excitable cells, however in other cells it stays constant it doesn't change. Is that small or not? Now to understand this we have said that the typical flash battery is1.5 V now let us say 1000mV= 1v according to metric system now let us round 70 mV into 100 Mv that would 0.1V which is less than the 1.5 but we have said that every single cell in your body is 0.1 multiply this number by 60 trillions. The amount of electrical energy in your body is amazing this electrical energy is basis of the powering our nervous system, muscle and our heart and it is the amount of what they are recording EMG, EEG, ECG.

<u>Slide 5 :</u>

The cell membrane of our body is semi-permeable is permeable to some chemicals than others. Now there are ion channels; normally all these channels are normally closed except for the potassium ion channels so the sodium ion channels are normally closed.

Now the sodium ion channels is normally closed; the sodium ion like to go down its concentration gradient but it couldn't because the sodium ion channels. Not only sodium channel but most other channels are closed (inions).

The major source negative charge inside the cell is protein. All the ions like to diffuse out or inside the cell but they couldn't because of the channel only the k can diffuse out because of the higher conc inside the cell.

As the k diffuses out due to the conc; inside the cell becomes negative and outside the cell is positive. Now every time positive charge diffuse outside the cell it makes the inside negative and the outside positive.

In biology we learned that In diffusion every chemical diffuse until equalizes every where but that is not going to happen why? Now in case of oxygen and carbon dioxide they don't have charges or their charges wouldn't affect their movement they will continue to diffuse until they equilibrium occur on both sides of the cell.

But in case of k if the potassium didn't have a charge it would move until for example 50 k inside and 50 k outside. But because the potassium has a charge it will first move according to con gradient outside the cell and as the k diffuses outside the cell a negative charge inside the cell would increase and form anther force called electrical force which is going to draw the k inside the cell thus eventually the k would stop to diffuse out because the electrical force that draw the k back into the cell would be equal to the con gradient that draws the k outside the cell .

Thus the k would be equal at both sides of the cell because of this two forces; concentration gradient and electrochemical force (equilibrium state). The force pushing the k outside the conc gradient is equal to the force draw potassium in (-90V).

But what about sodium potassium pump ? If the pump kick one potassium in and one sodium out that want change the polarity. The resting membrane exists in all your cell body and the time they changes in nerve and muscle cells they are going to change because there are other sodium channels can open up.

<u>Slide 10 :</u>

The Nerst equation can tell us what the voltage is for any battery. If you multiply all these constant it becomes 60. Now we have 60 times conc K out/ conc K in log 30 the answer is 88mV; (no net flow)thus a change in potassium con outside the cell changes the voltage of the cell.

<u>Slide 16 :</u>

The muscle and nerve cells are not like usual cells or round cells; these cells are fibers they are the only cells in our body called fibers. There are two never cells mylinated and unmylinated; all the muscle cells are unmylinted.

The action potential in unmylinated cells . A s the action potential travels along the nerve fibre the sodium channels start to open and replicate again and again along the nerve fibre and the repolarization will begin to develop in first depolarized area just before the adjacent area and the term is given here is propagation .

The action potential in myleinated fibre (oligodendrities if it around interneuron) and if it is around sensory and motor cells (Shawn cells).

The action potential along the myleinated nerve fibre is only occurring at the nodes of Ranvier this kind of conduction is called salutatory conduction (jumping conduction) thus it is faster than unmyleinated conduction.

How fast is the action potential in our living cells? Let us compare electrical conduction in a wire which is about 186000miles/sec and in the action potential is 100min/sec in thickest myleinated nerve cell ; in the wire it is electrons moving through a copper wire while in the cells it is moving through cytoplasm thus it is not even comparable.

Multiple sclerosis (MS) your immune cells attacks your myleinated cells; slowing the action potential in your body every thing is slowing; it involves degeneration of oligodendrities and schwan cells and replaced by fiber tissue (scar tissue at any injured place you have scar tissue) this action potential starts to slow down and eventually ceases. immunosuppressant (corticosteroids) as prednisone.

<u>Slide 18 :</u>

It is a short lasting reversal in the electrical polarity of the excitable cell.

Every cell has a membrane potential but only excitable cell; muscle and nerve can exhibit a reversal in electrical polarity. Where it goes for being negative on the inside to positive outside and positive outside to negative inside. And this acts like a signal that can be conducting along these nerve or muscle fibers.

The ion channels always open in all cells are potassium channels but in case of excitable cells nerve and muscle cells there is a momentary and briefly open up of sodium channels and when sodium start to flow in the cell that is going to change the polarity of the cell.

In depolarization where the cell becomes less polarized or less negative (what normally stimulated cells in our body is chemical (neurotransmitter)).

Repolarisation once the sodium channels rush into the cell they trapped on the inside because the sodium channels have closed but what happens there is addition sodium channels open up (voltage legand gated ion channels because what always opens a gate is a neurotransmitter) and the potassium ions rush out of the cell because of the conc gradient and the electrical force are pushing out and that is unlike resting membrane potential where the electrical gradient is on the opposite direction of positive gradient.

Depolarization goes from -70v to +30v and in repolarization more negative than positive and there is always over shoot that become more negative than it started out and eventually it be a kind of reorganize it self by sodium&

potassium pump in less than 10msec (Na/K doesn't affect the polarity)and it turn back to be normal electrical resting potential.

Let us say 1 action potential (AP) can generate in 10msec then in 1sec we can generate 1000 AP. Threshold is the maximum point where the stimulated nerve can cause an action potential by opening many sodium channels before that it is sub threshold. But what about super threshold this will stimulate more than action potential in row this is the way of our nervous system to communicate.

<u>Slide 19 :</u>

Voltage-gated Na+ channels have three main conformational states: closed, open and inactivated. Forward/back transitions between these states are correspondingly referred to as activation/deactivation (between open and closed, respectively), inactivation/reactivation (between inactivated and open, respectively), and recovery from inactivation/closed-state inactivation (between inactivated and closed, respectively). Closed and inactivated states are ion impermeable.

Before an action potential occurs, the axonal membrane is at its normal resting potential, and Na+ channels are in their deactivated state, blocked on the extracellular side by their activation gates.

In response to an electric current (in this case, an action potential), the activation gates open, allowing positively charged Na+ ions to flow into the neuron through the channels, and causing the voltage across the neuronal membrane to increase. Because the voltage across the membrane is initially negative, as its voltage increases to and past zero, it is said to depolarize. This increase in voltage constitutes the rising phase of an action potential.

At the peak of the action potential, when enough Na+ has entered the neuron and the membrane's potential has become high enough, the Na+ channels inactivate themselves by closing their inactivation gates. Closure of the inactivation gate causes Na+ flow through the channel to stop, which in turn causes the membrane potential to stop rising.

With its inactivation gate closed, the channel is said to be inactivated. With the Na+ channel no longer contributing to the membrane potential, the potential decreases back to its resting potential as the neuron repolarizes and subsequently hyperpolarizes itself. This decrease in voltage constitutes the falling phase of the action potential.

When the membrane's voltage becomes low enough, the inactivation gate reopens and the activation gate closes in a process called deinactivation. With the activation gate closed and the inactivation gate open, the Na+ channel is once again in its deactivated state, and is ready to participate in another action potential.

When any kind of ion channel does not inactivate itself, it is said to be persistently (or tonically) active. Some kinds of ion channels are naturally persistently active. However, genetic mutations that cause persistent activity in other channels can cause disease by creating excessive activity of certain kinds of neurons.

Mutations that interfere with Na+ channel inactivation can contribute to cardiovascular diseases or epileptic seizures by window currents, which can cause muscle and/or nerve cells to become over-excited.

<u>Slide 20 :</u>

One way of monitoring physical activity intensity is to determine whether a person's pulse or heart rate is within the target zone during physical activity.

For moderate-intensity physical activity, a person's target heart rate should be 50 to 70% of his or her maximum heart rate. This maximum rate is based on the person's age. An estimate of a person's maximum age-related heart rate can be obtained by subtracting the person's age from 220. For example, for a 50-year-old person, the estimated maximum age-related heart rate would be calculated as 220 - 50 years = 170 beats per minute (bpm). The 50% and 70% levels would be:

50% level: 170 x 0.50 = 85 bpm, and

70% level: 170 x 0.70 = 119 bpm

Thus, moderate-intensity physical activity for a 50-year-old person will require that the heart rate remains between 85 and 119 bpm during physical activity.

For vigorous-intensity physical activity, a person's target heart rate should be 70 to 85% of his or her maximum heart rate. To calculate this range, follow the same formula as used above, except change "50 and 70%" to "70 and 85%". For example, for a 35-year-old person, the estimated maximum age-related heart rate would be calculated as 220 - 35 years = 185 beats per minute (bpm). The 70% and 85% levels would be:

70% level: 185 x 0.70 = 130 bpm, and 85% level: 185 x 0.85 = 157 bpm

Thus, vigorous-intensity physical activity for a 35-year-old person will require that the heart rate remains between 130 and 157 bpm during physical activity.

<u>Slide 21 :</u>

the clinical related part is hypokalemic which developed by anorexia (the people who dont eat) or bulimia (when somebody eats and enforce himself to vomit). the other cause is diuretics (increase urine output) given to people with edema high blood have pressure (80%) of people who are on diuretics). hyperaldosteronism (hormone regulates sodium and potassium). the fourth reason and there are more excessive vomiting and diarrhea especially in infants.

<u>Slide 22 :</u>

Hyperkalemia caused by kidney failure as your kidney would be able to excrete excess amount of k anther cause is hypoaldosteronism a hormone that regulates your k level in the blood and third cause and there are more is excessive iv drop of k chloride instead of sodium chloride.

The treatment is much more difficult than the hypo one; one of the treatments is chelates and oxalate; it likes a jelly used to draw k from the blood stream into the elementary or put the patient on haemodialysis (artificial kidney). put a shunt on a patient arm and connect it into artificial machine to filter all excess electrolytes in the blood, it takes hours and they have to do it every other day.