Notes

Sonntag, 21. Januar 2018 12:47

More Na+ Channels at some place means lower threshhold for generating a action potential there.

Potentials
Equilibrium potential: The potential for which for a specific Ion, the flow through open channels is 0.
Reversal potential: same thing
Resting potential of the cell: The potential of the membrane when quiescent, counting all equilibrium potentials. This is the same as the reversal potential of the leakage current. The reversal potential of the leakage flow equals the resting potential of the cell.
The membrane potential of a cell is (see Goldman-Katz)

Nernst Equation

$$E_{ion} = \frac{RT}{zF} \cdot \ln\left(\frac{[K^+]_{extracellular}}{[K^+]_{intracellular}}\right)$$

E is the reversal potential of the specific ion type (K here).
Assuming room temperature:

$$\approx \frac{58mV}{z} \cdot \log_{10}\left(\frac{Ion_{extra}}{Ion_{intra}}\right)$$

V out is above the line.

Goldman-Hodgkin-Katz
The membrane potential is determined by the permeability of the membrane for certain Ions.

$$V_{membrane} = \frac{RT}{F} \cdot \ln\left(\frac{P_K \cdot [K^+]_{out} + P_{Na} \cdot [Na^+]_{out} + P_{Cl} \cdot [Cl^-]_{in}}{P_K \cdot [K^+]_{in} + P_{Na} \cdot [Na^+]_{in} + P_{Cl} \cdot [Cl^-]_{out}}\right)$$

If one Ion has way more permeability, it dominates the membrane potential.

Circuit Laws
Ohm's Law: $V = I \cdot R$
Kirchhoffs Current Law: The sum of all currents entering and leaving any node is zero.
Kirchhoffs Voltage Law: The sum of all voltages around a closed loop is equal to zero.
"Steady-State Condition": The sum of currents is zero, we can neglect the cell capacitance C_{mem}

$$I_{ion} = g_{ion} \cdot (V_m - E_{ion})$$

Hodgkin-Huxley Model
Describes how action potentials are generated.

$$I_{ionic} = I_{Leakage} + I_K + I_{Na} + \cdots = g_L(V_m - E_L) + \cdots$$

$$I_{ionic} = \underbrace{g_L(V_m - E_L)}_{I_L} + \underbrace{\overline{g_K} \cdot n^4 \cdot (V_m - E_K)}_{I_K} + \underbrace{\overline{g_{Na}} \cdot m^3 h \cdot (V_m - E_{Na})}_{I_{Na}}$$

The leakage current is constant
The conductance g can depend on time and can be written as $g_{\backslash \max} \cdot n,\ n \in [0,1]$.
We could also model a channel as a sequence of serial gates that are open or closed. Let n be the probability for a gate to be open. Then we could use $g_{\backslash \max} \cdot n^4, n \in [0,1]$ and the potence of n is still between 0 and 1. We take the power 4 because that is nice according to Hodgkin-Huxley.

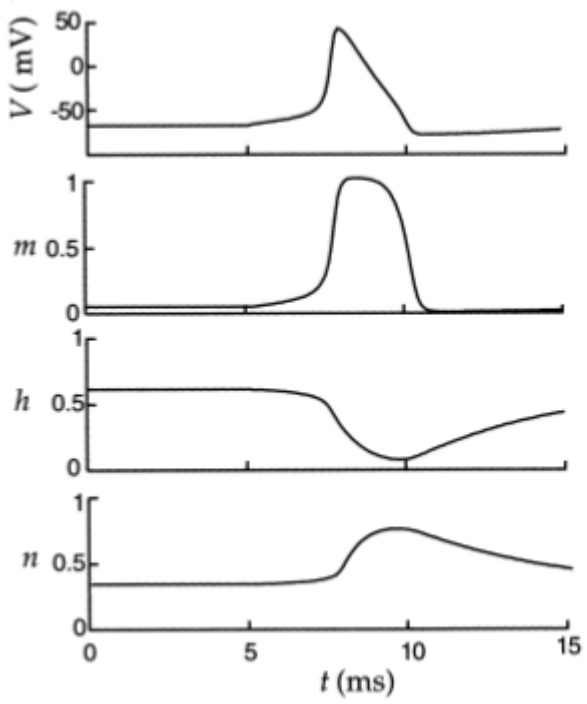
(g) Sodium channels open only transiently when the membrane potential is depolarized. Therefore Hodgkin & Huxley introduced another variable that can be interpreted as "blocking an open channel". The probability that an open channel is not blocked is denoted by h. Can you explain the expression for I_{Na}, last term of the model?

solution:

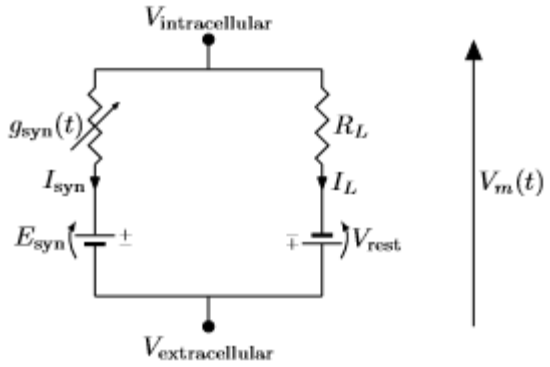
(f) n is the probability that a gate in the K^+ channel is open. Because it is modeled by 4 gates in series, n^4 is the probability that the whole channel is open; this is also the fraction of open channels. Multiplying this fraction with the maximal conductance $\overline{g_K}$ gives the actual conductance for K^+. This term is then multiplied with the driving potential for K^+, giving I_K.

(g) In this equation, $\overline{g_{Na}}$ is the maximum sodium conductance of the axon membrane. Sodium traverses the membrane through protein channels in the membrane, and these can open and close. $\overline{g_{Na}}$ is the conductance for sodium that is seen if all sodium channels are open. The gating variables m and h each change between 0 and 1 as functions of time and voltage. The product m^3h represents the fraction of the total sodium conductance at any given time. The sodium channel behaves as if it has two sets of gates. One set, the 'activation' gates (described by m), open rapidly when the cell is depolarised above a threshold voltage. The other gate, the 'inactivation' gate (described by h), closes slowly when the cell is depolarised. So m changes quickly and h changes more slowly.

h is low to block opening of **Sodium** channels while depolarized
n is how many channels are open
m is the probability of a channel being open



Direction of Current (Still unclear but whatever)
In Nernst equation and Goldman equation, we had the current in the numerator as the positive values outside. It was thus about positive charge ratio from out to in. **In the Circuit diagrams, the arrows point from in to out**, denoting **positive** ions flow. (The electrode currents however treat a current *into* the cell (depolarizing) as positive. At least one exercise sheet says that. a different ex. sheet says that the flux of positive charge going outward is positive current)
For the Voltage, there is a shitload of conventions how to choose the sign. In this course, the voltage arrows always go from the reference electrode to the measurement electrode (seemingly out to in). **But with respect to nernst, it seems to be $V_{in} - V_{out}$.** Still, in the nernst equation the enumerator is out and the denominator in (for positive ions).
(Almost) no current flows through the voltmeter.



So we actually have $V_{syn} = -I_{syn}R_{syn}$ here, which is confusing.

Fun Fact about conductance

If we use the equation $I = g \cdot (V_m - E_{ion})$, g is the slope of the current I.

Capacitance

Charge = Capacitance * Potential

$Q = C_{membrane} \cdot V_{membrane}$

$1Coulomb = 1nF \cdot 1mV$

1 Farad = 1 Coulomb/Volt = 1 Amperesecond/Volt

1 Volt = $1 \frac{kg\, m^2}{A \cdot s^4}$

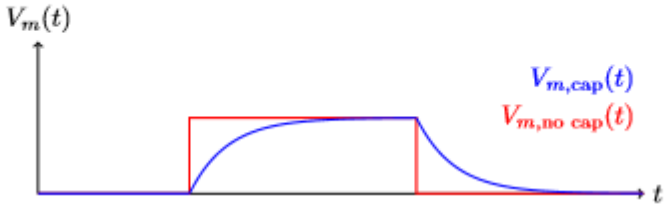
$$\frac{dQ}{dt} = I = C_m \frac{dV_m}{dt}$$

Current I changes membrane potential at a given rate mv/ms? Capacitance in nF, result in nA

Infinite resistance means no leaking current.

Denote Capacitor with two equally-long lines (like a battery)

4. By adding a capacitor we can make the time course of the voltage transition more realistic (not just a step function).



Cable Equation

See Ex. 04, 4.3

exercise04	solution04

Larger Axon radius means the same injected voltage propagates further.

capacitance only influences how long it takes for a signal to travel, not whether it arrives

Exercise 4.3: Cable Equation

For this exercise, nomenclature corresponds to the one used in Peter Dayan and L. F. Abbott, Theoretical Neuroscience, The MIT Press.
For a passive membrane, the membrane potential $V(x,t)$ is determined by solving the following partial differential equation (linear cable equation):

$$\tau_m \left(\frac{\partial v}{\partial t}\right) = \lambda^2 \left(\frac{\partial^2 v}{\partial x^2}\right) - v + r_m i_e$$

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where: $\tau_m = (r_m c_m)$ sets the scale for the temporal variation in the membrane potential
$\lambda = \sqrt{\frac{a r_m}{2 r_L}}$ sets the scale for the spatial variation in the membrane potential
(λ is called the *electrotonic length)*
with: c_m = specific membrane capacitance
a = radius of the axon (= 2 μm)
v = $V - V_{rest}$
r_m = specific membrane resistance (= 1 MΩ·mm^2)
r_L = longitudinal resistance (= 1kΩ·mm)
i_e = the current injected into a cell

We now assume an infinite cable and inject a constant current i_e locally at $x = 0$. The steady-state solution (so that $\frac{\partial v}{\partial t} = 0$) of the cable equation then is:

$$v(x) = \left(\frac{i_e R_\lambda}{2}\right) e^{-|x|/\lambda} \text{ , where } R_\lambda = \frac{r_L \lambda}{\pi a^2}$$

Mapping one thing I don't know to another

Resistor – Ion Channels
Capacitance – Lipid bilayer
Battery – Ionic concentration gradient

these correspond to each other

Postsynaptic Current

$I_{syn} = \frac{U}{R} = g_{syn}(t) \cdot \left(V_m(t) - E_{syn}\right)$

g is the synaptic conductance, V_m is the membrane potential, E_{syn} is the equilibrium potential of the synapse
We subtract E_{syn} from V_m because in the circuit graphic we can see that $g_{syn} I_{syn} + E_{syn} = V_m$

Units

Elektrischer Leitwert: $1S = \frac{1}{\Omega} = \frac{A}{V}$
nano <=/=> giga, piko <=/=> tera, milli <=/=> mega

Is a synapse excitatory?

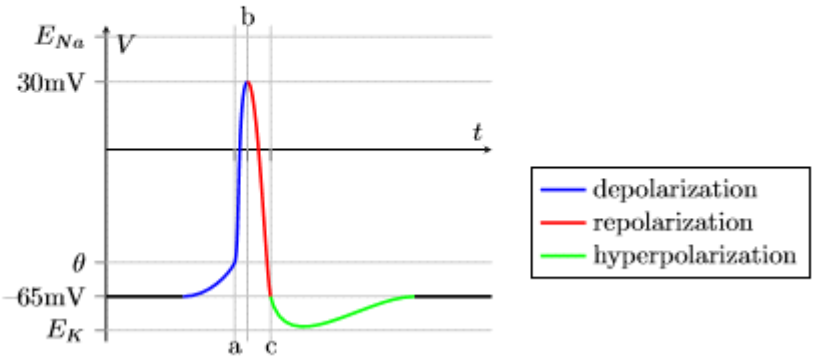
The AP threshhold is usually around $-55mV$. If the synapse brings the **membrane potential** V_m closer to above the threshhold than it would have been with only the resting potential of the cell (i.e. the reversal potential of the leakage current), the synapse is excitatory. E.g. $V_{Rest} = -70mV, V_m = -16.67mV \Rightarrow excitatory$.
They call the membrane potential also *postsynaptic potential*
If the synapse has a reversal potential of $E_{syn} \geq V_{Rest}, E_{syn} < threshhold$ then we call that **shunting inhibition** because the synapse does not hyperpolarize the cell, but it still makes the cell

less responsive.

Release Probability
If there is a very low extracellular amount of Ca^{++}, the probability of vesicle release is very low. If that probability is small and the number of synaptic vesicles available for release is large, then the expected number of released vesicles following a presynaptic action potential are poisson-distributed.

Action Potential
When the threshhold is exceeded, the Na+ and K+ channels open. but Na+ closes first, thus there's a hyperpolarization at the end.

At (a) and shortly before Na^+ channels open and K^+ channels start to open
At (b) Na^+ channels close (inactivation!)
At (c) K^+ channels start to close



This is also the reason why the action potential only moves in one direction. This is called the refractory period.
The K^+ that leave the axon during repolarization are pumped back in by the ATP pump. Same for the Na^+ that entered during the deporalization phase and need to leave again.
(Source: https://www.khanacademy.org/science/health-and-medicine/nervous-system-and-sensory-infor/neuron-membrane-potentials-2014-03-27T17:58:17.207Z/v/effects-of-axon-diameter-and-myelination)

Bayes Rule
$posterior = likelihood * prior : p(r)$
$$p(s|r) = \frac{p(r|s) \cdot p(s)}{p(r)}$$

Hebbian learning
Two main aspects in Hebbian learning are: joint activity and locality. Joint activity means that both pre- and postsynaptic neurons have to be active simultaneously to induce changes in the weight. Locality means that only neurons that are directly connected at a given synapse can change the weight of that synapse.

Spike Timing Dependent Plasticity
If $t_{post} > t_{pre}$, we have a probable depencency and increase the weight of that synapse. Otherwise decrease. We do that within some time window.

$F(\Delta t)$ is the amount of synaptic weight modification observed after a pair of pre- and postsynaptic spikes, as function of $\Delta t = t_{post} - t_{pre}$.

$$F(\Delta t) = \begin{cases} A^+ \exp(-\Delta t/\tau^+) & \text{if } \Delta t \geqslant 0 \\ -A^- \exp(\Delta t/\tau^-) & \text{if } \Delta t < 0 \end{cases}$$

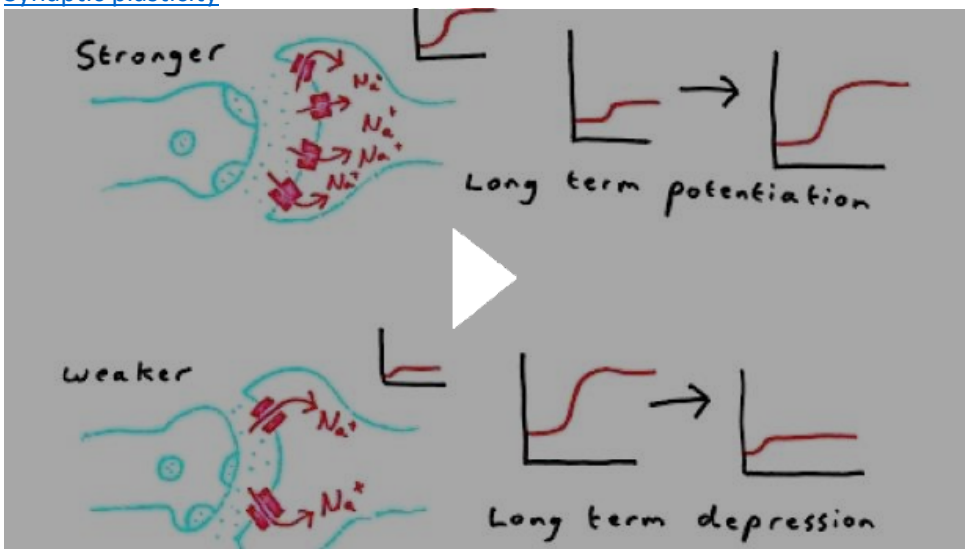
For the STDP window depicted in Fig 1, we have $A^+ = A^- = 0.5$ and $\tau^+ = \tau^- = 21.8$ms.

If we would only strengthen or only weaken, we would at some point have all (or no) neurons firing. that is worth nothing.

$$\Delta w = -A^+ \sum X(t)o(t) + A^- \sum Y(t)pre(t)$$

where o is the postsynaptic voltage, X and Y are the pre and postsynaptic spike train (1 or 0)

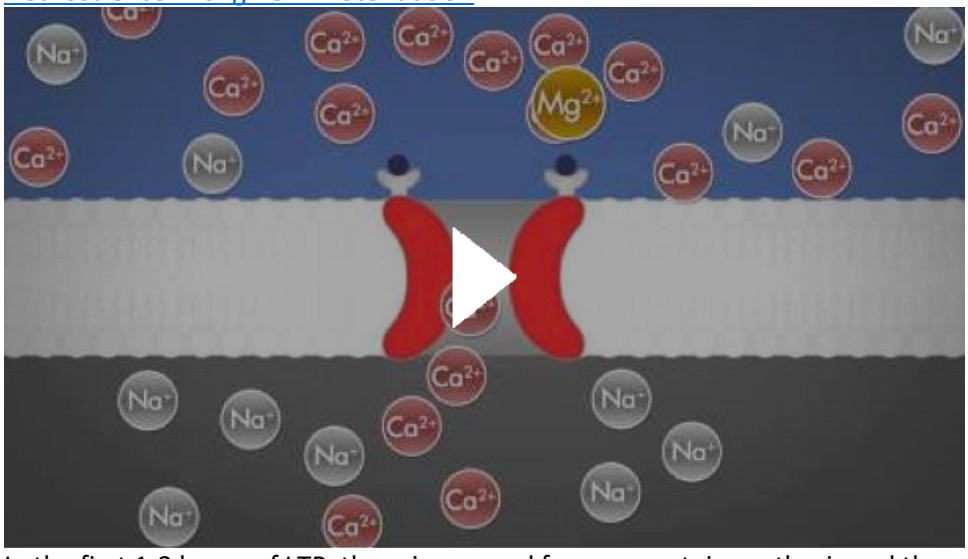
Synaptic plasticity



After a presynaptic AP, the cell becomes depolarized and releases Glutamate.
After a postsynaptic AP, the cell listens to Glutamate.
If the presynaptic AP was first, the postsynaptic cell receives Glutamate in the AMPA receptors and depolarizes. Also, they are more sensitive.
Flux through the AMPA can be both Na^+ and K^+, so the flux depolarizes the cell.
NMDA is usually blocked by magnesium, but once the cell is depolarized, the magnesium leaves and NMDA allows Na^+ and Ca^{2+} in and K^+ out once glutamate binds to it. This causes a large calcium influx into the postsynaptic cell.

If the postsynaptic cell was first, it is already repolarizing when the glutamate reaches it. There are thus fewer open NMDA receptors open and a more moderate calcium influx. If the Calcium influx is small, we get LTD (Long Term Depression) and if it is large, we get LTP (Long Term Potention).

LTP happens by moving more AMPA receptors to the synapse membrane and making them more receptive. This increases the current that is received.
LTD is the opposite.
If multiple synapses are weakly stimulating, they can together still cause LTP.
Neuroscience - Long-Term Potentiation



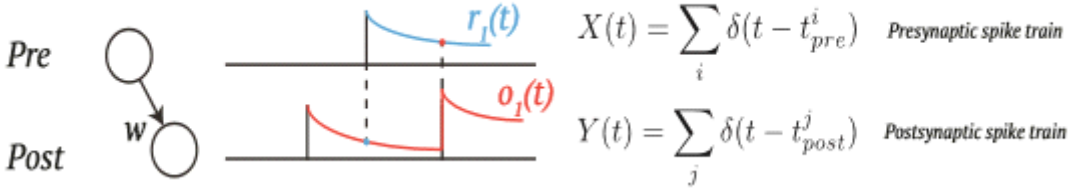
In the first 1-3 hours of LTP, there is no need for new protein synthesis and the conductance is still enhanced. The later phase of at least 24 hours requires new protein and RNA synthesis, which results in the construction of new postsynaptic receptors and presynaptic active zones (site of neurotransmitter releases).

This whole process happens only if the signals come in at a high enough frequency to reach a threshhold for LTP. The long phase (probably) only happens when the short phase is continually

triggered.
http://www.sumanasinc.com/webcontent/animations/content/ampa_and_nmda.mp4

How is the initial concentration restored?
Apparently the initial concentrations are restored mostly through the potassium ion leak channels (diffusion, which do not require energy,) but also through the active work of sodium-potassium-pumps in the cell membrane. The pumps require ATP and pushes 3 x Na+ ions out of the cell, at the same time allowing 2 x K+ ions to move into the cell.
Aus <https://www.khanacademy.org/science/health-and-medicine/nervous-system-and-sensory-infor/neuron-membrane-potentials-2014-03-27T17:58:17.207Z/v/neuron-action-potential-mechanism>
See also https://www.khanacademy.org/science/health-and-medicine/nervous-system-and-sensory-infor/neuron-membrane-potentials-2014-03-27T17:58:17.207Z/v/neuron-resting-potential-mechanism

Regarding Ex 9.1 (STDP)
We have the dirac delta which is 1 if its argument is 0 and else it is 0. So the *presynaptic spike train* $X(t) = \sum_{t_{pre}} \delta(t - t_{pre})$ is 1 if t is some t_{pre}. Thus, we consider only t that are at some t_{pre}, and multiple t_{pre} at the same t cause a larger consideration.
That means that the sum of all weights for learning at times t_{pre} is $A^- \cdot X(t) \cdot o(t)$ with A as a constant and o as the current postsynaptic voltage at time t.
This is because the closer t_{pre} is before t_{post}, the more they are related. That means, we actually change the weights by $-A^- X(t) o(t)$ because if pre is after post, we don't like that, but it is then that this formula is big.
Similarly but positive for the postsynaptic train $Y(t)$.



Consider any pair of pre and post. $pre_1, post_0$ is not related, so we subtract the voltage o_{pre0} because we later notice that $pre_1, post_1$ is related and add the voltage at $post_1$. There, we would have too much otherwise.
We only consider such pairs because any other t are excluded by X and Y.

McCulloch-Pitts Neuron Model
A neuron has inputs that are 0 or 1, which are weighted. if the results sum is larger than a threshhold, the output is 'activated', else it is not.
This allows NAND and AND, but not XOR and NOT(XOR)
Imagine with a graph where you draw a line to separate two groups.

Perceptron learning algorithm
(Example in exercise had 'no threshhold', i.e. threshhold fixed at 0. In that case, we add another - fixed - input to get our degree of freedom back by using its weight.)
The given binary inputs produce a binary solution. If the solution is as wished, test the next set of inputs. If it is not, first adjust the weights by some factor alpha. Any input that was 0 keeps its previous weight, any input that was 1 gets an additional/subtractional w_i in the direction of the desired output. (Actually for any nonzero input, because we just calculate $x_i \cdot w_i$). That means if the output should be 1, but was 0, we make the weight more positive. Otherwise more negative, but by zero.
Then test the next set.
The formula might contain a learning factor η, such that $\Delta_{w_i} = \pm x_i \cdot \eta$. In the below example, η is 0.3. We apply the changes only to active neurons (where the result was not as desired).
Repeat until all sets pass.

If the threshhold is 0 and the weighted inputs are equal to zero, the unit is still considered active.
If we don't use a threshhold that can be modified, we need a bias input instead
(https://stackoverflow.com/questions/1697243/perceptron-learning-algorithm-not-converging-to-0)

Can be thought of as - for mcCulloch-Pitts - a line that splits the inputs into output sets. Thus, AND can be done, XOR can not be done.
That means that every input provides one 'axis' of either 0 or 1, and for two inputs, the threshhold is decided by a line (described by both weights. $\theta = \alpha_1 x + \alpha_2$). In 3D with one input fixed, it's a plane and thus a fixed threshhold at 0 still allows enough freedom to choose a fitting line (in some plane).

Solution 11.2: The Perceptron Learning Algorithm

x_1	x_2	desired output	current output	w_1	w_2	w_3
1	0	1	0	–0.4	0.5	–0.4
0	1	1	1	–0.1	0.5	–0.1
1	1	1	1			
0	0	0	0			
1	0	1	0			
0	1	1	1	0.2	0.5	0.2
1	1	1	1			
0	0	0	1			
1	0	1	1	0.2	0.5	–0.1
0	1	1	1			
1	1	1	1			
0	0	0	0			

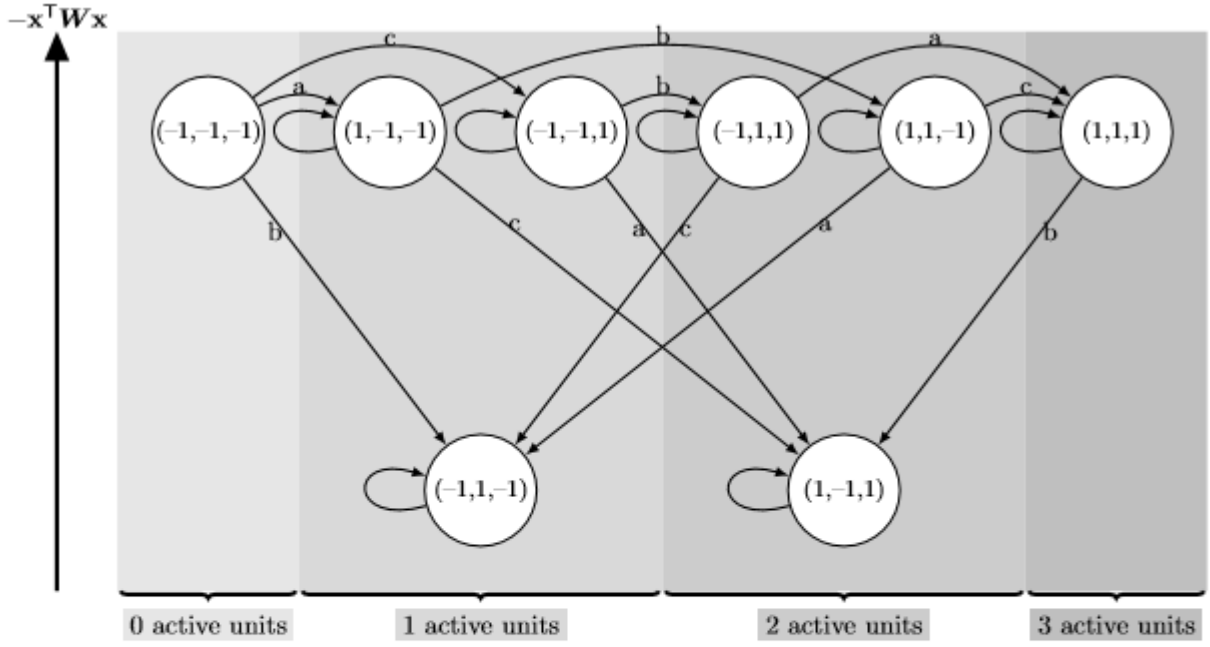
The Perceptron learned the OR function.

Hebbian Learning
let x_1 be a vector containing a pattern where the network should be stable. Same for x_2.
The neurons can either have a value of -1 or 1. Thus, $n_i \cdot n_j$ is 1 if both contain the same value.
Hebbian learning is defining the weight update step as $\Delta w_{ij} = \alpha x_j y_i$ where y_i is the output of the i-th neuron and x_j is some input to it. When input equals output, this delta is positive, else it's negative. This is good because we train the network so that it will in the end be able to find those inputs. (As opposed to the perceptron example above where we wanted a different output pattern). So we actually input the target patterns.
In Hopfield networks, that gives $W^{(m+1)} = W^{(m)} + x_k x_k^T - I$. That means that the connections to itself are always weighted 0 and the other weights are increased if the output was nice and decreased otherwise.
If we have a Hopfield Network, we apparently are not allowed to set some weights to 0 apart from the one from n_i to n_i. Thus, there might be some invalid results from hebbian learning, where perceptron learning would have worked.

Fundamental Memories (generalized Hebbian Model?) in Hopfield Networks
In hopfield networks, the weights are symmetric and all neurons are interconnected with all except themselves. The neurons can assume the output values -1, 1. A hopfield network has symmetric weights even though that is not entirely biologically correct. This is because we can then easier set up equations.
Learning cycles are not possible. The system can not get back to a previous state - there's always the same state, or more active neurons. Additionally, the energy always goes down, so it always reaches

a stable state.

Fundamental memories are stable states in a network that can be content-addressed by a noisy version of the data. If we update only one neuron weight and value at once, and then the next in the new state etc., we can get such a fundamental memory where no state change step actually changes the state.



$$E_{(-1,-1,-1)} = E_{(1,-1,-1)} = E_{(-1,-1,1)} = E_{(-1,1,1)} = E_{(1,1,-1)} = E_{(1,1,1)} = -\frac{4}{3}$$

$$E_{(-1,1,-1)} = E_{(1,-1,1)} = -4$$

The states are ordered in a way such that towards the right the number of active unit increases. It is no coincidence that the arrows:

- never go up in energy
- if energy stays the same, arrows never go from the right to the left (in this case, the number of active units never decreases).

Mind the bullet points! That energy is basically the sum of (weights times every neuron with every neuron product).

$$E = -\frac{1}{2}\sum_i \sum_j w_{ij} a_i a_j$$

The second point has the reason that 0 is also active, only below 0 is inactive - but we didn't look at the details :(

See also [Hopfield Energy Proof]

Synchronous vs Asynchronous update

In a synchronous update, all neurons are updated at the same time. In an asynchronous update, one random neuron is chosen and updated. The new value is then considered for the next update step.

$$s_i(t+1) = \begin{cases} +1, & if \sum_j w_{ij} s_j \geq \theta_{ij} \\ -1, & otherwise \end{cases}$$

"Neurons attract or repel each other" (attract if the weight is positive)

Training the network as storage

Training a Hopfield net involves lowering the energy of states that the net should "remember". This allows the net to serve as a content addressable memory system, that is to say, the network will converge to a "remembered" state if it is given only part of the state. The net can be used to recover from a distorted input to the trained state that is most similar to that input. This is called associative

memory because it recovers memories on the basis of similarity. For example, if we train a Hopfield net with five units so that the state (1, -1, 1, -1, 1) is an energy minimum, and we give the network the state (1, -1, -1, -1, 1) it will converge to (1, -1, 1, -1, 1). Thus, the network is properly trained when the energy of states which the network should remember are local minima.

- Note: In contrast to Perceptron training, the thresholds of the neurons are never updated.

Aus <https://en.wikipedia.org/wiki/Hopfield_network#Initialization_and_running>

The weights for when we have multiple patterns to store is $w_{ij} = \frac{1}{patterns}\sum_{p}^{patterns} a_i^{(p)} a_j^{(p)}$ where a_i is the i-th bit of the p-th pattern, i.e. the desired output of the i-th neuron. What this does: It causes the i-th and j-th neuron to tend to be equal iff they are equal in the desired pattern(s). We can only learn so many patterns per dimension

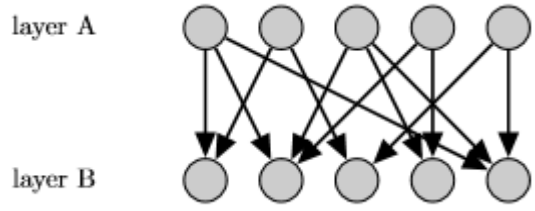
Differences Hopfield network to perceptron
Hopfield never updates threshholds.
It's a Feed-forward network (no cycles).

Error back propagation
The left neuron's output is fed into the right neuron's input. We define the error function as $\sum\left(f\left(x_1 w_1 + x_2 w_2\right) - d\right)$ where f is the activation function of the neuron and d is the correct solution that we want. The derivative of the error with respect to some weight consists of a constant 'delta term' multiplied by the input times the respective weight.
In general, the non-input part of the gradients is the same for all weights of one unit and is called the delta term or error term. The delta term of a lower, i.e. closer to the input, unit can be obtained from the higher units. The higher delta term multiplied by the connecting weight and by the derivative of the lower unit's activation function gives the lower unit's delta term wrt that weight. The delta terms represent the error contributed by each unit.
It is thus sufficient to compute the delta terms for each unit to get the derivatives wrt the weights.

3. We can think of the delta terms as travelling back through the wires. Whenever they leave a neuron through an input, they are multiplied by the weight of the input. Whenever they enter a neuron through its output, they are multiplied by the derivative of the activation function of that input.

   Whenever the output of one neuron is used several times, all delta terms that meet at a junction are simply added.

Convergence
A connection is *converging* if a neuron receives the input from several neurons. If it is sending to several neurons, it is called *diverging.* It **can** be both!
A 'connection' here means the arrows from the nodes of one layer to the next layer.

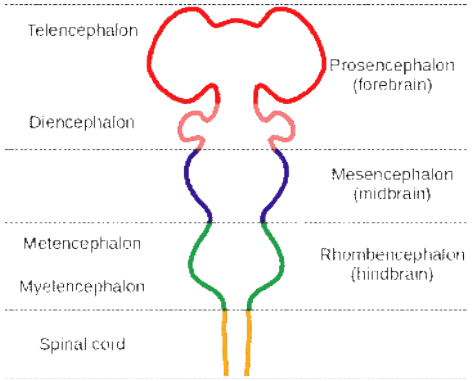


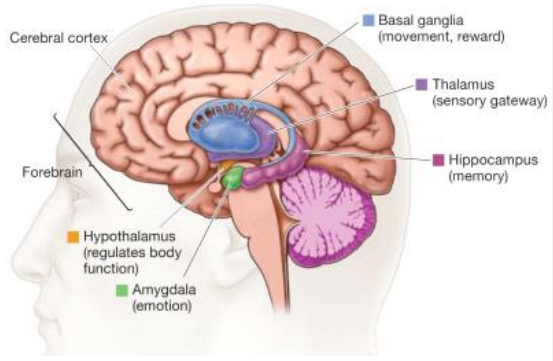
example that is both diverging and converging

Regarding 14.2.6
They talk about a weakly connected neuron. That means that it is further away, but it also means that it is *connected* and still under the influence of the actual event neuron. That might be why there are two spikes. one where it spikes and one where the correct neuron spikes.
The almost-correct neuron gets two spikes at almost the same location and thus one large spike.
I'm not sure why we're ignoring every other neuron that is connected then though. Maybe because the correct one spikes way harder and thus propagates well, while the others are almost not noticeable or cancel eachother out. (height everywhere instead of one spike)

VOCI

Brain during development





Prosencephalon

Telencephalon develops into the cerebral cortex, basal ganglia, hippocampal formation, amygdala

Diencephalon develops into the thalamus, hypothalamus, pituitary gland (just below the hypothalamus, regulates the endocrine system - i.e. hormones and sex organs)

Mesencephalon

Tectum and tegmentum. The mammalian tegmentum contains the superior and inferior collicullus.

Rhombencephalon

Metencephalon

pons, cerebellum

Myelencephalon

develops into the medulla oblongata

Lobes in the neocortex, which is in the Telencephalon

Frontal Lobe: Language processing (Brocas Area), motor/pre-motor areas, cognition

Higher mammals also have a pre-frontal lobe for cognitive processing

Parietal Lobe: behind central sulcus. Somatosensory processing and some sensori-motor functions

Temporal lobe: separated by the lateral fissure. Involved in auditory processing and language comprehension (Wernicke area). Visual object recognition. Contains a portion of the hippocampal formation which is involved in formation of new memories.

Occipital Lobe: Visual processing

What the things do

Pituitary Gland

controls hormones, sex organs

Hypothalamus

regulates autonomic bodily functions (e.g body temperature, food takein, sleep and circadian rhytms, sexual behaviour)

Cerebral Cortex

superficial grey matter (local connections, not myelinated) of the hemispheres. Serves various sensory, motor and cognitive functions and is subdivided into layers that organize input and output connectivity of resident neurons.

Increased surface area is thought to be important because this is where the grey matter is, which oversimplifiedly speaking does the computations.

Thalamus

relays information from and to the neocortex

Dopamine

Is not a part of the brain but a neurotransmitter and neuromodulator.

Significant for motor processes, pleasure, reward, attention, motivation

Neuromodulators are neurotransmitters that diffuse through neural tissue to affect slow-acting receptors of many neurons.

Aus <https://en.wikipedia.org/wiki/Neuromodulation>

Limbic System
- Cortical and subcortical structures concerned with emotions.
- Includes the gyrus cinguli, the hippocampus and the amygdala.

Basal Ganglia
- Voluntary movement
- Procedural Learning
- motor control
- When some of the dopaminergic neurons here degenerate, you get parkinsons

Cerebellum
- Motor behaviour
- Posture adjustment

Hippocampus
- consolidation of short-term memory into long-term memory

Amygdala
- Emotional Processing
- Sense of smell

Numbers

Number of genes in humans	$2.5 * 10^4$
Number of base pairs in human DNA	$3*10^9$
Average synapse count per neuron	10^4
Average input count needed for a neuron to fire	25
Human brain weight	1.5 kg
Human brain volume	1.1 liters
Human brain neuron count	86 billion $= \sim 10^{11}$
Human brain synapsecount	$\sim 10^{15}$
Human brain mass percentage of whole body mass	2%
Human brain energy consumption of whole body's	20%
Weight and #neurons of Smoky Shrew	0.18g, 36 million
Weight and #neurons of Short-tailed Shrew	0.35g, 52 million
Weight and #neurons of Star-nosed Mole	0.8g, 131 million
Weight and #neurons of Eastern Mole	1g, 204 million
Weight and #neurons of Mouse	0.4g, 71 million
Weight and #neurons of Hamster	1g, 90 million
Weight and #neurons of Rat	1.8g, 200 million
Weight and #neurons of Guinea Pig	3.8g, 240 million
Weight and #neurons of Marmoset	78g, 634 million

Weight and #neurons of Agouti	18g, 857 million
Weight and #neurons of Galago	10g, 936 million
Weight and #neurons of Capybara	76g, 1.6 billion
Weight and #neurons of Owl monkey	16g, 1.5 billion
Weight and #neurons of Squirrel Monkey	30g, 3.2 billion
Weight and #neurons of Capuchin Monkey	53g, 3.7 billion
Weight and #neurons of Macaque Monkey	87g, 6.4 billion
Proportion of neocortex to the entire brain	80-85%
Number of nerve fibers from the retina to the thalamus for carrying visual information	1 million for each eye
Meters of axons contained by $1mm^3$ of gray matter. And of white matter?	4'000m for gray matter, 9m for white matter
Neuron resting potential	$-70mV$
Neuron usual threshhold for AP	$-55mV$
Chloride reversal potential	$-65mV$
Potassium reversal potential	$-90mV$
Sodium reversal potential	$+55mV$
$\frac{RT}{F}$ expressed for base-10 log equation	58mV
Length of a synapse	$1\mu m\ \ (1\ micrometer)$
Length of a neuron	$100\mu m$
#nerve cells in a C. Elegans	300
#nerve cells in a Fruit Fly	100'000
#nerve cells in a Honey Bee	960'000
#nerve cells in a Frog	16 million
#nerve cells in a Mouse	75 million
#nerve cells in a Cat	1 billion
#nerve cells in a Chimpanzee	6.7 billion
#nerve cells in a human	85 billion
Size of the amoeba dubia genome	200x human genome

Hirnhaut (Meninges)
dura-mater, arachnoid-mater, pia-mater.
Dura mater is leathery and directly below the bone. Arachnoid mater is thin with spiderweb-like filamentous extensions to the pia mater.
The pia mater is part of the blood-brain-barrier. It's somehow rich in blood vessels though.

Between the pia mater and the arachnoid mater, there is the Cerebrospinal fluid **CSF.**

Local Field Potential (LFP)

Measurement of extracellular potential for a group of neurons (as opposed to a single neuron).
Dominated by dendridic synaptic activity.
Exicatory receptors at dendrites
AMPA, NMDA "Voltage(drive) = 0mV"
Inhibitory receptors at dendrites
GABA A, Chloride "Voltage(drive)=-65 mV"
GABA B, Potassium(K) "Voltage(drive)=-90 mV"

Functional Magnetic Resonance Imaging

Functional magnetic resonance imaging or **functional MRI** (**fMRI**) measures brain activity by detecting changes associated with blood flow.[1][2] This technique relies on the fact that cerebral blood flow and neuronal activation are coupled. When an area of the brain is in use, blood flow to that region also increases.[3]

Aus <https://en.wikipedia.org/wiki/Functional_magnetic_resonance_imaging>