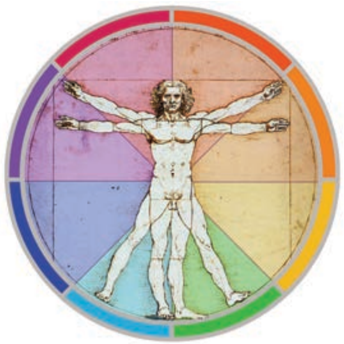
HUBS191 Lecture Material

This pre-lecture material is to help you prepare for the lecture and to assist your note-taking within the lecture,

it is NOT a substitute for the lecture !



Please note that although every effort is made to ensure this pre-lecture material corresponds to the live-lecture there may be differences / additions.

Physiological Principles of Human Movement and Sensation

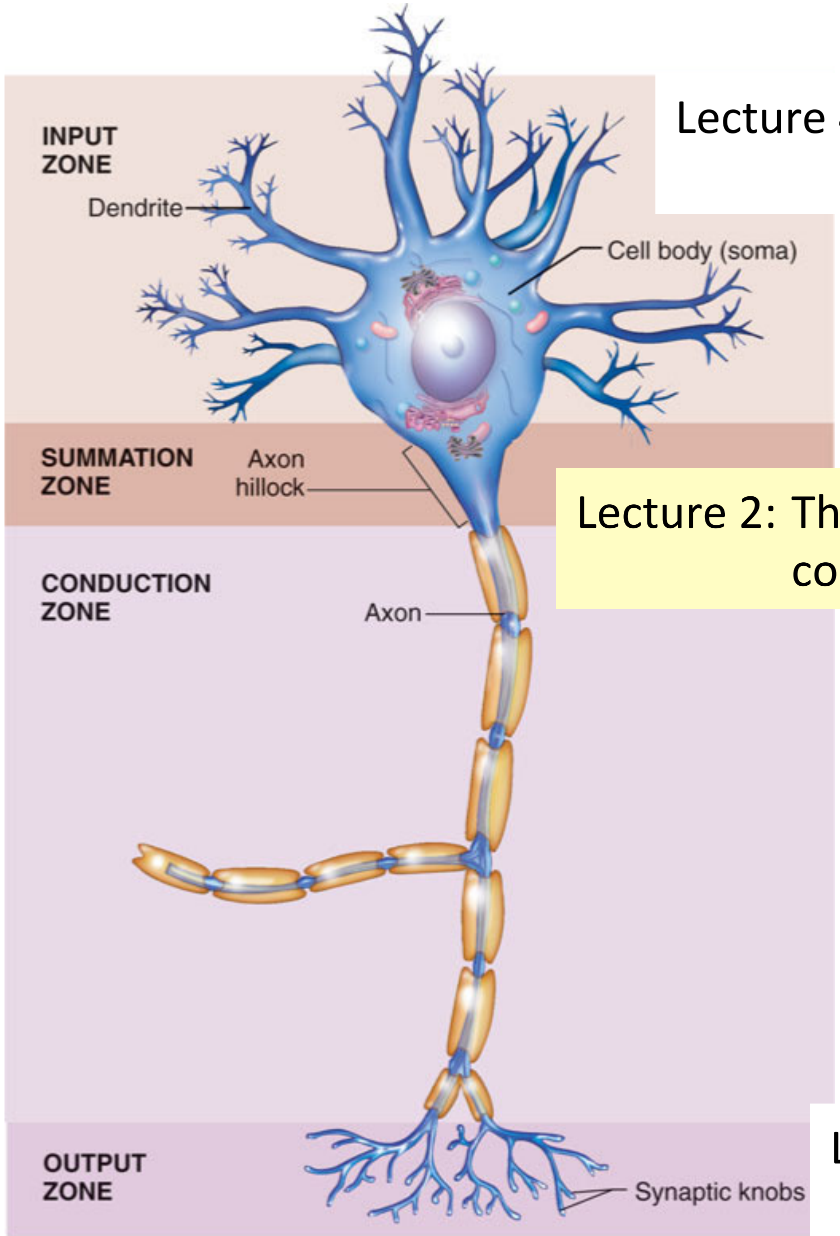
HUBS191

Lecture 22

Dr Martin Fronius
Department of Physiology

5. April 2017

Lectures outline



Lecture 4: Synaptic Networks and Integration (input zone)

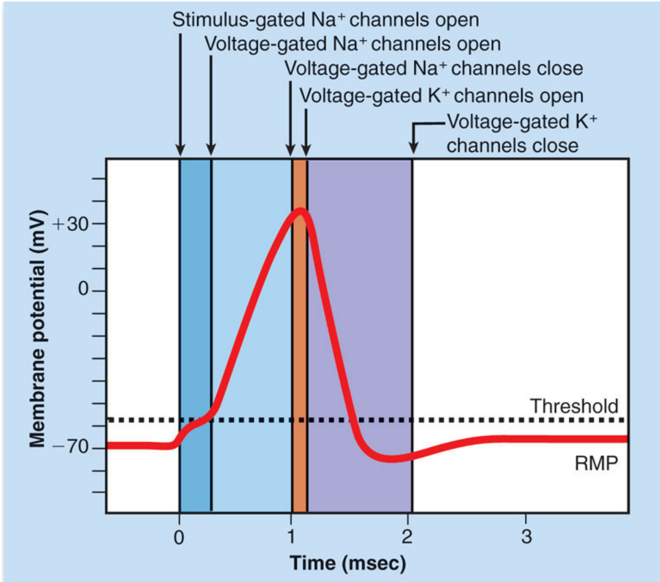
Lecture 2: The Action Potential and its conduction (axon hillock, axon)

Lecture 1: Bioelectricity and membrane potentials

Lecture 3: The Synapse – 'connecting' neurons (output zone)

Aims of Today's Lecture

Lecture 22 – The Action Potential



Outline

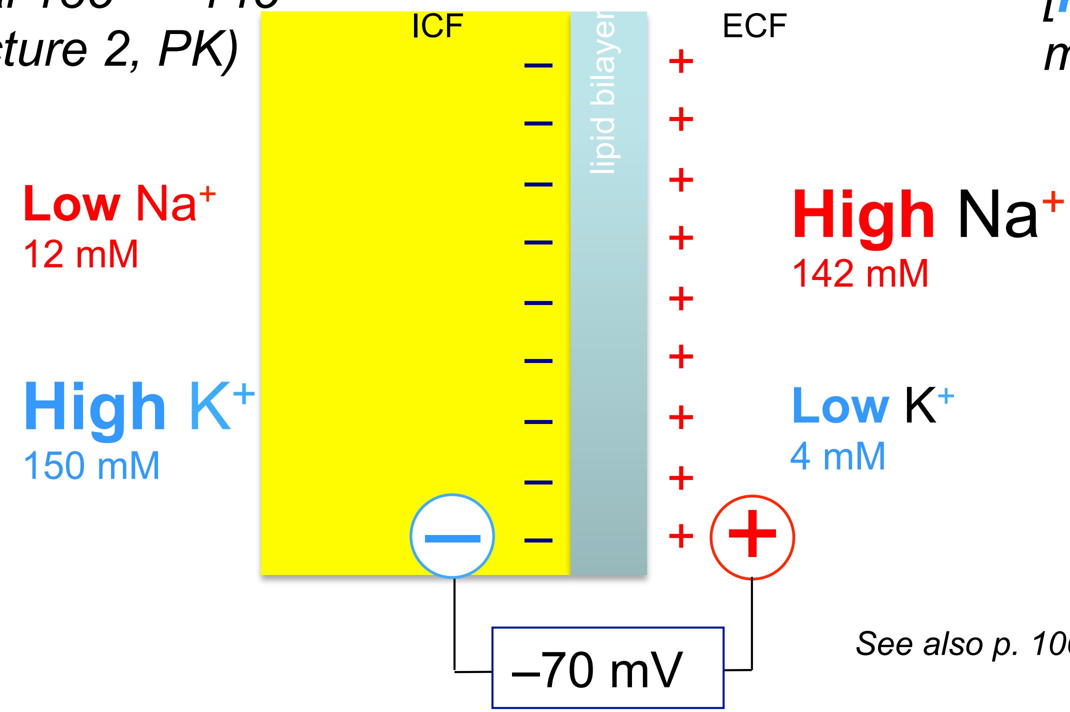
- Recap from lecture 1
- The Action Potential
- Propagation of the Action Potential
- Refractory periods – Why Action Potentials go one way?
- Ways to enhance the speed of Action Potential conduction (unmyelinated vs myelinated conduction)

Action potentials are the basis of all neuronal communication – they are the electrical impulses!

The uneven distribution of ions creates cellular electricity – the membrane potential

[Na^+] external 135 – 145 mmol /L (Lecture 2, PK)

[K^+] external 3.5 – 5 mmol /L (Lecture 2 PK)

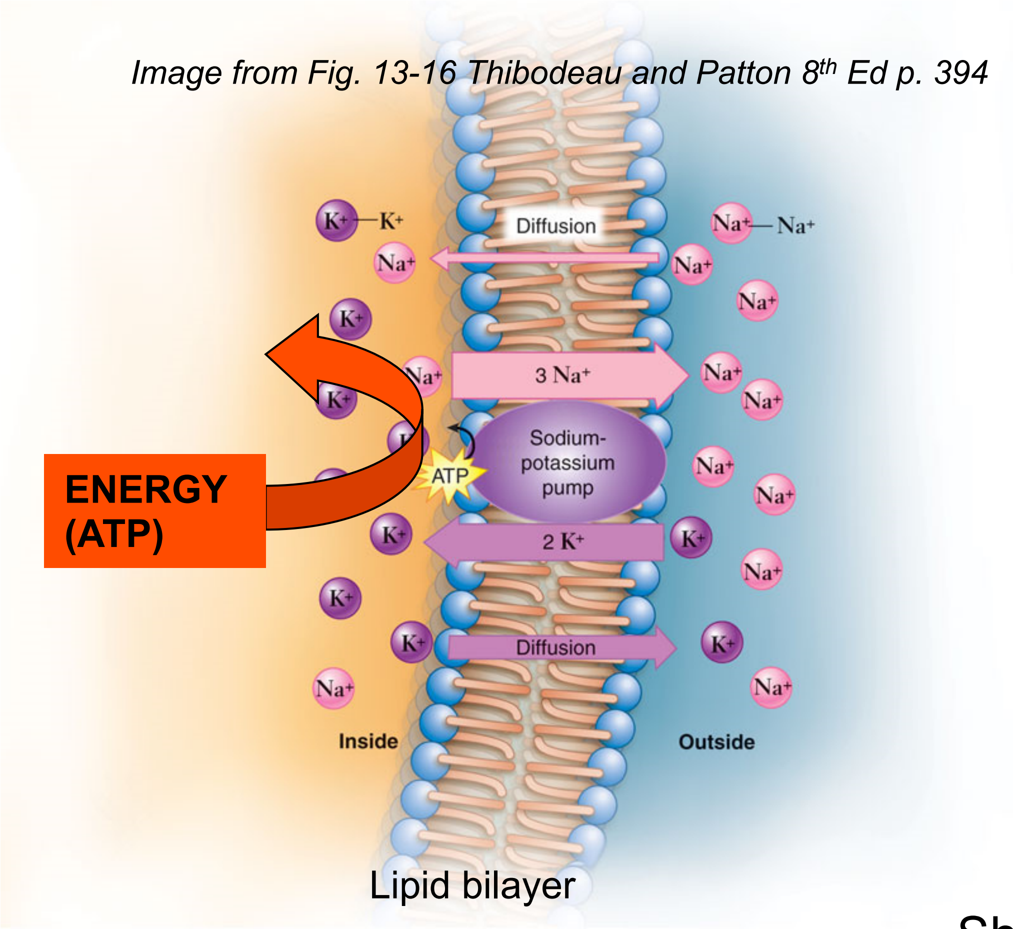


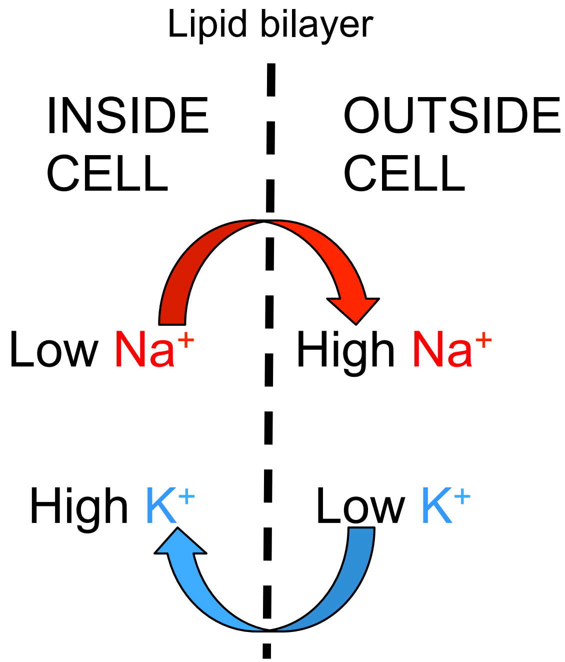
See also p. 1006 Patton&Thibodeau, 8th Ed.

- Charge difference between the two sides of the membrane → like a small battery
- THIS IS CALLED THE RESTING MEMBRANE POTENTIAL
  in living neurons it is about -70 mV, slightly negative inside
- Ion channels allow the flow of ions → electrical switches (**downhill** transport)

How is the membrane potential maintained?

Image from Fig. 13-16 Thibodeau and Patton 8th Ed p. 394





Shifts 3 x **Na^+** cations OUT of the cell

For

2 x **K^+** cations IN the cell

- Na^+/K^+ ATPase (aka Na^+/K^+ PUMP)
- Uses energy (ATP)
- '**uphill** transport'

Changes (types) of membrane potentials

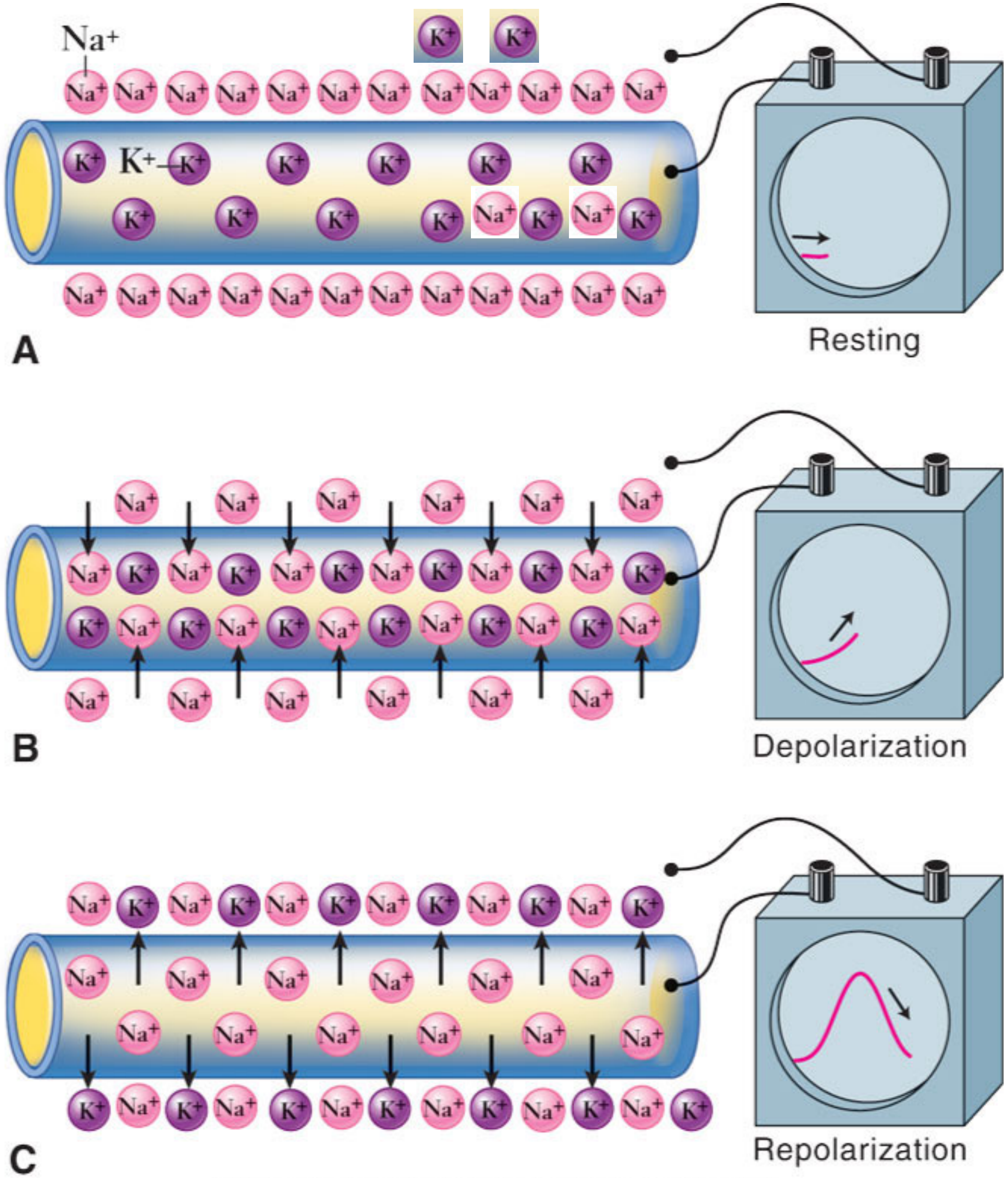


Fig. 13-18 Thibodeau and Patton 8th Ed. p.396.

- RESTING
- Low Na^+ inside and high K^+ inside
- Na^+ inflow = K^+ outflow
- **–70 mV**

- **Na^+** channels open
- Positive charges move IN
- DEPOLARISED → e.g. –60 mV

- **K^+** channels open
- Positive charges move OUT
- **REPOLARISED** → negative again
- **HYPERPOLARISED** → e.g. **–80 mV**

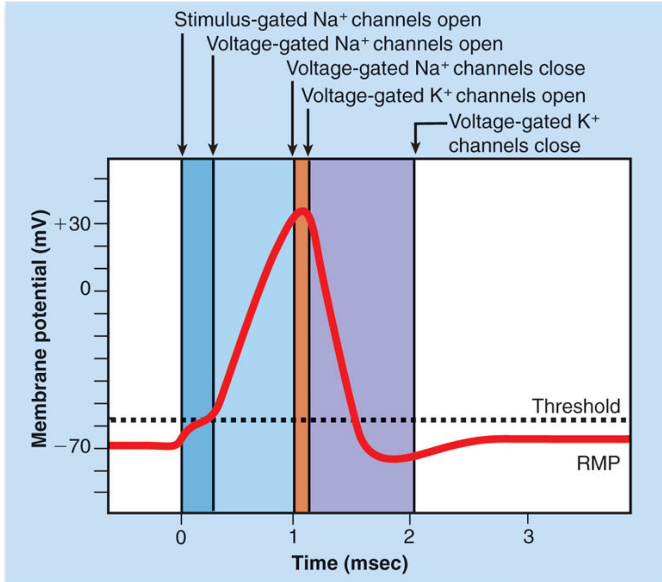
Types of Potentials

Type of Potential	**Polarisation**	**Typical value**	**Description**
Resting	Balanced	-70 mV	Neuron is at rest, not excited not conducting an impulse, Some K+ channels open.
Local	**DEpolarised** **EXCITATORY** **HYPERpolarised** **INHIBITORY**	**Varies**	Temporary fluctuation in a local region of the membrane Na+ and K+ channels open ACTIVE neurons
Threshold	Depolarised	**-59 mV**	Minimum local depolarisation that triggers an action potential
Action	**DEpolarised** **REpolarised**	+30mV	Temporary max depolarisation propagates along the axon without losing amplitude

See Table 13-3: *Patton & Thibodeau, 8th Ed p.397*

Aims of Today's Lecture

Lecture 22 – The Action Potential



Outline

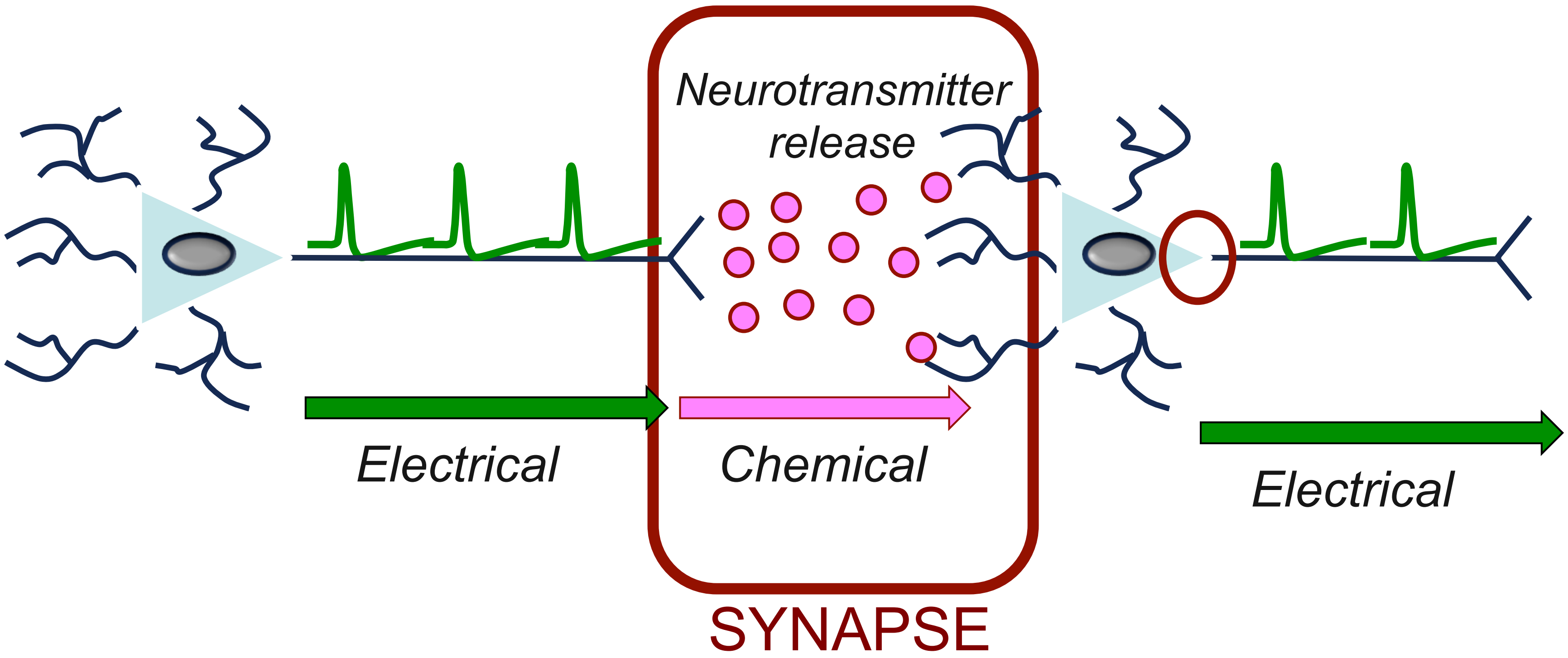
- Recap from lecture 1
- The Action Potential
- Propagation of the Action Potential
- Refractory periods – Why Action Potentials go one way?
- Ways to enhance the speed of Action Potential conduction (unmyelinated vs myelinated conduction)

Action potentials (APs) are the basis of all neuronal communication – they are the electrical impulses!

Objectives

- List the events that occur during the action potential
- Know the two types of refractory period of the axon
- Explain how the speed of conduction of the action potential can be enhanced

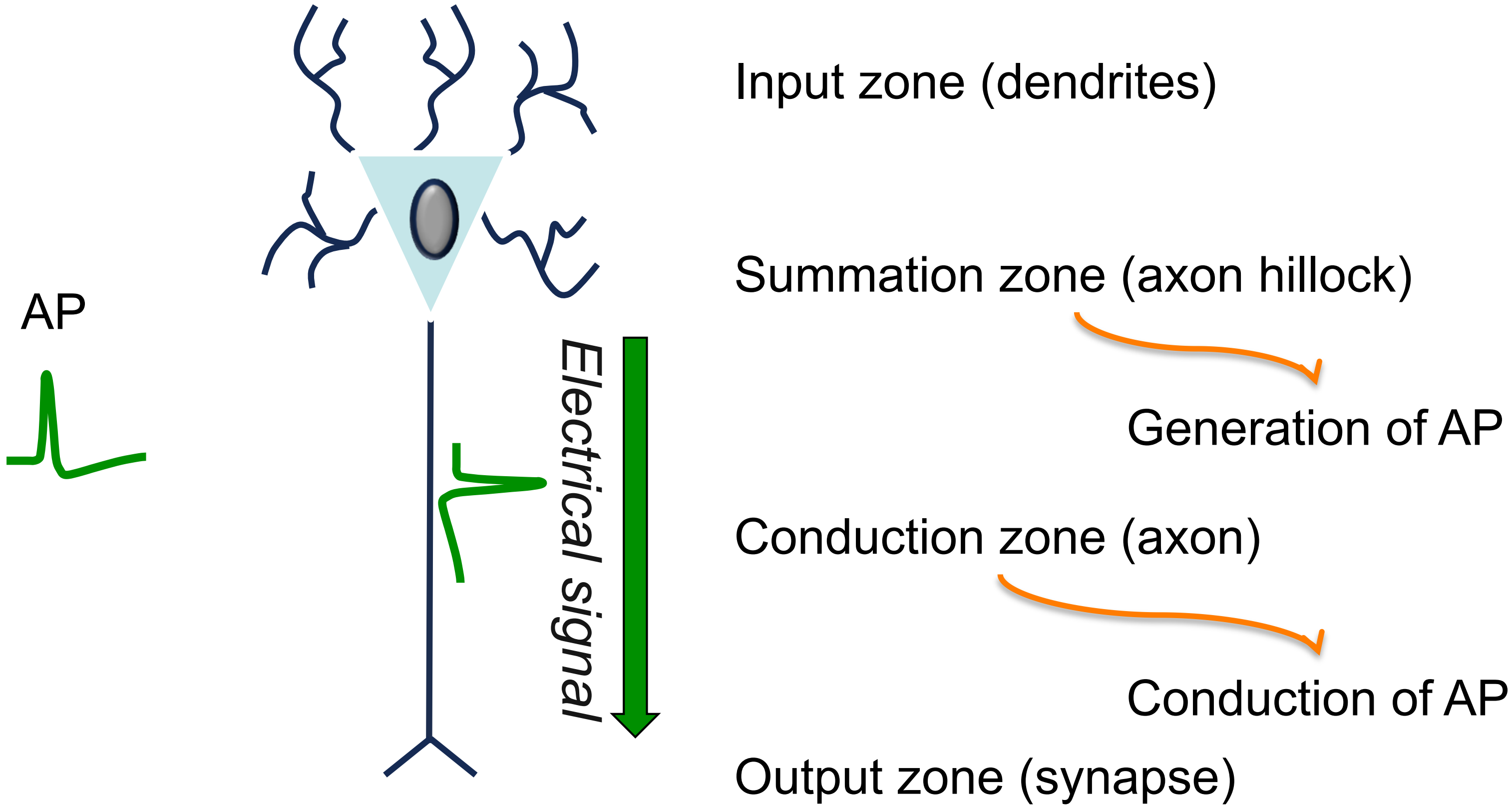
Cells of the nervous system: Neuron, basic structure



See also: Patton & Thibodeau 8th ed Fig 13-5 (7th ed Fig 12-5)

REMEMBER --- From A/Prof Jasoni's Lecture

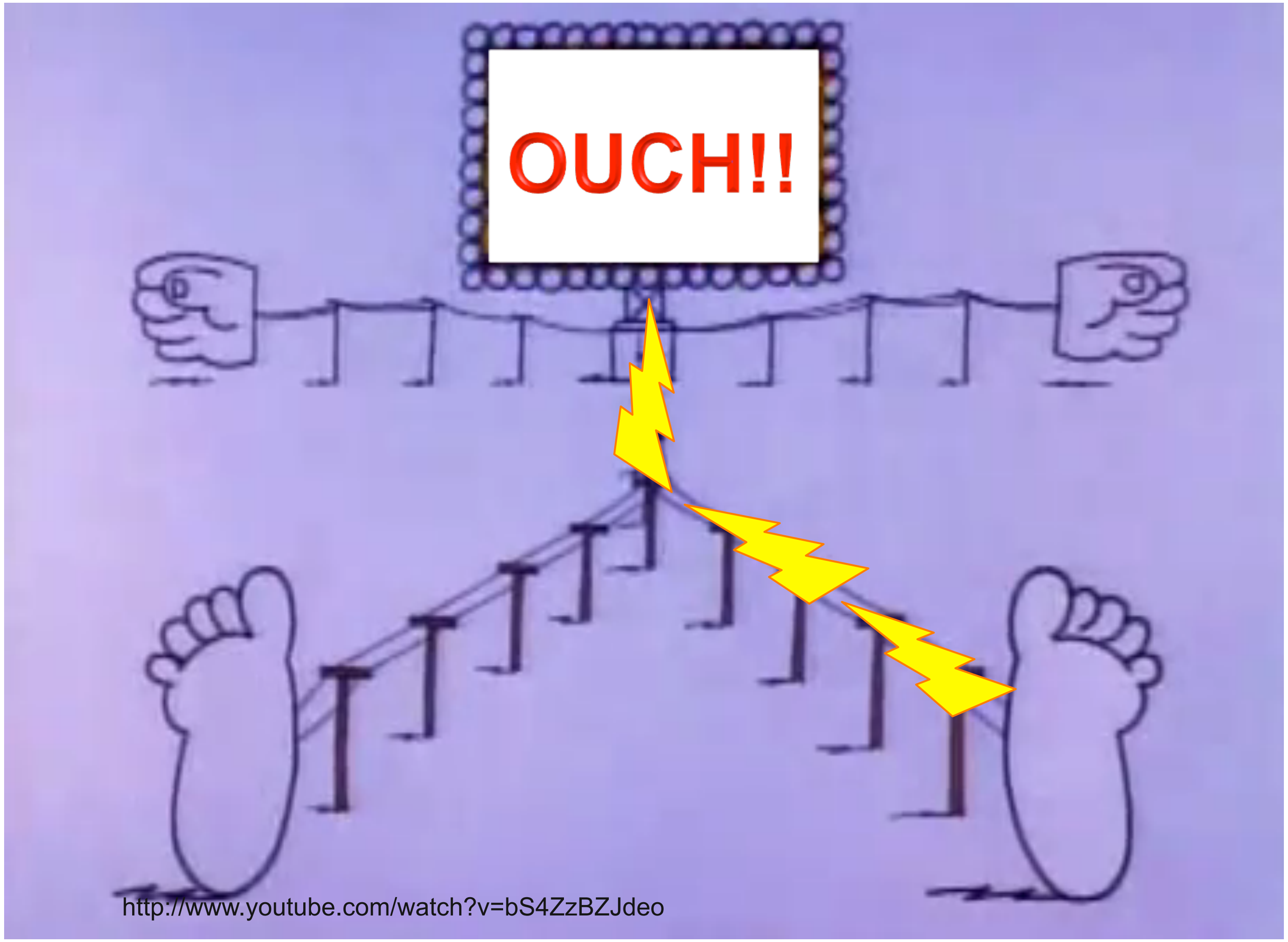
Recap *– Cells of the nervous system:* Neuron, basic structure



See also: Patton & Thibodeau 8th ed Fig 13-5 (7th ed Fig 12-5)

REMEMBER --- From A/Prof Jasoni's Lecture

The AP – sending information

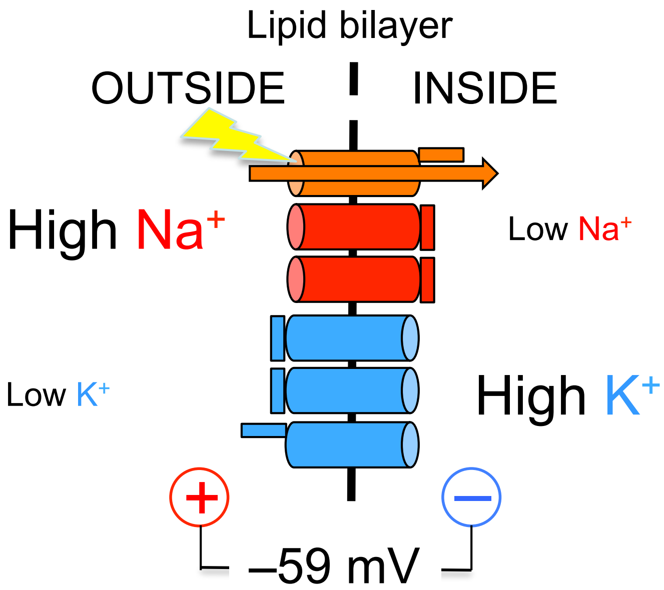


Sending ACTION POTENTIALS – EXCITATION (depolarisation)

REMEMBER --- From A/Prof Jasoni's Lecture

HOW DOES THE ACTION POTENTIAL OCCUR ?

It starts with an adequate stimulus causing a LOCAL DEPOLARISATION



- A small number of Na+ channels open (→ stimulus gated Na+ channels)
- … the membrane potential DEPOLARISES…
- … towards the THRESHOLD potential (**–59 mV**)

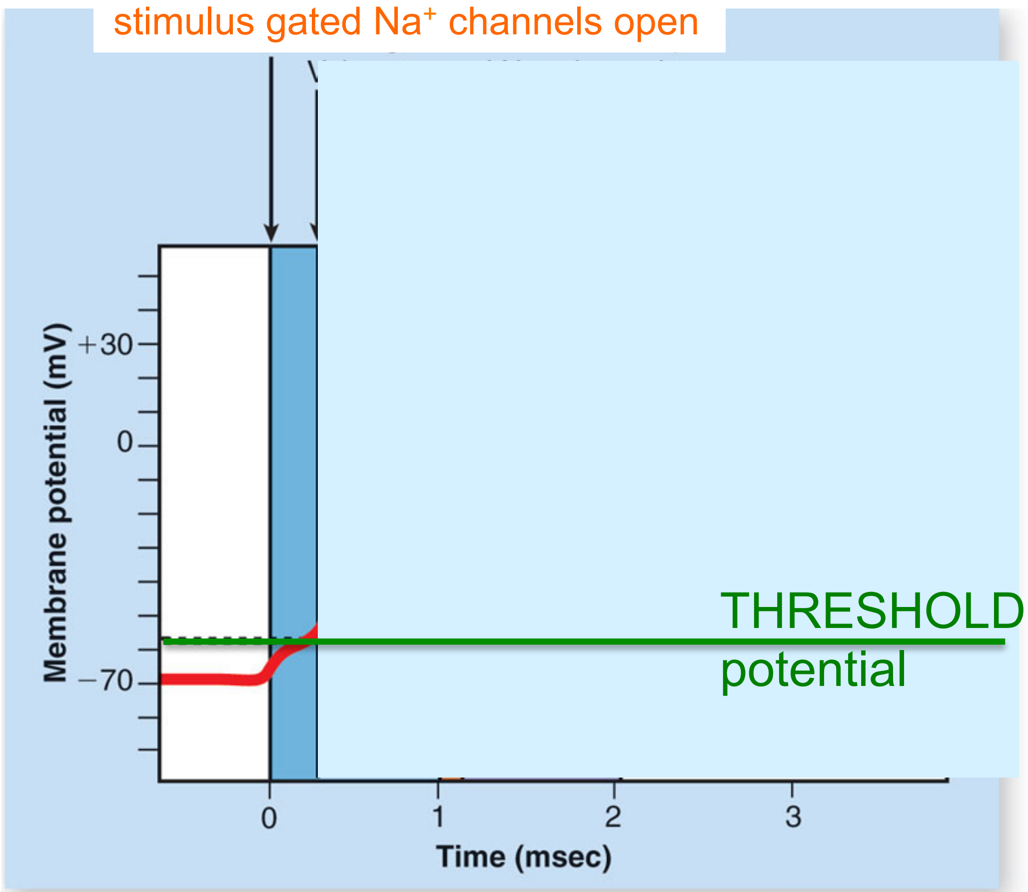
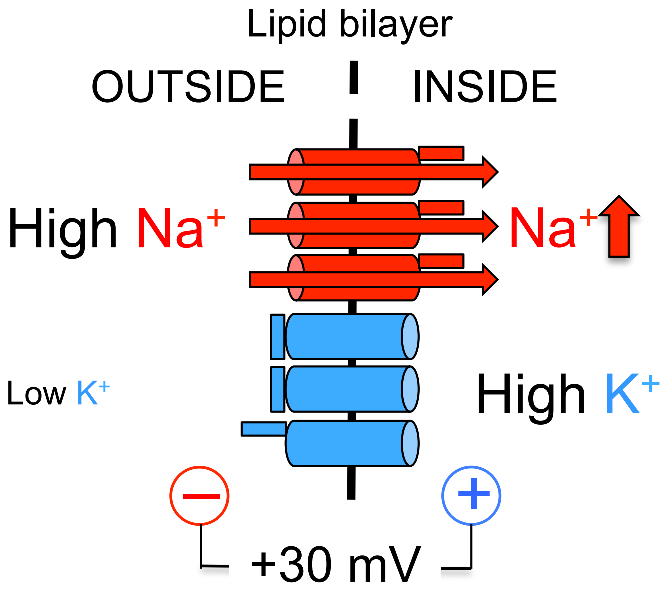


Fig. 13-19 Thibodeau and Patton 8th Ed p.396

The cell Depolarises: Na^+ enters the cell, the membrane potential becomes positive

At the threshold potential, voltage gated Na^+ channels open → further depolarisation



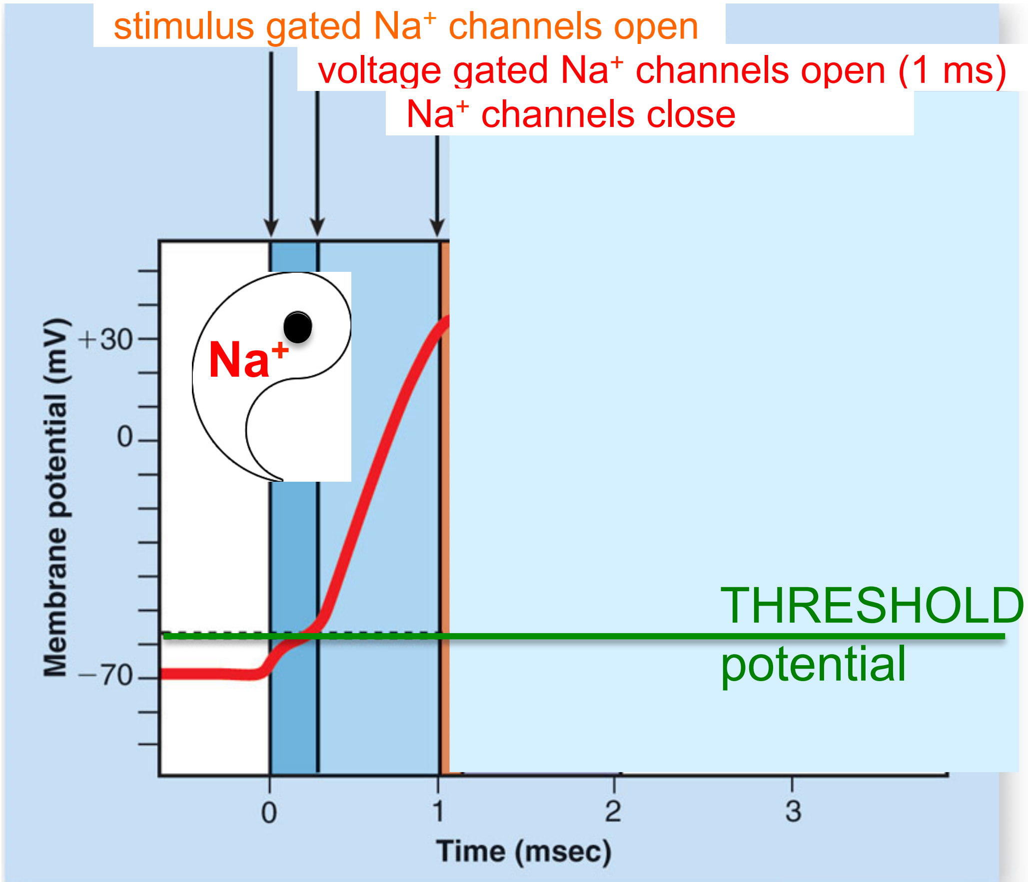
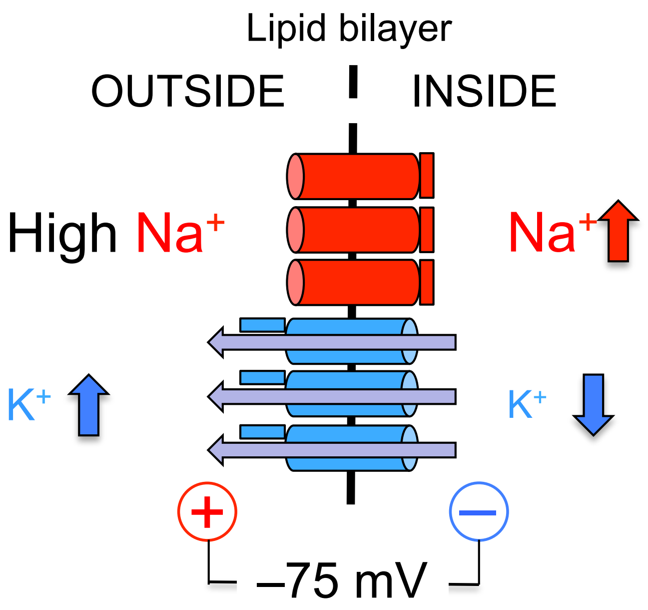


Fig. 13-19 Thibodeau and Patton 8th Ed p.396

- Voltage gated Na^+ channels open
- Once the membrane potential reaches MAXIMUM POSITIVE value the Na^+ channels start to CLOSE

The membrane potential peaks, K^+ leaves the cell to Repolarises



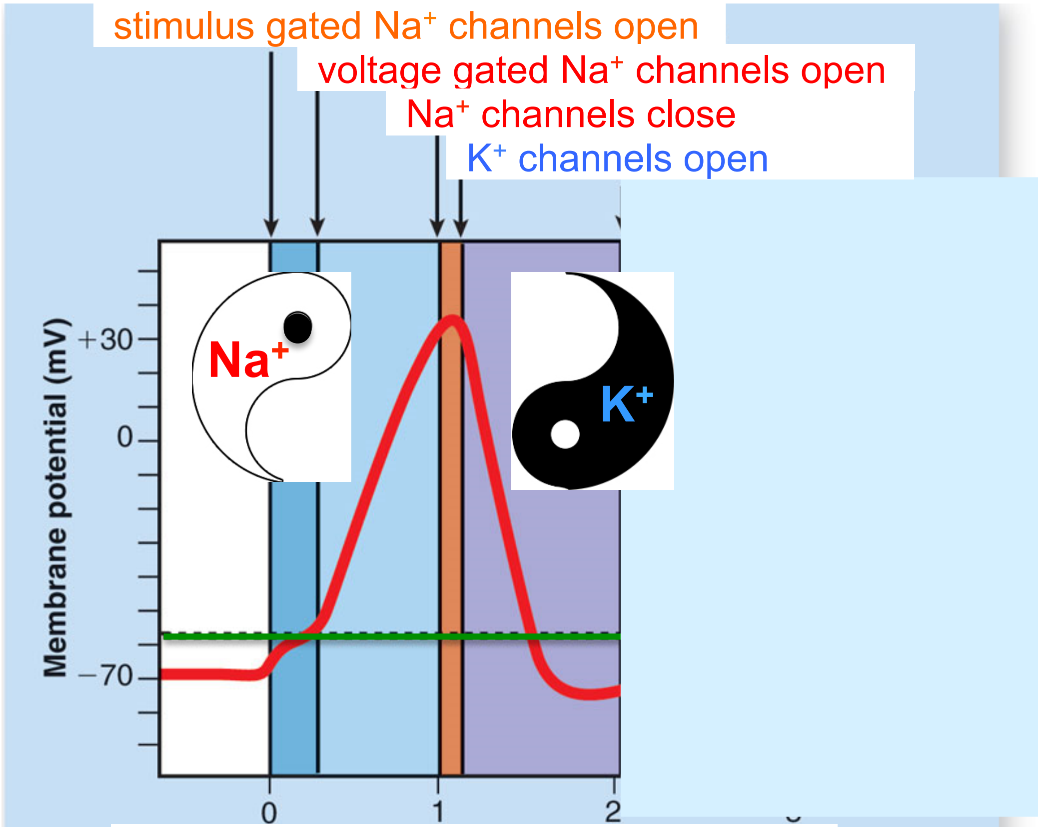
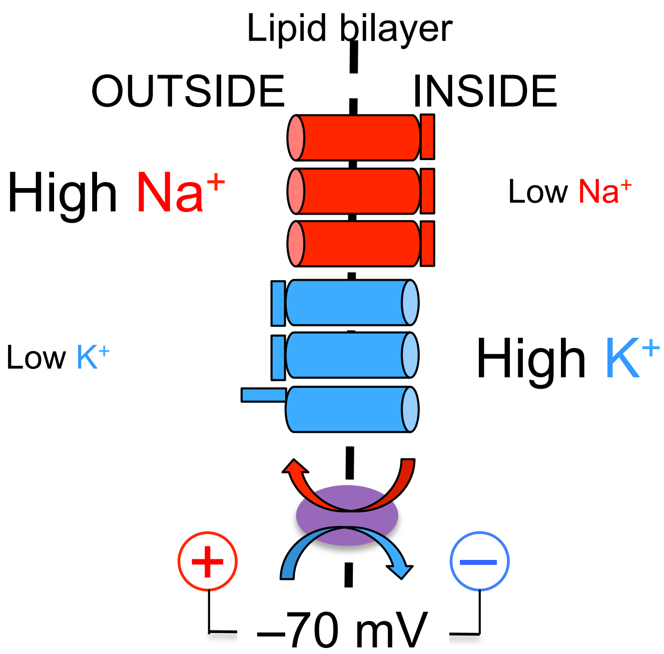


Fig. 13-19 Thibodeau and Patton 8th Ed p.396

- Na^+ channels shut
- Voltage gated K^+ channels open
- the membrane REpolarises as it becomes less positive inside (→ hyperpolarisation)
- the action potential recovers, RMP restored by Na^+/K^+ ATPase (‘recharge the battery’)

At high (–) membrane potential, K^+ channels close, the membrane potential recovers



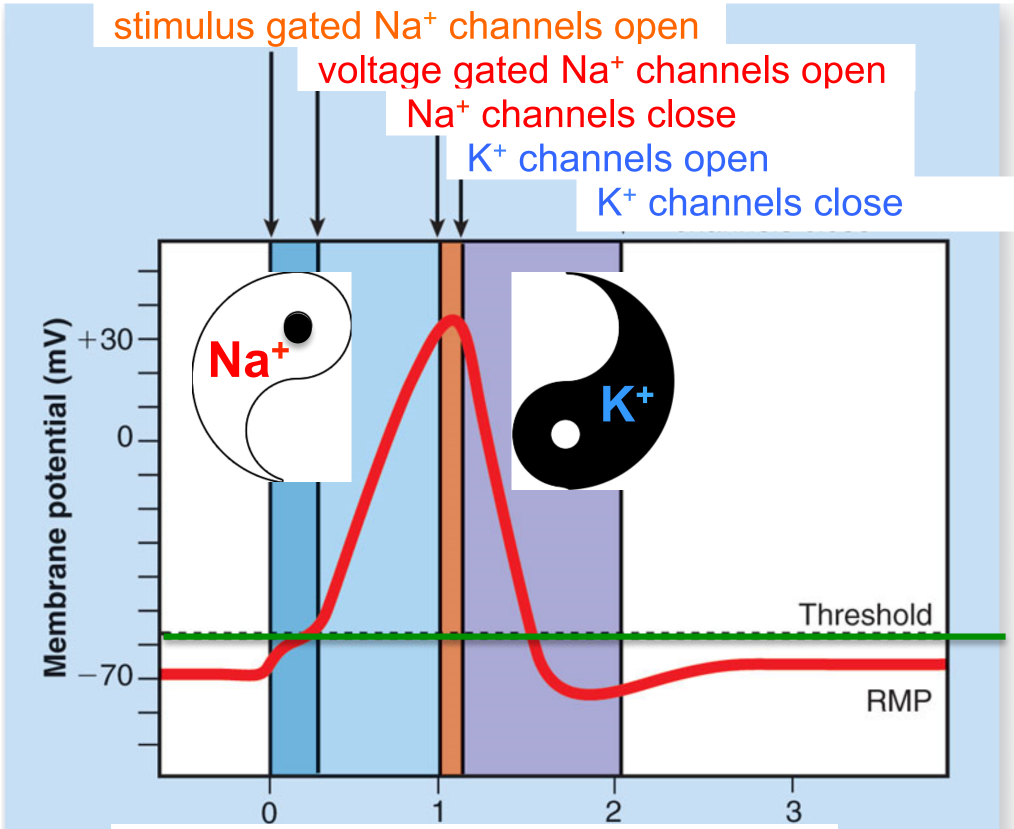


Fig. 13-19 Thibodeau and Patton 8th Ed p.396

- **Na^+/K^+ ATPase** activity
- Electrical and chemical gradients restored
- **R**esting **M**embrane **P**otential - RMP restored by Na^+/K^+ ATPase ('recharge the battery')

To summarise…

STEP	DESCRIPTION
1	A stimulus triggers stimulus-gated Na^+ channels to open and allow inward Na^+ diffusion. This causes the membrane to depolarize.
2	As the threshold potential is reached, voltage-gated Na^+ channels open.
3	As more Na^+ enters the cell through voltage-gated Na^+ channels, the membrane depolarizes even further.
4	The magnitude of the action potential peaks (at +30 mV) when voltage-gated Na^+ channels close.
5	Repolarization begins when voltage-gated K^+ channels open, allowing outward diffusion of K^+.
6	After a brief period of hyperpolarization, the resting potential is restored by the sodium-potassium pump and the return of ion channels to their resting state.

Table 13-2 from Thibodeau and Patton, 8th Ed p. 396

- **TIMING IS EVERYTHING !**
- Order of channel events → opening/closing!
- Different types of channels (e.g. stimulus, voltage-gated)

Table 13-1 from Thibodeau and Patton 8th Ed p. 395

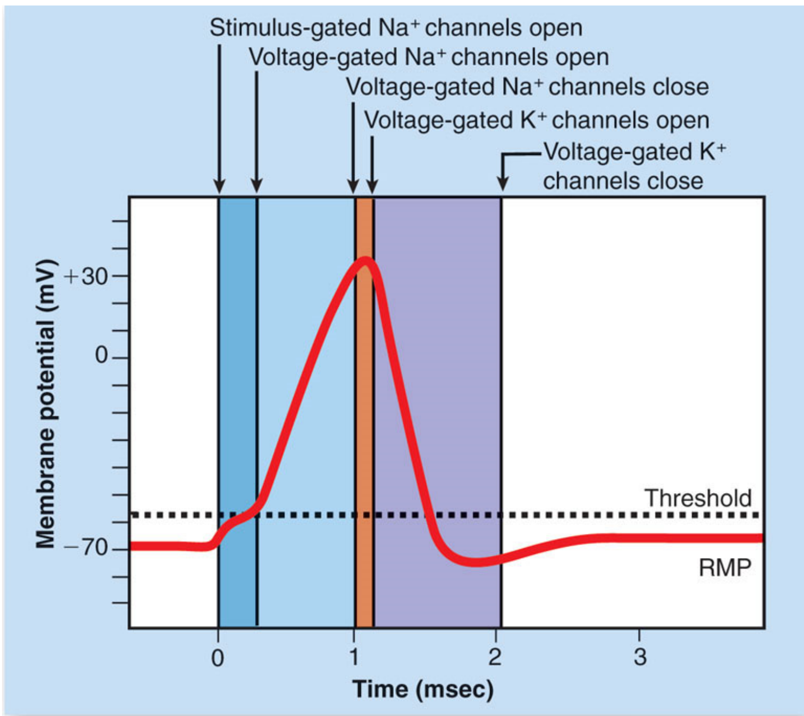


Fig. 13-19 Thibodeau and Patton 8th Ed p.396

TYPES of POTENTIALS

Type of Potential	**Polarisation**	**Typical value**	**Description**
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Threshold	DEpolarised	**-59 mV**	Minimum local depolarisation that triggers an action potential
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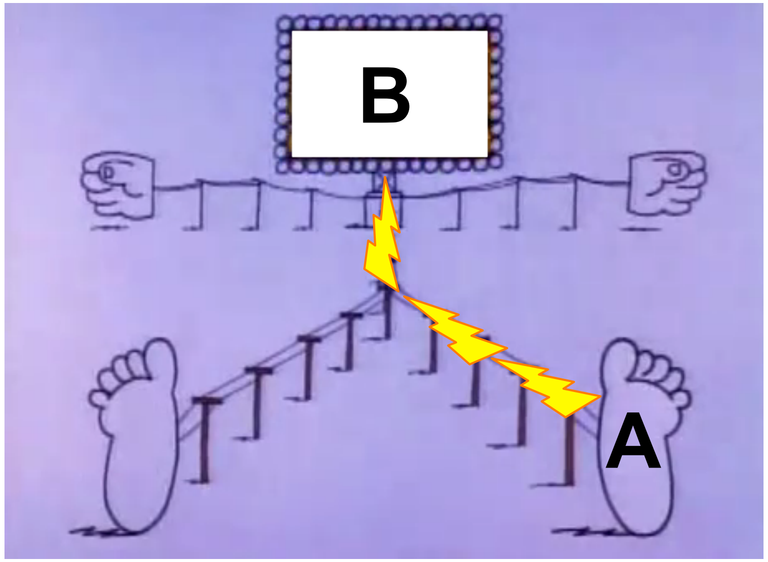
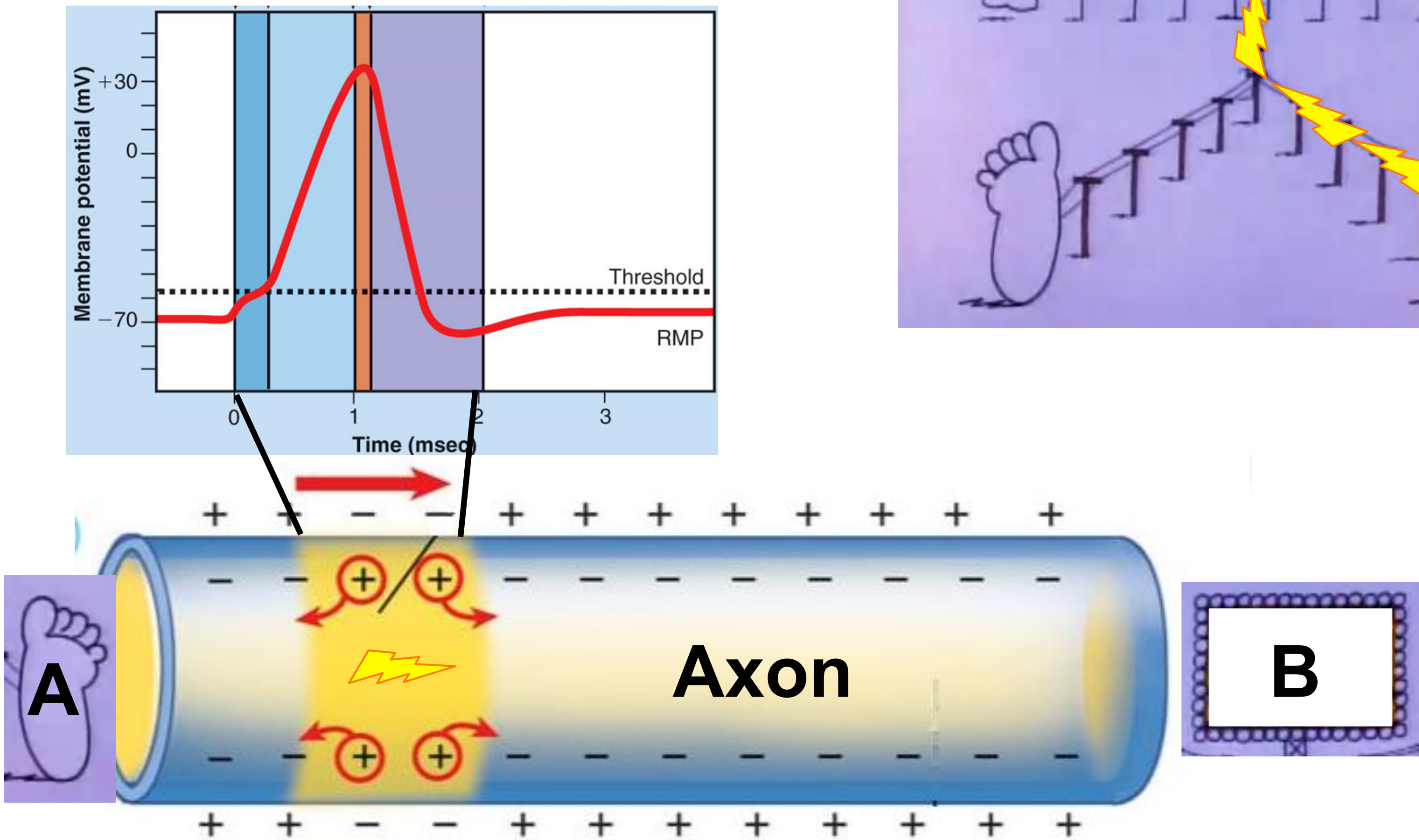
Adapted from Table 13-3 *Thibodeau and Patton p. 397*

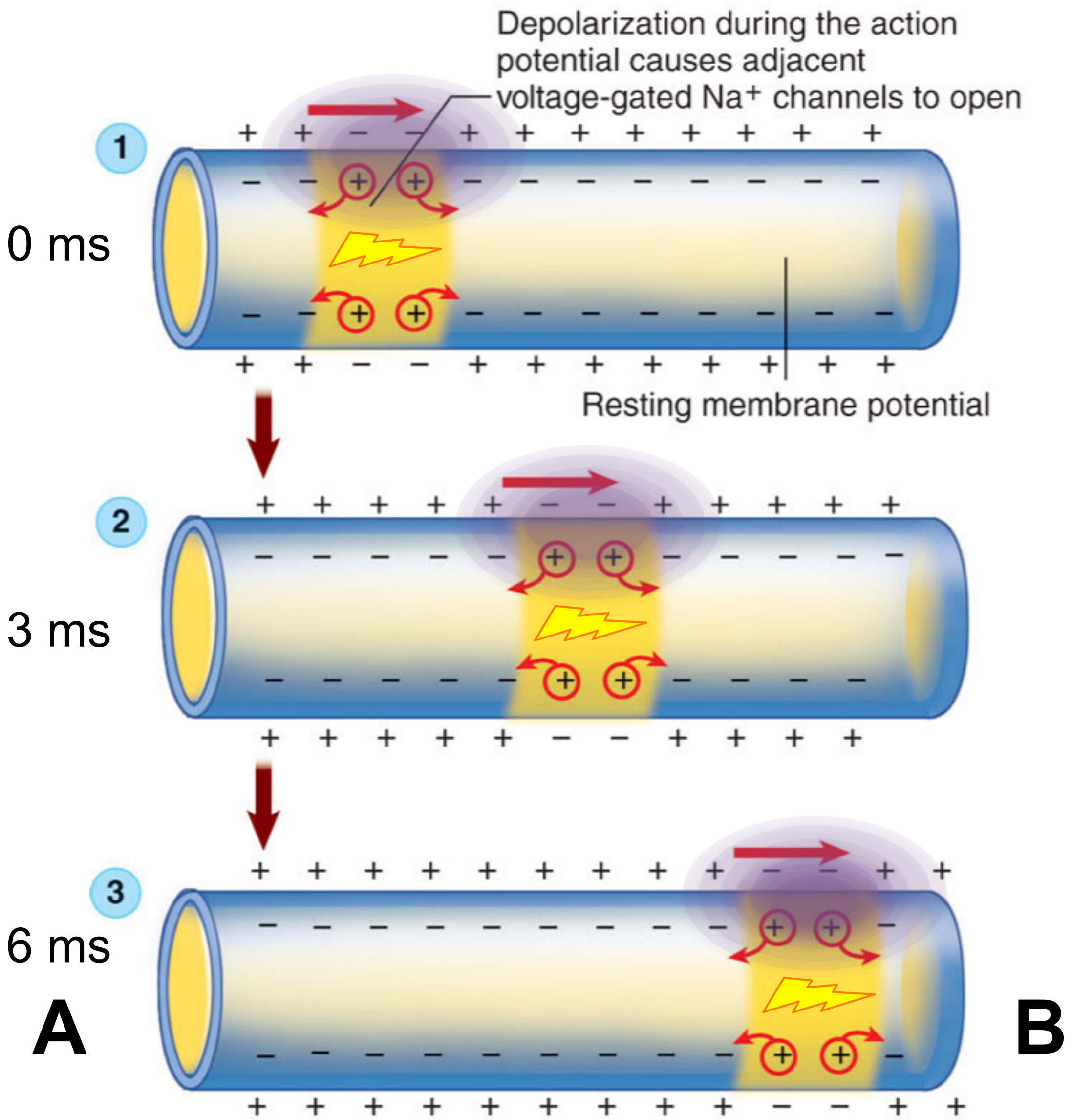
Conduction of the Action Potential down Axons





From Fig. 13-21 T+P 8th Ed p. 398

Conduction of the Action Potential down Axons



From Fig. 13-21 T+P 8th Ed p. 398

Relies on the spread of the DEPOLARISING electrical signal

Generation of an electric field

ALONG

the axon to instantly activate the next set of voltage gated Na^+ channels

Propagation of an action potential
➔ MEXICAN WAVE – or "La OLA"

Image from http://www.theage.com.au/news March 2007

What determines the propagation direction of an Action Potential

→ Refractory period

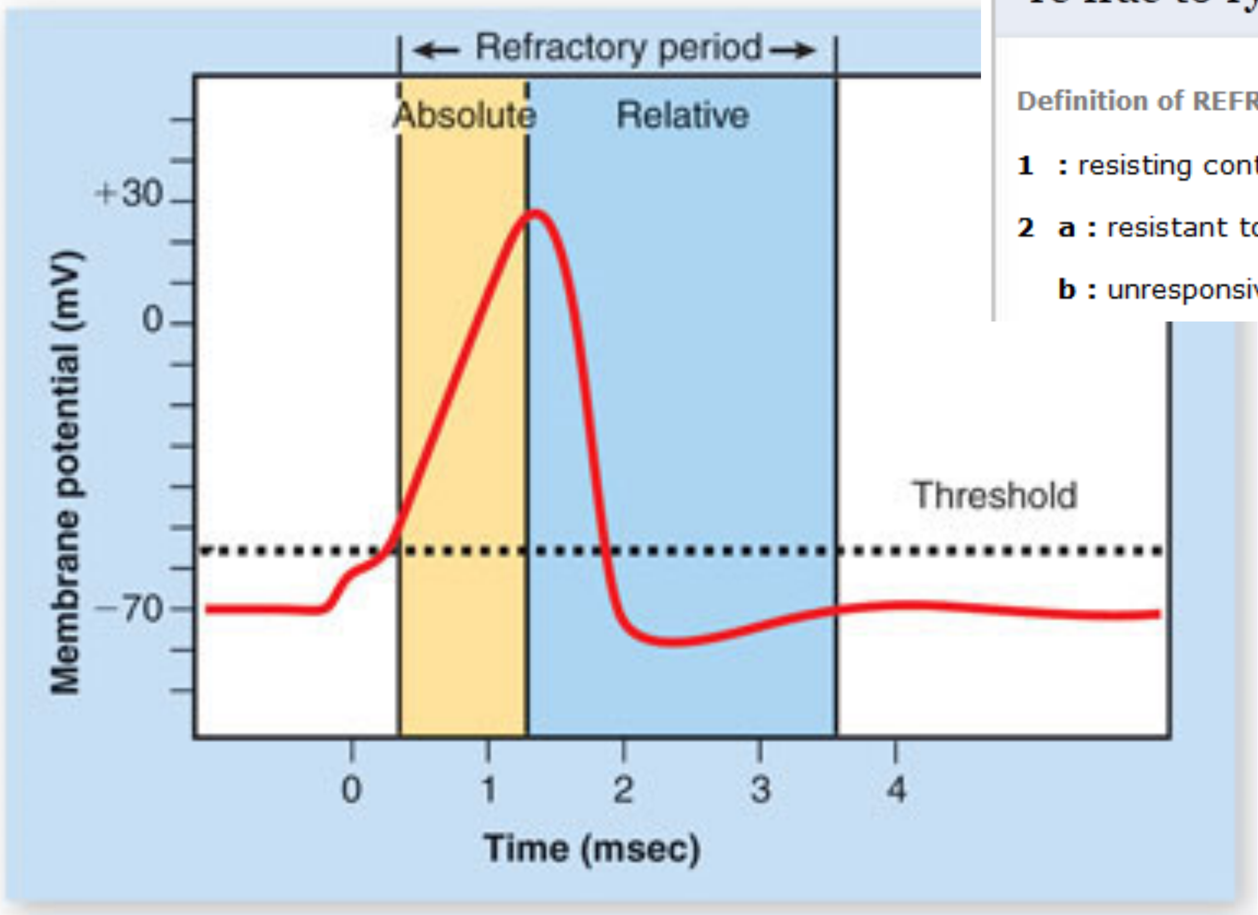
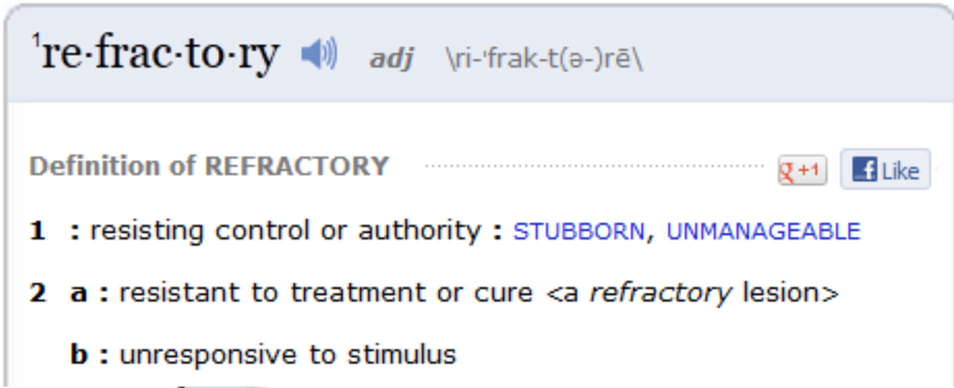


Fig. 13-20 Thibodeau and Patton 8th Ed p. 397



From http://www.merriam-webster.com

REFRACTORY PERIODS:

- **Absolute → no response**
- **Relative → large stimulus required**

REFRACTORY PERIODS:

- **Intrinsic feature of the Na^+ channels**

→ It also limits the number of action potentials at a given time (velocity of action potentials)

How does 'refractory period' work?

REFRACTORY PERIODS:

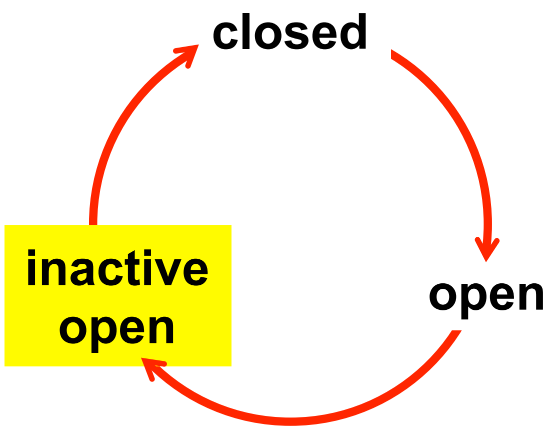
- **Intrinsic feature of the Na^+ channels**

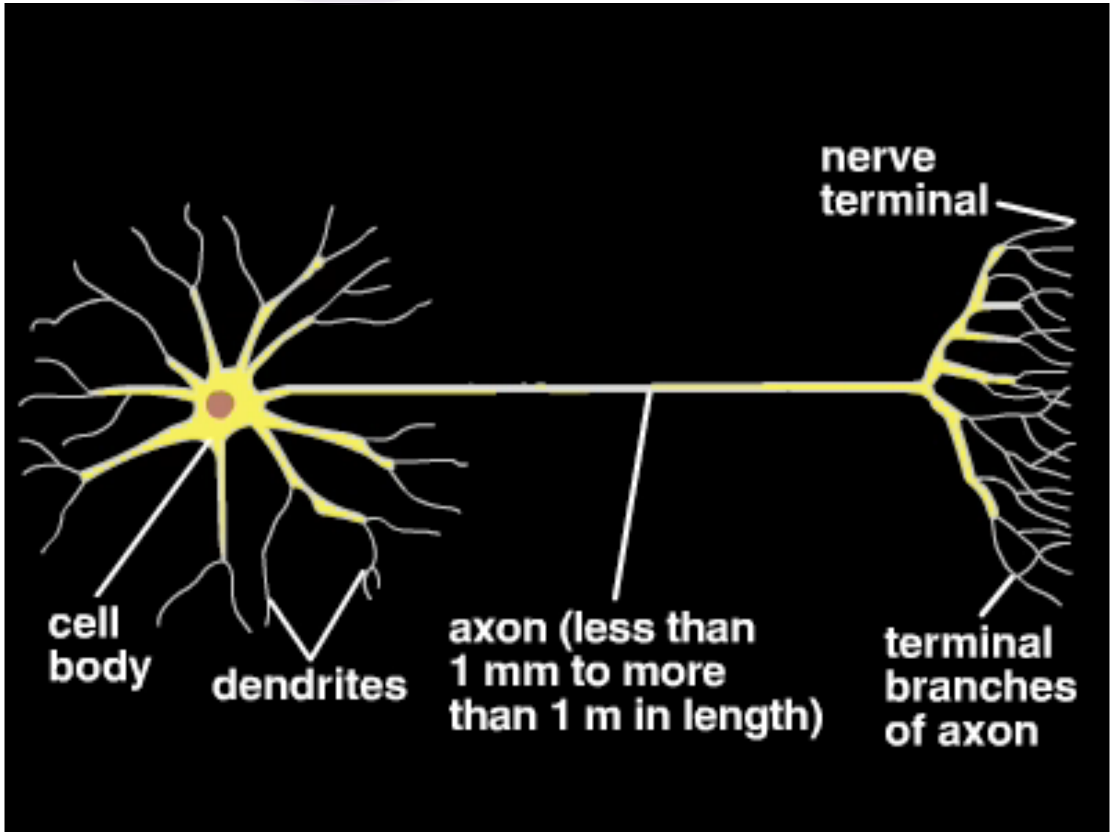
→ depend on the inactive state of the Na^+ channels following activation

→ 'inactive' channels do not respond to the electric field

→ Prevent backward movement

Activation cycle of a Na^+ channel:





Movie from: Molecular Biology of the Cell, 5th Edition, Alberts et al.

Ways to IMPROVE conduction speed

What do we mean by Speed ?

Maybe 1 m in 10 seconds 0.1 m/s

100 m in about 10 secs About 10m/s

50 km/hr is about 14 m/s

Conduction Speed of Axons

IF YOU ARE A FLY !

- An axon from a small invertebrate can conduct slowly e.g. **0.5 m/sec** Because its very small !
- **Unmyelinated axons**

IF YOU ARE A LARGE MAMMAL !

- An axon conducting at 0.5 m/sec – would be WAAAY too slow !
- Mammals: **50-70 m/sec**

→ Nature has found ways to improve action potential conduction speed!

Solution: Myelinated axons

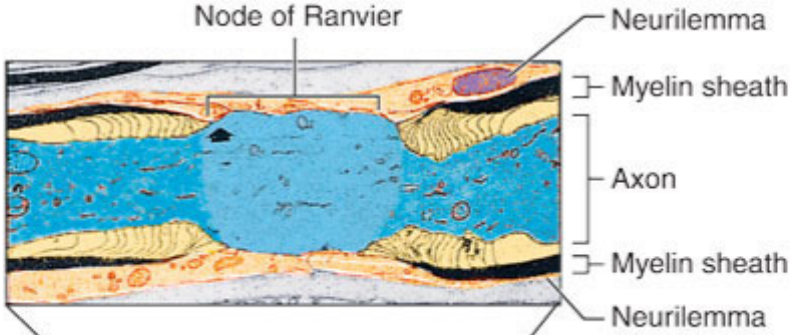
IMPROVE CONDUCTION SPEED → MYELINATED AXONS

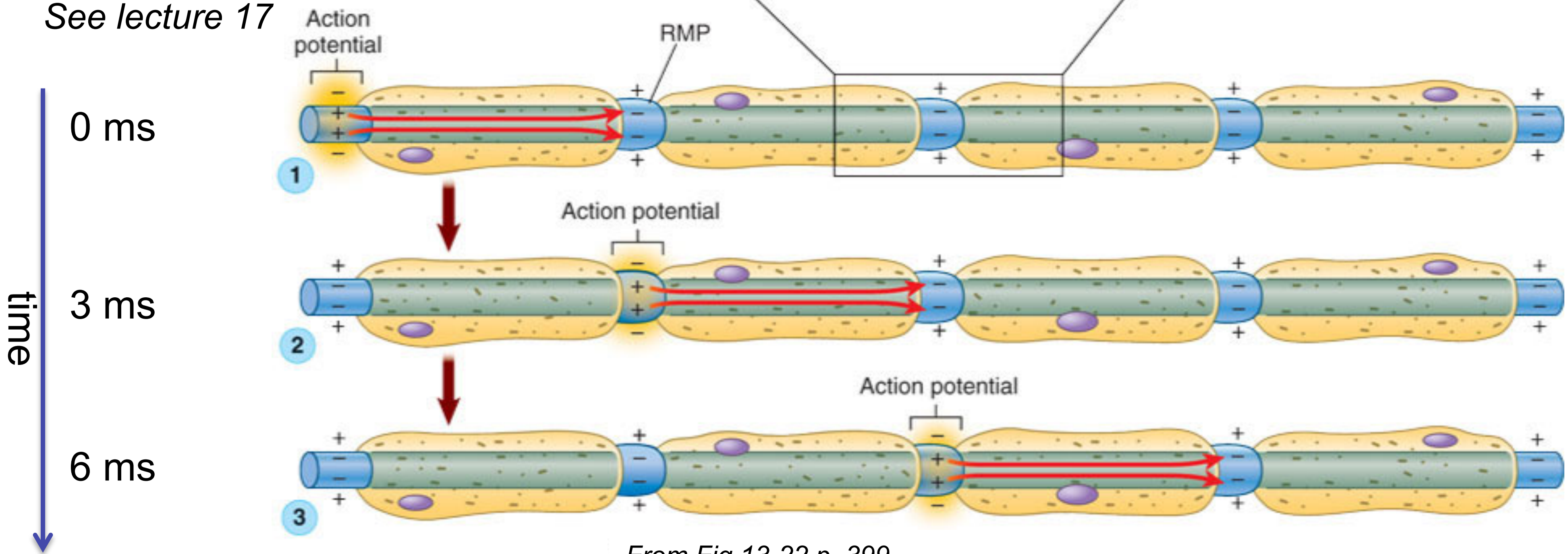
Myelination of the axon

→ Insulation of the 'wire'

- Schwann cells are a type of oligodendrocyte
- Provide insulation: **myelin sheath**

See lecture 17



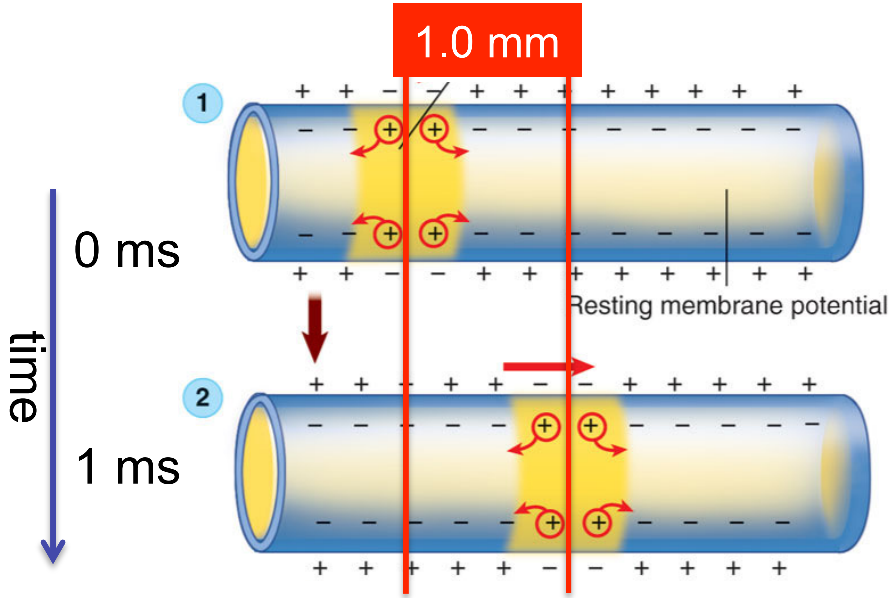


From Fig 13-22 p. 399

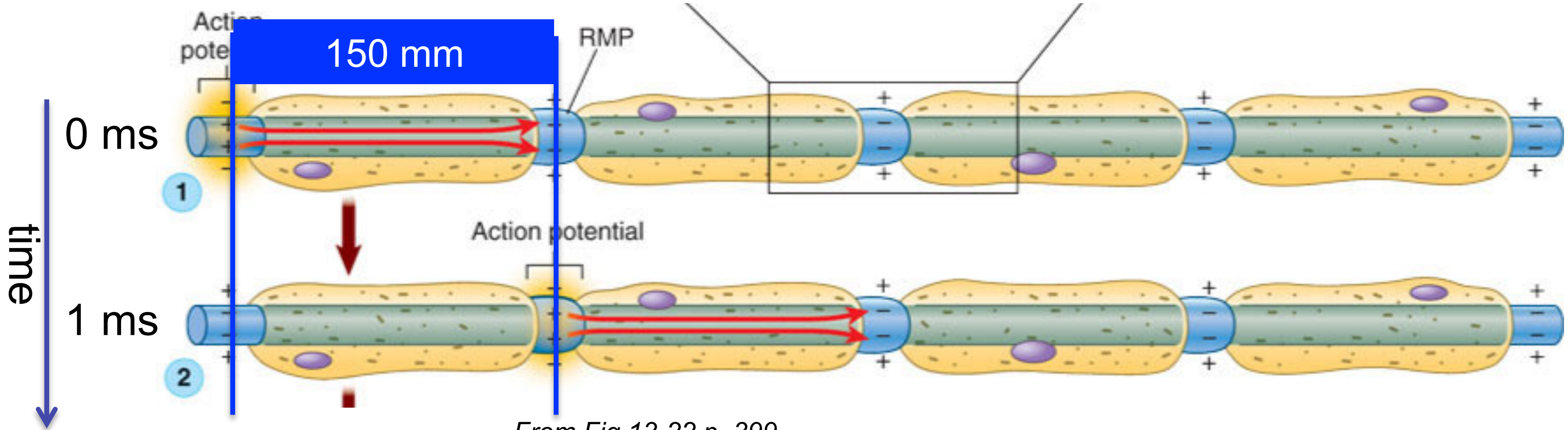
The action potential leaps between the nodes of Ranvier – saltatory conduction

Unmyelinated vs myelinated

Saltatory – latin: ‘saltare’, to leap



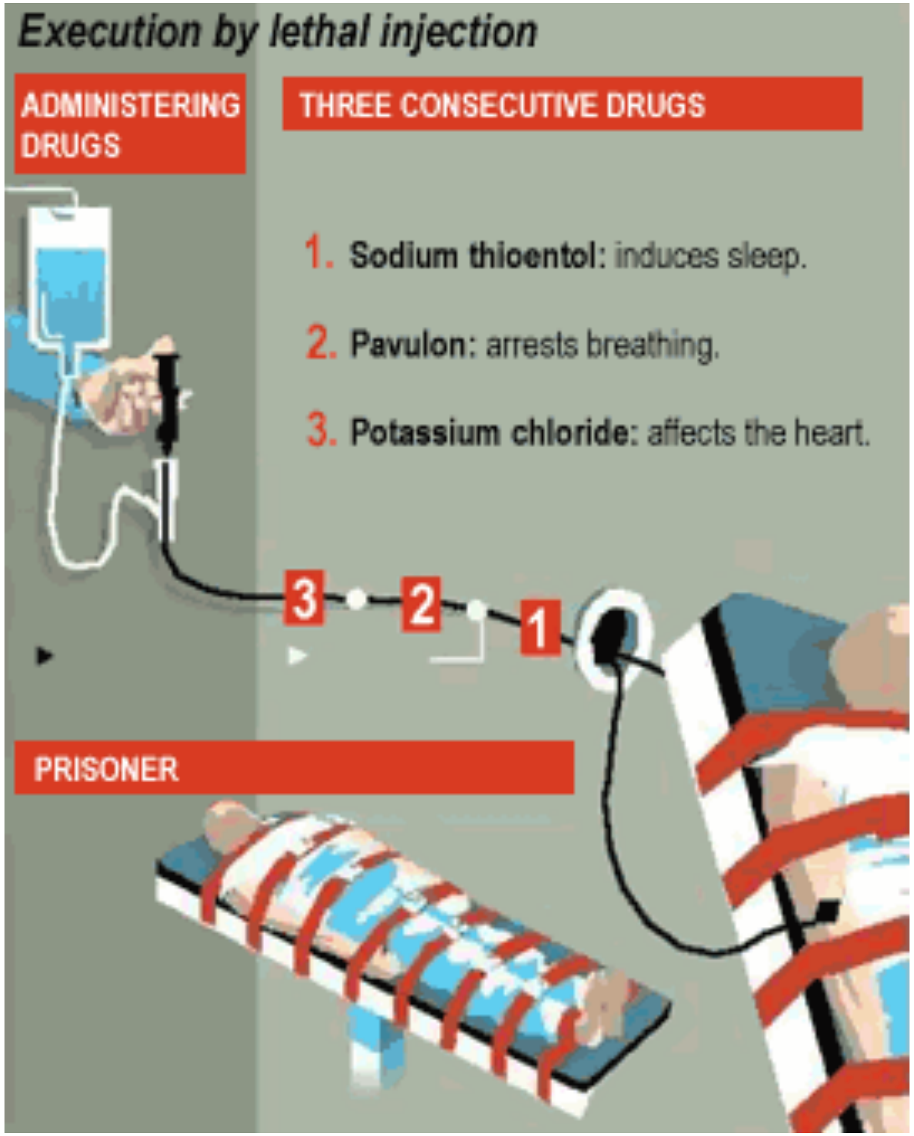
From Fig. 13-21 T+P 8th Ed p. 398



From Fig 13-22 p. 399

More distance at the same time!!!

Something to think about…



Why is KCl lethal ?

WHY ?????

From http://www.chm.bris.ac.uk/webprojects2006/Macgee/Web%20Project/lethal_injection.htm

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