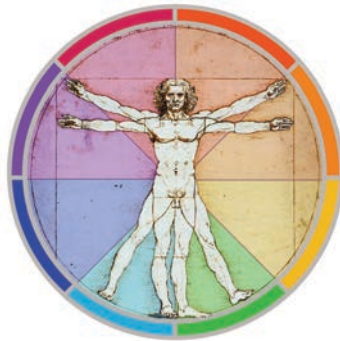


# HUBS191 Lecture Material

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



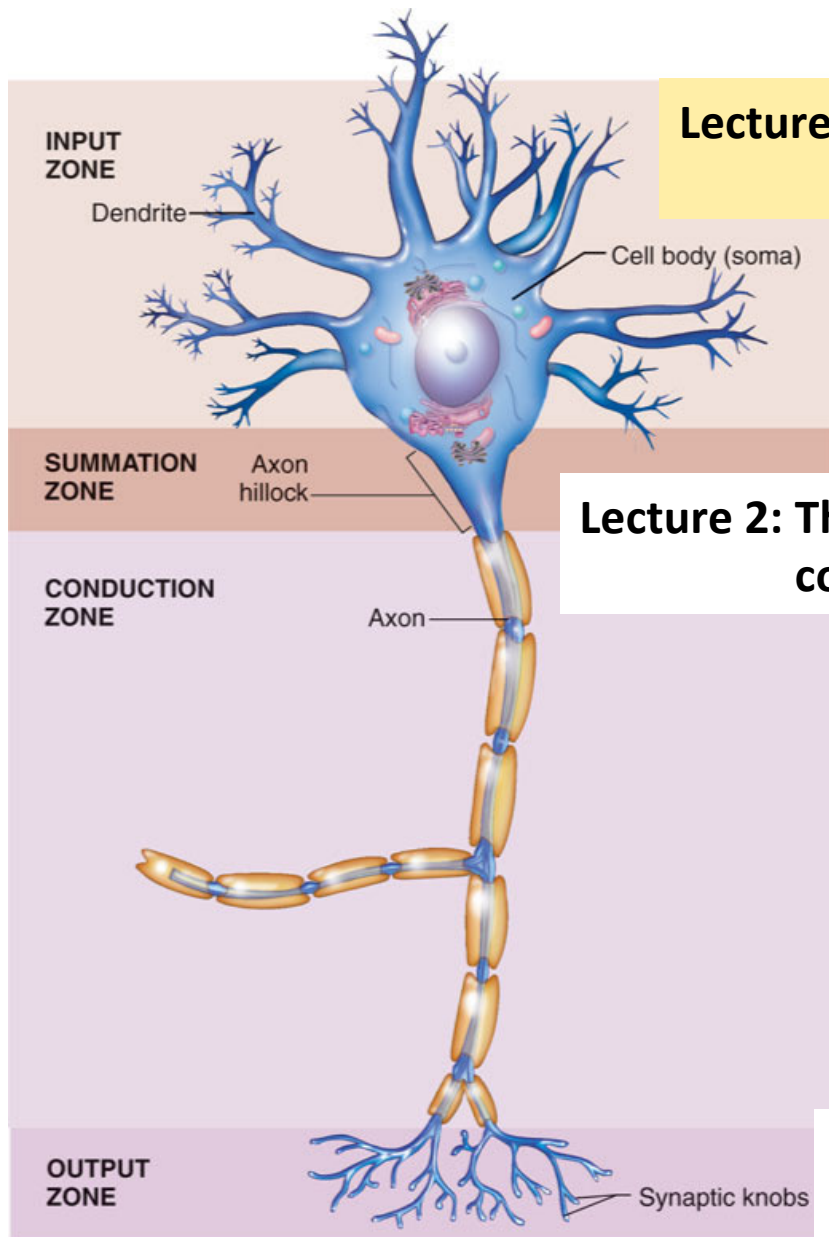
## **HUBS191**

### **Lecture 24**



Dr Martin Fronius  
Department of Physiology  
7. April 2017

# Aims of Today's Lecture



**Lecture 4: Synaptic Networks and Integration  
(input zone)**

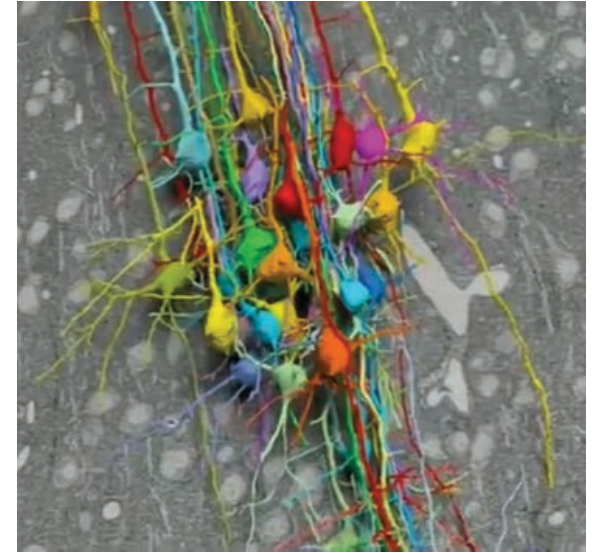
**Lecture 2: The Action Potential and its  
conduction (axon hillock, axon)**

**Lecture 1: Bioelectricity and membrane  
potentials**

**Lecture 3: The Synapse – ‘connecting’ neurons  
(output zone)**

# Aims of Today's Lecture

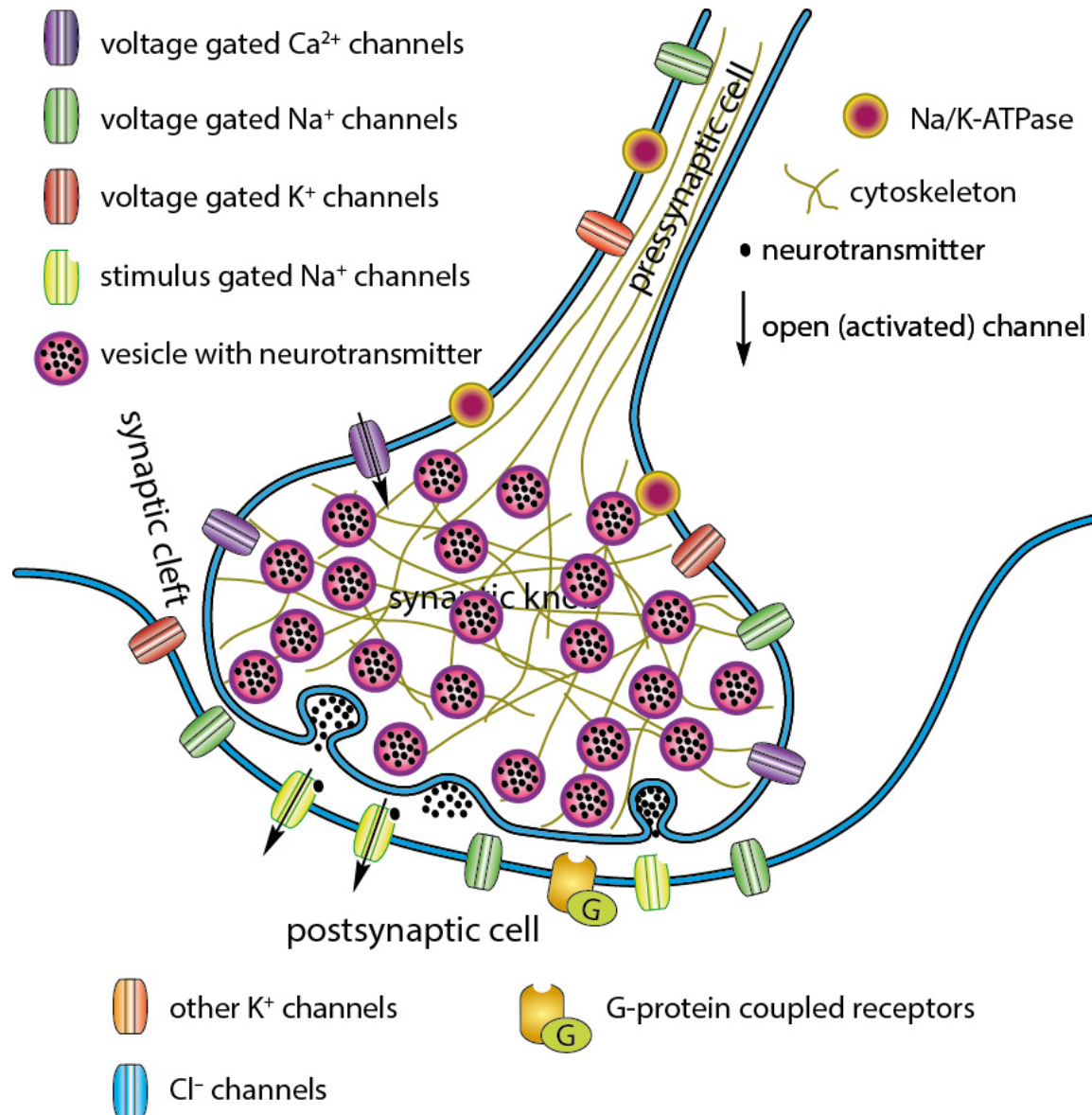
## Lecture 24 – Synaptic Networks and Integration



### Outline

- Recap from Lecture 24
- Excitatory and Inhibitory Transmitters and Synapses
- Synaptic Integration – Types of Summation (spatial and temporal)
- Types of neuronal network (Divergence, Convergence)

# The chemical synapse



1. Presynaptic cell 'output zone'
2. Synaptic knob
3. Synaptic cleft 'the gap'
4. Postsynaptic cell 'input zone'
5. Voltage-gated  $\text{Ca}^{2+}$  channels
6. Vesicles
7. Cytoskeleton
8. Na/K-ATPase
9. Mitochondria (not shown)

# The process of synaptic transmission – form and function

1. Action potential propagates down the axon – to the pre-synaptic knob
2. Pre-synaptic knob is depolarised – voltage gated  $\text{Ca}^{2+}$  channels open
3.  $\text{Ca}^{2+}$  ions enter and TRIGGER the release of the neurotransmitter from the vesicles
4. Neurotransmitter is released INTO the synaptic cleft
5. Neurotransmitter diffuses across the cleft and binds to its SPECIFIC receptors (stimulus gated channels) on the POST SYNAPSE
6. If  $\text{Na}^+$  channels open – LOCAL depolarisation of post synaptic cell
7. Net depolarisation – called the

EXCITATORY POST SYNAPTIC POTENTIAL – or **EPSP**

- 7'. Net hyperpolarisation (opening of  $\text{Cl}^-$  or  $\text{K}^+$  channels) – called the

INHIBITORY POST SYNAPTIC POTENTIAL – or **IPSP**

# HOW IS A SYNAPSE SWITCHED OFF ?

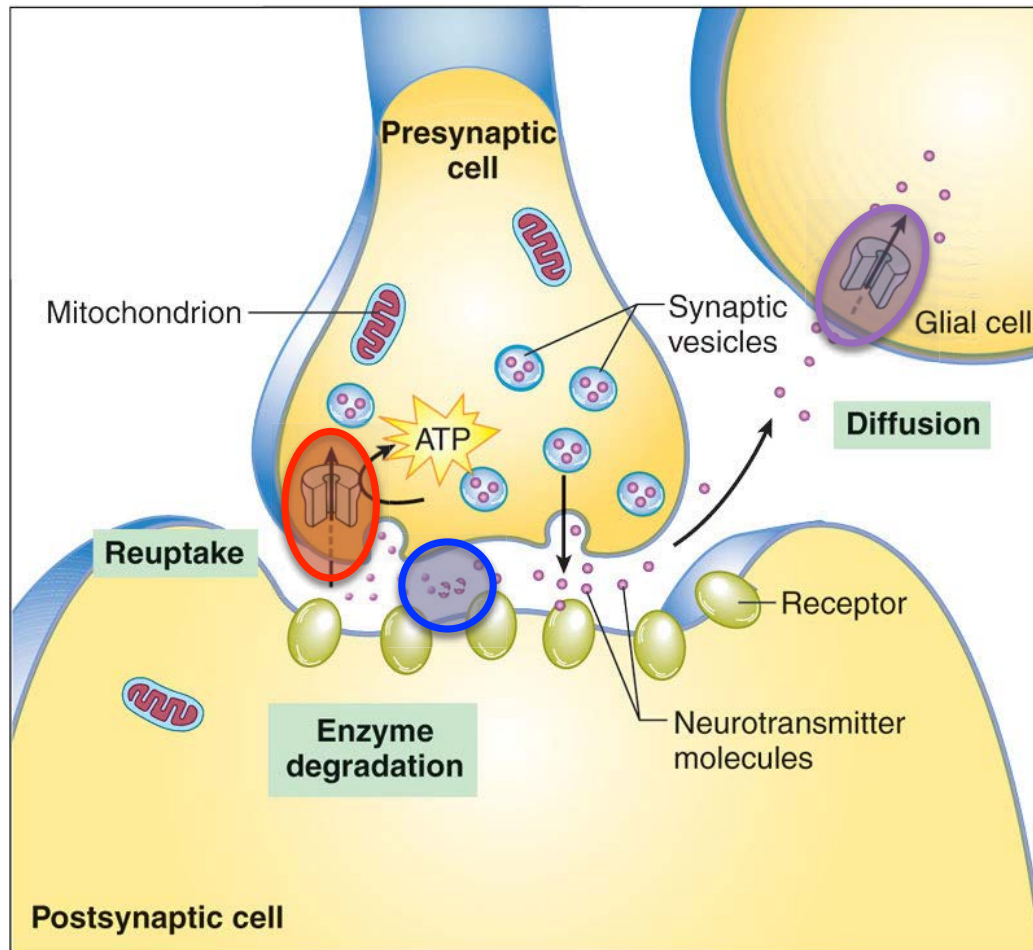


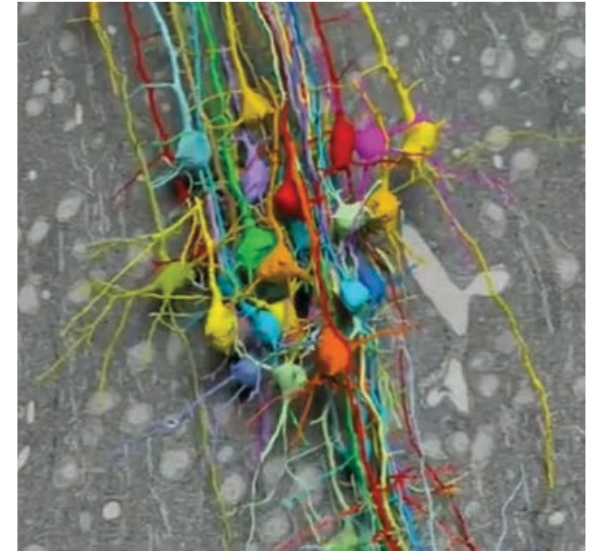
Fig. 13-26, Patton & Thibodeau, 8<sup>th</sup> Ed p. 402

- Excess transmitter released into the cleft.
- Excess transmitter must be removed
    - a. **Degradation** – by enzymes
    - b. **Reuptake** into the knob
    - c. Reuptake (diffusion) into **glia cells**  
(GLM3 – revisit)
  - Removal requires ATP – energy
  - Mitochondria in synaptic knob ...

Glia – the supporting cells – and revisit p. 384 T&P 8<sup>th</sup> Ed.

# Aims of Today's Lecture

## Lecture 24 – Synaptic Network



<http://www.nature.com/news/>

### Outline

- Recap from Lecture 24
- Excitatory and Inhibitory Transmitters and Synapses
- Synaptic Integration – Types of Summation (spatial and temporal)
- Types of neuronal network (Divergence, Convergence)

# Objectives

- List the main types of neurotransmitter and name some examples
- Explain the different types of synaptic integration
- Know the two types of synaptic network and explain their different advantages

# Types of Neurotransmitters



## Some Neurotransmitters:

- Acetylcholine (**EPSP**)
- Glutamate (**EPSP**)
- GABA (**IPSP**)
- Norepinephrine  
/Noradrenaline
- Dopamine
- Serotonin

See p. 407 P&T 8<sup>th</sup> Ed

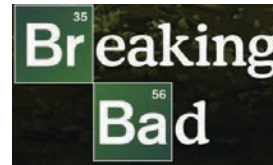
**A classical neurotransmitter is released from vesicles from within the presynaptic knob in response to  $\text{Ca}^{2+}$  influx**

# Drugs of abuse & neurotransmitter



from google.com

# Drugs of abuse & neurotransmitter



- Methamphetamine – P  
aka as 'crystal meth' or 'speed'
- Increases levels of
  - noradrenaline,
  - dopamine,
  - serotonin
- Blocks **reuptake**

Stimulates fight, flight, fright response

Acts in reward centres → **highly addictive**

Highly Neurotoxic!!!



Pervitin tablet container  
used in WW2 (en.wikipedia.org)

Obetrol: diet pill in the 1959/60  
in America



Image from <http://www.webinfo-net.com>

# Drugs of abuse & neurotransmitter

Almost all drugs target (hijack) endogenous neurotransmitter systems!

- Nicotine → nicotinic acetylcholine receptors (in the brain)
- Heroin → opioid receptors (endogenous endorphins peptides)
- Tetrahydrocannabinol → Cannabiod receptors (brain)

# Types of Neurotransmitters

EXCITATORY – cause **DEpolarisation**

- **Acetylcholine** – neuromuscular junction & brain,  
→ activates stimulus-gated  $\text{Na}^+$  channels (ionotropic receptors)
- **Glutamate** – most common excitatory neurotransmitter in CNS  
→ activates stimulus-gated  $\text{Na}^+$  channels  
→ activates stimulus gated  $\text{Ca}^{2+}$  channels

**EXCITATORY POST SYNAPTIC POTENTIALS - EPSPs**

**...due to the inflow of positive charged ions ( $\text{Na}^+$  and  $\text{Ca}^{2+}$ )!**

# Types of Neurotransmitters II

INHIBITORY – cause **HYPER**polarisation

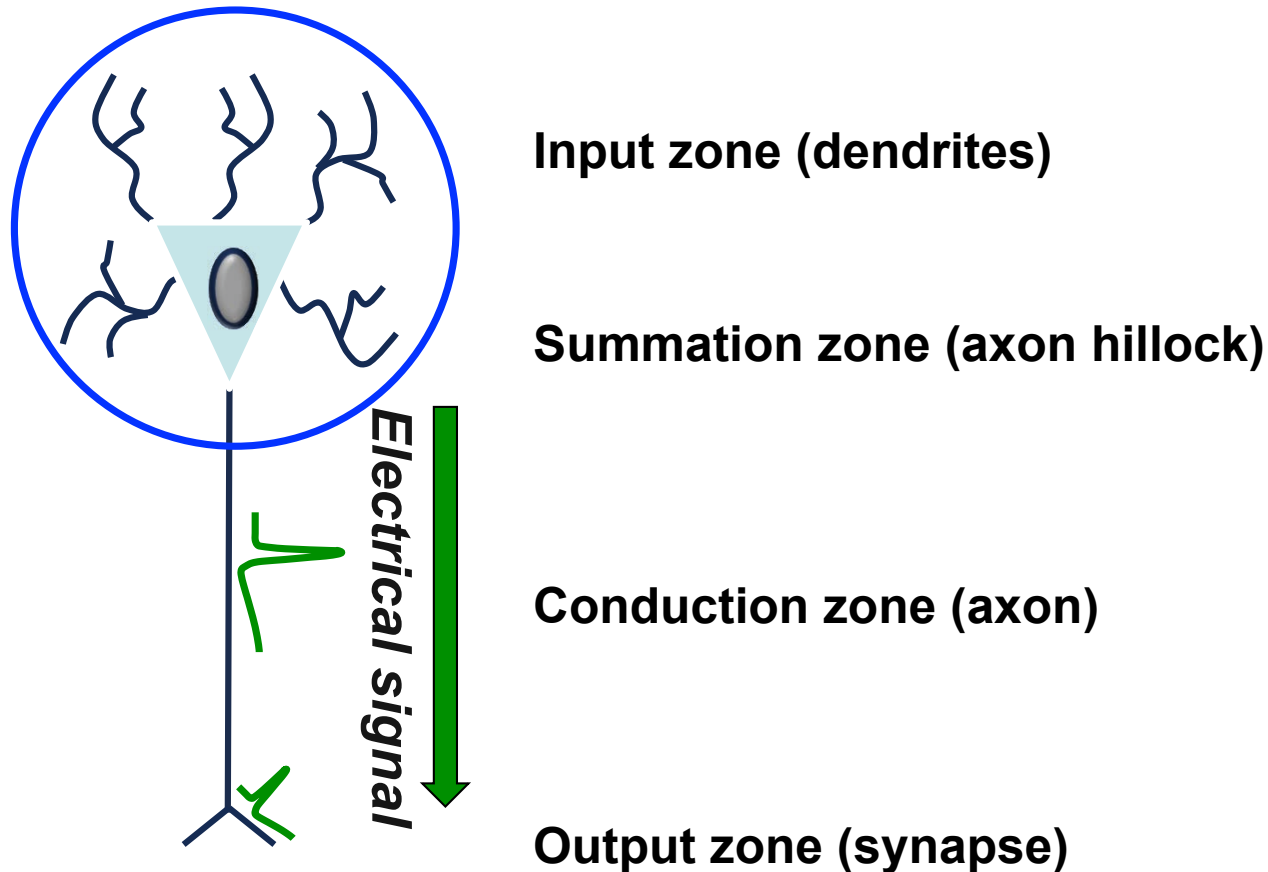
- **GABA** – gamma amino butyric acid,  
most common inhibitory neurotransmitter in brain.  
→ activates  $\text{Cl}^-$  channels (ionotropic receptor)  
→ activates  $\text{K}^+$  channels (metabotropic receptor)

**INHIBITORY POST SYNAPTIC POTENTIALS - **IPSPs****

**...due to the inflow of  $\text{Cl}^-$  or outflow of  $\text{K}^+$ !**

# ***Recap – Cells of the nervous system:***

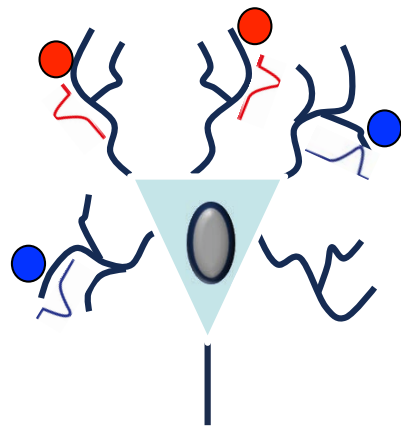
## **Neuron, basic structure**



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

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

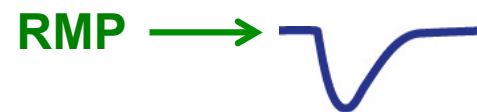
# Input zone – electrical signals in the postsynaptic neuron are EPSPs or IPSPs



- **Excitatory synapse**
  - excitatory neurotransmitter (presynaptic)
  - a lot of  $\text{Na}^+$  channels (postsynaptic)
  - local depolarisation
  - EPSP

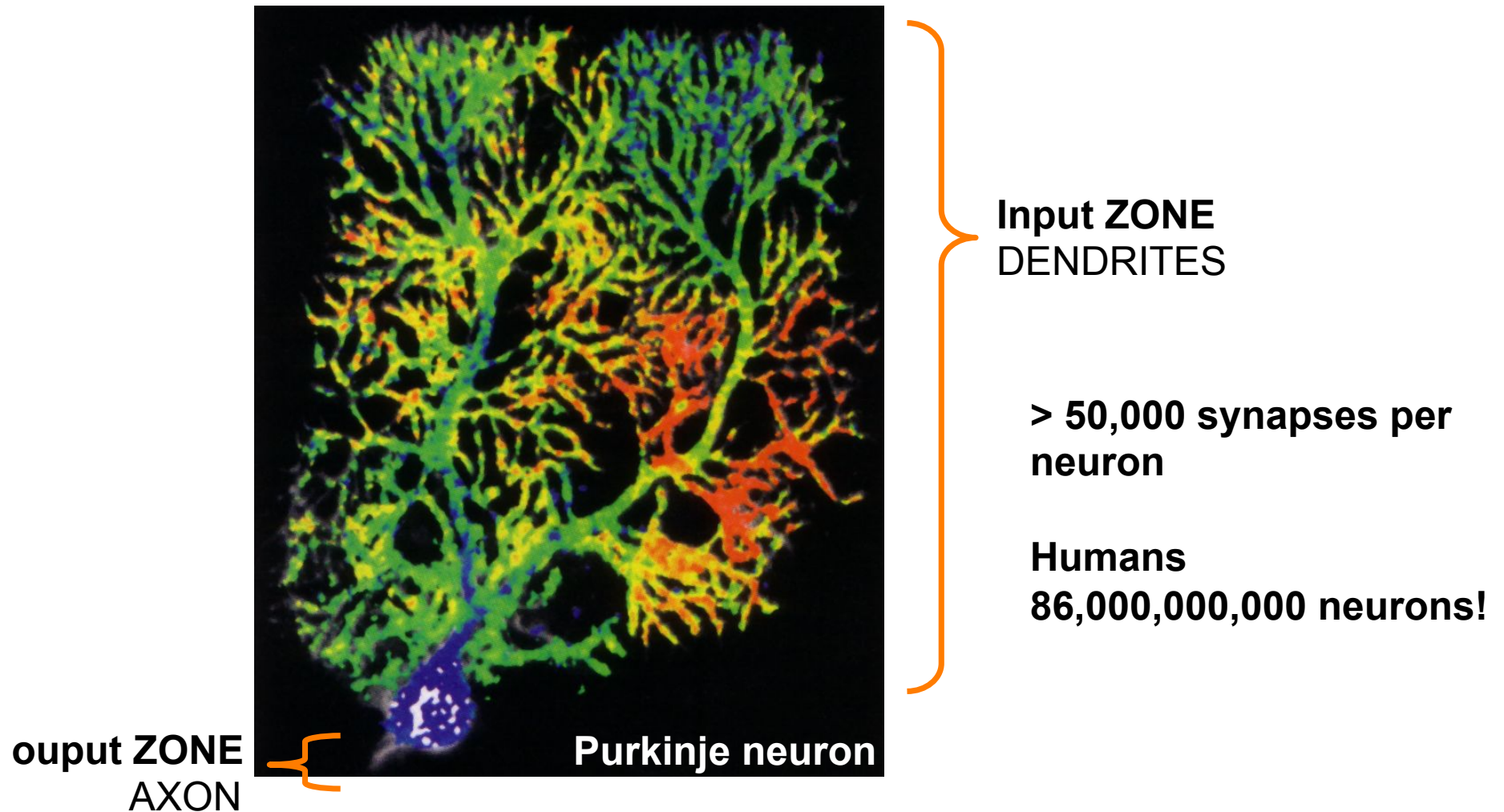


- **Inhibitory synapse**
  - inhibitory neurotransmitter (presynaptic)
  - $\text{Cl}^-$  and  $\text{K}^+$  channels (postsynaptic)
  - local hyperpolarisation
  - IPSP



**EPSPs and IPSPs are 'Local' potentials!**  
**And are caused by stimulus gated channels!**

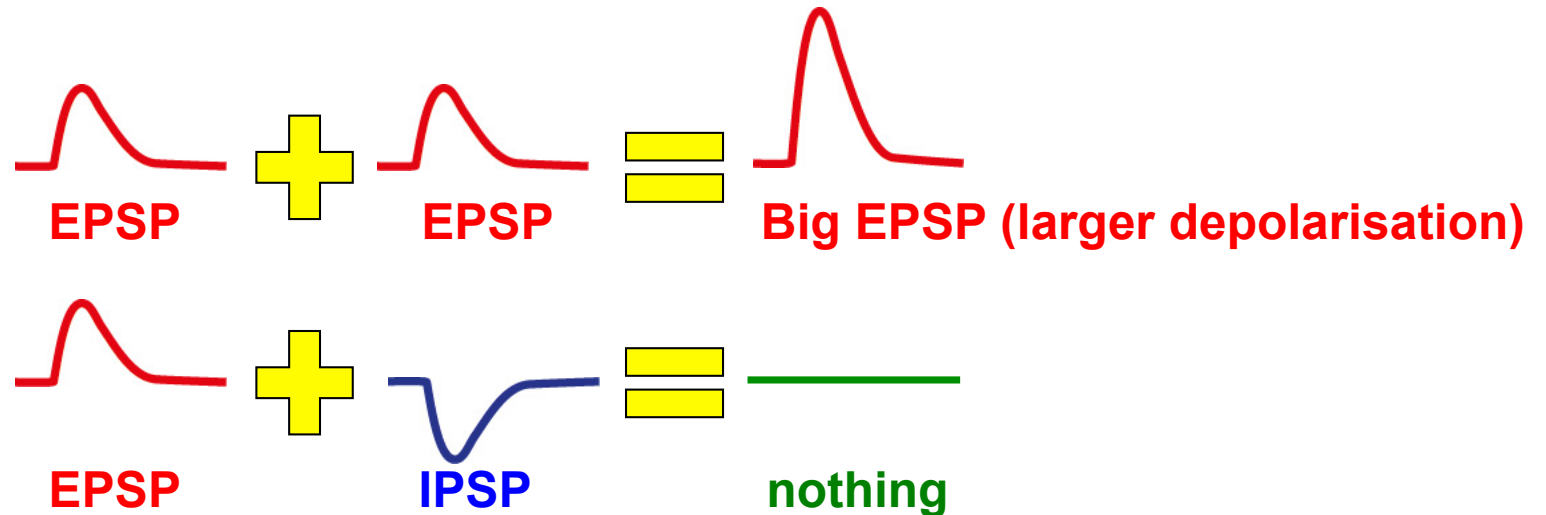
# Input Zone - Synaptic Summation



Local Synaptic Potentials (**EPSPs** and **IPSPs**) ADD UP  
– or SUMMATE within the Dendrites

# Two types of summation

Basic concept of summation (simple math):



## Spacial Summation

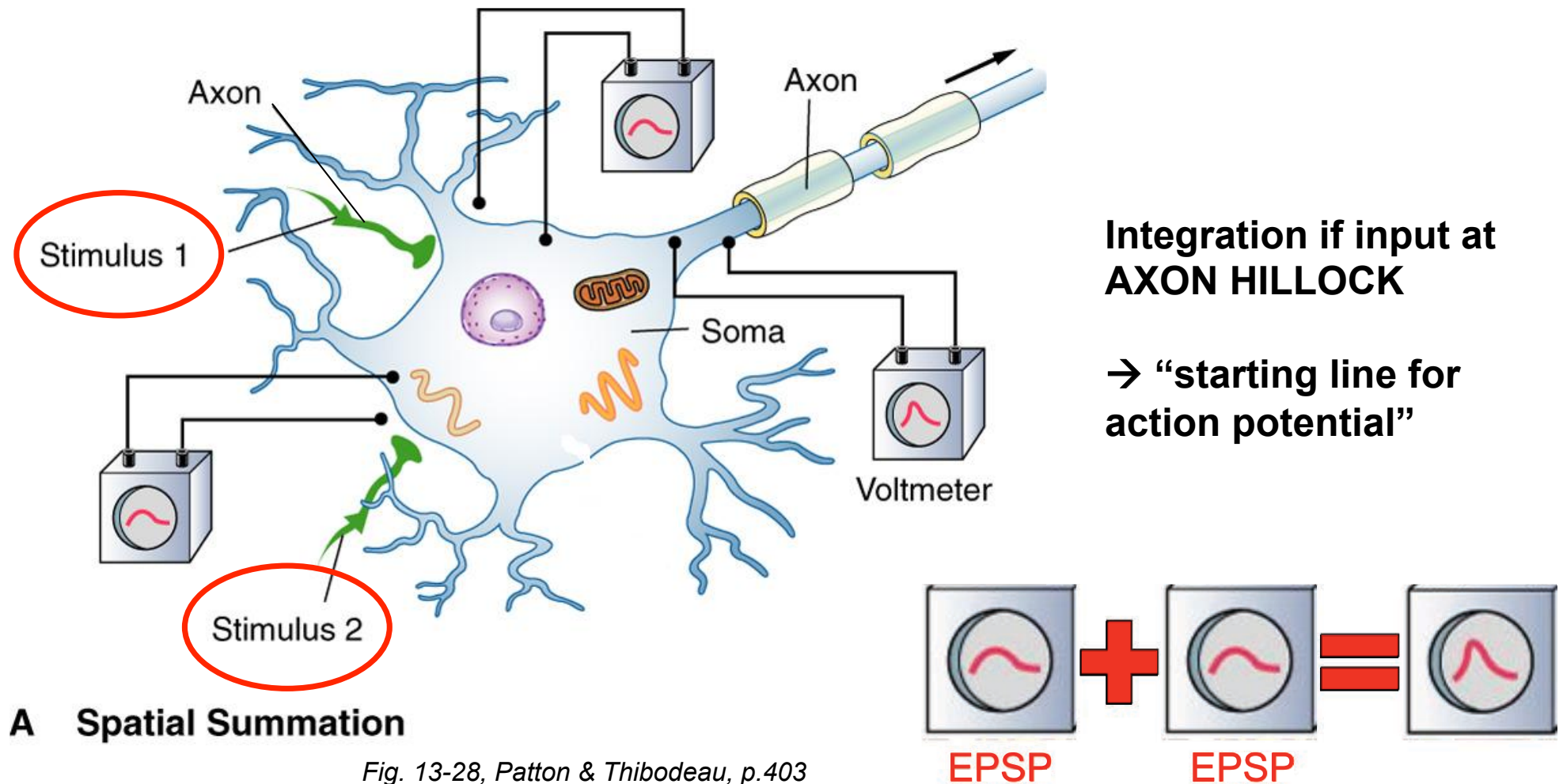
Spatial: latin – space, engl. space

Integration of inputs from different synaptic knobs (different localisation)

## Temporal Summation

Time dependent – integration of inputs within a short time  
at the same synaptic knob

# Spatial Summation of synaptic inputs



Integration of inputs from different synaptic knobs  
→ different localisations of the input zone

# AP generated and transmitted when neuron is above threshold...

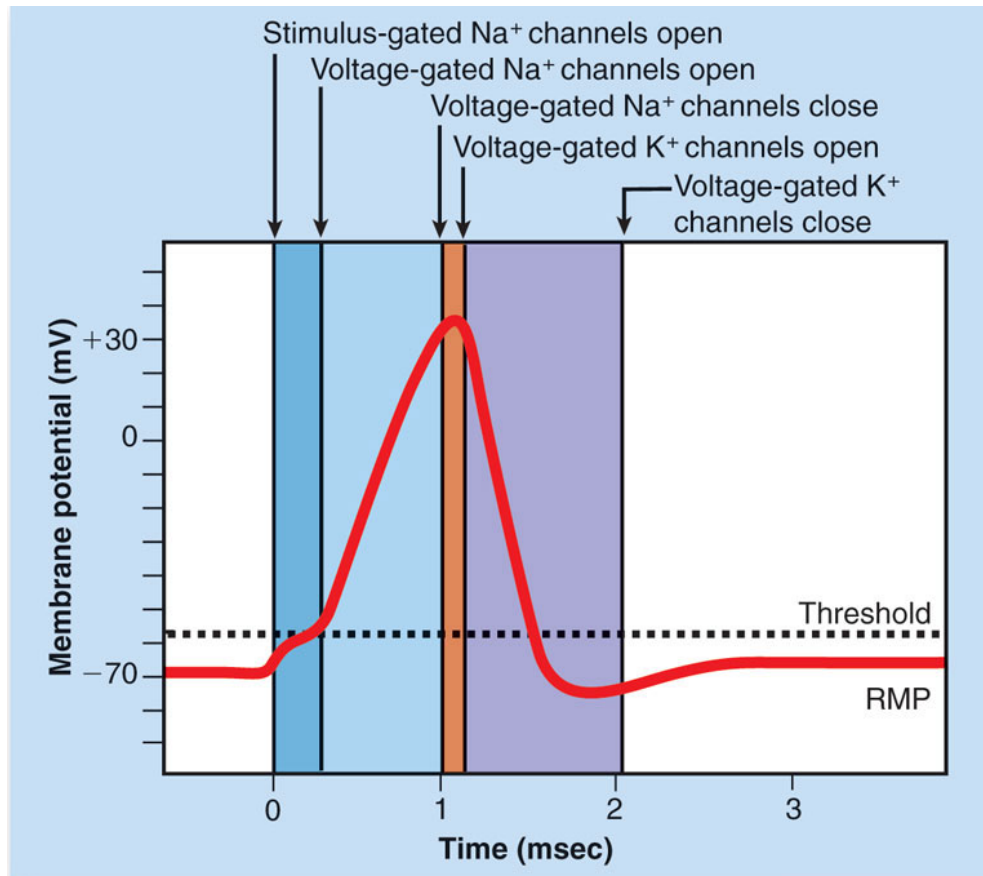


Fig. 13-19 Patton & Thibodeau, 8<sup>th</sup> Ed p.396

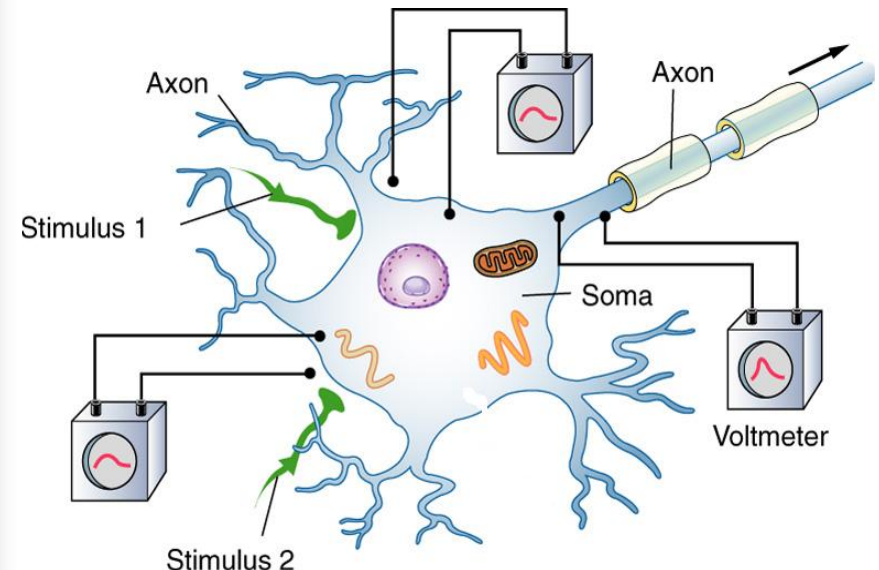
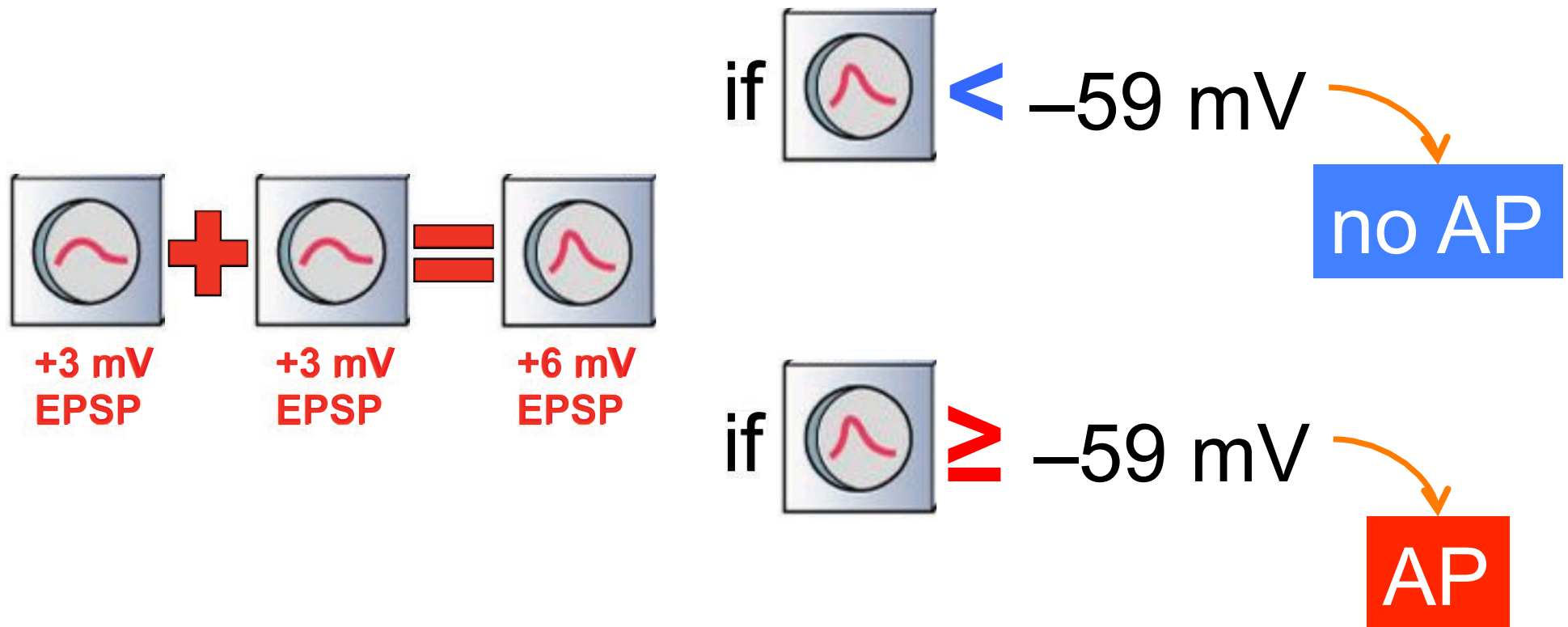


Fig. 13-28, Patton & Thibodeau, p.403

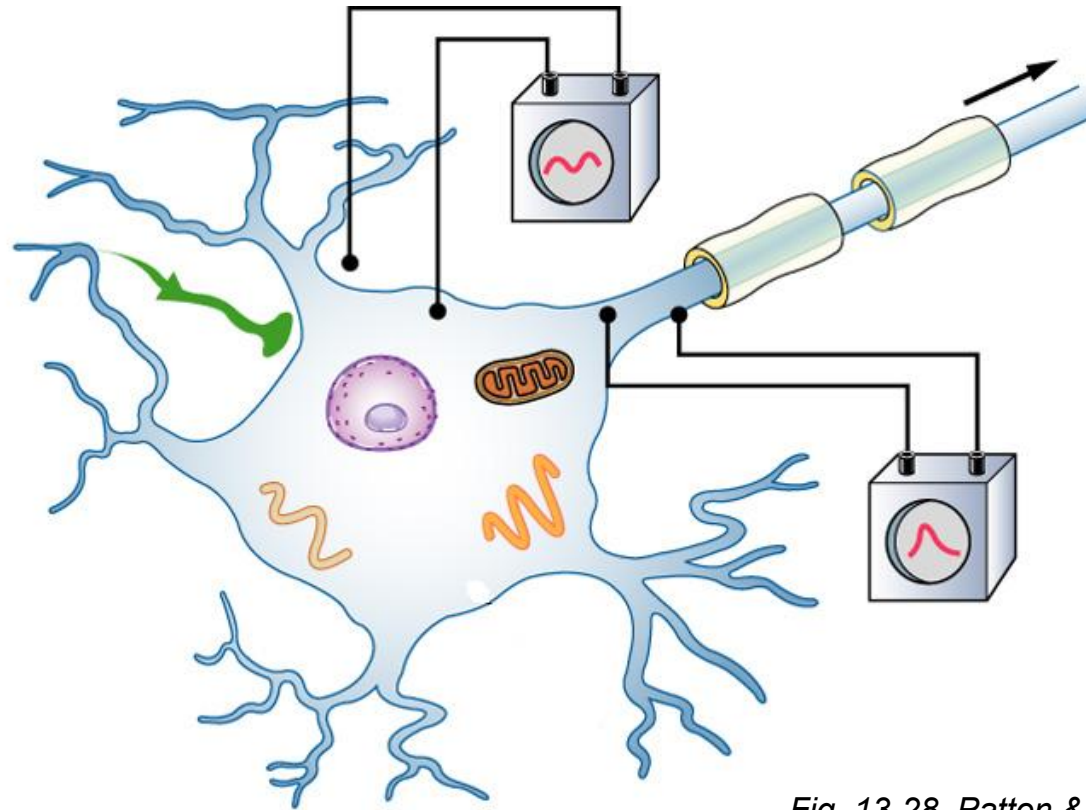
...at the AXON HILLOCK

# Action potential (AP) transmitted when neuron is above threshold...



...at the AXON HILLOCK

# Temporal Summation of Synaptic Inputs



*Fig. 13-28, Patton & Thibodeau, p.403*

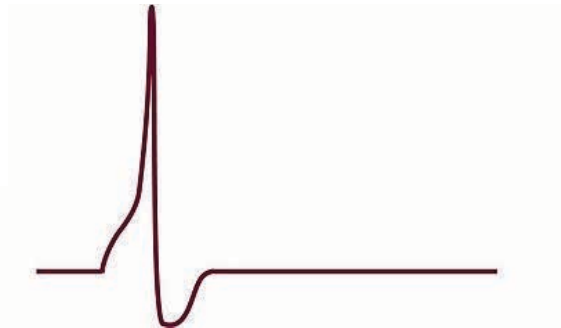
## **B Temporal Summation**

Mosby items and derived items © 2007, 2003 by Mosby, Inc.

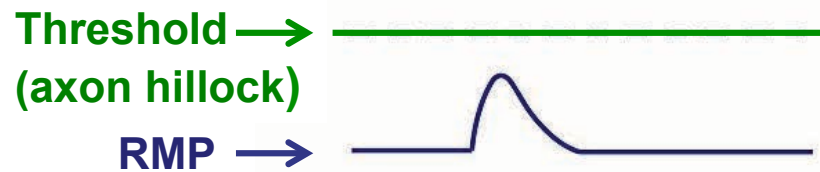
Time dependent – integration of inputs within a short time at the same synaptic knob (identical place)

# Temporal Summation of Synaptic Inputs

Neuron 1:  
Presynaptic  
action potential



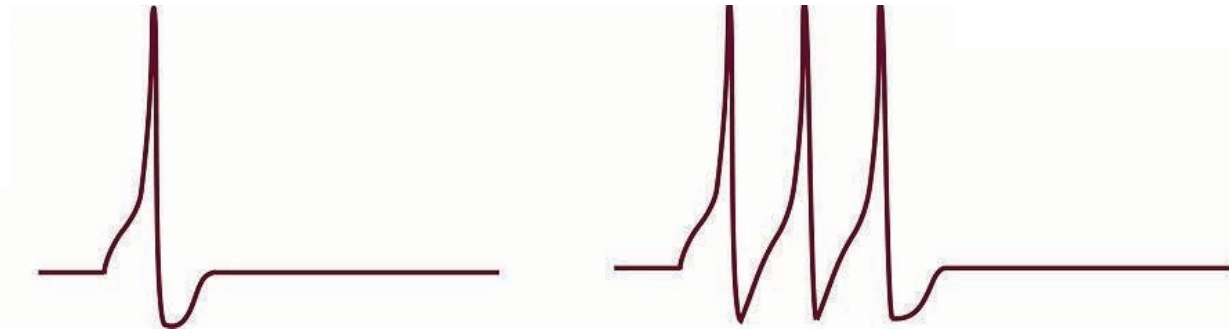
Neuron 2:  
Postsynaptic EPSP



no AP

# Temporal Summation of Synaptic Inputs

Neuron 1:  
Presynaptic  
action potential



voltage (mV)

Neuron 2:  
Postsynaptic EPSP

Threshold →  
(axon hillock)

RMP →

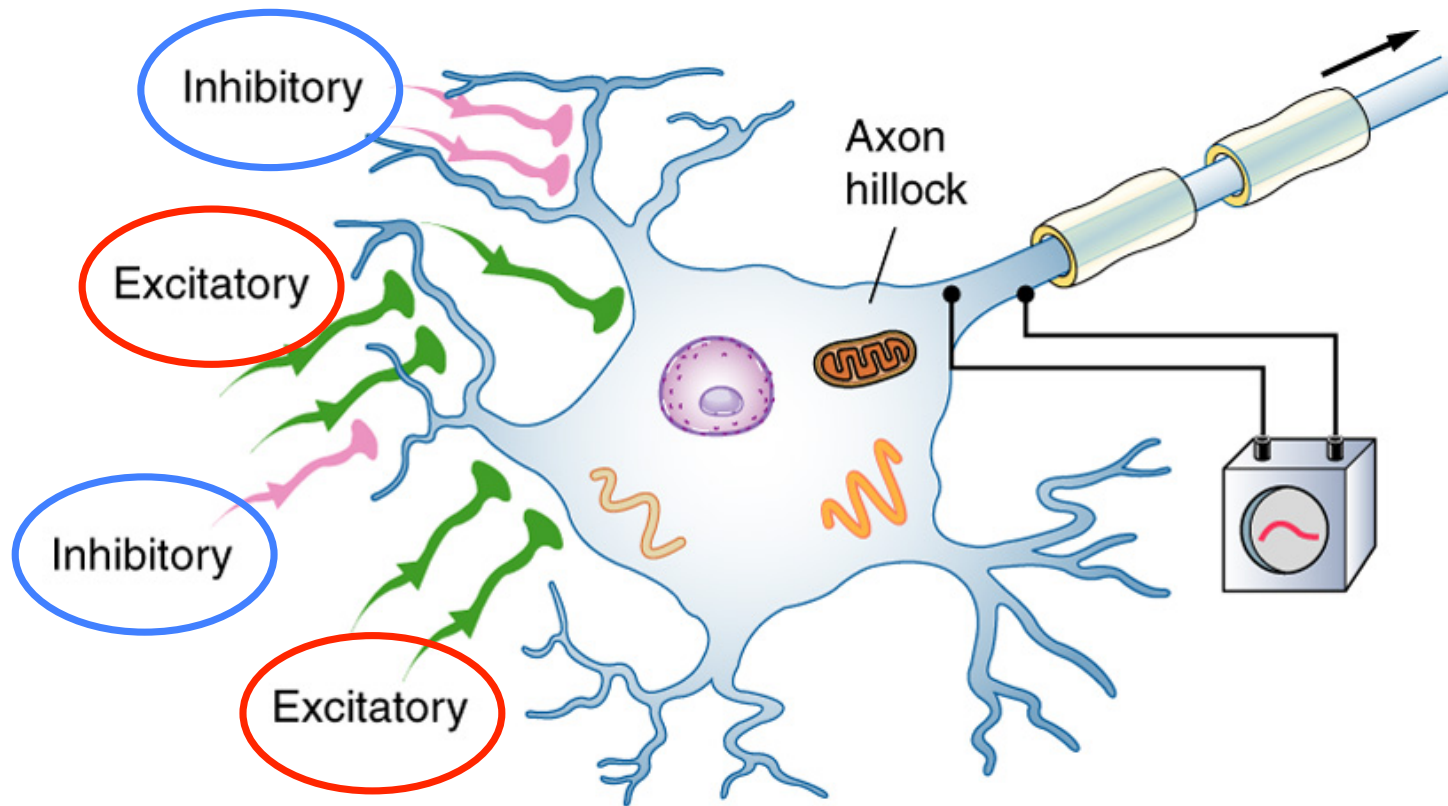
no AP

AP

voltage (mV)

- Time dependent – integration of inputs within a short time at the same synaptic knob
- EPSP last longer than a AP!

# Excitation & Inhibition

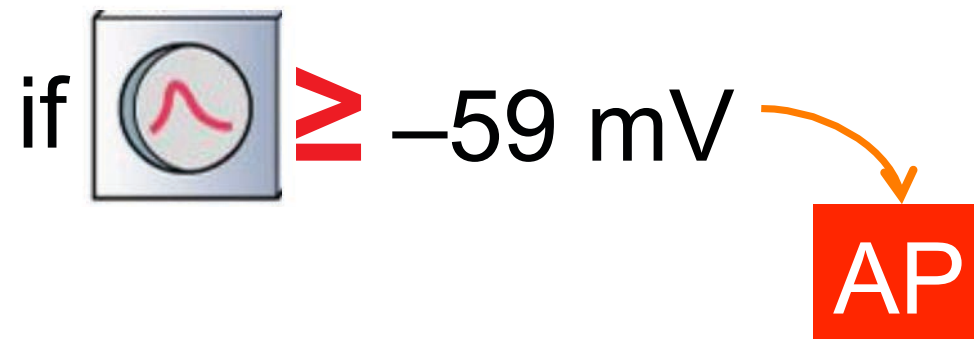
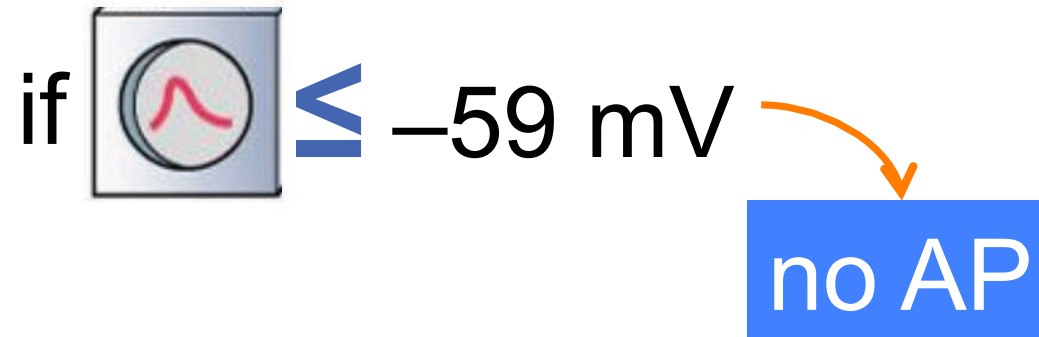
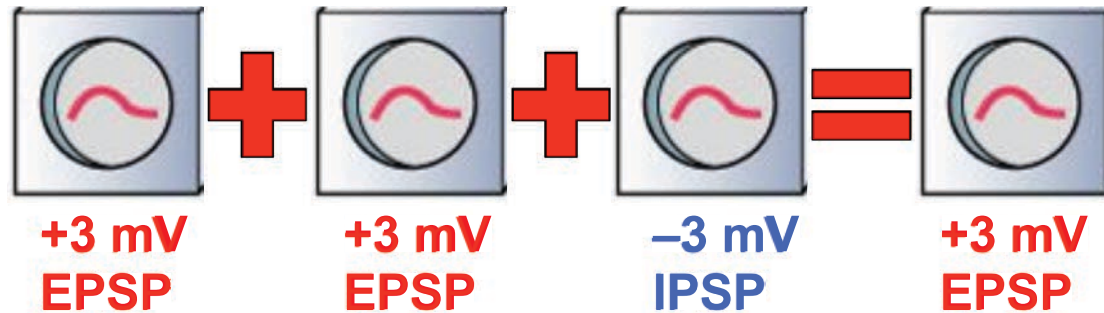


## C Summation of Excitatory and Inhibitory Signals

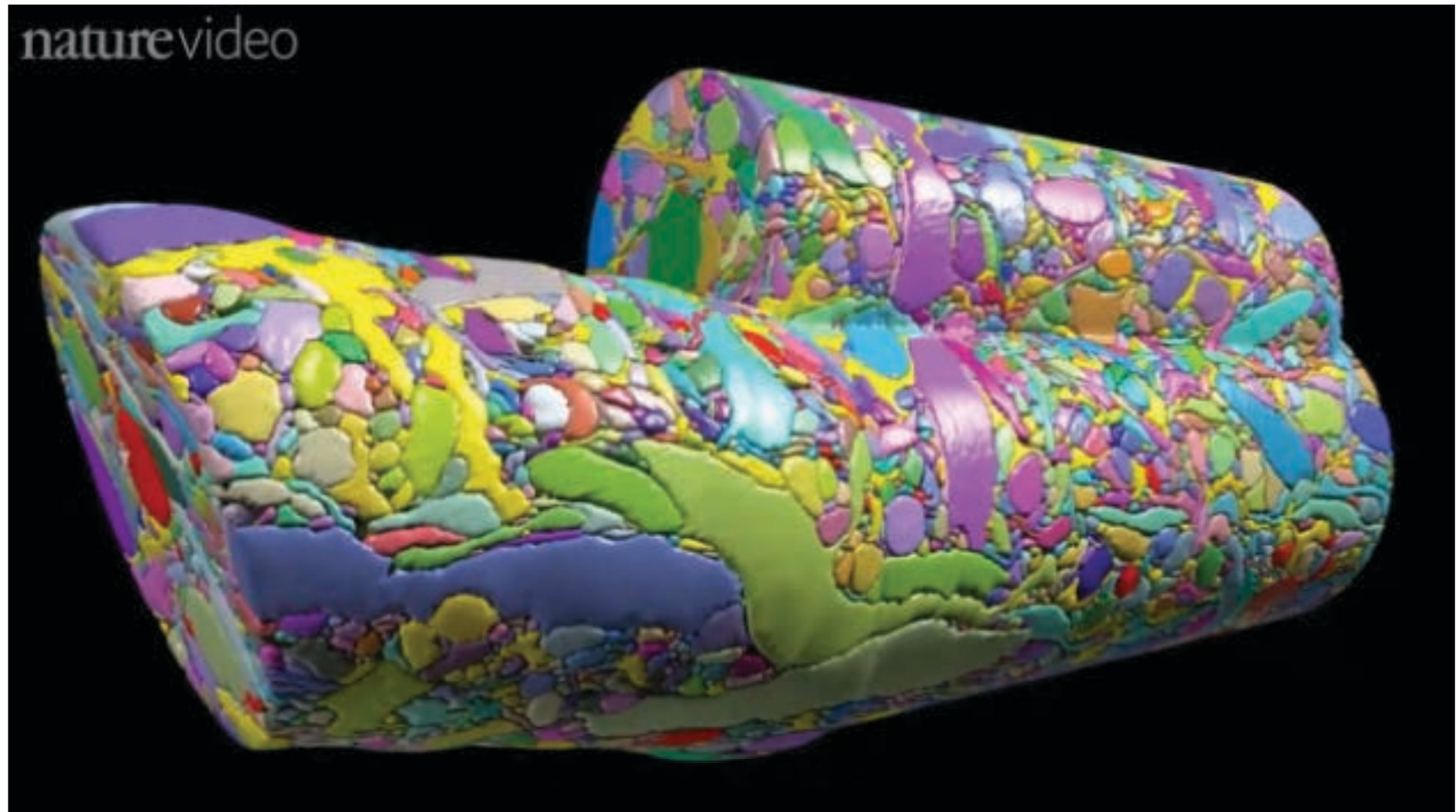
*Fig. 13-28, Patton & Thibodeau, p.403*

☺ *More of these in GLM3* ☺

# Excitation & Inhibition



# Neurones work in Networks



<http://www.nature.com/news/crumb-of-mouse-brain-reconstructed-in-full-detail-1.18105>

# Types of Network - Divergence

## Dictionary

### diverge

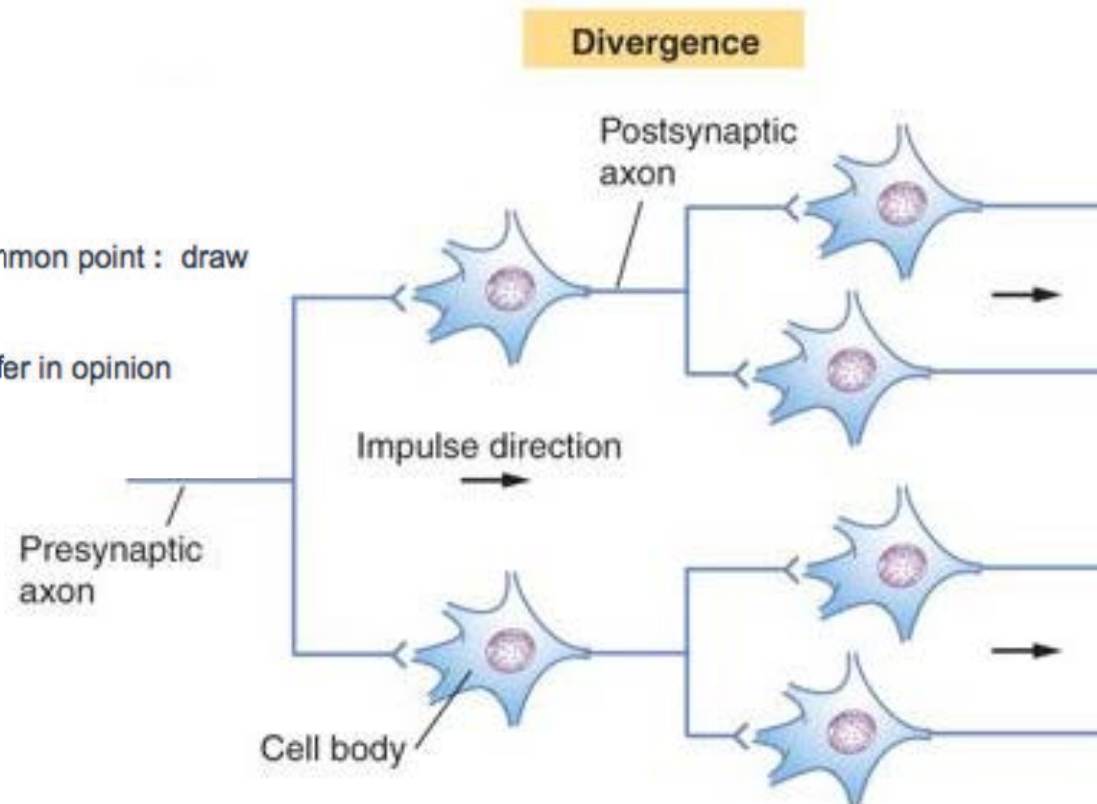
verb | di-verge | \de-'verj, dT-\

**1 a :** to move or extend in different directions from a common point : draw apart <diverging roads>

**b :** to become or be different in character or form : differ in opinion

**2 :** to turn aside from a path or course : deviate

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



Box 13-6, Patton & Thibodeau, p. 409

Information from a single neuron may **DIVERGE** to different brain regions (multiple neurons) or different body parts

Provides...

1. ... opportunity to **amplify** signals (multiple reactions in response to one stimulus)
2. ... few control points (precise coordinated control of muscles)

# Types of Network - Convergence

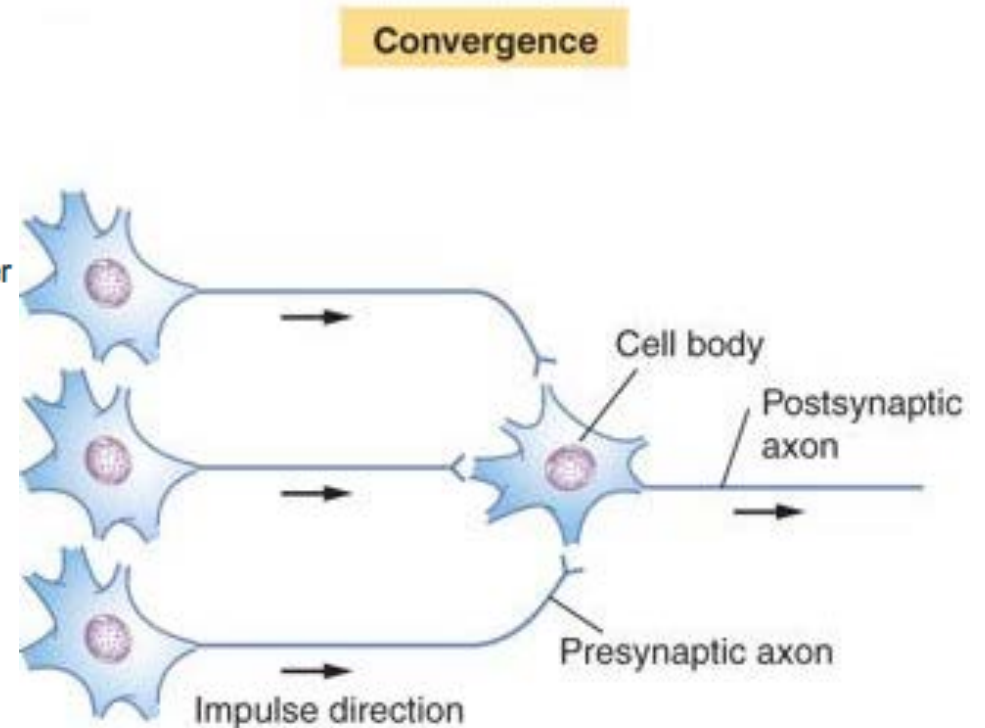
Dictionary

converge 

verb | con·verge | \kən-ˈvɜrj\

- 1 : to tend or move toward one point or one another : come together  
<converging paths>
- 2 : to come together and unite in a common interest or focus

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



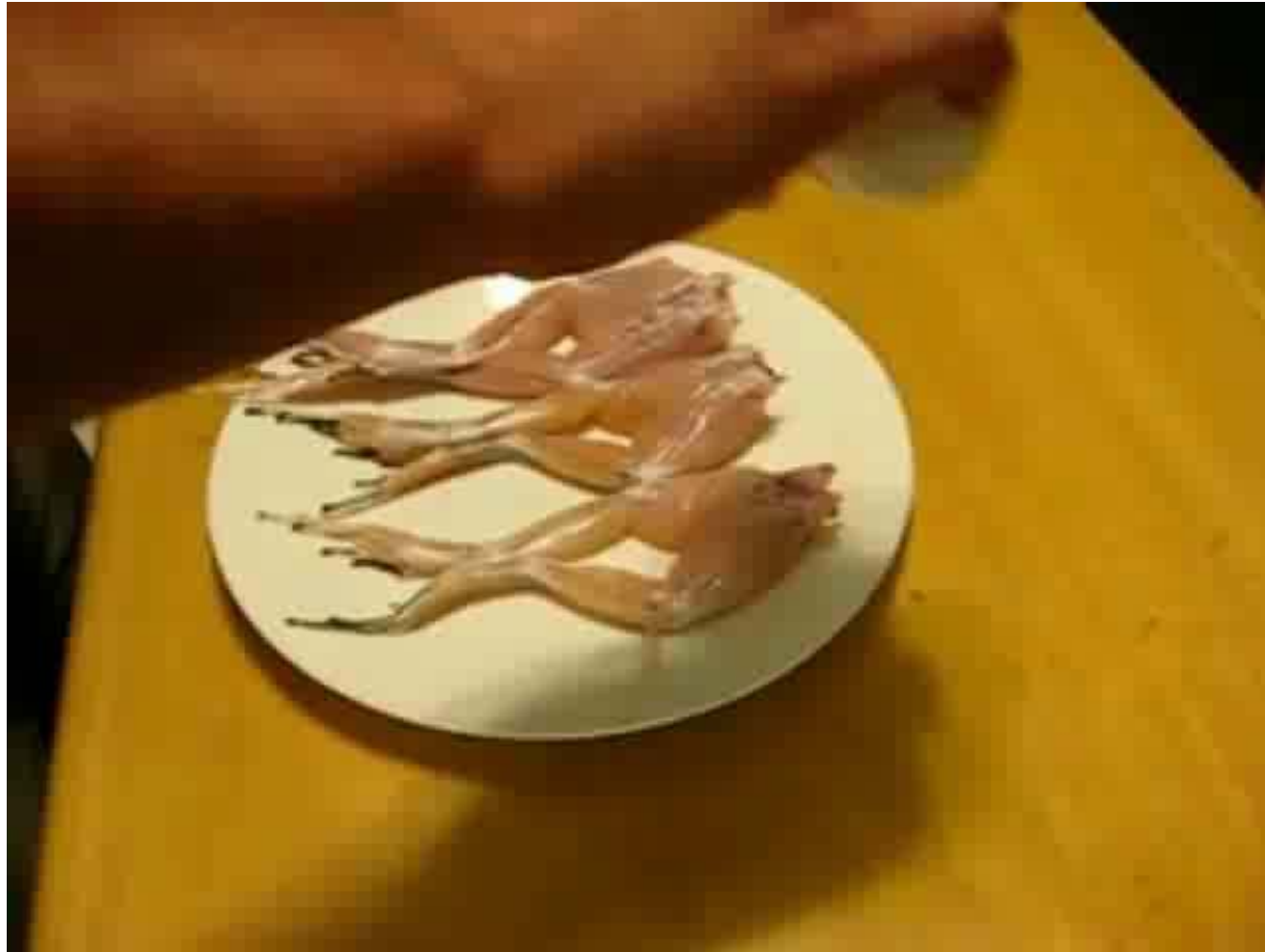
Box 13-6, Patton & Thibodeau, p. 409

Information from multiple neurons may CONVERGE on one single neuron (Summation!) that initiates a single response

Provides...

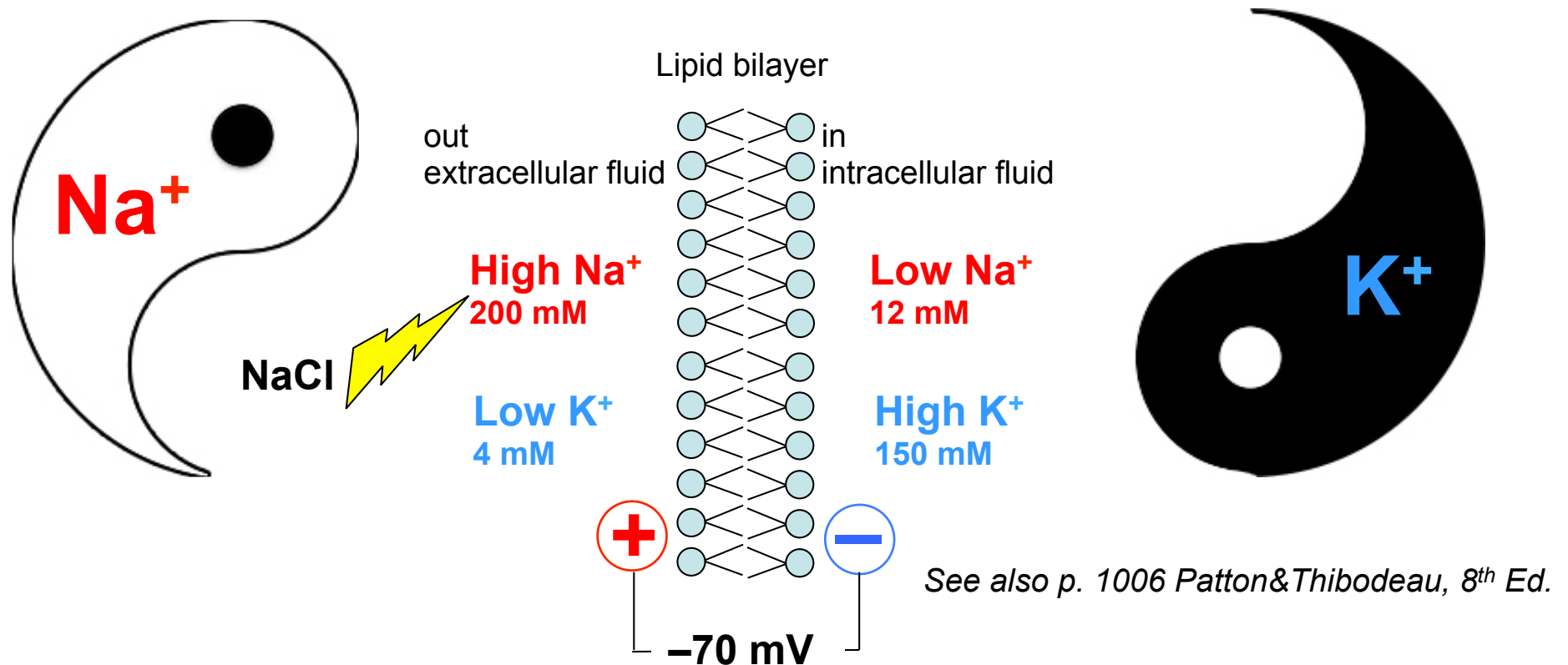
...redundancy → 'Back-up' route for the transmission of signals

*"system design that duplicates components to provide alternatives in case one component fails"*



Except from YouTube Frog Legs Dancing With A Little Salt  
Uploaded by [thearchipelagos](#) on 2 Jun 2009

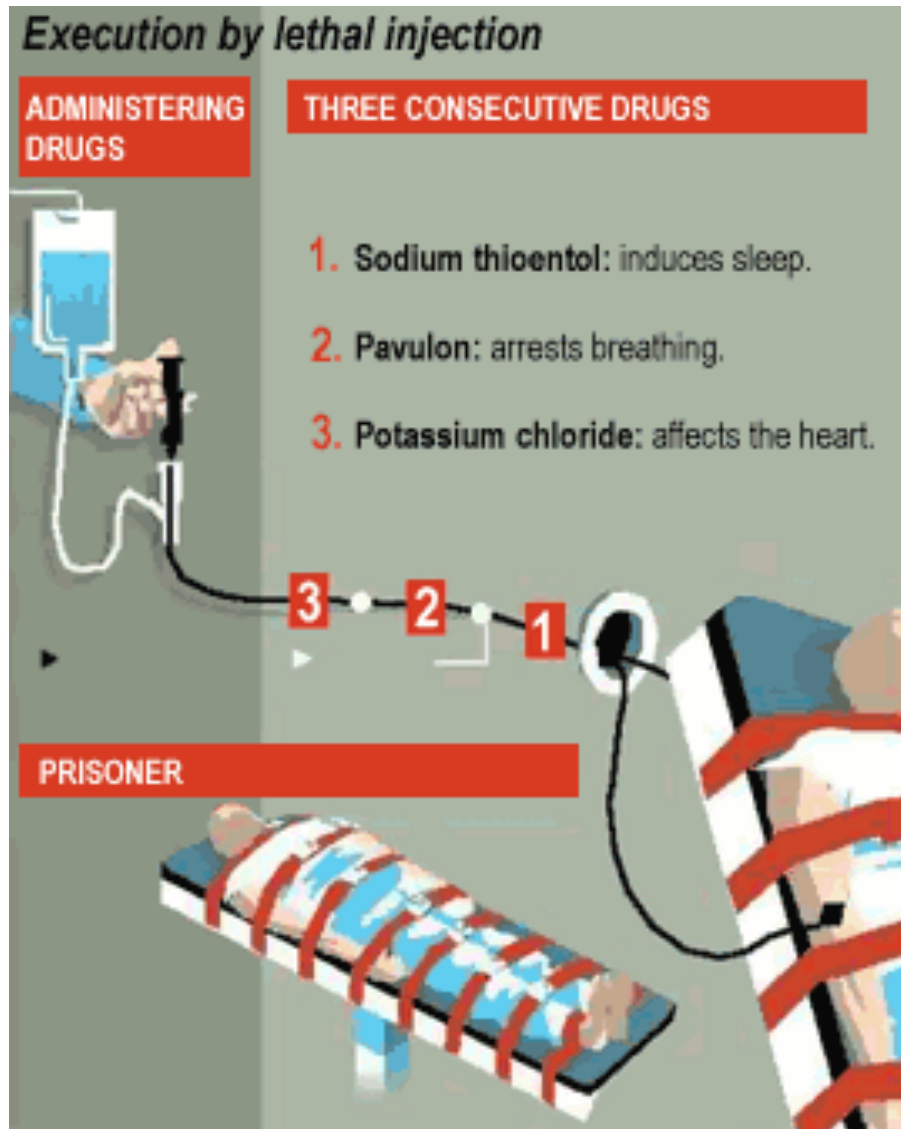
# What happens in the frog legs?



Increased external  $[\text{Na}^+]$  by the addition of salt – makes neuron cell membrane more excitable (as well as the muscle cells)

→ driving force for  $\text{Na}^+$  entry will be greater drives local **EPSP's**  
if **EPSP**  $> -59 \text{ mV}$  → AP → contraction (twitching)

# Something to think about



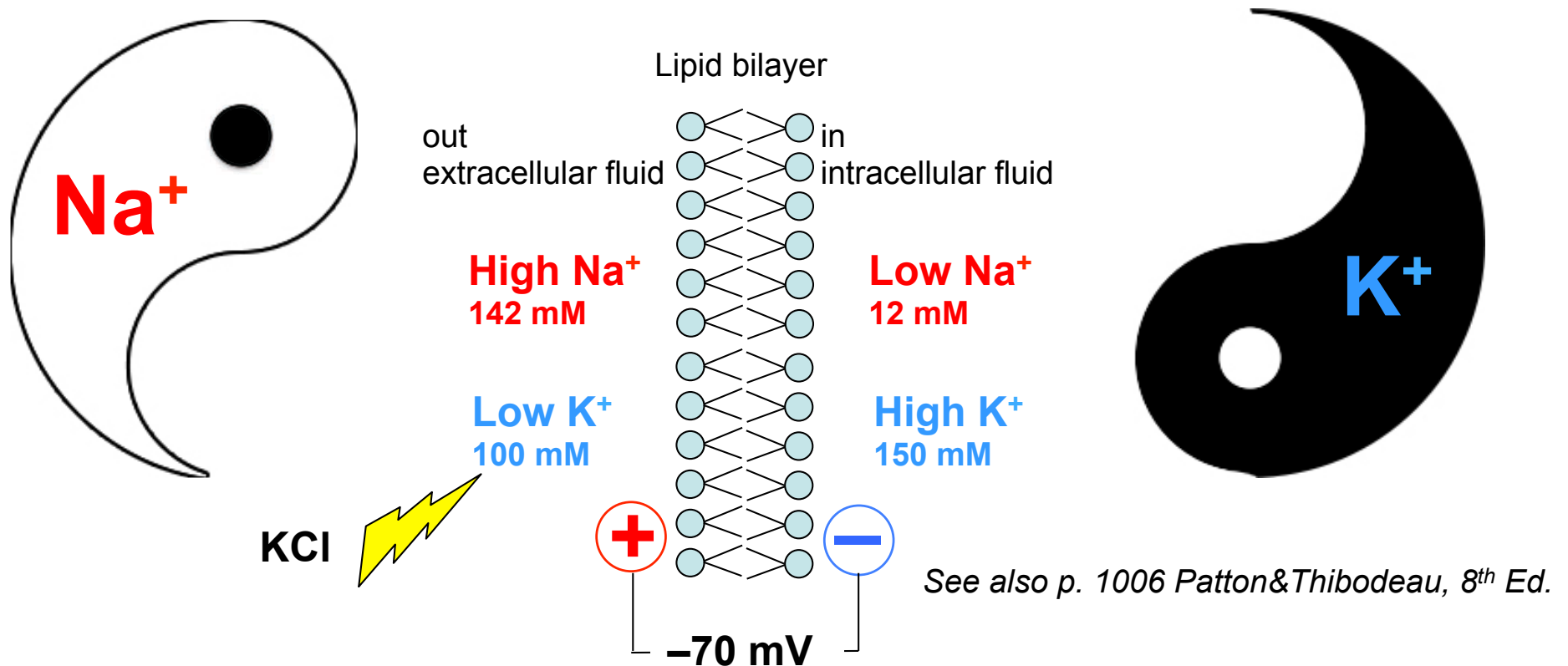
**Why is KCl lethal ?**

**WHY ??????**

From  
[http://www.chm.bris.ac.uk/webprojects2006/Macgee/Web%20Project/lethal\\_injection.htm](http://www.chm.bris.ac.uk/webprojects2006/Macgee/Web%20Project/lethal_injection.htm)

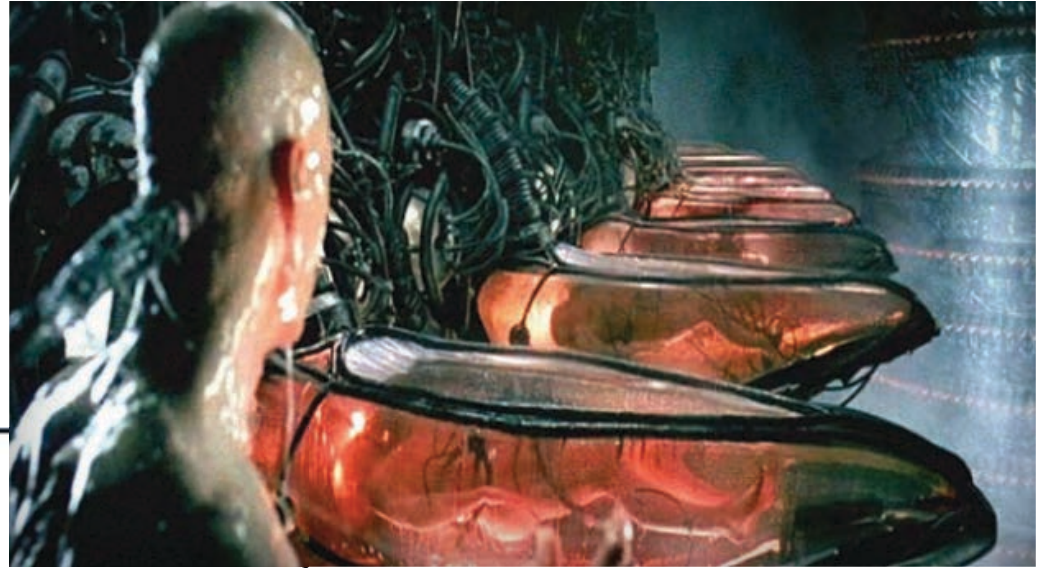
# Why is KCl lethal

→ disrupts ionic ( $K^+$ ) homeostasis!

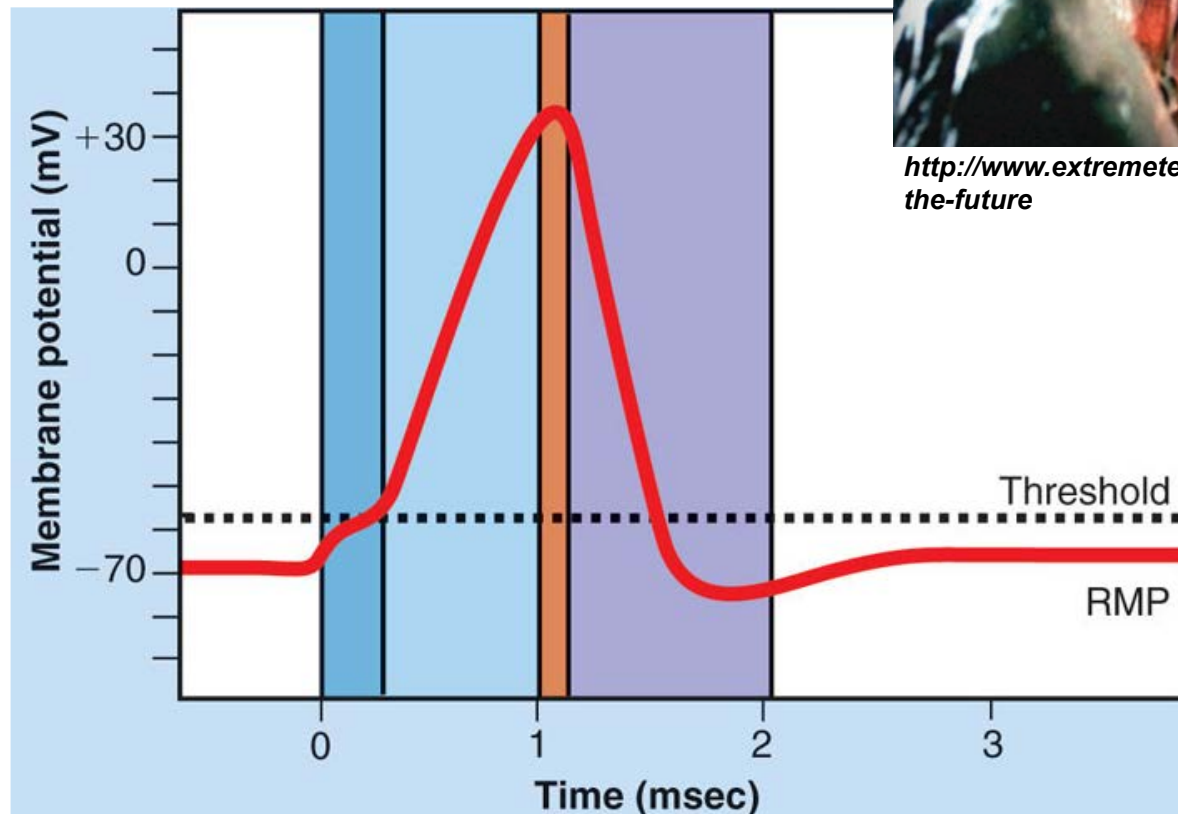


Raise  $K^+$  levels outside the cell → high  $K^+$  inside and **high  $K^+$  outside** –  
no  $K^+$  movement through the channels will be possible  
→ **cells do not repolarise, not enough  $K^+$  leaves the cell** (cardiac cells → no contraction, cardiac arrest)

# Bio-electricity!!!



<http://www.extremetech.com/extreme/135481-will-your-body-be-the-battery-of-the-future>



# Brief summary

Lecture 1. Bioelectricity, ion distribution, ion channels and pumps, membrane potential, depolarisation, repolarisation, hyperpolarisation

Lecture 2. Action potential, propagation/conduction of the action potential

Lecture 3. Synapses, signal transmission at chemical synapses

Lecture 4. Transmitters, synaptic integration (EPSP, IPSP), neuronal network types

**Reminder → online feedback questionnaire**

Thank you for you interest  
and attention!

# Bio–electricity!!!



# HUBS191

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