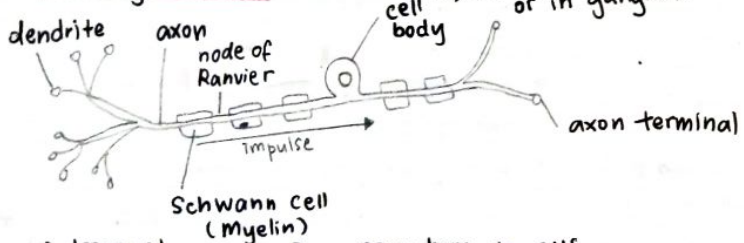
# COORDINATION
## CHAPTER 15

## NERVOUS COMMUNICATION

- Central nervous system (CNS) → brain, spinal cord
- peripheral nervous system (PNS) → cranial nerves (attached to brain), spinal nerves (" " spine)
- Nerve cells = neurones

## NEURONES

- Nucleus always in cell body.
- sensory neurone

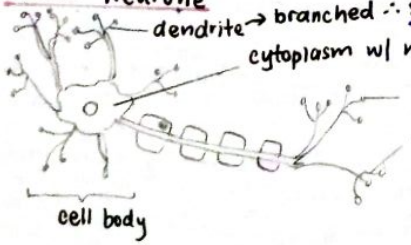



→ transmit impulses from receptors to CNS

- intermediate neurone / relay / connector

→ only found within CNS
→ transmit impulses from sensory to motor

- motor neurone




→ transmits impulses from CNS to muscle / gland

## MYELIN

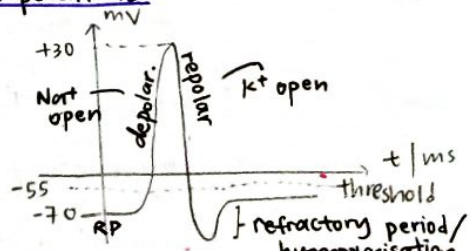
→ made when Schwann cells wrap themselves around the axon → csm mainly lipid & protein
→ Not all nerves myelinated
→ uncovered areas → Nodes of Ranvier
→ ↑ speed of nerve impulses

## REFLEX ARC

↳ pathway along which impulses are transmitted from receptor → effector w/ out 'conscious' regions of brain
→ involuntary respons → reflex action.

## TRANSMISSION OF NERVE IMPULSES

→ brief changes in distribution of electrical charge across csm → action potentials




## RESTING POTENTIAL (-60 → -70 mV)

→ electrical potential inside axon is 60-70mV lower than outside
→ maintained by sodium-potassium pumps in csm
→ energy from ATP
→ 3 $Na^+$ out, 2 $K^+$ in


→ more protein channels for $K^+$ than $Na^+$ ∴ $K^+$ diffuse back out faster
→ many large -vely charged molecules attract $K^+$ ∴ cannot diffuse out easily

∴ overall excess of -ve ion inside

## ACTION POTENTIAL

→ $Na^+$ move into axon ∵
  → Steep conc. grad
  → inside -vely charged ∴ attract $Na^+$
  → electrochemical gradient
→ electric current stimulates axon & $Na^+$ voltage-gated channels open
→ $Na^+$ diffuse into axon ∴ potential diff less -ve on inside. → depolarisation
→ depolarisation triggers more $Na^+$ voltage gated channels to open ∴ more depolarised
→ If P.d reaches -50 mV ∴ many open and P.d = +30mV
  ↖ -60 → -50 mV → threshold potential
→ Action potential only generated if reaches threshold potential.

→ After 1ms, $Na^+$ voltage gated channels close ∴ $Na^+$ cannot diffuse in
→ At same time $K^+$ channels open ∴ $K^+$ diffuse out. ∴ inside of axon more -ve & return to -70mV → repolarisation (removing +ve charge)

→ hyperpolarisation
→ pd drops below resting potential
→ $K^+$ channels then close & voltage gated $Na^+$ channels sensitive to depolarisation again
→ Na-K pump continues to pump $Na^+$ out $K^+$ in ∴ maintain distribution so more AP occur

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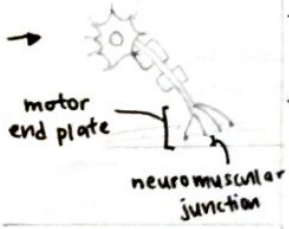
### FUNCTIONS OF SYNAPSES

- one-way transmission
  - ↳ neurotransmitters only on one side, receptors on other
- integration of impulses
  - ↳ motor neurone only transmits if threshold reached ∴ no overload
- connection of nerve pathways
  - ↳ info from one neurone can reach many relay neurones & effectors (eg. danger)
  - ↳ motor neurone have many dendrites ∴ SA ↑ for many synapses ∴ neurone can integrate info from many parts of the body
- depolarisation of postsynaptic neurone leads to AP only if above threshold
- Chances of AP↑ if more presynaptic neurone release more ACh.




- memory making and learning
  - ↳ new synapses form that link neurones from eyes & ears

### TRANSMISSION OF ACTION POTENTIALS

- temporary depolarisation of one part of membrane sets up local circuits with areas of either side
- local circuits depolarise adjoining regions ∴ AP generated in them → current flow depolarises next node of Ranvier
- In body, AP begins @ one end and 'new' AP generated ahead
  - ↳ ∵ $Na^+$ voltage gated channels are shut & cannot be stimulated to open
- Period of recovery → refractory period.
  - ↳ consequenses:
    - AP do not merge (discrete events)
    - min time between AP
    - Length of refractory period determines max frequency.

### HOW AP CARRY INFORMATION

- All AP have same
  - → amplitude
  - → speed of AP
- Differences
  - → frequency
  - → number of neurones carrying AP.

### SPEED OF CONDUCTION

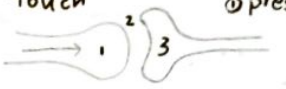
- myelin insulates axon membrane, preventing differences in potential across parts of membrane
  - ↳ speeds up rate of travel of AP
- local circuits exist from one node to the next ('jump')
  - ↳ called saltatory conduction
- Diameter of axon↑, Ω↓, transmission↑

### INITIATION OF AP

- A cell that responds to stimulus by initiating AP → receptor cell
  - ↳ convert energy to electrical impulse
- increase in +ve charge → receptor potential
- all or nothing law → transmit or not transmit

### SYNAPSES

- where 2 neurones meet but do not touch




① presynaptic neurone
② synaptic cleft
③ postsynaptic neurone

### MECHANISM OF SYNAPTIC TRANSMISSION

- impulses cannot 'jump' across synapse
- instead, neurotransmitter released to stimulate next neurone.

MECHANISM:

- AP arrives @ presynaptic neurone
- stimulates opening of voltage-gated calcium channels
- $Ca^{2+}$ diffuses into cytoplasm of presynaptic neurone
  - ↳ inside no $Ca^{2+}$ ∴ steep grad.
- Influx of $Ca^{2+}$ stimulates vesicles containing acetylcholine (ACh) to move to presynaptic membrane & fuse
- ACh released & diffuse across synaptic cleft
- ACh binds to receptor proteins temporarily on postsynaptic membrane.
  - ↳ chemically gated ion channels
- Proteins change shape, channel opens, $Na^+$ enter
- $Na^+$ depolarises membrane & generates AP
- Acetylcholinesterase catalyses hydrolysis of ACh to acetate & choline

$$ACh \xrightarrow{\text{acetylcholinesterase}} \text{acetate} + \text{choline}$$

- Choline taken back into presynaptic neurone, combined w/ acetyl coenzyme A to form ACh
- ACh transported into presynaptic vesicles

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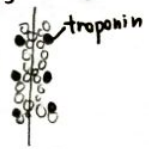
(continued filament structure)

- thin composed of actin
  - ↳ globular protein
  - ↳ many actin molecules form chain
  - ↳ two chains twisted → thin filament
- ↳ contains fibrous protein → tropomyosin
- ↳ troponin attached @ regular intervals

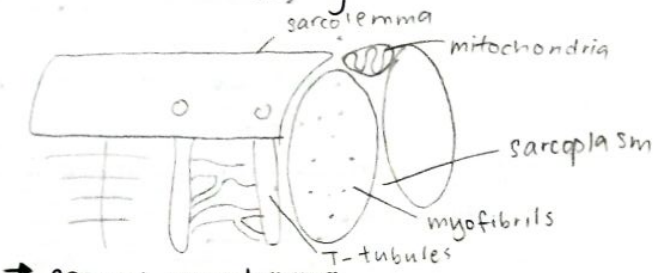



## MUSCLE CONTRACTION

- striated muscles → attached to skeleton
- neurogenic

## STRUCTURE OF STRIATED MUSCLE

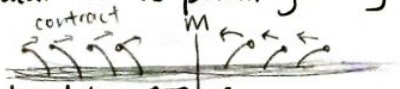
- muscle cell → syncytium (syncytium)
  - ↳ many nuclei




- CSM → sarcolemma
  - ↳ infolding into interior → transverse system tubules / T-tubules
- cytoplasm → sarcoplasm
- ER → sarcoplasmic reticulum (SR)
  - ↳ have protein pumps that transport $Ca^{2+}$ into cisternae of SR.
- mitochondria packed between myofibrils
  - ↳ ATP for muscle contraction
- striations produced by regular arrangement of myofibrils in sarcoplasm.

## STRUCTURE OF THICK & THIN FILAMENTS

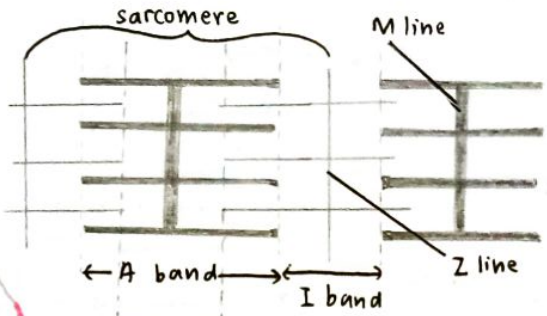
- thick composed of myosin
  - ↳ fibrous protein w/ globular head
  - ↳ many myosin molecules lie tgt w/ globular heads pointing away from M line




- ↳ each head has ATPase

## STRUCTURE OF MYOFIBRILS

- made of filaments
- thick filament → made of myosin
- thin filament → " actin




## HOW MUSCLES CONTRACT

- $Ca^{2+}$ released from stores in SR and bind to troponin
- Troponin molecules change shape
- Troponin & tropomyosin move to different position on the thin filaments ∴ exposing myosin binding site on actin chain
- Myosin heads bind ∴ forming cross-bridges between 2 types of filament
- myosin head tilts & pulls actin filament towards sarcomere centre.
- The heads hydrolyse ATP molecules, which provide energy so heads release actin
- Head springs back and bind to actin further along thin filaments.
- Process repeats as long as
  - ↳ troponin & tropomyosin do not block binding site
  - ↳ enough ATP supply

## PROVIDING ATP FOR MUSCLE CONTRACTION

- aerobic respiration in mitochondria
- lactic fermentation in sarcoplasm.
- produced from creatine phosphate
  - ↳ stored in sarcoplasm
- creatine phosphate + ADP → creatine + ATP
- creatine + ATP → creatine phosphate + ADP
  - ↳ demand ↓, ATP recharge
- when demand ↑, no ATP to recycle creatine
  ∴ creatine → creatinine (excreted in urine)

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### STIMULATING MUSCLE CONTRACTION

- Impulse moves along axon of motor neurone & arrives @ presynaptic membrane
- ACh diffuses across neuromuscular junction & binds to receptor proteins on postsynaptic membrane (sarcolemma)
- This causes chemically gated $Na^+$ ion channels to open
- $Na^+$ diffuse into sarcoplasm → depolarises membrane ∴ generates AP
- Impulses pass along sarcolemma & spreads down to T-tubules.
- Calcium ion channels in the SR membrane open
- $Ca^{2+}$ diffuse out down a steep conc. grad.
- $Ca^{2+}$ binds to troponin, troponin & tropomyosin move away & expose binding sites for myosin heads
- Myosin heads form cross-bridges w/ thin filaments & sarcomere shortens.

### HORMONAL COMMUNICATION

- Hormones = cell signalling molecules
- made in endocrine glands
- steroid hormones → lipid soluble.

### MENSTRUAL CYCLE

- ovulation ⇒ release of female gamete in oviduct
- menstruation ⇒ breakdown & loss of uterus lining
- primary → secondary → Graafian → ovulation → corpus luteum
- coordinated by glycoprotein hormones secreted by anterior pituitary gland & ovaries.

### BIRTH CONTROL

- steroid hormones that suppress ovulation
- Progesterone & oestrogen combined
  - ↳ FSH & LH highest when progesterone rise
  - ↳ suppress secretion of FSH & LH ∴ no ovulation
  - ↳ after 21 days not taken, ~~no suppression of FSH & LH~~, uterine lining not maintained ∴ menstruation
- Progesterone only
  - ↳ may allow ovulation to occur
  - ↳ reduce ability of sperm to fertilise egg
  - ↳ mucus in cervix more viscous ∴ egg less easily penetrated by sperm
- morning after (progesterone-like hormone)
  - ↳ ↓ chances of sperm reaching & fertilising egg
  - ↳ stop embryo implantation ∵ mucus

### HORMONAL CONTROL OF MENSTRUAL CYCLE

- During menstruation, anterior pituitary gland secretes LH & FSH
  - ↳ stimulates follicle to develop
  - ↳ stimulates secretion of oestrogen from cells surrounding follicle.
- Oestrogen ↳ stimulates endometrium to thicken & develop blood capillaries
  - ↳ decrease production of LH & FSH
- High oestrogen → surge of LH & FSH (little)
  - ↳ LH↑ → ovulation.
  - ↳ follicle develops into corpus luteum → secrete progesterone & oestrogen
  - ↳ FSH & LH maintain corpus luteum
- Progesterone & oestrogen
  - ↳ maintain lining of uterus
  - ↳ (progest.) inhibits anterior pituitary from secreting FSH ∴ no follicles develop
- Progesterone & oestrogen ↑, LH, FSH ↓ ∴ corpus luteum degenerates
- × corpus luteum = × progesterone & oestrogen
  - ↳ endometrium × maintained ∴ menstruation
- anterior pituitary gland no longer inhibited ∴ secrete LH, FSH, cycle begins again.

* each hormone has 1 peak in each cycle.

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## ELECTRICAL COMMUNICATION IN PLANTS

- outflow of $Cl^-$ → depolarisation
- out flow of $K^+$ → repolarisation
- AP travel along ESM through plasmodesmata

## MECHANISM OF ELECTRICAL COMM. IN PLANTS

- sensory hair deflected
- $Ca^{2+}$ channels @ base of hair opens ∴ $Ca^{2+}$ flows in and generates receptor potential.
- Within 20-30s, if 2 hair stimulated / 1 hair stimulated twice → action potential spread across lobe.
  - ↳ trap closes.
- Further stimulation → further AP → forces lobes to seal
  - → more $Ca^{2+}$ enter cells ∴ stimulates exocytosis of digestive enzymes (like synapse)
- once insect digested, cells on upper surface of midrib grow ∴ leaf reopens.

## ADAPTATIONS TO REDUCE ENERGY WASTE

- one hair stimulated will not close trap
- gap between stiff outer edges allow small insects to escape.

## PLANT HORMONES

- plant hormones = plant growth regulators
- carried in xylem sap / phloem sap

## AUXINS & ELONGATION

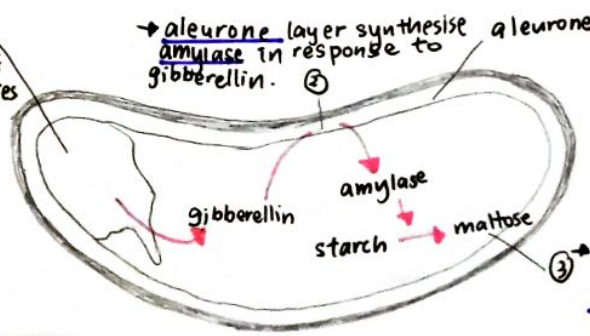
- principal auxin → IAA
- Synthesised in meristems
- Auxin binds to receptor
  - ↳ stimulates cells to pump $H^+$ into cell walls ∴ pH of wall ↓
  - ↳ stimulates $K^+$ channels to open ∴ $K^+$ enter cytoplasm ∴ $H_2O$ enters
- Expansins (in cell wall) activated by ↓ pH
  - ↳ loosen linkages between cellulose microfibrils
- Disruption occurs briefly → cell expands w/ out losing strength.

## GIBBERELLIN

- role in seed germination & stem elongation
- **STEM ELONGATION**
  - Tallness Le / le
  - Dominant Le codes for functional enzyme in a pathway that produces active form of gibberellin
  - Recessive le → no functional enzyme

## SEED GERMINATION

- @ first, seeds are dormant
  - ↳ little $H_2O$, metabolically inactive
- Embryo surrounded by endosperm
  - ↳ energy store containing Polysaccharide starch
- aleurone layer → protein-rich
  - ↳ NOT protective layer




① Absorption of $H_2O$ stimulates germination
- embryo synthesises gibberellin

② aleurone layer synthesise amylase in response to gibberellin.

③ Amylase hydrolyses starch → maltose ∴ mobilizes energy reserves
- maltose converted to glucose ∴ produce ATP