Neuronal Communication

Model Answers 2

Level	A Level
Subject	Biology
Exam Board	OCR
Module	Communication, homeostasis and energy
Topic	Neuronal communication
Booklet	Model Answers 2

Time allowed: **47 minutes**

Score: **/35**

Percentage: **/100**

Grade Boundaries:

A*	A	B	C	D	E
>69%	56%	50%	42%	34%	26%

Question 1

Which of the following describes the process that happens during **repolarisation** of a neurone during the action potential?

	Sodium channels	Potassium channels	Membrane potential
A	closed	open	decreasing
B	open	closed	decreasing
C	open	closed	increasing
D	closed	open	increasing

[1]

During repolarisation the sodium channels shut and potassium channels open allowing the membrane potential to decrease from +40mv to -70mv

Question 2

(a) Fig. 2.1 represents the end region of a neurone at a cholinergic synapse.

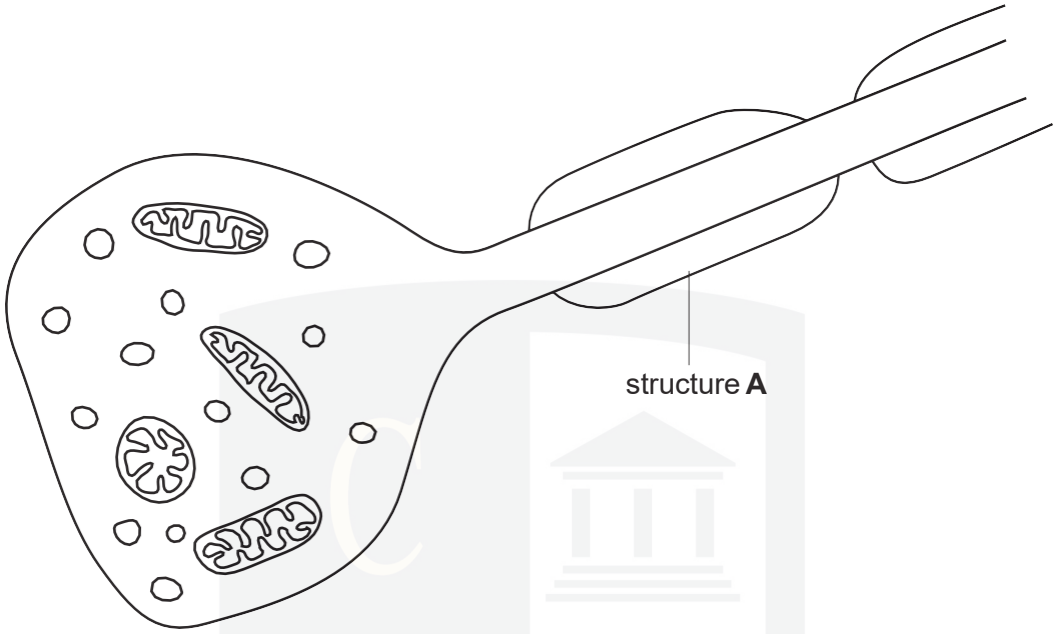


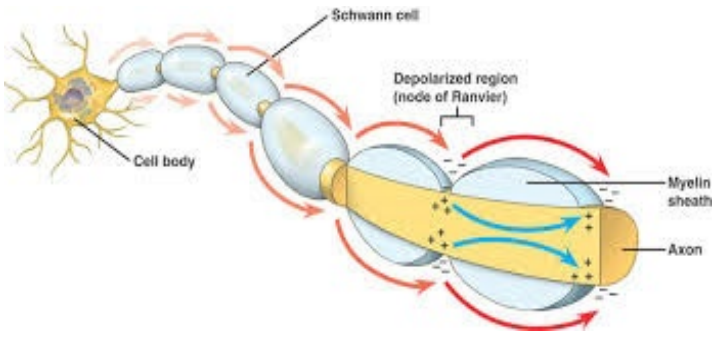
Fig. 2.1

(i) Describe the function of **structure A**.

In your answer, you should use the appropriate technical terms, spelt correctly.

[4]

- Structure a is a Schwann cell
- The Swan cell insulates the axon
- It prevents the movement of ions or depolarisation along the axon
- Sodium and potassium ion channels only occur at the nodes
- This speeds up the transmission of action potentials
- Action potentials only take place at the nodes
- This leads to the formation of longer local circuits so the impulse jumps from node to node
- This is known as saltatory conduction



(ii) Name the process by which acetylcholine leaves the neurone shown in Fig. 2.1. **[1]**

Exocytosis

(iii) Name the process by which acetylcholine travels across the synaptic cleft. **[1]**

Diffusion

(iv) A feature of synapses is that they allow transmission in only one direction.

State how this is achieved. **[1]**

- Only the presynaptic membrane/knob has neurotransmitter / acetyl choline / ACh / synaptic vesicles
- Only the presynaptic membrane has calcium ion channels
- Only the postsynaptic membrane has acetyl choline receptors
- Only Ach can be broken down at the postsynaptic membrane

This is a standard answer, but a more difficult one that often follows, is the role of synapses in the nervous system. Action potentials can in fact travel in both directions along the axon, so synapses act as valves only allowing one way transmission. Synapses also allow summation and responses from one area can travel to more than one part of the body. They are the way we learn and have conscious thought too.

(b) The chemical nature of synaptic transmission makes it susceptible to disruption by toxins.

(i) Atropine is a toxin produced by the deadly nightshade plant, *Atropa belladonna*.

Atropine is a similar shape to acetylcholine. The presence of atropine prevents the initiation of an action potential in the post-synaptic neurone.

Explain how the presence of atropine in the synapse will prevent the initiation of an action potential.

[3]

- Atropine competes with the acetyl choline for the receptor on the post synaptic membrane
- Atropine blocks the binding site and prevents ACh binding
- Sodium and potassium ion channels remain closed
- Sodium ions cannot enter and potassium ions cannot leave
- There is insufficient depolarisation of the post synaptic membrane which does not exceed the threshold potential

As the structure of atropine is similar to that of acetyl choline, it will have a complementary shape and bind to the receptors on the post synaptic membrane. These remain shut and prevent the entry and exit of ions, thus preventing the depolarisation of the post synaptic membrane.

(ii) Nerve gases have been used as chemical weapons. Some nerve gases act by inhibiting acetylcholinesterase, prolonging the effect of acetylcholine.

Suggest how atropine could act as an antidote to nerve gas. **[2]**

- Atropine will bind to the Ach receptors
- Less ACH can bind to the receptors so the constant depolarisation is prevented
- This prevents the constant firing of action potentials and reduces the effect of the nerve gas

[Total: 12]

Question 3

Fig. 5.1 is a trace that shows the changes that occur in the membrane potential of a neurone during the generation of an action potential.

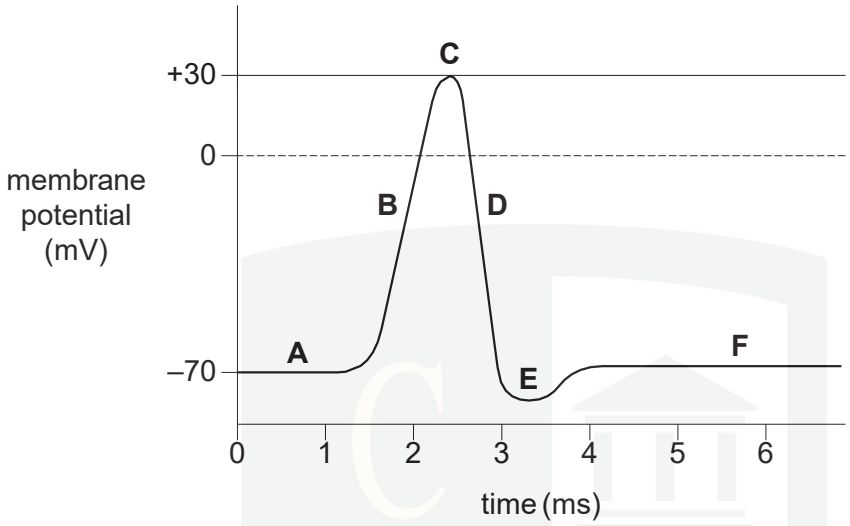


Fig. 5.1

(a) Using the letters **A** to **F**, indicate the point or points on the trace which correspond to the following:

(i) hyperpolarisation, E **[1]**

Hyperpolarisation immediately follows the action potential, where the potential difference drops below resting potential.

(ii) Resting potential, A and F **[1]**

Usual resting potential is at about -70mV

(iii) the membrane is most permeable to potassium ions, D **[1]**

During polarisation, the potassium ion channels are open

(iv) depolarisation. B **[1]**

During depolarization, the sodium ion channels are open, allowing sodium ions to flood into the axon, which takes the potential difference across the membrane toward 0mV

(b) Puffer fish, *Fugu spp.*, produce a powerful poison, tetradotoxin, and some species store it in high concentrations in their body tissues. Unless these fish are correctly prepared, eating them can be fatal.

Tetradotoxin is poisonous to humans because it blocks **gated** sodium channels in cell membranes, preventing action potentials. This does not happen in the fish themselves.

(i) With reference to Fig. 5.1, identify, using the appropriate letter, the part of the action potential trace that will be affected by tetradotoxin. **[1]**

B

As this is the stage at which the sodium ion channels are open

(ii) Suggest why tetradotoxin is **not** toxic to the puffer fish. **[1]**

The **channel/receptor/ion** is **different** in the puffer fish- so has no effect

(c) Multiple sclerosis (MS) is an auto-immune condition in which the nervous system is damaged. This damage leads to loss of sensation. One form of damage is shown in Fig. 5.2.

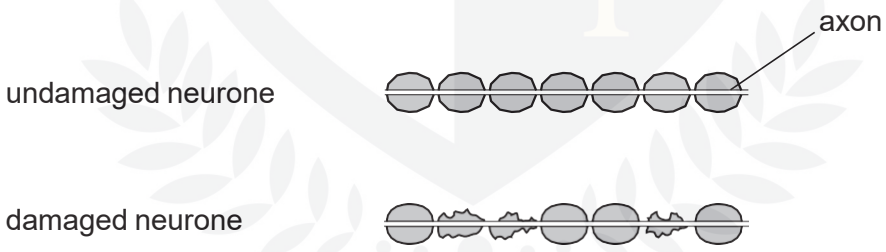


Fig. 5.2

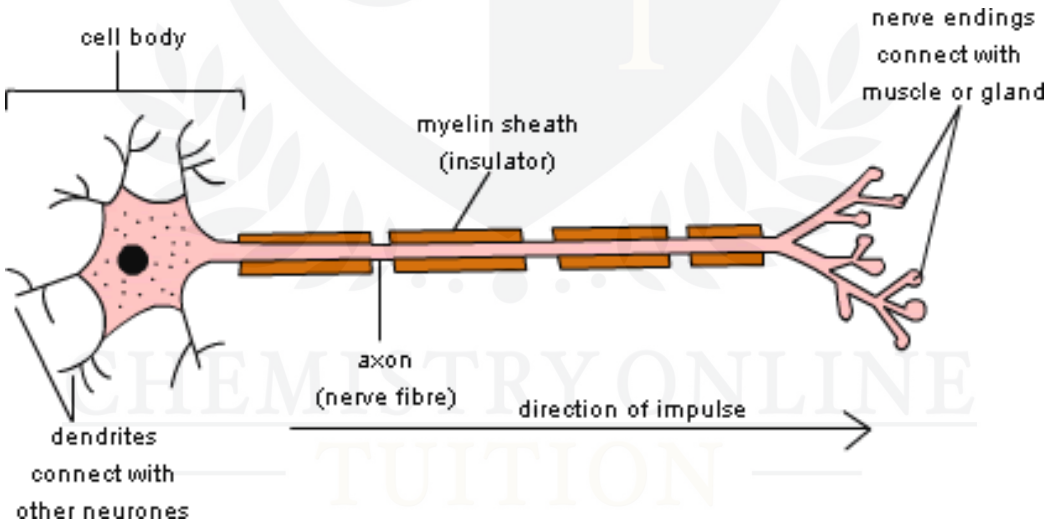
(i) Suggest why MS is described as an auto-immune condition. **[2]**

- An auto-immune condition is one where the body is attacked by **its own immune system**
- This is because the body treats cells- or in this case- neurons/myelin as **'foreign'/non-self**
- Therefore it **triggers antibodies/phagocytes/B lymphocytes/ T lymphocytes**

(ii) Explain why this damage leads to a loss of sensation. **[2]**

- This would cause **damage** to the **myelin sheath/ Schwann cells**
- Resulting in **less or no insulation**
- This therefore **slows down/stops conduction** of nerve impulses/action potentials, or **prevents saltatory conduction**
- This could occur in sensory neurons going towards the brain/CNS from sensory organs/receptors- which would therefore cause a loss of sensation

A motor neurone- insulated by myelin sheath, which speeds up impulse transmission:



[Total: 10]

Question 4

(a) Fig. 3.1 represents part of the axon of a neurone.

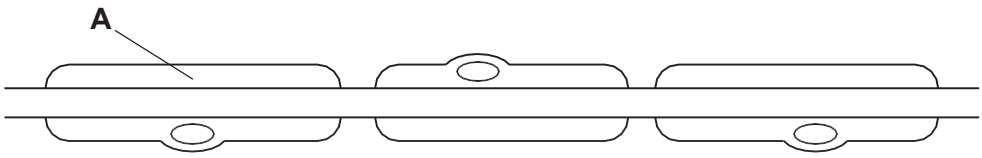


Fig. 3.1

Describe the **structure** of the feature labelled **A**. **[2]**

- The structure labelled A is the myelin sheath
- It consists of Schwann cells which are wrapped around the axon
- There are gaps between the cells known as the nodes of Ranvier

Table 3.1 shows details of the diameter and speed of conduction of impulse along the neurones of different animal taxa.

Table 3.1

type of neurone	axon diameter (μm)	speed of conduction (ms^{-1})	animal taxon
myelinated	4	25	mammal
myelinated	10	30	amphibian
myelinated	14	35	amphibian
unmyelinated	15	3	mammal
unmyelinated	1000	30	mollusc

(b) Using **only the data in Table 3.1**, describe the effect of each of the following on the speed of conduction:

(i) myelination, **[2]**

- The effect of myelination is to increase the speed of conduction
- Unmyelinated axons need a larger diameter to speed up the conduction

If you compare the myelinated axons in the amphibian with the unmyelinated axon in The mammal then the speed of the conduction is 12 times faster. It is better to compare these two axons as the diameter is similar

(ii) axon diameter. **[2]**

- A larger axon diameter produces greater speed of construction
- As the axon the diameter increases from 15 to 1000 in the unmyelinated axons of the mammals and the mollusc the speed of conduction increases by a factor of 10

It is important that your answer compares a similar diameter when comparing myelinated versus unmyelinated and it must also compare the effect of axon diameter in either myelinated or unmyelinated neurons

(c) Temperature also affects the speed of conduction of a nerve impulse.

(i) Suggest why an increase in temperature results in an increase in the speed of conduction.

[1]

- An increase in temperature increases kinetic energy, so ions diffuse across membranes more quickly
- Neurotransmitters will diffuse faster across the synaptic gap
- Neurotransmitters will be broken down by enzymes such as acetylcholinesterase more quickly
- Faster diffusion leads to faster depolarisation
- Action potentials may be shorter

Remember that any reference to a change in temperature may affect enzyme activity, in this question it will also relate to the speed of diffusion.

(ii) As the temperature continues to increase, it reaches a point at which the conduction of the impulse ceases. Suggest why.

[1]

- If temperature continues to increase then channel proteins and the sodium potassium pump may be denatured and no longer function
- Enzymes in the synapse may also be denatured

It is not enough to simply refer to enzyme activity In general terms. The question specifically refers to the effect of temperature on the conduction of the impulses and why it ceases, so your answer must be relevant to both synaptic transmission and the conduction of action potentials

(d) Outline the sequence of events following the arrival of an action potential at the synaptic knob until the acetylcholine has been released into the synapse.

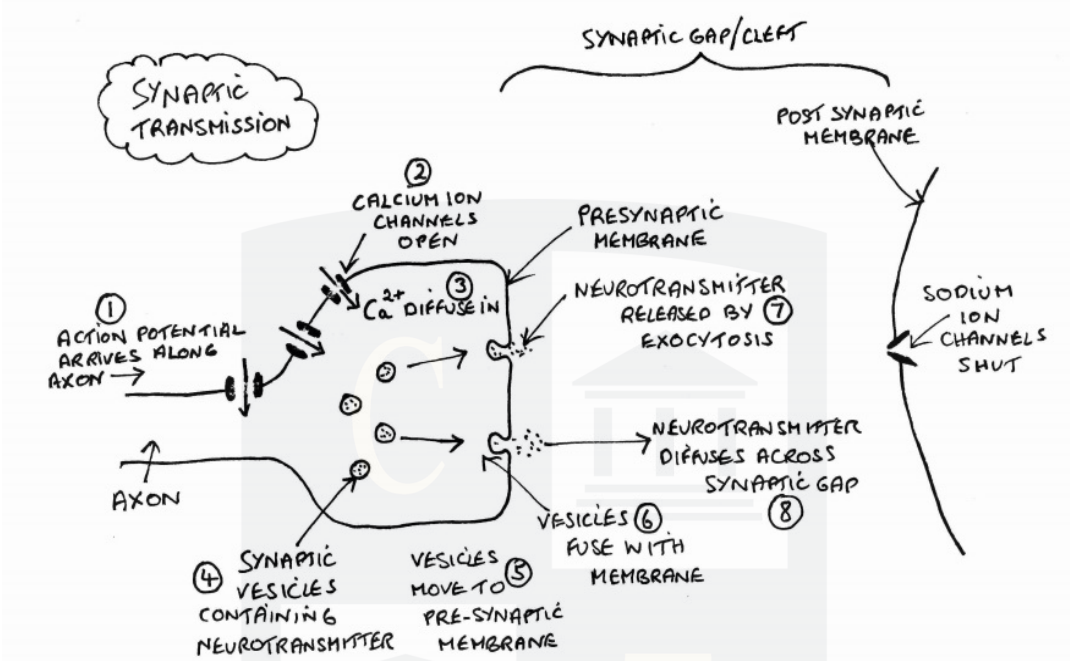
In your answer, you should use appropriate technical terms, spelt correctly.

[4]

Following the arrival of an action potential at the synaptic knob the following takes place:

- Calcium ion channels open
- Calcium ions diffuse into the synaptic knob
- Vesicles containing acetylcholine move towards the presynaptic membrane
- These vesicles fuse with the membrane
- The release acetylcholine by exocytosis into the synaptic gap

Note that the question asks you to outline the sequence of events up to the release acetylcholine into the synapse. So you must not include any reference to any events that occur at the post synaptic membrane, such as the binding of acetylcholine with receptors and the subsequent opening of the sodium ion channels



[Total: 12]