5.7 Neurones, nerve impulses and synapses

Name: _____ Class: _____ Date: _____ **Total: 20 marks**

KEY WORDS resting potential • action potential • threshold • saltatory conduction • synapse • refractory period

Objective

Practise statements 8.1-8.7: neurone structure, the resting and action potential, conduction speed and saltatory conduction, synaptic transmission, and how drugs act at synapses.

1 Key knowledge and worked examples

■ Three neurones, one plan

Sensatory, relay and motor neurones all carry a cell body, dendrites and a long axon; sensory carry impulses to the CNS, motor away, relay connect them inside the CNS.

■ The action potential

Resting potential -70 mV (the sodium-potassium pump, 3 Na+ out for 2 K+ in, plus K+ leak). A stimulus past threshold (-55 mV) opens voltage-gated sodium channels: depolarisation to +30 mV, all-or-nothing; potassium channels then open (repolarisation, overshoot to hyperpolarisation); the refractory period restores the pump and prevents backward travel.

■ Myelin speeds the message

Saltatory conduction : the impulse jumps between nodes of Ranvier, depolarising only at the nodes. Larger axon diameter also lowers resistance. Speed = myelin x diameter x temperature (up to the enzymes' optimum).

■ The synapse

Action potential arrives, Ca2+ enters the presynaptic knob, vesicles of neurotransmitter fuse with the membrane, transmitter diffuses the cleft, binds postsynaptic receptors, a new action potential fires if threshold is reached; enzymes (e.g. acetylcholinesterase) clear the cleft. The synapse is unidirectional - only the presynaptic side stores transmitter.

■ **Drugs at the synapse (exam favourite)**

SSRIs block serotonin re-uptake (signal persists); ecstasy floods then depletes serotonin; lidocaine blocks sodium channels (no impulse); nicotine mimics acetylcholine (agonist); botulinum toxin prevents vesicle fusion (no release).

2 Practice

2.1 State the value of a neurone's resting potential. [1]

2.2 Explain why an action potential is described as all-or-nothing. [2]

2.3 Name the ion that enters the presynaptic knob to trigger transmitter release. [1]

3 Exam-style questions

3.1 Describe the events that produce an action potential, from the resting potential to the start of the refractory period. [6]

3.2 Explain two ways the structure of a myelinated axon speeds conduction compared with an unmyelinated one, and state what limits the speed in both. [5]

3.3 Tetrodotoxin (TTX) from puffer fish blocks voltage-gated sodium channels. Explain why TTX stops nerve conduction, and predict its effect on the heart, whose muscle conducts electrical excitation between cells. [5]

Solutions

Original-task marking: M1 method, A1 accuracy, B1 reasoning.

2.1 About -70 mV.

2.2 Below threshold nothing happens; at or above threshold a full-size action potential fires regardless of stimulus strength.

2.3 Calcium ions (Ca2+).

3.1 Resting: pump holds -70 mV, interior negative; stimulus depolarises the membrane to threshold -55 mV; voltage-gated sodium channels open and Na+ floods in - depolarisation to about +30 mV; potassium channels open and K+ flows out, repolarising and overshooting to hyperpolarisation; the refractory period begins as channels reset and the pump restores the resting gradient.

3.2 Myelin insulates the membrane, so depolarisation jumps between nodes of Ranvier (saltatory conduction) - far less membrane to depolarise per unit distance; this saves the time and ATP that pumping at every point would need. Limits: the nodes' channel density and axon diameter (resistance), and temperature up to the channel proteins' optimum.

3.3 With sodium channels blocked, Na+ cannot enter and the membrane cannot depolarise; no action potential is generated or propagated, so nerve impulses stop entirely. Cardiac muscle's excitation also depends on ion-channel depolarisation passing cell to cell, so conduction fails and the heart's coordinated beat stops.