Question 1: Define membrane potential (V_m) and sodium equilibrium potential (E_{Na}). Which of these, if any, changes during the course of action potential?

Answer: The membrane potential (V_m) is the voltage across the neuronal membrane at any moment in time. The potential of the resting membrane is –75 mV. The sodium equilibrium potential (E_{Na}) is the steady equilibrium potential achieved when the membrane is permeable only to sodium ions. The value of E_{Na} is 62 mV. However, in its resting state, the membrane is not permeable to sodium. During the application of action potential, sodium channels open and sodium rushes into the cell. The large sodium current takes the membrane potential from its negative resting state toward E_{Na}. Sodium channels are deactivated after 1 msec, and the membrane repolarizes due to potassium efflux, which takes the membrane potential back toward the equilibrium potential of potassium.

Question 2: Which ions carry the early inward and late outward currents during the action potential?

Answer: During the early part of the action potential, the influx of sodium ions across the membrane briefly depolarizes the membrane. The brief inward sodium current is a consequence of opening the voltage-gated sodium channels for only 1 msec. Membrane repolarization is the result of potassium efflux, which is the outward potassium current because of opening voltage-gated potassium channels after a delay of 1 msec.

Question 3: Why is the action potential referred to as "all-or-none"?

Answer: Action potential is termed "all-or-none" because no partial action potentials exist. A physical or electrical event opens sodium permeable channels, but the resulting influx of sodium ions and the resulting depolarization – called a generator potential — must reach a critical level before the axon generates an action potential. The critical level is called a threshold. After achieving threshold depolarization, the cell fires an action potential.

Question 4: Some voltage-gated K^+ channels are known as delayed rectifiers because of the timing of their opening during an action potential. What would happen if these channels took much longer than normal to open?

Answer: Voltage-gated potassium channels open 1 msec after membrane depolarization. The resulting potassium conductance rectifies, or resets, the membrane potential. This conductance is called the delayed rectifier because of the 1 msec delay in rectifying the membrane potential. If these channels took longer than normal to open, the action potential would be wider, which means that it would take longer to restore the resting membrane potential.

Question 5: Imagine you have labeled tetrodotoxin (TTX) to be able to see it with a microscope. If we wash the TTX on to a neuron, what parts of the cell would you expect labeled? What would be the consequence of applying TTX to the neuron?

Answer: TTX is a natural toxin that interferes with the function of voltage-gated sodium channels. TTX blocks the sodium permeable pore by binding tightly to a specific part outside the channel and blocking all the sodium-dependent action potentials. Applying TTX to a neuron would block all impulses in that nerve, preventing it from firing any action potential, regardless of input. Labeled TXX could be visualized on the cell's axon, where voltage-gated sodium channels are concentrated.

Question 6: How does the conduction velocity of action potential vary with axonal diameter? Why?

Answer: The speed of action potential depends on how far depolarization spreads ahead of action potential. This, in turn, depends on the physical characteristics of axons. The two paths that a positive charge can take are inside an axon and across the axonal membrane. When the axon is narrow with many open pores, more of the current flows across the axonal membrane and is lost. When the axon is wide with a few open pores, the current flows inside the axon. The farther down the axon the current flows, the farther ahead of the action potential the membrane will be depolarized and the faster the action potential will propagate. As a result, the conduction velocity of axons increases with the diameter of axons.