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Impulses and pressure waves cause excitement and conduction in the nervous system

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ABSTRACT

It is general accepted, that nerval excitement and conduction is caused by voltage changes.

However, the influx of fluid into an elastical tube releases impulses or pressure waves. Therefore an influx of ion currents, respectively fluid motions into the elastic neuronal cells and fibres also induce impulses. This motion of charge carriers are measured by voltage devices as oscillations or action potentials, but the voltage changes may be an epiphenomenon of the (mechanical) impulses.

Impulse waves can have a high speed. As stiffer or inelastic a tube wall, the greater is the speed of the impulse. Myelin sheaths cause a significant stiffening of the nerve fibre wall and myelinated fibres have a conduction velocity up to 120 m/s. The influx of fluid at the nodes of Ranvier intensifies periodically the impulse wave in the nerve fibres.

The authors suggest that also the muscle end-plate acts as a conductor of axonal impulses to the inner of the muscle fibres and that the exocytosis of acetylcholine into the synaptic cleft may be an amplifier of the axonal impulse. It is discussed that intracellular actin filaments may also influence motions at the neuronal membrane.

Many sensory nerve cells are excited due to exogenous or endogenous mechanical impulses. It may plausible that such impulses are conducted directly to the sensory nerve cell bodies in the dorsal root ganglia without the transformation in electric energy. Excitation conduction happens without noteworthy energy consumption because the flow of ion currents through the membranes takes place equivalent to the concentration gradient.

Impulse waves cause short extensions of the lipid membranes of the cell- and fibres walls and therefore they can induce opening and closing of the included ion channels. This mechanism acts to "voltage gated" and "ligand-gated" channels likewise.

The concept of neuronal impulses can be helpful to the understanding of other points of neurophysiology or neuronal diseases. This includes e.g., the brain concussion and pathohistological findings in Alzheimer dementia.

To verify the concept of (mechanical) impulses in the nervous system it is necessary to carry out biophysical or mechanical investigations in very small dimensions and the authors hope to give for this a sufficient stimulus.

Introduction

It is general believed that a nerve fibre is a simple wire because of its electrical conduction properties, the cable like configuration and the proof of a membrane potential.

Cole and Curtis (1939) [1] and later Hodgkin and Huxley (1952) [2] investigated the flow of electric currents through the surface membrane of the giant nerve fibre of a *squid*. In addition sodium and potassium currents could be recorded with the voltage clamp technology, and their appearances showed a sharp dependence on the change of the membrane voltage. Hodgkin and Huxley developed a mathematical model which accounts for the conduction

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and excitation of the fibres. They were able to describe the relatively complex relationship in their famous differential equations. The calculated and experimental recorded action potentials as well as the sodium and potassium currents turned out to be largely identical. The Hodgkin and Huxley model remains the paradigm for conductance-based models of the nervous system [3].

Nevertheless there are fundamental differences between electrical cables and nerve fibres.

The electric propagation velocity of nerves (maximum about 120 m/s) is only a negligible fraction of propagation velocity of light, because ions move in nerve fibres and not free electrons. There are no storage batteries in highly developed organisms, which could send information in an electrical circuit (e.g., from the spinal cord to a peripheral muscle).

Hodgkin and Huxley's equations have been limited in their importance by the implementation of the patch clamp technique, and in this way, the possibility to examine small numbers of channels, or also of one channel alone. Up until now further channel types in particular for potassium, calcium and chloride as well as ligand-gated channels could be found by the patch clamp technique. However, this has not helped to understand the membrane kinetic in total (The Hodgkin and Huxley model is defined by a four dimensional vector field and there are dynamics in it, namely, the existence of a chaotic solution in the model with its original parameters [3].

The timing in opening and closing of the ion channels due to changes in the membrane voltage is difficult to comprehend since ion currents influence secondarily the membrane voltage.

In this paper, the authors want to demonstrate, that the elastic properties of cells, nerve and muscle fibres allow mechanical impulses to be carried (comparable with the blood pulse in the arterial vessel system), and therefore they can conduct energy or information. The impulse wave stretches the membrane, which reduces significantly the membrane resistance for ions. Through this mechanism, the "explosive" influx of sodium ions (with associated water) into the cell or the axon can create or can reinforce a mechanical impulse.

The impulse wave can open and close the ion channels in the membranes of fibres. The primary initiation of impulses in the nerve cells (e.g., at the axon initial segment) is an another question. However, the dendritic input and the great number of spines can act as a sum of minor impulses. The frequently described mechanosensitive channels in sensory receptors cells [4] are compatible with the assumption that the propagation of a great number of the neural circuits (e.g., polysynaptic muscle reflexes) is based entirely on mechanical impulses.

Electrophysical findings in neurophysiology

In order to maintain the cell specific electrolyte concentration, the ions flow through the membrane against their natural electrophysical distribution by the activity of ion pumps under the consumption of energy (ATP). The intra- and extracellular dissociation of the ion concentration is measurable as a cell specific membrane potential. A resting animal has to use one third of the available energy (ATP) to maintain its membrane potential [5]. The perpetuation of the cell specific dissociation of cations due to ion pumps enables the living organisms to have a response disposition.

At the time of excitation the ion currents flow gradients conform to the electrostatic potential through particular proteinaceous ion channels. Other ion channels are lined by cell specific receptors (ligand-gated ion channels) and react with specific neurotransmitters (acetylcholine, adrenalin, serotonin, GABA, glutamate, ATP and others) [6]. The transmitters may act as initiator, catalyst or modulator of the ionic flow in synaptic transmission and the efficacy is dependent on the dynamics of presynaptic transmitter release [7].

The typical action potential is sufficiently demonstrated in the physiology-textbooks: A rapid membrane depolarisation (+20–+30 mV) changes the resting potential (about −70 mV) to a peak overshoot potential of about +40 mV. After a few milliseconds the action potential returns to the level of the "resting potential".

The transmembranal current running during the action potential shows three phases: the first phase shows a mild outward current ("due to the passive cable spread of voltage and current" [8,4]); the second phase is caused by a fast and large sodium inward current (spike); and the third phase is due to a less intensive delayed outward potassium current, which occurs during the repolarizing phase [8].

It is assumed that the mechanism that ion channels open and close act by voltage changes (voltage-gated channels). For example: molecule groups of sodium-channel proteins can voltage dependent change their alfa-helix structure by torsion. It is supposed that the torsion is causing the opening of the ion channels.

According to an electrophysiological conception the propagation of the excitation is induced by a single action potential itself (voltage change). The voltage change in the excited area encroaches to the neighbouring unexcited area and induces there a depolarisation of the membrane with corresponding opening of the ion channels. In this way the action potential does a run continuously in non-excited regions.

Above it is stated, that a sodium-influx evokes the depolarisation of the membrane potential. The influx achieves the maximum in about 0.5 ms (measured at the giant axon of the *squid*), but the outward directed potassium flow achieves its maximum later (1.5 ms) and decreases more slowly [6]. May be because of the smaller diameter of the sodium, the sodium channels open first (mass number sodium 23, potassium 39, calcium 40, chlorine 35.5). In this assumption to the ions associated water molecules have to be taken into consideration [5], "especially" the tightly associated hydration water" [9]. Only minor amounts of Na^+- and K^+-ions flow during a period of an action potential through the membrane. Because the flow takes place equivalent to the concentration gradient and also the influx and efflux are equal it will be understandable, that the cell excitation occurs without noteworthy energy consumption. It is shown in experiments that cells after disruption of the energy supply (ATP) are still able to generate thousands of action potentials [6].

Nerve fibres can be covered by a myelin sheath like a coat sleeve. Thin fibres without a myelin sheath (e.g., vegetative fibres or type C-fibres) show a slower velocity of excitation propagation (about 0.5–2 m/s). The thickest fibres without a myelin sheath (giant axons of *squid* and *loligo* with an axon diameter of more than 1 mm) show a propagation velocity of about 20 m/s [8].

The Schwann cells generate the myelin sheath of peripheral nerve fibres. In the central nervous system this is done by oligodendroglial cells. Both cells wrap the axons with their own cell membrane (extruding the cytoplasm nearly completely), so that the sheath consists of numerous dense stacked lipid membranes. The ratio of the diameter of an axon cylinder to the diameter of the total fibre (including myelin sheath) is about 0.6–0.8 [8].

The thickest myelinated fibres (motoric fibres A alfa) reach a propagation velocity of up to 120 m/s. The myelinated fibre is interrupted at periodic distances by unmyelinated slim segments (1–2 μm in length) called nodes of Ranvier. In contrast to the myelinated internode the length of the node of Ranvier is 500–1000 times shorter.

The nodes of Ranvier show a very high density of sodium channels. However the internodal segments have only few sodium

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