Computer models of nerve membranes and axonal conduction were used to investigate the possibility of an electrical nerve block in both myelinated and unmyelinated nerves. The results suggest that a rapid and reversible electrical nerve block may be possible without activation of the nerve. This paper presents preliminary results from computer models used and their implementation.

Introduction

Present neural prostheses can impose only limited control over the peripheral nervous system. Neuralprosthetic control could be greatly enhanced if, in addition to being able to electrically excite axons, these devices could dynamically and selectively block axons. Applications -for such neural prostheses could include suppression of reflex activity in bladder and bowel, or mobility enhancement by suppressing spasticity in hemiplegia or cerebral palsy.

For a blocking technique to be used in these situations, ideally it must be quick to turn on and off and safe. The latter suggest biphasic waveforms and low frequencies of activation[1]. Tanner [2] noted that a 20 Kc alternating current, of an unspecified shape, appeared to suppress components of the compound action potential in dissected sciatic frog nerve. Woo and Campbell [3] confirmed Tanner's results. They also noted that the 20 Kc blocking could produce a tetanic muscular response without a characteristic compound action potential due to an asynchronous stimulation of large fibers.

Solomonow [4] described a block using rectangular monophasic pulses at frequencies below 20 kHz. This block was attributed to endplate depletion. Bowman [5] explored rectangular pulse blocking. He noted a rectangular pulse train could prevent nerve transmission in humans and cats and produce muscular responses that could not be attributed solely to endplate depletion. However, his results varied from trial to trial. Mortimer et. al. [6] demonstrated collision blocking in larger diameter axons through the generation of unidirectional pulse propagation using a cuff electrode and an intense quadratrapeziodal pulse. In the authors opinion the intensity of the quadratrapeziodal pulse could generate bidirectional action potentials in the smaller diameter axons. If this were so, this technique would be mainly suitable for pure motor nerves. Rattay [7], using a standard Hodgkin-Huxley model, demonstrated a local conduction block using in a 10µm axon using a monopolar electrode with a 2KHz waveform. In this paper we extend these computer simulations to include myelinated human nerves for the complete range of nerve diameters.

Methods

Models have been often used to predict experimental results. The parameters of a model can be changed to determine stability of these results. Hodgkin and Huxley described equations from which models of axons and action potentials could be constructed. This and other axonal models have led to the ability to test procedures without using animals. These models have been arrived at empirically, and hence are based on experimental results, not fundamental laws of nature. For this reason, a further demonstration of this technique is needed from animal trials. However, models are useful as a guide for potential animal testing.

Two myelinated axon and one unmyelinated axon models were used in this paper. The two myelinated models are the Frankenhauser-Huxley model for Xenopus Laevis axon and the Schwartz-Eikof model for rat sciatic nerve. The Hodgkin-Huxley model for the giant squid axon is the unmyelinated model. These models have been used previously to describe action potential generation.

To determine nerve blocking, the model must account for the propagation of the action potential from node to node. A separation of voltage across the membrane and external activating voltage field is made by this method. The voltage characteristics of an axonal node or element are also described by this method. To distribute the electrical nerve pulse, a monopolar electrode was investigated. Euler integration was used to solve the differential equations.

These computer models have allowed us to examine in detail the blocking affects. We were able to record the gating potentials of individual nodes of the axon and hypothesize the blocking mechanism.

The simulations were performed in MATLAB code and ran on 3 different pentium PC's of operating frequencies 60, 70, and 166 MHz. On the P166, a simulation using of 20 ms using 12 nodes and a 2 µs step took about 3 minutes.

Results

Localized high frequency blocking of action potentials is demonstrated on the three axon models. Slight differences of response to blocking stimulus were found for myelinated versus unmyelinated models. These are due to a greater distance between the nodes or node like elements that are to be simulated. The important difference, in response to the blocking stimulus, is that the Hodgkin-Huxley model can be excited by high-frequency current pulses which is in contrast to the Frankenhauser-Huxley and the Schwartz-Eikof models.

Figures 1 to 4 are examples of the block of a single axon's transmission. The Nodes of Ranvier for the myelinated
models, and segmented elements of neuron for the Hodgkin-Huxley model are separated vertically, so that an action

potential can be seen to travel on the figure up and to the right. The transmission upwards notes that the action potential propagates from one node to another. Time is on the ordinate axis. The action potential propagates from node to node as time increases to the right.

Figure 1 displays a train of action potentials in a from the Frankenhauser-Huxley model. Figure 2 displays the block of this transmission due to a high frequency blockade applied to node 6. Figures 3 and 4 are similar demonstrations from different axon models. It can be seen that the action potentials are blocked at the onset of the high frequency stimulation. Figure 5 represents the inactivation gate (h) probability of being open. This plot demonstrates that the h gate does not recover after the initial action potential to allow for the large influx of Na+ ions necessary for action potential propagation. Hence, the action potential does not pass through this node.

Discussion

The computer simulations demonstrate that there is the potential for a high frequency block of neural transmission not caused by endplate depletion. A single action potential at the onset of the blocking stimulation is seen in figure 1. This artifact is common to many simulations.

It is hypothesized that the high frequency current prevents the recovery of the h gate after an action potential from returning to a normal resting value. This affect can be thought of as an elongation of the refractory period of the neuron, during which action potential transmission is not possible.

This method of nerve blocking would have many advantages over other proposed methods of blocking. The pulses used were balanced and biphasic with no dc level. The block does not cause a high frequency repetitive firing which is known to be damaging to the neuron possibly due to abnormal Ca2+ levels due to the mass effect of all axons firing. The blocking is local to the area of the electrodes. It also follows the same type of recruitment order as FES, potentially allowing for selective stimulation of smaller fibers of the nerve. Clearly the next phase is to compare the results
with tests on living nerve.

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**References**


