A model of transduction in the frog muscle spindle

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Introduction

It has been known that a receptor potential may occur at the non-myelinated terminals in the frog muscle spindle and the receptor currents may be integrated in a Ranvier node of the myelinated branches at which afferent impulses may initiate.[1] The hypothesis that the receptor potential may be caused by an inward flow of sodium ions[2] is supported by recent works on mammalian muscle spindles[3] and crustacean stretch receptors.[4]

We, however, found calcium spikes at the sensory nerve terminal of the frog muscle spindle,[5] in addition to abortive spikes which were assumed to be sodium spikes propagating along the non-myelinated filaments in the terminal.[6,7] As the calcium spikes and abortive spikes can be induced by stretch of the muscle spindle and also they are supposed to play a role of trigger for afferent impulses,[5,7] several steps from the transduction to the encoding have to be considered in the frog muscle spindle. The present note describes a model of the ionic mechanisms for explaining the complex processes in the mechanoreception.

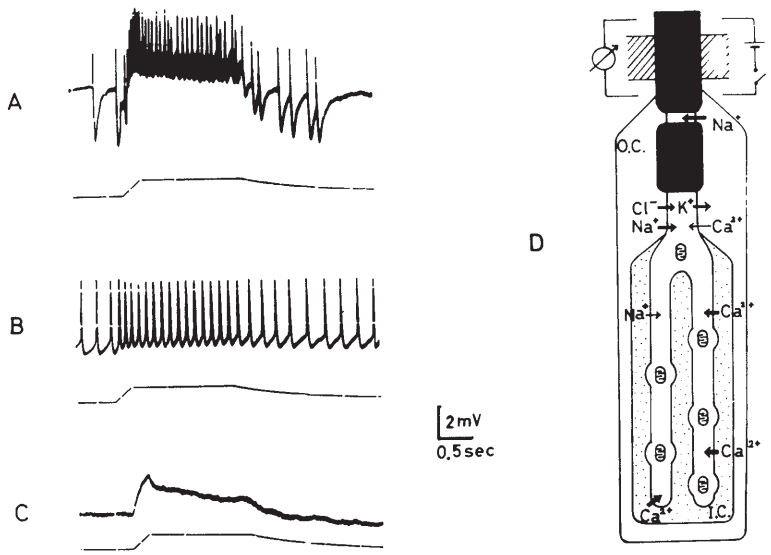

Fig. 1. Effects of 5 mM $CoCl_2$ (B) and Na^+ deprivation (C) on responses during stretch of a muscle spindle in comparison with the response in normal Ringer solution (A), and a schematic diagram of the distribution of ionic channels along the membrane of the nodes and terminals (D). O.C.; outer capsule. I.C.; inner capsule.

Methods

Muscle spindles were isolated from sartorius and semitendinosus muscles of the frog *Rana nigromaculata.* The sensory axon which innervates the spindle was isolated for 1 - 2 mm near the spindle capsule; the motor axon was removed. Just outside the capsule the myelinated segment of the axon was laid across an air-gap between two pools of Ringer solution made on two glass plates. Potential differences between the two pools were recorded through two Ringer-agar bridges immersed in the pools; each bridge was in turn connected to a calomel electrode which led to a differential high input impedance amplifier. The spindle was suspended between two stainless steel rods, connected to the tip of the levers of a differential electromagnetic puller, which stretched preparations symmetrically towards both poles at constant velocities (0.5 - 20 mm/sec).[8]

Results and Discussion

In a representative response of a muscle spindle during stretch in normal Ringer solution (Fig. 1A), dynamic and static discharges of sodium spikes occurred superimposed on the dynamic and static deflections of a spindle potential respectively. For 1 - 30 min after treatment of the spindle nerve terminal with 5 mM $CoCl_2$ or $MnCl_2$ which is known to block reversibly calcium spikes,[5] the discharges of sodium spikes occurred more rhythmic than that before the treatment, without any deflection of the spindle potential (Fig. 1B). The discharges and responses to stretch disappeared approximately 30 min after application of the calcium blockers. These effects were reversible if exposure to the drugs was not prolonged beyond 1 hr. Fig. 1C shows a spindle potential during stretch of the same preparation in Na^+-free Ringer solution after the recovery. The above results suggest that afferent discharges in normal Ringer solution may consist of two kinds of spikes triggered by calcium and abortive spikes and also that the spindle potential is probably a secondary response which may be due to a change in calcium permeability at the nodal membrane in the immediate vicinity of the site of origin of afferent discharges. The fact that all the responses to stretch are blocked by long-lasting application of calcium blockers leads us to another assumption that a genuine receptor potential may be due to an increase in calcium permeability at the non-myelinated terminals enclosed in inner capsule where is supposed to be more distal to the site of abortive and calcium spikes.

We would like to propose a model as shown in Fig. 1D for elucidating coherently the above suppositions. The density of Ca channels may increase instead of Na channels from the nodes of the myelinated branches to the terminal of the non-myelinated filaments. The Ca channels at the non-myelinated terminal may play a role as a mechano-transduction, but those near the Ranvier nodes as an encoding mechanism.

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