TEMPERATURE AND DENDRITIC RESPONSE OF SPINAL MOTONEURONS

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Well-established are the facts that the dendrites of spinal motoneurons carry impulses, at least in the cellulifugal sense, and that they do so with a decrement.¹-⁴ Whether or not they do these things in the cellulipetal sense is not a matter of present concern. The experiments to be discussed relate to “antidromic” conduction in dendrites of spinal motoneurons of the cat and the influence upon it of temperature change, an essentially unexplored problem.

Utilizing the fact that the dendrites of those motoneurons that supply the small plantar muscles extend laterally to the surface of the cord,¹ one can by chilling the cord surface produce a temperature change confined essentially to the dendrites themselves. In practice the warmed oil routinely employed to cover the exposed spinal cord was replaced by chilled oil which produced a rapid fall in temperature. During the ensuing 45 or so minutes, with the aid of an infrared lamp, temperature at the cord surface returned over a roughly exponential course to normal. Throughout this time dendritic responses were elicited and recorded from the point of maximal response⁵ on the cord surface, which is to say at the periphery of the motoneuron pool, by means of an electrode pitted against another at a distance. A thermocouple, by means of which temperature was recorded, was placed immediately adjacent to the electrode on the cord surface.

Figure 1 illustrates the manner in which dendritic response grows as temperature is lowered from 37° to 32°C. Since the superimposed recordings are identical until some time (indicated by an arrow) after the region ceases to be a source of current flow to impulse sinks in the more central parts of the motoneurons, it follows that there has been no influence upon conduction through the axons, the cell body, and the proximal dendrites. If, by way of contrast, body temperature generally is lowered, not only is the negative phase of dendritic response increased but so too is the prodromal positive phase, and the onset of each phase is later in time, all of which indicate the more widespread field of effect. Thus it would seem that the procedure employed reveals change in the more peripheral stretches of the dendrites and, furthermore, change that is primarily there rather than secondarily the result of change elsewhere.

In Figure 2 is plotted amplitude of the dendritic responses as a function of temperature in the range between 20° and 38°C. A ceiling of amplitude exists between 21.5° and 25.5°C above which temperature range there is a linear decrease in amplitude with increase in temperature. On the average in these experiments the maximum, and ceiling of, dendritic response occurred between 20.5° and 24.5°C. This result is in sharp contrast with that of Chang⁶ who found the cortical “dendritic potential” to be maximal in the range of normal body temperature and to fall off in the amplitude to practical extinction at 20°C. No simple explanation for this discrepancy presents itself, but the fact suggests that it is unwise to generalize concerning the activity of dendrites.
A striking aspect of the increment in amplitude with suddenly falling temperature is its rapidity. When the cord surface temperature was lowered suddenly to 21°C, dendritic response reached a ceiling within 20 seconds, remaining at that amplitude for 2 minutes during which time temperature rose to 25.5°C. With continued rise in temperature, response declined in the manner indicated by Figure 2.

That the decline in amplitude at temperatures below 21°C is real and not merely caused by the time required for the dendritic response to increment to ceiling is indicated by another experiment. Temperature at the cord surface was reduced to 19.6°C. In this instance a minute passed before ceiling was reached during which interval temperature rose from 19.6°C to 22°C. Ceiling amplitude was maintained as temperature rose to 24.5°C after which the decline with further temperature rise occurred. Thus, at temperatures below approximately 21° to 22°C there is a progressive establishment of cold block.

The response of dendrites is augmented in other circumstances: during impingement of presynaptic activity and during anoxia or asphyxia. Interaction between two causes of increment can be studied to best advantage by combining temperature change and presynaptic impingement. To do this, long spinal reflex activity has been employed to provide the requisite facilitatory background. Responses of the dendrites in isolation and in suitable temporal combination with a long spinal reflex volley were alternated throughout the period of temperature change. In this way the amplitude of response and the degree of facilitation produced by the long spinal reflex background can be measured at any given temperature. The result is depicted in Figure 3.
Figure 3 shows that the facilitation of dendritic response produced by long spinal reflex action is in degree inversely proportional to size of the unconditioned response. In the range of temperatures at which the unconditioned dendritic response is at maximum there is no increment of response consequent upon long spinal reflex bombardment of the motoneurons. These facts would seem to indicate that the region of the dendrites in which decremental conduction reliev- able by presynaptic action occurs is the same as that in which decrement reliev- able by cooling takes place. Were it not, in which case the region of decrement reliev- able by presynaptic action would necessarily be closer to the cell bodies, one would expect presynaptic action still to be effective when the temperature effect is maximal. Thus it would appear that decrement in the main is confined to the distal region of the dendrites.

![Figure 3](image-url)

**Fig. 3.**—Amplitude of dendritic response and degree of facilitation produced by presynaptic activity plotted as a function of temperature.

Clearly the three agencies presynaptic activity, aphyxia, and cooling all act to increase decrement of dendritic conduction by causing a depolarization of the dendritic membrane. Increase in membrane potential, such as occurs during the recovery cycle of motoneurons, or in the post-anoxic state, causes an increase in the decrement. In other words decrement in dendritic conduction is dependent upon the level of membrane potential. That fraction of membrane potential determining decrement is highly labile for the effects of temperature change, and of anoxia, are large and rapid. It is noteworthy in this connection that the change in membrane potential caused by cooling of nerves is confined largely to the L-fraction.

Presynaptic activity in bringing about depolarization acts directly upon the excitable membrane and only secondarily upon the metabolic mechanism to restore the membrane potential depleted by synaptic bombardment, or to maintain it in the face of "resting" activity. Temperature change and anoxia differ in that they act not directly upon the membrane, but rather upon the metabolic system that maintains the membrane potential. Since the decrement of dendritic conduction in degree is related (although the precise relation is not known) to the level of membrane potential, and since the membrane potential is determined by an equilibrium between electrostatic forces and chemical forces dependent upon oxidative metabolism, it follows that amplitude of the dendritic response between
zero and ceiling is an expression in the inverse sense of the rate at which the metabolic processes are operating.

It is of interest that the dendritic membrane, given adequate oxygen, normal carbon dioxide and maintained at normal body temperature, should be set at a level of polarization so far from that at which maximal response is possible. One must suppose that dendritic conduction with decrement toward the tips is the normal occurrence and important to the normal working of the motoneurons. It would occur, for instance, not only in artificial "antidromic" action, but also in association with monosynaptic reflex transmission, for the presynaptic endings concerned are located on the cell body and the thick proximal dendrites,11, 12 and the impulse generated there would travel toward the dendrite tips as well as along the axon to the periphery.

10 Lorente de Nó, R., A study of nerve physiology, Studies from The Rockefeller Institute, 131, 132, 1947.

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DECREMENTAL CONDUCTION IN PERIPHERAL NERVE. INTEGRATION OF STIMULI IN THE NEURON

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The purpose of this communication is to reinstate an important piece of knowledge, the doctrine of decremental conduction in peripheral nerve, which existed in the classical literature, but which was lost some 35 years ago, when, mistakenly, physiologists of prestige deemed it erroneous.