how be related to underlying vascularization is not new and was first pointed out by Redslob[6]. Whether other conditions such as those affecting the skin show a relationship between melanocyte migration and neovascularization is yet to be determined.

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End-plate Cholinesterase in Dystrophic Muscle

THE cholinesterase activity at the motor end-plate of dystrophic muscle was investigated histochemically by the thiolacetic acid method of Barrnett[1], a modification of that of Crevier and Belanger[2], which results in deposition of lead sulphide at the reactive site. A relatively quantitative method was developed to study this reaction by utilizing varying concentrations of physostigmine salicylate, a competitive inhibitor of cholinesterase.

The muscle tissue was obtained from dystrophic mice (strain 129, dydy) and unaffected litter mate controls. Excised tibialis anticus muscles were prepared histochemically for cholinesterase in varying concentrations (1×10^{-5}–1×10^{-9} molar). Two methods were used: exposure of whole muscle to physostigmine for 15 min, followed by the histochemical reaction, then sectioning; and exposure of fresh sections (after cryostat preparation with freezing) to the inhibitor for 20 min before histochemical reaction.

The reactivity of the fresh muscle sections was the most sensitive; total inhibition of cholinesterase activity in normal muscle requiring 1×10^{-6} molar physostigmine, and the dystrophic muscle 1×10^{-8} molar, a one-hundred fold difference. The whole muscle preparation was less sensitive, but similar orders of magnitude were obtained: total inhibition of cholinesterase activity in the normal requiring 1×10^{-5} molar physostigmine and at 1×10^{-7} molar an almost complete inhibition of dystrophic end-plate reactivity. Comparative results of end-plate counts from sections of prepared whole muscle at a concentration of physostigmine of 1×10^{-7} molar are presented in Table 1. At this concentration, the number of end-plates reacting was reduced to about one-third in normal muscle, but to less than one-tenth in dystrophic muscle. It should be noted that the actual number of end-plates in uninhibited dystrophic muscle is about one-third less than that in the control.

These findings lead to the conclusion that there is likely to be a marked reduction in the amount of available cholinesterase in this variety of dystrophic muscle. The effect of this lack could result in increased acetylcholine activity at the synaptic junction of the end-plate and can be correlated with previous studies indicating increased excitability of dystrophic muscle: spontaneous multiple action potentials[3], shortened refractory period[3], prolonged tetanic-like responses to nerve stimulation[4], delayed fatigue reactions with greater resistance and increased threshold to fatigue[4–6], increased time decay constant of miniature end-plate potentials[7], and hypersensitivity to anticholinesterase drugs[8].

Table 1. MUSCLE END-PLATE COUNTS FROM SECTIONS AFTER WHOLE MUSCLE EXPOSED TO 1×10^{-7} PHYSOSTIGMINE CONCENTRATION

Muscle	No. of sections	No. of end-plates	End-plates/section
Control uninhibited	10	650	65
Control inhibited	13	287	22
Dystrophic uninhibited	13	552	42
Dystrophic inhibited	14	48	3

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Effects of Various Ions on the Resting and Action Potentials of the Giant Nerve Cells of the Leech *Hirudo medicinalis*

IN each segmental ganglion of the leech, pairs of colossal cells (cell of Retzius) are found and bidirectional electrotonic type of transmission exists between these two neurones[1,2]. The amplitude of the spike is small, in the range of 20–30 mV. Because the resting potential of the cell is 30–50 mV, no obvious overshoot occurs. It is thought that no conduction of impulses occurs in the cell bodies of colossal cells[1,2]. This communication considers the effect of various ions on the electrical activity and electrical connexion between these cells.

The ganglion was isolated in physiological saline[3] and its connective tissue capsule was opened in order to expose the nerve cells and their glia. Two or three microelectrodes were inserted into the two nerve cells to record changes in the potential of the membrane and the transmembrane current.

Following substitution of sodium by sucrose, action potentials were absent (Fig. 1*A*).

The amplitude of the action potentials was increased by addition of barium chloride (10 mmolar) to the saline, the normal action potential changing to a prolonged action potential of 300–500 msec duration (Fig. 2, 1*b*). Membrane resistance of the cell was decreased as compared with its initial value in rest (Fig. 2, 1*a*).

Persistence in the calcium-free solution was a characteristic feature of these cells, and electrical activity was recorded for some hours. In these conditions resting and action potential increased and resting membrane resistance of the cell decreased (Fig. 1, *B*, *C*).

The voltage attenuation between the cells in calcium-free solution in practice was the same as in normal saline, so that the changes in membrane resistance were not caused by disturbance of the connexion between these cells (Fig. 1*D*). Penn and Loewenstein[4] have recently pointed out that the calcium ion participates in maintaining electrical activity between nerve cells. Calcium-free saline produces an increase in junctional resistance and upsets the synchronous discharge of these neurones.

When soaked continuously in a solution free of calcium, the cell spontaneously generated prolonged action potentials which are smaller in amplitude but greater in duration (5–10 sec) than those in barium solution (Fig. 2; 2, 3, 5). In the initial phase of prolonged action potential activity

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