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Influence of Various Ions on Fatigue Contracture.

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When 2 cc. of 1% sodium bicarbonate is injected intraperitoneally in frogs there results, in about one-half hour, a disorder of the skeletal muscles characterized by fascicular twitching and dystonic movements. This was described as tetany by Ringer[1] in 1884. A similar condition develops about one-half hour after the intraperitoneal injection of 2 cc. of a neutral 5% solution of sodium phosphates. We have not been able to produce this type of reaction by administration of sodium oxylate, sodium fluoride, potassium chloride, calcium chloride, magnesium chloride, ammonium chloride or hydrochloric acid, even when these substances were given in doses large enough to cause complete prostration.

We have studied the effect of all of these substances on the fatigue curve of the isolated gastrocnemius of the frog. The muscle was stimulated directly through silver chloride electrodes with faradic shocks delivered at the rate of 3 makes and breaks per second. Care was taken to have the stimuli always the same and strong enough so that the make shocks gave maximal contractions. The normal muscle when stimulated in this way responds at first with discrete contractions returning to the base line. After 15 or 20 seconds relaxation becomes delayed and fatigue contracture slowly develops resulting in a steadily maintained contracture of approximately the same height as the original discrete contraction.

A muscle from a frog treated with a neutral solution of sodium phosphates responds in a very different manner. Even after the first contraction, relaxation is much delayed and the muscle remains shortened with contractions of small magnitude superimposed upon the contracture curve. When the stimulation is discontinued, relaxation is slow and imperfect.

Strikingly different curves were obtained from the muscles of frogs poisoned with 0.4% sodium fluoride, the fluoride being substituted for that amount of sodium chloride in Ringer's fluid. When a frog was given 2 cc. of this solution prostration and muscular flaccidity occurred in about an hour and a half. Muscle isolated from such a frog responds in a normal manner for about a dozen contrac-

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[1] Ringer, Sidney, *Brit. Med. J.*, 1884, ii, 114.

tions and then rapidly fatigues at the base line. This type of fatigue curve is due to the failure of contraction and is to be sharply distinguished from the fatigue contracture of normal and phosphate muscle, where the fatigue shows itself as a failure of relaxation. A point of special interest is that shortly after fatigue is complete the muscle begins to shorten. This shortening continues after cessation of the stimulus and the muscle rapidly goes into rigor. The lifting power of the fluoride muscle is not impaired at first as was shown by tracings made with 50 gm. loads.

A 1% solution of sodium bicarbonate injected intraperitoneally into the frog alters the fatigue curve in much the same manner as phosphate. These observations are of interest in connection with the retention of phosphate in parathyroid tetany[2] and the development of tetany in normal individuals when alkalosis is produced by prolonged rapid and deep respirations.[3]

Sodium oxylate which is known to precipitate calcium in the blood stream[4] as well as *in vitro* and which might be expected to cause rigidity does not have this effect. Fatigue contracture develops just as slowly as in normal muscle. Calcium chloride, even when administered in quantities sufficient to prostrate the frog does not alter the fatigue curve from normal.

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Effect of Electric Currents on the Arteries.

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In a series of experiments the effect of electric currents of low tension (110 to 220 volts) on the femoral artery of the dog has been tested. These currents do not cause any anatomical changes if the blood is circulating freely. Combination of the passage of the current with a stopping of the circulation at a point distal to the electrodes leads to a complete destruction of the muscle fibres of the media, while the elastic fibres retain their specific staining properties but lose their elasticity. It is suggested that the heat that is liberated in the vessel wall produces this effect. The circu-

[2] Herring, W., and Kuhnan, J., *Endocrinology*, 1927, xi, 80.

[3] Grant, S. B., *Arch. Int. Med.*, 1922, xxx, 355; *Am. J. Physiol.*, lxvi, 274.

[4] Vines, H. W. C., *Endocrinology*, 1927, xi, 290.