In another experiment with bull frog muscle at all 3 frequencies $r = 198$, $C = .195$ and $R = 98$.

If r is made zero a balance may still be obtained but varies with frequency as shown in the following table (together with the change in resistance and capacity with stimulation).

It may be seen from the table that the resistance invariably decreased on stimulation. Change of shape may have an effect on resistance. Since the muscle placed between the electrodes had muscle fibers extending in various directions, it was thought that the effects of change of shape of the different fibers might tend to neutralize one another. The fact that there was always a decrease in resistance is considered as indicating that there was a decrease in resistance of the individual muscle fibers. The changes in capacity are not so regular.

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The Effect of Hypercalcemia on the Creatin Output in Myasthenia Gravis.

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The discovery by Fiske and Subbarow[1] of muscle phosphocreatine as a labile compound with a definite relation to the functional condition of the muscle, again awakened the interest in those diseases of the (neuro)muscular system, which are characterized by creatinuria, *viz.*, progressive muscular dystrophy and myasthenia gravis. The occurrence of acute attacks of muscular incompetence in myasthenia gravis versus the more steadily yet slowly progressive course of the muscular dystrophy made us choose the former disease for our experiments. The question whether the escape of the creatine from the muscle is due to an error of metabolism of the sarcoplasm or to an abnormal permeability of the sarcolemma presents itself. Through the parathyroid hormone preparation by Collip it has become possible to influence the blood calcium level sufficiently for the carrying out of cell-membrane permeability experiments in human subjects.

We report briefly an experiment designed to demonstrate the effect of hypercalcemia on the creatin output in myasthenia gravis. The patient was 45 years, with myasthenia gravis of one year's duration, the diagnosis having been made in the neurological divi-

sion. This patient is also the chief subject of the observations reported in the following paper. During the experiment the patient was on a creatine- and creatinine-free diet. The usual Folin methods were used for the urine determinations, and Clark's and Collips' method[2] for the plasma calcium.

TABLE I.

Day of experiment	Procedure	Serum Ca per 100 cc.	Creatinine per 100 cc. urine (daily average)	Creatine per 100 cc. urine (daily average)
		mg.	mg.	mg.
1-12	Fore period	10.6	1.05	.65
13-16	5 cc. 10% $CaCl_2$ intravenously daily	11.0	1.12	.55
	13-15 day	10.6		
17-19	Intermediary period		1.11	.65
20-29	Units of parathyroid hormone each day: 18, 20, 55, 55, 60, 75, 50, 75, 75.	10.7		
		12.9	1.16	.57
	Ca-lactate, 9 gm. daily	13.3		
30-36	Afterperiod		1.06	.58

The table is self explanatory. The blood calcium shows no increase on intravenous administration of $CaCl_2$ alone. On subcutaneous administration of parathyroid hormone (Lilly) in combination with large doses of Ca lactate by mouth (9 gm. a day) the blood calcium slowly rose from 10-11 mg. to 13.3 mg. per 100 cc. serum. At this point the patient complained of headaches and the hypercalcemia was no longer kept up. During no period, neither during the intravenous Ca treatment nor during the parathyroid treatment did the creatine or creatinine figures show any significant changes.

From the point of view put forward in the introduction we consider the experiment worth reporting.

[1] Fiske, C. H., and Subbarow, Y., *Science*, 1927, N. S. lxv, 401.

[2] Clark, E. P., and Collip, J. B., *J. Biol. Chem.*, 1925, lxiii, 461.