juncture by means of a right-angle iron attached to the anterior surface of the fore leg of a study chair. The top of the mantle conveniently passes through a circular hole in the writing ledge of the study chair. The writing ledge acts as an arm and finger rest for the subject. The mantle can be quickly removed from its supporting position and thoroughly cleaned without difficulty.

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# SPECIAL ARTICLES

## THE MYOGRAM OF THE ISOLATED SKELETAL MUSCLE CELL

THE determination of the contractile mechanism of a muscle cell from results procured in experiments upon an intact muscle presents the difficulty of differentiating between those factors attributable to the cellular process *per se* and those due either to a statistical distribution of the contractility of the fibers or to mechanical interference of the connective tissue enclosing the fibers. Moreover, the methods employed give no approach to the functional significance of the recognized structural elements within the muscle cell. The present investigation was undertaken, therefore, to develop a method of investigating the contractile and structural properties of single isolated muscle fibers. Opportunity is taken at this time to present a preliminary report of the results. A complete account will be published later.

### METHODS

The method is to mount the single muscle fiber upon the tips of two glass needles, one needle being rigid, while the second, the micro-lever, is flexible so that its tip is free to move when the muscle contracts. The needles, mounted in a Chambers' micro-manipulator, are held in the field of a microscope. The image of the movable lever is projected upon the slit of a recording camera and adjusted at right angles to the slit by means of a suitable optical system.

For the glass micro-lever used it has been determined that the displacement of the tip of the lever is a linear function of the force applied.[1] The contraction curve of the muscle, therefore, can be converted into absolute units when necessary.

The micro-levers have a period of about 0.0016 seconds. This is a sufficiently short period for recording the contractions of these muscle fibers.

The muscle cell is stimulated by break induction shocks applied by silver-silver chloride micro-electrodes.

Preparations of the isolated cells of the sartorius muscle, 1 cm to 1.5 cm in length, are used. These are mounted in a hanging drop of frog serum in the customary moist chamber.

[1] The method of calibrating the glass micro-levers will be published elsewhere by the junior author.

### RESULTS

The results obtained can be described to the best advantage by reference to the records of the contractions in Fig. 1. These records show the following:

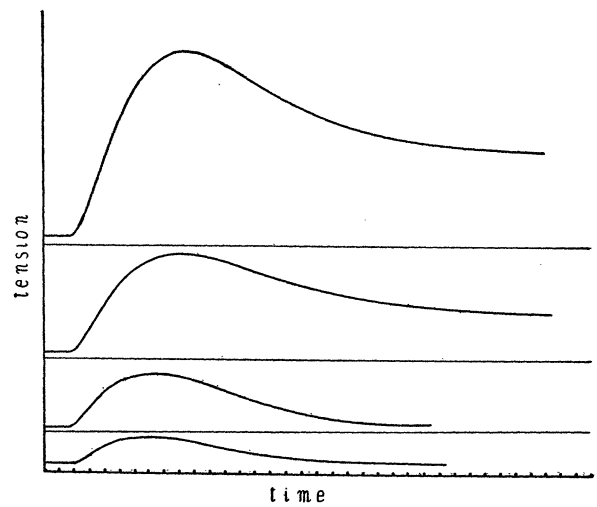


FIG. 1. Myograms from a single isolated fiber of the sartorius muscle showing a progressive increase in the magnitude of each response with increasing strength of stimulus. Temperature 23° ± 2° C. Time intervals represent 0.01 seconds.

(1) In the simple twitch of a skeletal muscle fiber the tension at the beginning of contraction rises abruptly, increases to a maximum, and then decreases to zero in a curve without discontinuities. No plateau or angle such as described by Fulton[2] for the intact muscle exists. These results are in agreement with those of Cooper and Eccles,[3] who ascribe Fulton's results to frictional interference in the muscle lever he used.

(2) An increase in the strength of stimulation results in an increase in the total tension developed and in the duration of the contraction. With strong stimuli the recovery from contraction is not complete, a new base line being reached.

[2] J. F. Fulton, Williams and Wilkins Co., 1926.
[3] S. Cooper and J. C. Eccles, *J. Physiol.*, 69, 1930; *Proc. Physiol. Soc.*, III.

The variation in the magnitude of contraction with variation in the strength of stimulus indicates that the all-or-none law does not apply to the electrically stimulated muscle cell. Gelfan[4] has reached a similar conclusion for the fibers of the retrolingual membrane of the frog stimulated *in situ*. Before a definite conclusion can be reached in the present investigation it is necessary to determine whether the stimulating current is affecting only the excitable system involved in any type of stimulation no matter how produced, or whether there are also direct electrochemical effects of the current upon other components of the cell. Some evidence that such changes are involved has been obtained and the subject is now under investigation from the view-point of the relation between the chronaxie of the cell and the magnitude of the response. In any case a more rigid statement of the all-or-none law in terms of the excitable system of the cell than is customarily given is necessary.

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## THE MYOCARDIUM IN YELLOW FEVER[1]

### A PRELIMINARY REPORT

A SERIES of twenty rhesus and cynomolgus monkeys has been studied electrocardiographically before inoculation with yellow fever virus (Asibi strain) and at varying intervals during the course of infection with yellow fever. Of these animals only two recovered from the disease. The functional changes in the sino-auricular node, auricular muscle, auriculoventricular bundle and ventricular muscle recorded by electrocardiography have been compared with the histopathological changes in the corresponding cardiac tissues of each animal.

Bradycardia, regular in rhythm, absolute in degree and progressively more marked on succeeding days of the disease, has been a constant finding in experimental yellow fever in the monkey. The heart rate in these animals has been reduced in varying degrees ranging from 75 to 30 per cent. of the normal frequency. The bradycardia persisted independently of ether anesthesia and sodium iso-amyl-ethyl barbiturate anesthesia, and following bilateral section of the vagus nerves. This retardation of cardiac rhythm was associated with hyaline, granular, vacuolar and fatty degenerative lesions in the musculature of the sino-auricular node. In the animals which recovered from the disease the normal heart rate was gradually resumed.

Occasionally reduplication of the P wave of the electrocardiogram was observed in experimental yellow fever; more rarely this deflection was seen to be inverted. Well-marked degenerative changes have been found in the auricular muscle.

Prolongation of the conduction time of the auriculoventricular bundle, as measured by the P-R interval of the electrocardiogram, was observed in slight or moderate degree in 84 per cent. of cases. With few exceptions the delay in conduction was progressive, increasing from day to day of the disease. The P-R periods regained their normal values in the animals which recovered. This impairment of conduction was associated with the appearance of granular, fatty and vacuolar degeneration in the fibers of the auriculoventricular bundle.

Among electrocardiographic alterations in the ventricular muscle during the course of the disease, changes in ventricular preponderance were commonly observed. The R-T period was lengthened in 94 per cent. of cases, and frequently it was deformed. The T wave commonly took an early origin from the descending limb of the R wave or the ascending limb of the S wave with concurrent distortion of the R-T segment of the ventricular electrocardiogram. The normal upright T wave was replaced in 76 per cent. of the cases by a deflection either negative in direction or diphasic in contour, the latter change being represented either by two waves above the line of equipotential or by one positive and one negative wave in relation to the base line. In addition to these types of deformity of the terminal deflection, an upright T wave of increased height was frequently observed. These abnormal variations of the ventricular electrocardiogram appeared most frequently on the later days of the disease; but these disturbances were transient, fleeting in nature, present in one tracing and not in another, one type of variation being inscribed upon one occasion, a different deformity appearing during subsequent annotations. Their occurrence was associated with the presence in the ventricular muscle of these animals of hyaline, granular, vacuolar and fatty degenerative lesions.

A detailed account of which the above is a summary is in preparation and will be published in the near future.

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[4] S. Gelfan, *Amer. J. Physiol.*, 93: 1, 1930.

[1] This work was performed under the tenure of a grant from the Banting Research Foundation. The electrocardiographic studies were carried out in the Yellow Fever Laboratory of the Rockefeller Foundation and in the Physiological Laboratory of the Rockefeller Institute in New York, through the kindness of the directors of these laboratories, Dr. W. A. Sawyer and Dr. A. E. Cohn.