quently taken at what was assumed to be the "height" of shock, namely 5 to 15 minutes after the shocking dose of serum. Consequently we had many experiments of moderate degrees of shock in which the blood samples examined were negative, although the thoracic duct lymph samples were positive. These latter were, however, collected continuously from the onset of shock. In view of our findings on the rapidity of the disappearance of injected histamine we have repeated a number of experiments on anaphylactic shock and are now able to report that in anaphylactic shock of moderate degrees of severity, blood samples taken at one, 2 or 3 minutes after the shocking dose of serum are usually active, although later samples may be negative. It is also seen from this work that an occasional animal may die from a dose of histamine (1.5 mg. per kilo) which is insufficient to be regularly detected in the thoracic duct lymph. In our original report on anaphylactic shock it was noted that in 2 of 9 cases of fatal shock we failed to find any activity in the thoracic duct lymph. These findings with histamine therefore afford circumstantial evidence of the validity of the histamine theory of anaphylactic shock in the dog.

## 8124 P

### Muscle Atrophies. I. Water and Nitrogen Studies in Simple Disuse Atrophy.

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Histologic, chemical, and physiologic studies have been undertaken in various types of experimentally produced muscle atrophies in young *Macacus rhesus* monkeys. In a series of 6 animals, simple disuse atrophy was produced in the right leg of each animal by immobilizing it in a plaster cast. The left leg was used as the control in each case. The gastrocnemius and soleus muscles were completely removed by careful dissection from their proximal and distal attachments while the animals were under light ether anesthesia. The excised muscles (6 atrophied and 6 controls) were then weighed and split longitudinally, one-half being used in the preparation of

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histologic sections and the other used for chemical studies. The half used for chemical analyses was weighed immediately, sliced into several pieces, and desiccated over sulphuric acid under reduced vapor pressure (20-30 mm. Hg.) until a constant weight was obtained. Nitrogen was then determined quantitatively by the Kjeldahl method, duplicate determinations being made for each of the 12 specimens, and the protein content calculated.

The loss in muscle weight varied from 4.9% in the one-week atrophy to 32.5% in the 10-week atrophy. Despite the decrease of muscle bulk in the atrophied muscle as compared with the respective controls, the relative proportions of water and protein remained substantially the same as in normal muscle. In the 10-week atrophy, for example, the variation in protein content between the control and the atrophied leg was but 0.3% and the difference in water content negligible.

Histologically the chief finding was a uniform shrinkage of the muscle cells. The muscle fibers were narrower but the characteristic appearance of the transverse striations remained unaltered. There was no increase in sarcolemmal or muscle nuclei nor in the connective tissue. The intramuscular blood vessels, nerves, and nerve endings were likewise unaltered. The significant change observed consisted of a diminution of the cross-section area. This change seemed to involve primarily the sarcoplasm, as evidenced by a more compact arrangement of the sarcostylic elements within the individual muscle cells. This was especially marked in cross-section in which Cohnheim's areas were no longer visible. The sarcostyles appeared distributed in a uniform manner throughout the muscle cells.

Electrical stimulation of the atrophied muscles with faradic and galvanic currents gave prompt responses, similar to those obtained in normal skeletal muscle.