located in the zero horizontal plane and while one was in the midline the other 2 were 5 mm. on either side of the midline. Four other wires were inserted vertically in a plane 17.5 mm. in front and 2 more in a plane 7.5 mm. in front of the zero point.

The brain was then dissected out and imbedded in celloidin. The horizontal wires were withdrawn, leaving minute tunnels through the brain and celloidin in the zero horizontal plane. The 2 wires in the frontal plane located 7.5 mm. rostrally were also pulled out, leaving similar tunnels marking the position of that plane. The block was then cut along the rostral surface of the 4 wires in the frontal plane 17.5 mm. in front of the zero point and the brain cut with the microtome into serial sections parallel to the frontal plane thus established.

Each frontal section has in it 3 small round holes produced by the wires inserted on the zero horizontal plane. One centimeter divided by the number of sections included between the 17.5 and 7.5 mm. planes gives the thickness of each section in terms of the unimbedded brain. This eliminates the error which would be introduced by shrinkage during imbedding and makes it possible to assign each section to its correct frontal plane.

Since the frontal plane to which each section belongs is accurately known and since each section contains 3 small round holes in the zero horizontal plane from which measurements can be made it is possible to fix with great accuracy the location of any nucleus or fiber tract and to express it in terms of rectilinear coordinates with reference to the zero point.

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Carbohydrate Metabolism in Degenerated Striated Muscle.

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(Introduced by J. P. Simonds.)

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Twenty-three rabbits used in these experiments were put under light ether anesthesia, and the gastrocnemius muscle injured either by freezing with carbon dioxide snow or by multiple light blows. After 24 to 72 hours the animals were given amytal intraperitoneally and the gastrocnemii removed after being frozen solidly with

carbon dioxide snow and ethyl chloride.[1] The muscles were analyzed quantitatively for glycogen,[2] lactic acid,[3, 4] and organic and inorganic phosphorus.[5]

The average glycogen value in normal rabbit gastrocnemii was 603 mg. per 100 gm. of muscle. This dropped to 258 mg. in those muscles injured by previous freezing, and to 103 mg. in the contused muscles. From microscopic studies of the lesions it was evident that degenerative changes were more profound in the contused than in the frozen muscles. With this decrease of glycogen there was a concomitant increase of lactic acid from a control average of 19 mg. per 100 gm. of muscle, to 53 mg. in the frozen muscles and 66 mg. in contused muscles. Phosphocreatine decreased markedly from an average control value of 66.3 mg. per 100 gm. to 7 mg. in the contused muscles. Contrary, however, to expectations the inorganic phosphorus likewise decreased slightly, from 44.5 to 36.1 mg. per 100 gm.

Microscopically there was extensive molecular degeneration of the muscles, with consequent interference with the capillary blood circulation, although the larger vessels had free circulation.

The results given suggest damage to the glycogenic function or to the glycogen storing capacity of the muscles. The still-active glycogenase depleted the muscle glycogen store, with the subsequent formation of lactic acid. While some of the acid may have diffused out into the surrounding tissues or larger vessels, enough remained to show the character of the change. This increase of lactic acid was accompanied as usual by a marked drop of phosphocreatine. The decrease of inorganic phosphorus also may indicate loss by diffusion.

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[1] Davenport, H. A., and Davenport, H. K., *J. Biol. Chem.*, 1928, **76**, 651.

[2] Pflüger, E., *Arch. f. die Ges. Physiol.*, 1909, **129**, 362.

[3] Friedemann, T. E., Cotonio, M., Shaffer, P. A., *J. Biol. Chem.*, 1927, **73**, 335.

[4] Davenport, H. A., and Cotonio, M., *J. Biol. Chem.*, 1927, **73**, 359.

[5] Fiske, C. H., and Subbarow, Y., *J. Biol. Chem.*, 1929, **81**, 629.