Glycogen Content in Normal and Hypertrophied Rat Heart.* (24375)

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It has been established(1,2,3) that glucose is one of many substrates which may provide energy for contraction of cardiac muscle. The degree to which oxidation of glucose contributes to energy requirements of the heart is affected by physical activity, state of nutrition and endocrine balance(3). Cardiac muscle glycogen is maintained at a remarkably stable level under conditions of increased cardiac stress associated with hypoglycemia(3), but varies with excessive stimulation such as severe anoxia and administration of epinephrine or thyroxin. The following experiments indicate that cardiac hypertrophy caused by exercise increases the glycogen content of rat hearts.

Methods. It has been demonstrated that cardiac hypertrophy can be produced experimentally in animals(4,5,6). In the present experiment, 22 male albino rats (Wistar Strain) were used. The animals were randomly divided into control and experimental groups of 11 each, weighing average 306 (242-353) and 280 (256-327) g respectively. All were fed Ranger Dog Pellets *ad lib*. The experimental rats were exercised at one mile/hour on motor-driven treadmill. On the first day, they were exercised two 15 minute periods with interval of 4 hours between periods. This time was doubled each day thereafter until they were running 2 one hour periods with interval of 4 hours between periods. Such a daily schedule was followed for 6 days a week until a total of 64 hours of running time was achieved(6). At the end of this period, both experimental and control rats were fasted 24 hours, weighed, and anesthetized with sodium phenobarbital. The thorax was opened, the heart removed, trimmed, drained and blotted free of blood and frozen by compression between blocks of dry ice. This procedure was completed within 30 seconds. The hearts were stored at –20°C. No change in content of glycogen could be detected as the result of this storage. The hearts were ground at approximately 0°C in a Potter-Elvehjem Homogenizer. Aliquots of the homogenate were taken for determination of glycogen by the method of Kemp and Van Heijningen(7). Duplicate samples were run on each heart. All samples were taken from the ventricular myocardium.

Results. Cardiac hypertrophy was produced by exercise. This hypertrophy was determined by comparing mean heart wt/body wt. ratio of experimental and control groups. The mean HW/BW ratio of the experimental group was 3.84 (3.09-4.22) as compared to 3.13 (2.72-4.10) for the control group. Calculation of the "t" value (4.14) shows this difference to be statistically significant. Mean concentration of glycogen in the experimental and control group was 717 ± 39 and 499 ± 66 mg/100 g of cardiac tissues respectively. A "t" value of 3.93 shows a significant difference between glycogen concentration of control and experimental groups.

Discussion. The results indicate that exercise produces a significant cardiac hypertrophy in albino rats as found previously in this laboratory(4). Our data indicate that there is a significant increase in concentration of cardiac glycogen in hypertrophied rat hearts as compared with normal controls. The increase in cardiac glycogen is an interesting observation since a distinguishing feature of cardiac muscle is its stable glycogen content.

Summary. 1. Cardiac hypertrophy was produced by exercise in albino rats. 2. There was a significant increase in concentration of glycogen in rat hearts with cardiac hypertrophy.

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Influence of Reticulo Endothelial Blockade on Turnover Rate of Homologous Plasma Proteins in Mice. (24376)

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Studies on formation of plasma proteins have established that parenchymal cells of liver are the main site of synthesis of serum albumin, α and β globulin and fibrinogen(1) and that part of the β globulin and all of the γ globulin is formed by basophilic cells (plasma cells) in lymphoid tissue and bone marrow(2,3,4). However, the cells primarily responsible for the catabolism of plasma proteins have not been identified. Cells of the reticulo endothelial system, particularly those localized in liver and spleen, are specialized to phagocytize different substances like carbon particles(5,6), bacteria, saccharated iron oxide(6), and cholesterol(7). Previous experiments have shown that heat denatured serum proteins(8), or proteins denatured by other methods, are taken up by the reticulo endothelial cells. It was demonstrated that such proteins can compete with clearance of carbon particles from blood by these cells(9). Possibly cells of the reticulo endothelial system also have a function in catabolism of normal, circulating serum proteins. Therefore, it was decided to study whether an impairment of the clearing function of the reticulo endothelial system by blockade would affect the turnover rate of S^{35} labelled homologous plasma proteins.

Materials and methods. Mice were used because turnover rate of their serum proteins is so rapid that, in a comparatively short time, these experiments could be carried out through several serum protein half lifes. The use of internally S^{35} labelled proteins seemed advisable, since externally labelled iodinated proteins may contain fractions that are slightly denatured and, therefore, handled differently by the animals. *R.E.S. blockade.* During experiments, blockade of R.E.S. was maintained by intravenous injections of 1 ml/100 g of a 25% solution of thorium oxide (thorotrast*) every 48 hours. In the first part of the experiments, effectiveness of such a blockade was investigated in about 20 animals. Carbon clearance rates were determined in groups of mice every 24 hours during 5 days of the experiment. Extent of blockade was investigated by measuring rate of clearance from blood of a colloidal carbon suspension (C11-1431 a†, particle size 250 Å), prepared as described previously(5). In these mice a dose of 16 mg carbon/100 g of body weight was used. Blood samples of 0.025 ml were taken from the retro-orbital venous plexus at various time intervals after intravenous injection of carbon. Carbon concentration in the samples was measured spectrophotometrically at 675 mμ wave length. It has been shown that rate of phagocytosis of a carbon suspension as related to time follows an exponential equation $K = \frac{C_o - C_t}{t}$, where K is a measure of phagocytic activity for the injected dose. Clearance curves were drawn for each animal and values for K calculated according to this equation. Mean values for K in control animals and in blocked animals at specific times are recorded in Fig. 1. Values of K in blocked animals are much smaller than those observed in control mice. This shows that an effective blockade of the R.E.S. has been successfully maintained during the experiment, when turn-

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