8070 P

Creatine and Glycogen Content of Normal and Infarcted Heart Muscle of the Dog.

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In our studies of the effects of perfusing the rabbit hearts with Ringer-Locke solution to which various amino-acids were added,[1] we were struck by the fact that infarction of the myocardium caused a sharp drop in the pH of the perfusate. This was apparently due to the liberation of a non-volatile acid metabolite giving the qualitative test for lactic acid. The inevitable result of the drop in pH was earlier failure, and, furthermore, low heart muscle creatine values. These findings seemed to be a corollary of our previous observation[2] that myocardial infarction in the dog leads to a creatinuria.

We therefore decided to undertake chemical studies of the infarcted as compared with the normal heart muscle of the dog. In addition to the creatine content we thought it worth while to determine the glycogen content, since Riesser and Brentano, we found, had demonstrated a close relationship between creatine mobilization and glycogenolysis.[3] Himwich and Goldfarb have demonstrated a decrease in glycogen content and an increase in lactic acid in infarcted heart muscle coincident with a rise in blood lactic acid.[4]

Dogs were anesthetized with barbiturates intraperitoneally and the chest opened under artificial pressure respiration. A nick was made in the pericardium over the first part of the main anterior descending branch of the left coronary artery. The artery was isolated and tied off, and the pericardium and chest closed. The heart was then allowed to continue beating for periods varying from 15 minutes to 12 hours. The infarcted beating heart was removed and sections of infarcted and normal muscle were minced in cold alcohol for glycogen determinations; similar portions were removed for creatine and total solid determinations.

The infarcted muscle showed an immediate drop in glycogen

[1] Decherd, G., Herrmann, G., and Davis, O., PROC. SOC. EXP. BIOL. AND MED., 1935, **32**, 1302.

[2] Herrmann, G., and Decherd, G., PROC. SOC. EXP. BIOL. AND MED., 1934, **32**, 478.

[3] Riesser, O., and Brentano, C., *Arch. f. exp. path. u. pharm.*, 1930, **155**, 1.

[4] Himwich, H. E., and Goldfarb, W., *Am. J. Physiol.*, 1934, **109**, 403.

TABLE I.
Comparison of Normal and Infarcted Heart Muscle.

Duration Min.	Loss in Infarcted Muscle % Creatine	Glycogen	Gain in Water Content in Infarcted Muscle
18	0	56	0.0
20	4	22	0.66
30	2	21	1.60
120	5	52	2.10
200	2	25	0.54
315	38	87	3.98
340	43	76	3.08
420	32	65	2.55
450	20	19	4.14
690	52	70	3.38
720	40	83	2.70

content as compared with the normal muscle (Table I). The drop seemed to have reached its maximum in 5 hours. Edema detectable by a drop in total solids apparently began to appear in the infarcted anoxic muscle within a half hour and likewise reached its maximum in 5 hours. The creatine loss was slight up to 5 hours, after which diffusion from the damaged muscle was considerable, and increased slowly.

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Reactions of Ant. Pituitaries of Male Rats to Administration of Ant. Pituitary-Like Substance and to Oestrin.*

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Recent studies[1] indicated that injections of massive amounts of oestrin into normal female rats induced weight and morphologic reactions in the anterior pituitary similar to those obtained by the injection of the anterior pituitary-like substance of pregnancy urine. Both of these substances induced a marked weight increase in the gland, a marked loss of granules from the basophiles and a less evident loss of granules from the eosinophiles (in A.P.L. rats whose ovaries contained active corpora lutea). Furthermore, it has been found that administration of the A. P. L. factor has no action on

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[1] Wolfe, J. M., PROC. SOC. EXP. BIOL. AND MED., 1935, **32**, 1192.