6337

Experimental Infarction of the Interventricular Septum of the Heart in the Dog.

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Infarction of the interventricular septum of the dog's heart by ligation of the front septal branch of the left coronary artery was successfully produced in 4 of 28 animals. The changes involved from one to two-thirds of the surface of the left side of the septum, and about one-tenth of the right. The infarct was widest about 1 cm. below the attached margin of the aortic cusps, its edges jagged, though well defined, and tapering in the direction of the apex. In one heart, 10 days after operation, the course of the left branch of the bundle of His was plainly visible just beneath the endocardium and over the infarcted muscle. Roughening or other alteration of the lining of the cavities of the heart was not observed.

Microscopically the tissues immediately beneath the endocardium, including the Purkinje fibers of the bundle, were unaltered, but the subjacent muscle had undergone coagulation necrosis and was in varying stages of organization, according to the age of the lesion. Approximately one-tenth of the A-V node was involved in the infarction, regions of varying size disseminated irregularly throughout the node.

There was no auriculo-ventricular dissociation demonstrated in post-operative electrocardiograms, and no significant irregularity in rhythm noted by other methods. The earliest after operation that an initial post-operative electrocardiographic tracing was made was 2 days, the latest, 10 days; a transient effect on the rhythm may have escaped recognition.

It seems probable that obstruction of the septal branch of the left coronary artery in the dog does not produce permanent dysfunction of the A-V node or the His bundle. The endocardium and tissues immediately beneath it, including part of the conduction system, may have received nourishment by seepage of blood in the same direct way that the inner lining of some of the arteries is nourished. It is possible that if the animals were kept alive over a period of several months, scar formation and contraction would damage the conduction system sufficiently to produce lasting heart block.

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A noteworthy observation was the absence of any thrombosis of the lining of the cavities of the hearts of these dogs, a state of affairs in marked contrast with that in the human heart where mural thrombosis is a frequent sequence of interference with the coronary circulation.

Further similar experiments are in progress.

6338

Effects of Pituitary Antidiuresis on Non-Cardiac Edema.

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From a previous study, describing the induction of pituitary antidiuresis in non-edematous subjects,[1] we were impressed by the magnitude of the net loss in body weight at the end of the recovery period. The obvious fact, that the reduction in weight which follows withdrawal of the antidiuretic agent is due under these conditions to loss of body water with its accompanying mineral elements, suggested the desirability of determining the effects of the procedure on the water and mineral exchanges in the presence of edema.

This has been done in 2 cases of typical lipoid nephrosis, one of chronic glomerulonephritis with superimposed nephrosis and one of edema of undetermined etiology. The total serum proteins were low in these subjects at the time of the tests. The case of edema of undetermined cause differed from the other 3 in showing no proteinuria and no increase in the blood lipoids. Throughout the entire period of observation all 4 patients were maintained in the metabolism ward under practically uniform conditions as regards room temperature and humidity, diet and routine for obtaining the body weight and for collecting samples of the excreta. The total water exchange was determined according to the method outlined by Newburg and Johnston.[2] After a preliminary control period of several days on a standard diet relatively low in NaCl, the patient was given a medium-sized dose of the antidiuretic pituitary extract (pitressin P. D.) subcutaneously at 3-hourly intervals for one or 2 days, the exact body weight being measured every 6 hours.

[1] McQuarrie, Irvine and Peeler, D. B., *J. Clin. Invest.*, 1931, **10**, 915.

[2] Newburg, L. H., and Johnston, M. H., *J. Clin. Invest.*, 1930, **8**, 161.