

Effect of Coronary Artery Occlusion on Dog's Heart with Total Coronary Sinus Ligation.*

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One of us (L.G.)¹ described 3 vascular mechanisms in the blood supply to the human heart which probably serve as compensatory means to ward off the results of coronary artery narrowing or occlusion. The first and most important of these is a gradual and consistent widening of anastomotic channels which occurs with increasing age. It was shown that many of these anastomotic channels are situated in the interventricular septum. The second compensatory mechanism is the age period development of rami telae adiposae, vessels which lie in the epicardial mantle and anastomose with the myocardial vascular ramifications on the one hand, and with periaortic and peripulmonic vessels on the other. The third mechanism is the existence of anastomoses between the myocardial and pericardial vascular ramifications and the extracardiac vessels (bronchial arteries, arteriae mammae internae and diaphragmatic vessels). These studies were subsequently confirmed and extended by Campbell,² Davis,³ Robertson,⁴ Hudson, Moritz and Wearn,⁵ and by Gross and Kugel.⁶

Recently, augmentation of these extracardiac anastomoses was attempted by Moritz, Hudson and Orgain⁷ by means of epicardiectomy (stripping of epicardium) with resulting pericardial adhesions. Using this procedure, Beck, Tichy and Moritz⁸ observed that some dogs may recover after gradual coronary artery occlusion covering a period of 2 years and that no infarctions were produced in the hearts. Robertson⁹ produced pericardial adhesions by tying off

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¹ Gross, Louis, *The Blood Supply to the Heart*, New York, 1921, Paul B. Hoeber.

² Campbell, J. S., *Quart. J. Med.*, 1929, **22**, 247.

³ Davis, D. J., *Arch. Path. and Lab. Med.*, 1927, **4**, 937.

⁴ Robertson, H. F., *Am. J. Path.*, 1930, **6**, 209.

⁵ Hudson, C. L., Moritz, A. R., and Wearn, J. T., *J. Exp. Med.*, 1932, **56**, 919.

⁶ Gross, Louis, and Kugel, M. A., *Am. Heart J.*, 1933, **9**, 165.

⁷ Moritz, A. R., Hudson, C. L., and Orgain, E. S., *J. Exp. Med.*, 1932, **56**, 927.

⁸ Beck, C. S., Tichy, V. L., and Moritz, A. R., *PROC. SOC. EXP. BIOL. AND MED.*, 1935, **32**, 759.

⁹ Robertson, H. F., *Arch. Path.*, 1934, **18**, 575.

coronary veins in stages. In many cases such dogs tolerated coronary occlusion, also produced in stages. If the adhesions were separated, however, the dogs died with fresh infarcts. Robertson believed that the progressive adhesions supplied nutrition to the heart.

Apart from the fact that these techniques involve extensive operative procedures, the question arises as to whether the results obtained were due to the added vascular supply by way of the pericardium or to the compensatory vascular dilatation within the myocardium made possible by the slow occlusion of the coronary arteries.

The experiments here described indicate a method which apparently produces a striking increase in the blood supply to the heart by means of a simple operative procedure. For certain theoretical reasons it was believed that tying off the coronary sinus would hasten the physiological age period widening of anastomotic channels in the myocardium such as normally occurs in the human heart. With this in view, the following experiments were undertaken.

Under Pernoston or Nembutal anesthesia, the left anterior descending coronary branch was divided between ligatures in a number of dogs approximately 2 cm. below the aortic ostium of the left coronary artery. Of 20 dogs which survived this procedure for longer than one hour, 19 presented a large vascular filling defect (incomplete filling of the coronary tree) as determined by subsequent injection of the hearts with a barium sulphate suspension in gelatine followed by roentgenography.¹ In 16 of these dogs which were sacrificed at intervals varying from 18 hours to 12 weeks, an infarct, usually measuring 5x5.5 cm., was observed on the surface of the left ventricle. This infarct generally involved a large part of the interventricular septum. One dog showed a small infarct measuring 1x1 cm. The 3 remaining dogs died or were killed within a few hours after coronary ligation, a period of time too short to produce visible infarction. Apparently, therefore, this procedure produces infarction practically invariably in the dog's heart. This becomes grossly visible 18 hours after the occlusion.

In another group of 41 dogs coronary sinus ligation was performed. This was followed almost immediately by considerable dilatation and widening of existing vascular channels on the surface of the heart. A number of these animals were sacrificed for anatomical studies. Barium sulphate gelatine injection of the coronary tree in these hearts showed a conspicuous increase in the extent of the vascular bed. In 10 surviving animals the left anterior de-

scending coronary branch was subsequently occluded in the manner described above. Eight of these animals died or were sacrificed in from 18 hours to 4 weeks after the arterial occlusion. The remaining 2 dogs died in less than 18 hours. These showed neither vascular filling defect (as determined by the injection technique) nor visible infarction. Of the 8 animals which died or were sacrificed 18 hours to 4 weeks after the arterial occlusion, 7 showed no vascular filling defect and 5 showed no infarction. The eighth animal showed a small vascular filling defect and infarction; another of the 8 animals showed a small infarct and one showed a myocardial hemorrhage. Apparently, therefore, in the majority of instances preliminary coronary sinus ligation prevents the appearance of infarction following acute occlusion of the left anterior descending coronary branch, and materially reduces the size of the infarct in others.

The operative mortality (within 48 hours after coronary sinus ligation) was 44% during our earlier experiences. This dropped to 20% in the last 10 dogs. Subsequent reports will deal with the technique employed and with certain characteristic electrocardiographic and other changes which follow these operative procedures.

It is hoped to apply this procedure to the human heart in order to increase the vascular tree.

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Birth Weight Criterion of Dwarfism in the Rabbit.

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The procedure practiced in this laboratory with reference to birth weight determinations is as follows: Each morning rabbits born during the previous 24-hour period are identified by color markings or toe amputations and weighed to the nearest gram on a balance calibrated in one gram intervals. The average elapsed time from birth to weighing is 12 hours, the mid-point of the 24-hour interval between determinations. It is apparent that this routine introduces an error which is dependent on the amount of nourishment which each individual in the litter has obtained between birth and the determination, since the female rabbit suckles her young immediately following parturition. This error will vary with the