A METHOD FOR CAUSING A CHRONIC MYOCARDIAL INFARCT

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The most reliable method of causing a myocardial infarct experimentally is to ligate a coronary artery, which causes necrosis of the myocardium of controllable size and location. However, the production of an infarct in an acute experiment does not enable the various functional changes related directly to the necrosis of the heart muscle to be studied, because opening the chest is itself a cause of severe disturbance, and the inflammatory and regenerative changes around the suture of the wound occur at the same time as the necrotic processes and the subsequent scar formation in the heart.

Many authors who have been concerned with the pain mechanism in stenocardia have caused a temporary disturbance of the circulation in the coronary artery. Sutton and Lueth [4, 5] passed a long ligature beneath the descending branch of the left coronary artery and then drew it through a glass tube which they left buried in the thoracic cavity; they brought the ends of the ligature to the outside. E. S. Shakhvazyan [3] passed the ligature beneath the coronary artery and brought it to the outside without a tube, having first resected two ribs.

These workers made no attempt to establish a chronic myocardial infarct. Their methods cannot be used to induce a necrosis in the myocardium long after the operation in which the ligature is applied, because the knot of the ligature cannot be fixed.

The ligature was drawn tight, usually not less than eight hours after the operation. Sutton and Lueth introduced a glass tube into the thoracic cavity, but because it was only very feebly attached to the pericardium it invariably caused damage to the heart, both on account of cardiac contractions and of movements of the thorax. The considerable displacement of the heart and large vessels which occurred when the ligature was tightened was also harmful. E. S. Shakhvazyan did not pass the ligature through a tube, which made it difficult or even impossible to tighten it long after the operation, because of the tissues which had grown on it. Also, resection of the ribs caused a disturbance of normal respiration.

In the experiments with thermoelectrodes implanted into the coronary arteries [1], permanent ligature of these arteries was effected by means of a long thread bearing a loop. The loops could not be tightened completely without considerable displacement of the heart.

We have found no published description for producing a chronic myocardial infarct, i.e., a necrosis of the myocardium of predetermined position caused by the primary impairment of the patency of the coronary artery at a particular point.* To study the function of the adrenal cortex during the development of myocardial necrosis, we have developed a method of producing a chronic myocardial infarct experimentally. The ligature was tightened permanently 1-3 weeks after the operation when the animal's condition had returned almost to the preoperative state.

Under intratracheal ether-oxygen anesthesia an incision was made into the fourth left intercostal space. The pericardium was divided in a line parallel to the phrenic nerve for a distance of 2-3 cm. Beneath the descending branch of the left coronary artery and at the level of the left auricular appendage a long ligature was tied with capron. Both ends of the latter were brought into the central opening of a plastic plate (Fig. 1); the threads then passed up the vertical arm of the tube and out through a hole in it. The pericardium was pulled up by two silk

* We are here not concerned with methods of producing necroses by means of primary biochemical damage to the cardiac muscle (use of electrolytes, hormones, coagulation, etc.).

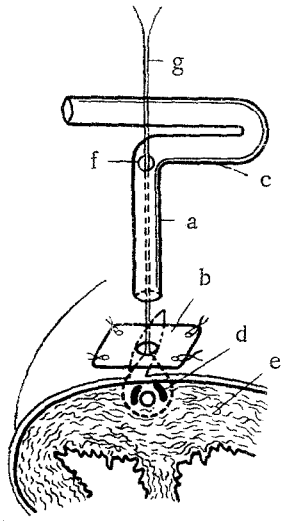

Fig. 1. Diagram showing the device for placing a permanent ligature on the coronary arteries. a) Plastic tube bent into a T-shape; b) plastic plate with central hole for ligature; c) wire from a metal paper clip used to bend the tube into a T-shape; d) coronary arteries; e) heart muscle; f) output aperture of the vertical limb of the tube; g) capron ligature.

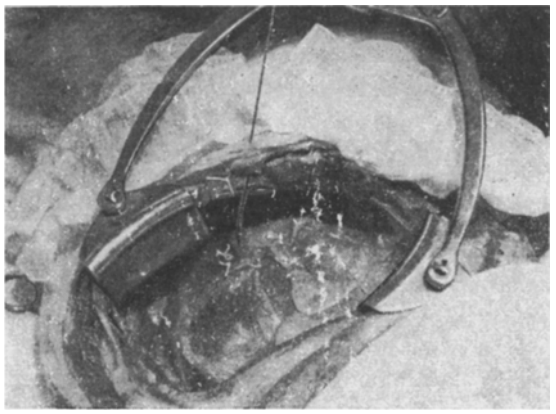

Fig. 2. T-shaped tube fixed to a rib. A ligature is passed beneath the tube and then through the muscle and skin to the outside.

sutures. Before the end of the tube was pulled tight it was kept about 0.7 cm from the heart so as not to interfere with contractions; in order to prevent any possible damage to the heart by the vertical limb of the tube, over the cut in the pericardium was sewn a plastic plate measuring 1 × 1.5 cm. The plastic tube 3-4 mm in diameter (the one we used was made of polythene) was bent into the shape of a T by means of the wire of an ordinary paper clip. The ligature passed through the vertical limb, and the horizontal limb was used to fasten the whole tube between the two ribs. Before the thorax was sewn up, the horizontal limb was sewn with two stitches to one of the ribs (Fig. 2), the ribs were then released from the force holding them apart, the muscles were sewn up in layers, and the skin sutured. The ends of the ligature which were drawn through the opening in the tube were then brought out through the muscle and skin to the outside. These ends were then covered with gauze and held on the surface with a bandage. The bandage was soaked in collodion and when it had dried it formed a compact mass which prevented damage to the ligature.

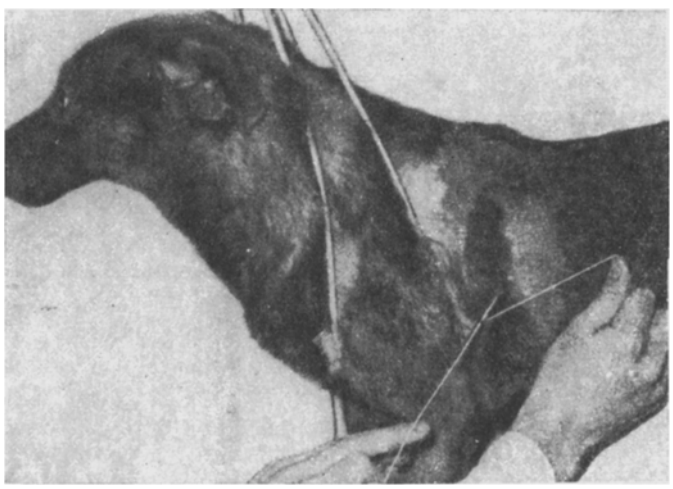

Fig. 3. The ends of the ligature are pulled tight over a piece of tube. The ligature remains permanently tight.

The ligature was pulled tight 7-22 days after the operation. The dog was placed on a stand, the bandage removed, and both ends of the ligature freed. While the electrocardiogram was recorded, maximum tension was applied to both ends of the ligature; then a piece of tube was placed between the ends, which were tied over it; the ligature was then left under permanent tension (Fig. 3). The electrocardiographic changes and the subsequent anatomical investigation of pathological changes confirmed that in all the animals a myocardial infarct had developed. Control experiments on three dogs showed that when the ligature was introduced beneath the artery but not subsequently tightened no necrosis of the myocardium developed.

The method we have described has enabled us in every case to obtain a myocardial infarct of determinate size and location. It was used to produce a model of artificial stenocardia, because when the ligature was slackened, it returned to its original position. This method is comparatively simple, and is entirely satisfactory experimentally in producing a model of a myocardial infarct or stenocardia in a prolonged experiment.

SUMMARY

A new method of inducing a chronic myocardial infarct is described. A T-shaped plastic tube is inserted into the thoracic cavity and a ligature introduced under the left coronary artery; it is brought to the outside, and then, at some long time after the operation it is tightened, and produces myocardial infarction at the moment when the animal's state of health has practically recovered to the preoperative condition.

LITERATURE CITED

1. G. N. Aronova and T. A. Maeva, in the book: Problems of the Physiology and Pathology of the Coronary Circulation [in Russian] (Kiev, 1960), p. 22.
2. S. A. Vinogradov, Arkh. pat., Vol. 1 (1955), p. 76.
3. E. S. Shakhvazyan, Experimental Contributions to the Problem of Disturbance of the Coronary Circulation in the Heart. Dissertation for Doctorate. (Moscow, Leningrad, 1940), p. 7.
4. D. C. Sutton and W. W. King, Proc. Soc. exp. Biol., Vol. 25 (New York, 1928), p. 842.
5. D. C. Sutton and H. C. Lueth, Arch. intern. Med., Vol. 45 (1930), p. 827.