DEVELOPMENT OF AN ANASTOMOSIS BETWEEN THE CORONARY VESSELS AND A TRANSPLANTED INTERNAL MAMMARY ARTERY*

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THE human coronary circulation has engaged the attention of investigators for a number of years and many attempts have been made to augment it. In 1935 Beck[1] attempted to improve the circulation of the left ventricle by the suturing of a muscle graft to the surface of the heart. In 1937 O'Shaughnessy[2] tried to bring arterial blood to the left ventricle by the suturing of the omentum to the ventricular muscle. More recently, Fauteux[3] in 1940 introduced ligation of the vena magna cordis for this purpose. In 1946, one of us[4] described an anastomosis between the left internal mammary artery and the left coronary circulation. This occurred in a dog 99 days after transplantation of the left internal mammary artery into the wall of the left ventricle. Since publishing the last report on this subject we have continued these experiments. The basic objective is to supply a fresh source of arterial blood to the heart muscles. In order to do this the left internal mammary artery has been partially removed from its normal position on the chest wall and implanted into the myocardium of the left ventricle. In this way it was thought that a communication might be developed between the implanted internal mammary artery and the left coronary circulation. Three technical variations of this procedure have been employed which have been designated as Experiments A, B and C. respectively.

Experiment A. — The internal mammary artery was tied at its distal end between two ligatures and cut. The free end was drawn through a previously prepared tunnel in the wall of the left ventricle and fixed in this position.

Experiment B. — The internal mammary artery was not cut but freed from its bed in its central portion. A slit was made in the wall of the left ventricle and the artery slung into the wound where it was held by a free pleural graft, or by sutures.

Experiment C.—The vena magna cordis and the anterior descending branch of the left coronary artery were tied. The internal mammary artery was sutured into the wall of the ventricle as in Experiment B.

RESULTS (SEE TABLE I.)

The results obtained in Experiments B and C were uniformly poor and will not be discussed here. This report will be confined to a description of the results obtained in 10 dogs treated as described in Experiment A. These animals were killed at the end of three or four months. In addition there are 17 animals which had the internal mammary artery transplanted into the left ventricular wall according to the technique described in Experiment A, which are still alive and many of these were operated upon more than a year ago. It is too early in this work to discuss morbidity or mortality statistics.

(*a*) *Anastomosis between the transplanted left internal mammary artery and the left coronary vessels.*—Of the 10 dogs killed, 2 or 20% showed a definite communication between the left internal mammary artery and the left coronary circulation. The first of these dogs, 8A, was killed 99 days after operation. The heart was removed from the thorax along with the attached left internal mammary artery. This artery was injected with pink Schlesinger's solution and the entire left coronary tree was filled (see Figs. 1a and 1b). Note that only the vessels of the left ventricle were injected and that no injection fluid entered the right coronary tree. The injection fluid flowed out of the coronary orifices into the aorta and then into the ventricular lumen. In order to obtain an x-ray of the injected vessels it was necessary to tie the left coronary orifice and then introduce the injection fluid. By this means, x-ray pictures were taken which showed clear outlines of the coronary vessels in the left ventricle (see Fig. 3).

Sections were taken through the site of implantation of the left internal mammary artery into the wall of the left ventricle. They showed three definite openings in the wall of the implanted internal mammary artery; these appeared to be vessels and were traced from the internal mammary artery out into the myo-

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TABLE I.

Dog lab. No.	*Duration of implant*	*Cause of death*	*Injection medium*	*Condition of internal mammary artery*	*Structures vascularized by transplant of internal mammary artery*	*Evidence of an anastomosis between the transplanted internal mammary artery and the left coronary circulation*
8A	99 days	Killed	Schlesinger's solution. Pink	Patent throughout	Left coronary artery	1. Injected medium filled the left coronary vessels and flowed into the aorta (Fig. 1) 2. X-ray evidence of filled coronary tree (Fig. 2) 3. Serial sections taken through the area of transplantation showed definite anastomotic channels (Fig. 3, a, b, c)
47	89 days	Killed	Schlesinger's solution. Blue	Patent throughout	Left coronary artery. Intercostals 3, 4, 5. Pericardial vessels	1. Injected medium filled the left coronary circulation (Fig. 4, a, b) 2. X-ray showed left ventricle filled with a radio-opaque mass due to failure of tying off the left coronary orifice 3. Section.
3A	46 days	Killed	Barium sulphate	Patent throughout	Intercostal vessels. Left coronary artery?	1. Pulsations transmitted to the area anastomosis through the injected medium. 2. Presence of injected medium in ventricular lumen demonstrated by x-ray.
31	132 days	Killed	Gross solution	Patent to within ¾" of entrance into heart	Intercostal vessels 3 to 5 inclusive	1. Gross solution present in intercostal vessels which had previously been detached from the internal mammary artery
34	110 days	Killed	Gross solution	Patent down to area of scar and beyond	Intercostal vessels 3 and 4	1. Intracardiac segment of the transplanted artery showed three visibly patent lumina
35	86 days	Killed	Gross solution	Patent although it had pulled away from the heart	Intercostal vessels and pericardial vessels	1. Visible evidence of an injected chest wall and pericardium. 2. X-ray visualization of pericardial vessels.
39	92 days	Killed	Gross solution	Patent to scar and beyond scar to heart. Narrow lumen within the heart	Intercostal 4 and 5	1. Visible evidence of injection medium in newly formed intercostal vessels.
6A	89 days	Killed	Schlesinger's solution	Patent throughout		1. No proved anastomosis.
41	89 days	Killed	Gross solution	Artery patent to kink 1" prox. to heart. Thrombosed beyond this point	Intercostal vessels 3 and 4	1. Visible evidence of injection medium in newly formed intercostal vessels.
43	93 days	Killed	Gross solution	Artery kinked ½" prox. to heart. Patent to this point. Partially revascularized thrombus beyond this site.	Not checked	1. No proved anastomosis.

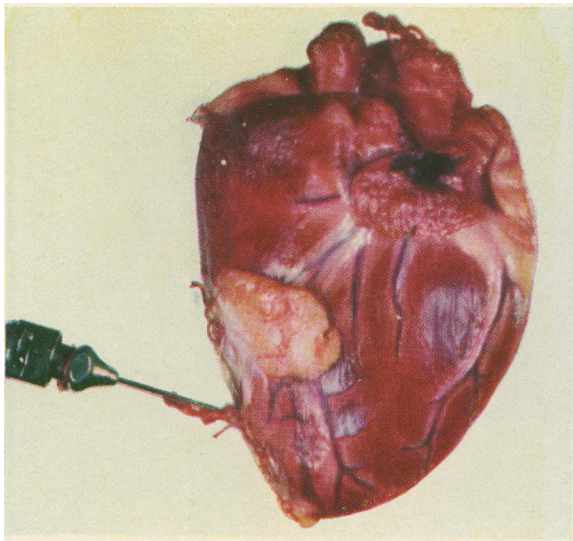

Fig. 1a

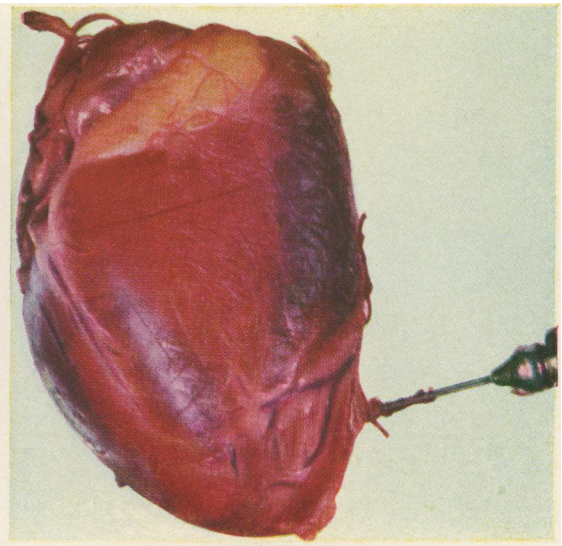

Fig. 1b

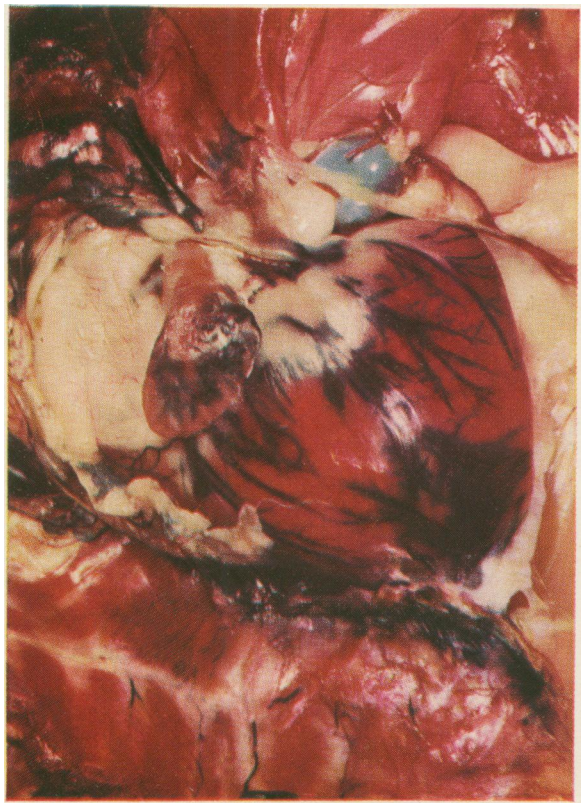

Fig. 2a

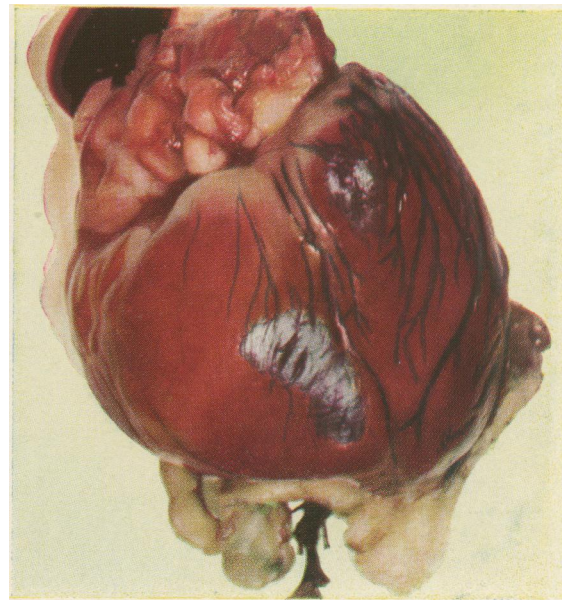

Fig. 2b

Figs. 1a and 1b.—Show the heart of a dog killed 99 days after the left internal mammary artery had been implanted in the wall of the left ventricle. The internal mammary artery has been injected with pink Schlesinger's solution. Note that the vessels of the left ventricle only have been filled by the injection fluid.

Fig. 2a.—Shows a cannula in the left internal mammary artery 89 days after the artery had been implanted in the wall of the left ventricle. Note that the injection fluid has flowed down the artery into the left coronary circulation and out into the pulmonary vein. It has also flowed into the chest wall.

Fig. 2b.—Shows dog 47 with the heart removed from the thorax. It demonstrates that only the left coronary vessels were injected, to the complete exclusion of the vessels of the right ventricle.

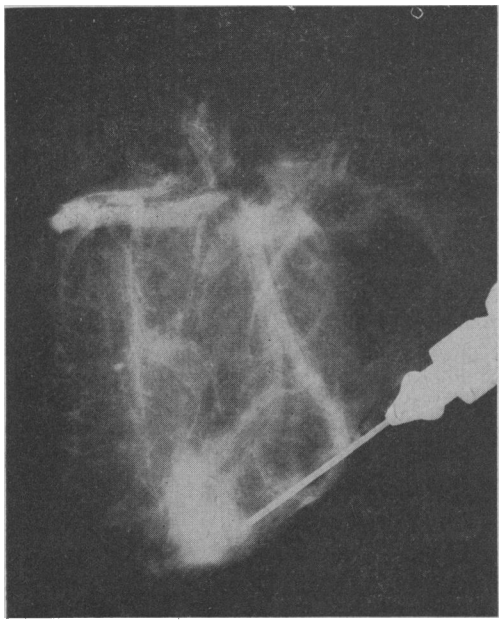

Fig. 3.—Shows x-ray of the left coronary tree which has been filled by injecting the left mammary artery 99 days after it was implanted into the left ventricular wall.

cardium (see Figs. 4a, 4b and 4c). Note that the internal mammary artery at the end of 99 days is lying in the myocardium of the left ventricle and has a completely patent lumen. The only change observed in the arterial wall is that of minimal intimal thickening.

The second dog, 47, was killed at the end of 89 days. The heart was left *in situ* and a cannula inserted into the left internal mammary artery well distal to the point at which the artery had been previously freed. The vessel was injected with blue Schlesinger's solution at a pressure of 120 mm. of mercury. The injection fluid flowed down the internal mammary artery and the left coronary vessels were seen to fill immediately as did the pulmonary vein. Simultaneously, intercostal vessels 4 and 5 on the left side were injected (see Fig. 2a). The heart and its attached internal mammary artery were then removed from the thorax and it was observed that only the left coronary vessels were injected, to the complete exclusion

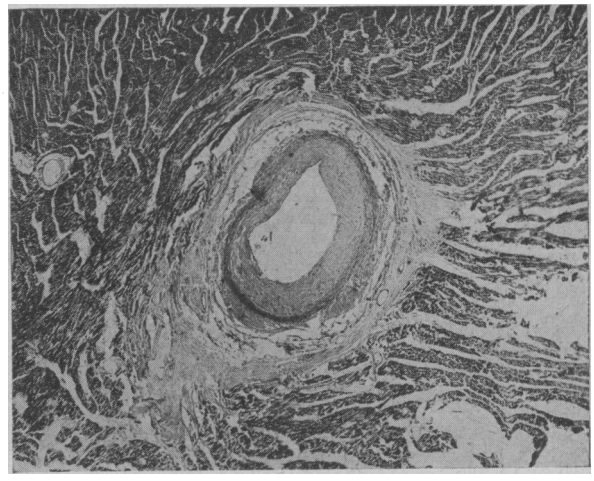

Fig. 4a

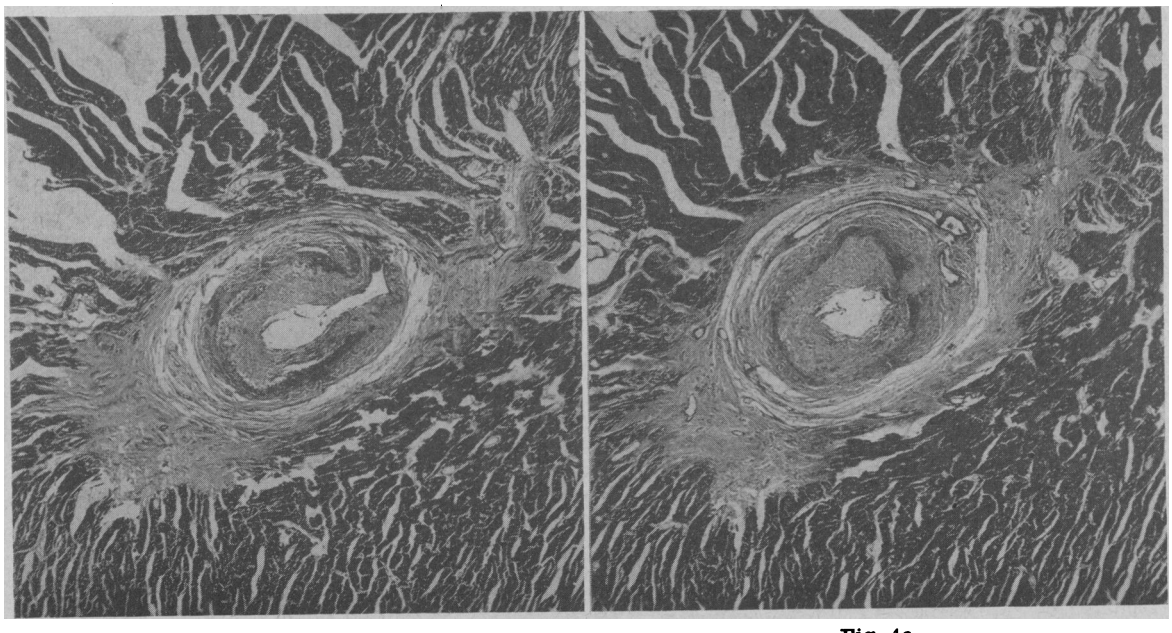

Fig. 4b **Fig. 4c**

Fig. 4.—Shows the transplanted internal mammary artery lying within the muscle of the myocardium at the end of 99 days. Note that the only change to be observed is a thickening of the intima. (a) It will be noted that in the upper portion of the section there is an indentation which is the beginning of a branch. (b) Shows the internal mammary artery with the branch well developed. (c) Shows a branch of the internal mammary artery lying along side of the parent vessel.

of those of the right ventricle (see Fig. 2b). Sections were taken through the site of implantation and showed a well-injected internal mammary artery—serial sections are now being made.

(*b*) *Patency of transplanted internal mammary artery* (see Table I).—In 8 of 10 animals the internal mammary artery became partially adherent to the chest wall in its former bed. Distal to this the artery was free for a distance of a half to one inch before entering the heart. A constricting scar developed at the junction between the free arterial portion and that part of the artery which re-adhered to the chest wall. The artery was patent throughout in 5 animals and was patent to the site of the scar and beyond it in 2 animals. In 3 dogs it was patent to the site of the scar but thrombosed beyond it (see Table II).

TABLE II.
PATENCY OF TRANSPLANTED INTERNAL MAMMARY ARTERY

No. of dogs	*Condition of transplanted internal mammary artery*
5	Patent throughout.
2	Patent to the site of the scar and beyond.
3	Patent to the site of the scar but thrombosed beyond.

(*c*) *Ability of transplanted internal mammary artery to form an anastomosis with vessels of surrounding structures.*—In 9 of 10 animals the internal mammary artery after transplantation revascularized the surrounding structures. In two instances there was an anastomosis with the left coronary vessel. In the 5 animals in whom a constricting scar formed, the artery proximal to this area developed communications with the chest wall and intercostal vessels of the left side. This occurred in spite of the artery having been, originally, completely disconnected from its habitual vascular bed. In 1 animal, where the artery had pulled out of the heart, a communication was developed between the artery and the vessels of the chest wall and the pericardium.

SUMMARY

A transplanted vessel, internal mammary, when placed in contact with another vascular bed will develop a communication with the vessels of that vascular bed, left coronary artery, pericardium and intercostal arteries.

CONCLUSION

The left internal mammary artery, after implantation into the left ventricular wall, formed a communication with the circulation of the left coronary vessels. The frequency of this would appear to depend upon the formation of scar tissue around the transplanted internal mammary artery. Work is now in progress to attempt to diminish the frequency of this scar formation.

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RÉSUMÉ

Les auteurs font un exposé de leur travail experimental en tentant de développer l'anastomose entre l'artère mammaire interne et les vaisseaux coronaires. Ce travail a été commencé l'an passé par le docteur Vineberg.

On a trouvé que l'artère mammaire interne ainsi implanté a formé une communication avec la circulation des vaisseaux coronaires gauches en différent degré. Un facteur de cela a été la formation d'un tissu cicatrisé autour de l'artère transplanté. Des essais sont faits à présent pour en diminuer la fréquence.

MALIGNANT LYMPHOMA: CUTANEOUS SYMPTOMATOLOGY*

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THE group of diseases formerly known as lymphoblastoma is now better known as malignant lymphoma. It is generally agreed that they spread by invasion or metastasis and that they terminate fatally. Their acceptance as neoplastic is gradually prevailing over the view long held in some quarters that they are of inflammatory origin. As yet however Warner,[1] an authoritative British pathologist, prominently supports the opinion that Hodgkin's disease is inflammatory in nature, stating that it stands in an intermediate position between the granulomas and the neoplasms.

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