1-1-1943

Collateral coronary circulation : with particular reference to surgical revascularization of the heart

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COLLATERAL CORONARY CIRCULATION WITH PARTICULAR REFERENCE TO SURGICAL REVASCULARIZATION OF THE HEART

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Senior Thesis presented to The College of Medicine, University of Nebraska, December 1943.

cardium has only two coronary arteries, and (2) that the heart has been deprived of continuity with the rest of the body - can be interpreted as two important defects in the structure of the human machine. So the most important muscular structure of man's body is perversely the most defenceless and most devoid of the chance to develop collateral circulation in obstruction of the normal blood supply.

Osler observed that coronary sclerosis is not a condition which affects the working classes to any great extent, and described it as a disease of the intelligentsia. Among the famous physicians who have been afflicted by it are Doctors John Hunter, Charcot, Nothnagel, and Pepper. At the Mayo Clinic, a statistical survey was made of diagnoses in over three hundred consecutive patients in each occupation, to determine the relative incidence of coronary sclerosis among farmers, laborers, clergymen, lawyers, bankers, and physicians. The criteria for diagnosis were the same in all cases, so the diagnostic error should be the same in the various groups. The findings of heir survey are herewith tabulated.

Occupation	No. of Records Studied	Incidence of Coronary Sclerosis Cases	Per Cent
Farmers	308	8	2.5
Laborers	306	8	2.6
Clergymen	306	14	4.6
Lawyers	304	14	4.6
Bankers	300	16	5.3
Physicians	307	33	10.7

It has log been recognized that stress, strain, intensity of work, and mental worries are important contributory factors in occurrence of coronary sclerosis. Surgeons have perhaps the greatest responsibility of all the physicians, and the strain which is more or less routine to them is probably comparable to the strain on a banker when there is a run on his bank.

Treatment of this condition in the past has been generally unsatisfactory. Of course the ideal solution to the problem is the prevention of atherosclerosis, but at present, practically no progress has been made in this direction. The usual medical management is directed at relief of the associated discomfort which is usually in the form of angina pectoris. This consists principally of general hygiene and the use of vasodilators in attacks of angina. It is also directed at prevention of progression of coronary sclerosis which would lead to occlusive incidents. Increased rest and a general slowing down of the tempo of the patient's existence accomplishes much in this direction, but is frequently not compatible with the patient's economic status nor with his personal wishes. Indeed these measures assume considerable therapeutic proportion by protecting the individual from occlusive incidents while intercoronary channels of significant size and number develop in the manner to be subsequently considered.

Surgical measures which have been used with varying degrees of success fall into three main classes. The first of these is surgical interruption of the nervous pathways along which pain producing impulses from the heart are conducted to the central nervous system. This is accomplished by alcohol injection of the upper four or five thoracic sympathetic ganglia, two cc. of alcohol to each, by resection of these ganglia, which is too formidable a procedure for the average patient with this disorder, or by resection of the stellate ganglia, or by section of the upper thoracic posterior spinal roots. These procedures are purely palliative. The second is total thyroidectomy, the rationale of which leaves something to be desired. The rationale is essentially decreasing the rate of metabolism which makes demands on the heart which are too great to be permitted by the limited coronary blood supply. The patient must have some thyroid hormone, but this can be administered oraly, a level being established which is just below that at which symptoms appear. So this is another means of slowing he patient down. The third of these consists of the sur ical attempt to promote anastomosis between branches of the two coronary arteries and the production of new pathways to bring blood to the myocardium. The last appears to be the most fertile field in which extended investigation is now proceeding. It seems to hold

some promise of being ultimately developed to a degree which will allow a patient with an otherwise crippling sclerotic lesion to lead a nearly normal existence.

Anatomic, Physiologic, and Pathologic Considerations

The heart muscle is supplied with blood from the two coronary arteries, right and left, which arise directly from the aorta close to its origin. The right coronary artery appears between the roots of the pulmonary artery and aorta and curving to the right in the auriculo-ventricular groove runs toward the apex in the inferior (posterior) interventricular sulcus. The part of the vessel in the auriculo-ventricular groove is sometimes referred to as the right circumflex artery and that portion in the interventricular sulcus as the posterior descending branch. The right coronary artery sends branches to the right auricle, to two thirds of the anterior surface of the right ventricle, and to the inferior or diaphragmatic aspects of both ventricles; a special branch goes in the majority of cases to the sino-auricular node, another to the auriculo-ventricular node and bundle. The left coronary artery, after a short forward course, divides into a circumflex and an anterior descending branch. The circumflex branch is distributed to the left auricle and to the left margin and inferior aspect of the left ventricle. The anterior descending branch runs toward the apex in the superior (anterior) interventricular sulcus and supplies the anterior wall of the left ventricle and the left third of the anterior wall of the right. The interventricular septum is sup-

plied by branches from both right and left coronaries. The sino-auricular node is supplied in a certain proportion of cases by the left coronary instead of the right. As a rule, the auriculo-ventricular node and bundle receive twigs from both arteries. Besides the main branches which have been described, there are a number of interesting and important branches which apparently occupy both anatomically and functionally a category of their own, because considered from both these viewpoints they seem to occupy a place halfway between that of the vasa vasoum and the cardiac coronary branches proper. These are the arterial telae adiposae or fat branches. They are seen in greatest number in fat found under the epicardium, namely in the grooves between the chambers where they occur as an irregular feltwork, and over the sites of the main coronary branches where they exist as delicate parallel accompanying vessels whose distance from the main branches varies directly with the amount of fat present. The rich inter-anastomosing network of delicate vessels, superimposed upon the roots of the pulmonary artery and aorta, falls under this category. These fat vessels arise largely from the first portions of the rightand lef circumflex arteries as well as from their branches soon after their origin.

A detailed description of the veins of the heart is not pertinent to this discussion except as concerns the Thebesian vessels. Except for the oblique vein of the

left auricle, ther are practically no veins of large caliber draining the auricles. This lack is largely made up for by the existence of tiny venous channels known as the Thebesian veins according to their discoverer, Thebesius, who described them in 1708. These are direct connections between the veins and the chambers of the heart, being seen in greatest numbers in the interauricular septum. In the left auricle they are not so numerous, but generally larger. In the ventricles, foramina Thebesii are most frequent at the bases of the papillary muscles, the region of the conus on the right side and the apical musculature.

Little was known about the ultimate disposition of blood to the myocardium until 1928 when Wearn made some interesting perfusion studies. In perfusion of the dead human heart with a suspension of Berlin blue, he found that 60 per cent of the perfusate was returned from the chambers of the heart directly, only 40 per cent coming out through the coronary sinus. On section of these hearts, he found only patchy areas in which the capillaries were injected. It was then found that human hearts obtained at autopsy within three or four hours of death, when perfused with warm oxygenated Locke-Rosenheim solution would soon start to beat regularly and with good force. Introduction of Berlin blue into the perfusion fluid soon stained these hearts a deep blue without stoping them. The hearts were stopped by introduction of

glacial acetic acid into the perfusate, sectioned, measured, fixed in formalin, and measured again to enable correction for shrinkage to be accomplished. Microscopic sections were made, and comparative counts of muscle fibers and capillaries were done. It was found that there is regularly one capillary vessel for each ventricular fiber, and one for every two auricular and Purkinje fibers. This ratio was not found to be altered in diseased hearts. During normal growth, it was found that muscle fibers enlarge and capillaries multiply, thus providing a relatively constant blood supply; but in cardiac hypertrophy, the fibers enlarge, but the capillaries increase neither in size nor number, so there is a mechanical impediment to the exchange of metabolic substances. This led Wearn (1939) to suggest that cardiac hypertrophy may be as much a handicap as a true compensatory mechanism.

The extracardiac anastomoses of the coronary arteries were described by Hudson et al (1932) as quite extensive and quite constant. Injection studies with an India ink suspension were done in 31 normal hearts and surrounding tissues. Some of these were made directly into the coronary arteries and others into the thoracic aorta which was tied off just distal to the coronary ostia. Plates 1 and 2 are reproductions of their findings in a typical study and show most of the constantly encountered anastomatic channels which include:

1. Coronary fat branches emerging around the root of the aorta and anastomosing with the aortic vasa-vasorum and the vessels of the periadventitial connective tissue from the aortic ring to the diaphragm.

2. Vessels emerging at the base of the pulmonary artery and closely adherent to it even well within the substance of the lung.

3. Many mall arteries leaving the heart around the root of the pulmonary veins in the pericardial reflection. These anastomose with pericardial, bronchial, and mediastinal arteries, and gave extensive mediastinal injection.

4. Larger and more numerous injected vessels leaving theheart around the ostia of the superior and inferior venae cavae than around the large arteries, being applied to the adventitia of the veins. Their course and number is not constant. The right auricular branches of the right coronary artery which arborize around the ostia of the venae cavae after supplying the auricle are notable, especially around the superior veno-cava.

5. A few small branches leaving through the intervascular reflections of the pericardium. These are inconstant, and no large vessels were observed. These (most of them) are continuous with the arteriae telae adiposae.

There was an extensive network of vessels injected over the entire parietal pericardium. The largest mediastinal arteries injected were the pericardiacophrenic branches of the internal mammary arteries. Small anterior branches of the aorta were also found to contribute many branches to the posterior and lateral surfaces of the parietal pericardium. The most extensive anastomoses were found to be around the veins entering the heart. In the mediastinum, the usual order of prominence of injected vessels was found to be:

1. Pericardiacophrenic arteries
2. Anterior mediastinal vessels
3. The Pencardial vessels
4. Bronchial vessels
5. Superior and inferior phrenic arteries
6. Intercostal and oesophageal branches of the aorta.

The coronary circulation undergoes a series of changes with age and development which are quite constant, and which have been described by Dr. Louis Gross and others following careful injection and dissection studies. The smaller figure in Plate 3 represents a roentgenogram of the circulation in an average heart at birth and shows that both sides of it are approximately equally supplied with blood, so that, if it were not for the characteristic right circumflex branch, it would be impossible to discern the left from the right side of the heart. Upon stereo-

scopic examination, this and the other hearts at birth failed to show macroscopic septal anastomosis. The branches show uniformity of lumen and are without tortuosity except in their extremities. No arterial telae adipose can be foundat birth or in the first decade. There is a beginning clearing of the right side during the first decad,but no septal anastomoses can be found. In the second decade, the distribution of blood becomes a little more marked on the left side, and a few delicate septal anastomoses begin to appear as do a few stray fat vessels parallel to the main coronary branches. The third decade of life shows a definite left sided preponderance. Septal anastomoses can be much more clearly made out in the clearedspecimen. The tortuosity of the vessels is quite discernible,and rami telae adiposae can be readily observed. The fourth decade presents these changes in definite progress; the septal anastomoses are clearly developed and the left side of the heart is in the ascendant. Tortuosity of the vessels becomes marked, and a striking network of elicate arteriae telae adiposae makes it appearance. In the cleared specimen, the latter fills the whole auriculo-ventricular groove, and appears as a greyish mass of vascular channels. In the fifth decade, the preponderance of left side over right is striking, as is also th tortuosity of the vessels. At this period of life the minbranches are occasionally seen projecting beyond

the mass of hart musculature into the fat. This is due to a beginning regression and atrophy of heart muscle, leaving the vessels relatively too long. The septal anastomoses are distinct and abundant, being arranged somewhat in the fashion of a row of harp strings. Fat vessels are well developed and numerous. The sixth decade of life shows an ever increasing left sided vascular predominance and tortuosity of the vessels. The septum shows a system of very patent and free arterial anastomoses. The larger figure in Plate 3 shows these features of the seventh decade of life; the increasing relative anemia of the right side, the obvious tortuosity of the vessels, and the septal anastomoses. A cleared specimen in this age group shows the well developed fat-vessel network.

Intercoronary anastomoses have been mentioned rather casually but the existence of these has been the subject of long and hot controversies in the past century.

Thebesius, in 1708, followed by Haller, Morgagni and Senac, on the basis of careful dissections, came to the conclusion that anastomoses exist between both coronary arteries. Haller stated that these were quite rich and occurred with frequency at the root of the pulmonary artery, in the posterior longitudinal sulcus, in the vntricles, at the apex, and through the vaso-vasorum of the great vessels.

In 1799 Parry and Jenner first interpreted the clinical sundrome known as angina pectoris as due to calcification of the coronary arteries and the autopsy on John Hunter's heart, after Jenner had diagnosed his condition, corroborated this view.

In 1810 Coldani's dissections revived the claim that anastomoses exist, particularly at the root of the pulmonary artery. Cruvelhier again described wide anastomoses between both coronary arteries as well as with bronchial arteries.

In 1855 Hyrtl, on the basis of injection and corrosion experiments denied the existence of anastomoses between the coronary arteries and this was confirmed in 1866 by Henle, who stated however, that capillary anastomoses do occur.

Krause was the first to oppose the views of Hyrtl, but meanwhile Beraud had found anastomoses exist between the coronary arteries and vessels from adjoining organs.

In 1880 Langer showed that anastomoses exist between the coronary arteries and those of the pericardium, and through these, with the internal mammary arteries. He showed further that connection occurr with the bronchial arteries via the vasa-vasarum of the pulmonary arteries, andwith the diaphragm through branches from the auricles.

In 1881 Cohnhein and A. von Schulthess-Rechberg repeated their experiments on the clamping of coronary

arteries in curarized dogs. Their conclusions which profoundly influenced the opinion of future observers and which are still being held by some, were that clamping of either main coronary artery caused the ventricles to stop in diastole within two minutes. They accordingly argued that the coronaries were end arteries, and that if any anastomoses exist, they must consist of five capillaries. These experiments were confirmed by subsequent investigations, and more injection experiments confirmed Hyrtl's work. These helped to lend considerable support tothe opposers of the view that anastomoses exist.

It was not long, however, before other observers, notbly McWilliam, Bickel, and Porter, after performing numerous careful ligation experiments in dogs, came to conclusions opposed to those of Cohnhein and von Schalthess-Rechberg. They held that many of the results of the previous investigators were due to the trauma of the operation, and tying off branches, and even a main coronary artery does not necessarily lead to instantaneous death. In 1892 Kolster gave an accurate description of the processes of infant formation and healing by scarring.

Moreover, this opposition gained further strength by collection of numerous clinical cases and pathological material by Dock, Osler, Herrick, and others. These showed that obliteration of coronary branches, and in ome cases of a main coronary artery produced results

which varied in different cases from almost instantaneous death to those which experienced no symptoms and showed no clinical sign, the condition being recognized only at autopsy

After these investigations, it could scarcely be denied that intercoronary anastomoses exist, and it remained to describe their location and appearance and to explain why, if anastomoses exist, infarcts occur.

In 1907 Spolteholtz employed a chrome yellow-gelatin suspension for injections with subsequent dehydration in benzal and carbon disulfide, and obtained a reconstruction of the cardia circulation vastly superior to anything previously obtained. His conclusions were:

(a) No end arteries exist in the heart

(b) Rich anastomoses occur in all layers of the heart, and through the vasa vasorum, on the great vessels

(c) In the thick muscle of the left ventricle, perpendicular vessels penetrate to anastomose under the endocardium

(d) The papillary muscles are particularly rich in anastomoses

(e) With growth, the appearance of vessels on the surface shows a typical alteration.

In ligation experiments on dogs, he further showed that the infarcted area was much smaller than the area supplied by the tied off vessel.

In 1909 Miller and Matthews proved that many of the fatal results obtained by Cohnhein and others were due to the use of curare and morphine. By using ether anesthetic and strophanthus as a heart tonic, they obtained a mortality ofonly 8.7 per cent following ligation of the left descending ramus. They were therefore of the opinion that considerable anastomoses must exist between the coronary arteries.

In Herrick's clinical classification of angina pectoris (1912), one group of cases concerns patients who survived a coronary occlusion for a time varying from days to weeks, all of which were in persons over fifty years of age. He was of the opinion that anastomoses exist and that the p@tency of these vessels played an important part in determining the degree of compensation which can take place after obliteration. He argued that gradual occlusion allows the heart to adapt itself to the new conditions and allows collaterals to develop sufficiently to compensate. He suggested that the Thebesianvessels might serve as accessory nutritive channels in such cases.

In 1918 Smith corroborated the work of Spaltsholtz and of Miller and Matthews by experimental ligations in dogs and by interpretations of human autopsy material.

It remained for Gross, in 1921, to describe the nature and architectural arrangement of these anastomoses.

Employing barium-sulfate gelatine suspensions for injection, he was able to demonstrate the anastomoses by means of X-ray plates and in cleared specimens after injection. On the surface of the heart (Plate 4, Figs. 1 and 2) a good injection shows very open anastomosis occurring between branches from the anterior descending branch of the left coronary artery and those of the anterior rami of the right. A similar, though usually less conspicuous anastomosis occurs on the corresponding posterior surface of the heart. The interauricular and particularly the interventricular septa are the seats of very extensice, and in later age groups, very wide anastomoses. So far as communication between smaller vessels of the heart is concerned, the auricular walls and appendages, as well as the ventricular walls throughout, are the seat of very abundant anastomoses and interanastomoses between branches from both coronary arteries as well as between branches from each coronary. Capillary anastomoses are very numerousand rich and can be seen in any portion of cardiac musculature. Further, the arteriae telae adiposae were traced into the auricular and ventricular musculature and there were found distinct anastomoses between these and branches from the coronary arteries and vasa vasorum. In summary of Gross' significant work, the following statements can be made: -

(a) Anastomoses exist between the right and left

coronary arteries both in capillary and in pre-capillary distribution

(b) Anastomoses exist between the branches of the same coronary artery

(c) Anastomoses exist between the coronary arteries and vessels from adjacent organs.

(d) Anastomoses in the heart are universal and abundant.

Since it has been shown that end-arteries in the anatomical sense of Cohnheim do not exist in the heart, the question which now arises is how to explain on this basis the formation of infarcts.

Pratt (1898) formulated the concept of functional end arteries which are vascular structures in which the resistance to flow in the anastomatic area is greater than the pressure in the different contributing vessels. Probably the coronary arterial system falls at least to a large extent into this category.

In 1937 Mautz and Beck made some significant measurements of the size of intercoronary anastomoses. They used a barium sulfate-gelatine suspension injected at a pressure of 200 mm. of mercury into the ramus descendens of the left coronary artery. "Six-foot X-ray plates" were made of the normal human hearts studied which were obtained at autopsy of relatively young persons who died from causes not related to the heart. Considerable variation in size and extent of the anastomoses was encount-

ered by these investigators measured 70 micra in diameter (Plate 5).

In 1941 Blumgart, Schlesinger and Zoll made similar studies employing a lead-agar injection mass and arrived at the conclusion that the anastomotic channels in the normal heart are less than 40 micra in diameter and can therefore be of no significance in the case of a sudden extensive occlusion of a coronary artery.

Also in 1941 Dock devised a method of determining the capacity of the coronary bed. He used a perfusion apparatus with which he could measure the rate of flow, regarding rate of flow as being a direct function of the capacity of the coronary bed. He used kerosene as his perfusion fluid since this would not pass into the tissue spaces and encroach upon the cross-sectional area of the vascular bed by compression. He found that in spite of intercoronary anastomoses, the capacity of the coronary bed is 35 per cent lower in the hearts of those 60 to 80 years old than it is in young adults. This capacity also decreases per gram of tissue in cardiac hypertrophy, so the previously described work of Wearn is corroborated by an entirely different approach.

In the next year, Prinzmetal and his associates utilized the kerosene perfusion apparatus of Dock in a very ingenious quantitative determination of collateral

coronary circulation due to intercoronary anastomoses. They used two complete perfusion devices, one for each coronary artery, simultaneously and separately. By means of some relatively simple calculations using the data thus obtained, they were able to arrive at the volume of flow through the collateral channels. Obviously these values would not be the same for kerosene as for blood with its formed elements, but the value obtained with kerosene should be a direct function of the collateral volume flow of blood. In 12 normal hearts, the average value of the collateral flow, that is the proportion of perfusion fluid which passed from one coronary artery to the other, was 4.16 per cent of the total flow. Further, ome hearts were injected with a radio-opaque material of viscosity similar to that of blood via one coronary artery and X-rayed; the whole arterial tree was found to be filled. Oxalated blood was then used, and similar results were obtained, so it was concluded that the anastomatic channels are large enough to be of use.

In addition to the usual vascular channels consisting of arteries, capillaries, and veins more or less as found in other organs, there is a series of other direct vasular channels between the coronary arteries and the chambersof the heart. Like in the case of intercoronary collateral channels, the existence of these has been variously claimed and denied.

The concept of direct vascular channels between the coronary vessels and the chambers of the heart is not new. It was first advanced in 1705 in a personal letter written by Raymond Vieussens to the personal physician of the Dauphin of France, which was published in 1706. He described a large "polyp" in the heart which appeared to take root in certain holes in the wall of the heart which seemed to him to be the orifices of specific ducts. He injected an alcoholic solution of saffron dye into the left coronary artery and observed its passage into the left auricle and ventricle, while none escaped via the veins into the right chambers. He also dissected human hearts and the hearts of sheep and calves, and, with the aidof a microscope, found the small openings in the chambers of all the hearts examined, some being covered by delicate valves.

These vessels, clearly described by Vieussens, have since been confused with similar ones described by Thebesius two years later in 1708, and as a result, all such vessels have become commonly referred to as thebesian veins. Thebesius injected the coronary veins and noted the escape of his injection fluid into the heart cavities through small openings in the endocardium. He was familiar with Vieussens' work and so states in his paper.

Thebesius' belief that the vessels were connected only with the veins was supported by several anatomists

of the day and Vieussens' work was soon forgotten. Lancisi (1740) injected mercury into the coronary arteries and observed its appearance in the chambers of the heart, but believed with Thebesius that it escaped through the venus channels(thebesian veins). In 1798 John Abernethy, by making a "common coarse waxen injection" into the coronary arteries observed that it flowed readily into the cavities of the heart. He also injected masses ofdifferent colors into the arteries and veins, and concluded that vessels from the arteries and veins communicated with the chambers because the injection mass was too viscous to pass through the capillary bed.and yet the different colored solutions passed into the cavities of the heart unmixed.

No more work was apparently done with these vessels until Pratt in 1898, by means of perfusion of defibrinated blood through the ventricle of a cats heart, kept it beating for an hour. This important work furnishes the first experimental evidence that the thebesian vessels might serve as an entrance for blood from the ventricles into the capillaries.

In 1928 Wearn perfused the coronary arteries with india ink and observed that the perfusate for the most part escaped into the chambers of the heart through the thebesian vessels. He regarded these as channels through which blood is drained rapidly from the myocardial

vessels as systole commences, so that the latter are practically empty during systole and fill during the sustaind aortic pressure during diastole. He cited two cases which came to autopsy, of syphilitic aortitis which completely occluded both coronary arteries at their source, in which the heart had obviously been functioning for quite some time subsequent to the occlusion. This was interpreted as meaning that the slow occlusive process had allowed the thebesian vessels to take over the total circulation to the heart. Thus Wearn proposed a normal function and a function as collateral circulatory channels in coronary artery disease.

Grant and Viko (1929) injected the thebesian vessels through their endocardial foramina and through the coronary vessels. They used finely drawn glass cannulae of such sizes as to fit snugly into the endocardial foramina, and an injection pressure of 50 to 60 mm. of mercury and cleared the specimens in methyl salicylate. They confirmed the existence of anastomoses between thebesian vessels and coronary veins and capillaries, and described channels uniting neighboring foramina. They also injected the coronary arteries but failed to find evidence of direct arterial communication and concluded that the coronary arteries communicate with the thebesian vessels only through capillaries.

Stella (1931) employed the denervated heart-lung

preparation in dogs in an attempt to test the claim of the existence of communications between the arteries and heart chambers by a physiologic method. He was able to maintain the pressure within the ventricles and drop the pressure within the coronary arteries, and since under such conditions be was unable to demonstrate backflow from the chambers into the arteries, he concluded that his work did not support the claim of the existence of large channels connecting the thebesian veins with the coronary arteries.

Grant in 1926 found in a child's heart, which presented a congenital anomaly, persistent channels communicating freely with the heart cavities and with the coronary veins, arteries, and capillaries. Bellet, Gouley, and McMillan in 1933 reported dilated sinusoids or thebesian veins in a heart which showed an advanced tuberculous fibrocaseous infiltration.

The lack of agreement concerning the vascular communications between the coronary tree and the heart chambers led Wearn, Mettier, Klumpp, and Zschiesche to make some conclusive studies which they reported in 1933. Human hearts obtained at autopsy showing no gross evidences of pathologic process were used in these experiments. They were placed in the refrigerator till rigor mortis passed off, following which they were massaged in warm saline to break the remaining rigor and remove all blood

clots in the chambers. The coronary sinus and the tricuspid valve were closed and each coronary artery and the pulmonary artery cannulated. A fluid mass of celloidin dissolved in acetone dyed red with alkalin was maintained in the ventricles at a pressure of 180 mm. Hg while suction was applied to the coronary arteries. Then a similar mass dyed blue with crystal violet and brilliant green was injected at the same pressure into the coronary arteries. After the celloidin had solidified the tissue was digested away in hydrochloric acid and the cast obtained was mounted. In all of the 15 hearts thus studied, red tipped terminations of the blue casts of the coronary arteries in the ventricular mass were noted. These were numerous, 8 to 10 being regularly seen in the ramus descendens of the left coronary artery. Plates 6 and 7 represent the findings in photographic form.

Similar procedures were done including the coronary veins via the coronary sinus, and numerous venous connections with the ventricles were noted. To prove that these observations were not the result of artifacts, gelatine injections were similarly made and sections were examined microscopically. The injection mass was found to be confined to the vessels.

To complete this study, wax plate reconstructions were mde from serial sections. Plate 7 Fig. 3 is a photograph of one of these wax plates reconstructions of a

section of the left ventricular wall near the apex. The region enclosed by the broken line indicates the area from which the reconstruction shown in Plate 8 Fig. 1 was made. Fig. 2 of Plate 8 shows a typical microscopic section as used in the preparation of this reconstruction.

Study of these reconstructions confirmed the existence of arterio-luminal channels (vessels of Vieussens) and thebesian veins connecting the capillary bed and small veins with the heart chambers. But some other structures were also described in detail, which Wearn et al regard as the same myocardial sinusoids incompletely described by Minot in 1900. Their walls lie in close contact with the heart muscle, running between bundles of muscle fibers and at times between the fibers themselves. Arterial branches supplying these myocardial sinusoids were termed "arterio-sinusoidal vessels". The fact that these are arterial in character at their beginning, gradually changing to simple, irregular endothelial tubes makes it improper to term them either arteries or veins. The myocardial sinusoids run a meandering course, anastomose very freely with one another and with capillaries, and open into the lumen of the ventricle either directly or through a common opening with arterio-luminal channels.

The similarity of the walls of the myocardial sinusoids to those of capillaries, and the somewhat similar distibution between and in close contact with muscle bundles and fibers would indicate that these sinusoids

play some definite role in supplying parts of the heart with blood.

Thus the schema in Plate 9 may be regarded as representing the vascular channels of the heart, as far as present knowledge goes.

The coronary circulation in the resting individual carries about 5 per cent of the total volume flow of blood in the body. During extreme exertion, this may be increased to as much as 15 per cent. In general, coronary flow is directly proportional to the pressure within the aorta, the coronary circulatory bed filling when the myocardium is relaxed during diastole. During systole, this circulatory bed is rapidly emptied, much as one would squeeze water from a wet sponge. This is accomplished in large part by the special circulatory mechanisms just discussed. Indeed, Evans and Starling have calculated that 60 per cent of the blood of the coronary circulation is delivered into the right auricle by the coronary sinus, he remaining 40 per cent finding its outlet through the other channels described above.

Anrep and his associates in 1927 used the heart-lung preparation and the hot-wire anemometer to measure and describe the rates of circulation in the heart relative to the cardiac cycle. They found that the flow into the coronary system increases rapidly as the ventricle relaxes, but more gradually throughout the remainder of

the diastolic period and during the isometric period of ventricular systole. The flow is greatly and suddenly reduced during the ejection phase of ventricular systole, being almost completely arrested in the left coronary since the force exerted by the heart muscle is greater than the aortic pressure. This force is less in the case of the right heart, so flow in the right coronary is not reduced as much as in the left. The flow from the coronary sinus is increased slightly during the latter half of auricular systole and very greatly during ventricular ejection.

Batson and Bellet in 1930 found that there is considerable to-and-fro movement of blood in the coronary veins. They injected flakes of graphite too large to pass through the pulmonary circulation into the femoral vein of dogs. Examining the hearts, they found flakes impacted in the smaller coronary veins, but none in the arteries. This is probably due to the same extracoronary factors which produce the wide fluctuations in coronary artery flow.

From his perfusion experiments in the beating human heart, Wearn (1928) believes that all of the capillaries in the heart are functional all of the time, stating that the intermittency of capillary flow observed in skeletal muscle does not occur in the myocardium.

Green and Wegria (1942) made a significant study of the effects of asphyxia, anoxia, and myocardial ischemia

on the coronary blood flow of dogs. They demonstrated that ashyxia due to stopping the artificial respiration, local ischemia produced by interruption of blood flow to an area of myocardium, and anoxia due to local poisoning by sodium cyanide which interferes with intracellular respiratory enzymes caused increased coronary flow even in the absence of changes in aortic pressure. Local ischemia for 45 to 60 seconds increased the instantaneous systolic and diastolic flow 200 to 400 per cent and 200 to 350 per cent respectively. Longer ischemia had no effect. Similar results were obtained with asphyxia and cyanide poisoning, so it was concluded that the increase is due to anoxia, probably in the myocardium. The importance of this in coronary occlusion with respect to bringing in blood via collateral channels is obvious.

In addition to these chemical considerations, there are certain hydrodynamic factors involved in the development of collateral blood supply to an ischemic area, which Wiggers (1936) has pointed out. In the normal heart with no ischemic areas, there are no pressure gradients between areas of the myocardium. But when an area becomes ischemic due to partial or complete occlusion ofa coronary artery, the pressure within the arteries of this area is decreased, so a pressure gradient exists between this portion of the vascular tree and those portions adjoining it. Due to this pressure gradient blood

tends to flow tward the ischemic area through the small anastomatic channels which have been described. With the resultant increase in pressure within these channels, dilatation occurs and collateral circulation which previously was potential becomes functional. Wigger, further points out that when the size of a cornnary artery is reduced, flow from the ventricle into the coronary beyond the partial obstruction could occur during systole via arterio-luminal vessels; during diastole, flow would be back toward the ventricle thus resulting in a to-and-fro movement. When a coronary artery is completely occluded, no pressure gradient exists during diostole between the ischemic area and the ventricular cavity, so blood flow would be only toward the ischemic area in the appropriate arterio-luminal vessels. The pressure relationships which are involved in bringing blood to an ischemic area of myocardium from the ventricle and from extracoronary anastomatic sources are shown in the diagrams of Plate 10.

It is not the purpose of this paper to discuss the pathology of coronary occlusion per se, but a few important facts should be considered. Coronary occlusion is ommonly produced in three ways:

(1) Arteriosclerotic narrowing of the vessel

(2) Thrombosis in an already arteriosclerotic vessel

(3) Syphilitic aortitis.

In rare cases syphilitic arteritis of a coronary vessel or an embolus from a vegetation on a valve may block a vessel. Of these various causes by far the most important is atherosclerosis with or without an added thrombosis. The result may be slow ischemic atrophy of cardiac muscle with replacement fibrosis and scarring, or it may be sudden production of an infarct of the heart with the accompanying dramatic clinical phenomena which are so well known. But these do not always occur. Blumgart, Schlesinger, and Zoll (1941) found a series of cases at autopsy of patients dying from other causes in which there were complete occlusions of a coronary artery or one of its branches without infarction, and without a history of angina of any kind. Injection studies showed filling of the coronary artery distal to the occlusion when the injection mass was introduced through the other coronary, so that collateral circulation must have been adequate. Therefore, complete occlusion of one or more major coronary arteries may be compatible with comfort and longevity. In the entire series of 353 patients, every patient suffering from angina pectoris primarily, without valvular disease and without arterial hypertension, showed old complete occlusion of at least one and in most cases two major arteries before the terminal illness.

In one sense it is fortunate that the sclerotic lesions do not ordinarily involve the smaller ramifications of the coronary arteries, because if they did, the development of collateral circulation would be greatly embarrassed if not altogether impossible.

Pericardial adhesions are a common result of the pericarditis which is produced when an infarcted area includes the surface of the heart. Vascularization of these which is not uncommonly observed at autopsies may provide an additional but belated source of blood for the ishemic area. In 1935 Hosler and Williams made an experimental and pathologic study of the role of pericardial adhesions in cardiac hypertrophy. They were unable to produce circulatory embarrassment of any kind in dogs in which extensive adhesions were induced between the heart and pericardium, and to the right and left sides of the diaphragm, which in the dog places much more tractionon the heart than do similar adhesions in man. In several thousand autopsies which they reviewed, 1.7 per cent showed extensive cardio-pericardial adhesions. In all cases in which there was cardiachypertrophy, there was associated valvular disease. The cases without cardiac hypertrophy were free from valvular, myocardial, and vascular disease. They concluded in contradiction to the ommonly held viewpoint that adhesions per se do not cause circulatory embarrassment unless they are extensive

enough to cause cardiac compression, angulation, or torsion, these being very rare.

If the foregoing considerations are summed up, it can be stated that in th ordinary course of events and in the average young adults heart, the intricate systems of anastomosis are all in active function and are not prepared to act suddenly as entirely adequate compensatory agents. Nevertheless, when vascular obliteration takes place, a certain amount of compensation does occur so that the infarcted area is smaller than the region supplied by the obliterated vessel, the remaining portion receiving sufficient nutrition from the anastomoses. Moreover, if the occlusion is gradual and circulation good, sufficient dilatation of the anastomosing vessels can occur to preserve considerable or even all of the musculature. Where the occlusion occurs in a relatively older individuals heart, the patent and free anastomoses, as well as the well-developed arteriae telae adiposae and the vascular channels of Thebesius, Vieussens, and Wearn, can often amply supply the affected area so that the myocardium can be completely spared.

Th amount of blood brought to an ischemic area will depend upon: (1) the pressure gradient; (2) the number and size of collateral channels present; (3) the length of time over which the occlusion is developing.

The Problem of Experimentally Produced Collateral Coronary Circulation;
The Experimental Basis of the Coronary Operation.

As has been shown, numerous compensatory factors are brought into play when occlusion of a coronary artery or one of its branches occurs. But it is generally agreed that these are not sufficient entirely to compensate for the progression of coronary sclerosis or for its accompanying deleterious effects upon the myocardium in at least a large proportion of cases. The intracardiac compensatory factors include intercoronary anastomoses and the structures of Vieussens, Thebesius, and Wearn, the physical and chemical conditions for the development and utilization of these being present in occlusive coronary disease. Possible extracardiac soures of blood which are available through normally occurring anastomotic channels have also been enumerated. (Hudson, Moritz, and Wearn 1932)

In 1932 Moritz, Hudson, and Orgain reported the examination at autopsy of four human hearts surrounding which there were extensive pericardial adhesions. These patients had died of causes not related to the heart, and there was no significant coronary arteriosclerosis. They injected the coronary arteries with a colloidal suspension of lamp black and found that the presence of vascular channels in the fibrous adhesions and the normally occurring anastomoses between the coronary and extracardiac circulation permitted very extensive

injection of the mediastinal vessels. They reasoned that if extracardiac anastomoses actually constitute a significant reserve for the coronary circulation, then vascularized pericardial adhesions might provide some degree of protection in coronary artery disease. With this in mind, work was started at Western Reserve University to study the functional significance of such an experimentally produced collateral circulation in experimental coronary occlusion. This was probably the starting point of all the research which has been done in recent years concerning surgically induced collateral coronary circulation.

In 1934 Beck, also of Western Reserve University, was resecting a cardiac scar which was adherent to the parietal pericardium. Upon transection of this, brisk bleeding occurred from both cut ends. This appears to be the first observation at the operating table of extracardiac anastomoes with appreciable flow of blood, and is especially interesting because Beck had been doing considerable experimentation with surgical revascularization of the heart in dogs. Some of these experiments were reported by Beck and Tichy in 1935, and by Beck alone in the same year. It was attempted in these experiments on dogs to promote extracardiac anastomoses by grafting various vascular tissues to the surface of the heart. Among the vascular structures utilized as grafts

in these studies were pericardium, pericardial fat, mediastinal tissues, pleura, omentum, and pectoralis major, triangularis sterni, and intercostal muscles. When these grafts were applied to the heart, it was found to be necessary to produce some irritation of the epicardium in order to promote the development of atisfactory adhesions. A number of methods were tried to accomplish this, notable among which were assorted chemical irritants which were found to be too violent or too unpredictable in their action, sandpaper and special burrs for mechanical abrasion of the epicardium, the means of final choice being the burrs.

When these grafts were applied to the normal heart, a few anastomoses developed between the vessels in the graft and the coronaries, but these were few and small. In order to demonstrate these, the aorta was clamped and divided just distal to the coronary ostia, and the grafts were injected via the aorta with 0.4 per cent aquaeous solution of ferric ferrocyanide at a pressure of 100 mm. Hg. Plate 11 shows the normal heart after injection with dye through the grafts. This specimen shows the greatest amount of injection through the extracardiac anastomoses found in the whole series of 16 normal specimens; it is obvious that thisdegree of collateral circulation is grossly inadequate to provide any significant protection of the coronary circulation.

It would seem that although a satisfactory vascular bed was available in the grafts, a stimulus which would bring about continuity between the cardiac and extracardiac beds was lacking.

A pressure differential was created between the coronary and extracardiac vascular beds by placing adjustable silver clips about the three major coronary branches near their origin and tightening these progressively at repeated operations. Thus the hydrodynamic conditions essential to the development of collateral circulation were brought about. Grafts applied to the heart by the same methods as before were found to be more vascular; injection studies corroborated this observation. Plate 12 illustrates the slight degree of injection with dye via the grafts which occurred in response to a slight degree of coronary obstruction due to slight compression of all three major coronary branches, near their origins. The extent of the collateral circulation via the graft is greater than that in the normal dog, but is still far from satisfactory. It was found that as greater degrees of compression of the coronary arteries was produced, thereby increasing the pressure differential, there were proportionately greater extracardiac anastomoses. One typical example is shown in Plate 13. Slight compression of the three branches was done when the grafts were applied; at

reoperation four months later, the right coronary artery and the left ramus descendens were completely occluded. One year after the second operation, the dog, which had appeared normal in all respects, was sacrificed, and injection studies were done. A small cicatrix measuring 12 mm. in diameter in the lateral wall of the right ventricle was judged to represent a healed infarct. It was estimated that the total cross-sectional area of the coronary arterial system was reduced by about 85 per cent. All of the coronary vessels including the capillaries were completely filled with the dye. All of the attempts completely to replace the coronary circulation by collateral channels were unsuccessful. It was noted that the dye injections were not confined to the regions of the myocardium near the graft, but that the whole myocardium was injected, and it was suggested that the increase in intercoronary anastomoses might be promoted by the grafts as well as by the partial coronary occlusion.

Some interesting observations were made at operation also. One of these was that ligation of a small branch of a coronary artery very frequently was followed almost immediately by fatal ventricular fibrillation. This might account for the sudden death occassionally observd in patients who at autopsy show occlusion of only a small branch of a coronary artery. Ligation of

a large branch rarely resulted in disturbance of rhythm. Another of these was that destruction of areas of epicardium with the burr generally produced some disturbances in rhythm which were usually transient. These includedsinus tachycardia, heart block, extrasystoles, ventricular tachycardia, auricular fibrillation, and occasionally ventricular fibrillation.

Mautz (1936) showed that one cc. of procaine or metycaine in 10 per cent aquaeous solution invariably stopped auricular fibrillation and other arrhythmias induced by instrumentation and decreased cardiac irritability markedly if applied to the epicardium a few minutes before abrasion was started. He also showed that quinidine sulfate given orally in 30 mg. doses twice daily for a few days preoperatively decreased the incidence of ventricular tachycardia and fibrillation. These measures were adopted by Mautz, Beck, and their associates as routine in cardiac surgery henceforth. They further state that ventricular fibrillation can be stopped and normal rhythm restored in 80 per cent of occurrences by massage of the heart to fill the coronaries with fresh blood followed by brief application of 60 cycle alternating current at 1 ampere with contacts on either side of the heart.

Mautz and Beck (1937) studied the sites in which collateral coronary circulation developed in experimental

chronic obstruction of the left descending coronary branch in dogs, produced over a period of 6 months. The dogs were sacrificed and injections of a radio-opaque s substance made via the descending ramus distal to the occlusion; six-foot x-ray plates were then made of the hearts. In 100 dogs several large anastomatic channels were quite consistently observed. These were:

(1) Between the left distal and right proximal auricular branches over the base of the heart.

(2) Channels over the conus of the right ventricle.

(3) Between th right and left coronaries in the posterior auricular-ventricular sulcus.

These vessels were thin walled with little smooth muscle and practically no elastic connective tissue fibers. The normally occurring extracardiac anastomoses were only slightly increased. Plate 14 illustrates these findings. Fig. 2 of this plate illustrates in diagrammatic form the anatomic connections observed between the main coronary arteries and between coronary and extracardiac arteries.

In 1941 Stanton, Schildt, and Beck reported their latest experiments on dogs in which 100 animals were used in groups of ten dogs each. In 50 of these, the entire epicardium was thoroughly abraded with a metal burr so that cardio-pericardial adhesions would form. After one, two, and three weeks the descending ramus of

the left coronary artery was ligated and divided in groups of 10 dogs. These ligations were similarly performed in 50 hearts in which no abrasion had been done. In the group of non-abraded hearts, the mortality was 70 per cent; in the abraded hearts the mortality was 38 per cent. After two months, all of the remaining dogs were killed, and the hearts were injected by way of the right and the left circumflex arteries with a radio-opaque substance too viscuous to pass through the capillaries, so that only the arterial tree would be injected. The normal specimens injected showed infarction but no intercoronary anastomoses, whereas the abraded specimens contained extensive intercoronary anastomoses, quite large enough to provide a common arterial bed. It was further noted that in the abraded specimens, the size of the infarct was consistently reduced, in some cases, even to non-existence.

Experiments similar to those by Beck and his associates were done in England by O'Shaughnessy between 1936 and 1939. These were terminated by the untimely death of O'Shaughnessy who was killed during the British evacuation at Dunkirk in the present war. After experimenting with several types of tissue used as grafts, his final choice was a pedicle graft of greater omentum brought through an incision in the diaphragm. Beck agrees with O'Shaugnessy in regarding omentum as the

most effective structure available, but believed that the diaphragmatic hiatus produced was too conducive to diaphragmatic herniae involving abdominal viscera. O'Shaughnessy recognized this possibility, but never saw it occur. He stated that of all the structures in the body, the omentum has the greatest potentiality for vascularization, and cites cases of colossal uterine fibroids nourished by as many as 6 arteries each about the size of the brachial artery, passing to the tumor by way of omental adhesions (O'Shaughnessy 1937).

O'Shaughnessy avoided the use of mechanical abrasion of the epicardium in order to induce adhesions. He found that when an aleuronat-starch paste was applied to the epicardium, supple, vascular adhesions were formed, without systemic disturbances. Thus the grafts could be sutured to the pericardium after application of aleuronat to the graft where it touches the heart, thereby avoiding suturing the graft to a friable myocardium. Aleuronat is a preparation of aleurone granules which are found in the seeds of some plants and cereals.

Using greyhounds in his dog experiments enabled him to obtain a physiologic check on his results. These dogs subjected to ligation of the descending branch of the left coronary artery without application of an omental or other graft were found to have markedly reduced running capacity when tested on the track 4 months

after operation, showing abnormally great dyspnea and obvious signs of distress. But when dogs which had omental pedicle grafts applied at the same time the ligation was done were tested on the track, they were found to have approximately normal running capacity and generally finished the course in very good order.

The results of O'Shaughnessy's injection studies were, in essence, the same as those of Beck.

Thompson (1940) and Thompson and Raisbeck (1942) reported a series of dog experiments in which they obtained some rather remarkable results. In a control group of 20 dogs, the descending branches of the left coronary artery and vein were ligated with a mortality of 50 per cent. In 16 dogs the same ligations were done and sterile talc was dusted into the pericardial cavity at and about the ligation site with a mortality of 25 per cent. In another group of 16 dogs adhesive pericarditis was produced with talc, followed by the same ligations two to three weeks later with no mortality. In a group of 8 dogs the same operations were performed followed in 2 to 3 weeks by ligation of the right coronary artery at about its middle; 2 to 3 weeks later the left circumflex branch was ligated at its origin; 2 to 3 weeks after that, the right coronary artery was ligated at its origin, leaving practically nothing but the septal branch intact. The total mortality in this experiment

was 75 per cent, but the remaining two dogs ran and played normally, appearing clinically well. Unfortunately, no injection studies were made of these hearts.

Previous to these experiments, Robertson (1935) and Gross and Blum (1935) had demonstrated that ligation of the coronary sinus and major veins was followe d by a conspicuous increase in extent of the vascular bed and the arterial anastomoses, as shown by injection studies. Subsequent ligation of arteries produced smaller areas of infarction than in hearts with intact veins. Robertson was able to ligate all of the arteries and veins of the heart in multiple operations. In the surviving dogs the extensive pericardial adhesions were cut and tied to see if the thebesian-like structures could support cardiac function, many patchy areas of cyanosis developing at this time. Only one dog survived this last operation, and it died two months later in congestive heart failure. Mortality rates were not stated, but it was implied that they were high.

In 1941 Friedbacher reported an unsuccessful attempt to anastomose the internal mammary artery to the coronary artery by direct surgery in dogs. He suggested that this might be possible if anticoagulants which could be locally inhibited were used.

Burchell (1940) performed some experiments very similar to those of Beck, but his interpretation of

results was very different from Beck's. Injection studies after experimental occlusions agreed with Beck and others, the largest anastomotic vessels being found around the apex of the left heart, and around the crux of the heart. When grafts like those Beck uses were applied to the heart, Burchell was able to demonstrate no protective action in subsequent experimental occlusions. The grafts were seen to contain some small vessels, but these could not be shown consistently to carry more blood or appear of larger size when there was chronic coronary obstruction. Results were the same with omental grafts, and only a small amount of barium sulfate reached the coronary circulation in one heart of several injected by way of the omental grafts.

In three dogs with muscle, and three dogs with omental grafts and occlusion of the three main coronary arteries, section of the grafts was not found to inconvenience the dogs, and no electro-cardiographic changes could be shown which indicated loss of blood supply to the myocardium. Furthermore, in dogs in which these same conditions were produced, Burchell could demonstrate no significant volume of blood flow through the pericardial adhesions. When the thoracic and abdominal aortae were separately perfused with a mixture of Ringer's solution, blood, and dye at pressures of 110 to 130 mm. Hg. in the grafts and 60 to 80 mm. Hg. in the

heart-lung part, only small amounts of dye reached the heart. In three normal dogs, dye was recognized in the hearts of two, and this amounted to a flow of 0.5 to 2.0 cc. of flow via the graft per minute. In one dog with intact coronary circulation and a muscle graft, the amount of dye indicated a flow of 2.0 to 3.0 cc. per minute; in one with intact coronaries and an omental graft, flow in the graft was 2.0 to 4.0 cc. per minute. In two preparations in which the three main coronary arteries were progressively occluded over a period of 7 to 20 months with grafts in place, no dye at all could be recognized in the cardiac output.

Whereas Burchell did not deny the possibility that vascular channels in adhesions and grafts might develop functional value, he did not observe it to happen in experiments seemingly favorable to such a result.

From a consideration of the results of the experiments conducted by all of these investigators a few findings seem to stand out. All seem to agree that operations involving considerable areas of the hearts surface are conducive to the development of intercoronary collateral channels. Although post mortem anatomical studies appear discouraging in some instances, mortality figures and physiologic endurance tests such as those of O'Shaughnessy constitute undeniable evidence that the heart does receive ample blood by way of col-

lateral channels. Thus it would appear that perhaps, nearly as much is accomplished through stimulation of intercoronary anastomoses as by bringing in a new blood supply by way of grafts.

Direct surgical anastomosis of the internal mammary artery to a coronary artery is probably not practical at least by present technical methods, because the occlusive lesions so frequently extend in the arterial tree to branches smaller than can be satisfactorily operated upon. Artificially induced collateral circulation, however, could not be effective if there were significant disease in the very small arteries within the myocardium.

The Coronary Operations

Beck, O'Shaughnessy and Thompson and their associates having done the experimental work previously described, were the first surgeons to make any extensive attempts to accomplish surgical revascularization of the heart in man. Their criteria for selection of patients upon which these operations were to be performed were rather rigid and quite uniform. They were:-

(1) Unequivocal evidence of cardiac ischemia in clinical, electro-cardiographic, and roentgenologic findings.

(2) Absence of improvement with long, thorough medical treatment.

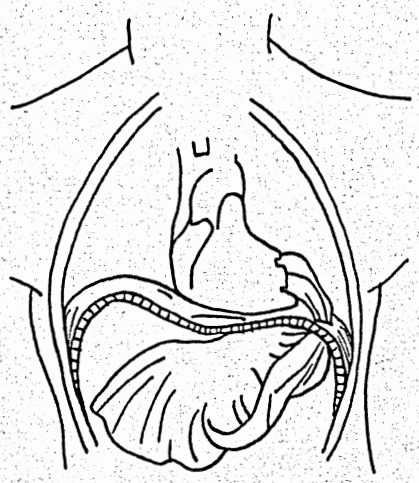
(3) Absence of congestive failure at the time of operation. In patients with a history of decompensation, pre-operative digitalization was done.

(4) Extreme disability, sufficient to place the patient in Class 3 of the classification proposed by the American Heart Association.

In 1935 Beck performed the first coronary operation as illustrated in Plates 15 nad 16. After incision through the skin and pectoralis major as shown, and resection of the third, fourth, and fifth costal cartilages, the pericardium was incised vertically. The pericardium and epicardium were thoroughly abraded with a metal burr. A pedicle graft from pectoralis major muscle was then applied to the abraded anterior and left surfaces of the heart. Closure was then made as shown. In subsequent

operations, quinidine 0.2 g. was given orally every four hours for several doses, and 5 cc. of procaine in 2 per cent aquaeous solution was applied to the heart for five minutes before proceeding, to decrease the incidence of arrhythmias. Later, Beck used two pedicle grafts of pectorolis major, one around each side of the heart as in plate 17, fig. 1 and also used vascular mediostinal fat in addition to this.

O'Shaughnessy (1937) used quite a different operative technic, namely cardio-omentopexy. He made his incision in the fifth intercostal space from the sternum to the anterior axillary line, dividing costal cartilages 5 and 6. The left phrenic nerve was crushed, and a small incision made in the left side of the diaphragm, through which a suitable portion of omentum was isolated and brought into the thoracic cavity, suturing the diaphragm about it. The omentum was then brought through a



pericardial incision and sutured to the edges of the pericardium and to the heart. In subsequent operations, aleuronat paste was used to produce adhesions to the heart, and no sutures were placed in the epicardium. Shock was largely avoided by minimizing trauma and blood loss and by interruption of nociceptive impulses carried by the phrenic nerve. The anatomic relations produced are shown in the accompanying diagram.

Thompson and Raisbeck (1942) used a much simpler operative technic. They resected one and one half inches of the fifth left costal cartilage and incised the pericardium. After aspirating the fluid, they introduced 5 cc. of procaine in 2 per cent aquaeous solution and waited 5 minutes before proceeding. Eight grams of sterile talc was applied to the anterior surface and left and inferior borders of the heart, and closure was done.

Beck and his associates operated on 30 patients between 1935 and 1937, in which his operative mortality was 33 per cent. It is noteworthy however that the mortality rate decresaed markedly as more experience was gained. Of the 20 patients who survived the operation, four died in the next three years, and of these, autopsies were performed on three. Two of these had showed poor clinical results, one dying of inanition and the other of coronary sclerosis, and injection studies did not demonstrate anastomoses in the grafts large or numerous enough to carry

any significant volume of blood. In the case which had had a good clinical result, a severe degree of coronary sclerosis was found. The injection studies showed ten or twelve communications between the arterial system and the grafts which were judged large enough to have explained the clinical improvement. Further anatomical studies must wait until more of the surviving patients die.

An evaluation of Beck's clinical results shows that 65 per cent of the 20 surviving patients were completely or almost completely relieved, going back to their previous occupations; 20 per cent were moderately improved; 15 per cent showed inconsequential improvement. These cases are summarized in the tables of Plate 18. The clinical courses of three typical cases of Beck are herewith summarized.

Case 1. Arteriosclerotic heart disease and angina pectoris of 9 years' duration, severe for 5 years; moderate generalized arteriosclerosis with hypertension; arterial pressure systolic 164, diastolic 92 mm. Hg.; moderate chronic pulmonory emphysema. A 48 year old farmer, with gradually diminishing exercise tolerance, finally became incapacitated for any work because of pain. Exercise tolerance tests brought on precordial pain, fatigue, and dyspnea. Operation was done Feb. 13, 1935. Four months later he began to do light work around the hospital as a gard-

ener. He has returned to farming, doing 12 hours work daily for four years, feeling no more than ordinary fatigue at the end of the day. The result in this, the first patient to have the operation was excellent. See Plate 17, Fig 2.

Case 24. Arteriosclerotic heart disease and angina pectoris of five years' duration. A tailor, aged 50 years, in November 1933, and again in August 1936, had myocardial infarcts. They were anterior and apical, and posterior and basal. Invalidism followed the second attack and he was barely able to go to his shop. Slight exertion and emotional strain brought on pain. The left ventricle was slightly enlarged. The patient had extensive myocardial fibrosis and was a doubtful candidate for operation. Operation was done April 9, 1937. At the operation the heartbeat was feeble, and it was apparent that the muscle was seriously damaged. The patient stated that he had been greatly relieved from pain following the operation, but his exercise tolerance was not increased. He died one year and two months later of myocardial infarction. Autopsy showed a poor anatomical result. The clinical result in this case must be considered poor.

Case 25. Arteriosclerotic heart disease and angina pectoris of 9 years' duration. A retired salesman, aged 52 years, had had some attacks which were sufficiently severe to have been caused by coronary

thrombosis. The conventional electrocardiogram was normal, but the T-wave in the chest lead was reversed in direction. The record taken during induced anginal attacks after exercise showed changes suggesting a posterior and basal infarct. Operation was done April 24, 1937. The patient states that there is improvement and he is able to carry on his work, without symptoms. The clinical result in this case should be considered fair.

O'Shaughnessy operated on six patients. There was no operative mortality. Two of these subsequently died, one of uremia, and one of hemorrhage from a duodenal ulcer. The other four patients were alive over a year after the operation and were much improved. He believed that this is a much less hazardous operation than Beck's pedicle grafts of pectoralis, and that omentum has marked advantages over muscle as a vehicle for blood vessels.

Thompson and Raisbeck (1942) reported operation on sixteen patients by the technic previously described. They state that all of these were in such condition that they had "nothing to lose and everything to gain". Of these, one died soon post-operatively, two on the second post-operative day, and one died three weeks later, so their operative mortality was 25 per cent. Of the rest, ten showed marked improvement, striking in some cases, while the improvement of two was moderate. Here again

anatomical studies must await the death of some of these patients. Follow-up examinations with exercise tolerance tests, arterial blood pressure, and direct venous blood pressure recordings, and fluoroscopic, radiographic, and electrocardiographic examinations failed to show any evidence of cardiac constriction, or compression, or hypertrophy due to adhesive pericarditis.

Summary and Present Status of Surgical Revascularization of the Heart in Coronary Sclerosis

There are two means available for inducing the development of intercoronary anastomotic channels. The first of these is chronic coronary obstruction which is impractical surgically. The second is by production of hyperemia due to inflammation on the surface. There is some difference of opinion as to the importance of surgically induced extracardiac anastomoses with the coronary circulation. Since the improvement in most of the patients who survived the various coronary operations cannot be denied, it remains to determine whether the clinical improvement is due mainly to intercoronary or extracoronary collateral channels, or if both are about equally responsible. It is remarkable that all of the patients in whom improvement was noted said that they felt better within 8 to 10 days post-operatively. This is too soon for anastomotic channels of important size to have developed. Beck suggests the possible explanations that the immediate hyperemic reaction might have allowed redistribution of the coronary blood and that nerves lying immediately beneath the epicardium might be torn or otherwise interrupted.

Certainly the mortality rates and the inconsistency of good operative results are such that the coronary operations canno be regarded as established therapeutic measures. But the number which showed striking improve-

ment is both significant and encouraging. All of the patients operated upon were chosen because they were cardiac derelicts, to whom life, as it was, was scarcely worth living, and were hence poor surgical risks at the outset.

It can be conservatively stated that work has advanced to the place where limited application to human patients should be done. Satisfactory data have been accumulated and the experimental evidence is generally good. The improvement in some of the patients is whom operation has been carried out has been almost incredible.

Speculations as to the Future of This Mode of Treatment of Coronary Sclerosis

Only the continued observation of the operated patients over a period of many years will tell us what happens to the collateral vascular channels produced by surgery. Their size might remain static once they have been established, they might increase in size due to the hydrodynamic factors which have been considered, or they might decrease in size due to contraction of the collagen fibers laid down by fibroblasts in the initial phase of inflammation. Since some of these patients have already been under observation for some years without apparent development of myocardial ischemia, it seems unlikely that the last named possibility will very frequently occur.

Beck feels that with constantly improving technic and methods the mortality will be very low - perhaps even negligible - in patients with good cardiac reserve, and quite low in seriously ill patients. Certainly the present mortality rates do not give a fair indication of the mortality to be expected in patients operated upon before their cardiac reserve is seriously impaired. But some of the experimental work discussed indicates that extensive collateral channels are not likely to develop unless there is already considerable embarrassment of the coronary circulation, so that prophylactic operation when

coronary sclerosis is not yet well advanced would not be expected to be very effective.

It is my opinion that the coronary operations will ultimately become valuable therapeutic measures in selected cases, but will probably not become a very widely used mode of treatment.

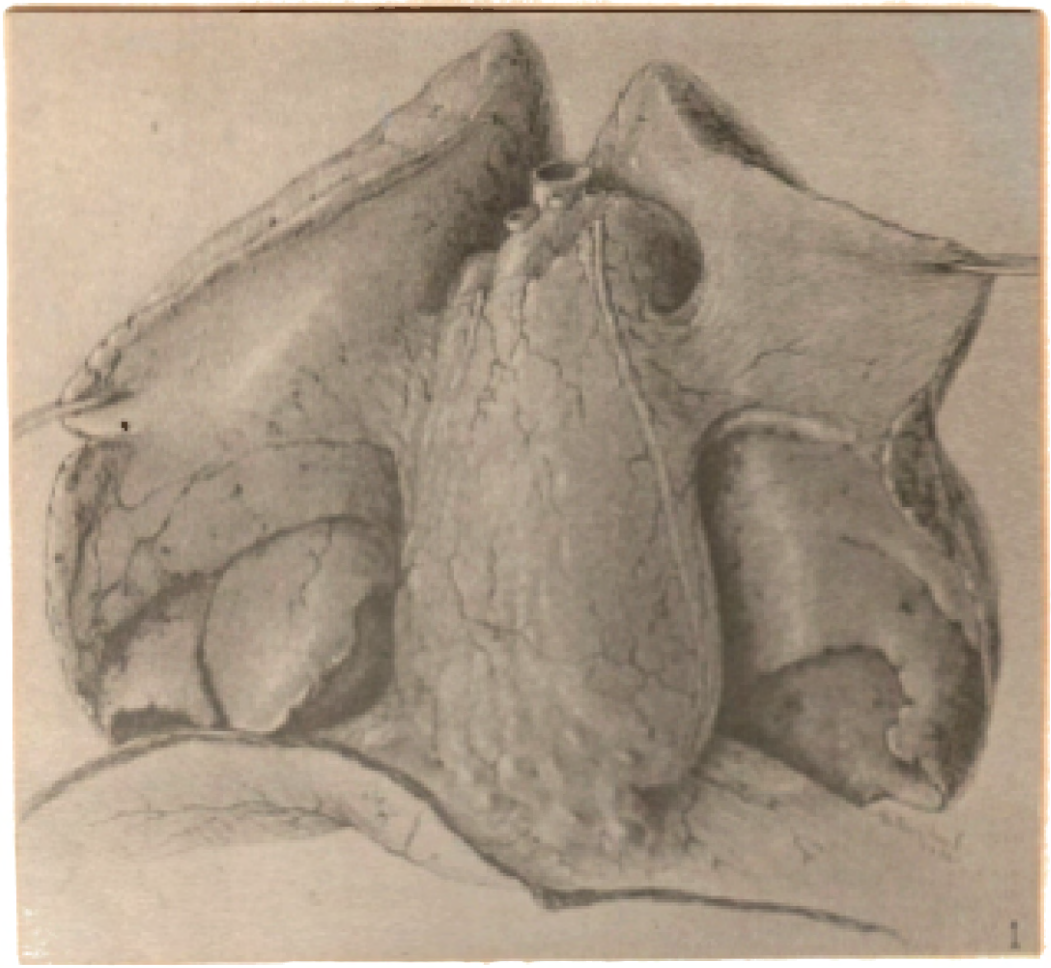


Plate 1

Dr wing of he rt, lungs, and diaphragm after injection of the coronary arteries with a colloidal suspension of carbon particles. net-work of injected pericardial and mediastinal vessels is seen. There are large injected pleur l and di phr tic vessels ab ut one h lf normal size.

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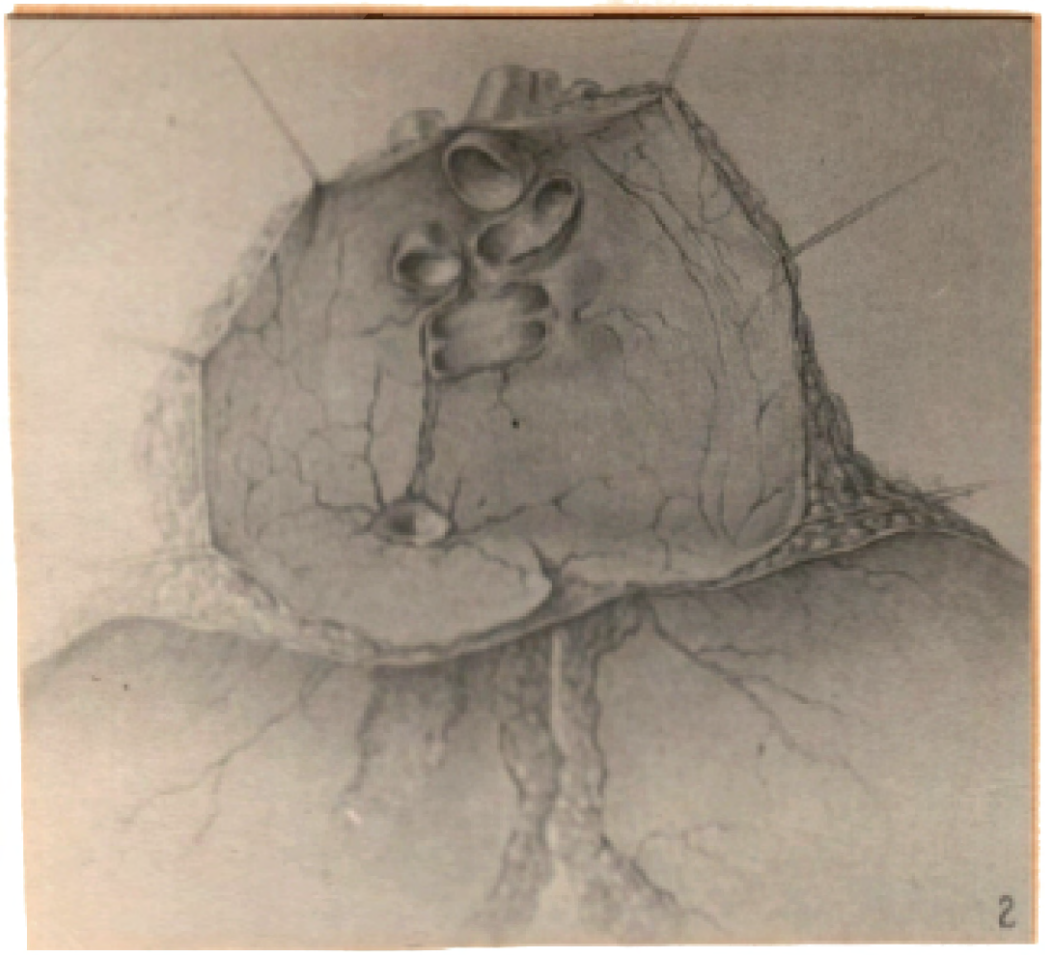

Plate 2

Drawing of the inner surface of the parietal pericardium and the inferior surface of the diaphragm. The coronary arteries were injected and the heart removed to expose the sites of anastomoses between coronary and extr - cardiac vessels. About one half natural size.

(Plates 1 and 2 after Hudson, Moritz, and Dearm.)

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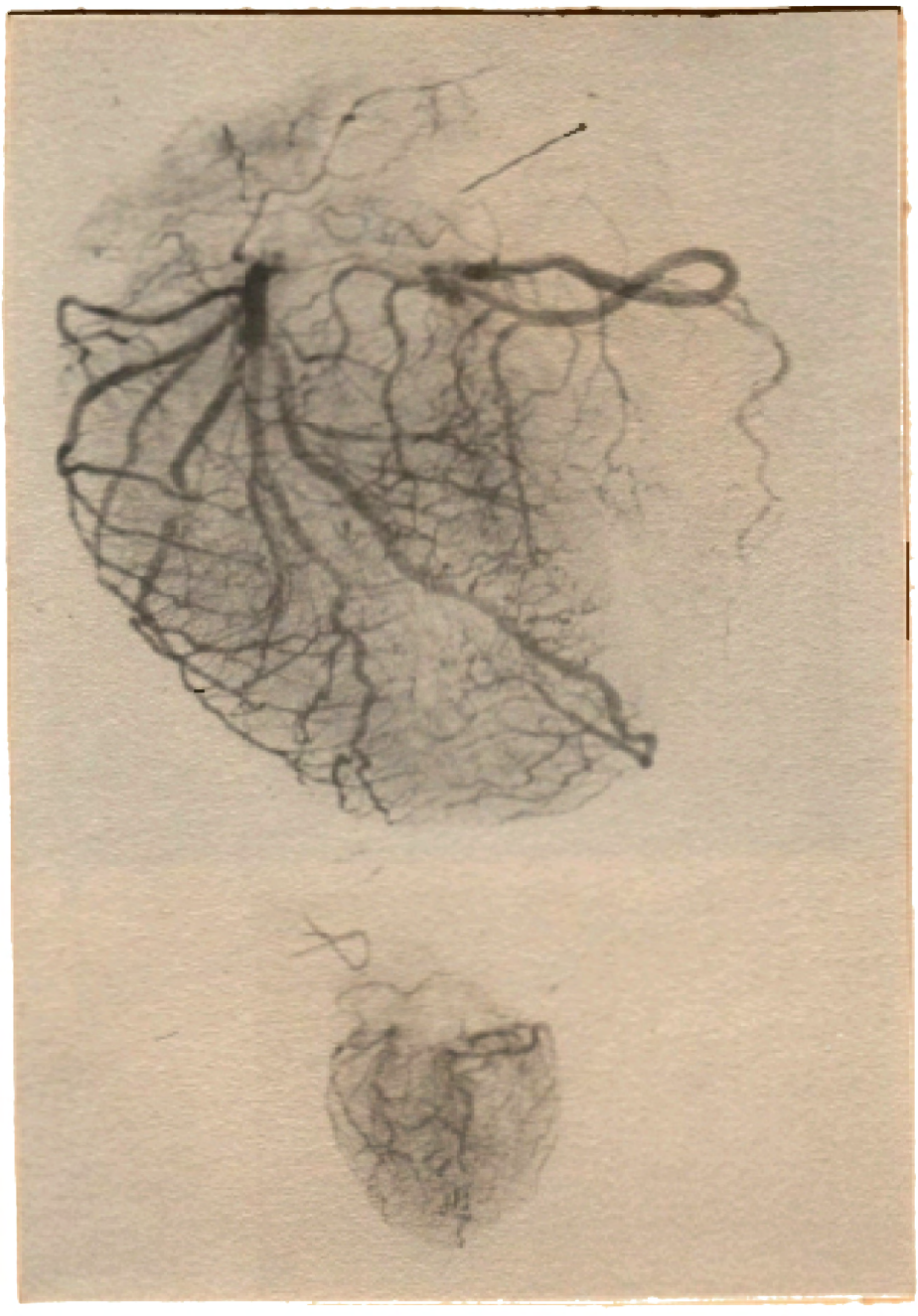


A composite contrasting the circulation at birth and in the seventh decade of life. Note the difference in the relative amounts of blood supplying both sides of the heart, in the tortuosity of the vessels and patency of the septal anastomoses. (After Gross)

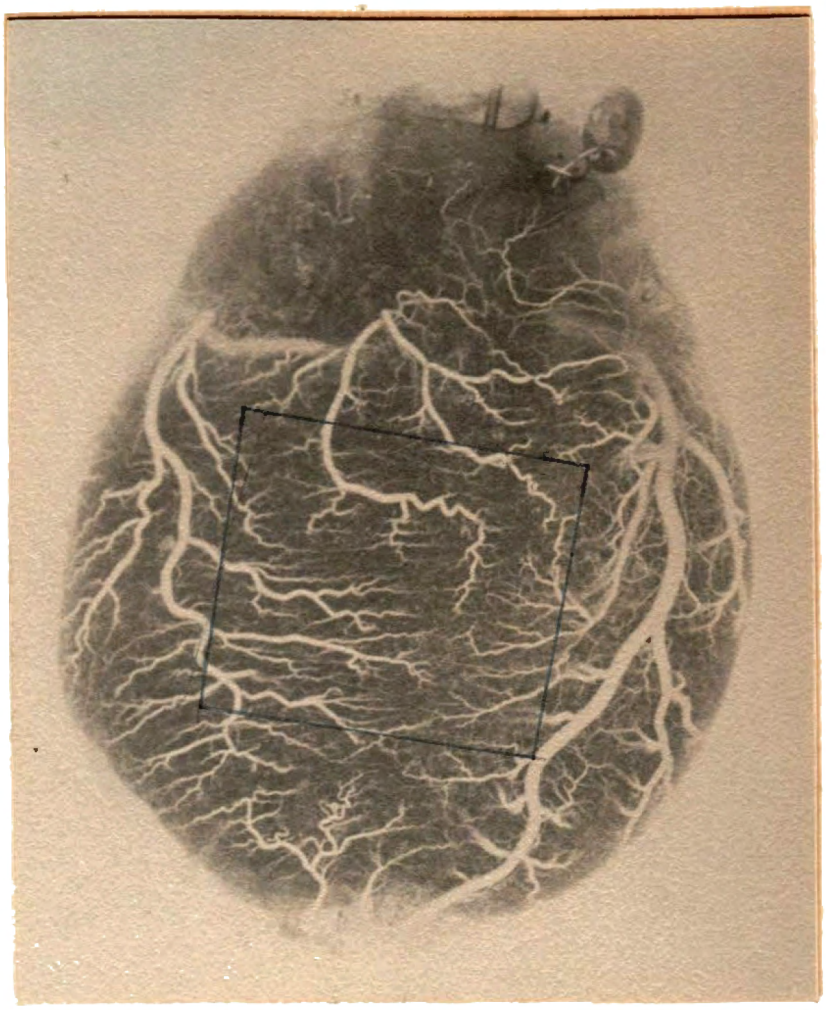


Fig. 1

Fig. 2

Plate 4

Plate 4

Fig.1. A good injection showing very open anastomoses occurring between branches of the ramus descendens anterior of the left coronary artery and those from the rami anteriores of the right. A similar though less conspicuous anastomosis occurs on the posterior surface.

Fig. 2. This is an enlargement of the central area of Fig. 1. (After Gross)

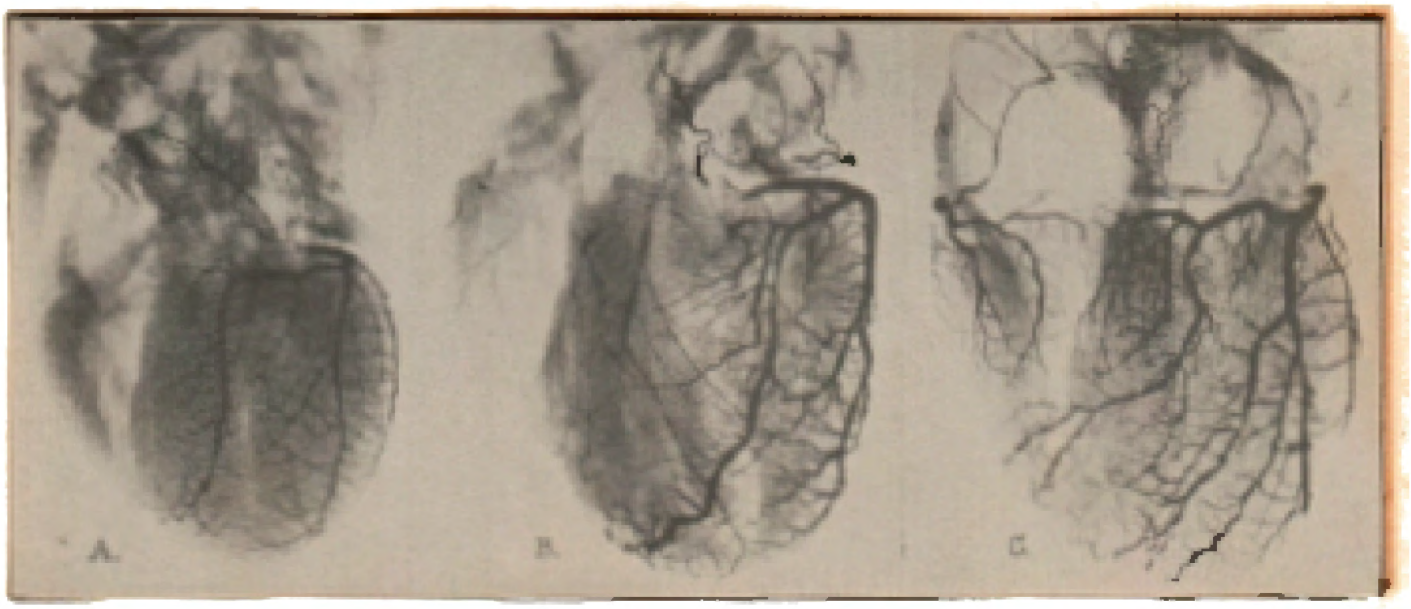


Plate 5

Variation in number an size of intercoronary anastomoses in normal hearts.

A. Shows minimal anastomosis.
B. Shows maximal anastomosis.
C. Shows average number and size of stomoses.

These are photographs of roentgenograms made after injection of the coronary tree with radio-opaque substance. (After Mautz and Beck)

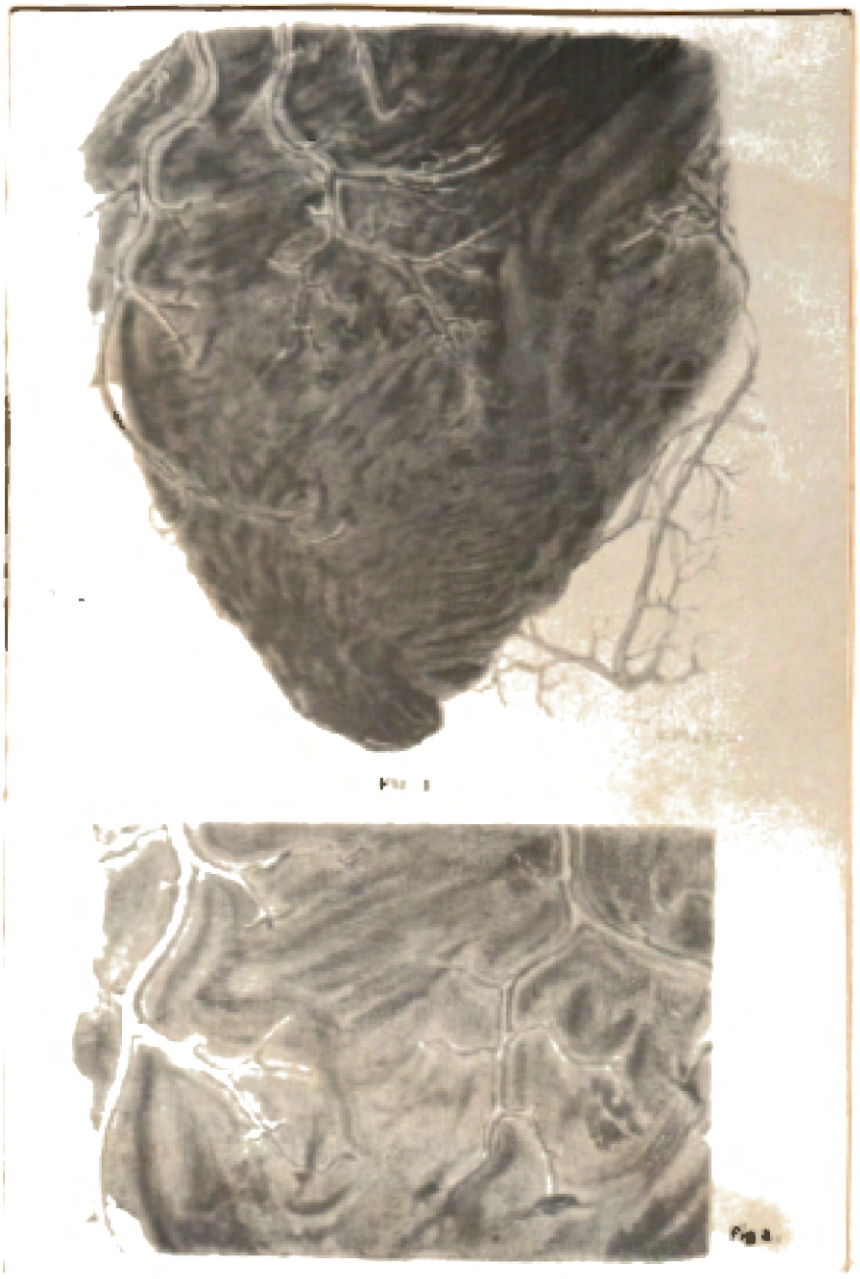


Plate 6

Fig. 1 Celloidin injected and digested heart. Note junctions of arterial casts with ventricular cast.

Fig. 2 An enlargement of the central area of Fig. 1. (After Weardn, Mettier, Klumpp, and Zachiesche)

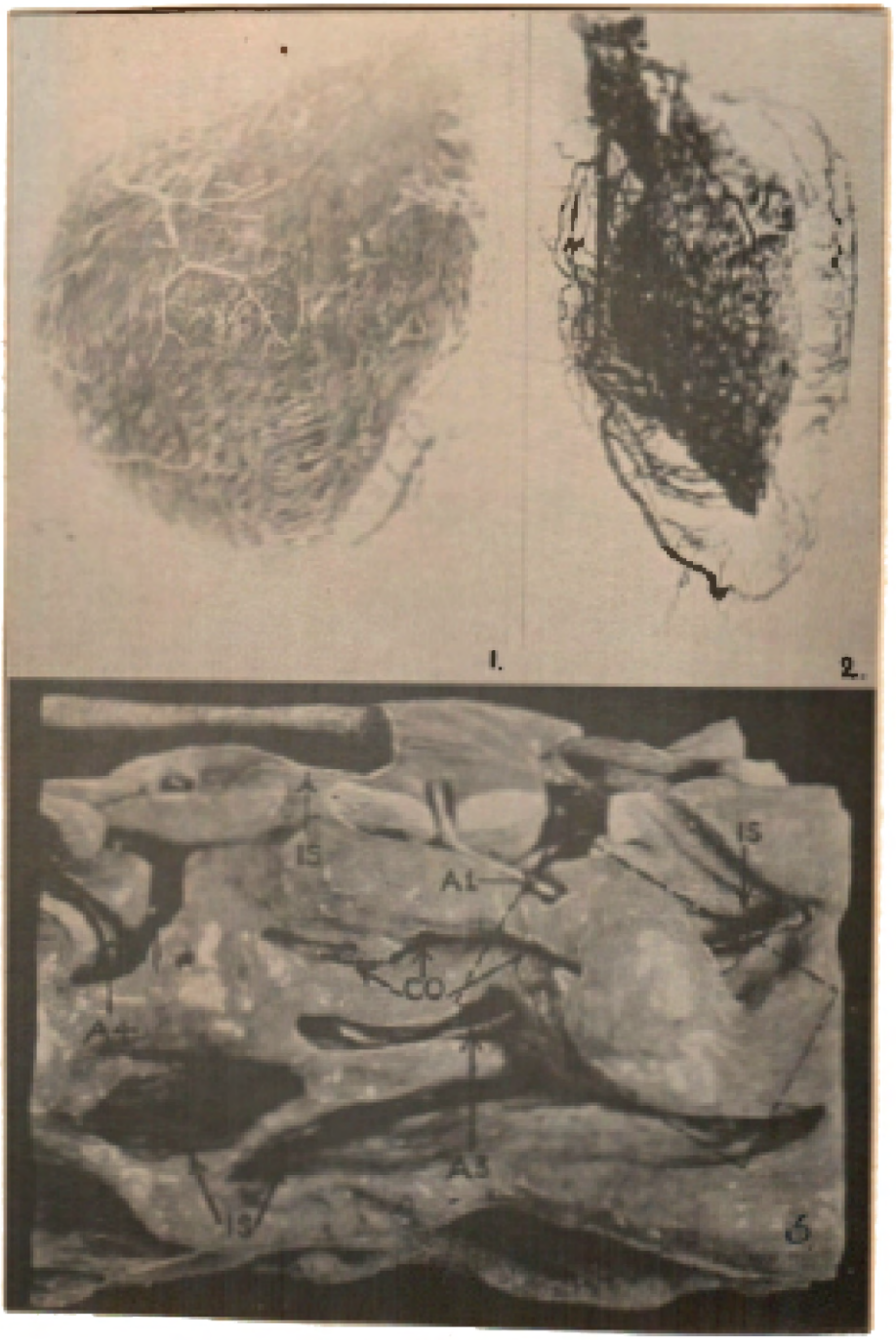
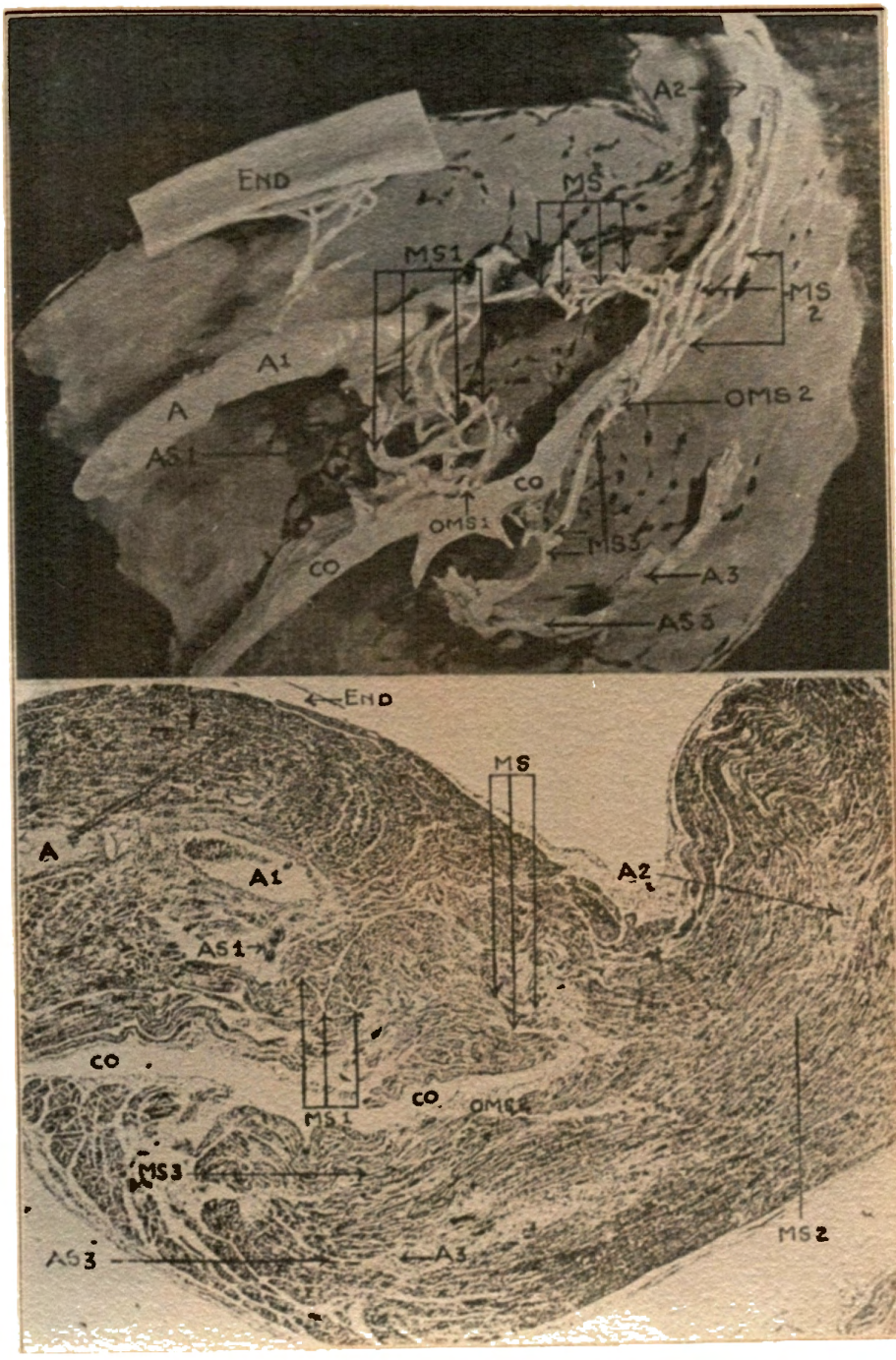


Plate 7

Plate 7

Figs. 1 and 2 are photographs of the heart in Pl. 6 before removal of irrelevant arterial casts. Fig. 2 shows arterio-luminal vessels in profile.

Fig. 3. Wax plate reconstruction through the left ventricular wall near the apex in which is shown the common opening (CO) through which several arterio-luminal vessels and myocardial sinusoids open into the lumen of the ventricle. The wax has been dissected away to show the arteries (A , A , and A .) which communicate with the cavity through the common opening. The dotted rectangle includes the tissue from which serial sections were made to prepare the wax reconstruction shown in Pl. 8. Labeling is identical. This photograph is three fifths the size of the original model which was constructed on a scale 20 times the actual size. (After Wearn, Mettier, Klumpp, and Zschiesche.)


END
A2
MS
MS1
MS 2
A1
A
AS1
OMS 2
CO
OMS1
MS3
A3
AS 3
1.
END
MS
A
A1
A2
AS1
CO
CO
OMS2
MS1
MS3
MS2
AS3
A3
2.

Pla e 8

Plate 8

Fig. 1. Photograph of wax plate reconstruction of the arterio-sinusoidal vessels. AS.is a branch of the artery which opens directly into the common opening (CO) which is an impocketing of the endocardium (END). MS is a myocardial sinusoid, OMS is an opening of myocardial sinusoid. (Reconstruction is 200 times actual size; photograph is one fifth the size of the model.)
Fig. 2. A photomicrograph of one of the serial sections from which the model in Fig. 1 was constructed. Labeling is identical. (X 41) (After Wearn, Mettier, Klumpp, and Zschiesche.)

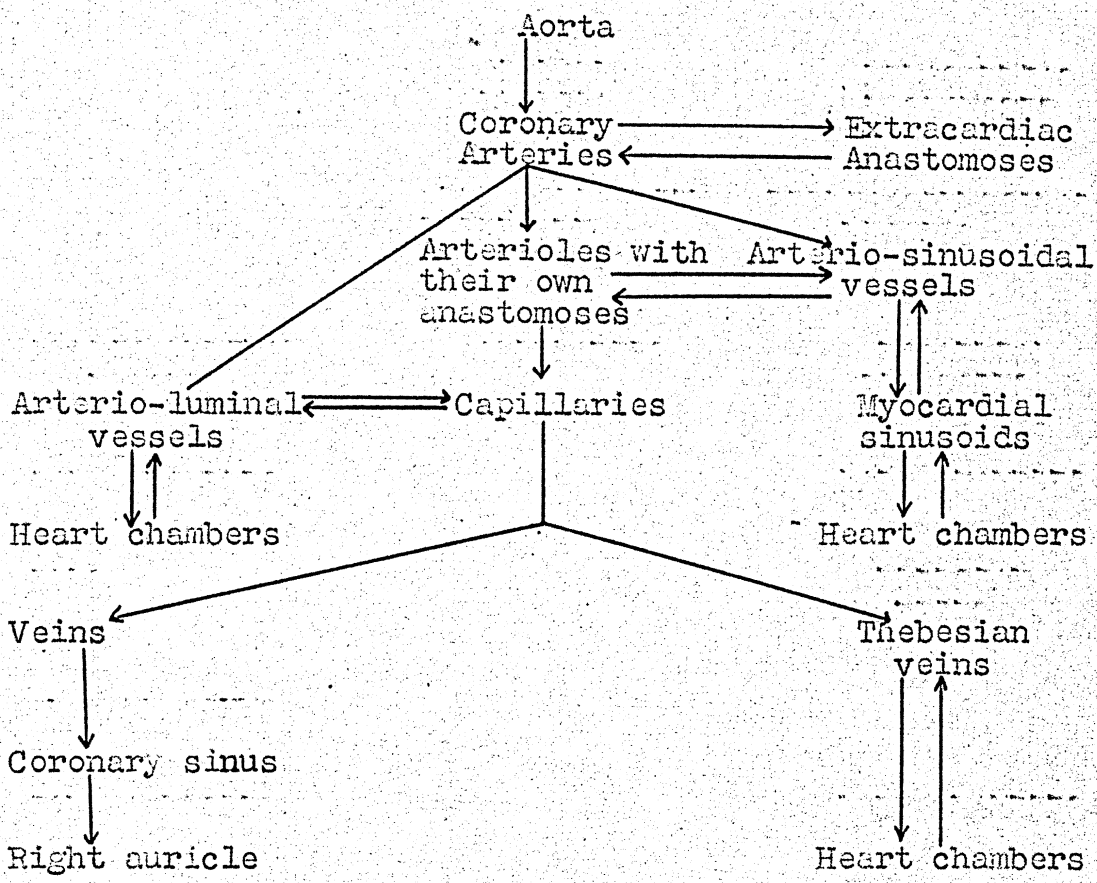
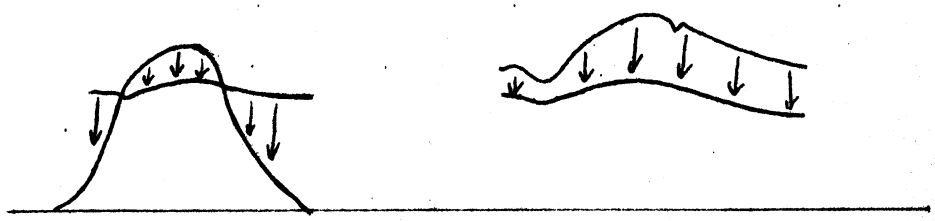
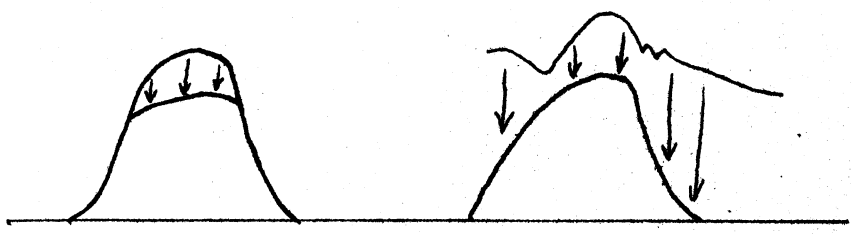


Plate 9

Diagram representing the circulation to the heart in its anatomic aspects.



In gradual narrowing of a coronary branch

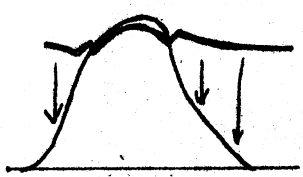


In complete occlusion of a coronary branch

Legend:
Intraventricular pressure indicated by blue

Intracoronary pressure indicated by red

Extracoronary pressure as in other aortic branches indicated by black.



Normal

Plate 10

(After Wiggers)

Plate 11

Normal dog's heart after injection with dye via the extracardiac anastomoses. This specimen shows the greatest amount of injection found in the series of 16 normal specimens. (After Beck and Tichy.)

Plate 12

A slight degree of injection with dye in response to a slight degree of coronary obstruction. (After Beck and Tichy.)

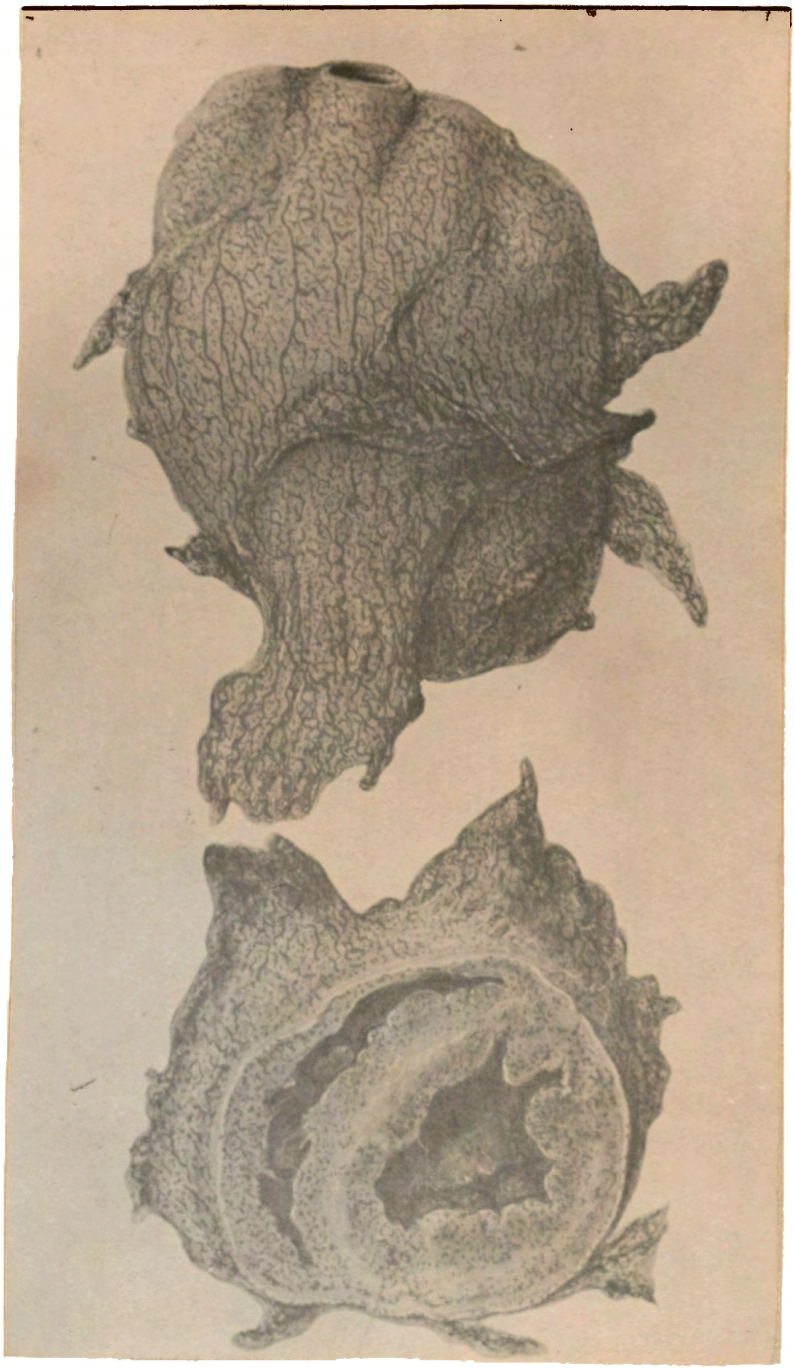


Plate 13

Extensive injection of the myocardium through the v.scular bed established by oper.tion. New blood vessels c n be seen. (After Beck and Tichy.)

Plate 14

Fig. 1. On the left is roentgenogram of the base of the heart with the heart in situ in the chest after injection of the coronary arteries. Severe constriction of the main left coronary artery has led to only a slight increase in size of the basal anastomoses which are here principally around the superior vena cava. On the right is roentgengram of the base of the heart showing the marked enlargement of the intercoronary communications as marked by numbers which correspond to the numbers in Fig. 2.

Fig. 2. Diagram showing anatomic connections between main coronary and between coronary and extracardiac arteries. R.C., right circumflex; L.C., left circumflex; L.D.R., left descending ramus; S., septal; P., pericardial; I.M. internal mammary; R.I., right innominate; I., intercostal. Note the fact that the extracardiac anastomoses in the normal heart are between the small auricular branches of the circumflex arteries and the extracardiac arteries. The common sites of anastomoses are indicated by the numbers in descending order of magnitude. (After Sautz and Beck.)

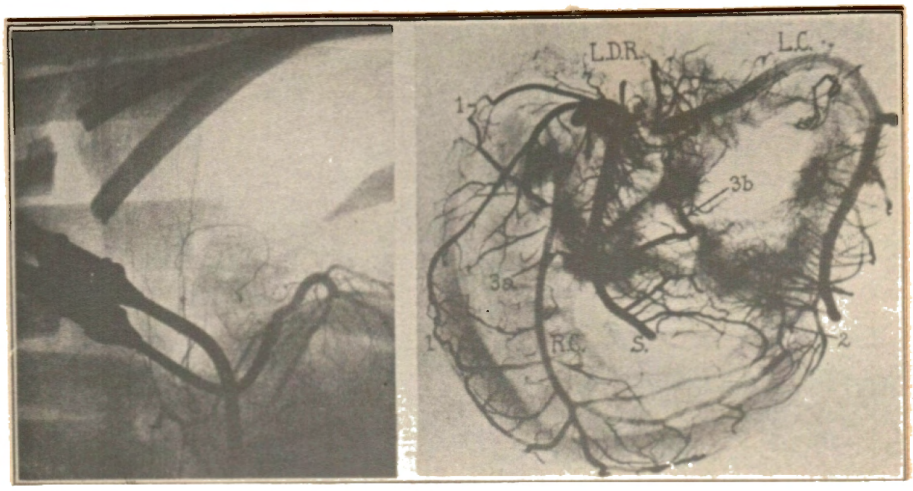


Fig. 1

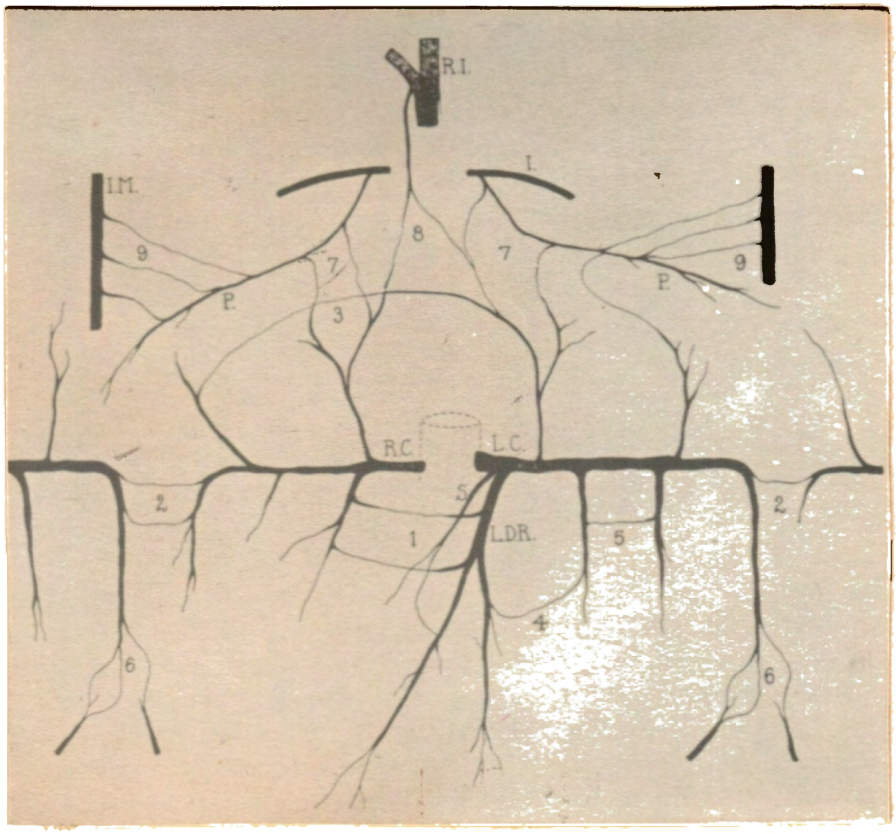


Fig. 2
Plate 14

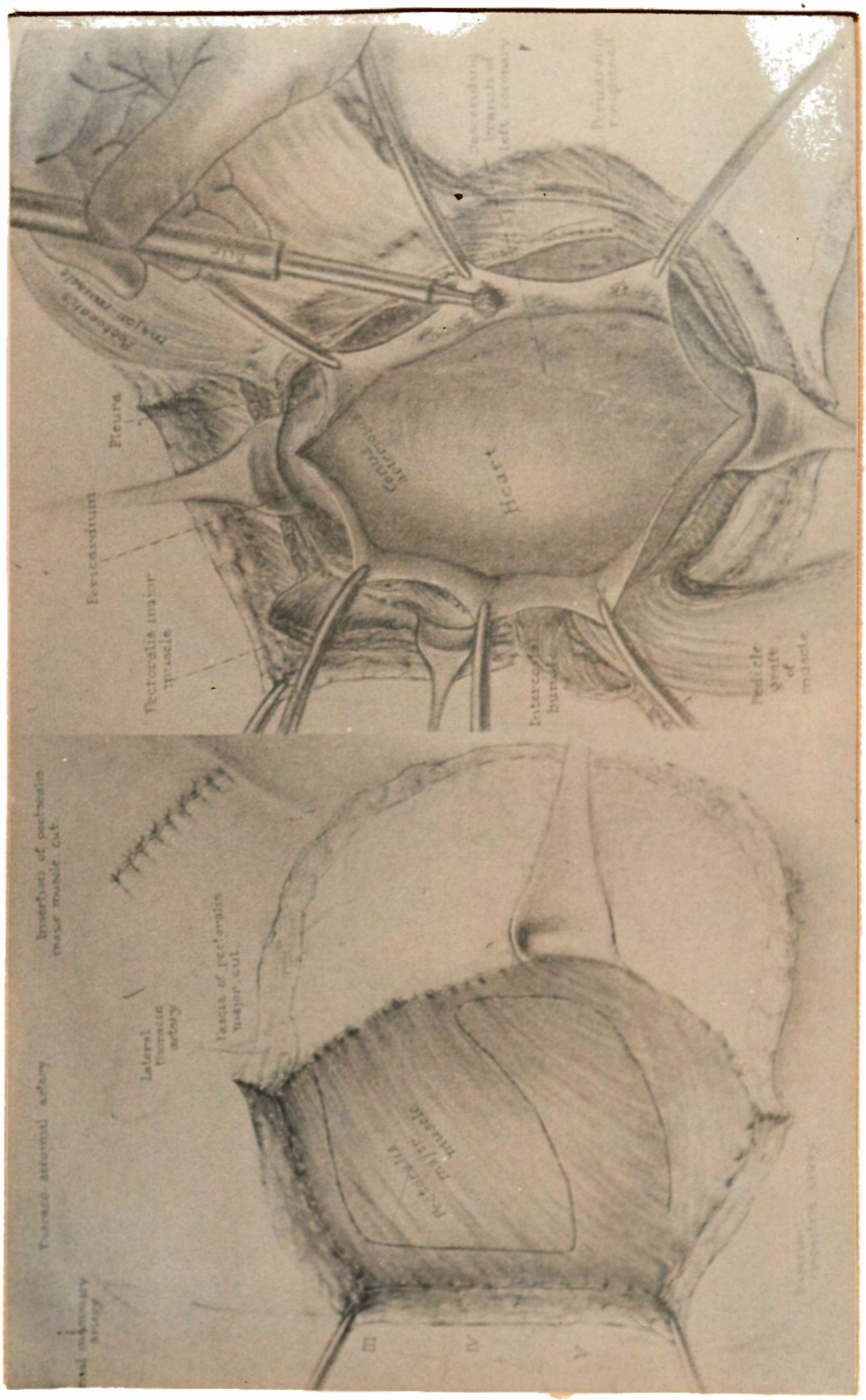


Plate 15

Steps in Beck's first coronary operation. (After Beck)

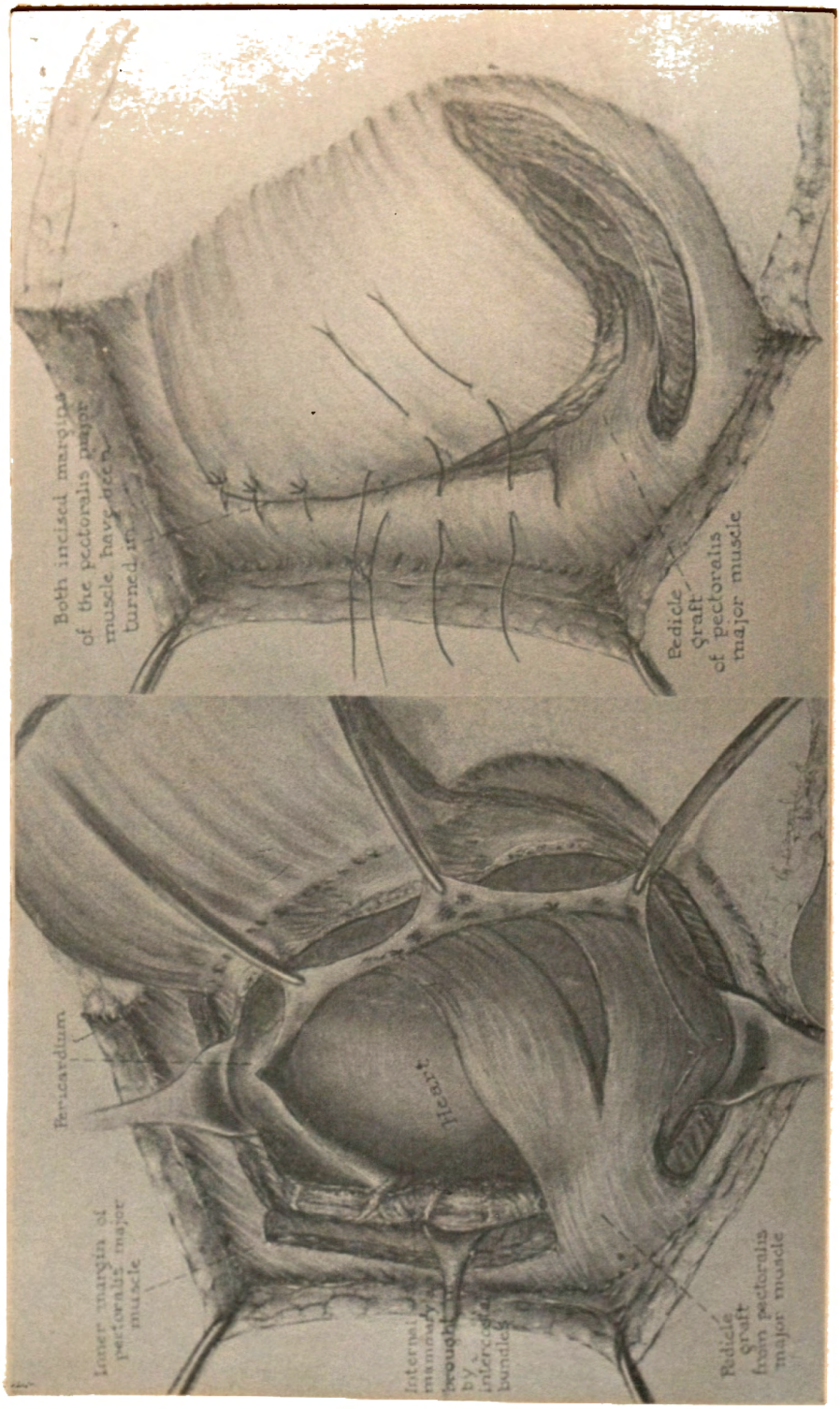


Plate 16

Steps in Beck's first coronary operation. (After Beck)

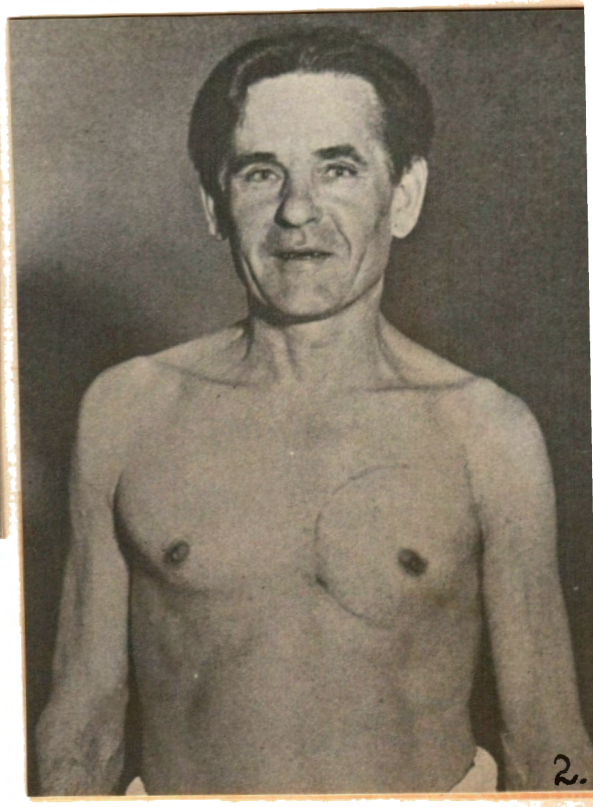


Plate 17

Fig. 1. Altered method of applying pedicle graft of pectoralis major muscle in Beck's subsequent cases.

Fig. 2. Photograph of Beck's first patient three and one half months post-operatively. (After Beck)

Table 1. Summary of the cases which did not survive the coronary operation.

Patient	Survival Period (Days)	Cause of Death
2	6	Thrombosis of abdominal aorta
5	2	B. Welchii mediastinal infection
7	4	Cerebral thrombosis, bronchopneumonia
8	1	Coronary thrombosis
10	2	Sudden death; aneurysm posterior left ventricle
12	9	Pneumonia
14	5	Pneumonia
16	1	Sudden death; advanced coronary artery disease
29	2	Pneumonia and pleural effusion
30	1	Sudden death; advanced coronary artery disease

Table 2. Summary of the twenty cases which survived the coronary operation and were able to leave the hospital.

Case	Period of Survival Yr.	Mo.	Days*	Cause of Death	Clinical Result of Operation
1	5	3	18		Good
3	2	5	29	†Inanition; Simmonds' disease	Poor
4	4	10	22		Fair
6	4	9	0		Good
9	4	4	25		Good
11	4	3	9		Good
13		5	29	Cardiac failure; myocardial degeneration	Poor
15	3	10	6		Good
17	3	6	13		Good
18	3	6	10		Good
19	3	5	28		Good
20	2	2	26	†Cerebral hemorrhage	Good
21	3	4	1		Good
22	3	4	1		Good
23	3	2	25		Good
24	1	2	25	†Coron. thrombosis	Poor
25	3	1	7		Fair
26	2	10	1		Fair
27	2	9	27		Fair
28	2	9	18		Good

*Time as of June 1, 1940

†Post-mortem examination performed (After Feil and Beck)

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