Coronary insufficiency may be defined as an unfavorable balance between the actual amount of coronary blood flow and that quantity required for adequate nutrition of the myocardium. This disparity between supply and demand is reflected clinically in a picture varying from the asymptomatic to the agonizing pattern of angina pectoris. From the viewpoint of pathology, the effects of the inadequate coronary circulation in some cases may cause no discernible damage, in others a discrete necrosis of the subendocardial tissue, or in still others a massive transmural myocardial infarction.

Actually, therefore, the term coronary insufficiency deserves further descriptive expression which qualifies its degree. To this end, Master et al. have employed the term "acute coronary insufficiency," designating a transient and partial decrease in blood flow in a segment of a coronary vessel and differentiating this condition from the prolonged coronary arterial insufficiency which results in massive infarction. Even the use of such qualifying adjectives as "acute," "chronic," "transient," and "prolonged" has failed to counteract the basic confusion which follows the application of the generic term "coronary insufficiency" to a clinicopathologic state of such varying proportions. The confusion is compounded when the additional terms of "thrombosis" and "occlusion" are used to imply an ability clinically to recognize the exact precipitating cause of decreased coronary blood flow when such diagnostic acumen does not exist.

For the purposes of this discussion, the term "coronary insufficiency" shall be considered to refer to a chronic imbalance between coronary blood flow and the nutritional demands of the myocardium usually manifested by a previous or by repeated myocardial infarctions, and by continuing symptomatic disability.

Approximately two-thirds of all heart disease is the result of actual or
relative failure of the coronary circulatory system. In 1949, in the United States, heart disease was responsible for 518,568 deaths, of which 313,757 (60.4 per cent) were attributed to arteriosclerotic heart disease. The next most frequent cause of death was cancer, responsible for 206,325. Master estimates that approximately 800,000 new cases of myocardial infarction appear annually in the United States.

In the vast majority of instances, impaired coronary blood flow results from varying degrees of sclerotic occlusion of the coronary vessels. The causes and speed of progress of this disease are determined by metabolic and perhaps endocrine issues which, for the present, remain obscure. The logical and ultimate solution to the problem of vascular sclerosis must rest with those measures which inhibit its development, and yet in that critical field all prophylactic efforts have been most disappointing. This fact, and the recent dynamic increase in the morbidity and mortality of the disease, particularly when it is referred to the coronary circulation, have been the constant stimulus for newer efforts in treatment.

An understanding of the cardiac pathology and abnormal physiology consequent to the development of sclerosis of the coronary arteries is essential before the role of surgery as a therapeutic tool can be considered fully. Coronary arterial sclerosis most commonly is the result of an atheromatous subintimal degeneration of one or more of the major coronary vessels. Although it occurs in all age groups, it is more frequent in the latter half of life. The actual obstruction to coronary blood flow may arise from the intrinsic atherosclerotic process which eccentrically or annularly elevates the endothelium, or from an associated superficial luminal thrombosis or an intramural hemorrhage. The obstructing lesions almost invariably are limited to the epicardial (visible) coronary vessels and generally occur within 3 to 4 cm. of the coronary ostia. Macroscopically, the involved arteries may not reveal their seriously deranged internal structure. However, they may be greatly thickened and hard. The microscopic changes are limited chiefly to the subintimal and intimal layers where any combination of necrosis, hemorrhage, ulceration, calcification, or connective tissue proliferation may be seen. Medial sclerosis also may involve these vessels and reduce or obliterate their lumina.

Established concepts have related coronary blood flow to the anatomic distribution and the caliber of the vessels, the size of the vascular bed, the head pressure in the arterial system, the vascular resistance, and the venous back pressure. All of these factors are exquisitely interrelated but which one exerts the predominant influence under various normal and abnormal conditions is unknown. It is apparent immediately that the effective caliber of the vessels and the size of the vascular bed are adversely altered by coronary artery atherosclerosis with a consequent reduction in blood flow. However, presumably, there is no fixed relation between this reduction and the degree to which the caliber of the vessel has been reduced. Other factors related to the arterial and venous pressures and vascular resistance are brought to bear which affect considerably the relation of vessel size to blood flow.
The ultimate result of atheromatous narrowing of a coronary arterial segment is modified primarily by the anatomic distribution of the major vessels. Unrolled, fixed, and injected human hearts reveal three basic patterns. In individuals with a predominant right coronary artery, this vessel supplies the right ventricle, the posterior wall of the left ventricle, and the posterior portion of the interventricular septum. In those in whom the left coronary artery is predominant, the left ventricle, the entire interventricular septum, and the posterior portion of the right ventricle are supplied by that vessel. With the balanced type of coronary circulation, each ventricle is supplied by its respective artery. Since atheromatous changes are most common in the left coronary artery, it appears that the potential gravity of coronary artery disease is predetermined at birth and is increased significantly in the 20 per cent of the population who are thought to have a predominant left coronary circulation (Fig. 1).

The end result of coronary arterial sclerosis is further modified by the normally existing but minute intercoronary arterial vessels which function as a collateral system. Although ranging in size from below 40 up to 180 micra, they are capable of considerable dilatation. Actually, these intercoronary vascular communications make up an anastomotic network of considerable size and when artificially or naturally overdeveloped, increase the physiologic size of the vascular bed of the myocardium. While it has been presumed by some that they do not increase the total blood flow quantitatively, they certainly provide a means for the more efficient distribution of blood to areas that otherwise would have been denied adequate nutrition. The circulation provided through these collateral vessels may prevent necrosis of the myocardium when the primary source of blood flow has been gradually and chronically reduced. Even when this is not possible initially because of the acuity or extent of the occlusive process, the collateral circulation may increase locally and reduce the size of the anoxic area. This partial response, however, may fail fully to equalize blood supply and demand, and then the area remains ischemic, a potential focus for the initiation of a fatal ventricular fibrillation. When large portions of the coronary arterial system are involved by the obstructing progress and the collateral system is generally inadequate to supply all myocardial areas, extensive fibrosis results with ultimate disabling or lethal effect upon cardiac output which is expressed usually as left ventricular failure.

However, it constantly must be realized that there normally exist at least two natural mechanisms of myocardial nutrition other than the coronary arterial system. The first, and more important, is the extensive system of vascular communications between the lumina of the various cardiac chambers and the myocardial capillary bed. This represents a vestigial remnant of the original nutritive system and the spongy structure of the primitive heart such as may be seen in the fishes and in certain of the amphibia. The myocardium in these species receives the major portion of its nourishment directly from the blood which it propels and in which it is bathed. Coronary arteries may be rudimentary or absent.
A similar condition is seen in the heart of the 5 weeks old human embryo.\textsuperscript{11} This mechanism, in part, persists in the adult mammalian heart, but comparatively is of much less importance. However, in some instances it may be capable of maintaining life and activity (even in man) in the presence of complete chronic obliteration of both coronary arteries.\textsuperscript{12}

Figure 2 clearly shows the loose and apparently disorderly arrangement of the intramyocardial vascular channels of the human heart. This is exactly the sort of set-up which logically might have been expected in consequence of its phylogenetic origin. The lack of clear-cut anatomical differentiation between these various vascular elements suggests that naturally or deliberately stimulated alterations in the size of certain of these purely artificially differentially designated vascular channels surely must extend into and involve the others. Thus, we may presume confidently that a demonstrated over-all augmentation of the intercoronary arterial collateral vessels will be attended by a similar overdevelopment of the other blood pathways including those which communicate directly with the cardiac lumina. This phenomenon undoubtedly enhances the potential myocardial nutrition which may be derived from this source in a major degree.

![Figure 1](attachment:image.jpg)

FIGURE 1: Demonstration of the varying types of coronary arterial distribution. (Schlesinger, Gregg's Coronary Circulation In Health And Disease, courtesy of Lea & Febiger.)
The second natural source of auxiliary nourishment of the heart is the arteriolar and capillary vascularity existing in the junctional tissue at the base of the heart (junction of the visceral and parietal pericardial reflections). These vessels originate from various intercostal, bronchial, esophageal, and other mediastinal arterial branches. When, due to disease (or surgical trauma), adhesions develop between the visceral and parietal layers of the pericardium, a new capillary vascularity from this source may also become applied to the surface of the heart. This may be considered as forming part of an augmented mediastinal vascular bed. In the presence of a general dilatation of the minute intramyocardial vessels (intercoronary communications), anastomotic communion between the mediastinal and the general myocardial capillary bed becomes free. This concept has been presented previously.13

Therapeutic Objectives

The major and logical objective in the treatment of chronic occlusive vascular disease of the heart is correction of the imbalance between the existing blood flow and the nutritional needs of the myocardium. The most obvious method of accomplishing this purpose would seem to involve augmenting the quantity of blood delivered per unit of time by the coronary circulation and improving its distribution throughout the myocardium. Alternatively, blood might be admitted to the myocardial vascular bed by another route. Less desirable would be a method of reducing the nutritional demands of the myocardium so that the limited existing supply might become more nearly adequate for its needs. Either approach to the problem, effectively carried out, should result in the disappearance or significant modification of the manifestations of the disease. When the major objective is beyond realization, modification of the clinical manifestations of the disease without alteration of the basic pathophysiology may become a reasonable and acceptable goal of therapy.

The surgical procedures devised and practiced for the relief of coronary insufficiency may be classified according to the objectives they seek to accomplish. Measures are available which are capable of modifying (1) the manifestations of the disease only, and (2) the actual degree of coronary insufficiency. The latter techniques are divided further into those which diminish the demands of the myocardium, and those which increase the physiologic flow through the coronary vascular bed.

1. Measures Which Modify the Manifestations of Coronary Insufficiency

The painful impulses which arise from a chronic imbalance between coronary blood flow and myocardial demand may be blocked by destruction of the sympathetic pathways which mediate pain from the heart. This may be accomplished by postganglionic resection of the upper dorsal and lower cervical sympathetic chain, by division of the preganglionic fibers to these same areas, or by paravertebral injections of alcohol. Obviously, these measures have a limited but sometimes worthwhile applicability in this disease.
2. Measures Which Modify the Degree of Coronary Insufficiency

(a) By Diminishing Myocardial Demand. Reduction of cardiac work and hence diminution in the nutritional demands of the myocardium have been accomplished by reducing the basal metabolism by total thyroidectomy, by ligation of the arteries to the thyroid gland, and by destruction of thyroid function by roentgen therapy or the administration of radioactive isotopes of iodine.

(b) By Increasing the Effective Flow Through the Myocardial Capillary Bed. This may be accomplished by bringing a new source of vascularity to the myocardium, and/or by stimulating an overdevelopment of the intercoronary collateral circulation.

The intrinsic vascularity of the heart may be increased either at a superficial level or throughout all the layers of the myocardium. Attempts have been made to apply a new source of blood supply to the epicardium by omentopexy, myopexy, pneumopexy, the superficial application of an open excluded loop of vascularized jejunum denuded of its mucosa, and the intrapericardial insufflation of irritants. A variation of these procedures designed to vascularize the myocardium beneath the epicardium has been suggested by Vineberg\(^4\) who has recommended the implantation of a bleeding internal mammary artery into a tunnel created within the thickness of the left ventricular wall. An increased vascularity of the deeper myocardial layers following this procedure is thought to arise from proliferation of small collateral branches of the artery or from a terminal luminal opening.

Beck\(^18\),\(^19\),\(^20\),\(^21\) has developed a method of utilizing the coronary sinus and its tributary venous channels as an auxiliary system for the distribution of arterial blood throughout the full thickness of the myocardium by establishing retrograde flow from the ascending aorta by way of a vascular graft to the coronary sinus. At a second operative stage, the sinus is incompletely ligated at its ostium. This procedure has been modified by Kralik\(^22\) who advises a direct lateral anastomosis between the thoracic aorta and the coronary sinus. At the suggestion of Cowley,\(^23\) we have been successful both in the laboratory and clinically in overcoming the need for a second stage with either of these techniques by applying activated cellophane about the terminal end of the coronary sinus so that its progressive sclerosing action gradually obliterates the lumen, thereby assuring the retrograde passage of arterial blood into the venous channels. However, long term studies by Eckstein et al.\(^24\),\(^25\) and by Bakst et al.\(^26\),\(^27\),\(^28\),\(^29\) have shown that such retrograde flow is abolished in experimental animals within three months. Ultimately, the venous radicles of

\(\text{Figure 2A: Sectional view of the structure of the ventricle of a frog (Rana pipiens).}\)

\(\text{Figure 2B: Enlarged segment showing spongy structure of frog ventricle. The coronary arterial system is relatively primitive in this species.}\)

\(\text{Figure 2C: Sectional view of the structure of the ventricle in a 5 weeks old human fetus. (Keith, Human Embryology And Morphology, courtesy of William Wood and Company, after His).}\)

\(\text{Figure 2D: Diagrammatic representation of the loose and apparently disorderly arrangement of the intramyocardial vascular channels in the human heart. (Gould's Pathology Of The Heart, courtesy of Charles C Thomas).}\)
the coronary sinus become obliterated completely. However, in the meantime, a 4 plus overdevelopment of the intercoronary collateral vessels will have been stimulated (Figs. 3A, B, C, D). In our opinion, the prolonged benefits observed following these operations may be attributed largely to this effect.

Ligation of the coronary sinus or of its major tributary (great cardiac

Figure 3A: 175X magnification of interfascicular space in a 45 year old patient showing normal sized intercoronary arterial channels.—Figure 3B: 80X magnification of interfascicular space in a 48 year old patient who had 5 years previously suffered from infarction, and who died 18 days after the first stage of a Beck II procedure. Note great dilatation and increase in the number of the intercoronary channels.—Figure 3C: 80X magnification of interfascicular space in a 46 year old patient who died 5 days after the second stage of a Beck II operation. Note great dilatation of intercoronary collaterals.—Figure 3D: 80X magnification of interfascicular space in a 54 year old white male who died 3 months after the second stage of a Beck II operation. Note tremendous dilatation of intercoronary vessels amounting to a pseudo-angiomatous change. (Bailey's Surgery Of The Heart, courtesy of Lea & Febiger).
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vein) has been advised and practiced\textsuperscript{30, 31, 32} as a means of altering the venous back pressure and the head pressure in the arterial system in order to stimulate the size and efficiency of the intercoronary collateral system. In our experience (experimentally), this leads to a 2 plus overdevelopment of these vessels.

\textit{Discussion}

Destruction of the sympathetic innervation of the heart is a palliative measure designed to relieve the pain of coronary insufficiency when it is easily provoked and incompletely benefited by nitroglycerin. Although contrary claims have been made, there is no satisfactory evidence that this method of treatment alters the size of the vascular bed by dilating the arterial or capillary system. The improvement in functional capacity which results in over half of the patients more properly may be attributed to an observed significant decrease in the incidence and severity of bouts of anginal pain. Serious objection has been raised to the modification of this symptom without altering its basic cause since it may serve as a warning against continued and unwarranted activity.

Diminution in thyroid function brings about an over-all decrease in bodily metabolism and, hence, in the demands for myocardial nutrition. Thus, an incompetent coronary circulation may be enabled to meet its minimized physiologic obligations. However, this form of surgical treatment implies the creation of an abnormal metabolic state with disadvantages potentially as disturbing as the disease state it is designed to alleviate. The conversion of vibrant human endeavor to a state of sluggish inactivity is a matter of grave concern, particularly when it cannot be regulated without difficulty and danger. Myxedema, in itself, is a cause of heart disease. Furthermore, presumably hypothyroidism may increase the rate of progress of the established sclerotic processes by its effect upon cholesterol metabolism. Even if this rate is not significantly augmented, the inexorable natural progression of the disease eventually must reduce the coronary blood flow below the level of the reduced myocardial demands. In a strict sense, artificially induced hypothyroidism is but a palliative measure sharply limited in effectiveness by the serious proportion of its side effects. When truly indicated, this form of treatment would seem to be accomplished with less initial risk by the use of radioactive isotopes of iodine than by surgical techniques.

Surgical measures designed to increase the size and rate of the flow through the myocardial vascular bed are best termed \textit{revascularization techniques}. The theoretical potential benefit to be derived from these procedures must be evaluated in relation to the depth and the area of the myocardium receiving the additional blood supply from the new vascular channels, and to the degree to which the natural intercoronary arterial communications become expanded.

The term \textit{surface revascularization} applies to those surgical measures devised to bring an additional arterial blood supply to the epicardium. Included in this group are omentopexy,\textsuperscript{33} myopexy,\textsuperscript{34} pneumopexy,\textsuperscript{35} and jejunopexy,\textsuperscript{36} each entailing the grafting of richly vascularized living tis-

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sue directly upon the epicardial surface of the ventricle. Arteriolar, or at least capillary, communications develop between the vascular bed of the grafted tissue and that of the most superficial layer of the ischemic ventricle.

The intrapericardial instillation of a powdered silicate produces new arteriolar channels which arise within the inflammatory adhesions which develop between the pericardium and epicardium and, therefore, also is a technique for surface revascularization. Moreover, the work of Schildt, Stanton, and Beck clearly indicates that powdered asbestos instilled into the pericardium also stimulates at least a 1 plus generalized enlargement of the intercoronary collateral vessels.

Critical questions arise in considering surface revascularization concerning whether the obtained increase in the size of the vascular bed is effective quantitatively, whether it continues to function physiologically after it has been established, and finally, whether it has been applied to vital areas of the myocardium. Although new vascular communications between the graft and the superficial layers of the heart can be demonstrated with dye or radiopaque material, reasonable doubt remains concerning the quantity and direction of blood flow through them. Even the firm proof of a flow of arterial blood through these arteriolar communications would not negate the criticism that perhaps only the superficial layers of the myocardium might be revascularized thereby.

The common observation of isolated subendocardial necrosis occurring in the absence of overt disease of the more superficial layers of the myocardium and of the visible coronary vessels emphasizes the limited protection provided by adequate circulation to the epicardial surface alone. This is not surprising from an anatomic point of view. Fundamentally, the coronary vascular bed is segmental in character since the normal subdivisions of the three main arteries terminate independently in specific layers of spiral musculature and function as end-arteries. A lash of coronary arterial branches is found between each pair of adjacent layers of myocardium, but as groups these are not in free communication with one another. Under such circumstances of anatomic distribution, it is apparent that an increase in the vascularity of certain specific myocardial segments need not increase the vascularity of others.

One, therefore, may question the total amount of benefit so provided unless there is a coincident overdevelopment of the intercoronary collateral system which is capable of distributing some of this new blood to all myocardial layers.

Physiologically, it would appear that the deeper layers of the myocardium are more urgently in need of revascularization than are those close to the epicardium when sclerotic coronary arterial disease is present. Gregg and DePalma have pointed out that the vessels embedded within the deeper myocardial layers are subjected to a higher intramyocardial pressure during systole than are the more superficially placed channels. This implies a greater likelihood of functional impairment to flow and consequently of subendocardial necrosis, particularly when disease of the
vessels is already present.

In spite of theoretical weaknesses in these concepts, attempts to promote extracardiac vascular communications have prolonged life and reduced mortality in the experimental animal when they have been used before or during partial occlusion of a coronary artery. These procedures have reduced the area of induced infarctions, and have enhanced the size of the collateral system. Applied clinically, these same methods have produced encouraging results in decreasing the morbidity and mortality of coronary artery disease. Conceivably, the benefits that have accrued may be based upon the extracardiac vascular communications themselves. However, they may have been accomplished mainly by virtue of an effective augmentation of the size of the intercoronary arterial system and its ability to decrease the threshold of ventricular fibrillation by a more efficient and judicious distribution of blood throughout the myocardium.

Schildt, Stanton, and Beck have indicated that the use of a powdered silicate, asbestos, applied intrapericardially stimulates an expansion of the intercoronary communicating system which was not observed with other substances tested or with other methods of simple surface revascularization. Because only by the existence and function of such a distributing system can blood from a new source be distributed uniformly to the myocardium, and because of the relative ease and safety of the operation, pericardial poudrage probably remains the most satisfactory of the surface revascularization techniques.

The implantation of the bleeding end of the mobilized left internal mammary artery into the thickness of the left ventricular wall has been classified as a surface revascularization technique although the additional blood supply is extended into the deeper myocardial layers. The operation has been advocated by Vineberg on the premise that direct vascular union will take place between the branches and end of the bleeding implanted artery and the smaller tributaries of the coronary arterial system. At the same time, it has been proven satisfactorily by all investigators that this implantation does not produce a purposeless hematoma of the myocardium.

Again the question arises whether this technique results in a quantitatively effective increase in the size of the coronary vascular bed. Actual communications between the systemic artery and the coronary arterial system have been demonstrated by Vineberg in 60 per cent of the experimental animals. However, in our own laboratory, grave doubt has arisen as to whether these anastomotic channels are adequate to provide a meaningful increase in blood flow to the myocardium. Flow studies during life indicate that not more than 2 to 4 cc. of blood per minute are distributed through such an implanted vessel to a relatively localized region of the anterior portion of the left ventricle. This implies that while the Vineberg procedure may benefit ischemia which is limited chiefly to that area of the myocardium, it is relatively inadequate in altering the end results of sclerotic disease involving the circumflex branch of the left coronary artery or the right coronary artery. Furthermore, the size of...
the lumen of the implanted portion of the internal mammary artery ultimately seems to undergo progressive diminution due to intimal hyperplasia and medial thickening (Fig. 4A, B).

Pratt\(^{40}\) first demonstrated that rhythmic contractions of the extracorporeal heart of a cat can be sustained by retrograde perfusion of blood into its capillary bed by way of the coronary sinus. Roberts\(^{47}\) proposed clinical application of this observation, and Beck and his associates\(^{18,19}\) developed a satisfactory two-stage technique applicable both in the experimental animal and in man. Basically, the first operative stage consists of establishing an anastomosis between the descending thoracic aorta and a contiguous portion of the coronary sinus by the use of an autogenous vascular graft. At a second stage, when the anastomotic stomata are well healed and the tributaries of the coronary sinus have become adjusted to an increased intraluminal tension, the coronary sinus is ligated incompletely at its terminus. Thus, a retrograde flow system is set up by which arterial blood passes directly from the aorta into the myocardial capillary bed by way of venous radicles.

Kralik\(^{22}\) has recommended a technical modification in which a direct lateral (side-to-side) anastomosis between the descending aorta and coronary sinus is accomplished without benefit of an intervening vascular graft. This enables one to utilize a smaller arteriovenous fistula since there is less danger of thrombosis of the anastomosis. The small size of the

![FIGURE 4A](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21263/)

**FIGURE 4A** Photomicrograph of first portion of implanted internal mammary artery in a dog after 6 months. Note great reduction in size of residual lumen due to intimal hyperplasia and medial thickening. (Bakst et al, unpublished work).—**FIGURE 4B**: Similar view of first portion of implanted internal mammary artery in another dog after 6 months. Note great reduction in size of lumen. (Bakst et al, unpublished work).

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created stoma avoids the possibility of high output failure sometimes encountered in these cases since a large arteriovenous fistula may persist if the ligature cuts through the sinus permitting recannulation of its lumen.

More recently, in our hands, a further technical modification based upon the gradual sclerosing effect of activated cellophane apparently has eliminated the need for a second stage operation to reduce the size of the terminus of the coronary sinus.

The term total revascularization of the heart is applied to the Beck II procedure and its various modifications inasmuch as these measures attempt to make available additional blood supply to its total myocardial mass. The revascularization of the left ventricle, which is the primary objective, extends in part to the right ventricle as well. Initially, arterialization of the coronary sinus establishes a retrograde perfusion of the coronary veins with arterial blood. It has been demonstrated experimentally by Bakst and associates\textsuperscript{26, 27, 28, 29} in our laboratory, and by Eckstein and Leihninger,\textsuperscript{24, 25} independently, that this arterial blood entering the venous system actually does traverse the capillary bed of the heart. A great reduction in mortality, a decrease in the frequency of ventricular fibrillation, and minimal myocardial infarction have been demonstrated by Beck et al.,\textsuperscript{20, 48} by Angulo et al.,\textsuperscript{19} and by Bakst et al.,\textsuperscript{28} when arterialization of the coronary sinus precedes experimental ligation of a large coronary arterial branch in dogs (Fig. 5A, B, C).

However, it has been demonstrated also that even after ligation of a large coronary arterial branch the retrograde perfusion of arterial blood through the capillary system ceases in the experimental animal after about three months. This is brought about by progressive enlargement of the intercoronary collateral arteries which now bring blood to the area of myocardium which has been deprived of its normal source of blood supply. Later, the smaller tributaries of the coronary sinus system develop medial and intimal hyperplasia and frank thrombosis in response to the persistently elevated pressures (Fig. 6A, B). Ultimately, these changes cause obliteration of the venous vascular channels. From an anatomic standpoint, this development is to be expected since the venous capillary bed, in contrast to the arterial, is not protected by a system of vessels of progressively decreasing caliber but is derived almost directly from large veins and, therefore, bears the full brunt of any significant increase in pressure within these major vessels (Fig. 7A and B). Also, there are intrinsic differences in the structural make-up of arteries and veins which differentially influence their ability to long tolerate arterial pressures.

The ultimate benefit to human patients from total revascularization, therefore, cannot depend upon continued retrograde capillary perfusion. Indeed, it appears to be related more accurately to the overdevelopment of the intercoronary collateral system, and, in part, to the remarkably enhanced extracardiac vascularity which is derived from the pericardial and mediastinal adhesions following surgery. Characteristically, both in the experimental animal and in patients, the produced adhesions can be demonstrated to bear a maze of sizable blood vessels when the anastomosis be-
Figure 5A: Diagramatic illustration of intracardiac myocardial circulation showing a "shunt" between the arterial (light) and the dependent (depleted) blood within the myocardial circulation during hypoxia. The myocaridal blood within the dependent, essentially under hypoxic conditions, must contract essentially under hypoxic conditions. The myocaridal arterial blood within the dependent arterioles brought about by the creation of a small amount of retrograde flow as established has been shown to be transient.

(Bailey et al., J. Thoracic Surg., courtesy of C. V. Mosby Co.)

Figure 5B: Illustration depicting excessive proprion of myocaridal blood due to stegation of blood flow caused by diminished in the effective size of the coronary arterial lumina. The myocaridal blood within the myocardial circulation is shown essentially hypoxic under hypoxic conditions.

Figure 5C: Illustration showing a "shunt" between the arteriolar (light) and the dependent (depleted) blood within the myocardial circulation during hypoxia.
between the aorta and coronary sinus remains patent, and, in comparison, are strikingly avascular if the graft has become thrombosed soon after its establishment. The functional role of these extracardiac vascularized adhesions appears to be a considerable one since bleeding extensive enough to require replacement transfusions invariably follows their separation in the experimental animal after a successful sinus arterialization. A similar situation has been discovered during reoperation upon two patients for large output failure, respectively fifteen and eighteen months after establishment of the shunt (Fig. 8 and Fig. 9A, B, C, and D).

Ligation of the coronary sinus or a major tributary without arterialization is also classified as a total revascularization technique. Such procedures greatly increase the pressure within the superficial veins of the left ventricle. However, they limit left coronary arterial inflow unpredictably. There is evidence that the chronic passive congestion so created acts as a stimulus to the overdevelopment of the intercoronary arterial system. We consider this overdevelopment to be of 2 plus degree in comparison with the 1 plus augmentation produced by poudrage with a silicate and the 4 plus produced by arterialization of the coronary sinus. The extracardiac adhesions resulting from this surgical procedure are neither as extensive nor as vascular as those following sinus arterialization.

As indicated earlier in this communication and previously, it is felt by the authors that there is no clear-cut anatomical differentiation between the various smaller intramyocardial vascular channels. There are marked ebb-and-flow currents in the course of the circulation of blood through them. It is deemed impossible to augment the size of the collateral intercoronary arterial channels without simultaneously stimulating an enlargement of all the other minute intrinsic myocardial vessels. By continuity

![FIGURE 6A](image-url)  ![FIGURE 6B](image-url)

**Figure 6A**: Extreme reduction in size of lumen of a sizable tributary of the coronary sinus in a dog 6 months following its arterialization. Note great hyperplasia of the intima as well as medial thickening. (Bakst et al, unpublished work).—**Figure 6B**: Section showing the anterior descending coronary artery (central vessel) in a dog, flanked by its two accompanying veins, 6 months after arterialization of the coronary sinus. Note extreme thickening of walls of veins with consequent reduction in the size of the lumina. (Bakst et al, unpublished work).
this process may be presumed to extend both to the luminal communications and to the normal anastomotic mediastinal vessels found at the base of the heart, and also to those found in any newly formed surface adhesions.

Reasoning thus, one must consider that all procedures which have the effect of stimulating or creating intercoronary overdevelopment not only improve the distribution of blood within the various myocardial layers and segments, but simultaneously enhance the effect of the normal or created auxiliary luminal and surface mechanisms of cardiac nutrition (Fig. 8). These secondary circulatory factors independently may be capable of maintaining life indefinitely in extensive progressive occlusive disease of the coronary arteries.

One might consider that the stimulated increase in the effectiveness of these primitive mechanisms of supplying blood to the myocardium had produced a reversion to the “amphibian” type of heart. This raises the question of how useful an amphibian heart would be to a man. Certainly the heart of even the most vigorous amphibian is less competent, especially for the performance of prolonged or violent activity, than is the normal mammalian heart. However, in individuals with coronary arterial disease the existing mammalian heart is markedly deteriorated and its function is unpredictably subject to episodes of painful or fatal depression. In such a person, a more reliable although less efficient heart of amphibian type might well be preferable to the existing diseased mammalian one. This concept is especially applicable in this situation since

![Figure 7A](image1.png) ![Figure 7B](image2.png)

*Figure 7A:* Injection specimen showing structure of the smaller coronary arterial vessels and their relationship to the capillary bed. (Dr. Armand W. Angulo, Hahnemann Medical College, unpublished work).—*Figure 7B:* Injection study of coronary venules showing that the capillaries arise directly from sizable venous branches thus being particularly vulnerable to the effects of intraluminal pressures significantly higher than normal. (Dr. Armand W. Angulo, Hahnemann Medical College, unpublished work).
most of these patients are of middle age or older, and may be willing to accept a life of less than full normal vigor. In any event, there is nothing entailed in any of these revascularization procedures which would seem in any way capable of reducing further the diminished but remaining flow through the natural coronary channels. In other words, any new

FIGURE 8: Diagram showing what the authors believe to be the ultimate changes in the intrinsic myocardial circulation following arterialization of the coronary sinus. A, there is great enhancement in the mechanism of myocardial nutrition from the lumen due to generalized dilatation of the intercoronary arterial channels and all the rest of the intrinsic minute vascularity of the myocardium which connects on one side directly with the various cardiac lumina. B, there is an enormous increase in the natural vascularity of all adhesions forming between the surface of the heart and the surrounding tissues. C, the coronary sinus tributaries ultimately become obliterated completely. However, the created arteriovenous fistula persists. (Bailey's Surgery Of The Heart, courtesy of Lea & Febiger).
vascularization of the heart, of any type, represents a clear net gain.

The possible mechanisms by which augmentation of the size of the intercoronary arterial channels takes place have been subjected to considerable investigation. Zoll and associates have designated local or generalized anoxia of the myocardium as the chief or primary natural stimulus capable of stimulating overdevelopment of these vessels. Generalized anoxia leading to generalized augmentation of the intercoronary communications may be caused by such systemic processes as anemia, chronic pulmonary disease, or circulatory impairment due to valvular or other type of heart disease. Local elaboration of this system of vessels occurs about areas of marked local ischemia or of frank infarction (usually due to atheromatous blockage of a sizable coronary arterial branch).

However, it is felt that artificial (surgical) stimulation of intercoronary overdevelopment may be related to other factors than pure tissue anoxia. It has been suggested that the augmentation in size of these channels observed following the intrapericardial instillation of a powdered silicate may be consequent to the specific chronic irritative effect characteristic of these chemicals. This would seem analogous to the chronic pulmonary

![FIGURE 9A](image-url)

**FIGURE 9A**

*Figure 9A*: Great initial vascularity of epicardio-pericardial adhesions due to newly formed vascular loops. Subsequently most of these vessels become obliterated. (Forbus, Reaction To Injury, courtesy of Williams and Wilkins Company). *Figure 9B at left*: Distended capillary bed of myocardial surface following Beck II procedure, and primitive capillary loops forming in surface adhesions. *Figure 9C*: Direct vascular communication between the congested myocardial capillaries and these primitive loops brings about great enlargement of the latter by distention and prevents their normal involution and disappearance. They then act as "run-off" vessels tending to relieve myocardial congestion. *Figure 9D*: After the retrograde flow of arterial blood is abolished by progressive obliteration of the coronary sinus venous system, the direction of flow of blood through the markedly vascularized adhesions becomes reversed, blood then flowing from the adjacent tissues into the now ischemic myocardium.
FIGURE 10: Development of progressive cardiac enlargement and large output failure due to creation of a sizable arteriovenous fistula in the Beck II procedure. Correction by ligation of the communicating vascular graft. Serial radiograms in C.T., A, taken before arterialization of the sinus and AA, 15 months afterward, showing cardiac enlargement due to the excessively large created arteriovenous fistula. Serial radiograms in C.M., B, taken respectively before arterialization of the sinus, BB, 7 months afterward, C, 18 months afterward, and finally CC, 3 months after ligation of the communicating graft.
damage caused by inhalation of silicate dusts. There appears to be no primary element of tissue anoxia involved in this process.

On the other hand, simple ligation of the coronary sinus creates a marked degree of myocardial congestion of passive type. Undoubtedly, some actual aggravation of the pre-existing ischemic state may be produced by the pressure of the congested vessels upon the parenchymal myocardial tissue. Frank interstitial edema also may develop. Furthermore, mere prolongation of the time required for drainage of the exhausted myocardial blood due to the partial venous obstruction probably entails some additional tissue anoxia.

However, it would seem reasonable to presume that the long maintained mechanical distention of the capillaries and other small intramyocardial vascular channels might easily bring about a permanent generalized dilatation of these structures. This process logically might become extended to

*Figure 11A:* P-A and right anterior oblique views of heart in V.F. 3 months following myocardial infarction, showing early aneurysm formation.—*Figure 11B:* Similar radiograms taken 10 months later showing increase in size of ventricular aneurysm.
Figure 12A: Artist's reconstruction, upon a photograph of a heart, of the appearance of heart and superimposed ventricular aneurysm as seen at operation in the case of V.F. — Figure 12B: Top view sketch showing method of applying first row of mattress sutures at the base of the aneurysm. The approximating clamps are not shown. By traction on the ends, the aneurysmal walls become approximated at its base. — Figure 12C: Sac being resected distal to two rows of mattress sutures; the outer row was placed in interrupted fashion. — Figure 12D: Cut myocardial edges finally oversewn. — Figure 12E: Resected specimen of aneurysm alongside an inch ruler. (Bailey's Surgery Of The Heart, courtesy of Lea & Febiger).
involve their cardiac luminal apertures, thus enhancing the size of these communications. Indeed, this was the original concept and objective of Gross and associates who investigated the effects of experimental ligation of the coronary sinus in 1937.

Furthermore, such a generalized turgid congested condition of the myocardial capillaries would seem materially to influence the development of adhesions forming upon the surface of the heart. Normally the initially very numerous proliferated capillary loops characteristic of early adhesion formation undergo prompt involution and ultimate obliteration. Unless there is a stimulus toward persistence, such as may be seen in certain chronic inflammatory or infectious states, such adhesions soon become relatively avascular. However, the existence of distended capillaries upon the surface of the heart would seem to facilitate or predispose toward excessive vascular communication by way of the forming capillary loops with the intrinsic vascularity of the adjacent superimposed or "grafted" extracardiac tissues. A tendency toward persistence of these congested "hypertensive," and undoubtedly relatively large vascular communications then must logically be expected, not only because of the increased size of the anastomotic vessels but also because they would tend to function as a partial "run-off" for the congested myocardium. In time, with progressive overdevelopment of the non-obstructed deeper myocardial venous drainage channels, the passive myocardial congestion must tend to diminish and ultimately disappear. Then, the free vascular communion between the extracardiac "grafted" tissues and the superficial layers of the myocardium might well permit reversal of the previous direction of flow so that arterial (capillary) blood might pass from the graft into the ischemic heart muscle.

FIGURE 13: P-A and right anterior oblique radiograms of V.F. taken 6 weeks following surgery. Note disappearance of aneurysmal bulge from cardiac outline and over-all reduction in the size of the cardiac silhouette.
A similar explanation may be advanced for the demonstrated great augmentation (4 plus) of the intercoronary vessels following retrograde perfusion of the coronary sinus by a created aortic fistula. However, the resultant myocardial vascular congestion is even more pronounced and persistent (Fig. 5C) due to the constant addition of an appreciable amount of arterial blood to the limited capacity of the myocardial vascular bed (at least 200-500 cc. per minute). Therefore, the physical "dilating" effect upon the size of the intramyocardial vessels due to mechanical congestion must be enhanced correspondingly. Again, once the initial retrograde transvenous perfusion becomes abolished by progressive obliteration of the tributaries of the coronary sinus, all of the enlarged luminal and surface vascular pathways, formerly run-off channels, become available for use as afferent passages or sources by which blood can reach the ischemic myocardium (Fig. 8 and Fig. 9A, B, C, D).

Both the surface and the total revascularization techniques alter the effective anatomic distribution of the coronary vessels and increase the size of the vascular bed. Their effects upon arterial head pressure, vascular resistance, and venous back pressure are either incompletely understood or unrecognized. It is apparent that a thoroughly dependable surgical procedure must influence favorably either all or the most predominant determinants of coronary blood flow. However, it is entirely reasonable that the major initial efforts in the field should be dedicated to the obvious objective of increasing the size of the vascular bed.

Surgical Experience

Because theoretically, and in the experimental laboratory, arterialization of the coronary sinus most profoundly increases the size of the coronary vascular bed, we have applied the technique clinically in a total of 71 patients. Of these, 53 have had the two-stage Beck II procedure which employs a vascular graft placed between the aorta and coronary sinus, and 18 have had the Kralik modification which eliminates the use of the graft.

There have been 8 operative deaths in the 53 patients subjected to the Beck II procedure, 6 prior to the completion of the second stage and 2 subsequent to it. In 8 of the series, the vascular graft was found to be thrombosed at the time of the second operative stage. Two patients developed high output failure within fifteen months because of the excessive size of the arteriovenous fistula so created but improved immediately after the coronary sinus was completely ligated at a third operation (Fig. 10).

There has been one operative death in the 18 patients subjected to the Kralik modification of the Beck II procedure, which creates a smaller arteriovenous fistula that in our experience has neither become thrombosed nor produced high output failure. The ability gradually to obliterate the coronary sinus ostium with activated cellophane, thus reducing the operation to a single stage technique, unquestionably will decrease further the risk and morbidity involved in the Kralik modification. All surviving patients have been clinically benefited with respect to the cardiac condi-
tion, although one developed a spinal cord complication, presumably due to too extensive mobilization of the aorta with development of a paraplegia. This generally satisfactory clinical experience certainly is related to judgment in patient selection and to the choice of the operative technique.

*Criteria for Selection of Cases*

In our present state of knowledge, it would seem that the surgical treatment of chronic coronary arterial insufficiency should be directed toward those patients whose clinical pattern implies an ominous prognosis, or whose manifestations seriously interfere with their ability to meet the requirements of ordinary living. Although occlusive coronary artery disease always implies an uncertain prognosis and the possibility of sudden death, surgery cannot be advised lightly merely because of the presence of the disease alone. However, it seems reasonable to suggest that the occurrence of a definitely established myocardial infarction is a sufficient manifestation of an ominous failure of the coronary circulation to serve as a primary indication for surgical therapy particularly when it is found in patients who have not yet reached 50 or 55 years of age and hence presumably are suffering with a greater than an average tendency toward progression of the disease.

The more common indication for surgical treatment is easily provoked angina pectoris which seriously limits ordinary activity. Since this clinical picture often is encountered just prior to the development of a myocardial infarction, it is necessary to establish the fact that the incapacitating nature of the angina pectoris has been a chronic event. Under no circumstances should surgery be considered within several months of an episode of acute myocardial infarction. Although the Kralik-Beck technique promises the most extensive revascularization, it remains the most trying procedure to withstand surgically. Hence, it is best limited to patients less than 55 years of age who have not suffered from more than two previous episodes of infarction, who are not handicapped by other severe systemic disease such as diabetes or hypertension, and who do not have more than a 20 per cent enlargement of the heart (radiographic demonstration). Furthermore, since this technique creates a fistula between the aorta and coronary sinus, it should not be applied in patients with past or present evidence of congestive heart failure, or even severe reduction in myocardial reserve.

While a lesser degree of revascularization (1 plus) is promised by the intrapericardial insufflation of a powdered silicate, this entails only a relatively simple surgical technique that can be applied through a limited extrapleural anterior thoracic incision. This is the more justifiable procedure in patients over 60 years of age who have had more than two myocardial infarctions, a greater degree of cardiac enlargement, or a history of previous congestive heart failure.

Undoubtedly there is an intermediate group of patients in whom either the Vineberg technique or the combination of pericardial poudrage and coronary sinus ligation (Beck I procedure) should be used. Where the
**Figure 14**: Experimental creation of complete heart block. A, right atrium opened under hypothermia (or more quickly at normal temperatures) after obstructing vena caval flow. Location and course of A-V conduction system indicated. B, division of common conduction (A-V) bundle. If septum is penetrated inadvertently, it is repaired immediately by suturing. C, atrial wall closed and circulation restored. Many animals die within 24 hours due to onset of acute ventricular fibrillation.
myocardial involvement is highly localized to the anterior surface of the left ventricle and the patient is not considered an adequate risk for total revascularization, the Vineberg operation may be considered. On the other hand, the Beck I technique may be used where the need for total revascularization exists but the past history of, or present, or incipient congestive heart failure contraindicates the creation of an arteriovenous fistula.

**Surgical Complications of Coronary Arterial Disease**

**Ventricular Aneurysm**—In view of the unhappy prognosis associated with a ventricular aneurysm complicating coronary arterial disease, it appears that the very presence of the lesion is the indication for its sur-

![FIGURE 15: Electrocardiographic studies of Aixala and Sanchez showing the creation of complete heart block in a mongrel dog, and its correction through the formation of an artificial bundle of kent. A, leads in the normal dog. B, extrasystoles with the knife in the left ventricle. C, complete heart block created by surgical division of the bundle of His. D, during suturing of muscle graft from left auricle to left ventricle. E, reconversion to normal sinus rhythm. (Courtesy of Drs. Aixala and Gabriel Sanchez, Havana, Cuba, unpublished material).](image)
Figure 16: Correction of spontaneously appearing heart block in C.R., a 36 year old rheumatic white male. A, lead 2 of electrocardiogram taken 2 years previously. Note normal conduction time with P-R interval of .22 sec. B, 2 to 1 heart block seen in C.R. on Nov. 10, 1954. C, 4 to 1 heart block seen in C.R. on Nov. 15, 1954. D, lead 2 taken 24 hours after surgical creation of bundle of Kent. There is no observable change in conduction mechanism. E, electrocardiogram taken 6 days postoperatively shows reestablishment of atrioventricular conduction. Note that P-R interval is .12 sec. in duration. Occasional premature atrial contraction is present. QRS complexes are not bizarre.
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gical removal. Although present experience in the successful removal of
a ventricular aneurysm is limited to the one patient (apparently the first
in medical annals) operated by one of us (C. P. B.) on the 15th of April,
1964, at the Hahmann Hospital, in Philadelphia,13,51 significant knowl-
dge was obtained in the problem of patient selection for such a pro-
cedure.

Case Report: V. F., Case No. 54-04387, a 56 year old coffee planter, had suffered a
myocardial infarction in January, 1953. Progressively increasing disability from per-
sistent anginal syndrome and the appearance of evidences of congestive heart failure
led to consideration of definitive excision of an enlarging anterolateral ventricular
aneurysm (Fig. 11A and B). A persistent diastolic gallop rhythm existed prior to
operation. At the time of surgery, a large noncrushing clamp was applied across most
of its poorly demarcated base. After placement of two rows of mattress sutures, the
sac was excised and the cut edges of the myocardial wound were approximated with
running sutures (Fig. 12A, B, C, D and E).

Comment

The convalescence was essentially uneventful. Gallop rhythm disap-
peared within one week. Marked clinical improvement ensued and has
persisted. The heart size became reduced markedly (Fig. 13).

Since no revascularization procedure was carried out, it would seem
that the observed benefit was brought about entirely by the removal of
the deleterious physiological effects produced by the paradoxically pulsating
aneurysmal sac. This experience suggests that patients with enlarging
aneurysms and those with symptoms of progressive circulatory impair-
ment with a coexisting cardiac aneurysm should have an opportunity to
have surgical intervention at an earlier stage.

As a general rule, overt congestive failure and gallop rhythm are recog-
nized as basic contraindications to cardiac surgical procedures. However,
in patients with sizable ventricular aneurysms these clinical findings fail
to respond to the ordinary measures of therapy and, although deterrents
to the successful conclusion of the operation, represent an indication
rather than a contraindication to surgical intervention.

Heart Block and Adams-Stokes Syndrome—Since the specialized group
of myocardial fibers which make up the so-called bundle of His represent
the only normal myocardial connection between the musculature of the
atria and the ventricles, impairment of its physiological or anatomical
continuity will tend to dissociate their respective functions.

While blockage of transmission of the excitation impulse through the
bundle of His and its branches may be brought about in several ways
(congenital defect, surgical or accidental trauma to the heart, involvement
by a rheumatic nodule), the most frequent cause of such interference is
coronary atherosclerosis. Either ischemia or frank infarction involving
the septum may affect the conduction system producing various types of
bundle branch block, delayed conduction, or even complete heart block.
In this latter condition, the ventricles develop an independent contraction
rhythm with a rate which may vary from 30 to 50 per minute. Mean-
while, the atria continue to contract at their own usual rate (70 to 100
per minute). In some individuals, the type or degree of block may vary
from day-to-day or week-to-week.
While those persons who have a permanent complete heart block seem to do reasonably well for prolonged periods of time, the over-all life expectancy in cases due to atherosclerotic heart disease is estimated to be between 12 and 24 months. Furthermore, these individuals experience a significant loss of reserve for vigorous activities. Perhaps this is due to an inability to increase their cardiac output on demand by an acceleration of rate. In some patients, transient asystole may occur resulting in fainting and convulsions (Adams-Stokes syndrome).

On the other hand, individuals who exhibit heart block which varies from day-to-day and from week-to-week tend more frequently to develop such episodes of transient asystole, any of which may eventuate fatally from severe depression of ventricular irritability due to the prolonged anoxia, or from the sudden onset of ventricular fibrillation.

Hence, it was deemed desirable to attempt to develop a method of creating surgically a new conduction pathway between the atria and ventricles. Anomalous congenital communications of this type are occasionally seen (bundle of Kent). Such an abnormal conduction pathway usually represents an area in which the development of the atrioventricular fibrous ring (annulus fibrosus) has been deficient so that a direct (embryonic) atrioventricular myocardial communication persists in addition to the normal bundle of His. In such cases, the observed time of atrioventricular conduction (P-R interval in the electrocardiogram) is observed to be abnormally short. Presumably this is to be explained by the passage of the excitation wave from atrial tissue directly into the continuous muscle fibers of the ventricle without need for passage through the node of Tawara (A-V node) and the common conduction bundle. It was deemed possible to create a similar communication surgically.

**Experimental Technique**

Complete atrioventricular dissociation was brought about in experimental animals (mongrel dogs) by surgically dividing the common conduction bundle as it passes along the lower interatrial septum. This was done with the venae cavae temporarily occluded under direct vision (Fig. 14A, B, C). Using this technique, we have been able to produce a complete and lasting heart block in several dogs, which we have employed in testing various grafting techniques. At a later date we have carried out procedures designed to correct the block.

Prior to the initiation of this investigation (September 1954), and unknown to us, Aixala and Sanchez in Havana, Cuba, had independently undertaken a similar study (March 1954). We in no way desire to deprive them of proper priority for this concept or for the experimental

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**Figure 17:** Technical steps in the creation of a "Bundle of Kent." A, isolation of a portion of the right atrial wall with formation of pedicle. B, oversewing of the defect and elevation of a partial thickness of the ventricular wall below the coronary artery. C, insertion of the everted atrial pedicle into the ventricular bed. Note the sutures placed around the ventricular flap to guarantee complete surface apposition of the "sandwiched" graft. D, second full thickness pedicled graft raised from right atrial wall for implantation without eversion. E, closure of second atrial incision and insertion of non-everted "isoperistaltic type" graft into a myocardial tunnel.
demonstration of its validity. Figure 15 clearly shows the restoration of atrioventricular conduction in an 11 lb. mongrel dog in which they had previously produced heart block at the same operation by the instrumental transventricular technique of Sodi Pallares.54 Our work has been directed toward producing a complete and permanent block at one operation, and then, after a suitable interval has elapsed to avoid any possibility of spontaneous remission, toward producing a permanent correction at a second procedure.

Case Report: On November 15, 1954, at Hahnemann Hospital, in Philadelphia, an operation to form an artificial bundle of Kent12 was done on C. R., Case No. 54-13211, a 36 year old white male with a heart block which varied from complete to 2 to 1 (Fig. 16A, B, C, D, E). A portion of the right atrial wall was excluded from the circulation by a noncrushing clamp technique. This elongated excluded segment was then separated from its atrial attachment except at one extremity. The residual defect in the atrial wall was closed by sutures.

FIGURE 18: Photograph of operative field in C.R. showing diagonally placed (arrows) strip of mobilized atrial wall.
CORONARY INSUFFICIENCY

Then, a selected portion of the thickness of the right ventricular wall was split and the epicardial portion was elevated. After freshening both its epicardial and endocardial surfaces, the end of the pedicled atrial tissue was placed between the two layers of the ventricular wall in "sandwich" fashion. Several interrupted sutures were placed to maintain the displacement of the atrial tissue permanently (Fig. 17A, B, C, and D). A second longer pedicled graft of atrial tissue was prepared and placed similarly but without eversion.

While no change was noted in the patient's cardiac rhythm or conduction for five days following operation, suddenly on the sixth day a nearly normal type of atrioventricular conduction developed with an increase in ventricular rate from 50 to 82 per minute. The QRS complexes presented a fairly normal over-all configuration, but the P-R interval was only .12 second. It had been .22 second in duration on examination two years previously. This very short P-R conduction time would seem to confirm the existence of a much shorter conduction path than normal, just as is seen with a naturally occurring bundle of Kent.52

Comment

These experimental demonstrations and this first report of an apparent clinical surgical success in treating the medically relatively recalcitrant and incapacitating entity of complete heart block suggest a widespread applicability of such a surgical technique in individuals with this unfortunate complication. The actual surgical procedure performed is neither shocking nor physiologically burdensome. One would not expect to encounter significant operative mortality (Fig. 18).

CONCLUSIONS

1. The surgical procedures devised for the treatment of coronary arterial insufficiency may be classified according to the objectives they seek to accomplish.
2. Measures which modify only the clinical manifestations of the coronary arterial insufficiency consist of those which destroy the sympathetic pathways mediating pain sensation from the heart by postganglionic resection of the upper dorsal and lower cervical chains, by division of the preganglionic fibers to these same areas, or by paravertebral injections of alcohol.
3. The only available method which modifies the degree of coronary insufficiency by decreasing myocardial demand is destruction of thyroid function by surgical or medical therapy.
4. Coronary insufficiency may be decreased by a variety of procedures which are designed to increase the size of the coronary vascular bed. These are best termed revascularization techniques, and represent the most physiological approach to the problem. They are classified according to their ability to increase the vascularity at a superficial level or throughout all layers of the myocardium.
5. The blood supply to the epicardium, or the layers immediately beneath the epicardium, may be augmented by omentopexy, myopexy, pneumonopexy, the intrapericardial insufflation of irritants, or the implantation of the bleeding end of the mobilized left internal mammary artery into the left ventricular wall. The intrapericardial instillation of a powdered silicate appears to be the most promising of the surface revascularization procedures.
6. The term total revascularization is applied to the techniques which
create an anastomosis between the aorta and the coronary sinus either through a vascular graft (Beck II) or directly (Kralik modification) thus establishing a retrograde flow of arterial blood to the myocardial capillary bed by way of the venous channels. Such retrograde flow is transient but great augmentation of the adequacy of the intercoronary collateral system is produced and remains. Simple ligation of the coronary sinus also may be considered as a total revascularization procedure although a lesser degree of intercoronary overdevelopment is obtained by this procedure.

7. The selection of patients for the revascularization procedures is based upon those manifestations of the disease which indicate an ominous prognosis or seriously interfere with the ability of the patient to meet the requirements of ordinary living.

8. Resection of a ventricular aneurysm represents a promising surgical development in the management of a relatively recalcitrant and untoward cardiac complication which nearly always is the result of occlusive coronary arterial disease. The first historically successful case is herein presented.

9. The development of a seemingly satisfactory and rather safe surgical treatment for complete or varying heart block is detailed with presentation of the first apparently successful human operation.

CONCLUSIONES

1. Los procedimientos quirúrgicos ideados para el tratamiento de la insuficiencia coronaria pueden clasificarse de acuerdo con el fin que se propenem.

2. Son medidas que sólo modifican las manifestaciones clínicas de la insuficiencia coronaria, aquéllas que consisten en destruir las vías simpáticas aliviando la sensación dolorosa del corazón por medio de la resección postganglionar de las cadenas superior cervical e inferior cervical dividiendo las fibras pregangliónares hacia estas áreas o bien por las inyecciones para vertebrales de alcohol.

3. El único método que existe para modificar el grado de la insuficiencia coronaria haciendo disminuir la demanda del miocardio, es la destrucción de la función tiroidea ya sea por procedimiento quirúrgico o médico.

4. La insuficiencia coronaria puede decrecer mediante varios procedimientos que tienden a aumentar el calibre de los vasos del lecho vascular coronario. Estos pueden mejor llamarse técnicas de revascularización y representan el modo más fisiológico de atacar el problema. Se clasifican de acuerdo con su aptitud para aumentar la vascularización en un plano superficial o a través de todas las capas del miocardio.

5. La provisión sanguínea al epicardio o a las capas inmediatamente debajo de él puede aumentarse por la omentopexia, la miopexia, neumopexia, la insuflación intrapericárdica de irritantes o la implantación del cabosangrante de la arteria mamaria interna movilizada dentro de la pared ventricular izquierda.

La aplicación intrapericárdica de un silicato en polvo parece ser el más prometedor de los métodos de revascularización superficial.
6. El término revascularización total se aplika a las técnicas que—crean anastomosis entre la aorta y el seno coronario por medio de un injerto vascular (Beck II) o bien directamente (Modificación de Kralik) estableciendo así un flujo retrógrado de sangre arterial hacia el lecho capilar del miocardio a través de las vías venosas. Tal flujo retrógrado es transitorio pero se produce un gran aumento de la eficacia del sistema colateral intercoronario y esto persiste. La simple ligadura del seno coronario también puede considerarse como un procedimiento de revascularización total aunque menor grado de nuevo desarrollo intercoronario se obtiene por este método.

7. La selección de los enfermos para la revascularización se basa—sobre las manifestaciones de la enfermedad que indican un mal pronóstico o sería interferencia de la capacidad del enfermo para los requerimientos de la vida ordinaria.

8. La resección del aneurisma ventricular es una adquisición quirúrgica prometedora para el tratamiento de las complicaciones relativamente recalcitrantes que resultan casi siempre de la enfermedad coronaria oclusiva. El primer caso históricamente con éxito satisfactorio se presenta aquí.

9. El desarrollo de un procedimiento quirúrgico al parecer seguro para el bloqueo completo o variable del corazón se detalla con la presentación del primer caso humano aparentemente satisfactoriamente realizado.

RESUME

1. Les traitements chirurgicaux de l'insuffisance de l'artère coronaie peuvent être classés selon les objectifs qu'ils cherchent à atteindre.

2. Certains ne modifient que les manifestations cliniques de l'insuffisance de l'artère coronaie: ce sont ceux qui détruisent les trajes sympathiques transmettant la douleur venant du coeur. Il s'agit de la résection postganglionnaire des chaînes dorsales supérieures et cervicales inférieures, de la division des fibres préganglionnaires dans ces mêmes régions ou des alcoolisations paravertébrales.

3. Une seule méthode valable modifie le degré de l'insuffisance coronarienne en diminuant les besoins myocardiques: c'est la destruction de la fonction thyroidienne par la chirurgie ou la thérapeutique médicamenteuse.

4. On peut diminuer l'insuffisance coronarienne par une série de procédés qui sont destinés à accroître l'importance du lit vasculaire coronarien. Ce sont les procédés qui sont le plus exactement désignés sous le terme de moyens de revascularisation. Ils représentent la façon la plus physiologique d’aborder ces problèmes. Ils sont classés selon qu’ils sont susceptibles soit d’augmenter la vascularisation des plans strictement superficiels, soit d’atteindre toutes les couches du myocarde.

L'instillation péricardique de silicate en poudre semble être le procédé de revascularization superficielle le plus satisfaisant.

6. Le terme "revascularisation totale" est appliqué aux procédés qui créent une anastomose entre l'aorte et le sinus coronaire, soit par greffe vasculaire (Beck II) soit directement en établissant un courant rétrograde de sang artériel vers le lit capillaire myocardique par les conduits veineux. Un tel courant rétrograde est passager, mais il produit une grande augmentation de l'importance du système collatéral intercoronarien qui persiste. La simple ligature du sinus coronaire peut aussi être considérée comme un procédé de revascularization totale, bien qu'il donne un plus faible degré de développement intercoronarien.

7. Le choix des malades pour le procédé de revascularization est fondé sur l'existence de manifestations telles qu'elles laissent supposer un pronostic sérieux, ou qu'elles empêchent manifestement le malade de mener une vie normale.

8. La résection d'un anévrysme ventriculaire représente un progrès chirurgical plein de promesse pour obtenir la guérison d'une complication cardiaque relativement récalcitrante et insurmontable, qui presque toujours provient de la thrombose de l'artère coronaire. Les auteurs rapportent le premier cas historiquement traité avec succès.

9. Ils exposent en détail le développement d'un traitement chirurgical également satisfaisant et sans risque syndrome d'Adams-Stokes complet ou partiel, avec présentation d'un cas qui semble avoir été opéré avec succès.

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