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Surgical revascularization of the myocardium

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**The Surgical Revascularization
of the Myocardium**

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INTRODUCTION

Coronary artery disease holds the dubious distinction of being the major cause of deaths and disability in the field of cardiac disorders. The prevalence of the disease seems, paradoxically, to be increasing despite mounting efforts to modify its ravaging, but insidious, course. The gravity of the prognosis of this disease has been emphasized by the disheartening reports of investigators who have followed large series of patients treated conservatively. Parker and his associates (1) in 1946, reported only a 5 year survival in 3,440 cases with angina pectoris. The prognosis becomes even more disheartening in patients who have suffered a myocardial infarction. Katz et al (2), in 1949, reported 25% of these people to be dead within two months, 50% within one year, and 75% within three years.

Basically, the offending lesion is an arteriosclerotic vascular degeneration with its resultant gradual or sudden occlusion of portions of the coronary arterial tree. Although advances have been made toward the modification of this sclerotic process by metabolic means, the ultimate solution seems remote at the present. In the interim, both

physicians and surgeons have attempted to increase myocardial vascularity, despite the natural progression of the occlusive process, by augmenting its diminished blood supply from neighboring vascular structures or by redistribution of myocardial blood from unaffected to affected areas.

The problem of surgical revascularization of the myocardium has been approached by almost as great a variety of methods as there has been surgeons interested in the problem. This thesis is not intended to be a comprehensive report on all the methods advocated, but rather, most will be mentioned but only a few in detail. The recent literature on the anatomic and physiologic principles necessarily involved in a successful myocardial revascularization will also be presented. The neurosurgical relief of angina pectoris as advocated by Danielopolu (3) in 1926 and Leriche and Fontaine (4) in 1928 will not be discussed. There still remains the question whether or not these procedures, such as stellate ganglionectomy and paravertebral block, actually increase the circulation to the myocardium. Investigations by Beck and Leighninger (5) in 1954 would suggest

they did not.

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

The presence of intercoronary arterial communications has long been established by the work of Gross(6). Their significance, however, was not established until the work of Blumgart, Zoll, Schlesinger and co-workers (7,8). Their work indicated the presence of intercoronary communications in only 10% of normal hearts. Thus, functionally, at least, the coronary arteries are end-arteries. Under the influence of certain stimuli, these communications become functional by increasing both in number and in size. The chief stimulus of this response seems to be a relative insufficiency of blood; insufficiency originating in the blood itself (anemia), in the aeration of the blood (pulmonary disease), in the myocardium (hypertrophy, in the endocardium (valvular disease), and in the coronary arteries themselves (coronary atherosclerosis). Other factors leading to the development of significant collaterals consequent to the narrowing process in coronary atherosclerosis may be: 1) metabolites acting directly on the communications or indirectly through reflex action, and 2) the lowered pressure

distal to the narrowing producing a pressure differential favoring widening of the collaterals and inflow from other arteries.

The studies by Blumgart et al (7) revealed that a decrease of from 10% to 27% of the cross sectional diameter of a major coronary artery is commonly followed by the production of interarterial anastomoses. Using pig hearts, these investigators partially ligated the anterior descending branch of the left coronary artery. Sacrifice of these animals at a later date revealed a time interval of from 10 days to 21 days between ligation of the artery and the production of functionally significant intercoronary anastomoses. Although other circulatory channels have been implicated, intra- and extra-cardiac in origin (9), the collateral circulation to the myocardium is primarily a function of these communications.

Brofman and Beck (10) have likened coronary heart disease to a "tug-of-war, or battle for survival, between the relentless occlusive process on the one hand and a compensating collateral circulation on the other". Under optimal conditions, a patient may become stable. This stability does

not represent a cessation of the occlusive process, but rather the ability of the collaterals to keep pace with it. It is to this delicate balance that surgery can add a little extra protection against an otherwise fatal change in coronary circulation.

There is no obligate relationship between the degree of reduction of coronary circulation and the resulting consequences (11). In only a relatively small group of deaths due to coronary artery disease can the cause of death be explained on the basis of the degree of reduction of coronary inflow and extensive myocardial destruction. A much larger group die due to a loss of the coordinated cardiac beat. Brofman, Leighninger and Beck (12) referred to the former as "muscle deaths" and to the latter as "mechanism deaths". These investigators, in their experimental preparation on dogs of the Beck No. 1 and No. 2 operations, formulated and enlarged upon many of the present concepts of the electrical stability and instability of the heart.

According to Brofman, Leighninger and Beck, the electric manifestation of injury to the myocardium is due to the interaction of two factors: 1) a vulnerable area of ischemic myocardium (trigger area), and 2) a

"current of oxygen differential" produced by two contiguous areas of myocardium. This current in turn is dependent upon: 1) the degree of difference of oxygenation in the "trigger" area and in the surrounding myocardium, 2) the abruptness of the transition between the two areas, and 3) the size of the "trigger" area. It can be seen, therefore, that the electrical instability of the heart is not a function of the absolute oxygenation of the myocardium, but of the oxygenation differential between two areas of myocardium. In a young man with only moderately impaired coronary arterial flow, exertion would cause a perfusion of highly oxygenated blood to all areas capable of responding with vaso-dilatation thus often setting up relative anoxia in the area supplied by the impaired coronary vessel resulting in an electrically unstable heart. In this manner do deaths occur with adequate total coronary inflow and with functional myocardium.

The reverse was demonstrated by these investigators also to be true. A coronary artery, on a dog heart made cyanotic by occluding the dogs trachea, was perfused with highly oxygenated blood with resultant fibrillation. In a patient with a generalized lower oxygenation of the myocardium, such as, congestive

heart failure, anemia, and cor pulmonale, an area completely deprived of blood supply would not have as great an oxygen differential as would be present in a similar occlusion in an otherwise normal heart, and hence a lesser degree of electric instability. By the same token, if a patient with a previously normal myocardium develops an area of ischemia, the administration of oxygen to this patient, rather than being beneficial, might raise the oxygenation of the surrounding myocardium predisposing to a greater electric instability.

These authors state that the sharper the transition between the ischemic and oxygenated areas, the higher is the current of oxygen differential and the more electrically unstable is the heart. Again using dogs, they found that multiple small coronary artery ligations produced a more completely anoxic area than large artery ligation. This is presumably due to the fact that multiple small artery occlusion more completely occludes the arterial inflow; inter-arterial collaterals bringing in enough blood to marginal areas, in the case of large artery ligation, to destroy an abrupt transition. Since 69% of coronary artery occlusions occur within the proximal 4 cm. of the main stem coronary branches (13), this factor

would appear to be favorable for the patient.

Whereas the presence of intercoronary collaterals prior to occlusion may be beneficial, Brofman and Beck (10) present a concept of "malignant" collaterals developing in fibrosed and scarred areas of healed myocardial infarction. Such an area would present a lower circulatory resistance, and preferential flow to it might serve as a functional arteriovenous fistula depriving surrounding viable myocardium full access to the oxygenated blood. According to these authors, such a mechanism would explain the clinical picture often seen in a patient with myocardial infarction who convalesces well until the tenth to fifteenth day. At this time symptoms of increased dyspnea, cyanosis, and orthopnea appear.

SELECTION OF PATIENTS

Since the proper selection of patients for these surgical procedures directly influences their statistical success, the authors reviewed allotted considerable space in their papers describing their method of such selection. The following is a composite of the ones described by several authors (5, 15, 16):

Group 1. These are the patients with

mild or intermittent angina, experiencing angina with exercise or with emotion but capable of living comparatively normal lives.

Group 2. These are the patients with progressive coronary artery insufficiency but without disability. They show an increased frequency of anginal attacks or progressive dyspnea.

Group 3. Those patients with partial or total disability but without angina at rest. Many of the patients in this group may experience angina at rest associated with emotion, straining at stool, or after a heavy meal.

Group 4. These are the patients with status anginosus.

The patients in the first three groups can be considered candidates for surgery. The cases falling in Group 4. should not be operated upon. In these cases there is insufficient myocardium remaining to revascularize. What is left is vascularized by a quantity of blood insufficient to sustain the patient through the operative procedure and the post-operative period.

The most acceptable candidate can be visualized as a lean person in the fourth or fifth decade having had one, or at most, two myocardial

infarctions. Younger patients tend to have a more rapidly progressive disease. His blood pressure should be normal or only slightly elevated. A severe hypertension would not be acceptable, especially in regards the Beck No. 2 operation in which high arterial pressures would have a deleterious effect on the coronary venous system. Ideally the patient should have no evidence of cardiomegaly or congestive failure. A slight degree, however, was acceptable by most surgeons.

Contraindications to these procedures are: a myocardial infarction within 6 months of the contemplated date for operation, severe hypertension, advanced congestive heart failure with or without cardiomegaly, status anginosus and angina decubitus, and associated conditions; such as, generalized arteriosclerosis, pulmonary disease, hepatic and renal failure.

SURGICAL METHODS

The surgical methods designed to increase the effective coronary circulation fall into three main categories:

1. Those designed to increase the extrinsic coronary collateral circulation by extracardiac communications. This includes such proce-

dures as: the production of anastamoses with such vascular structures as omentum (17,18), lung (19-21), skin (22), jejunum (23-25), and pectoral muscle (26-28); the implantation of a systemic artery, such as the internal mammary, into the wall of the left ventricle (29-33); the production of vascular adhesions by abrasion of the pericardium or by pericardial irritants (34-43); and, the ligation of the internal mammary arteries (44,45).

2. Those intended to effect a better distribution of the oxygenated blood. Among these are: the technics employing stenosis or partial ligation of the coronary sinus associated with or without arterIALIZATION of the coronary sinus (5, 14,35,36,46); chemical or mechanical de-epicardIALIZATION (39); and, the development of a direct connection between the ventricular cavity and the myocardial circulation (47,48).

3. Those procedures designed to remove the occluding lesion of the coronary artery (49,50).

GRAFTING OF VASCULAR EXTRACARDIAC STRUCTURES

This group of procedures in general represents the first attempts by surgeons to revascularize the myocardium. The first such operation was by Beck (26), in 1935, using a pectoral muscle graft

to the myocardium of the left ventricle. This was the start of experimental work by him which finally culminated in the Beck Operations No. 1 and No. 2, which will be discussed later. O'Shaughnessy (17,18), in 1936-37, reported on experimental work done on dogs using an omental graft to the myocardium. He used this cardio-omentopexy on six humans with one postoperative death (due to causes other than cardiovascular), the remaining five patients experiencing symptomatic relief of their myocardial ischemia.

In 1957, Bakst and associates (28), experimenting on dogs, used Beck's pectoral muscle graft technic. In order to measure the increased vascularity contributed to the canine coronary circulation following this procedure, they employed the Mautz-Gregg method of measuring backflow (51). This method consists of ligating one of the branches of the left coronary artery, either the circumflex or anterior descending. This artery is then cut distal to the ligature and the amount of blood drained from the distal end is measured as the backflow. In their series of dogs, the average backflow in normal controls was 1.7 cc per minute. In the dogs protected by the pectoral graft the backflow was 5.4 cc, a substantial increase.

In 1948, Kownacki et al (21) presented the results of their work with 18 dogs on which a cardiopneumopexy and lingular vein ligation was done. Their technic consisted of ligating the lingular vein and suturing the lingular lobe of the left lung to an area on the left ventricle that had been painted with a 30% acriflavine solution (a caustic and inflammatory agent). The second stage of the operation, done 2-3 weeks after the first, involved ligation of the anterior descending branch of the left coronary artery. Their results are difficult to evaluate statistically in that first three dogs died in the very early postoperative period and the next fifteen survived. The results probably represent the increased skill of the operator. Nonetheless, it would seem that considerable protection is given a dog against a sudden occlusion of a major coronary artery.

The degree to which grafts such as these actually contribute to myocardial circulation has been doubted by some authors. Gregg and Sabiston (52), in their laboratory, prepared several dogs with pedicled skin grafts to the heart. The coronary sinus was cannulated and the venous return measured. When the skin graft was clamped they observed no decrease in

coronary sinus flow. They were of the opinion that the effectiveness of such procedures as these may rest on their acting as bridges between one coronary artery and another.

INTERNAL MAMMARY ARTERY IMPLANTATION

The technic and use of this operation was first advocated by Vineberg (15,29-33) in 1946. The technic consists of mobilization of the left internal mammary artery as far distal as possible leaving about $1\frac{1}{2}$ cm. intercostal artery stumps. The artery is ligated and cut as far distal as feasible and swung into the pericardial cavity to be buried in a tunnel made in the left ventricular myocardium. Just prior to burying the artery the intercostal artery stumps are cut flush with the internal mammary and allowed to bleed freely. The artery is then buried confining the bleeding within the myocardium. The success of this operation rests on the fact that the myocardium, especially in the subendocardial layers, consists of a spongelike network of vessels. Thus, when the mammary artery is buried with a bleeding intercostal branch, a hematoma is not formed. The blood disappears immediately into this spongy myocardium. In this way, according to Vineberg, blood flows continually down the internal mammary artery and out the

opening in its wall, thereby keeping the artery open until it forms new branches which join with the coronary arterioles.

At latest publication (33), Vineberg reports having treated 45 patients suffering from coronary artery insufficiency with internal mammary implantation. In the 33 patients having no angina at rest, there were 2 deaths, a mortality rate of 6.6%. In the 12 patients suffering from angina decubitus, there have been 7 deaths, a mortality rate of 58.3%. The mortality of the entire group was 20%. Of the patients with no angina at rest, 30 were followed from six months to four and one-half years following operation. Prior to operation 23 (77%) were totally disabled. In the follow-up period, 20 (71.4%) showed clinical improvement and 23 (77%) have returned to work. Of the 10 patients with angina decubitus followed during this period, 4 showed clinical improvement and 3 returned to work.

These statistics, as presented by Vineberg, at first glance are very encouraging, a returning to work of 77% of the group with no angina at rest. Of this group prior to the operation 77% were totally disabled, the remainder of the group (23%) carrying out useful activity. If we can assume,

that of these patients capable of activity pre-operatively the majority will survive the operation without impairment of this activity, then the number of patients brought back to work must be reduced to approximately 55-56%.

The success of this operation would seem to center on the patency of the arterial implant allowing a flow of oxygenated blood into the myocardial circulation. Glenn and Beal (53) in 1950 experimenting with dogs, reported that an artery implanted in the myocardium became occluded by an obliterative endarteritis and tended to fibrose and disappear after a period of 6 weeks. Bakst et al (54), in 1955, found in 6 month follow up studies on dog hearts protected by this procedure, that the lumina of the vessels in 95% of the cases studied were obliterated by intimal proliferation. The studies by Sabiston, Fauteux and Blalock (55,56), in 1957, however, revealed patency in 60% of arterial implants in the right ventricular myocardium and patency in 92% of the vessels implanted in the left myocardium. The disparity of results found by these investigators is difficult to explain. The answer may rest on difference of technic.

INTERNAL MAMMARY ARTERY LIGATION

In 1932, Hudson, Moritz, and Wearn (9), by injecting the coronary arteries of human cadavers with a mixture of methylene blue and india ink, demonstrated communications from the major coronary vessels with the pericardiophrenic branches of the internal mammary arteries as well as the anterior mediastinal, pericardial, bronchial, superior and inferior phrenic, intercostal, and esophageal branches of the aorta. In 1939, at the suggestion of Fieschi in Italy, Zola and Cesa-Bianchi ligated the internal mammary arteries of a patient suffering from coronary artery insufficiency (cited in ref.45). After this procedure the patient experienced considerable relief of symptoms. In 1955, Battezzatti, Tagliaferro, and De Marchi (44) reported work on eleven patients treated with bilateral internal mammary artery ligation. In each, anginal symptoms were relieved as well as the electrocardiographic and balistocardiographic evidence of myocardial ischemia.

Glover et al, (45), in 1957, reported his experimental and clinical work with bilateral internal mammary artery ligation (BIMAL). These authors again demonstrated significant communications in dogs between the coronary arteries and the pericardiophrenic

artery by perfusing this artery with a mixture of Evan's blue, fluorescein, and the millicuries of radio-iodine. Applying this surgical procedure to dogs, their results were: of 14 control dogs (unprotected), 13 died after ligation of the anterior descending branch of the left coronary artery; of 19 dogs protected by BIMAL, 11 (58%) survived the subsequent ligation of a major coronary vessel.

Glover applied this procedure, clinically, on 50 patients, many of whom had had multiple infarcts and episodes of congestive heart failure. Of these, 5 (10%) died in the early post-operative period. Of the total group of fifty, 68% were clinically improved with disappearance or decrease in anginal attacks and resumption of normal activity. Twenty-two per cent of the group were clinically unchanged.

Recently, Lord et al (57) have described a method of measuring the contribution to coronary circulation of extracoronary sources. Their procedure consists of clamping the aorta proximal to the coronary ostia and measuring the venous return to the right atrium. This return in the right atrium should represent extra-cardiac contribution. When such measurements are made on a dog having undergone a BIMAL, the coronary return with the aorta clamped was substantially

increased over the normal control dogs.

Glover and associates were uncertain as yet whether bilateral internal mammary artery ligation would remain a surgical procedure in its own right or would be used as an adjunct to other surgical procedures.

PRODUCTION OF VASCULAR ADHESIONS

The production of vascular adhesions, cardio-pericardiopexy, has been used alone (37,38,40,41, 42,43) as well as an adjunct to other technics; for example, as used in the Beck No. 1 operation. The rationale of the operation is the production of a bread and butter pericarditis resulting in fibrous adhesions between the pericardium and the epicardium by the use of a nonabsorbable inflammatory agent such as talcum (41), asbestos (36) and alleuronol (18).

Thompson (41), in 1939, reported the use of powdered magnesium silicate (talc) for the first time. In 1954 (42) he stated that talc, instilled intapericardially, produced a foreign body reaction resulting in an adhesive granulomatous pericarditis without constriction, and with a vascular bridge between extracardiac structures and the myocardial vessels. Since the particles of talc are not removed by phagocytes and by the lymphatics, Thompson believed the connections

formed by this procedure would remain throughout the patients life since the talc would remain as a constant source of irritation.

Later in 1954, Thompson and Plachta (43) reported their results over a fourteen year period. To evaluate the benefit derived in the survivors, four items were considered: 1.) decrease in anginal attacks, 2.) increase in exercise tolerance, 3.) increase in ability to attend to daily needs and, 4.) return to gainful employment. Of fifty-seven patients operated upon there were seven hospital deaths and deaths attributed to the operation giving a mortality rate of 12%. Of the remaining fifty patients followed to the time of publication "poor results obtained in 5-10%, moderate results in 50%, and marked results in 40%".

Gorelik and Dack (37,38), in 1954, reported their results in the use of cardiopericardiopexy using powdered talc. These authors used the procedure on 47 patients. Three subjects died in the early postoperative period giving a mortality rate of 6.4% (four patients died of noncardiac causes during the early postoperative period, all with improved cardiac status). Thirty-seven of these patients were followed over an extended period with excellent results reported in 51% and good results in 17%.

Gorelik and Dack, believing that much of the myocardial ischemia seen in patients with rheumatic heart disease was due to the repeated episodes of rheumatic myocarditis, performed cardiopericardiopexies on 18 patients with rheumatic heart disease (38). The immediate post-operative mortality rate was 22% (four patients). Of the remaining fourteen patients all but two had moderate to marked improvement of cardiac status as shown by increase in exercise tolerance and decrease or complete disappearance of congestive failure. In none of these cases were attempts made to correct valvular defects. Several of their patients with tight mitral stenosis showed clinical improvement comparable with results seen with mitral commissurotomy. Because of these, they felt that cardiopericardiopexy might be used in cases of valvular lesions where mitral valvulotomy was not applicable; e.g. in cases of free mitral or aortic regurgitation, aortic stenosis, severe tricuspid lesions, or marked left ventricular hypertrophy.

Cardiopericardiopexy, as well as the grafting of vascular structures to the myocardium, has been discredited on the basis that such procedures produced additional circulation only to the surface of the heart, whereas, most areas of ischemia are deep or

subendocardial in position (58). Burchell (59), in 1940, published studies indicating that, in dogs who have undergone a talc poudrage, the epicardium presents a barrier that prevented the penetration of vascular channels of substantial size. Using this work, as a basis, Harken et al (39) suggests removing the epicardium with 95% phenol, doing a talc cardiopericardiopexy, and a cardiopneumopexy. These investigators used this procedure on eighteen patients with no mortalities and relief of pain in all.

BECK OPERATION NO. 1

The Beck Operation No. 1 consists of abrading the inner surface of the pericardium and the epicardium in areas devoid of large coronary vessels, partially ligating the coronary sinus, installation of 0.2 Gm. of powdered asbestos into the pericardial cavity, and grafting of the abraded pericardium to the surface of the heart. All of the components of this procedure, except partial ligation of the coronary sinus, are procedures that have been discussed as operations in their own right earlier in this paper. The rationale behind partial ligation of the coronary sinus is as follows: the ligation of this vein is not beneficial when the arterial circulation in the heart is normal because it reduces arterial

inflow, but when a major coronary artery is occluded ligation of the sinus affords protection against loss of coordinated mechanism, and it also provides for greater utilization of oxygen from the blood in the vascular bed (36).

Using this procedure on dogs Beck has shown that the myocardial circulation is significantly increased, using the Mautz-Gregg back flow method to measure the collateral thus produced (36). They found in 67 normal dogs that the backflow from the anterior descending branch of the left coronary artery averaged 3.8 cc. per minute. After the No. 1 operation was done, the backflow in 41 dogs was 8.5 cc. per minute, or an increase of 4.7 cc. When a period of about 6 months to 1 year was allowed to elapse after the No. 1 operation was done, the backflow had increased to 55 cc. per minute.

In a series of thirty normal dogs not protected by the operation, 70% died following ligation of the anterior descending branch. Of thirty dogs protected by the procedure, 73.4% survived such a ligation. It was felt by these authors that apparently this procedure supplied enough blood 4.7 cc. to prevent the development of an electrically unstable heart thus allowing time for the development of natural inter-

arterial communications.

In a comparative study, Beck and Leighninger (36) report a backflow of 4.8 cc. per minute obtained with a talc poudrage on 20 dogs, 1 cc. greater than the average normal but 3.7 cc. less than obtained with the No. 1 operation. Using the Vineberg internal mammary implantation on 20 dogs, the average backflow was 3.9 cc. per minute. Thus, these investigators obtained no increase in backflow measurements over the normal, the measurements being the same with artery occluded or not occluded.

Brofman (60), a cardiologist, reported the results obtained on 185 consecutive patients operated on by Dr. Beck from 1951 to 1956. In this series there have been eleven deaths associated with the operation (two during the operative procedure and nine during the early post-operative period) for a mortality rate of 6%. The follow up studies were done over a period of from six months to five years with an average follow up of two years. During this period eighteen were known to have died giving a mortality rate of 13.1%. Citing the work of Lindgren (61), a comparable mortality rate of 30% was reported in patients after two years of medical management. Of the remainder of the patients surviving the operation, Brofman reports 90% to have

definite amelioration of symptoms and 10% having obtained no observable improvement.

BECK OPERATION NO. 2

Beck first reported using this procedure on humans in 1948 (35) and at that time reported on his animal research leading up to the development of this technic. The operation is divided into two stages: the first stage consists of shunting arterial blood into the coronary sinus via a free vein graft between the descending thoracic aorta and the coronary sinus or a direct anastomosis of these structures; the second stage, done 2-3 weeks later, involves partial ligation of the coronary sinus at its entrance into the right atrium. This procedure takes advantage of the fact that venous drainage of the heart is primarily accomplished by two sets or systems of veins, 40% of the drainage by the Thebesian and deep venous system and 60% by the superficial venous system (anterior cardiac veins and the coronary sinus). The superficial system, thereby, offers a nice portal for arterialization (14).

Beck and Leighninger (36), again using the Mautz-Gregg backflow measurements, reported a backflow of 13.7 cc. per minute in dogs protected by this procedure compared to 3.8 cc. per minute in unprotected dogs.

When a period of 6-12 months is allowed to elapse after the operation, the backflow had increased to 62 cc. per minute. In a series of 45 dogs, 70% died following ligation of a major coronary artery. Of 45 dogs protected by this operation, 91.1% survived such a ligation. In dogs, at least, the Beck No. 2 produced results superior to the Beck No. 1.

In clinical application, however, this is not the case. During the period of 1951-1953 Beck reports a mortality of 7.5% for the Beck No. 1 and a mortality of 26.1% for the No. 2 (36). Because of this, he has set the procedure aside, to be used, possibly, in those patients who still have pain following the No. 1 operation. Other authors have agreed with Beck on this point. Feil et al (62) reported a mortality rate of 5% for the Beck No. 1 and 25% for the Beck No. 2. They state that in addition to providing more inter-coronary collaterals, this procedure actually adds a burden to the heart and circulation, the functioning arteriovenous fistula necessitating an increase in cardiac output and blood volume. In some of their patients cardiac failure was induced by the operation. Bakst and Bailey (46) reported that, while reversal of blood flow was good in the initial stages, later there was a diminution either of the amount of retrograde

flow or impairment of the patency of the new anastomotic channels. Harken et al (39) summarized their objection to this operation as follows: 1.) it involves two major operations either of which are strenuous for coronary patients, 2.) many of the venous grafts thrombose, 3.) if the patient survives the operation with a patent graft, there is the possibility of the formation of high venoarterial shunts thus carrying blood more or less directly from the aorta into the chambers of the ventricles without being distributed to the myocardium, and finally, 4.) postmortem examinations in many patients show that atheromatous plaques have occluded the arterialized venous system suggesting that these vessels cannot withstand high arterial pressures.

ENDARTERECTOMY

This procedure is essentially as yet in experimental stages. The feasibility of endarterectomies was established by the investigations of Absolon and his associates (50) working with cadaver hearts. The operation, as described by Bailey et al (49) consists of the insertion of a May cannula (a curette tipped cannula) into a coronary artery through a small branch distal to the occluded area. The cannula, or curette, is passed up the artery past the occluded

area. As it is withdrawn the intima and the atheromatous occlusion is stripped away leaving a patent artery. The clinical application of this procedure has not been extensive enough for proper evaluation. Bailey reports having performed the operation on two patients with clinical improvement in both. The limitations of this procedure are obvious. Best results would be obtained in patients having one, or at most, two isolated areas of arterial occlusion. The exact incidence of death due to a single coronary occlusion is not known but is estimated by Hellerstein (63) to be about 10%. This procedure may be valuable in such cases with a demonstrable single lesion, preferably in the acute stage.

MUROLUMINAL ANASTOMOSES

These procedures utilize Vineberg's concept mentioned earlier, that the myocardium consists of a fine spongelike vascular network surrounding the myocardial fibers. Essentially, these procedures are still in the experimental stage and as yet no evaluation of clinical application has been made.

In 1956, Goldman and co-workers (47) reported investigations with dogs using U-shaped arterial grafts, straight arterial grafts and straight polyethylene grafts. The procedures are described as

follows: a U-shaped segment of carotid artery about 3 cm. in length is perforated at several sites along its length. This graft is then imbedded into the myocardium in such a way that the ends of the artery open into the left ventricular cavity, the remainder of the artery containing the perforations being buried in the myocardium. Similarly, the straight arterial grafts and polyethylene grafts were placed with one end in the ventricular cavity and the rest of the graft buried in the myocardium. In this fashion, oxygenated blood is forced directly from the left ventricle during systole, into the myocardial circulation.

In the dogs studied by these authors, these procedures resulted in a significantly larger number of survivors and smaller infarct size than was seen in unprotected dogs when the anterior descending branch of the left coronary artery was ligated.

Massimo and Boffi (48), in 1957, reported using a T-shaped tube as a graft, the horizontal arm embedded in the myocardium parallel with the long axis of the left ventricle and the vertical arm extending into the left ventricular cavity. These authors state the possible disadvantages to procedures such as these are: the damage done by an incision into the already

damaged myocardium and, the possibility of a callous formation or eventual development of a ventricular aneurysm at the tube site.

DISCUSSION

The experimental work on animals done by the investigators in this field have proven the protective effect these various procedures afford when used prior to ligation of a main coronary artery. Mortality rate was lowered from 70 to 90% to approximately 10%, and the size of the infarct was also reduced. Caution should be used, however, in applying concepts based on animals with normal coronary arteries, to humans suffering from occlusive coronary disease. An excellent example is the difference in effects of aorta-coronary sinus anastomosis in dog and in man. This is feasible and tolerable in the former, but not in the latter (36,46,62). Another example is the conjecture that surgery protects the heart from fatal ventricular fibrillation by producing a minute improvement in the intercoronary collaterals resulting in a more uniform oxygenation of the myocardium and thus prevents the development of electrical potentials between ischemic and nonischemic areas. While it is true that acute infarction in dogs causes death by ventricular fibrillation,

according to Hellerstein (64), in more than 60% of humans dying from acute coronary occlusion, the mechanism of death is atrioventricular block, and ventricular and atrial asystole.

The most powerful stimulus for the development of coronary collaterals is local tissue anoxia (7). In none of the surgical procedures described above has the volume of the injected anatomic collateral had, or the retrograde flow, been of the magnitude observed with chronic ligation of the coronary artery. Blumgart et al (7) reported that of the normal cadaver hearts studied by them, only 15% contained inter-arterial anastomoses large enough to be demonstrated by the Schlesinger lead-agar mass. In contrast, 98% of the hearts showing occluding lesions in the arteries had collateral channels in much greater number and of larger size. This work therefore suggests that patients with advanced coronary atherosclerosis prior to surgery, may already have a maximally developed intercoronary vascular network.

If such maximal formation of the interarterial collaterals does in fact occur, the question then arises concerning the significance of the slight increase provided by surgery. No experiments have yet been reported which demonstrate the protective value of

surgery in dogs with coronary artery disease, simulated by ligature stenosis or by experimental intimal disease. Such a study would be of great value in the evaluation of these procedures.

The evaluation of these surgical procedures has, thus far, been essentially subjective. The diminution of pain (angina) has been reported in from 45 to 70% of the survivors of each of the various procedures (36,37,39,43). The psychological effects of cardiac surgery have been recognized by several authors (36). The patients consider that the most has been done for them and often acquire a zest for living and an increased confidence resulting in the patients resumption of previous activities and a desire to work. The studies of survival and mortality presented thus far in the literature have been made in comparison to a series of medically managed patients published a decade ago (61). Thompson (43) has claimed that his terminal and incapacitated cases have survived an average of 9.5 years after the appearance of symptoms and compared this to 4.5 years for medically managed patients. According to Hellerstein (63), the survival rate recently for non-operated cases is much better, an average survival of 8 to 10 years. A recent comparative study of medically

and surgically treated patients for coronary artery disease is an important, but missing, item in the literature today.

SUMMARY

The primary objective of surgical revascularization of the myocardium is to increase the extra-cardiac and intra-cardiac collateral communications to the myocardium thereby preventing the development of ischemic areas causing pain, the development of dangerous oxygenation differentials, and the destruction of myocardium. Such operations as discussed in this paper are merely palliative procedures, they are not curative. The atherosclerotic process proceeds unabated and the development of new coronary occlusions is not prevented.

BIBLIOGRAPHY

1. Parker, R.L.; Dry, T.J.; Willius, F.A.; and Gage, R.P.: "Life Expectancy in Angina Pectoris." *J.A.M.A.* 131: 95, 1946
2. Katz, L.N.; Mills, G.Y.; and Cisneros, F.: "Survival After Recent Myocardial Infarction." *Arch. Int. Med.* 84:305, 1949.
3. Danielopolu, D.: "The Surgical Treatment of Angina Pectoris." *Bri . M. J.*, Jan. 30, 1926, p.180.
4. Lerliche, R.; and Fontaine, R.: "The Surgical Treatment of Angina Pectoris." *Am. H. J.* 3:649, 1927-28.
5. Beck, C.S.; and Leighninger, D.S.: "Operations for Coronary Artery Disease." *J.A.M.A.* 156:1226, 1954.
6. Gross, L.: The Blood to the Heart in its Anatomic and Clinical Aspects. New York, Paul B. Hoeber, 1921, Pp. 171.
7. Blumgart, H.L.; Zoll, P.M.; Freedberg, A.S.; and Gilligan, D.R.: "The Experimental Production of Intercoronary Anastomoses and their Functional Significance." *Circulation* 1:10, 1950.
8. Zoll, P.M.; Wessler, S.; and Schlesinger, M.J.: "Interarterial Coronary Anastomoses in the Human Heart with Particular Reference to Anemia and Relative Cardiac Anoxia." *Circulation* 4:797, 1951.
9. Hudson, C.L.; Moritz, A.R.; and Wearn, J.T.: "Extracardiac Anastomoses of the Coronary Arteries." *J. Exper. Med.* 56:919, 1932.
10. Brofman, B.L.; and Beck, C.S.: "Coronary Heart Disease. I. Hemodynamic Principles and their Therapeutic Application." *J. Thoracic Surg.* 35:232, 1958.
11. Yater, W.M.; Beck, C.S.; Leighninger, D.S.; and Brofman, B.L.: "Symposium on Coronary Arterial Disease." *Am. Rev. Tuberc.* 71:904, 1955.
12. Brofman, B.L.; Leighninger, D.S.; and Beck, C.S.: "Electrical Instability of the Heart: The Concept of Oxygen Differential in Coronary Heart Disease." *Circulation* 13:161, 1956.
13. Schlesinger, M.J.; and Zoll, P.M.: "Incidence and

Localization of Coronary Artery Occlusions." Arch. Path. 32:178, 1941.

14. Bailey, C.P.; Geckeler, G.D.; Truex, R.C.; Likoff, W. Antonius, N.A.; Angulo, A.W.; Redondo-Ramirez, H.P.; and Neptune, W.: "Arterialization of the Coronary Sinus." J.A.M.A. 151:441, 1953.
15. Vineberg, A.M.: "Internal Mammary Artery Implantation in the Treatment of Angina Pectoris: A Three Year Follow Up." Canad. M. J. 70:367, 1954.
16. Antonius, N.A.; Crecca, A.D.; and Massarelli, L.G.: "Clinical Evaluation of the Surgical Treatment of Thirty-two Cases of Coronary Artery Disease." J. Thoracic Surg. 35:68, 1958.
17. O'Shaughnessy, L.: "An Experimental Method of Providing a Collateral Circulation to the Heart." Brt. J. Surg. 23:665, 1935-36.
18. O'Shaughnessy, L.: "Surgical Treatment of Cardiac Ischaemia." Lancet, Jan. 23, 1937, p.185.
19. Carter, B.N.; Gall, E.A.; and Wadsworth, C.L.: "An Experimental Study of Collateral Coronary Circulation Produced by Cardiopneumopexy." Surgery 25: 489, 1949.
20. Reimann, D.L.; and Mansberger, A.R., Jr.: "A Technical Consideration of Cardiopneumopexy: Comparative Study." Bull. School Med. Univ. Maryland 38:118, 1953.
21. Kownacki, R.J.; Kownacki, V.R.; KenneI, A.J.; Imbriglia, J.E.; and Martin, W.L.: "Collateral Circulation to the Heart by Means of Cardiopneumopexy and Lingular Vein Ligation." J. Thoracic Surg. 76:106, 1958.
22. Von Wedel, J.; Lord, J.W., Jr.; Neumann, C.G.; and Hinton, J.W.: "Revascularization of the Heart by Pedicle Skin Flap." Surgery 37:32, 1955.
23. Baronofsky, I.D.; Sprafka, J.L.; and Noble, J.F.: "Use of Intestinal Loops for Revascularization of the Heart." Circulation Res. 2:506, 1954.
24. Key, J.A.; Kergin, F.G.; Martineau, Y.; and Leckey, R.G.: "A Method of Supplementing the Coronary Circulation by a Jejunal Pedicle Graft." J. Thoracic Surg. 28:320, 1954.

25. Hannon, D.W.; and Baronofsky, I.D.: "A Comparison of Cardiojejunopexy with Other Methods of Revascularizing the Heart." *Surgery* 40:1111, 1956.
26. Beck, C.S.: "Development of a New Blood Supply to the Heart by Operation." *Ann. Surg.* 102:801, 1935.
27. Beck, C.S.; and Tichy, V.L.: "The Production of a Collateral Circulation to the Heart. I. Experimental Study." *Am. H. J.* 10:849, 1935.
28. Bakst, A.A.; Boley, S.J.; Morse, W.; and Loewe, L.: "Experimental Surgical Treatment of Occlusive Coronary Artery Disease. I. Use of Pedicled Pectoral Muscle Graft." *Angiology* 8:308, 1957.
29. Vineberg, A.M.: "Development of Anastomosis Between Coronary Vessels and Transplanted Internal Mammary Artery." *Canad. M. J.* 55:117, 1946.
30. Vineberg, A.M.; and Jewitt, B.L.: "Development of an Anastomosis Between Coronary Vessels and a Transplanted Internal Mammary Artery." *Canad. M. A. J.* 56:609, 1947.
31. Vineberg, A.M.: "Treatment of Coronary Artery Insufficiency by Implantation of the Internal Mammary Artery into Left Ventricular Myocardium." *J. Thoracic Surg.* 23:42, 1952.
32. Vineberg, A.M.: "The Treatment of Angina Pectoris by Internal Mammary Artery Implantation Supplemented by Pericardial Fat Wrap." *Connect. St. M. J.* 19:281, 1955.
33. Vineberg, A.M.; and Buller, W.: "Technical Factors Which Favor Mammary-Coronary Anastomosis: Report of Forty-five Cases of Human Coronary Artery Disease Thus Treated." *J. Thoracic Surg.* 30:411, 1955.
34. Beck, C.S.: "Principles Underlying the Operative Approach to the Treatment of Myocardial Ischemia." *Ann. Surg.* 118:788, 1943.
35. Beck, C.S.: "Revascularization of the Heart." *Ann. Surg.* 128:854, 1948.
36. Beck, C.S.; and Leighninger, D.S.: "Scientific Basis for the Surgical Treatment of Coronary Artery Disease." *J.A.M.A.* 159:1264, 1955.
37. Dack, S.; and Gorelik, A.N.: "Cardiopericardiopexy

for the Treatment of Coronary Artery Disease."
J.A.M.A. 45:772, 1953.

38. Gorelik, A.N.; and Dack, S.: "Revascularization of the Myocardium by Cardiopericardioplexy." *Internat. Coll. of Surg.* 21:167, 1954.
39. Harken, D.E.; Black, H.; Dickson III, J.F.; and Wilson III, H.E.: "De-Epicardialization: A Simple Effective Surgical Treatment for Angina Pectoris." *Circulation* 12:955, 1955.
40. Mazel, M.S.; Bernstein, M.M.; Callen, I.R.; Schnaer, I.R.; Wu, L.T. and Bonk, A.: "A Simple Operation for the Treatment of Chronic Coronary Artery Disease." *Arch. Surg.* 70:309, 1955.
41. Thompson, S.A.: "Development of Cardio-pericardial Adhesions Following the Use of Talc." *Proc. Soc. Exper. Biol. and Med.* 40:260, 1939.
42. Thompson, S.A.; and Akopiantz, L.A.: "Cardiopericardioplexy in the Surgical Treatment of Myocardial Ischemia." *J. Internat. Coll. Surg.* 22:551, 1954.
43. Thompson, S.A.; and Plachta, A.: "Fourteen Years' Experience with Cardioplexy in the Treatment of Coronary Artery Disease." *J. Thoracic Surg.* 27:65, 1954.
44. Battezzati, M.; Tagliaferro, A.; and De Marchi, G.: "The Ligature of the Internal Mammary Arteries in Disorders of Vascularization of the Myocardium." *Minerva Med.* 46(Part Two):1173, 1955.
45. Glover, R.P.; Davila, J.C.; Kyle, R.H.; Beard, J.C. Jr.; Trout, R.G.; and Kitchell, J.R.: "Ligation of the Internal Mammary Arteries as a Means of Increasing Blood Supply to the Myocardium." *J. Thoracic Surg.* 34:661, 1957.
46. Bakst, A.A.; and Bailey, C.P.: "Arterialization of the Coronary Sinus in Occlusive Coronary Artery Disease. IV. Coronary Flow in Dogs with Aorticocoronary Sinus Anastomosis of Twelve Months' Duration." *J. Thoracic Surg.* 31:559, 1956.
47. Goldman, A.; Greenstone, S.M.; Preuss, F.S.; Strauss, S.H.; and En-Shu, Chang: "Experimental Methods for Producing a Collateral Circulation to the Heart Directly from the Left Ventricle." *J. Thoracic Surg.* 31:364, 1956.

48. Massimo, C.; and Boffi, L.: "Myocardial Revascularization by a New Method of Carrying Blood Directly from the Ventricular Cavity into the Coronary Circulation." J. Thoracic Surg. 34:257, 1957.

49. Bailey, C.P.; May, A.; and Lemmon, W.M.: "Survival After Coronary Endarterectomy in Man." J.A.M.A. 164:641, 1957.

Absolon, K.B.; Aust, J.B.; Varco, R.L.; and Lillehei, C.W.: "Surgical Treatment of Occlusive Coronary Artery Disease by Endarterectomy or Anastomotic Replacement." Surg. Obst. and Gyn. 103:180, 1956.

51. Mautz, R.R.; and Gregg, D.E.: "The Dynamics of Collateral Circulation Following Chronic Occlusion of Coronary Arteries." Proc. Soc. Exper. Biol. & Med. 36:797, 1937.

52. Gregg, D.E.; and Sabiston, D.C.: "Current Research and Problems of the Coronary Circulation." Circulation 13:916, 1956.

53. Glenn, F.; and Beal, J.M.: "The Fate of an Artery Implanted in the Myocardium." Surgery 27:841, 1956.

Bakst, A.A.; Maniglia, R.; Adam, A.; and Bailey, C.P.: "The Physiologic and Pathologic Evaluation of Implantation of the Internal Mammary Artery into the Left Ventricular Myocardium for the Treatment of Coronary Artery Disease." Surgery 29:1, 1955.

55. Sabiston, D.C. Jr.; and Fauteux, J.P.: "Fate of Experimental Arterial Implants into the Right Ventricle." Arch. Surg. 75:479, 1957.

56. Sabiston, D.C. Jr.; Fauteux, J.P.; and Blalock, A.: "An Experimental Study of the Fate of Arterial Implants in the Left Ventricular Myocardium With a Comparison of Similar Implants in Other Organs." Ann. Surg. 145:927, 1957.

57. Lord, J.W. Jr.; Coryllos, E.; Lowenfels, A.B.; Dysart, R.; Neumann, C.G.; and Hinton, J.W.: "Evaluation of Operations for Revascularization of the Myocardium by the Study of Coronary Blood Flow Using Extracorporeal Circulation." Surgery 43:202, 1958.

58. Bailey, C.P.: Surgery of the Heart. Lea and Febiger, Publishers, 19

59. Burchell, H.S.: "Adjustments in Coronary Circulation After Experimental Coronary Ligation with Particular Reference to Revascularization of Pericardial Adhesions." *Arch. Int. Med.* 65:240, 1940.
60. Brofman, B.L.: "Medical Evaluation of the Beck Operation for Coronary Artery Disease." *J.A.M.A.* 162:1603, 1956.
61. Lindgren, I.: "Angina Pectoris: A Clinical Study with Special Reference to Neurosurgical Treatment." *Acta. med. scandinav.* (supp. 243) 138:1, 1950.
62. Feil, H.; Pritchard, W.H.; Hellerstein, H.K.; Watts, R.W.; Hornberger, J.C.; and Helfrich, H.M.: "The Beck Operations for Coronary Heart Disease: An Evaluation of 63 Patients Selected for Operation." *Ann. Int. Med.* 44:1:271, 1956.
63. Hellerstein, H.K.: "Evaluation of Surgical Methods in the Treatment of Coronary Artery Disease." *Mod. Concepts Cardiovas. Dis.* 26:411, 1957.
64. Hellerstein, H.K.: "What is the Mechanism of Death?" Report to Postgraduate Seminar, University of Kansas, Feb. 26, 1957.