Study of the Hemodynamic Effects of the Aorto-
Coronary Sinus Graft Operation in Patients
with Coronary Artery Disease

By T. W. Moir, M.D., and W. H. Pritchard, M.D.

A group of patients with coronary artery disease have been studied preoperatively and after
surgical creation of an arteriovenous fistula between the descending aorta and coronary sinus
(Beat II, revascularization operation). Preoperatively, basal studies of cardiac output and blood
volume were done with radioisotope technics. Postoperatively, catheterization studies of cardio-
vascular function were obtained and abnormalities, including heart failure, were found in patients
with functionally significant arteriovenous fistulas.

A CLINICAL evaluation of surgical pro-
cedures for revascularization of the heart
in 63 patients with arteriosclerotic heart disease
with angina pectoris has been reported re-
cently by Feil and associates.1 Two different
surgical procedures were utilized after each
had been studied extensively in normal dogs.2, 3
Although the results in 6 patients who had peri-
cardial abrasion, asbestos application to the
abraded epicardium, and partial ligation of
the coronary sinus were reported, the report
emphasized the clinical results in 57 patients
who had arterIALIZATION OF the coronary sinus
through a vein graft from the aorta. This paper
is concerned primarily with the patients in the
latter group who had arterIALIZATION of the
coronary sinus as described by Beck.2

This surgical procedure was performed in 2
stages: (1) a homologous vein graft was first
placed between the aorta and the coronary sinus (fig. 1); and (2) after a period of from 2 to
4 weeks, the coronary sinus near its entry into
the right atrium was narrowed, allowing a
variable arterial flow into the right atrium to
remain.

The purposes of the present study were to
evaluate certain measures of basal cardiac
function in some of these patients with coro-
nary artery disease before surgery and to ascer-
tain the hemodynamic consequences of the
surgically created arteriovenous fistula upon
the circulation of these subjects.

PATIENTS AND METHODS

It was not possible to study a representative
sample of patients undergoing the 2-stage operation
sequentially at appropriate times before and after
operation. Many of the patients studied preoper-
avely were not restudied after surgery because of
nonpatency of the vein graft, operative mortality,
or loss from the study for other reasons. Similarly,
some of the postoperative patients who had had a
successful graft procedure had not been studied
preoperatively. Since our primary interest was in the
effects of the fistula, patients who survived and
achieved a functionally patent graft were studied
regardless of whether a preoperative study had been
performed.

All patients had coronary artery disease mani-
fested by angina pectoris or previous myocardial
infarction, or both. None had preoperative evidence
of cardiomegaly or clinical evidence of cardiac
failure. All were normotensive.

Patients were studied in the postabsorptive, basal
state. Preoperatively, cardiac output was calculated
from arterial dilution curves, following injection of
iodinated (I131) human serum albumin.4 Blood
volume was determined before and after operation
with the same isotope.4

Postoperatively cardiac catheterization was per-
formed to study pressure and flow relationships.
Pressures were recorded with Sanborn elec-
tronometers, and cardiac output was estimated by
the Fick principle. Oxygen consumption was mea-
sured by analysis of expired gas while the subject
breathed room air. The systemic arteriovenous
difference was calculated from the femoral artery
oxygen content and the averaged values of the
oxygen content of superior and inferior cavae. The
pulmonary arteriovenous difference was calculated

From the Department of Medicine, Western Re-
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versity Hospitals of Cleveland.

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Institute, U. S. Public Health Service.
Fig. 1. Posterior view of the heart showing vein graft in place between the descending aorta and the coronary sinus. The constricting ligature placed around the coronary sinus (stage II) is not shown.

from the femoral artery oxygen content and the best mixed sample beyond the right atrium, usually the pulmonary artery, but occasionally the right ventricle. Thus, the shunt flow was the difference between calculated pulmonary and systemic flow. Cardiac work was calculated in Kg. M./hr., from the relationship, work = total cardiac output \times mean arterial pressure. The kinetic factor, $\frac{mV^2}{g}$, was not used. Total peripheral resistance was calculated by the formula:

$$T.P.R. = \frac{\text{Mean arterial pressure} \times 1332}{\text{Cardiac output (ml. per second)}}.$$ 

In the postoperative catheterization study 3 groups were designated: 1. Thrombosed grafts. These patients had had a vein graft initially placed, but at the second stage operation the communication was found to be thrombosed, or at a later date was completely ligated. In this group no arteriovenous communication was found at the time of the catheterization study. These patients served as controls for the patients with functioning aorto-coronary sinus grafts. 2. Patent grafts, immediate study. Catheterization was done from 13 to 28 days after placing the graft, and before the second-stage operation. 3. Patent grafts, remote study. Catheterization was performed from 11 to 26 months after the second stage operation, at which time the coronary sinus had been narrowed at its entrance to the right atrium.

Results

Figure 2 shows the preoperative data for blood volume and cardiac output and demonstrates the wide range of individual determinations. The mean blood volume was $70.3 \pm 12.1$ ml. per Kg. of body weight. The mean cardiac index was $2.86 \pm 0.82$ L. per M.$^2$ per min. No difference in blood volume or cardiac output was found that could be related to the presence of previous myocardial infarction.

Postoperative Studies

Blood Volume. In figure 3 the blood volume-body weight relationship has been plotted and lines of regression have been drawn for the postoperative patients. The blood volume determinations were done at varying times after surgery. Three groups of patients are compared: 17 patients with thrombosed grafts; 13 patients with patent grafts without heart failure; 13 patients with patent grafts with heart failure.

No significant increase in blood volume is discernible in patients with patent grafts when compared with those having thrombosed grafts unless cardiac failure is present. The increase in blood volume in patients with patent grafts
and cardiac failure is significant, \( p \) (adjusted means) = <0.01. There was no increase in blood volume in patients with thrombosed grafts when compared to the preoperative study group.

**Cardiac Output and Shunt Flow.** Table 1 shows the individual data and mean values in the postoperative catheterization study. The mean cardiac index in the 6 patients with thrombosed grafts was within normal limits (2.96 L per M.\(^2\) per min.) and is comparable to the 2.86 L per M.\(^2\) per min. value found preoperatively. In the 6 patients with open grafts catheterized shortly after the first stage of the operation (immediate study group), the cardiac index was slightly elevated, averaging 3.50 L per M.\(^2\) per min., and the mean shunt flow was 1.75 L per min. In the 5 patients with patent grafts of longer duration (remote study group), the cardiac index was markedly elevated, averaging 6.21 L per M.\(^2\) per min. with a mean shunt flow of 6.52 L per min. The effective systemic flow progressively decreased in spite of the augmentation of the total cardiac output in all patients with a functioning shunt and was most pronounced in those patients with grafts of longer duration.

**Table 1.—Cardiac Catheterization Data in the Postoperative Patients**

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<th>Patient</th>
<th>Mean pressure (mm. Hg)</th>
<th>Cardiac output (L./min.)</th>
<th>Cardiac index (L./M.(^2)/min.)</th>
<th>Systemic flow (L./Min.)</th>
<th>Shunt flow (L./Min.)</th>
<th>Pulse rate</th>
<th>Stroke volume (ml.)</th>
<th>Mean arterial pressure (mm. Hg)</th>
<th>Oxygen consumption (ml./M.(^2)/min.)</th>
<th>Cardiac work (Kg.M./hr.)</th>
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Three of the 6 patients with patent grafts studied shortly after the first-stage operation were in cardiac failure which developed following surgery. In 2, the cardiac indices were elevated (4.37 and 4.20 L. per M.² per min.). Three of the 5 patients with patent grafts studied in the remote period were also in heart failure at the time of the study. Two of these 3 had increased cardiac indices (4.80 and 6.57 L. per M.² per min.).

Pulse Rate and Stroke Volume. The pulse rate and stroke volume of the patients whose grafts were thrombosed at the time of the study were essentially normal. In those patients with patent grafts of short duration, the stroke volume was not elevated, but there was a consistent increase in pulse rate. The stroke volume became elevated to a mean value of 145 ml. in those patients whose grafts remained functioning from 11 to 26 months; the pulse rate in these subjects was normal.

Intracardiac Pressure. Mean right atrial and right ventricular pressures were normal in the postoperative patients with thrombosed grafts. In the patent graft group, pressures were variably increased and no direct relationship could be found between the volume of shunt flow into the right atrium and right atrial pressure. Rather, atrial pressure was related to mean right ventricular pressure and any increase was due to cardiac failure rather than volume of shunt flow.

Arterial Pressure, Cardiac Work, and Total Peripheral Resistance. Although the mean arterial pressure was, on the average, lower in the patients with patent grafts than in those patients without a shunt, there was no consistent change.

Cardiac work as shown in figure 4, increased proportionately to the volume of shunt flow in the patients with patent grafts. Peripheral resistance was in the upper ranges of normal in the operated patients with closed grafts, but was slightly lowered in patients with grafts of shorter duration, and decreased in those with shunts present for longer periods of time.

Coronary Sinus Pressure. In 1 patient with a functioning graft, the coronary sinus was catheterized. The coronary sinus pressure was 97/62 (mean 60) mm. Hg; arterial pressure was 140/80 (mean 80) mm. Hg. A blood sample from the coronary sinus was 91 per cent saturated.

Discussion

The study of cardiac output preoperatively demonstrated a mean cardiac index that was in the lower range of normal for the resting state when compared with determinations obtained in normal subjects. The results are similar to those of Chapman and Fraser, and indicate that in patients with coronary artery disease, but free of heart failure, the cardiac output at rest is not significantly altered from normal. However, the experience of Smith and associates is at variance with this conclusion. The mean blood volume of the preoperative patients, expressed in milliliters per kilogram of body weight, was within the limits of normal for the method used. Operation alone caused no significant change in blood volume, since patients with thrombosed grafts had mean blood volume values comparable to the preoperative group.

Of major interest in this study was the opportunity to observe the hemodynamic alterations produced by the aortic-atrial communication in patients with coronary artery disease. Operation per se produced no significant change in the measurements of cardiovascular function, since these values were well within the range of normal in the patients with thrombosed grafts. However, in the patients with patent grafts circulatory abnormalities incident to the arteriovenous fistula became apparent.

It is now generally held that the cardiac output is elevated in the presence of an arterio-
venous fistula. Animal and human studies support this view. The present study confirms the fact that this augmentation of cardiac output is due to an increased stroke volume when the shunt flow is of great magnitude. However, in our patients with patent grafts of short duration, and with a relatively low volume of shunt flow, the cardiac output rose only moderately and was due to an increase in pulse rate. As the volume of shunt flow through the patent aorto-coronary sinus fistula increased, cardiac output rose as the result of an increased stroke output (table 1).

Considerable speculation remains concerning the reason for augmentation of stroke volume in arteriovenous fistulas, and particular attention has been directed toward the role of an increased pressure gradient between the right atrium and ventricle. The present study in patients with coronary disease showed no obvious relationship between an increase in cardiac output and mean right atrial pressure; elevations in right atrial pressure were more related to the right ventricular pressure than to the volume of shunt flow and consequent increases in cardiac output. Mean pressure in the atrium, however, may not mirror end-diastolic pressure or volume in the ventricle. In addition, the presence of cardiac failure in some of the patients in the present study vitiates any firm conclusions regarding pressure-volume changes between the right atrium and ventricle relative to increase in stroke volume.

In spite of augmentation of the total cardiac output in these patients, the effective systemic flow was decreased, the degree of which was inversely related to the volume of shunt flow. Similar changes have been described in experimental studies where “body flow” has shown a progressive decrease as the volume of shunt flow has increased.

It is generally believed that the blood volume is elevated in the presence of functionally significant arteriovenous fistulas and it has been suggested that this may be an essential factor in the maintenance of an effective “body flow” in the presence of large fistulas. In experimental animals it has been shown that this rise in blood volume is due to an expanded plasma volume. In human subjects with arteriovenous fistulas, an elevation in blood volume has been variable and inconstant; Warren and associates found only 44 per cent of their patients with any significant blood volume increase, with no good correlation of this change with the magnitude and duration of shunt flow. This elevation of blood volume was observed in the absence of detectable cardiac failure. Epstein, Post, and McDowell have demonstrated decreased renal excretion of sodium in patients with arteriovenous fistulas, and in Hilton’s study in dogs, arteriovenous fistulas led to renal vasoconstriction, reduced glomerular filtration rate, and a decrease in sodium, chloride, and water excretion. In these data one finds a suitable setting for fluid retention with subsequent blood volume expansion without cardiac failure. However, in the present study the elevation of blood volume was related to the presence of cardiac failure and lends support to the thesis of Reid and McGuire that elevation of the blood volume in arteriovenous fistulas is due to cardiac failure, at least in patients with pre-existing heart disease.

Since the cardiac work increased proportionally to the volume of shunt flow, the precipitation of cardiac failure in some of these patients is not surprising. However, the incidence of overt heart failure could not be entirely correlated with the volume of shunt flow and it would appear that a major determinant of cardiac decompensation in these patients with coronary disease was the poor “functional reserve” of the myocardiun. It is apparent that this surgical procedure, which increases cardiac

| Table 2.—Hemodynamic Data before and after Surgical Obliteration of the Fistula, in a Postoperative Patient in Heart Failure |
|----------------------------------|------|------|
| Cardiac output (L./M.²/min.) | 4.8  | 3.2  |
| Systemic output (L./M.²/min.) | 2.2  | 3.2  |
| Shunt flow (L./min.)           | 3.9  | —    |
| Right atrial pressure (mm. Hg) | 7/3 (5) | 3/1 (2) |
| Right ventricular pressure (mm. Hg) | 60/5(24) | 38/4(15) |
| Arterial pressure (mm. Hg)     | 150/70(99) | 156/96(116) |
| Cardiac work (Kg.M./hr.)       | 612  | 510  |
| Total peripheral resistance (dynes-cm.-sec.-²) | 1040 | 1680 |
| Blood volume (ml./Kg.)         | 83   | 73   |
work due to the arteriovenous fistula, may precipitate cardiac failure in patients with myocardial disease. Table 2 illustrates studies performed in a patient whose graft remained open and who 18 months after operation developed congestive heart failure. The patient was subsequently reoperated upon, and the fistula was closed; the signs of cardiac failure, together with the hemodynamic abnormalities, regressed.

It is interesting that the volume of shunt flow through the aorto-coronary sinus graft increased with time and resulted in a markedly elevated cardiac output in the patients with grafts of long duration (table 1). If we may assume that the coronary sinus has remained narrowed at its egress into the right atrium, it appears likely that the progressive enlargement of a collateral venous bed in the area of the aorto-coronary sinus graft accounts for the augmentation of shunt flow. Such venous collateral circulation is known to occur in peripheral arteriovenous fistulas in man and has been observed in animals subjected to the present operative procedure.34

**Summary**

A group of patients with coronary artery disease in whom an aorto-coronary sinus graft was surgically created have been studied to determine the functional effects on the cardiovascular system. Preoperatively, mean values for basal cardiac output were within the lower ranges of normal and the average blood volume values were normal.

Postoperatively, no circulatory abnormalities were demonstrated in patients in whom the aorto-coronary sinus graft was thrombosed at the time of study. In those patients with patent grafts, hemodynamic changes of an arteriovenous fistula were demonstrated. Cardiac output and work increased, effective systemic flow decreased, peripheral resistance fell, and blood volume became greater. The increase in cardiac output was related to an increase in pulse rate in cases with small shunt flows and to augmentation of stroke volume when fistulas were of greater magnitude and duration. Elevation of the total blood volume was shown to be related to the presence of cardiac failure and not to the presence of the arteriovenous fistula per se.

Cardiac failure developed in some of the patients with a functioning aorto-coronary sinus graft and could be reversed with subsequent surgical obliteration of the shunt. The development of cardiac failure in these patients with pre-existing heart disease could not be entirely related to the volume or duration of shunt flow, but rather seemed dependent on a lack of “myocardial reserve” to withstand the added load of an arteriovenous fistula.

Increase in volume of shunt flow was related to duration of the functioning aorto-coronary sinus graft and suggested increased venous collateral flow into the right atrium.

**Acknowledgment**

The authors wish to thank the members of the cardiac catheterization unit of The University Hospitals of Cleveland who assisted in the catheterization studies of this project.

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**Summary in Interlingua**

Un gruppo de patientes con morbo de arteria coronari in qui un grafto de sinus aorto-coronari habeva essite create per un operatione chirurgic esseva studiate con le objectivo de determinar le resultante effectos in le sistema cardiovascular. Ante le operation, le valores medie del basal rendimento cardiac esseva basse-normal, e le valores del volumine medie de sanguine esseva normal.

Post le operation, nulle anormalititates circulatori esseva demonstrate in patientes in qui le grafto aorto-coronari esseva thrombositate al tempore del studio. In patientes con graffos patente, alterationes hemodynamic de un fistula arterio-venose esseva demonstrate. Le rendimento e le labor cardiac se augmentava, le effective fluxo systemic se reduceva, le resistentia peripheric se reduceva, e le volumine de sanguine se augmentava. Le augmento del rendimento cardiac esseva relateante a un accele ration del pulso in casos con paue fluxo derivational e a augmentation del volumine per pulso quando le fistulas esseva plus considerabile in magnitude e duration. Esseva monstrate que le augmento del volumine de sanguine total
eseva relationate al presentia di disfallimento cardiac e non al presentia de un fistula arteriovenose per se.

Disfallimento cardiac se disveloppiava in certes del pacientes con funcionante graffos de sinus aorto-coronari e poteva esser revertite per le subsequente obliteration chirurgic del derivation. Le disveloppanimento de disfallimento in iste pacientes con pre-existent morbo cardiac non eseva completely relationable al volumine o al duration del fluxo derivational sed pareva depender plus tosto de un manco de “reservas myocardial” capace a manipuler le carga additional de un fistula arteriovenose.

Le augmento del volumine del fluxo derivational eseva relationate al duration del funcionante graffo de sinus aorto-coronari e suggereva un augmento del fluxo collateral venose a in le atrio dextere.

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T. W. MOIR and W. H. PRITCHARD

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