Interarterial Coronary Anastomoses

Occurrence in Normal Hearts and in Certain Pathologic Conditions

By Bertram Pitt, M.D.

It is generally recognized that interarterial coronary anastomoses of functionally significant size occur with great frequency in the presence of occlusive coronary artery disease and in other conditions such as anemia and cardiac hypertrophy in which myocardial anoxia may be present. Disagreement has been expressed by some investigators regarding the incidence of these anastomoses in normal and abnormal hearts. The problem has therefore been re-investigated by means of a technic of infusion of saline solution and injection of wax spheres of known size. The results of these observations in 75 hearts are presented and their significance is discussed.

CHANNELS connecting the right and left coronary arteries were first called attention to by Richard Lower of Amsterdam in 1669. The Swiss anatomist Albrecht von Haller demonstrated these anastomoses by dissection of the coronary arteries. The existence of these anastomoses was subsequently denied by Hyrtl, Henle, and Cohnheim, the latter stating that the coronary arteries were true end-arteries. In opposition to the endartery theory Krause, Langer, West, and others again claimed that the coronary arteries communicated through precapillary anastomoses.

It is generally recognized that interarterial coronary anastomoses (greater than 40 μ) occur with great frequency in the presence of occlusive coronary artery disease and its sequelae. Anastomoses have also been found in such conditions as hypertrophy of the myocardium, valvular disease, anemia, and emphysema of the lungs. Cardiac hypoxia has been suggested as the common cause of all these conditions.

The important question as to the frequency of occurrence of interarterial coronary anastomoses in normal hearts is however unsettled. Schlesinger et al. using a roentgen-plus dissection method in their investigation of over 1,000 hearts were able to demonstrate interarterial coronary anastomoses (greater than 40 μ) in only 9 per cent of normal hearts. They did, however, assume the presence of abundant fine capillary anastomoses. This is shown by the fact that aqueous solutions injected into one coronary artery are always seen elsewhere in the heart. Their work has been confirmed by Ravin and Greever and by Maili and Bledsoe using similar methods. A second view is that interarterial coronary anastomoses (larger than 40 μ) are present in a majority of normal hearts. This view is supported by the work of Prinzmetal et al. using an injection method. The same conclusion was reached by Baroldi and Mantero who used a corrosion method. Vastesaeger and Vander-Stratten using a refined stereoscopic x-ray method, and most recently by Laurie and Woods using a modification of the roentgen-plus-dissection method.

On the basis of the work of Schlesinger and Zoll, Beck assumed that only 9 per cent of the normal population have interarterial anastomoses and that the anastomoses resulting from myocardial hypoxia are inadequate for the prevention of infarction. On the further assumption that these channels are an important factor in the fate of patients with coronary artery disease, Beck and others have designed a number of operations to

From The Department of Medicine, Professor H. Staub, Director, and the Pathological Institute, Professor A. Werthemann, Director, University of Basel, Switzerland.
increase the collateral circulation of the ischemic heart.27 It has been said that "the justification for Beck's operation rests on experimental and postmortem evidence that occlusive coronary artery disease cannot promote an effective intercoronary circulation until it is severe."

Since the indication for surgery and the prognosis of occlusive coronary artery disease may be dependent on the presence or absence of interarterial coronary anastomoses (greater than 40 μ) in normal hearts, a reinvestigation of the problem with another method seems justified.

**METHODS**

Seventy-five hearts (table 1) were chosen at random from the autopsy material of the University Pathological Institute. The case material consisted of Swiss Caucasians varying in age from 0 to 90 years. After the investigation for anastomoses a routine pathologic examination was performed.

The heart was removed from the cadaver 5 to 45 hours (average 24 hours) after death and inspected for the presence of rigor mortis. The aorta was then dissected free from the pulmonary artery, and ligatures were placed under the coronary arteries at a point a few millimeters distal to their exit from the sinuses of Valsalva. In adipose hearts it was necessary first to free the coronary arteries from the overlying fatty tissue before placing the ligatures. The aorta was then opened by means of 2 longitudinal cuts that passed within 1 mm. of the valvular ring. A metal catheter was inserted into each of the coronary ostia after which the ligatures were secured. The heart was then suspended from a cross-bar by means of a wire passing through one of the pulmonary veins. A constant pressure flask (fig. 1), adjusted to the appropriate height to exert a maximum pressure of 100 mm. Hg, was then connected to the metal catheters and 100 ml. of physiologic saline were injected into the left coronary artery. If fluid was not seen flowing from the right coronary artery the direction of perfusion was changed and 100 ml. of physiologic saline were injected into the right artery. Once the fluid flowed from the opposite coronary artery a suspension of wax spheres,29 35 to 45 μ and 75 to 90 μ in diameter (fig. 2) was injected into the tubing. Another 100 ml. of fluid were then passed through the coronary arteries at the same pressure, and the fluid that

<table>
<thead>
<tr>
<th>Table 1.—Occurrence of Interarterial Coronary Anastomoses in Normal Hearts and in Certain Pathologic Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>-------------------</td>
</tr>
<tr>
<td>Normal hearts</td>
</tr>
<tr>
<td>1. History of anemia</td>
</tr>
<tr>
<td>2. No history of anemia, &quot;normal series&quot;</td>
</tr>
<tr>
<td>Pathologic hearts</td>
</tr>
<tr>
<td>1. Oclusive coronary sclerosis* with fibrosis of the myocardium</td>
</tr>
<tr>
<td>2. Oclusive coronary sclerosis with complete occlusion and infarction</td>
</tr>
<tr>
<td>3. Oclusive coronary sclerosis without fibrosis or infarction</td>
</tr>
<tr>
<td>4. Fibrosis of the myocardium without occlusive coronary sclerosis</td>
</tr>
<tr>
<td>5. Infarction without occlusive coronary sclerosis</td>
</tr>
<tr>
<td>6. Valvular lesion plus hypertension</td>
</tr>
<tr>
<td>7. Hypertensive heart disease plus hypertrophy</td>
</tr>
</tbody>
</table>

*Oclusive coronary sclerosis refers to those cases in which the coronary arteries were found to be markedly narrowed.
came out of the artery was collected on a filter. This filter lay in a funnel that had been inserted into a suction bottle. The difference in height of the coronary ostia and that of the free end of the tubing suspended over the suction flask was 5 cm. The filter paper was then removed from the suction flask and examined microscopically. The presence of spheres was considered evidence for the occurrence of interarterial coronary anastomoses. The investigation was completed in 15 minutes, so that this method could be used in conjunction with the routine autopsy. In contrast to other methods, the heart remains intact for further pathologic examination.

A disadvantage to this method is that the anastomoses cannot be localized. The possibility also exists that anastomoses may not be demonstrated even if present. This would apply particularly when one of the main branches of a coronary artery is occluded proximal to its collateral channel. The suspension of spheres could then pass from the patent artery through the anastomoses but could not leave through the occluded ostium. Another technical problem is that perfusate escaping from the vascular bed can enter the myocardium, and simulate infarction. This necessitated the histologic confirmation of all suspected infarcts.

RESULTS

Results are shown in table 1. Anastomoses were found in only 6 per cent (1 out of 15) of the hearts in the “normal series.” In those cases with occlusive coronary artery disease, and fibrosis of the myocardium or infarction, anastomoses were found in 75 to 100 per cent of the cases. With occlusive coronary disease but without fibrosis or infarction, anastomoses were found in 25 per cent of the cases. In hypertensive heart disease and in hearts with valvular lesions, anastomoses were demonstrated in 43 to 50 per cent of the cases.

Of the 3 cases of normal hearts with anastomoses 2 were not included in the “normal series,” since anemia (hemoglobin 70 per cent or less)\textsuperscript{14, 21} is known to be a factor in the production of anastomoses. The other patient with anastomoses in a normal heart had a history of epileptic attacks for the past 9 years. The inclusion of this case in the “normal series” may also be questioned, since epilepsy can produce hypoxemic changes.\textsuperscript{30}

No relationship could be found between the age at time of death or sex and the presence of absence of anastomoses.

DISCUSSION

Anastomoses were found in only 6 per cent of normal hearts but could be readily demonstrated in many pathologic hearts especially with occlusive coronary artery disease and fibrosis of the myocardium. These results are in agreement with those of Schlesinger, Blumgart, Zoll and others using the roentgen-plus-dissection method.\textsuperscript{14, 15, 18, 23}

It is interesting that our results do not agree with those of Prinzmetal,\textsuperscript{19, 24} who used a similar injection method. A comparison of the 2 methods disclosess some factors that could account for the differences of results. 1. In the present study the hearts were examined on an average of 24 hours post mortem at a temperature of 22 C., whereas Prinzmetal injected the hearts after keeping them for 24 hours at 4 C. and 4 hours at 37 C. 2. We used wax spheres (fig. 2) (35 to 45 and 75 to 90 $\mu$) in a perfusate of physiologic saline, whereas Prinzmetal used glass spheres of varying diameters (40 to 200 $\mu$) in a perfusate with a viscosity close to that of blood. The glass spheres used by Prinzmetal have
been shown to have jagged and sharp speeules.31 3. We used a maximum pressure of
100 mm. Hg both for perfusion and injection, whereas Prinzmetal used a pressure of 200
mm. Hg for perfusion and 160 mm. Hg for injection.

It is unlikely that the first 2 factors are
mainly responsible for the differences in re-
results. Although the author examined the
hearts for the absence of rigor mortis, there
may have been some residual vascular spasm
remaining in the smaller vessels; but this can-
not account for the finding of anastomoses
only in the pathologic hearts. The second and
third factors, perfusion and injection pressure
in conjunction with the sharp spicules on the
spheres, perhaps account for the difference in
results. Wiggers has stated that a pressure of
100 mm. Hg is slightly higher than should
be used.32 The pressure in an occluded cor-

nary artery is not that of 0 but 20 to 30 mm.
Hg during diastole and 40 to 50 mm. Hg
during systole. The difference between the
pressures in the functioning and in the oc-
ccluded coronary artery is therefore less than
would be expected and is perhaps within the
range of 60 to 80 mm. Hg. We have therefore
used a pressure of 65 mm. Hg for both per-
 fusion and injection and found it to be ade-
quate in many cases. Baroldi et al.17 and Vas-
tesaeger et al.25 have also used excessively
high pressures for injection. Although
Schlesinger et al.14, 15 have used pressures up
to 200 mm. Hg, they were able to demonstrate
anastomoses in only 9 per cent of normal
hearts. This can perhaps be explained by their
use of a perfusate that did not penetrate uni-
formly to vessels smaller than 40 μ. The walls
of the smaller arterioles and capillaries were
therefore not subjected to the high pressures.
An adequate explanation of Laurie’s and
Woods’26 finding of frequent anastomoses in
normal hearts, with use of a slight modifica-
tion of Schlesinger’s method must await fur-
ther study. It is therefore possible with a
pressure of 200 mm. Hg that the anastomoses
demonstrated with such frequency by Prinz-
metal and associates19, 24 are the result of
stretching pre-existing capillary channels and
of creating artificial communications.

It is probable that the anastomoses (greater
than 40 μ) found in hearts with occlusive
coronary artery disease are functional in
many instances and that coronary arteries are
not physiologic end-arteries as suggested by
Porter in 1896.9 This view is supported by
the experimental work of Kolster10 and by
many observed cases that have complete oc-
cclusion of a coronary artery without evidence
of ischemia or infarction.33-35 Further evi-
dence is that infarction may occur in an area
whose primary vessel is patent but whose col-
leral supply is occluded (“infarction at a dis-
tance”).15, 36-37 In normal hearts the abun-
dant capillary network connecting the cor-

nary arteries cannot be expected to serve as
a functional collateral blood supply because
of the high resistance across these channels.9
If there are interarterial anastomoses in nor-
mal hearts (greater than 40 μ), no substantial
evidence of their function has as yet been
presented. Spalteholz and Hirsch showed ex-
perimentally that the size of an infarcted area
is smaller than that of the area supplied by
the obstructed vessel.12, 38 Prinzmetal used
this observation to justify the presence of
anastomoses in normal hearts, stating, “the
degree of collateral circulation in the (nor-
mal) heart is not sufficient to prevent an in-
farction following obstruction to a major

Fig. 2. Spheres 75 to 90 μ, 100 × enlarged.
coronary artery but may limit the size of the infarction.  

Wiggers, on the other hand, suggested that it is more likely that the size of the infarction is smaller because of diffusion from the surrounding myocardium.  

It has also been shown that the size of the infarct cannot be correlated with survival or death of the individual after infarction.  

On the basis of present evidence the functional significance of these anastomoses in normal hearts, even if present, is uncertain.

Although the present results support those who advocate procedures for establishing prophylactic anastomoses in normal hearts, a certain amount of caution is in order. Beck has applied his operations mainly to patients with angina pectoris or previous myocardial infarction.  

It has been adequately shown that these are the very people who have an anastomotic circulation. It can be argued that the anastomotic circulation is inadequate for the needs of the myocardium and that additional help afforded by the operation might give the heart sufficient reserve to relieve angina pectoris or to prevent infarction. The evaluation of whether the actual increase in anastomoses or whether psychologic factors are responsible for the results obtained by Beck and others will have to await careful follow-up of patients and controlled studies.

**Summary**

A method is presented for the study of interarterial coronary anastomoses. Wax spheres (35 to 45 and 75 to 90 μ) were injected into one coronary artery at a maximum pressure of 100 mm Hg. The finding of spheres in the opposite coronary artery was considered positive evidence for the presence of anastomoses.

A total of 75 hearts randomly selected were studied with this method. Of the 15 normal hearts only 1 (6 per cent) was found to have anastomoses. In those cases with occlusive coronary artery disease, fibrosis of the myocardium and infarction anastomoses were found in 75 to 100 per cent of the cases. In hypertensive heart disease and in hearts with valvular lesions, anastomoses were demonstrated in 43 to 50 per cent of the cases.

These results are in agreement with those of Schlesinger, Blumgart, and Zoll, who used the roentgen-plus-dissection method but are in disagreement with the finding of anastomoses (greater than 40 μ) in the majority of normal hearts by Prinzmetal by means of an injection technic similar to the one used in this study.

The factors accounting for the difference in results between this study and others are discussed.

**Acknowledgment**

The author wishes to express his gratitude and appreciation for the constant help and encouragement of Dr. W. Schweizer, Department of Medicine, University of Basel, and to thank Professors Werthemann and Scheidegger, Pathological Institute of Basel, for the generous use of the autopsy material and the pathologic diagnoses.

**Summario in Interlingua**

Es presentate un metodo pro le studio de anastomoses inter arterias coronari. Spheres de cera (con diametros de 35 a 45 e de 75 a 90 μ) eseva injicite in un arteria coronari sub un pression maximal de 100 mm de Hg. Le constatation del presentia de spheres de cera in le opposite arteria coronari eseva considerate como prova positive pro le existentia de un anastomose.

Un total de 75 cordes, seligite per randomisation, eseva studiate per medio de iste methodo. Inter le 15 cordes normal inclusit in le serie, anastomose eseva constatate in solmente 1 caso (6 pro cento). In le gruppos de cordes con morbo occlusive de arteria coronari, con fibrosis del myocardio, e con infarciamento, anastomoses eseva constatate in inter 75 e 100 pro cento del casos. In casos de hypertensive morbo cardiae e in cordes con lesiones valvular, anastomoses eseva presente con un frequentia de inter 43 e 50 pro cento.

Iste resultatos es de accordo con le constataziones de Schlesinger, Blumgart, e Zoll, qui laborava con un metodo a roentgenographia e dissection, sed illos non concorda con le constatazione de Prinzmetal quie reporta le pre-
sentiad de anastomose (de plus que 40 μ) in le majoritate del cordes normal super le base de investigations con un technica injectional simile al technica usate in le presente studio.

Es discuite le factores que pote explicar le differentia inter le resultatos del presente studio e illos de studios per altere auteurs.

REFERENCES

2. Haller, Albrecht van: Quoted by von Redwitz, E. F. 13
30. Scheidegger, S.: Personal communication.


Neither is it true which is commonly believ'd, that the heart by any motion or distention of its own doth draw blood into the ventricles, but that whilst it is moved and bended, the blood is thrust forth, and when it is relax'd and falls, the blood is received in manner as follows.—William Harvey. De Motu Cordis, 1628.
Interarterial Coronary Anastomoses: Occurrence in Normal Hearts and in Certain Pathologic Conditions
BERTRAM PITT

Circulation. 1959;20:816-822
doi: 10.1161/01.CIR.20.5.816
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1959 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/20/5/816

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/