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 reinvestigated 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 end-artery 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 Greetchev, 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, Vastesager and Vander-Straten 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
INTERARTERIAL CORONARY ANASTOMOSES

increase the collateral circulation of the ischemic heart. 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." 28

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 re-investigation 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 adipo 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 coronary 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><strong>No. of cases</strong></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. Occlusive coronary sclerosis* with fibrosis of the myocardium</td>
</tr>
<tr>
<td>2. Occlusive coronary sclerosis with complete occlusion and infarction</td>
</tr>
<tr>
<td>3. Occlusive 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 hypertrophy</td>
</tr>
<tr>
<td>7. Hypertensive heart disease plus hypertrophy</td>
</tr>
</tbody>
</table>

*Occlusive 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) 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.

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.

It is interesting that our results do not agree with those of Prinzmortal, who used a similar injection method. A comparison of the 2 methods discloses 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 Prinzmortal 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 μ) in a perfusate of physiologic saline, whereas Prinzmortal used glass spheres of varying diameters (40 to 200 μ) in a perfusate with a viscosity close to that of blood. The glass spheres used by Prinzmortal have...
been shown to have jagged and sharp spicules.\textsuperscript{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 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 cannot 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.\textsuperscript{32} The pressure in an occluded coronary 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 occluded 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 perfusion and injection and found it to be adequate in many cases. Baroldi et al.\textsuperscript{17} and Vastaesager et al.\textsuperscript{25} have also used excessively high pressures for injection. Although Schlesinger et al.\textsuperscript{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 uniformly to vessels smaller than 40 \(\mu\). The walls of the smaller arterioles and capillaries were therefore not subjected to the high pressures. An adequate explanation of Laurie's and Woods\textsuperscript{26} finding of frequent anastomoses in normal hearts, with use of a slight modification of Schlesinger's method must await further study. It is therefore possible with a pressure of 200 mm. Hg that the anastomoses demonstrated with such frequency by Prinzmetal and associates\textsuperscript{19, 24} are the result of stretching pre-existing capillary channels and of creating artificial communications.

It is probable that the anastomoses (greater than 40 \(\mu\)) 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.\textsuperscript{9} This view is supported by the experimental work of Kolster\textsuperscript{10} and by many observed cases that have complete occlusion of a coronary artery without evidence of ischemia or infarction.\textsuperscript{33–35} Further evidence is that infarction may occur in an area whose primary vessel is patent but whose collateral supply is occluded ("infarction at a distance").\textsuperscript{15, 36–37} In normal hearts the abundant capillary network connecting the coronary arteries cannot be expected to serve as a functional collateral blood supply because of the high resistance across these channels.\textsuperscript{9} If there are interarterial anastomoses in normal hearts (greater than 40 \(\mu\)), no substantial evidence of their function has as yet been presented. Spalteholz and Hirsch showed experimentally that the size of an infarcted area is smaller than that of the area supplied by the obstructed vessel.\textsuperscript{12, 38} Prinzmetal used this observation to justify the presence of anastomoses in normal hearts, stating, "the degree of collateral circulation in the (normal) heart is not sufficient to prevent an infarction following obstruction to a major.
coronary artery but may limit the size of the infarction.77,19 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.32 It has also been shown that the size of the infarct cannot be correlated with survival or death of the individual after infarction.39 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.40 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 technique 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 Scheiddegger, 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 metodo. Inter le 15 cordes normal includite 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 infariciamento, anastomoses eseva constatate in inter 75 e 100 pro cento del casos. In casos de hypertensive morbo cardiac e incorde con lesions valvular, anastomoses eseva presente con un frequentia de inter 43 e 50 pro cento.

Iste resultatos es de accordo con le constatazioni 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-
sentia de anastomose (de plus que 40 μ) in le
majoritate del cordes normal super le base de
investigationes con un technica injectional
simile al technica usate in le presente studio.

Es discutite le factores que pot explicar
le differentia inter le resultatos del presente
studio e illos de studios per altere autores.

REFERENCES
1. LOWER, R.: Quoted by Zoll, P. M., et al.14
2. HALLER, ALBRECHT VAN: Quoted by von Red-
witz, E. F.13
3. HYRTL, J.: Quoted by von Redwitz, E. F.13
4. HENLE, J.: Quoted by von Redwitz, E. F.13
5. COHNHEIM, J., von SCHULTHEESS-RECHBERG,
   A.: Ueber die Folgen der Kranzarterien
   verschliessung für das Herz. Virchows Arch.
   Path. 85: 503, 1881.
6. KRAUSE: Quoted by von Redwitz, E. F.13
7. LANGER, L.: Quoted by von Redwitz, E. F.13
8. WEST, S.: The anastomoses of the coronary
   arteries. Lancet 1: 945, 1883.
9. PORTER, W. T.: Results of ligation of the
coronary arteries. J. Exper. Med. 1: 46,
   1896.
10. KOLSTER: Experimentelle Beiträge zur Lehre
der Myomalacia cordis. Skandinav. Arch.
    Physiol. 4: 1, 1892.
11. MERKEL, H.: Zur Kenntnis der Kranzarterien
    des menschlichen Herzen. Verhandl. deutsch.
12. HIRSCH, C., and SPAETENHOLZ, W.: Koronar-
kreislauf und Herzmuskelarterien. Deutsche
    med. Wechschr. 33: 790, 1907.
13. von REDWITZ, E. F.: Der Einfluss der Er-
krankung der Koronararterien auf den
   Herzmuskel mit besonderer Berücksichti-
gung der chronischen Aortitis. Virechows
   Arch. Path. 197: 433, 1909.
14. ZOLL, P. M., WESSLER, S., and SCHLESINGER,
    M. J.: Interrarterial anastomoses in the
    human heart, with particular reference to
    anemia and relative cardiac anoxia. Circu-
    lation 4: 797, 1951.
15. SCHLESINGER, M. J.: An injection plus dissec-
tion study of coronary occlusions and anas-
16. BLUMGART, H. L., SCHLESINGER, M. J., and
    DAVIS, D.: Studies of the relation of the
    clinical manifestations of angina pectoris,
coronary thrombosis and myocardial infarc-
tion to the pathological findings. Am. Heart
   J. 19: 1, 1940.
17. BAROLDI, G., MANTERO, O., AND SCOMAZZONI,
    G.: The collaterals of the coronary arteries
   in normal and pathological hearts. Circula-
18. MAILL, J. B., AND BLEDSOE, A.: Pathological
    anatomy of coronary heart disease. Arch.
    Path. 56: 577, 1953.
19. PRINZMETAL, M., SIMKIN, B., BERGMAN, H. C.,
    and KRUGER, H. E., Studies on the coro-
    nary circulation. II. The collateral circu-
    lation of the normal human heart by coro-
    nary perfusion with radioactive erythro-
cytes and glass spheres. Am. Heart J. 33:
    420, 1947.
20. MANTERO, O., BAROLDI, G., AND SCOMAZZONI,
    G.: The coronary arterial circulation in the
    hypertrophic heart. Cardiologica 1: 48,
    1958.
21. ECKSTEIN, R. W.: Development of interar-
terial anastomoses by chronic anemia. Cir-
22. ZIMMERMAN, H. A.: The coronary circulation in
    patients with severe emphysema, cor pul-
    monale, cyanotic congenital heart disease,
    and severe anemia. Dis. of Chest 22: 269,
    1952.
23. RAVIN, A., AND GREEVEN, F. F.: Coronary ar-
teriosclerosis, coronary anastomosis and
    myocardial infarction. Arch. Int. Med. 78:
    125, 1946.
24. PRINZMETAL, M., KAYLAND, S., MARGOLES, C.,
    AND TRAGERMAN, L. J.: A quantitative
    method for determining collateral coronary
    circulation. J. Mt. Sinai Hosp. 8: 933,
    1941-2.
25. VASTESAEGER, M. M., STRAETEN, P. P., VAN
    DER, FRIART, J., CANDAELLE, G., GYTS, A.,
    AND BERNARD, R. M.: Les anastomoses in-
tercoronariennes telles qu’elles apparaissent
    a la coronarographie post mortem. Acta
26. LAURIE, W., AND WOODS, J. D.: Anastomosis of
    the coronary circulation. Lancet 2: 812,
    1958.
27. BECK, C. S., AND LEIGHNIGER, D. S.: Opera-
tions for coronary artery disease. Lancet
    1: 1025, 1957.
28. EDITORIAL: Surgical treatment of ischemic
29. EMMENEGGER, H., HURLIMANN, B., AND
    BUCHER, K.: A simple method of producing
    radioactive spheres for the investigation of
    pharmacol. 9: 254, 1951.
30. SCHEIDEgger, S.: Personal communication.
31. ZOLL, P. M.: Personal communication.
32. WIGGERS, C. J.: The problem of functioning
    coronary collaterals. J. Exper. Med. 8: 402,
    1950.

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/