Editorial

Anatomy and Functional Importance of Intercoronary Arterial Anastomoses

The development of interarterial coronary anastomoses profoundly influences the clinical course of angina pectoris and acute myocardial infarction and is important in lessening the extent of possible pathologic myocardial changes. The studies by Pitt recorded elsewhere in this issue are a contribution to the problem of the anatomic and functional characteristics of the coronary arterial system that has been debated for centuries. In 1669, Lower stated, "Hence it sometimes happens that, when the lumen of some artery has been too long obstructed or ligated, the blood busies itself in opening a wider channel for its passage in this vessel, must drive and buffet all the more into the next ones, until it has considerably dilated them to give itself room."

Lower did not state, however, whether he had observed interarterial anastomoses between the coronary arteries in the normal heart. In the normal heart, the anatomists have maintained that the coronary arteries are not end arteries.

Proof of their contention is afforded by the observation that a watery solution injected into one coronary artery is immediately seen in all the other cardiac arteries. The physiologists, however, maintain that functionally the coronary arteries are end arteries inasmuch as sudden ligation or occlusion of a main stem always produces myocardial infarction.

If a coronary artery is gradually occluded experimentally or by atherosclerotic narrowing, numerous observers have observed the development of functionally important intercoronary communications. These anastomotic collateral channels communicate between coronary arteries serving to nourish areas beyond complete occlusions that would otherwise undergo necrosis; others serve as bypasses or detours connecting the artery proximal to the occlusion with the distal portion. Experimentally it has been shown that an animal may survive gradual occlusion of one or more main coronary arteries with little myocardial damage. Indeed, complete occlusion of a main coronary artery gradually accomplished in successive stages often was not accompanied by myocardial infarction in the dog. Similar experimental observations were made by Blum, Schauer, and Calef; by Burchell; by Gregg, and by us. The clinical counterpart of these experiences, i.e., the occurrence of complete coronary artery occlusions without myocardial infarction, has been noted by Saphir and his associates, by Bean, and by ourselves. Recently, however, Snow, Jones, and Daber have questioned the degree to which the collateral vessels protect the myocardium from necrosis. They always found gross myocardial infarction of some extent in association with occlusions, although often considerably smaller than might otherwise have been expected. Since their observations were made in a series of 25 patients, limited to those with clinical manifestations of coronary disease, instances of complete protection from infarction may well have been eliminated from consideration at the outset by the method of selection. In any event there is no question about the development of intercoronary anastomoses in response to obstructive coronary disease and their importance in lessening the consequences of coronary artery occlusion. By the Schlesinger lead-agar or barium-gelatin injection technic, the functionally important collateral channels were

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found to be generally more than 40μ in diameter. Using a wholly different technic of injection of wax spheres, Pitt has reached similar conclusions. Likewise, Baroldi et al., utilizing a corrosion technic, found increased anastomoses in relation to atherosclerotic occlusions and narrowings.

The hearts of patients with angina pectoris usually show one or more occlusions of the coronary arteries, a rich collateral development, and variable myocardial fibrosis or infarction. While there is a general relationship between the incidence of coronary occlusion and the occurrence of angina pectoris, other modifying factors, such as the exact site of the occlusion, the importance of the vessel involved, the adequacy of the collateral circulation, the rates at which such occlusions or narrowings develop, and the temporary influence of emotion and of vasomotor reflexes, are also of great importance. Although damage to the heart is minimized by the development of the collateral circulation, the margin of safety, or, as it may be termed, "the coronary reserve," is reduced.

In contrast to the general agreement of the findings in the hearts with atherosclerotic narrowings and occlusions, considerable divergence of results has been reported regarding the presence of intercoronary anastomoses in the normal heart. In an extensive series of over 1,500 consecutive normal hearts studied by the Schlesinger technic, the incidence of anastomoses was 9 per cent after exclusion of anemia, cardiac hypertrophy, valvular heart disease, and other categories in which hypoxia evidently leads to an increased incidence of anastomotic development. Although this experience has been confirmed by many others, including the current report by Pitt, in normal hearts Baroldi and co-workers using a corrosion technic, Vastesaeger and collaborators by stereoscopic radiography after injection of Lipiodol, and Laurie and Woods, who used an injection technic, found anastomoses of greater size and with greater frequency. Laurie and Woods, indeed, alone among investigators, observed sizable inter-

coronary arterial anastomoses in 75 per cent of patients over 4 years of age and in only 23 per cent of patients with severe atherosclerosis. As they state, "much of the difference of opinion has probably arisen due to differences in technique." It is to be hoped that these workers will study a series of cases, observing the precautions and experimental methods found by many other investigators to be reliable, in order to determine whether their exceptional results are due to racial differences in the Bantu, whether the presence of anemia in their cases accounts for their results, or whether the differences are due to experimental errors.

Parallel to the anatomic studies described above are the results of the physiologic studies of Wiggers, Gregg, and others that amplify the meaning of the morphologic observations disclosed by the injection technics. An indication of the magnitude of intercoronary collateral development can be demonstrated experimentally by measurement of retrograde flow and pressure from a severed main coronary branch. Immediately following abrupt occlusion of a main coronary branch, the retrograde coronary flow approximates 5 to 5.8 ml. per minute, as compared to control values of 2 to 3 ml. per minute, and is relatively constant in any one dog for a few hours. Measurements of retrograde flow during temporary clamping of the other coronary arteries indicate that these arteries are the major source of flow.

After long-continued obstruction of a coronary artery or a branch in an otherwise normal animal heart, the flow of blood from the cannulated end of the artery becomes quite large. It begins to increase within a few hours, may double within 2 days, and become 3 to 4 times the control level within a week. Within a few weeks, the flows approximate the values for the normal rate of inflow before occlusion in that coronary artery or branch. The observation that the retrograde blood has the same content of oxygen and carbon dioxide as that in a
systemic artery leaves no room for doubt that the collateral circulation is on the arterial side of the coronary capillary bed. The gradual augmentation of retrograde flow is attended by similar elevations of systolic and diastolic pressures in the peripheral end of the occluded coronary artery.

Additional evidence of the functional significance of the collateral vessels is afforded by experimental myographic studies. In the normal dog heart, ligation of a main coronary artery causes the ischemic area to stretch rather than to contract in systole. The intercoronary communications are too small to transfer adequate quantities of blood. Myographic records taken from an area supplied by a slowly occluded artery in dogs indicate that the region is undergoing shortening and that the newly developed collateral channels are functioning in adequate fashion.

Observations of the extent and size of the intercoronary anastomoses in the heart post mortem in man are in accordance with these experimental studies. That the collateral circulation protects the myocardium from damage is generally agreed; a difference of opinion exists only in regard to the degree of protection that is conferred. Available evidence indicates that although sizable intercoronary anastomoses are visible within days after a sudden complete coronary occlusion or sudden narrowing, several weeks are necessary for their rich development and several months may elapse before their full potentiality is realized.

The slow development of these collateral channels emphasizes the importance of rest and reduced activity for many weeks after acute myocardial infarction, contrary to the current tendency to earlier ambulation. Ample evidence exists that reduced cardiac work favors healing of the infarct, reduces the extent of myocardial damage, lessens liability to rupture, and provides time for the development of these anastomotic channels. The slow development of a richer anastomotic circulation is also apparently responsible for the occasional clinical improvement of patients with angina pectoris, the collateral channels acting to offset the narrowing or even the occlusion that has occurred.

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REFERENCES


'The knowledge which a man can use is only real knowledge, the only knowledge which has life and growth in it, converts itself into practical power. The rest hangs like dust about the brain or dries like rain-drops off the stones.' (Froude)—William Osler, M.D. After Twenty-five Years. Montreal Med. Journal, 1899.
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