EDITORIAL

Inequalities of Myocardial Perfusion in Coronary Artery Disease ("Coronary Steal")

HYPOXIA HAS been long known to cause a dramatic decrease in resistance in the coronary vascular bed. In the presence of localized ischemia due to coronary artery disease, pronounced reduction of vascular resistance would presumably result in a marked drop in the pressure head in the parent artery, distal to a proximal stenotic lesion. Such reduction in the pressure head conceivably could reduce perfusion in the terminal reaches of that parent vessel. The concept is simple; the hemodynamic possibilities are not.

Renewed impetus was given to study of the role of arterial collateral channels by the report that exercise of an arm, in the presence of stenosis at or near the origin of the subclavian artery, could give rise to neurologic symptoms.1 This syndrome, related to an increased reversed flow through the vertebral artery, became known by the catchy title of "subclavian steal." Presumably vascular dilatation in the arm muscles decreases the pressure in the subclavian and vertebral arteries, in turn decreasing the perfusion pressure in the brain stem. Thus blood is "stolen" by the upper extremity, with resulting symptoms of an ischemic brain. It is apparent that the nature of the communications in the circle of Willis is important in the development of the syndrome. The concept of vascular "steal" has become popular, and aortoiliac2 and mesenteric steals3 have been described.

For a long time it has been known that, when a coronary artery arises from the pulmonary artery, it may be subject to a low pulmonary artery pressure, inadequate for myocardial perfusion. Brooks4 early pointed this out. Amongst others, Effler and associates5 suggested the term "coronary steal" for this situation, wherein flow is reversed in an anomalous coronary artery connected to the cardiac chambers or pulmonary artery. Measurement of pressures within these aberrant coronary vessels in my laboratory confirmed the theoretical pressure relations.6 Indicator dye, injected into an aberrant artery while the attachment to the pulmonary artery was occluded, progressed to the myocardium and through collateral vessels to the normally attached adjacent coronary artery. When the experimental occlusion of the pulmonary artery was released, pressure in the anomalous coronary artery fell, and the indicator was flushed from the myocardium into the anomalous vessel and into the pulmonary artery. One could thus say that blood was "stolen" from the coronary circulation and lost into the pulmonary artery with resultant inadequate pressure in the coronary system to perfuse the myocardium.

The dynamics of flow must be quite complicated in a diseased, multi-obstructed coronary arterial tree. When the proximal portion of one coronary artery is obstructed, however, coronary arteriography commonly reveals filling of its distal portions through collaterals arising from the opposite coronary artery. Similar collaterals are demonstrable when both coronary arteries are partially occluded; contrast material injected into the right coronary artery then may fill the left anterior descending coronary artery from a collateral, and injection into the left coronary artery will also show it to fill through the residual lumen. Under these circumstances the flow in a collateral vessel would depend upon the pressures at either end, and the blood would flow toward the bed which had the lower resistance because of local vasodilation. When local vasodilatation occurs in the distal ramifications of an artery, flow might well be reversed in the collaterals joined to that artery.
Available data indicate that certain coronary vasodilator drugs may precipitate angina pectoris even though they are known to increase total coronary blood flow and the oxygen content of coronary sinus blood. In dogs with a well-developed collateral circulation, dipyridamole has resulted in a significant reduction in retrograde flow into a coronary artery distal to its occlusion. A recent report indicates myocardial lactate production during administration of Isuprel at a time when the coronary sinus oxygen content was increased. It has been postulated that these phenomena occur because of dilatation of the resistance vessels in the myocardium which usually restrict flow through normal segments and thus maintain the perfusion pressure required to supply the myocardium distal to coronary artery constrictions. If the normal arterioles are dilated and thereby permit rapid flow, the pressure in large coronary arteries decreases and blood is "stolen" from ischemic areas. Wean's description of intramyocardial vascular network suggests that shunting of blood could occur through normal channels within the myocardium. Whether this is important in drug-induced myocardial ischemia is not known.

It is not yet certain that "coronary steal" occurs although illustrations have been given of circumstances where it seems likely, viz., as a result of congenital coronary anomalies, acquired coronary artery disease, and pharmacologically induced readjustments within the intramyocardial vascular network. We probably will not know whether "steal" occurs until direct measurement of blood flow can be made in individual arteries and collateral vessels. Meanwhile the circumstantial evidence for steal is reasonably good, and if the concept is useful in stimulating further thought and research, it should be retained.

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References

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_Circulation_. 1970;42:193-194
doi: 10.1161/01.CIR.42.2.193

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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http://circ.ahajournals.org/content/42/2/193.citation