Regional Myocardial Perfusion during Ischemia

To the Editor:

In a recent article Maseri et al. report their data on regional myocardial perfusion and its response to atrial pacing in a selected group of patients with coronary artery disease. Their conclusions include two postulates worth closer scrutiny: 1) some patients have ischemic myocardial areas even at rest, and 2) during ischemia induced by right atrial pacing the myocardial perfusion drops in some areas below the level prevailing at rest.

Analysis of the initial 133Xe slopes did not disclose any systematic difference between normal and poststenotic areas. This finding is in accord with the experience of others. The conclusion of ischemia at rest was based on the 133Xe residual activity at the time when 90% of the tracer was washed out. This activity was systematically greater in poststenotic areas than in normal areas, whereas the activities were similar 15 minutes after the injection. The authors conclude that the latter finding rules out the impact of fatty tissue. This may not be necessarily true since the last part of the decay, deviating from the initial monoeponential slope, may well be due to the amount of nonmuscular tissue in the respective segments. Another possibility is the lack of collaterals in these patients, a very strange clinical situation since almost all patients with ≥ 90% coronary obstructions exhibit collateral vessels. If the collaterals were really absent it would explain the difference in the final levels of the tracer by not contributing to the washout. In any event, a somewhat higher residual 133Xe activity in the poststenotic segments does not justify the conclusion of ischemia at rest, since this functional state is not analogous to a reduced myocardial perfusion in any given area.

The postulate that during ischemia the poststenotic regional perfusion drops below the basal level is not convincingly supported by the fragmentary data of Maseri et al. They say, "Although there was an increase in flow calculated for the whole heart during angina, similar to that reported in the literature and that observed in the control group, a severe reduction of myocardial perfusion relative to control areas occurred in poststenotic areas." My interpretation of this statement (no exact flow data presented) is that the flow increased in poststenotic areas to a lesser degree than in the normal areas. This would be in accord with the data of others. But the authors conclude in the last chapter of their discussion that "... during severe ischemia induced by raising myocardial demands above the potential supply, flow to the ischemic area may become dramatically impaired because of the addition of functional factors which in some areas of the myocardium reduce blood supply far below the resting level." This would indicate a vasoconstrictive component during pacing-induced ischemia effectively counteracting the potent ischemic vasodilatation, a possibility which I consider very remote, but of great pathophysiological interest. An explanation based on raising left ventricular end-diastolic pressure upon ischemia is insufficient since this variable would affect the entire left ventricular subendocardium. Since the postulates of Maseri et al. concern the whole "cold spot" imaging currently widely practiced, the presentation of the relevant flow data at rest and upon ischemia with appropriate statistical treatment would be imperative and superior to and more convincing than portraying one single patient in two separate figures (figs. 1 and 5).

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References


The author replies:
To the Editor:

Dr. Frick questions our interpretation of the Xenon data reported in Circulation. For the studies at rest he raises three points:

1) He suggests that the finding of a consistently greater residual activity at 90% Xenon washout in poststenotic areas with similar Xenon accumulation at 15 minutes may be related to "... nonmuscular tissue in the respective segments ..." rather than to the presence of localized areas of reduced perfusion. This hypothesis appears hardly tenable because the presence of tissue with high Xenon solubility should also affect the 15 minutes distribution of the indicator.

2) Then Dr. Frick proceeds to suggest that the reduced flow in poststenotic areas may be explained by the absence of collaterals. This hypothesis is conceivable, although the absence of large visible collaterals does not rule out the presence of several nonvisible collaterals.

3) He points out that the presence of a regional reduction of perfusion cannot be equated with ischemia in its strict metabolic meaning. We share his opinion because a reduced flow may be associated with reduced local metabolic demand.

For the studies during ischemia caused by pacing-induced tachycardia Dr. Frick questions our conclusion that "... during severe ischemia flow to the ischemic area may become dramatically impaired because of the addition of functional factors." We derived this conclusion from the findings of reduced poststenotic washout rates when pacing above the anginal threshold was started immediately after the injection of Xenon. The findings, illustrated in figure 9 of our article, do not seem "fragmentary," as suggested by Dr. Frick, since all five patients in whom ischemia appeared soon after the onset of pacing, behaved in the same way. By contrast, regional Xenon washout rates observed following Xenon injection during pacing-induced angina show only a lesser degree of increase in poststenotic than in normal areas rather than a reduction relative to control. The interpretation of this apparent paradox is to be found in a severe degree of inhomogeneity of perfusion in the myocardium included within the solid angle of poststenotic regions which results in very little deposition of indicator in the areas with very low flow relative to those with high flow. Model simulations indicate that following bolus injection, initial washout rates reflect adequately average flow when differences in perfusion range from 1 to about 0.2, but they are negligibly influenced by flow values that...
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