assessments of only non-Q-wave MIs, which dates from a period when the non-Q-wave MI was assumed to be a valid subset with unique characteristics. It is to be hoped that this misconception has been permanently put to rest and, further, that investigations of outcome after MI will avoid the egregious error of combining random mixtures of first and subsequent infarcts.

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REFERENCES


Does Flow Reserve Match Contractility?

I have read with great interest the report by Barilla et al. (1). The data reported are intriguing because, to the best of my knowledge, this is the first report indicating that the restoration of regional contractility during low-dose dobutamine administration may occur despite different perfusion patterns, depending on the presence or absence of collateral filling.

Let me raise an issue not addressed in the Discussion of Barilla’s article. I definitely agree with Bonow (2) that the increase in flow in patients with collateral filling is expected, because the drop of pressure beyond the fixed obstruction can increase the flow, despite the coronary driving pressure’s being unchanged. The no-measurable-flow response in patients without collateral channels can also be expected. In fact, why should the flow increase through a stenosis or an occlusion? Irrespective of flow regimen, the authors (1) noted an amelioration in contractility of dysfunctional myocardium—one that was still present at 2-methoxy-isobutyl-isonitrile (MIBI) administration and during the time allowed for it to distribute to the myocardium (i.e., up to 8 min), I presume, because no mention was ever made to subsequent deterioration of wall motion. This is an astonishingly long time, which would more appropriately define the response to low-dose dobutamine of stunned myocardium (but this was not the case, as indicated by the low sestamibi uptake). It seems inconceivable that such a prolonged increase in contractility may occur in the absence of an adequate increase in blood flow, the situation being absolutely different from the postextrastolic potentiation of contractility, when myocytes burn their energy stores all in one go. By contrast, the increase in contractility of ischemic but viable myocardium at low-dose dobutamine is a short-lived phenomenon: it may begin at very low dosage (as low as 2.5 μg/kg body weight per min, in our experience) and usually fades away at 10 μg/kg per min, seldom at 20 μg/kg per min. In patients with very severe coronary stenosis or coronary occlusion without collateral blood filling, a biphasic response to dobutamine should be seen at a dosage even lower than that at which the authors injected technetium-99m sestamibi. Given this, as well as the notion of the ischemic cascade (3,4), I make the point that Barilla et al. (1) described an intermediate phase of the biphasic response phenomenon—that is, the time when the flow reserve is exhausted, but wall contractility has not yet deteriorated in response to forthcoming or ongoing ischemia, or both. I suggest that this possibility is whispered to the reader.

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REFERENCES


REPLY

We are grateful to Dr. Barletta for his comments. Obviously, the findings we have reported constitute an intermediate step of a biphasic response phenomenon, as stated by Dr. Barletta. However, a 5-min step protocol for low-dose dobutamine echocardiography is common (1,2), and a biphasic response (i.e., wall motion improvement followed by worsening) is rarely observed at low doses of 5 to 10 μg/kg body weight per min (3). Nevertheless, no change in wall motion and thickening occurred during the 3 min after tracer injection, even when we used 10 μg/kg per min of dobutamine.

We also wish to emphasize that our study was not intended to describe the behavior of inotropic contractile reserve during low-dose dobutamine infusion, but it was aimed at investigating the pathophysiologic and clinical implications of the presumed mismatch between perfusion and contractility in areas with severely hypoperfused viable myocardium.

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REFERENCES

2. Panza JA, Dilsizian V, Laurienzo JM, Curiel RV, Katsiyannis PT. Relation between thallium uptake and contractile response to dobutamine: implications regarding myocardial viability in patients with