

## Editor's Note

# Do You Need Demonstrable Ischemia for Evidence-Based Decision-Making in Chronic Stable Angina?



**Key words:** myocardial ischemia, chronic stable angina

The key words in the question of whether demonstrable ischemia is necessary for evidence-based decision-making in chronic stable angina are “evidence-based.” My short answer to that question is yes, myocardial ischemia must be demonstrated in order to make evidence-based decisions.

Next question is “What is myocardial ischemia?” In the context of a high-grade stenosis, transient ST depression, radionuclide perfusion abnormalities, or left ventricular (LV) wall motion abnormalities observed independently prior to or after coronary angiography in the area perfused by the coronary artery in question are strong evidence for a pathologically significant coronary narrowing.

Next question is “How do evidence-based data from clinical trials relate to decision-making?” As everyone knows, in all clinical trials “evidence-based data” relate to populations, whereas decision-making is an individual exercise, not population based. For example,

1. The diagnosis of coronary artery spasm, requires evidence for transient narrowing of an epicardial coronary artery plus electrocardiographic (ECG) changes suggesting ischemia, usually ST-segment elevation.

2. In acute coronary syndromes, there is no question that many diagnostic and therapeutic decisions are made based on evidence; for example, (a) in transient ST-segment depression the evidence for ischemia is confirmed, and an aggressive medical strategy is employed; (b) in ST-segment depression associated with troponin I leak, the patient is placed into a higher-risk category, and although aggressive medical therapy is warranted, a revascularization strategy based on evidence is also warranted; (c) in ST-segment elevation, the elevation generally equates with occlusion of an epicardial coronary artery—thus, a revascularization strategy, either percutaneous coronary intervention (PCI) or thrombolytic therapy, based on evidence is warranted.

3. In patients with chest pain of uncertain etiology, clearly the first step is to establish the presence or absence of myocardial ischemia, either by stress ECG, stress echo, or stress nuclear studies.

Since we utilize evidence to make diagnostic and therapeutic decisions in the conditions outlined above, why should we not use the same approach in patients with chronic stable angina?

If decisions involving PCI are not based on evidence for myocardial ischemia, one needs to ask another question, “Why should anyone undergo catheterization without a clear-cut indication?” Obviously, the main reason for performing coronary angiography is the need to know a patient’s anatomic pathology in order to determine whether revascularization is an option. In 1975, Gofredo Gensini commented that “the primary goal of coronary angiography is the identification, localization, and assessment of obstructive lesions present within the arteries of the heart.”<sup>1</sup> The operative word here is assessment—that is, what is the significance of the stenosis observed? Is it the cause of the patient’s symptoms?

The following are the guidelines of the American College of Cardiology, the American Heart Association, the American College of Physicians, and the American Society of Internal Medicine for invasive testing of patients with chronic stable angina:<sup>2</sup>

Class 1 indications include Canadian Cardiovascular Society (CCS) class 3 or 4, despite medical therapy; left ventricular dysfunction and/or strongly positive stress tests; sudden cardiac death or ventricular tachycardia survival; and angina plus heart failure symptoms.

Class 2A indications include symptoms of uncertain diagnosis after stress testing when it is important to know the coronary pathology; inability of the patient to undergo noninvasive testing due to disability, illness, or morbid obesity; an occupational requirement for a definitive diagnosis; and inadequate prognostic information after noninvasive testing.

In Class 3, invasive testing is not indicated. Nonindications include a risk of angiography that outweighs the benefits; CCS class 1 or 2, response to medical therapy, and no evidence of ischemia on noninvasive testing; and patient preference.

I still believe angiography is the best single test to determine the presence or absence of coronary artery disease and prognosis, and do not keep my symptomatic patients from undergoing coronary angiography and ventriculography. However,

one must question the significance of a specific stenosis and must use additional parameters other than stenosis severity to help make decisions. For example, findings of a high-grade proximal stenosis and TIMI II or less flow are highly suggestive of a physiologically important lesion, and thus the patient should probably undergo PCI or surgical revascularization. However, findings of a high-grade proximal stenosis and TIMI III flow raise the question of the physiologic significance of the coronary stenosis. This patient should probably undergo revascularization; however, the stenosis should be assessed in multiple views since it may not be quite as severe as perceived in one view and thus may not be responsible for myocardial ischemia. In a third situation, findings of a high-grade proximal stenosis, TIMI III flow, but also a dilated ventricle and a poor ejection fraction raise the question of myocardial viability. In such a patient, there should be some evidence to show myocardial viability before proceeding with angioplasty and/or stent implantation or surgical revascularization. This can be done in the catheterization laboratory, providing the angiography is performed properly. This requires careful observation of the coronary angiogram and ventriculogram. Improved LV contraction after nitrate infusion or after a PVC suggest viability. Collateral flow to the area in question is also highly suggestive of ischemic viable myocardium, as is visualization of a myocardial blush of the microcirculation with a contrast agent.

Some asymptomatic patients are considered for coronary angiography and ventriculography. For example, a patient with

a strongly positive stress test (silent myocardial ischemia) or LV dysfunction determined by echocardiography or other non-invasive means has a high risk for future cardiac events. If high-grade stenoses are found, PCI should be carried out if possible.

Another example would be a patient with a stress test not strongly positive but who has moderate LV dysfunction determined noninvasively. This patient is at intermediate risk for future cardiac events and may warrant coronary angiography.

An example of a patient who does not warrant coronary angiography is one with a stress test that is weakly positive or negative, with a normal LV ejection fraction. This is a low-risk patient, and probably does not deserve coronary angiography.

In summary, my long answer to the question posed in the title of this editorial is the same as my short answer. There should be evidence for ischemia for decision-making in the individual patient with chronic angina whose angiography shows coronary stenosis.

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## References

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