**Editorial Comment**

**Ergonovine Echocardiography for Coronary Spasm: Facts and Wishful Thinking***

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In this issue of the Journal, Song et al. (1) describe their enthusiasm for a novel noninvasive test strategy as a screening evaluation for the diagnosis of coronary artery spasm. Their strategy was designed to confirm the diagnosis of coronary spasm among patients with chest pain syndromes suggesting variant angina. Patients with myocardial infarction, unstable angina, bundle branch block or rhythm disturbances were excluded. After discontinuation of cardioactive drugs, a noninvasive stress test (exercise electrocardiogram [ECG] or thallium-201 imaging) was applied. Echocardiography was performed in patients with negative noninvasive stress test results. In patients with acceptable acoustic windows, ergonovine was administered intravenously, in bolus doses, until either ischemia or a total dose of 0.35 mg occurred. Development of either reversible ECG ST segment changes or regional wall motion abnormalities was taken to represent criteria for coronary spasm. The authors report reasonable specificity and sensitivity and no complications in 80 patients compared with that for provocative testing done during coronary angiography.

The first consideration for any test for coronary spasm must be the safety of the test. Clearly, one cannot judge safety in 80 patients. There is a small but finite risk for adverse outcome associated with ergonovine testing when performed in the catheterization laboratory (2). Abrupt occlusion, profound hypotension or ventricular tachycardia/ventricular fibrillation that does not respond to intravenous nitroglycerin or calcium antagonists may develop. Presumably, this is the result of more intense spasm, plaque disruption and thrombosis at or near sites of spasm, as suggested by a recently developed experimental model proposed by Picon et al. (3) using a triggering vasoconstrictor substance. Other experimental models indicate that intense vasoconstriction (e.g., vasoconstrictor applied topically to arteries) may result in changes in endothelial structure (4-6) and function that could also favor platelet aggregation and coagulation at the lumen surface. Refractory coronary spasm requiring intracoronary nitroglycerin for relief has been described by us (7,8) and others (2), and sometimes there is need for an intracoronary calcium antagonist or even emergency percutaneous transluminal coronary angioplasty. During a noninvasive test, if refractory coronary spasm were to occur at the site of ergonovine-induced spasm, a serious problem could result with no direct access to the coronary arteries. Even if such a problem occurred in only 1 of 1,000 patients with spasm, the outcome could be catastrophic. However, patients undergoing testing in a catheterization laboratory would have the option of intracoronary administration of not only nitroglycerin and calcium antagonists, but also thrombolytic agents as well as balloon angioplasty if needed.

The second consideration with any test is its value as a strategy in patient management. As a general principle, diagnostic tests are most effective when utilized in patients with an intermediate pretest likelihood of the suspected condition. Conversely, a test is less indicated at extremes of pretest likelihood. The authors identified angiographic evidence for coronary spasm in 56 (74%) of the 76 patients examined in a 1-year period. Thus, this is not a cohort where anything less than a perfect test result is likely to provide additional usefulness. These data suggest that the patients chosen for evaluation of the new test were highly selected (e.g., study population was enriched in patients with coronary spasm). This high percentage also raises the issue of selection bias related to how the patients were chosen to evaluate this new test. How the test would perform in the usual population of patients with coronary artery disease undergoing evaluation in our country is unknown. Although coronary spasm and the need to know were prominent questions two decades ago, this issue is no longer common in contemporary practice, perhaps because of the frequent use of calcium antagonists, better nitrate regimens or attenuations in the virulence of coronary artery disease processes. Questions about coronary spasm contributing to a clinical syndrome have become relatively infrequent. Furthermore, the few patients that we are asked to evaluate at present, relative to possible coronary spasm, often have very refractory chest pain syndromes that have eluded multiple physician evaluations. It is unlikely that a noninvasive test, with unknown sensitivity and specificity in such a population, as well as uncertainties about safety, will satisfy the requirement for a definitive evaluation to sort out such a complex clinical problem. However, this is speculation and what is needed is a study that investigates the relative usefulness of a strategy of management that is guided by a noninvasive test for coronary spasm compared with a strategy where such a test is omitted. Said another way, Is this knowledge of value in the subsequent cost-effective management of such patients? Certainly, knowledge about the presence or absence of important coronary disease is important in certain patients, but such patients are likely to undergo coronary angiography anyway, and if needed, an invasive test for spasm may then be carried out.

Until these issues are addressed, my recommendation is that in the few patients requiring a test for spasm, it is probably safer to perform such a test in the catheterization laboratory.

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This would reserve the new test for the very few patients that have either had a recent coronary angiogram or in whom, for some reason, knowledge of coronary anatomy would not be needed. In my view, it is therefore wishful thinking to believe that this new noninvasive test for spasm could have much clinical utility even if the facts about safety, sensitivity and specificity were resolved.

References