Papillary Muscle Dysfunction During Dipyridamole-Thallium Imaging

TO THE EDITOR: I found the article by Lette et al. (1) most interesting. They described a patient who developed reversible exacerbation of mitral regurgitation due to papillary muscle dysfunction and scintigraphic evidence of posterior wall ischemia during dipyridamole-thallium imaging.

In their discussion, they suggested that in papillary muscle dysfunction the anterior papillary muscle is less frequently involved than the posterior and cited my article in 1969 (2) as a source of reference. Unfortunately this was a misquotation, because there was no difference in the occurrence of papillary muscle dysfunction between anterior and posterior or inferior myocardial infarction or ischemia in my series (2).

The equal incidence of papillary muscle dysfunction in anterior and posterior-inferior myocardial involvement is understandable when one considers the blood supply of the papillary muscles. The anterior papillary muscle is supplied primarily by one or more branches of the left anterior descending artery including the diagonals, but additional branches enter that area from the marginal branches of the circumflex artery. The posterior papillary muscle is supplied by the arteries, which supply the diaphragmatic surface of the left ventricle. In 70% of the cases, these arteries are branches from both the right coronary and left circumflex arteries, in 20% they are exclusively from the right coronary artery, and in 10% from the left circumflex artery. Since coronary arterio-sclerosis and occlusions are just as common in left anterior descending as in right coronary and left circumflex arteries, ischemic changes should therefore involve the posterior papillary muscle as frequently as the anterior papillary muscle (2).

It was also mentioned by Lette et al. that cardiac auscultation for transient mitral regurgitation, a sign of reversible papillary muscle dysfunction, is a simple and practical adjunctive test for myocardial ischemia during dipyridamole-thallium imaging, and, thus, it may confirm that an isolated reversible posterior wall myocardial perfusion defect is truly ischemic in nature as opposed to an artifact resulting from attenuation by the diaphragm. However, it should be noted that because of the dual-blood supply of both the anterior and posterior papillary muscles, some myocardial infarcts or ischemia are accompanied by the apical systolic murmur of papillary muscle dysfunction but others are not (2).

REFERENCES


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REPLY: We thank Dr. Cheng for his letter and recommend his excellent paper on the syndrome of papillary muscle dysfunction (1). We would like to make the following clarifications:

1. In revising and shortening our original manuscript (2), we misplaced the reference to Dr. Cheng's article. We intended to use his article as a reference for our description of the blood supply to papillary muscles (it should have been placed three lines higher). Our most sincere apologies to Dr. Cheng for the misquotation.

2. Dr. Cheng's study (1) showed an equal incidence of papillary muscle dysfunction in anterior and postero-inferior myocardial involvement. Heikkila (3) also found an equal incidence of anterior and postero-inferior involvement by electocardiography in patients developing a mitral systolic murmur after myocardial infarction. However, anterior myocardial infarction was more common in his series (75%), indicating that a patient with a postero-inferior infarction was more likely to develop mitral insufficiency. Burch (4) found a predominance of anterior wall involvement, but his study was limited to two cases. In reviewing the literature, we found a predominance of infero-posterior involvement in a majority of studies (5,6,7), prompting us to state that "the anterior papillary muscle is less frequently involved." Considering that ischemia of either papillary muscle can cause transient mitral regurgitation, Dr. Cheng would probably agree that transient mitral regurgitation during dipyridamole-thallium imaging may confirm that an isolated reversible posterior wall defect is ischemic in nature as opposed to an artifact resulting from attenuation of the diaphragm (as stated in our paper), and may also confirm that an isolated reversible anterior wall defect is not a breast artifact in a female patient.

3. We definitely agree with Dr. Cheng that only a small subgroup of patients with transient myocardial ischemia develop transient mitral insufficiency on auscultation. A prospective study on the incidence of transient mitral regurgitation during dipyridamole-thallium imaging is presently under way at our hospital.

REFERENCES