DEPARTMENTS

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 posteroinferior 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 arteriosclerosis 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 dipyridamolethallium 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

 Lette J, Gagnon A, Lapointe J, Cerino M. Transient mitral regurgitation: an adjunctive sign of myocardial ischemia during dipyridamole-thallium imaging. J Nucl Med 1989; 30: 1271-1272. Cheng TO. Some new observations on the syndrome of papillary muscle dysfunction. Am J Med 1969; 47:924-945.

> Tsung O. Cheng George Washington University Washington, DC

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 posteroinferior myocardial involvement. Heikkila (3) also found an equal incidence of anterior and postero-inferior involvement by electrocardiography 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 from 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

 Cheng TO. Some new observations on the syndrome of papillary muscle dysfunction. Am J Med 1969; 47:924-945.

- Lette J, Gagnon A, Lapointe J, Cerino M. Transient mitral regurgitation: an adjunctive sign of myocardial ischemia during dipyridamole-thallium imaging. J Nucl Med 1989; 30:1271-1272.
- 3. Heikkila J. Mitral incompetence complicating acute myocardial infarction. Br Heart J 1967; 29:162-169.
- Burch GE, De Pasquale NP, Phillips JH. Clinical manifestations of papillary muscle dysfunction. Arch Int Med 1963; 112:112-117.
- Bashour FA. Mitral regurgitation following myocardial infarction: the syndrome of papillary mitral regurgitation. *Dis Chest* 1965; 48:113-122.
- Orlando MD, Wooley CF, Scarpelli D, Ryan JM. Mitral regurgitation caused by infarcted papillary muscles: report of fifteen cases. *Circulation* 1964; 30(suppl III):136.
- De Busk RF, Harrison DC. The clinical spectrum of papillary muscle disease. N Engl J Med 1969; 281:1458-1467.

Jean Lette Maisonneuve-Rosemont Hospital Montreal, Canada

Hyperperfusion of a Lower-Lobe Pneumonia by Positive Pressure Ventilatory Support

TO THE EDITOR: Kim and Heyman (1) recently discussed an unusual scintigraphic pattern of ventilatory/perfusion (V/ Q) mismatch in a 4-mo-old infant on positive end-expiratory pressure (PEEP) mechanical ventilatory support. Blood flow was redistributed away from the well-ventilated upper lungs to the underventilated atelectatic lower lobes. They attributed this redistribution directly to PEEP, which expands ventilated lung and increases its resistance to blood flow. These effects are most pronounced in the upper lungs where perfusion pressures normally exceed alveolar pressures only during the peak of the cardiac cycle. Normally, the vascular resistance of the lower lungs is similarly increased by PEEP and the degree of redistribution is limited. However, a large area of poorly ventilated lung that cannot expand may act as a low resistance vascular shunt and allow a significant redistribution of blood flow to occur. Regions of lung that are atelectatic, consolidated, or occluded by an endobronchial obstruction may, thus, become inappropriately hyperperfused with PEEP and appear as an area of reverse V/Q mismatch on perfusion/ ventilation scintigraphy with Q > V. If a large region of lung is involved, an extensive right-to-left shunt may occur resulting in a profound deterioration of respiratory status characterized by a marked hypoxemia and a large Arterial-alveolar oxygen partial pressure gradient (A-a gradient). In this event, the upper lungs will generally be left significantly underperfused by the resulting redistribution of blood flow and may appear on scintigraphy as broad areas of V/Q mismatch with V > O.

Regions of reverse V/Q mismatch, per se, are not altogether uncommon and have been previously attributed to a variety of factors including metabolic alkalosis, pulmonary venous and arterial hypertension, paralysis or impairment of the respiratory center, inhalational anesthetics, nitrate vasodilators, mucous plugs, atelectasis, and even lung disease such as COPD, pneumonia and asthma (2-6). However, the association with PEEP described by Kim and Heyman (1) has

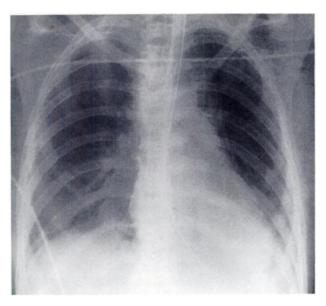


FIGURE 1

Chest radiograph findings include ventilatory apparatus, endotracheal and nasogastric tubes, and subcutaneous emphysema at the left shoulder/upper thorax. Neither the descending aorta or left hemidiaphragm are visualized, and infiltrates are seen near the left base, consistent with a diagnosis of left lower lobe pneumonia.

only been previously reported in one adult case (7) and one other infant case (8). Nuclear medicine physicians should be familiar with this pattern of V/Q mismatch and its significance since pulmonary scintigraphic studies may be ordered for ventilatory-dependent patients to rule out pulmonary thromboembolism (PE) as a cause of respiratory deterioration. Fur-



FIGURE 2

Left posterior oblique perfusion image following the administration of [^{99m}Tc]MAA through a patent i.v. catheter in the distal right arm. Clinically, the arm appeared unremarkable without evidence of thrombophlebitis or venous occlusion and the unusual degree of retained venous activity may reflect endothelial damage from prior i.v. medications. The entire left lower lobe appears markedly hyperperfused. Both lungs show some redistribution of activity away from the upper portions toward the bases.