INVITED COMMENTARY

The Chronic Perfusion Defect: Our Knowledge Is Still Hazy, but the Message Is Clear

It is late in the afternoon, and you get a call from your emergency department requesting a ventilation-perfusion (V/Q) scan for a patient with a sudden onset of dyspnea and tachypnea. You look at the chest radiograph and find only minimal abnormalities, with a small area of left-sided linear atelectasis and a slight elevation of the left diaphragm. The lungs show no opacities. The results of the ventilation scan are normal. A mismatched perfusion deficit is obvious, which you characterize as large segmental lesions involving both the posterior and the lateral basilar segments and at least 1 large segmental deficit involving the inferior lingula and a subsegment of the superior lingula. You inform the emergency department that the V/Q scan clearly shows a high probability of pulmonary embolism. Anticoagulant therapy is begun, and the patient is admitted to the hospital.

Approximately 1 wk later, the patient's pulmonologist comes to consult you. He says, "The patient with the high-probability scan told me he might have had a blood clot in the lungs a few years ago. I know the physician who was taking care of him, so I called and found that other lung scans were obtained at that time. They were just sent to me; would you like to see them?"

Naturally, you say yes. You are dismayed to find that 2 old V/Q scans are available. The first scan shows a large mismatched-segment perfusion deficit involving at least 5 full segments with bilateral deficits, and the second is a 3-mo follow-up scan with findings identical to those of the scan you read 1 wk previously. Given this information, you admit that your reading of a high probability of pulmonary embolism was caused by a chronic perfusion deficit because you see no new mismatched lesions. You conclude that this patient's anticoagulant therapy is probably best discontinued. "I wish I had known that sooner," says the pulmonologist. "The patient had intracerebral bleeding last night."

This example illustrates the reason the data presented by Wartski and Collignon (*1*) in this issue of *The Journal of Nuclear Medicine* are extremely important and unfortunately often ignored. Although the scenario is fictional, the appearance of the V/Q scan is not. I carry around slides of this patient to illustrate the point that Wartski and Collignon made. The patient was young and in excellent health except for his pulmonary status, which included an angiographically proven large left lung base and a lingular embolus. (He was part of the Prospective Investigation of Pulmonary Embolism Diagnosis.) His follow-up V/Q scan showing a still-high probability of pulmonary embolism was obtained almost 1 y later, when the patient was no longer receiving therapy and was asymptomatic. This was a rare, successful instance of my being able to badger a referring physician into obtaining a follow-up V/Q scan.

I think the reader would be wrong to get bogged down in a comparison of the magnitude of the results of Wartski and Collignon (*1*) with those of other studies, such as the Urokinase-Streptokinase Pulmonary Embolism Trial (USPET) of the 1970s. In USPET, the size of the chronic perfusion defects was scored entirely differently. A 10% chronic defect was equivalent to a 2-segment defect. Wartski and Collignon did not make clear the number of remaining mismatched segments or the degree of remaining mismatching, because ventilation scanning was not performed. Furthermore, the use of ^{131}I-macroaggregates as the perfusion tracer in the USPET trial probably obscured subtle perfusion changes that Wartski and Collignon uncovered in their patients. Although the authors compared their results with those of other series, I am not concerned about these correlations. To me, the important point is that a significant (high-probability–sized) chronic perfusion deficit exists after pulmonary embolism. Any patient unlucky enough to have a deficit can present to an emergency room with chest pain from nonembolic causes such as adenovirus. When the emergency department staff finds out about the prior pulmonary embolism, the patient clearly will undergo V/Q scanning, and because of the persistent, high-probability–sized chronic deficit that will be seen, the patient will needlessly receive anticoagulant therapy and risk serious hemorrhagic complications. Therefore, I choose not to quibble about the magnitude of the problem, because variables such as scan technique, patient selection, and assessment of perfusion defect size differ from series to series.

I believe that a chronic perfusion defect is a serious problem. How does one deal with it? Wartski and Collignon (*1*) indicated that the best way to handle this problem is to obtain a routine pulmonary lung scan after completion of anticoagulant therapy. I agree with the concept but suggest that this utopian approach will not happen in the United States in these days of managed care. In fact, what is more likely is that unless we—as nuclear medicine physi-

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cians—push the concept, it will hardly happen at all. Therefore, the real question becomes whether a way exists to predict who is likely to have a chronic perfusion deficit. Unfortunately, knowledge about this aspect remains hazy, but Wartski and Collignon provided many valuable clues to solve this perplexing problem. They suggested that an in-hospital ventilation perfusion scan be obtained on day 8 because "a closer correlation was found between residual obstruction and [a pulmonary vascular obstruction on day 8]." They found that comorbid cardiopulmonary disease significantly influenced the incidence of chronic perfusion deficit. They also found that the larger the initial perfusion deficit, the more likely the patient was to have a residual chronic deficit. Finally, they found that age was not an important variable. Therefore, assuming one cannot devise a plan for V/Q follow-up of all patients, the data that Wartski and Collignon provided give clues to allow a push for follow-up of those patients who need it the most. The larger the initial perfusion deficit, the more likely is a chronic perfusion deficit. In patients with a large perfusion deficit, one should try to obtain a follow-up lung scan before the patient leaves the hospital. One should worry most about, and push the hardest for, patients with persistent large perfusion defects and comorbid cardiopulmonary disease regardless of age.

Another serious problem for those with a chronic perfusion deficit needs to be addressed. The United States has a relatively mobile population. Pulmonary embolism is an episodic disease, and a patient may likely get a second pulmonary embolus in a locale far different from that of a first embolus. The patient can be well into a course of anticoagulant therapy by the time records are transferred from 1 location to another, allowing ample time for hemorrhagic complications in a patient who does not need anticoagulant therapy. I suggest that any patient who has a chronic perfusion defect be instructed as to the nature of this finding and its significance for future treatments. Providing the patient with an analog copy of the V/Q scan is even better. Another possibility—an easy alternative in these days of computer literacy—is to give to the patient a diskette with computerized images of the last lung scan to keep as part of personal medical records. These can be brought to the hospital if any subsequent symptoms of pulmonary embolism develop.

In conclusion, Wartski and Collignon (*1*) are correct, and their conclusion is clear: any patient being treated for pulmonary embolism deserves follow-up lung scanning when anticoagulant therapy is completed. However, if my experience is any indication, this procedure will never become standard in this country. I would be thrilled to find that 10% of the patients diagnosed with embolism undergo appropriate follow-up baseline studies. I believe that anyone with a V/Q abnormality should receive a copy of the scan, either on a computer diskette or as an analog image, to facilitate future care. I congratulate Wartski and Collignon for bringing this serious problem to our attention again and for providing additional insight into potential management strategies we all should use.

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REFERENCE

1. Wartski M, Collignon M-A, for the THESEE Study Group. Incomplete recovery of lung perfusion after 3 months in patients with acute pulmonary embolism treated with antithrombotic agents. *J Nucl Med.* 2000;41:1043–1048.