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Re: Ventilation-Perfusion Studies and the Diagnosis of Pulmonary Embolism: Concise Communication

An article in this *Journal* (1) linking the scintigraphic results and clinical findings in patients suspected of pulmonary embolism suggests that the information provides the referring physician with a rational basis for patient management. Other recent reports on the role of nuclear medicine in the diagnosis of pulmonary embolism have provided sophisticated analyses of their data, and the most commonly used perfusion scan parameters include: the size of the largest perfusion lesion, the degree of involvement of individual segments, and the correlation of the perfusion defects with radiographic and ventilation scan abnormalities (2-4). One important pattern recognition feature of perfusion images has been ignored, however, and we believe that knowledge of the frequency distribution pattern of segmental defects would be helpful as a diagnostic criterion of pulmonary embolic disease. Furthermore, this pattern may explain some of the reported advantages of different imaging techniques.

We reviewed our recent experience of six-view perfusion lung scans in patients suspected of having pulmonary embolic disease.

Twenty-two patients with the typical scan findings were treated for pulmonary embolism. The frequency distribution of the 121 segmental perfusion defects is shown in Table 1, with some liberties taken in grouping various bronchopulmonary segments. The average number of perfusion defects per patient was 5.5; 67% were located in the lower lobes, 15% in the right middle lobe or lingulae of the left upper lobe, and 18% in the upper lobes.

The posterior oblique views were compared with the lateral views to determine which resulted in better lesion definition. As seen in Table 1, if lower-zone defects are better visualized in any one view, then the posterior oblique view is superior, whereas in the upper zones the lateral view is superior. In the middle zone of either lung field no one view was found to be superior. Fifty-two (43%) of all segmental defects were better visualized in one view, and in 41 (79%) the oblique view gave superior results.

Table 1 indicates that the superiority in visualization by the oblique view, a similar finding in other studies (5), is directly related to the distribution of these perfusion defects. Since the majority of perfusion defects are located in the lower lung zones and are better defined in the posterior oblique views, this view is more productive for visualizing defects. These data indicate, however, that when the perfusion abnormality is in the middle or upper lung zone (superior segment of lower lobes, middle lobe or upper lobes), then the posterior oblique view may not be the view to best define the lesion.

In summary, we believe that the frequency distribution of segmental defects is an important feature of pattern recognition of the scan findings in pulmonary embolism and should be included among the diagnostic criteria currently used. Based on our findings this distribution pattern explains the superiority of posterior oblique views in defining scan lesions in pulmonary embolism.

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TABLE 1. FREQUENCY DISTRIBUTION OF SEGMENTAL PERFUSION DEFECTS. TABULATION OF THE OBLIQUE OR LATERAL VIEW IN WHICH A DEFECT IS BETTER VISUALIZED

Segment*	Total defects	Oblique better visualizes defect	Lateral better visualizes defect
Posterobasal	25	12	—
Medial and/or anterobasal	20	10	—
Lateral basal	17	11	—
Superior	19	7	6
Medial/inf. lingula†	5	1	2
Lateral/sup. lingula†	12	—	—
Apical and/or posterior	13	—	2
Anterior	9	—	1
Total	120	41	11

* Individual or groups of bronchopulmonary segments.

† The right middle lobe and the lingular segments of the left upper lobe are grouped together.

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5. NEILSON PE, KIRCHNER PT, GERBER FH: Oblique views in lung perfusion scanning: Clinical utility and limitations. *J Nucl Med* 18: 967-973, 1977

Reply

Dr. Wilson and his colleagues are right in indicating that patterns of perfusion defects may be helpful as diagnostic criteria in pulmonary embolic diseases. I am not sure, however, that the data in their table are useful for this purpose. First, such data would be useful only if the relative frequency of perfusion defects in various bronchial pulmonary segments was known for patients with pulmonary embolism as well as for patients without pulmonary em-

bolism. Second, as in the evaluation of any diagnostic procedure, independent verification of the final disease state is essential. Dr. Wilson and his colleagues imply that their 22 patients had a subjective diagnosis of pulmonary embolism. Thus, I would conclude that while their supposition may be direct, their data do not yet support their conclusions.

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Re: Correlation of Contrast Angiography and Histological Pattern with Gallium Uptake in Primary Liver-Cell Carcinoma: Noncorrelation with Alpha-Feto Protein

In their article, Waxman et al. comment that "the hepatitis A antigen also did not correlate with the findings of the gallium scan" (1). As there is no known association between hepatitis A (as opposed to hepatitis B) and hepatocellular carcinoma, the relevance of this observation escapes me. Did this represent a repeated typographical error or a misunderstanding?

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REFERENCE

1. WAXMAN AD, RICHMOND R, JUTTNER H, et al: Correlation of contrast angiography and histological pattern with gallium uptake in primary liver-cell carcinoma: Noncorrelation with alpha-feto protein. *J Nucl Med* 21:324-327, 1980

Reply

In response to Dr. Cooney's letter, the statement should read, "the hepatitis B antigen showed no correlation with the findings of the gallium scan." The hepatitis B surface antigen has been shown to have a high association with hepatocellular carcinoma, especially in nonalcoholic cirrhosis (1,2). The hepatitis B surface antigen has also been called the hepatitis associated antigen or HAA. The abbreviation was initially used and subsequently changed in the final draft of the paper and incorrectly referred to as hepatitis antigen A, instead of hepatitis associated antigen for hepatitis B surface antigen. We are indebted to Dr. Cooney for his observation. We too know of no known association of hepatitis A and hepatocellular carcinoma.

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Re: A Radiometric Microbiologic Assay for the Biologically Active Forms of Niacin

Concerning the paper by Judith A. Kertcher et al: "A Radio-

metric Microbiologic Assay for the Biologically Active Forms of Niacin" published in this *Journal* (1), we have been seriously misquoted. They state: "Another microbiologic assay using the protozoan *Tetrahymena pyriformis* is said to be more sensitive and more specific. This organism, however, is slow and difficult to grow and was shown to respond to several naturally occurring, biologically inactive derivatives of niacin." Their reference for such statements was to our paper "Nicotinic acid assay in blood and urine" (2).

The point of our paper was that the niacin requirement of *T. pyriformis* closely—hence usefully—parallels that of man and higher animals (our paper, p. 575), and that niacin derivatives having *no animal activity* were *inactive* (our italics) for *T. pyriformis*, in contrast to their incorrect allusion (*vide supra*) to our work (2). Indeed, we stated that "Trigonelline (the betaine of nicotinic acid), which has *no animal activity, proved inert* for *T. pyriformis*" (our italics), and "*T. pyriformis* does not respond to nicotinuric acid, whereas *Lactobacillus plantarum* does, another point of specificity favoring *T. pyriformis*." We never stated, as these authors wrote, that *T. pyriformis* responded to "biologically inactive" niacin derivatives. We did note, as they confirm, that *L. plantarum* does respond to animal-inactive niacin derivatives, e.g., trigonelline and nicotinuric acid (an excretory product), thus making *L. plantarum* less specific than *T. pyriformis* for detecting and estimating biologically active niacin.

In contrast to what the authors state in their paper (1), *T. pyriformis* is easy to grow. The maintenance medium consists of glucose and proteose-peptone, and gives as heavy a growth in 3 days as does the basal medium (2). Also the *T. pyriformis* method does not require elaborate, costly arrays of equipment and supplies—as for example, a radioactivity counter, radiolabeled metabolites, etc.; one needs only flasks, tubes, pipettes, and a simple photometer. For over 20 years we have routinely used the *T. pyriformis* assay for niacin, and we process over 100 blood or tissue samples with ease in 2 hr, with a turnover time of 3 days. We think their present method (1) cannot render as good performance.

We reiterate that *T. pyriformis* mirrors the response of only biologically active niacin; *L. plantarum* does not. The authors should be aware of this point; their quotations badly misguide the reader. Indeed, the protozoan response to vitamins that are biologically active only for man makes them suitable and specific microanimals, parallel to the higher animals, for analyzing vitamins in biologic fluids and tissues. Their clinical correlations make them far superior to radionuclide methods (3,4), as for example in the comparison with radioisotope assays for vitamin B₁₂ (4), a point they left unmentioned before decrying the use of protozoan assays (1).

It has long been recognized that assays for B vitamins, mainly based on protozoan assays, including that of *Tetrahymena* for niacin, yield results of especially close clinical validity, as recognized by our invitation to describe assays for vitamins in this (3) and the previous edition.

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