tion. If one wants to evaluate the utility of a diagnostic maneuver, it goes without saying that the maneuver cannot be assumed accurate a priori and that another independent criterion for diagnosis must be used. To be sure, the authors used clinical findings, but a clinical diagnosis of pulmonary embolism is next to impossible (*1*). Seven of the 28 patients had the diagnosis confirmed by angiography and this is, of course, important, but an inductive conclusion based on seven out of seven is far weaker than one based on the implied 28 out of 28. The further conclusion that pulmonary embolism can be excluded by a pattern of abnormal ventilation in areas of abnormal perfusion is based on 43 patients with suspected embolism whose final diagnosis of obstructive airway disease was made by scan and clinical findings. I would not deny that such a diagnosis of obstructive airway disease can be accurate, but I do wonder how the positive diagnosis of one disease can exclude the superimposition of another suspected condition. Again, angiographically proven cases are needed. (I do not assume angiography is 100% accurate, but at this point it is still the standard for comparison, short of autopsy.)

The same sort of fallacy may be present in the paper "Evaluation of a ^{133}Xe ventilation technique for diagnosis of pulmonary disorders" by Farmelant and Trainor (*J Nucl Med* 12: 586–590, 1971). Whether the fallacy really is present is difficult to judge because the diagnostic criteria for various patients are not stated in the article.

The above-mentioned papers are useful in that they catalog the variety of patterns that might be observed with ventilation and perfusion scanning and, indeed, the authors may be correct in thinking that a certain combined scan pattern indicates the diagnosis of pulmonary embolism, but this conclusion is not a logical consequence of the presented data.

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REFERENCE

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REPLY BY FARMELANT AND TRAINOR

Dr. Schneider raises a germane point that we slighted. The criteria for classifying patients, particularly those categorized as having pulmonary embolization, should have been stated.

However, we believe that the diagnosis of pulmonary embolism can be quite certain in some clinical situations without angiography or postmortem examination. In our group of 15 patients classified as having pulmonary embolism, all showed clear chest x-rays and none had clinical evidence of bronchospasm, i.e., wheezing was not present. One had angiographic confirmation and serial changes in the perfusion scan. Four had active thrombophlebitis and serial changes in the perfusion scan. Three had only serial changes. Four patients had active thrombophlebitis and multiple perfusion defects, but repeat scans were not obtained. In three additional patients, one of whom had active thrombophlebitis, the diagnosis rests on the purely clinical considerations. These latter seven patients had combinations of acute onset of cough, chest pain, dyspnea, hemoptysis, and fever with no evidence of pneumonic infiltrates. Our problem was in getting angiograms in patients whose primary physician was convinced of the diagnosis on clinical grounds backed by the perfusion scan.

In short, while absolutely convincing evidence of pulmonary embolization may be absent in seven of our 15 patients, the diagnosis did not rest on the discrepancy between the perfusion and ventilatory defects at the time the study was in progress. At present, rightly or wrongly, this criterion is being relied on quite heavily in this hospital.

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REPLY BY ISAWA

Because of the ready availability of lung scans in the diagnosis and management of pulmonary embolism, frequency in the use of pulmonary angiography is certainly decreasing unless surgery is contemplated. It was true in the patients reported in our recent article (*1*). As questioned by Dr. Schneider, we do not think that concurrent small emboli were completely excluded in the group of patients who were diagnosed to have obstructive airways disease, but perfusion abnormalities in these patients were mostly explained on the basis of obstructive airways disease as evidenced on aerosol inhalation scans.

When alveolar ventilation is disturbed by airway obstruction, perfusion is promptly diminished (*2–4*).

This is probably induced by alveolar hypoxia (*5*) and can well be demonstrated by perfusion lung scans (*6,7*). On the other hand, when the pulmonary artery is occluded, normal ventilation exists in the ischemic lung regions after 6–8 hr if parenchymal complications do not develop (*8*). These experimental findings substantiate our clinical observation that normal ventilation in the areas with absent perfusion indicates pulmonary vascular disease, most likely pulmonary embolism under the clinical circumstances in which there is a strong clinical and laboratory suspicion of pulmonary embolism in patients without radiological evidence of parenchymal consolidation or pleural changes. With these physiologic and clinical observations in mind we think that combined perfusion and inhalation lung scans are of great help in interpreting perfusion abnormalities especially by obtaining aerosol scan evidence for or against airways disease.

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BENEDICT CASSEN, Ph.D.

November 13, 1902—March 21, 1972

The Society of Nuclear Medicine announces with great sadness the death of Dr. Benedict Cassen, who died in the Santa Monica Hospital within a few hours of suffering a myocardial infarction. Dr. Cassen was the recipient of the first Distinguished Scientist Award given by the Society at its annual meeting in July 1970.

He was born in New York City and lived during part of his childhood in the tobacco farming region of Connecticut. While on a visit to relatives abroad, he enrolled in the Royal College of Science in London from which he received a degree in physics and mathematics in 1927. He went on to receive his doctorate (magna cum laude, 1930) from the California Institute of Technology, working on high potential x-ray tubes. He became a hospital physicist and worked for the Westinghouse Research Laboratories and the Harper Hospital in Detroit. Later he became a physicist in the U.S. Naval Ordinance Test Station in Pasadena, and in 1947 became Research Physicist at the UCLA Atomic Energy Project in the School of Medicine, now the Laboratory of Nuclear Medicine and Radiation Biology.

Most recently he was Professor Emeritus of Biophysics and co-developer with Dr. Norman S. MacDonald of the UCLA Medical Cyclotron Facility which was dedicated by Dr. Glenn Seaborg at the time of the Society's annual meeting in Los Angeles in July 1971. He also served as a consultant to the Medical Division of the Oak Ridge Institute of Nuclear Studies and to the Los Angeles Veterans Administration Hospital.

Among Dr. Cassen's many important contributions to nuclear medicine was his development of the prototype scanner for directional gamma-ray detection which was described in *Nucleonics* in August 1951. He had a great influence on a host of friends and colleagues including many members of the Society. Among his other contributions to the Society, Dr. Cassen served as a member of the editorial board of the *Journal* for several years. His hobby was his work in which he expressed a keen and continuing interest until the end.

He leaves his wife, Wylie, of Pacific Palisades and son, Balfour, of Los Angeles.