TABLE 1. ^{133m}Ba DOSE CALCULATIONS*

Radionuclide	Rads/μCi injected: Skeleton	Rads/μCi injected: Total body	Injected dose to bone (%)
^{133m}Ba-^{133}Ba	0.00257	0.000459	60
^{135m}Ba	0.001176	0.000178	60
^{131}Ba-^{131}Cs	0.00648	0.001840	60
^{85}Sr	0.02759	0.009367	70
^{87m}Sr	0.0000640	0.0000151	70
^{18}F	0.0001113	0.00002556	53

* The assumption is that 60% of the injected dose goes to bone and decays there with a $T_{1/2}$ of 38.9 hr. The ^{133m}Ba deposited in bone is assumed to decay to ^{133}Ba which decays with an effective half-life equal to the physical half-life (7.2 yrs).

TABLE 2. ^{133m}Ba EQUILIBRIUM ABSORBED DOSE CONSTANTS

Radiation$_i$	No/dis n_i	Energy$_{(MeV)}$ E_i	Δ_i (gm-rad/μCi-hr)
Electron capture	1.0	—	—
γ_1	0	0.01129	—
e_{L_I}, γ_1	0.67	0.00669	0.0095
e_M, γ_1	0.33	0.01123	0.0078
γ_2 0.276 keV	0.175	0.2757	0.1027
e_K, γ_1	0.595	0.2383	0.3019
e_{L_I}, γ_1	0.172	0.2701	0.0989
e_M, γ_1	0.057	0.2756	0.0334
$K_{\alpha 1}$ x-ray	0.283	0.0322	0.0193
$K_{\alpha 2}$ x-ray	0.147	0.0318	0.0099
$K_{\beta 1}$ x-ray	0.080	0.0364	0.0062
$K_{\beta 2}$ x-ray	0.017	0.0374	0.0013
L—x-ray	0.180	0.0045	0.0017
KLL Auger electrons	0.047	0.0263	0.0026
KLX Auger electrons	0.021	0.0308	0.0013
KXY Auger electrons	0.003	0.0353	0.0002
LMM Auger electrons	1.206	0.0034	0.0086
MXY Auger electrons	3.087	0.0011	0.0071
38.9 hr	^{133m}Ba	0.288	
	↓	γ_2 0.01229	
	↓	γ_1 0	

γ_1 assumed to be completely converted in M and L shells.

$\gamma_2 \frac{e_K}{\gamma} = 0.34$, K/L + M . . . = 2.6.

This yield of ^{133}Ba is ~6 × 10^{-4} μCi/μCi of ^{133m}Ba. The calculations for ^{133}Ba have been published in Dillman LT: Radionuclide decay schemes and nuclear parameters for use in radiation dose estimates, Part 2. MIRD Pamphlet No 6, *J Nucl Med* 11: Supplement 4, 5–32, 1970.

gamma ray is emitted in only 17% of the disintegrations. One millicurie of retained ^{133m}Ba decays to only 0.6 μCi of ^{133}Ba. Dose calculations are shown in Tables 1 and 2.

A sample of ^{133m}Ba, cyclotron produced, was kindly supplied by Dr. Larry Brown of the Oak Ridge National Laboratory as $^{133m}BaCl_2$ in 0.1 *M* HCl (2.2 mCi/ml). This was neutralized, filtered and boiled, taken to dryness, and dissolved in sterile isotonic saline. Dogs were injected with 1 mCi each, and blood samples were drawn periodically. The blood disappearance curve was similar to that described with ^{131}Ba (*1*). Bone scans of good quality were obtained with both rectilinear scanners and a gamma-ray camera. It should be noted that ^{133m}Ba, like other barium and strontium isotopes, is excreted in the bowel; scans of the pelvic area should be preceded by thorough cleansing of the gastrointestinal tract.

The short physical half-life of ^{133m}Ba contributes to the low radiation dose (even with the contribution from ^{133}Ba). Despite the highly converted emission there is a significant dose reduction over the commonly used ^{85}Sr. The ability to produce ^{133m}Ba in either a nuclear reactor or cyclotron is of particular interest.

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ACKNOWLEDGMENT

This work was supported by USPHS CA06519 and by T-492 from the American Cancer Society.

REFERENCES

1. SPENCER RP, LANGE RC, TREVES S: Use of ^{135m}Ba and ^{131}Ba as bone-scanning agents. *J Nucl Med* 12: 216–221, 1971

2. HOSAIN F, SYED IB, WAGNER HN: Ionic ^{135m}Ba for bone scanning. *J Nucl Med* 11: 328, 1970

3. LEDERER CM, HOLLANDER JM, PERLMAN I: *Table of Isotopes*, 6th ed, New York, John Wiley & Sons, 1967, pp 76, 478

INHALATION LUNG SCANNING

There appears to be a logical fallacy in two papers recently published in the *Journal of Nuclear Medicine*. In the paper, "Radioaerosol inhalation lung scanning: Its role in suspected pulmonary embolism" (*J Nucl Med* 12: 606–609, 1971) Isawa, et al begin with the premise that "a diagnosis of pulmonary embolism cannot be made by perfusion scanning alone." The conclusion of the paper seems to be that aerosol inhalation scanning helps to make the diagnosis of pulmonary embolism. This conclusion is derived from studies on 28 patients with a final diagnosis of pulmonary embolism "based on the discrepancy between perfusion and aerosol deposition patterns." This appears to be begging the ques-

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

1. POULOSE KP, REBA RC, GILDAY DL, et al: The diagnosis of pulmonary embolism: A correlative study of the clinical, scan, and angiographic findings. *Brit Med J* 3: 67–71, 1970

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*).