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Diagnosis of Pulmonary Embolism and Infarction By Photoscanning[1,5]

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The diagnosis of pulmonary embolism and infarction is a frequent and formidable challenge. Symptoms may mimic a variety of other disorders or diseases, and the condition may exist in a grave form for a considerable time without objective signs being present on physical examination or routine chest films. For these reasons, there has been a long search for better and simpler methods for the diagnosis of this condition. Radioisotope scanning seems to offer one possibility for help in this diagnostic problem. Lung scans have been performed using radiating microspheres (1, 2,) and macroaggregated particles of human serum albumin labeled with radioisotopes (MAA) (3-6). Both techniques have revealed pulmonary embolism or infarction as "cold" areas in animal experiments. Because ceramic microspheres are insoluble and remained permanently in the lung and macroaggregated albumin is eliminated from the lungs after scanning, MAA has become the agent of choice. Lung scans performed with radioactive MAA are easy to perform and demonstrate changes attributable to pulmonary embolism and infarction in experimental animals and in patients (7-10). This report summarizes our experience in the first twenty patients examined by ^{131}I macroaggregated albumin lung scans in our institution.

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MATERIALS AND METHODS

1. *Patient Material:* All patients were hospitalized at the University of Texas Medical Branch Hospitals. Most were suspected of having pulmonary embolism by their referring physician. A few patients were referred with other pulmonary diseases.
2. *Radiopharmaceuticals:* ^{131}I macroaggregated Albumin[1] was obtained in a ready-to-inject form. Regular shipments were received and the material was kept refrigerated at 10° C until ready for use. Just prior to the test, the vial was gently agitated to insure mixing, and the necessary volume for a dose of 300 μC was withdrawn into a disposable syringe and injected intravenously with the patient in the recumbent position.
3. *Technique of Scanning:* Ten to fifteen minutes later, the patient was placed beneath the detector of a photoscanning device.[2] Appropriate count rates were taken from the region of the lung. The photoscanning device was then set accordingly and the scanning begun starting at the lung apex. The scans were performed routinely from the anterior aspect, and in selected patients, scans were also performed in the lateral and posterior positions.
4. *Interpretation:* The developed photoscan, dot scan and in some cases, color scan were interpreted by one of several physicians. In making this review, all photoscans were interpreted by at least two of us separately before the final interpretation was agreed upon.
5. *Classification of Patients:* The 20 patients were classified on the basis of all information available, except scanning, into one of three groups: 1. Nine patients had definite or probable pulmonary embolic disease; 2. Four patients had possible pulmonary embolic disease; 3. Seven patients had other pulmonary disorders. Each of the patients with definite or probable pulmonary embolic disease had a classic history of pulmonary embolism, compatible physical and radiographic findings, and in most cases suggestive ECG changes. Patients in Group 2 had clinical histories suggestive of pulmonary embolism, but without confirmation of the disease. Group 3 patients had histories not suggestive of pulmonary emboli and laboratory tests suggesting other diagnoses.

RESULTS

Table I outlines the clinical summaries of these first twenty patients having lung scans performed with macro aggregated albumin ^{131}I at the University of Texas Medical Center. The clinical data of Group 1 (definite or probable pulmonary embolic disease) will be analyzed in detail in order to evaluate the diagnostic accuracy of various procedures. Because of the lack of a firm diagnosis, Group 2 patients have been set apart. In Group 2, the lung scan was normal in two patients. This relatively low percentage of positivity of the lung scan in this group may reflect either the small size of the emboli or the absence of pulmonary embolic disease.

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[1]Albumotope LS—Kindly furnished by E. R. Squibb & Sons, New Brunswick, New Jersey.

[2]Picker Magnascanner, Picker Nuclear Corp., White Plains, New York; Nuclear Chicago Pho-Dot Scanner, Nuclear Chicago Corp. Des Plaines, Ill.

TABLE I

CASE HISTORIES OF TWENTY PATIENTS HAVING LUNG SCANS PERFORMED

(GROUP 1)

Case No.	*Initials*	*Age*	*Sex*	*Clinical Findings*	*ECG Findings*	*Radiographic Findings*	*Lung Scan Findings*	*Clinical Course and Final Diagnosis*
1.	H.G.	50	F	Mitral commissurotomy for rheumatic mitral stenosis in 1954 & 1956. Chest pain, dyspnea, hemoptysis, cardiomegaly.	Atrial fibrillation. Incomplete right bundle branch block	*Chest x-ray:* Multiple bilateral pulmonary infiltrates. Minimal cardiomegaly	Multiple "cold" areas more frequent on right side, general reduction in concentration right lung.	*Treatment:* anticoagulants. *Clinical Diagnosis:* bilateral pulmonary emboli and infarction.
2.	R.D.	36	M	Myocardopathy with congestive heart failure. Complaint of chest pain and dyspnea. Cardiomegaly.	Biventricular hypertrophy	*Chest x-ray:* Infiltrate right mid lung. *Angiocardiogram.* Embolus in right and middle lung.	Marked reduction in concentration right upper lung.	*Treatment:* anticoagulants. *Clinical Diagnosis:* pulmonary emboli and infarction.
3.	D.H.	58	M	Chronic congestive heart failure secondary to arteriosclerosis. Dyspnea, hemoptysis and cardiomegaly.	Incomplete right bundle branch block. Right atrial and ventricular enlargement.	*Chest x-ray:* Pulmonary emphysema with prominent pulmonary arteries. *Angiocardiogram:* marked pulmonary hypertension.	Multiple "cold" areas throughout both lower lung fields.	*Treatment:* for cardiac failure. *Clinical Diagnosis:* pulmonary emboli and infarction.

TABLE I

(GROUP 1, cont'd)

Case No.	*Initials*	*Age*	*Sex*	*Clinical Findings*	*ECG Findings*	*Radiographic Findings*	*Lung Scan Findings*	*Clinical Course and Final Diagnosis*
4.	J.P.	31	F	Chest pain, dyspnea and hemoptysis. Diminished breath sounds right lower lung.	S_1–Q_3–T_3 Pattern	*Chest x-ray:* Left pulmonary artery cutoff secondary to embolus. *Angiocardiogram:* embolus in left upper and obstruction right lower pulmonary artery.	Decreased concentration in both lower lung fields. Follow up scan showed improvement.	*Treatment:* anticoagulants and venal caval ligation. *Clinical Diagnosis:* bilateral pulmonary emboli.
5.	M.J.	38	F	Fracture cervical spine with quadriplegia. Nine days later hypotension, dyspnea and disorientation.	S_1–Q_3–T_3 Pattern	*Chest x-ray:* negative on admission. None obtained at time of acute episode.	Marked decrease in concentration right upper and left lower lobes.	*Treatment:* anticoagulants. *Autopsy:* large pulmonary emboli in right upper and left lower lung.
6.	E.P.	56	M	Chronic atrial fibrillation in obese patient (450 lbs). Chest pain, dyspnea, hemoptysis. Decreased fremitus left lung.	Atrial fibrillation	*Chest x-ray:* infiltrates in both lower lobes.	"Cold" areas in right and left lower lung field.	*Treatment:* anticoagulants. *Clinical Diagnosis:* pulmonary emboli and infarction.

Case No.	*Initials*	*Age*	*Sex*	*Clinical Findings*	*ECG Findings*	*Radiographic Findings*	*Lung Scan Findings*	*Clinical Course and Final Diagnosis*
7.	J.U.	30	M	Fever unknown origin and splenomegaly. Chest pain, hemoptysis and phlebitis. Cardiomegaly. Hepatomegaly.	Right axis deviation. P. pulmonale. Right precardial T wave inversion.	*Chest x-ray:* infiltrate in right midlung. *Angiocardiogram:* bilateral loss of peripheral vessels.	"Cold" area right upper lung and general decrease in concentration in left lung.	*Treatment:* anticoagulants. *Final Diagnosis:* recurrent pulmonary emboli and infarction.
8.	R.O.	40	F	Pelvic thrombophlebitis chest pain, dyspnea, hemoptysis. Right calf and femoral tenderness.	T wave inversion lead 3 and AUF	*Chest x-ray:* normal *Angiocardiogram:* obstruction to right midlung vessels.	Normal limits	*Treatment:* anticoagulants. *Final Diagnosis:* pulmonary emboli and infarction.
9.	R.C.	40	M	Fractured right ribs in auto accident. Chest pain, hemoptysis.	Normal	Infiltrate, left lower lung.	Decreased concentration in left lung with patchy concentration upper and lower lungs.	*Treatment:* anticoagulants. *Final Diagnosis:* pulmonary embolism.

TABLE I

(GROUP 2)

Case No.	*Initials*	*Age*	*Sex*	*Clinical Findings*	*ECG Findings*	*Radiographic Findings*	*Lung Scan Findings*	*Clinical Course and Final Diagnosis*
10.	A.G.	20	F	Chest pain, dyspnea and cardiomegaly. P2 greater than A2. Holosystolic murmur and edema.	Right ventricular hypertrophy. P. pulmonale	*Chest x-ray:* prominent pulmonary arteries suggesting pulmonary hypertension.	Normal	*Treatment:* for congestive heart failure. *Final Diagnosis:* primary pulmonary hypertension. Questionable multiple small pulmonary emboli.
11.	M.B.	18	F	Chronic glomerulonephritis. Edema, uremia, congestive heart failure, chest pain, dyspnea, and hemoptysis.	S_1–Q_3 pattern	*Chest x-ray:* infiltrate in right lower lobe.	Decreased concentration right lower lobe, anterior and lateral scan.	*Treatment:* anticoagulants and urokinase. *Final Diagnosis:* possible pulmonary emboli.
12.	G.F.	70	M	Bilateral calf pain Chest pain, hypotension. Bilateral rales in lungs. Positive calf tenderness	Subendocardial injury and ischemia	*Chest x-ray:* normal	Normal with scalloping of left border	*Treatment:* anticoagulants. *Final Diagnosis:* thrombophlebitis with possible pulmonary emboli.
13.	A.B.	57	F	Dyspnea, chest pain, hemoptysis.	Antero-lateral myocardial infarction	*Chest x-ray:* left pleural effusion with minimal effusion on the right.	Decreased concentration in left lower lobe.	*Treatment:* symptomatic *Final Diagnosis:* possible pulmonary embolus.

TABLE I

(GROUP 3)

Case No.	*Initials*	*Age*	*Sex*	*Clinical Findings*	*ECG Findings*	*Radiographic Findings*	*Lung Scan Findings*	*Clinical Course and Final Diagnosis*
14.	F.H.	32	F	Raynaud's phenomenon, arthritis, dyspnea, gross cardiomegaly, hepatomegaly. Positive LE Test	Sinus tachycardia	*Chest x-ray:* cardiomegaly, suggestive of pericardial effusion. *Angiocardiogram:* large pericardial effusion.	Decreased concentration in left lower lobe due to a large heart.	*Treatment:* pericardial tap revealing bloody effusion. *Autopsy:* disseminated lupus erthymetosis.
15.	R.C.	49	M	Dyspnea, hypertension bilateral pulmonary rales.	S_1–T_3 pattern	*Chest x-ray:* emphysema with bleb and/or fibrosis in right lower lobe.	Patchy decrease in concentration right lower lobe.	*Treatment:* directed to pulmonary disease. *Final Diagnosis:* pulmonary fibrosis and emphysema with bleb in right lower lobe.
16.	H.H.	33	F	Chest pain, dyspnea, hypertension, pedal edema. Low grade azotemia.	Wolff-Parkinson-White Syndrome	*Chest x-ray:* normal	Normal with scalloping of left lateral lung.	*Treatment:* steroids. *Final Diagnosis:* glomerulonephritis.
17.	F.T.	57	M	Syncopal episodes with seizures. 7th nerve weakness. Bilateral Babinski sign.	Normal	*Chest x-ray:* mass in right midlung.	Decreased concentration in right and left midlung.	*Treatment:* chemotherapy *Final Diagnosis:* metastatic sarcoma to the lung.

TABLE I

(GROUP 3, cont'd)

Case No.	*Initials*	*Age*	*Sex*	*Clinical Findings*	*ECG Findings*	*Radiographic Findings*	*Lung Scan Findings*	*Clinical Course and Final Diagnosis*
18.	E.G.	39	F	Postpartum subcapsulary hematoma of liver drained surgically followed by chest pain and dyspnea.	S_1–Q_3–T_3 Pattern	*Chest x-ray:* subdiaphragmatic abscess.	Decreased concentration right lower lung with separation of lung and liver.	Surgical drainage of abscess. *Clinical Diagnosis:* right subphrenic abscess.
19.	S.S.	20	F	Pulmonary arterial venous fistula present for ten years with clubbing and cyanosis of fingers	Right ventricular hypertrophy	*Chest x-ray:* shows rounded lesions in both lower lobes. *Angiocardiogram:* bilateral A-V malformations.	Normal	*Treatment:* none *Clinical Diagnosis:* pulmonary A-V fistulas.
20.	L.P.	37	M	A normal volunteer without symptoms	None	Normal	Normal with scalloping of left lateral lung border.	No pulmonary disease.

PULMONARY EMBOLIC DISEASE

Age and Sex: In Group 1, the mean age was 38 years with a range of 18 to 56 years. There were five males and four females.

Clinical Symptoms: In Group 1, the most common presenting symptoms were dyspnea and chest pain (seven patients). Hemoptysis (six patients) and phlebitis (two patients) were also common symptoms.

Physical Findings: Among patients with pulmonary embolism there were six with cardiomegaly and five with abnormal breath sounds. Two patients had physical evidence of phlebitis.

Laboratory Findings: In Group 1, five patients were tested for elevations of serum glutamic oxalocetic and pyruvic transaminase; two patients had elevated levels. The serum bilirubin was elevated in two of five patients tested.

Electrocardiogram: The ECG tracings were abnormal in eight out of nine patients studied in Group 1. The changes noted were a typical S_1-Q_3-T_3 pattern (two patients), ventricular hypertrophy (two patients), T wave inversion (two patients) and P pulmonale (one patient).

Radiographic Findings: Of the nine patients in Group 1, eight had chest x-rays performed during the acute symptomatic episode; of these, seven were abnormal (five showed infiltrates, one showed a pulmonary artery "cut-off" sign and one showed prominent pulmonary arteries). One patient had a normal chest x-ray.

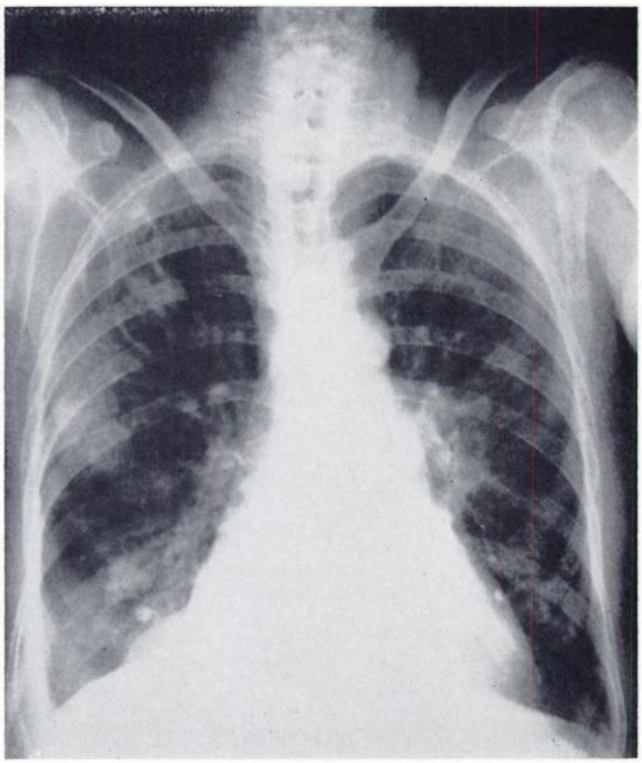


Fig. 1A Case No. 1—(H.G.)—Chest x-ray showing bilateral pulmonary infiltrates more marked in the right lung and cardiomegaly.

Five of the nine patients had venous angiocardiagrams and these were abnormal in all five cases. Three of the angiocardiograms revealed obstructive arterial lesions and two showed loss of peripheral vessels and pulmonary hypertension (prominent pulmonary hilar vessels with abrupt attenuation of vascular lumens just distal to the hilar area).

Lung Scan Findings: In Group 1, eight out of nine patients had abnormal lung scans. The abnormalities noted were "cold" areas which were multiple in seven patients and single in one patient. The patient with a normal lung scan had obstruction of a small pulmonary artery with an otherwise normal chest x-ray and was thought clinically to have a small pulmonary embolus.

The findings typical of pulmonary embolism and infarction were epitomized by a patient with chronic congestive heart failure secondary to rheumatic heart disease (Fig. 1). The smaller peripheral infarcts appeared as wedge-shaped areas in the left lung of this patient. The larger more central emboli caused generalized reduction in the concentration of radioactivity throughout the entire right lung.

In one patient serial lung scans were performed which revealed a change in the appearance of the scan in pulmonary embolism (Fig. 2). The initial scan revealed marked reduction in concentration of activity in the entire left lung and lower right lung. The venous angiocardiogram confirmed this finding. Eighteen days later after clinical improvement had taken place, the lung scan revealed a more uniform distribution of macroaggregated albumin though some abnormality persisted. The patient was treated with anticoagulants and vena caval ligation.

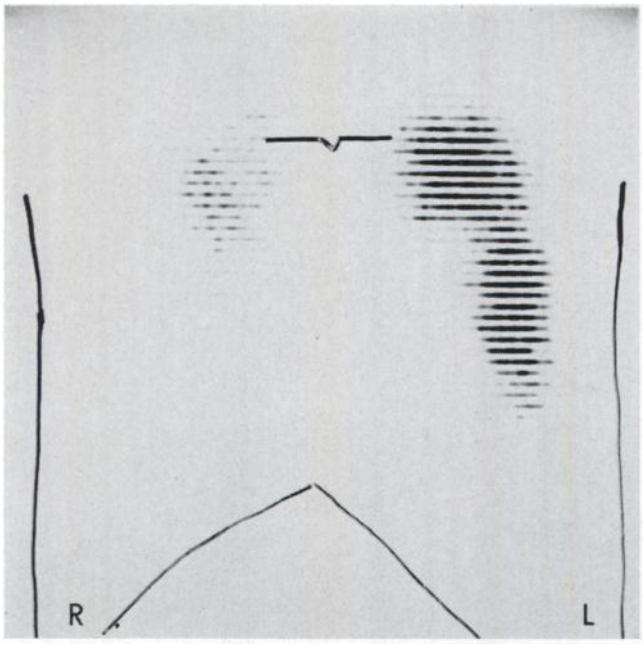


Fig. 1B Lung scan in the same patient showing two "wedge-shaped" cold areas in the left lung and generalized reduction of concentration in the right lung compatible with multiple pulmonary infarcts.

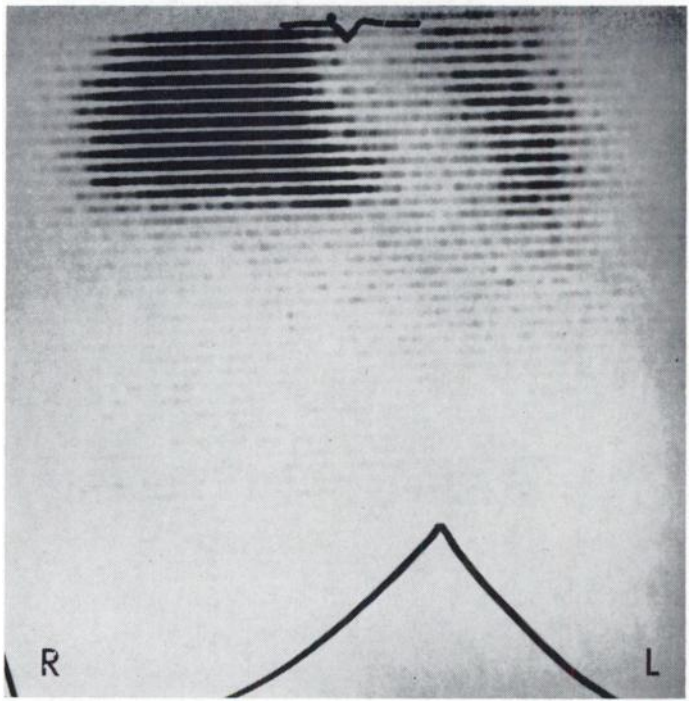


Fig. 2A Case No. 4 (J.P.)—Lung scan during acute episode showing cold area in right lower lung and generalized reduction in concentration in the left lung.

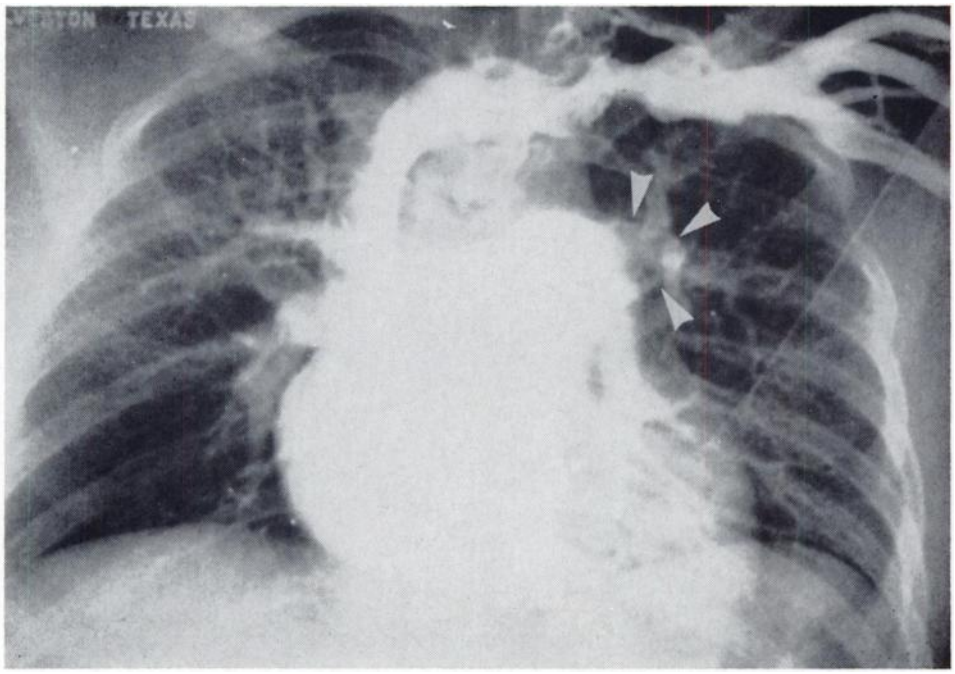


Fig. 2B Venous angiogram done 15 days later revealing large embolus in left main pulmonary artery, (arrow), and diminished vascularity in right lower lung.

The majority of patients in Group I survived the acute episode with conservative medical therapy (anticoagulants). One patient received additional therapy with fibrinolytic agents and one patient required a vena caval ligation. Only one patient died in Group I. This patient had massive pulmonary embolism and expired several hours after the scan was performed. The autopsy revealed the presence of pulmonary emboli in the exact locations suggested by the scan.

MISCELLANEOUS PULMONARY DISEASE

Radiographic Findings: In Group 3 patients, a variety of radiographic findings included the following: pericardial effusion (one patient), pulmonary emphysema and fibrosis (one patient), pulmonary neoplasm (one patient), subdiaphragmatic abscess (one patient), A-V malformation (one patient) and normal (two patients).

Lung Scan Findings: In Group 3, the lung scan was abnormal in four of six patients (excluding one normal volunteer). There was patchy concentration which was related to an emphysematous bleb with localized fibrosis in one patient and metastatic pulmonary neoplasm in another. In one patient, the left lower lobe was "cold" due to the presence of a pericardial effusion (Fig. 3). A large subdiaphragmatic abscess, (Fig. 4), however, produced marked "coldness" in the

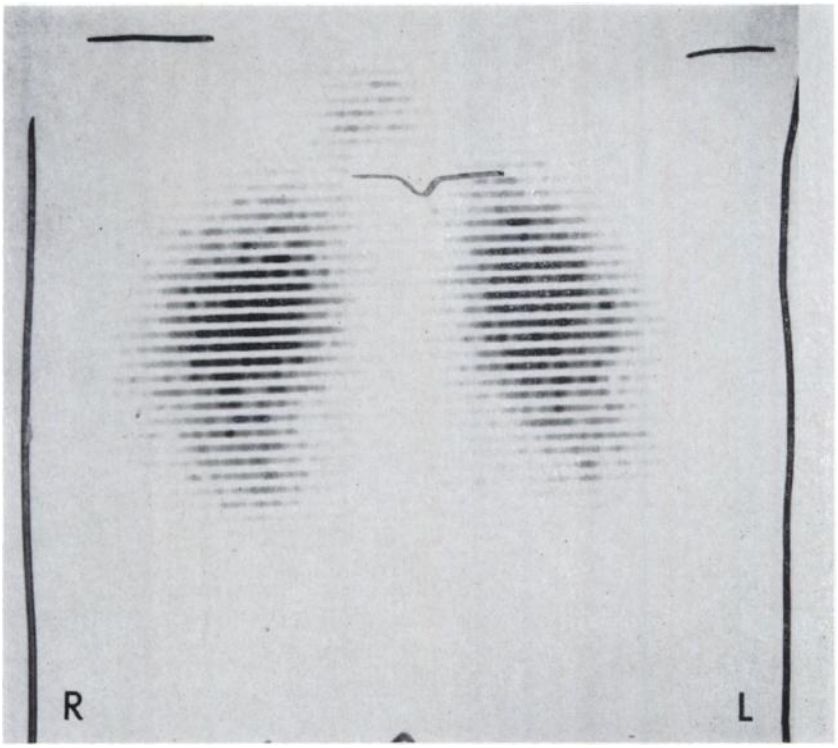
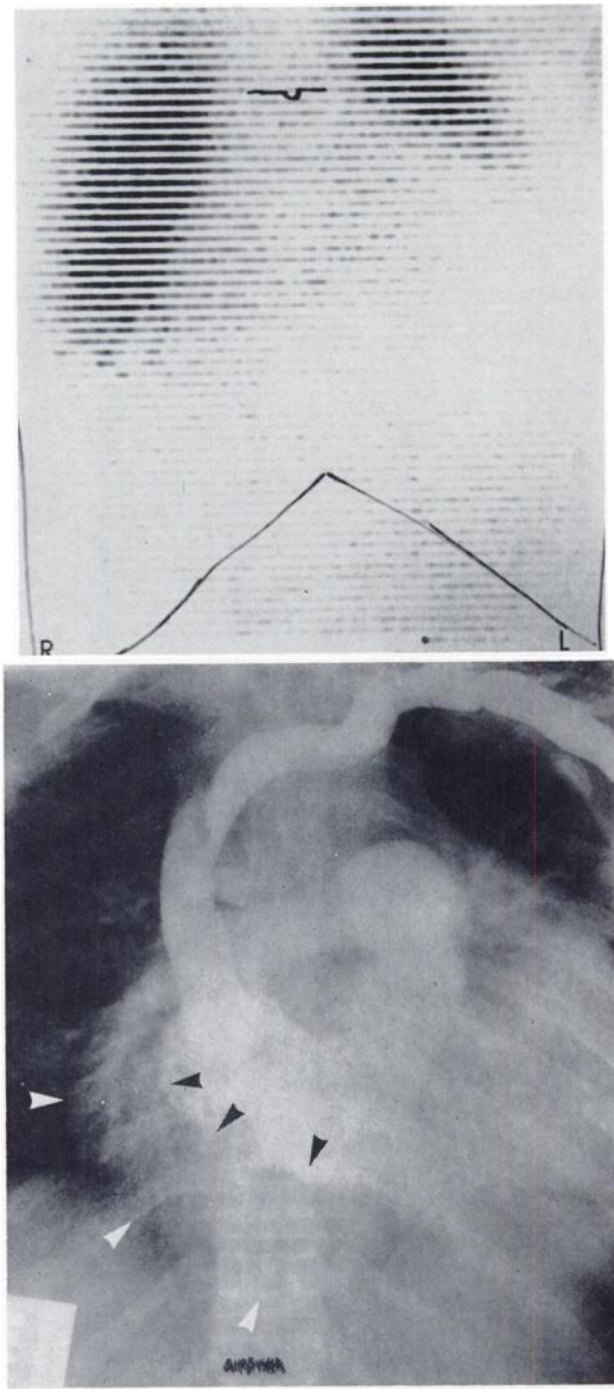


Fig. 2C Lung scan performed 18 days after 1st scan after clinical improvement. The concentration is now equal in both lungs with still some decrease in concentration in the lower lobes bilaterally.



A.

B.

Fig. 3 Case No. 14—(F.H.) (a) Lung scan showing decreased concentration of radioactivity in the left lower lobe. The heart area is increased in size. (b) Venous angiogram on the same patient demonstrating the large pericardial effusion that caused the abnormality on the lung scan. White arrows point to the outer aspect of the pericardium; black arrows point to the lateral extent of the cavity of the right heart.

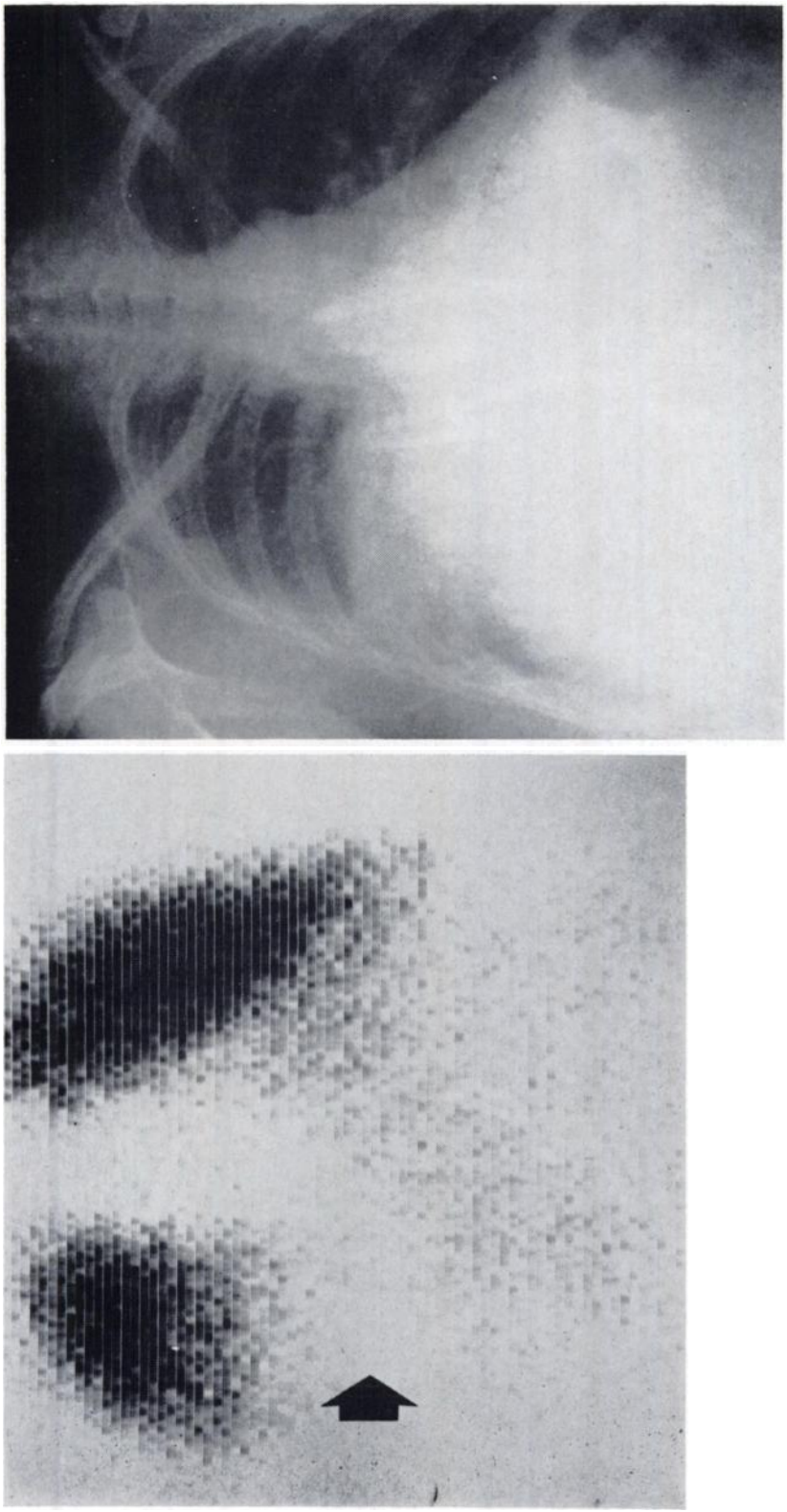


Fig. 4. Case No. 18 (E.G.)—(a) Lung scan demonstrating decreased concentration of radioactivity in the right lower lung. Note the liver concentration in the central lower portion of the scan and the space separating the lung and liver activity (arrow). (b) Chest x-ray on same patient showing the elevation of the right hemidiaphragm and pleural effusion. At exploration a large right subphrenic abscess was found.

right lower lobe and increased separation between the right lung and liver uptake. In one patient, the presence of multiple A-V malformations failed to produce any change in the lung scans. Of the remaining patients with normal lung scans and no evidence of pulmonary emboli, two had scalloping of the left lateral lung border. One of these patients was a normal volunteer while the other had glomerulonephritis with hypertension and azotemia. One other patient in this series (G. F. in Group 2) had the finding of irregularity along the left lung border (Fig. 5). Although this could be interpreted as an abnormal finding, we consider it a normal variation which cannot be used to support the diagnosis of pulmonary embolism.

In Group 3, one patient with a massive pericardial effusion secondary to disseminated lupus erythematosis expired several days after lung scanning. The autopsy revealed no evidence of pulmonary embolism, also as suggested by the scan.

CLINICAL CORRELATIONS IN PULMONARY EMBOLI

The incidence of presence of certain symptoms, signs, and laboratory findings in the nine patients with pulmonary embolic disease is summarized in Table II. The lung scan compared favorably to other studies employed in the evaluation

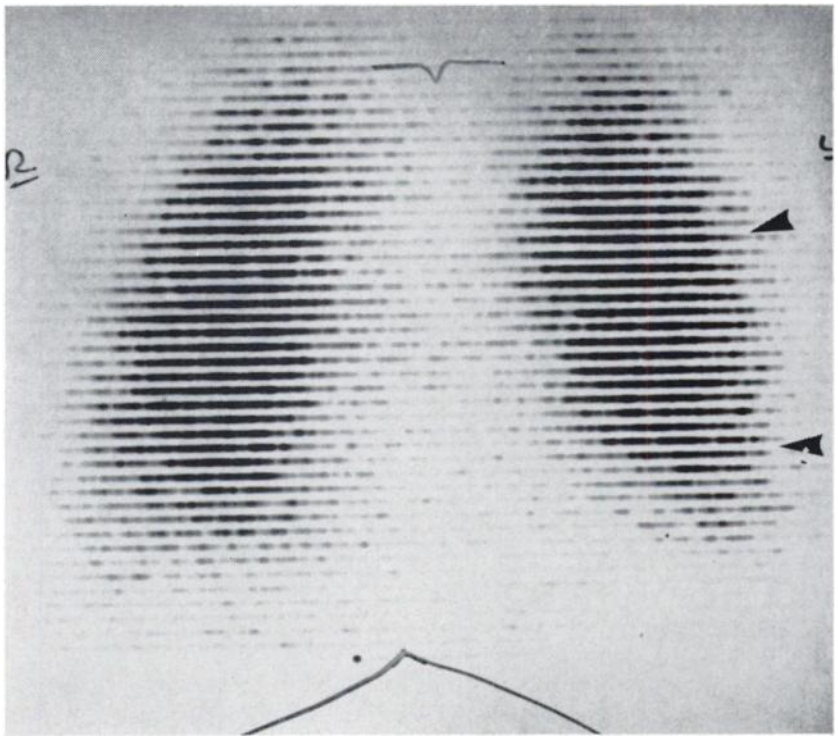


Fig. 5. Case No. 12 (G.F.)—Lung scan interpreted as normal in a patient with possible pulmonary emboli (Group 2). Some irregularity is noted along the left lateral lung border (arrows). This may be related to movement of the patient during the scanning process. This type of change has been observed in normal patients and probably should be interpreted as a normal variation.

of these cases. The pulmonary angiogram appears to be the study with the most accuracy in pulmonary embolic disease.

Specificity: The incidence of the presence of symptoms or abnormalities of laboratory tests, although of interest, does not tell the whole story. Of equal interest and importance are the abnormalities observed and their specificity for pulmonary embolic disease. Those abnormalities which were felt to be reasonably suggestive of pulmonary emboli are listed in Table III. In this analysis, the lung scan compared more favorably to these other procedures. Of course, this series is small and our experience in other conditions limited. However, the presence of a lung scan abnormality, particularly if there are multiple defects in the presence of a negative or compatible chest film, is strongly suggestive of pulmonary emboli. One caution here is that the lung scan should not be superimposed over the chest film, unless care is taken to prevent magnification of the chest x-ray, and chest x-rays taken in deep impression and upright will always show more lung area than the lung scan performed during quiet respiration. Also, the upper and lower borders of the lung scan are frequently "patchy" due to the movement of the lungs by respiration during the scanning process.

DISCUSSION

The present study indicates a sufficiently high degree of correlation between the lung scan and the clinical diagnosis of pulmonary embolism to recommend it as a routine screening procedure in the diagnosis of this disorder. Because abnormalities are observed in other pulmonary diseases, caution should be exercised in drawing conclusions from a positive or a negative study.

A number of reports have appeared in the literature which also indicate a role for this procedure in the diagnosis of pulmonary embolism. Wagner and

TABLE II

CLINICAL AND LABORATORY FINDINGS IN 9 PATIENTS WITH PULMONARY EMBOLIC DISEASE

Finding	*No. Patients Positive*	*No. Patients Negative*	*% Positive*
Dyspnea	7	2	78%
Chest pain	7	2	78%
Hemoptysis	6	3	67%
Phlebitis	2	7	22%
Cardiomegaly	6	3	67%
Abnormal breath sounds	5	4	56%
Serum Bilirubin	2	3	40%
ECG	8	1	89%
Chest x-ray	7	1	87%
Lung scans	8	1	89%
Angiograms	5	0	100%

associates found a good correlation between massive pulmonary embolism diagnosed by "cold" areas on the lung scan and autopsy findings in five patients (10). They also found abnormalities in a variety of other pulmonary disorders. In the one patient in our series who came to autopsy with the diagnosis of massive pulmonary embolism, there was an excellent correlation between the two findings. Quinn and associates also found a good correlation between pulmonary embolism and infarction and findings on lung scans (7) and like Wagner, also found abnormalities in a variety of other pulmonary disorders. Taplin and associates found abnormalities to exist in pulmonary emboli, infarcts, pneumonia, atelectasis, cysts and abscesses which all appear as areas of reduced radioactivity on the scan (8). They felt that the scan was of diagnostic value especially when used in conjunction with the chest x-ray. From all of these studies and from our own experience, it appears that the lung scan can point to the site of embolic lesions before signs of lung infarction are recognizable on plain chest films.

From our own experience, we can confirm that the lung scan will be positive in a high percentage of patients with pulmonary embolism and infarction. Out of nine patients who were classified as probable or definite pulmonary infarction, the lung scan was abnormal in eight. In four patients with possible pulmonary emboli, the scan was abnormal in two. In one case of idiopathic pulmonary hypertension thought possibly due to multiple small pulmonary emboli, the lung scan was normal. In another patient with clinical signs of pulmonary infarction,

TABLE III

ABNORMALITIES SUGGESTIVE OF PULMONARY EMBOLIC DISEASE OBSERVED IN 9 PATIENTS IN GROUP I

ECG		
S_1–Q_3–T_3 Pattern	2 patients	
Right Bundle Branch Block	2 patients	
Right Axis Deviation	1 patient	
	5 patients out of 9	56%
Chest x-ray		
Pulmonary Infiltrates	5 patients	
Pulmonary artery "cut-off"	1 patient	
	6 patients out of 8	75%
Lung Scan		
Decreased Blood Flow to Multiple Areas	7 patients	
Single Area	1 patient	
	8 patients out of 9	89%
Venous Angiocardiogram		
Obstruction to Pulmonary Artery	3 patients	
Decreased Pulmonary Blood Flow to Multiple Areas	2 patients	
	5 patients out of 5	100%

but no roentgenographic findings, the scan was normal except for some indentations along the left border of the lung. These were not of sufficient size to warrant an impression of the crescent sign seen on the lung borders in Wagner's series and fall into what we consider a normal variation. This consideration was necessitated by a similar finding in a normal volunteer with no evidence of any pulmonary disease.

The importance of considering other causes for abnormal lung scans is emphasized in our Group 3 patients who showed a 67 percent incidence of abnormality. We found abnormalities on the scan due to pericardial effusion, subdiaphragmatic abscess, neoplasms and pulmonary emphysema and fibrosis of the lung. In one patient with multiple pulmonary A-V fistulas, there was no abnormality. One patient with glomerulonephritis had scalloping of the left lateral lung border.

In our experience, the lung scan has been safe and simple to perform. No adverse reaction is observed in any of these patients which could be attributed directly to the performance of the lung scan. Indeed the animal studies performed by others indicate that the number of stable particles injected in a lung scan is far below that which could cause any difficulty from mechanical blockage of pulmonary capillaries (6). The only other significant hazard from such an examination is the radiation hazard. Wagner has calculated a dose to the lungs of .3 rads for ^{131}I macroaggregated albumin (10).

The place of lung scanning in the workup of patients with suspected pulmonary embolism and infarction will have to be defined from a larger series of cases. Our current feeling is that patients suspected of having pulmonary embolism should receive all available studies including angiography in addition to the lung scan. It is only in this way that we will be able to correlate the accuracy and specificity of these tests. We have been successful in obtaining good pulmonary angiograms using a large amount of contrast material injected intravenously (80–100 cc) and have not felt it necessary to perform selective catheter studies.

The lung scan offers a method, not only of initial diagnosis, but also of following patients after the initial episode. In one patient in whom follow-up examinations were performed, it appeared that greater pulmonary blood flow was present on the second scan than on the first, indicating some improvement. In this patient, treatment has been with anticoagulants alone. There is current interest in the evaluation of urokinase as well as surgical procedures in the treatment of pulmonary emboli. Carefully controlled studies of the natural history of pulmonary embolism will be necessary before the role of any specific agent can be definitely ascertained. The lung scan will offer valuable information in this regard.

SUMMARY

A series of twenty patients studied with lung scans performed with ^{131}I macroaggregated albumin has pointed out some areas of usefulness and potential limitation of this procedure in clinical practice. In nine patients with probable to definite pulmonary infarction, the lung scan was abnormal, showing "cold" areas, in all but one. The chest x-ray was abnormal in seven out of eight pa-

tients examined. Angiocardiograms were performed in five patients and were abnormal in all five patients. In seven patients with a variety of other pulmonary disorders, including one normal volunteer, the lung scan was abnormal in five showing decreased concentration in areas of pathology ranging from subdiaphragmatic abscess to pericardial effusion and pulmonary neoplasms. Irregularity of the left lung border was found in a normal patient as well as in several patients with equivocal pulmonary disease and is felt to be a normal anatomic variation.

The lung scan is simple to perform and can be carried out as a bedside procedure on the critically ill patient. It is safe, no immediate reaction appearing and no delayed sequelae developing during the period of follow up in this series. It was possible to perform serial studies to follow the results of therapy. The lung scan, though showing a good correlation with clinical diagnosis of pulmonary embolism and infarction should not be relied upon as the only procedure in the diagnosis of this disorder. The final place of this procedure in clinical practice will await the accumulation of further data, but preliminary reports indicate a great deal of promise for the procedure.

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