

NEW PATTERNS OF ABNORMAL PULMONARY PERFUSION ASSOCIATED WITH PULMONARY EMBOLI:

1. SCINTIGRAPHIC MANIFESTATIONS

S. Boyd Eaton, A. Everette James, Reginald E. Greene,
Joseph H. Lyons, Majic S. Potsaid and Felix G. Fleischner

Harvard Medical School and Massachusetts General Hospital, Boston, Massachusetts

Isotope radiography has established itself as a simple, safe and reliable method for showing pulmonary perfusion abnormalities, particularly those associated with pulmonary embolism. The "characteristic" scintigraphic pattern of this pathology is a discrete, localized area of decreased radioactivity. In some instances the isotopic pattern complements the roentgenographic signs of pulmonary infarction as originally described by Hampton and Castleman (1) and recently amplified by Fleischner (2). When the chest roentgenogram is negative, scintigraphic demonstration of underperfusion is of even greater value. This communication describes a scintigraphic pattern (noted by M. S. Potsaid) which is apparently associated with pulmonary embolism and which has not been previously discussed.

MATERIALS AND METHODS

Over 200 lung scintigrams performed on patients clinically suspected of having pulmonary embolic disease were reviewed. The examinations were performed at the Massachusetts General Hospital between August, 1966 and April, 1968. High specific-activity macroaggregated human albumin labeled with ^{131}I was the radiopharmaceutical used; in nearly all instances an intravenous dose of 300 μCi (0.3–1.0 mg of albumin) was administered. The macroaggregated particles are predominantly 25–50 microns in diameter, and they display the normally perfused portion of the pulmonary arterial bed by impacting for a time in alveolar capillaries which are 8–15 microns in diameter.

An Anger scintillation camera (3) was used for all examinations. This stationary detector is an 11.5-in. NaI(Tl) crystal with a parallel-hole collimator. The apparatus images isotope distribution without mechanical scanning, and the detector can be oriented throughout a broad range. Because this feature simplifies patient positioning, multiple views (i.e. both laterals and two anterior and/or posterior projections) are invariably obtained. In most cases,

175,000 counts were accumulated for each view, but if the counting rate was unusually low (e.g. as over areas of decreased perfusion), the cathode-ray-tube intensity was increased and fewer counts were accumulated.

RESULTS

The appearance of the normal lung, as depicted by the scintillation camera is one of regularly distributed radioactivity throughout both lung fields. On frontal projections, the heart, mediastinum and occasionally the hila can be identified as areas in which radioactivity is essentially absent. The left lateral view may show decreased activity in the area of the heart, particularly if cardiomegaly is present. The fissures are not normally visible (Fig. 1).

The conventional scintigraphic pattern of a major pulmonary embolus is that of decreased perfusion in the area served by the involved artery, be it segment, lobe or entire lung (Fig. 2). Embolization of smaller arteries causes smaller, half-spindle-shaped or crescentic areas of underperfusion (Fig. 3). All these involved areas extend to the periphery of the lung and are "pleural based."

Our review reveals an additional pattern commonly observed in cases with a high probability of pulmonary embolism. This finding is a circumferential zone of decreased radioactivity corresponding to diminished perfusion in the periphery of one or more lobes. Decreased activity may be apparent around the circumference of the involved portion of the lung, causing the lobe to appear small in volume (Fig. 4), or it may be identified as a curvilinear zone along an interlobar fissure (Fig. 5). Where several lobes and/or segments are involved, a striking pattern of decreased activity along the fissures is produced (Fig. 6). There are instances in which

Received Oct. 22, 1968; revision accepted March 18, 1969.

For reprints contact: S. Boyd Eaton, M.D., Massachusetts General Hospital, Boston, Mass. 02114.

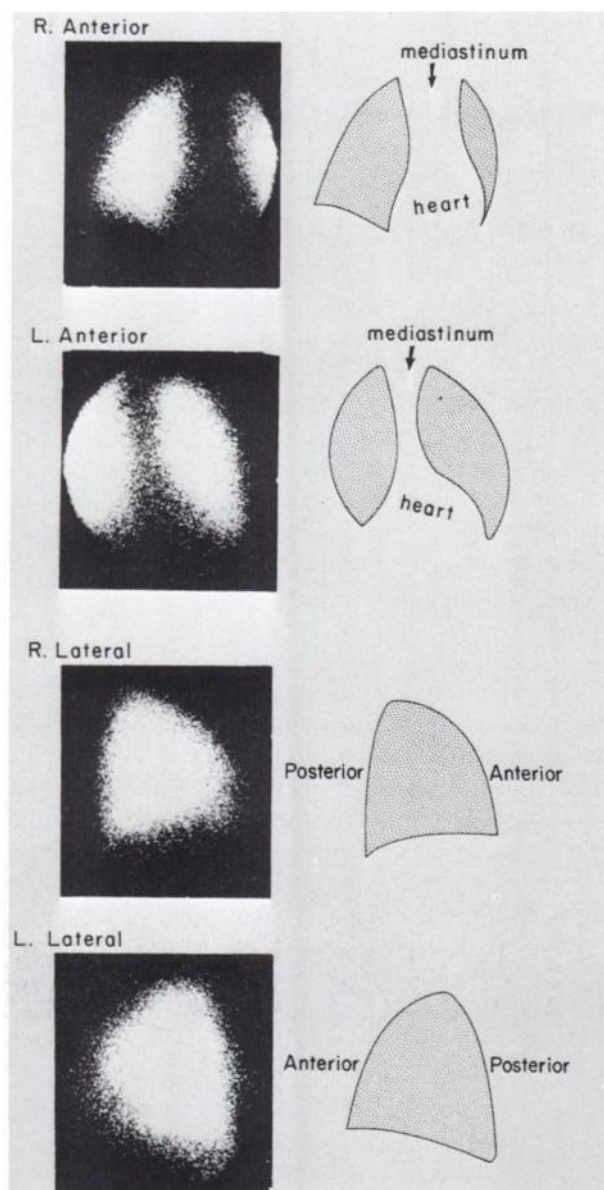


FIG. 1. Normal lung shown by scintillation camera.

circumferential ischemia can be seen in one region of the pulmonary vasculature and a crescentic defect elsewhere in the same patient (Fig. 7).

The scintigrams chosen for illustration are of patients whose concurrent roentgenograms were essentially normal or without apparent abnormalities, such as pleural effusion, which might account for the defects noted. Most of the patients had strong clinical evidence of pulmonary embolism. Two of these patients later came to postmortem examination and were indeed found to have multiple pulmonary emboli in the region of the perfusion defects.

DISCUSSION

To present certain concepts more clearly, a brief review of the pertinent anatomy and pathology is

in order. The pulmonary arteries ramify through about 20 orders of successive branchings from the hila to the periphery of the lung (4). Two types of branches occur (5). Axial vessels diverge to form gentle oblique angles and constitute the direct pathway for blood flow. The lumen of these vessels gradually diminishes, particularly in the periphery of the lung; such vessels inevitably catch and hold microemboli carried to them by the pulmonary circulation. Numerous side branches arise from the axial arteries almost perpendicularly at all levels to supply the capillaries of adjacent lung tissue (6). Microemboli tend to bypass the side branches and lodge relatively selectively in the peripheral axial vessels.

These relationships establish the anatomic basis for scintigraphic findings where pulmonary microembolism has occurred. Multiple microemboli tend to preferentially decrease blood flow to the peripheral portion of the lung relative to what is seen centrally.

Pulmonary microembolism has been increasingly recognized as a significant medical problem (7-9). The radiographic, arteriographic and scintigraphic manifestations of massive pulmonary embolism have been carefully characterized, but small and microscopic emboli occur more commonly than is generally appreciated and usually remain undiagnosed. In a study using postmortem pulmonary arteriogra-

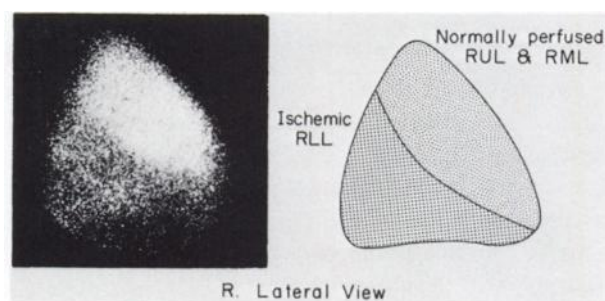


FIG. 2. Decreased perfusion throughout entire right lower lobe.

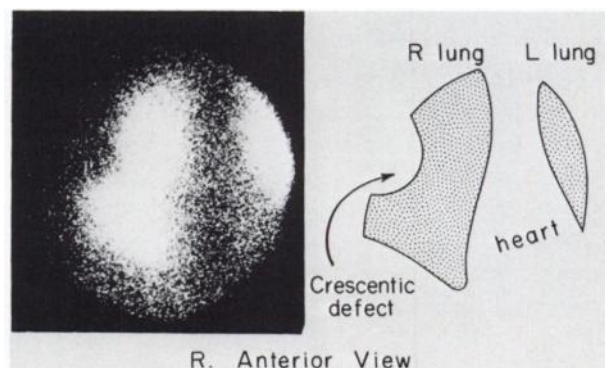


FIG. 3. Classic crescentic defect along lateral border of lung.

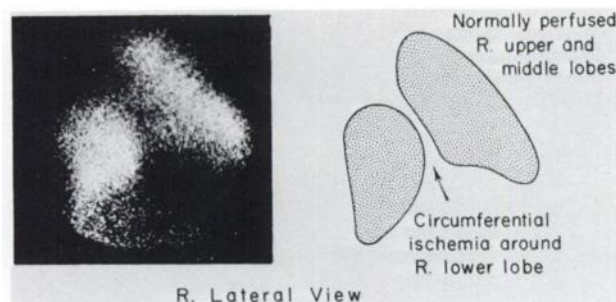


FIG. 4. Circumferential ischemia around periphery of right lower lobe.

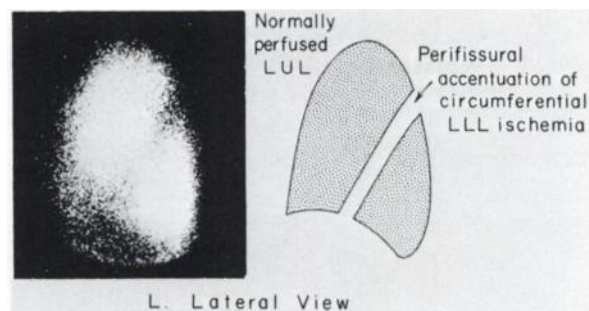


FIG. 5. Periffissural accentuation of circumferential left lower lobe ischemia.

phy, serial sectioning of lung slices and intensive microscopic examinations, Smith, Dexter and Dammin found multiple thromboemboli in arteries smaller than 1 mm in diameter in all cases of histologically proven pulmonary embolism. Thromboemboli were present in the larger hilar and segmental arteries in only one third of the cases (10,11).

Several mechanisms can be postulated for microembolism. Multiple small emboli may originate in the extremities or pelvic veins. Alternatively, discrete clots formed peripherally may fragment (during their passage through the heart) on the chordae tendineae and papillary muscles of the tricuspid valve (12). In both instances, small emboli would be distributed to all regions of the lungs in a fairly random fashion. Finally, larger emboli arrested in central pulmonary arterial branches may disintegrate. The resulting fragments will be distributed to more peripheral vessels in the involved lobe or segment.

The condition or conditions that might produce the patterns described in this communication have not been precisely defined. However, there is little doubt that circumferential and periffissural hypoperfusion are related to pulmonary embolism. This contention is supported not only by our lung isotope radiography but also by detailed studies of histologically proven specimens using postmortem pulmonary angiography (10,11).

While the relationship to pulmonary embolism seems clear, the mechanisms for the observed changes are not so clear. There are at least three ways in which peripheral pulmonary hypoperfusion might be produced. In a purely mechanical manner microemboli lodging in the peripheral pulmonary axial arteries may be expected to produce circumferential hypoperfusion with relative sparing of the central portion of the involved lobe or lobes. Alternatively, Smith and his coworkers have shown the development of bronchopulmonary collateral circulation after pulmonary thromboembolism (13). Such collaterals form around the periphery of the involved lobe. Blood reaching the periphery

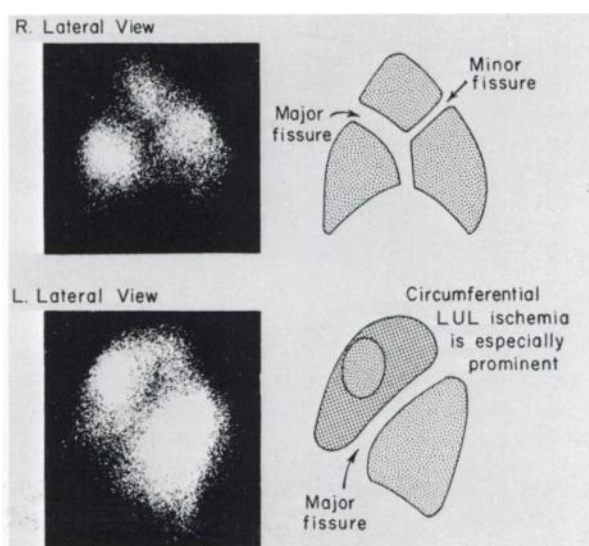


FIG. 6. Generalized circumferential ischemia involving multiple lobes in both lungs.

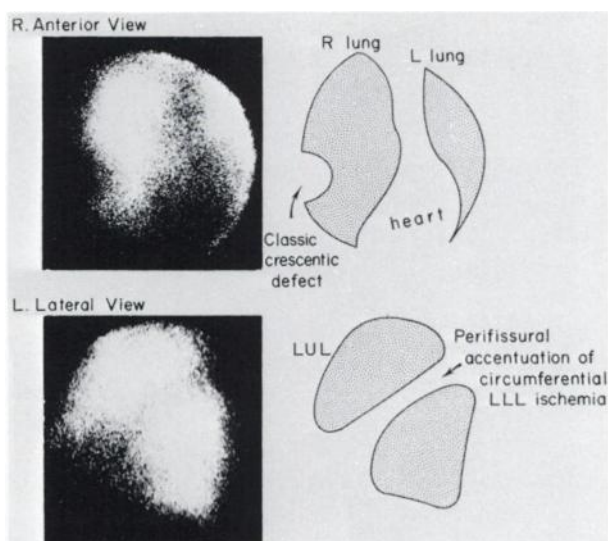


FIG. 7. Classic crescentic defect along lateral border of right lung occurring in conjunction with periffissural accentuation of circumferential left lower lobe ischemia.

of the lung through the bronchial arteries will not contain radioactivity because radioactive particles reach the lungs through the pulmonary arteries after an i.v. injection. Finally, reflex vasoconstriction may occur in association with pulmonary embolism (14, 15). This phenomenon could create or augment peripheral hypoperfusion. We are currently investigating the role of each of these mechanisms in experimental animals (16).

SUMMARY

A scintigraphic pattern consisting of peripheral pulmonary hypoperfusion is presented. This curvilinear or circumferential zone of decreased radioactivity is usually best recognized along the interlobar fissures. It is thought that the observed pattern results from numerous microemboli lodging in the small axial arteries at the lung periphery. In cases where multiple views, especially lateral projections, are obtained with the gamma camera, it is almost as common as the generally accepted discrete local defect.

ACKNOWLEDGMENTS

The authors gratefully acknowledge the valuable assistance of the following persons: our physicist, Miss Hing Har Lo; our technicians, Mrs. Norita Leary, Miss Sally West and Miss Diane Wathen; and our secretaries, Miss Lorraine Zagami and Miss Toni-Marie Condangelo. This work was supported by Training Grant GM-1339 from the National Institute of General Medicine, National Institutes of Health.

REFERENCES

1. HAMPTON, A. O. AND CASTLEMAN, B.: Correlation of postmortem chest teloroentgenograms with autopsy findings with special reference to pulmonary embolism and infarction. *Am. J. Roentgenol.* **43**:305, 1940.
2. FLEISCHNER, F. G.: Roentgenology of the pulmonary infarct. *Seminars in Roentgenology* **2**:61, 1967.
3. ANGER, H. O.: Scintillation camera with multichannel collimators. *J. Nucl. Med.* **5**:515, 1964.
4. KRAHL, V. E.: The lung as a target organ in thromboembolism. In *Pulmonary Embolic Disease*, Sasahara, A. A. and Stein, M., eds. Grune & Stratton, New York, 1965, pp. 13-22.
5. ELLIOTT, F. M. AND REID, L.: Some new facts about the pulmonary artery and its branching pattern. *Clin. Radiol.* **16**:193, 1965.
6. PRICHARD, M. M. L., *et al.*: Peripheral ischaemia of the lung. *Brit. J. Radiol.* **27**:93, 1954.
7. Pulmonary microembolism, leading articles. *Lancet* **1**:429, 1967.
8. BLAISDELL, F. W., *et al.*: Pulmonary microembolism. *Arch. Surgery* **93**:776, 1966.
9. MADDISON, F. E., *et al.*: Pulmonary microembolism—radiologic findings. *Radiology* **90**:1,176, 1968.
10. SMITH, G. T., *et al.*: Postmortem arteriographic studies of human lung in pulmonary embolism. *J. Am. Med. Assoc.* **188**:143, 1964.
11. SMITH, G. T. *et al.*: Postmortem quantitative studies in pulmonary embolism. In *Pulmonary Embolic Disease*, Sasahara, A. A. and Stein, M., eds. Grune & Stratton, New York, 1965, pp. 120-130.
12. WESSLER, S., *et al.*: Experimental pulmonary embolism with serum induced thrombi. *Am. J. Pathol.* **38**:89, 1961.
13. SMITH, G. T., *et al.*: Human systemic—pulmonary arterial collateral circulation after pulmonary thromboembolism. *J. Am. Med. Assoc.* **188**:452, 1964.
14. DALEN, J. E., *et al.*: Cardiovascular responses to experimental pulmonary embolism. *Am. J. Cardiol.* **20**:3, 1967.
15. WIENER, S. N., *et al.*: Observations on pulmonary embolism and the pulmonary angiogram. *Am. J. Roentgenol.* **98**:859, 1966.
16. JAMES, A. E., *et al.*: An experimental model to study pulmonary microembolism. *Radiology* **92**:924, 1969.