Clinical Use of Ultrasound in the Early Diagnosis of Pulmonary Embolism

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IT IS NOT always appreciated that the mortality rate from pulmonary embolism in this country each year is truly staggering. The lethal issue may occur suddenly from massive embolism, or over the course of months or years from repeated small emboli, producing chronic cor pulmonale and death from right heart failure. It has been stated that "pulmonary embolism is now the most common lethal pulmonary disease in adults seen at necropsy in a general hospital population. It is the third most common nonlethal pulmonary lesion after pneumonia and emphysema."[34] At the Peter Bent Brigham Hospital, in 1960 it was the third most common cause of death, the main cause in 15% of patients autopsied, and a contributory cause in an additional 19%. Coon and Willis have estimated that 47,000 deaths occur annually from embolism to the lungs.[9] From autopsy statistics alone, the overall incidence rates of fatal and non-fatal pulmonary emboli, range between 6 and 64%.[3-5, 16, 17, 24, 25, 29, 36] In various reported series, embolism has been the cause of death in 2 to 15% of all patients autopsied.[4, 17, 19, 20, 24, 26, 34] The disease occurs in the young[6] as well as in the old, but individuals in the later years of life clearly bear an increased risk.

The problem assumes more urgent proportions when one considers an apparent rise in incidence rates in recent years.[13, 21, 27] In fact, one group concluded that this increase represented "one aspect of an epidemic of thrombotic disease at present affecting Western society."[27] Although a heightened index of suspicion and a significant improvement in diagnostic acumen may reasonably be assumed to play a part in the increasing number of diagnoses, the rise in incidence appears to be greater than that accounted for by these considerations alone. One may speculate that the changing characteristics of modern living predispose to thromboembolism. With the increase of life expectancy the so-called degenerative diseases have assumed increasing importance. Several afflictions of the elderly appear to give rise to thromboembolism, notably heart disease,[8, 36] hemiplegia,[7] carcinoma,[18, 35] emphysema,[30] varicosities,[1] and prolonged immobility, particularly after certain operative procedures.[12, 32] In any of these conditions, an apparently healthy or cured individual, or one with presumed years of useful life ahead, may succumb to a lethal embolic episode which was potentially preventable.

Faced with this growing problem, multiple therapeutic measures continue to become available for various presentations of the disease. While advances have been

Presented at the Annual Meeting of the American Surgical Association, May 11–13, 1967, Colorado Springs, Colorado.

made, ideal treatment is yet to be realized. Anticoagulants, venous interruption by surgical means, and pulmonary embolectomy are available. Although safe and effective delivery of fibrinolytic agents to the embolus appears to be the most fruitful area of therapeutic investigation, the total applicability of this technic to man is not yet determined.

With therapeutic measures available for both prophylaxis and treatment, what then is the cause of the alarming death rate? The primary reason, it seems to us, was summarized by De Bakey, in 1954: "It becomes increasingly apparent that much of the prevailing confusion on the subject of thromboembolism derives from the difficulty of establishing the diagnosis, and consequently, a firm basis for the disease." [10] The application of known ameliorative measures has been limited by the lack of a rapid, simple, and accurate diagnostic technic. We believe that the use of reflected ultrasound will help to fill this need.

Principles and Technic

The principles involved in the use of the ultrasound echo technic are relatively simple. Ultrasound frequencies greater than 20,000 cycles per second, and above the upper limit of detection by the human ear are utilized. The electronic apparatus consists of a pulse generator which drives a piezo-electric crystal to emit sound waves at various frequencies, generally from 1 to 10 million cycles per second. The crystal, housed in a cylindrical transducer, not only transmits the sound impulses, but acts as the receiver of returning echoes. Sound travels in a relative straight beam through any uniform medium with little divergence until an interface is encountered, representing the boundary between media of different acoustical impedances or densities. When there are different densities, and, therefore, different acoustic impedances between two media, as exists between air and a liquid or solid, the interface acts like a poorly silvered mirror, so that a portion of the energy is reflected back to the source of the impulse, i.e., the transducer. The proportion of the ultrasound energy which is reflected from the interface of two media is related to the magnitude of the difference in acoustical impedance, or the density, of the two media. The difference in the acoustical impedance of human tissue and air is extremely high, and results in a large mass of echoes which are returned from superficial areas of lung tissue. Second, the absorption of ultrasound by gas is relatively high compared to the absorption of most liquid or solid media. Therefore, the wave energy which is not reflected is absorbed rapidly in passage through gas-filled alveoli. Even a thin rim of normal air-filled lung tissue at the periphery would be enough to attenuate or reflect the beam, and prevent penetration of the ultrasonic wave far into the substance of the pulmonary tissue at the low gain of the instrument specifically used for this study.

Ultrasound patterns were obtained with two instruments, one of which was previously developed in our laboratory for the study of cardiac motion. In this instrument, the B-scan instrument, the transducer is a 2 megacycle/second barium titanate crystal which emits 1 microsecond pulses of ultrasound with a repetition rate of 2,000 pulses per second. The returning echoes, which are received by the transducer during the interval between pulse transmissions, are converted to electrical signals which are displayed on the photographic paper of an Electronics for Medicine recorder. Continuous recording of the returning echo is displayed as the transducer is maintained perpendicular to the chest wall and moved in a vertical or horizontal path about the thorax. This instrument permits a linear bidimensional scan of the lung. By moving the scanning transducer systematically over the thorax in

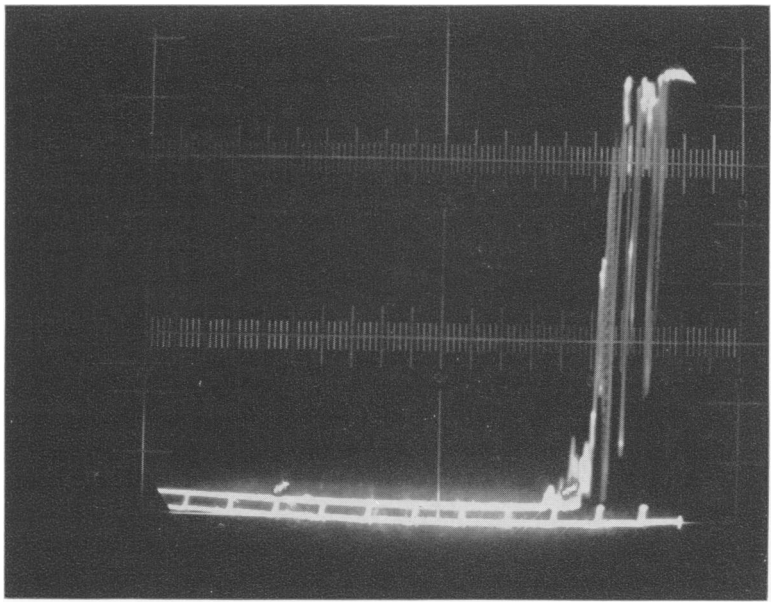

FIG. 1. Control ultrasound pattern from normal human lung. One cm. range marks can be seen at the bottom of the record. The transducer-skin interface is to the right. Decreasing echoes to the left, fading at 3–4 cm., are from the underlying chest wall and, probably, the most peripheral rim of lung tissue.

various directions and in different planes, a three-dimensional impression of the lung parenchyma can be attained, localizing the embolized area segmentally. The instrument is relatively fixed in position, because of its size.

A portable A-scan instrument is available * also and the quality of the record obtained from it is approximately equal to that of the larger scanner. At present, however, the portable instrument, which is about the size of an EKG machine, allows only unidimensional point scanning. By moving the transducer at many points along an intercostal space, the pattern can be read directly and immediately from the oscilloscope screen, but a permanent record can be obtained only by taking a Polaroid photograph of each point. Modifications can be made, however, in the portable instrument which will allow it to perform and record the continuous scan, which is somewhat more convenient and faster, although not essentially different in terms of accuracy. The inter-rib placement of the transducer is vital, since recording over the high density surface of a rib can give rise to misleading, artifactual patterns. We have found it most convenient to utilize the instrument at low gain, adjusted to give an end inspiratory pattern of decreasing echoes, fading at approximately 3–4 cm. from the face of the transducer, representing echoes from the chest wall and, probably, the most superficial surface of underlying lung. The entire examination takes 10 to 15 minutes and is no more inconvenient to the patient than an electrocardiogram.

* "Reflectoscope," Sperry Division of Automation Industries, Danbury, Connecticut.

Results

A normal pulmonary echogram from the portable machine is illustrated in Figure 1. The skin-transducer interface is to the right of the record. In contrast to the normal pattern of echoes fading at 3 to 4 cm. within the chest, areas of ischemia are distinguished by an echo pattern originating from much deeper within the chest (Fig. 2). The usual peripheral localization of ischemic zones of the lungs from pulmonary emboli, with a resultant increase in lung density, probably account for the characteristic ultrasound pattern. Either a

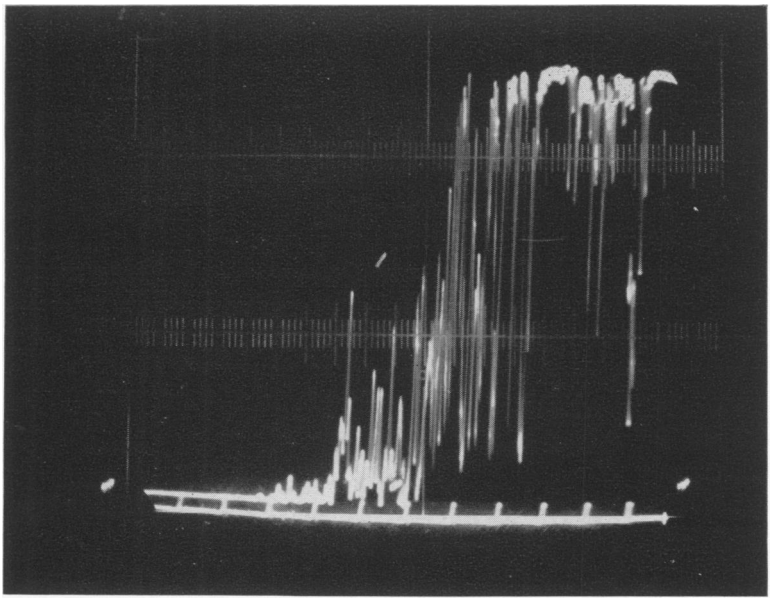

FIG. 2. A characteristic ultrasound pattern obtained over an area of embolized lung in the same patient as Fig. 1. Echoes increase markedly in prominence and depth as the transducer is placed over a portion of ischemic lung.

decrease in absorption of ultrasound by the lung and/or a loss of strong reflecting surfaces from the superficial pulmonary tissue could result in change from a normal to an abnormal pattern. A decrease in air content may be the fundamental physical event. If absorption were decreased, more ultrasonic energy would be available to penetrate deeper within the lung. Also, if strong reflecting superficial air-tissue interfaces were eliminated, less of the beam would return from the superficial tissue allowing greater penetration and echo return from deeper structures. Succinctly, with a pulmonary embolus which always produces a zone of increased focal density in the peripheral or sub-pleural area of the lung distal to it, we obtain a deep, thick band of returning echoes. This can be easily differentiated from the thin band seen from normal lung and representing echoes returning from the chest wall itself. The significance in this change of density in terms of the pathology and physiology of pulmonary embolization, so inadequately understood at present, seems worthy of further investigation.

Our studies began in dogs [23] embolized by autologous clot. The method proved 99% accurate in localizing the area of pulmonary ischemia in 21 dogs, confirmed by pulmonary arteriography and postmortem examination. On no occasion did the emboli produce an abnormality on routine chest radiographs, an invariable finding after embolization of the canine lung in the absence of pulmonary hypertension. Changes in the ultrasound pattern usually became apparent 2 minutes after clot injection and stabilize at approximately 10 minutes postembolization.

We then applied the method to patients, and thus far have studied and correlated results with other diagnostic parameters in 183 individuals. Thirty-five were normal control subjects, 63 had suspected pulmonary emboli, 28 had confirmed pleural effusion, 18 had pneumonia by clinical and radiographic criteria, and 10 had carcinoma of the lung. The remaining patients had pneumothorax, pulmonary fibrosis, tuberculosis, pulmonary hypertension, pulmonary alveolar proteinosis, calcified granuloma, nodular silicosis, previous pulmonary resection, emphysema, chronic congestive failure, lung cyst, atelectasis, as-

pergillosis (with fungus ball), or were postthoracotomy patients with characteristic postoperative pulmonary difficulties.

Thirty-five normal control patients showed a normal lung pattern virtually identical to that obtained in the laboratory animals. In like manner, the pattern obtained over areas of pulmonary embolization was in all ways similar to the abnormal patterns seen over the experimentally-induced embolus. Figure 3 is a tracing taken with the compound, or B-scan, instrument, inscribed upon the continually moving paper of an Electronics for Medicine recorder. Depth of penetration of the beam is read in the vertical axis, from top to bottom. The transducer was moving in the eighth interspace from an area of normal lung to an area of ischemic lung, and back to a normal pattern again.

Confirmation of the presence of emboli in the patients was obtained by several parameters, including I_{131}-MAA lung scanning, clinical findings, pulmonary arteriography, necropsy, and surgery (Table 1). Pulmonary arteriography was performed in two subjects and the area of pulmonary vascular obstruction corresponded to the area of lung from which an ultrasound pattern of the "embolic" type had been obtained. Absolute diagnostic confirmation was obtained in 11 patients. Areas of pulmonary embolization, which had been located from pulmonary echograms, were found at autopsy in nine patients and seen at thoracotomy in two. Thirty-six patients in the "embolus" group had I_{131}-MAA scans and positive correlations were obtained in 34. Four patients were judged too sick for the study. In one patient, the radioactive scan failed to detect an embolism which had been noted on the ultrasound scan. An infarction was confirmed at necropsy in the exact location predicted by ultrasound. The second patient thought not to have an embolus on the basis of a radio-

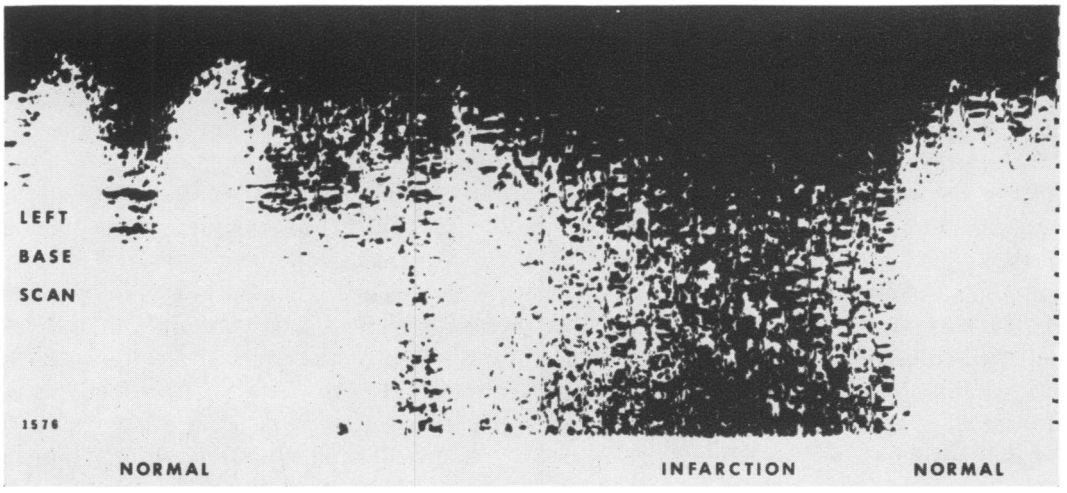

FIG. 3. Tracing taken with the compound, or B-scanner. Depth of penetration of the beam is read in the vertical axis, from top to bottom. The patient was a 54-year-old man with aortic stenosis, aortic insufficiency, and mitral insufficiency, who developed severe pleuritic left chest pain and hemoptysis while under treatment for cardiac decompensation. An I_{131}-MAA scan revealed decreased isotope concentration in the right apex with no abnormal areas seen in the left lung. The ultrasound pattern disclosed two areas of apparent pulmonary infarction in the left lower lung field. As shown above, the scanning path entered one of these areas of infarction from the left, moved across the area of infarction, and then passed into normal lung to the right of the scan. The patient had a cardiac arrest from which he could not be resuscitated. At necropsy, the right lung was normal but occlusive emboli were found associated with recent infarcts in the left lower lobe. The location of the lesions corresponded with that anticipated from the ultrasound recordings.

TABLE 1. *Correlation: 63 Patients with Ultrasound Pattern of Pulmonary Embolism*

Clinical Pattern of Embolism		Chest Roentgenogram		I 131-MAA Scan (40 Patients)		Autopsy (11 Patients)		Surgery (2 Patients)		Pulmonary Arteriography (2 Patients)	
Classical	39	Normal	21	Confirmed	34	Confirmed	9	Confirmed	2	Confirmed	2
Suspicious	24	Non-specific changes	35	Negative	2	Other Disease No embolus	2				
		Diagnostic	7	Too ill to study	4						

active scan had sickle cell anemia with classic clinical signs of infarction and a chest x-ray strongly suggestive of a small infarcted area.

The findings on routine chest x-rays, in this series, were variable and frequently were delayed for several days after the suspected time of embolization. Any abnormalities usually were ephemeral, nonspecific densities, reported as compatible with several different pulmonary disease processes, including pulmonary embolus. In only seven cases, was the radiologist firm in his diagnosis. Since the technic is still in the stage of experimental evaluation, the patients we have seen, particularly in the earlier phases of the study, have been unduly weighted in favor of classic symptomatology. They by no means reflect the usual proportion of patients with this disease who present themselves to the clinician. The group with classic symptoms—pleuritic chest pain, friction rub, hemoptysis, tachypnea, localized asthmatic wheezes, etc.—often had no definitive confirmatory examination other than ultrasound scan because of the clarity of the diagnosis.

In 10 patients with suggestive symptoms but negative, or normal ultrasound scans, confirmation was obtained by means of a normal I_{131}-MAA lung scan. In two other instances of negative ultrasound scan, however, I_{131}-MAA lung scan showed a suggestive perfusion defect in an area of lung that contained an obvious carcinoma in one, and emphysematous changes alone in the other. In three patients with congestive failure, myocardial infarction and emphysema, respectively, autopsy confirmed a negative ultrasound scan for embolus.

In our study we encountered several individuals especially worthy of note who have added to our optimism about the technic. One was a 39-year-old woman with a clinical and radiological diagnosis of right upper lobe pneumonia, who was scanned to add to our ultrasound experience with pneumonia. The echo pattern in her right upper lobe where the pneumonic process was easily diagnosed by chest x-ray was not remarkable, but surprisingly, a pattern considered classic for pulmonary embolus was seen consistently at the left base of the lung, where she had no symptoms and no abnormality on chest film. At first, this was believed to represent the first false positive ultrasound tracing. However, the following morning, the patient developed hemoptysis with pleuritic chest pain and a friction rub in the left lower lobe at the exact site of the positive ultrasound scan. Chest x-rays were again negative but a radioactive scan was consistent with pulmonary embolism. Thus the scan picked up an embolus in an area where infarction was apparently developing, but which had not reached the point where it produced recognizable symptoms or x-ray findings. In several succeeding cases, we have unquestionable evidence that infarction of lung tissue is not necessary to produce the pattern seen with the ultrasound scan. Furthermore, the scan

will pick up emboli in the absence of any findings on routine chest films. The scan may prove of particular value in a patient with a negative chest x-ray, but with suggestive symptoms of pulmonary embolism. Moreover, the ultrasound scan may aid considerably in following patients with proven embolic disease simply and safely, until complete resolution of the clot is obtained. Serial tracings have been obtained in several patients. Such a case (Fig. 4), was scanned on multiple occasions after probable pulmonary infarction until restoration of the normal pattern was obtained.

An understanding of the fundamental physical principles of the technic would indicate that a similar pattern to that of embolism should be obtained from areas of established sub-pleural consolidation from other causes, such as pneumonia or atelectasis. To date, we have studied 18 patients with pneumonia, of whom six showed a "false positive" ultrasound tracing. One of 5 patients with atelectasis also showed a pattern similar to that obtained from areas of pulmonary ischemia due to emboli. One patient with pulmonary alveolar proteinosis, and one with extensive subpleural nodules from metastatic carcinoma, proven at necropsy, also had scans suggestive of an "embolus-type" picture. In the majority of these individuals, with "embolus-type" scans, the differentiation from pulmonary infarction was not difficult because of the distinctive clinical and roentgenographic findings. It is entirely conceivable, however, that conditions such as extensive pleural thickening or lung contusion may prove confusing in the future. In this regard, scans of the area of the chest wall immediately adjacent to or over a recent thoracotomy wound may be difficult to interpret. All other pulmonary diseases studied showed a normal pattern. We were surprised to find normal scans in the majority of patients with diseases such as pneumonia or atelectasis. Theoretically, we would anticipate a normal pattern, if

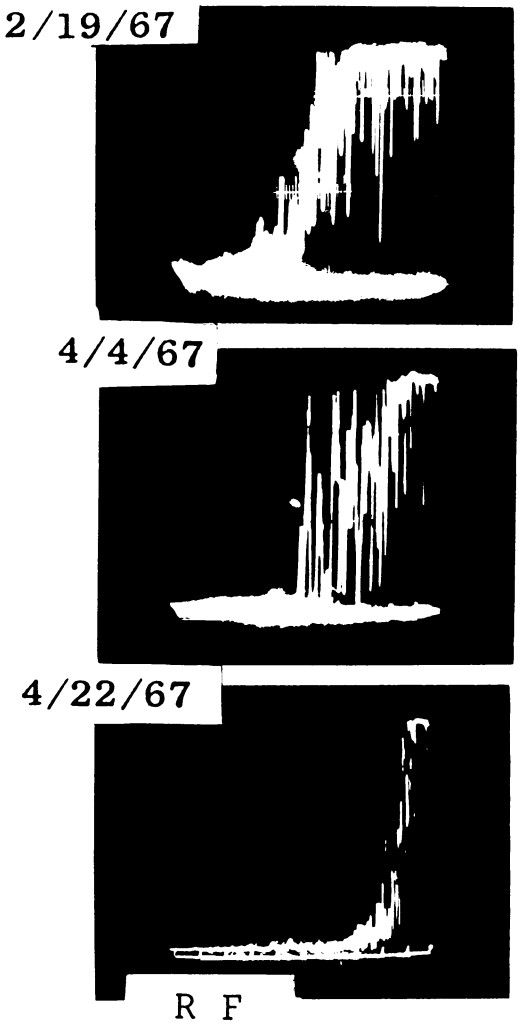

FIG. 4. Representative serial tracings taken over the same area in a patient with a proven pulmonary embolism, with probable infarction. The depth of the echoes progressively decreases until a normal pattern was restored on 4/22/67.

the consolidation did not extend to the pleura. With the low instrument gain, intentionally selected for this study, the superficial pulmonary tissue would, therefore, reflect and attenuate the sound waves before the beam reached the disease area.

Pleural fluid, which is relatively free of acoustical interfaces, is easily recognized as an echo-free zone interposed between the band of echoes from tissue interfaces of the chest wall and those returning from

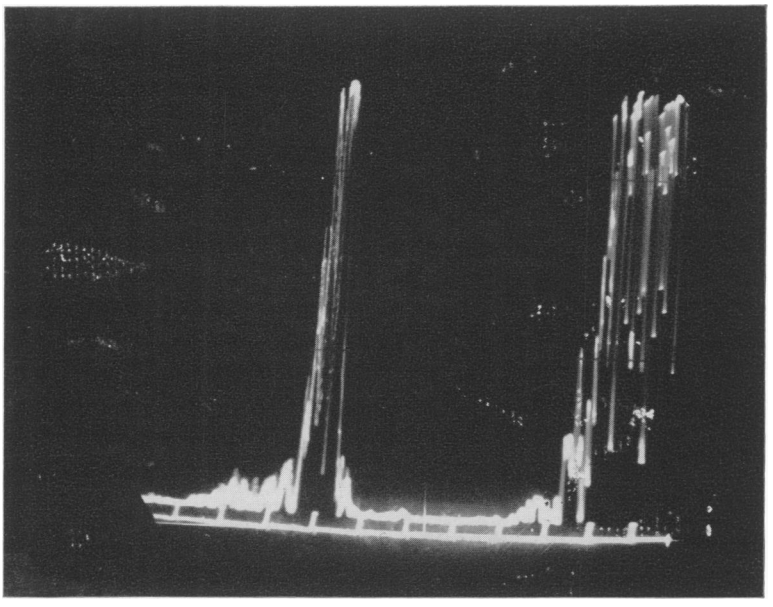

FIG. 5. Typical ultrasound pattern of pleural fluid. The width of the echo-free zone in the center of the scan is linearly related to the width of the fluid layer overlying the lung. The echoes to the left of this zone represent normal underlying lung; the echoes to the right represent chest wall reflections.

pulmonary tissue (Fig. 5.). As Joyner and coworkers [22] have indicated, this band of poor echo return is the pattern expected from relatively homogeneous and uniform fluid which does not present the multiple reflecting interfaces of tissue. The depth of the band conforms to the thickness of the fluid layer overlying the lung. It is generally accepted that 300 to 500 ml. of free pleural fluid are necessary for diagnosis on the conventional erect roentgenogram. In two of our 28 patients with pleural fluid, the chest film was negative and the ultrasound scan was confirmed by diagnostic thoracentesis. We do not yet know whether the ultrasound scan can detect areas of pulmonary ischemia under a band of fluid. However, Figure 6 illustrates the

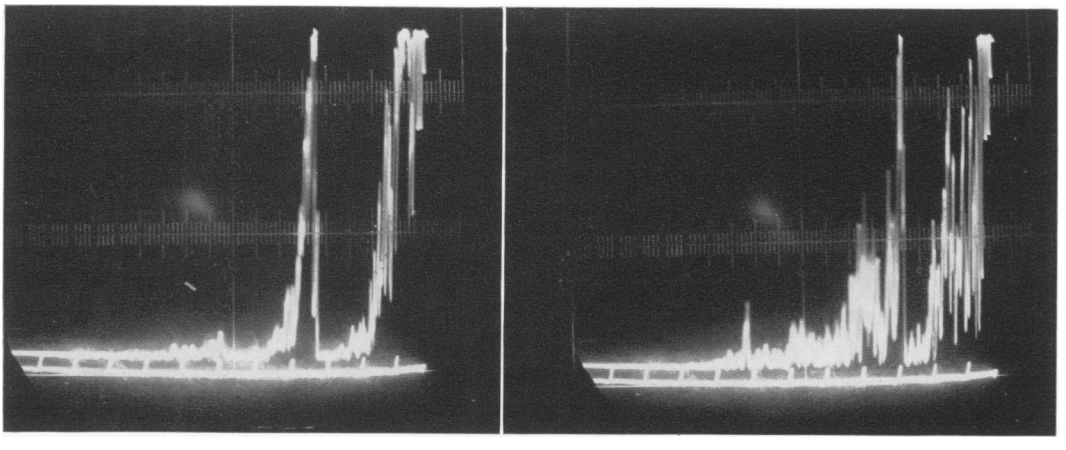

FIG. 6. Patient with fluid at both bases, but an embolus, also, at the right base. In the upper Polaroid photograph, chest wall echoes are to the right of the scan followed by an echo-free zone of fluid, and then, to the left a tall, spiking echo representing the peripheral rim of normal lung beneath. Note the increased depth of echo return from the underlying lung at the right base over the embolus. It is not clear, as yet, whether this pattern of embolus under fluid will be consistent in every patient.

pattern in a patient with fluid at both bases, but with a pulmonary embolus in the right lower lobe only. The relatively thick band of echoes returning from the underlying lung tissue in this area is seen in contradistinction to the thin band deep to the fluid on the opposite, non-embolized, side.

Discussion

It is apparent from the literature that pulmonary embolism is a ubiquitous, lethal and poorly-controlled disease. Frequently the diagnosis is not suspected from clinical findings, and is first made at autopsy. The fact that often there are no signs or symptoms to make the diagnosis of venous thrombosis suspect prior to a catastrophic, massive pulmonary embolism, has been well-documented.[2, 28] In the 606 patients with pulmonary emboli proved at autopsy, reported by Coon and Willis, a definitive diagnosis was made prior to death in only 7%.[9] Diagnostic failure was due either to a short time between the first symptoms and death, or to the obscure nature of the clinical manifestations of the disease. The so-called classic triad of chest pain, shortness of breath and hemoptysis was found in only 3% of the major embolic episodes. Hypotension, cyanosis, pleuritic chest pain, and hemoptysis occur in only a few individuals. Frequently tachypnea is the sole manifestation of a major embolic episode. True infarction or hemorrhagic necrosis of the lung, probably occur as well in a minority of patients with embolism.

A prevailing difficulty of applying known therapeutic technics is the time factor. Donaldson and co-workers reported that only 25% of the 271 patients reviewed, in whom the time factor could be ascertained, survived embolism for at least 1 hour; 22% of this entire group survived for 2 hours, and 17% lived for 6 hours or longer.[11] Therefore, one of five patients who suffer massive fatal embolism may be expected to live long enough to allow institution of a planned surgical procedure. The vagaries of the disease and its rapid progression to death combine to make effective treatment extremely difficult.

In an attempt to increase the salvage rate from both the massive and the sublethal recurrent forms of the disease, a variety of diagnostic aids have been proposed. The plain chest x-ray usually shows no abnormality.[33] Transient fluffy radiologic densities, produced by focal areas of pulmonary hemorrhage and edema occur frequently, but are generally not recorded because of lack of stability. The radiologist can rarely differentiate embolism from pneumonitis or atelectasis even in patients in whom any definite x-ray abnormality exists. Changes in the electrocardiogram have been reported[21] in the majority of cases, although they, too are rarely diagnostic. Electrocardiographic changes suggest the diagnosis by demonstrating the cardiac effects of the pulmonary vascular occlusion, primarily acute cor pulmonale. However, changes in the EKG may be seen in several primary cardiac conditions which are prominent in the differential diagnosis in an acute situation, making interpretation extremely difficult. Elevations of serum bilirubin and lactic dehydrogenase (LDH), in conjunction with a normal serum glutamic oxalacetic transaminase (SGOT) add credence to the diagnosis, but the combination does not occur frequently enough, in our experience, to make these determinations of significant value. Furthermore, in acute cases these determinations cannot be done in sufficient time to embark upon a major surgical procedure.

Pulmonary angiocardiography is often extremely helpful if larger caliber arteries are occluded.[31, 39] However, with this technic, several difficulties are apparent. It requires highly sophisticated instrumentation in the hands of specialists in radiology and cardiac catheterization, and it necessitates transporting a sick patient. It is time-con-

suming, complicated, and somewhat cumbersome for an extremely ill individual. Angiograms are highly diagnostic for major embolisms but are subject to considerable difficulty in interpretation at levels lower than the second order lobar vessels. Thus, small emboli involving the muscular arteries, approximately 1 mm. in internal diameter, are impossible to discern by this technic. The majority of emboli are not in the major pulmonic vessels [34] but are in the distal limbs of the pulmonary tree. Furthermore, interpretation of angiograms in the presence of heart failure or without recent previous chest films to rule out other pulmonary pathology, also subjects this technic to error. Angiography is normally negative in the presence of multiple small emboli and is useless to the clinician attempting to choose between long-term anti-coagulation therapy or venous interruption. Accentuation of hypotension in an already hypotensive patient, as a result of dye injection or arrhythmias, secondary to manipulation of the catheter, occasionally occurs.

Macroaggregates of human serum albumin, isotopically labelled, have been proposed [37, 38] as effective for radioactive lung scanning. The technic is safe and useful in demonstrating a perfusion defect in massive embolism, but usually requires 1 to 2 hours to perform. In addition, the "cold areas" of diminished or absent pulmonary arterial blood flow "furnishes no definitive information concerning patency of pulmonary arteries" [15] but are seen in lesions featuring consolidation, atelectasis and, in lesions that displace tissue, and in situations slowing blood flow. The left border of the heart, particularly in cardiac enlargement, obscures a significant portion of the scan in the left inferior-medial portion of the lung field making diagnosis of embolism in this area extremely difficult. Since the majority of emboli migrate to the lower lobes, obscuring the scan in this area is detrimental. We have experienced a few instances in which an obvious clinical infarct was present in the left lower lobe, only to be faced with an interpretation such as "slightly less isotope concentration seen adjacent to the left lower cardiac border. I am not sure this is large enough to be significant." Conversely, in another case, with a similar radioactive scan interpretation, both pulmonary arteriography and the ultrasound scan were negative. The presence of other pulmonary abnormalities on the chest film in the area of a perfusion defect, makes the diagnosis of embolism hazardous. In one recent study,[15] the majority of patients with cardiorespiratory illnesses *other than* pulmonary thromboembolism, had scans characterized by decreased radioactivity over regions in which the pulmonary arteries were patent. Furthermore, administration of radioactive material to a pregnant or nursing woman should be discouraged.

It is apparent that what is needed is a technic for diagnosis which has the advantage of simplicity, accuracy, speed, and minimal manipulation of the patient. Both lung scanning and pulmonary arteriography require a patient to be transported to the Radiology Department for at least 1 to 2 hours. We believe that the application of ultrasonic principles to the diagnosis of pulmonary embolism may be a significant advance. The difficulties that we have experienced have primarily been the necessity of correlating this totally new technic with older, established technics that, perhaps, may not be as accurate as ours, in the long run, particularly in the presence of small, recurrent emboli. At least in terms of projection of the ischemic area on the chest wall, we have, in a few cases, picked up areas of embolization no larger than 1 to 2 cm. in diameter. In one case this very small area conformed in exact, pin-point localization to the only spot on the chest wall where a friction rub could be heard. The diagnosis of these small areas of embolism would seem to make the

technic advantageous in large-scale epidemiologic studies of embolic disease. The diagnostic inadequacies of the technics now in common usage, as well as the inadequacies of the ultrasound scan, have been delineated above, making correlation in some of our cases difficult. Admittedly, we have not utilized angiography to the extent that it deserves. Even pathologic examination at autopsy, however, unless it is done by extremely meticulous prosectors, may easily miss small emboli in the distal pulmonary tree, where most emboli, in point of fact, actually reside. Furthermore, it has been shown by serial arteriography [14] that even larve emboli tend to disappear in 9 to 19 days and thus show few traces at necropsy. Therefore, even the pathologist, supposedly the "highest court of diagnostic appeal," may have his judgment altered by the ephemeral nature of the disease. It is hoped that, as additional clinical experience is acquired at our institution and by others who may be encouraged to apply this technic, that ultrasound lung scanning will stand the test of time, and fulfill the current urgent need for a simple, reliable adjunct to the early diagnosis of pulmonary embolism.

Summary

The advantages of this new technic, which needs further evaluation and verification, would appear to be: 1) it is simple enough to be done by an intelligent technician with a minimal amount of training; 2) the tracings can be done rapidly, in approximately 10–15 minutes, which may expand the applicability of pulmonary embolectomy; 3) it requires no manipulation or moving of the patient, since it can be done at the bedside; 4) positive diagnoses are made in the presence of completely negative chest x-rays; 5) small emboli, at least in terms of projection of underlying areas of ischemia on the chest wall, can be diagnosed, which may allow, in the future, for a fuller appreciation of the true incidence of the disease and the true significance of supposedly "minor" chest symptoms as indicators of occult emboli; 6) positive tracings can be obtained within 10 to 20 minutes of the time of embolization, judging by the animal studies; 7) by the nature of the technic, the most prominent confusing diagnoses—myocardial infarction, dissecting aneurysm, acute pericarditis with tamponade, bacteremic shock, etc.—should not be logically expected to show an ultrasound pattern similar to that of embolus; 8) it is not necessary to have necrosis of lung tissue; to obtain an "embolus pattern"; 9) the technic is perfectly safe, does not involve the injection of radioactive or opaque material and poses virtually no inconvenience to the patient; 10) it can be used as a safe, simple method of following the progression of a known embolus to its eventual resolution. The major disadvantages of the technic appear to be: 1) the underlying principle of the technic allow for "false positives" in the presence of marked increases in sub-pleural density from other pulmonary disease processes. Hopefully, in the majority of instances, these can be differentiated easily on clinical grounds alone; 2) as with any other new technic, some experience in the technical performance of the scan is necessary to eliminate artifacts in interpretation. Considering the magnitude of the problem, it is likely that continued and judicious use of this new diagnostic approach will give us greater insight into its limitations and, hopefully, into possibilities for extension of its usefulness.

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