THE VARIED CLINICAL MANIFESTATIONS OF PULMONARY EMBOLISM *

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PULMONARY embolism is a common finding at necropsy. It is nevertheless regarded in many hospitals as an unusual disorder, infrequently considered in the differential diagnosis of cardiopulmonary diseases.

The frequent failure to recognize pulmonary embolism clinically can best be explained on the basis of widely held misconceptions regarding the prevalence of this disorder and the evidence necessary for its diagnosis.

If the diagnosis is considered only when all the classic clinical and laboratory features are demonstrable, most instances of embolism will go unrecognized. Where physicians are familiar with the variety of clinical, radiologic and electrocardiographic manifestations which characterize pulmonary embolism, the frequency and importance of this disorder in modern practice become evident.

Excellent pathologic studies of embolism and infarction have been numerous,[1-3, 9] clinical investigations few.[4, 17, 20] In clinical studies, moreover, the approach has varied according to the interests of the investigators. In some, pulmonary embolism has been regarded chiefly as a cardiovascular catastrophe, and attention has been concentrated on the electrocardiographic criteria of diagnosis. In other studies, embolism has been regarded as a pulmonary disease, and attention has been confined largely to the radiologic aspects of diagnosis. No data have been reported concerning the relative frequency of the respiratory and circulatory manifestations of pulmonary embolism in general hospital practice.

As a result of growing interest in this disease at the Graduate Hospital, the diagnosis has been made with increasing frequency in recent years. During the last 18 months the entire house staff has coöperated in an effort to detect clinically instances of pulmonary embolism on all services of the hospital. The number recognized exceeded all anticipation, and provided a unique opportunity to measure the relative frequency of the various respiratory, cardiovascular, abdominal and neurologic guises which pulmonary

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embolism may assume. These observations are reported in the belief that greater familiarity with these varied manifestations and more widespread appreciation of the frequency of pulmonary embolism in modern hospital practice are essential if improved diagnosis of this important and common disease is to be achieved.

The Frequency of Pulmonary Embolism

Statistics on the occurrence of pulmonary embolism vary widely, according to whether studies are made on (1) necropsy material in hospitals, (2) necropsy material in nonhospitalized patients, or (3) on clinical material. The range of observations is shown in table 1, utilizing representative studies from well known hospitals and institutions.

The significance of emboli is often indicated by classifying them (a) as the cause of death, (b) as a contributory cause of death, and (c) as incidental. The frequency with which pulmonary embolism is detected at necropsy will be influenced by the thoroughness and interest of the pathologist, and in the routine work of an overtaxed pathologic laboratory small emboli and infarctions may be overlooked.

The degree of variation is indicated by Hampton and Castleman's observation that pulmonary embolism was detected in 9% of routine necropsies at Massachusetts General Hospital, and in 14% of a specially studied group of 400 cases.[2] The similarity of findings in the various pathologic studies included in table 1 is notable.

It will be seen in table 1 that massive embolism is observed in approximately 4% of all hospital necropsies, while contributory embolism and incidental embolism are noted in 3%. The detection of pulmonary embolism in about 10% of patients dying in general hospitals has been remarkably constant over a period of years. American studies reported in 1934,[1] 1945 [6] and 1952 [8] have each shown pulmonary embolism to be present in about 10% of cases. It should be noted, however, that Short [4] surveyed autopsy records of two English hospitals and noted a sharp rise in the frequency with which massive embolism caused death between the years 1913 to 1917 and the years 1944 to 1948. It is possible that this rise is merely the result of

Table 1

Frequency of Pulmonary Embolism Reported in Various Hospitals and Institutions

Population Studied	Method of Study	Massive Embolism, Per Cent	Other Embolism, Per Cent	Total Embolism, Per Cent
Hospital deaths[1-9]	Necropsy	1.0–7.0	3.0–5.4	6.2–14.0
Homes for indigent[10,11]	Necropsy	6.8–14.2	11.5–16.3	23.1–25.7
Postoperative patients[4,7,8,12-17]	Clinical, Necropsy	0.03–0.37	0.02–0.35	0.2–0.5
Medical patients[4,19-22]	Clinical, Necropsy	0.12–0.5	0.32–2.0	0.6–2.5

increasing age at death, with more deaths due to degenerative disease and fewer due to infection.

Two excellent studies of necropsies performed on inmates dying in homes for the indigent demonstrate an even higher occurrence of pulmonary embolism and infarction. Both in Pittsburgh [10] and in Columbus,[11] embolism or infarcts were found in 25% of necropsies, and in approximately 10% of necropsies massive embolism was the immediate cause of death. Towbin [11] has emphasized that these observations in homes for the indigent are more typical of the population at large than are observations made on hospital patients.

Clinical estimates of the frequency of pulmonary embolism are of a much lower order of magnitude. Pulmonary embolism is more dramatic in surgical patients who might but for this catastrophe have fully recovered. For this reason, and because the period of risk in surgical patients is shorter and more easily measured, the problem of thrombo-embolism has in the past been given more study on surgical than on medical services. The frequency of postoperative embolism reported in most studies, however, is low; the average incidence of massive pulmonary embolism following surgical procedures is approximately 0.18%. The reported incidence of nonfatal postoperative embolism is little higher, averaging 0.20%.

The frequency of embolism is greater among medical patients,[4, 8] but direct comparison of ratios is misleading, since the age of medical cases is greater and their stay in the hospital usually longer. Although the reported incidence varies over a wide range, the mean embolism rate is 1.1% of all medical admissions. Approximately 0.4% represent massive emboli, fatal within minutes or hours; the remaining 0.7% represent smaller emboli which are nonfatal, or are merely contributory to death.

In another important group of patients there is a significant prevalence of embolism, but no quantitative data are available. These are the ambulatory patients who, either because of underlying disease or because of trauma, often trivial, or without known predisposing factors, develop thrombo-embolism. More than a third of our patients with pulmonary embolism were of this type, and a third of our fatal cases died of thromboembolic disease with which they had entered the hospital.

Relative Frequency of Embolism and Other Pulmonary Diseases

With the decline in frequency of infection in medicine and surgery, pulmonary embolism has increased in importance as a cause of illness and death to a degree that is not generally appreciated. Short,[4] in an excellent study of the occurrence of pulmonary embolism in a general hospital in England, made the observation that pulmonary embolism was the most common acute pulmonary disease encountered between 1947 and 1950 at the Southmead Hospital in Bristol, exceeding in prevalence lobar pneumonia, bronchogenic carcinoma and idiopathic pleurisy with effusion. A review of records

TABLE 2

Frequency of Diagnosis of Certain Pulmonary Diseases
Graduate Hospital, 1942–1957

Years	Diagnosis	Annual Rate
1944–50	Pulmonary embolism	5.6
1951–52	Pulmonary embolism	12.0
1955–57	Bronchogenic carcinoma	30.0
1955–57	Pneumonia	33.3
1955–57	Pulmonary embolism	60.0

at the Graduate Hospital in 1952, however, indicated that despite special interest in the diagnosis of pulmonary embolism, pneumonia and bronchogenic carcinoma far outnumbered pulmonary embolism (table 2).

In 1955 a study was instituted at the Graduate Hospital to ascertain the value of serum transaminase determinations as an aid to the differentiation of pulmonary embolism and myocardial infarction. It was quickly evident that the frequency of pulmonary embolism was far greater than was anticipated, and a total of 90 cases was detected in the next 18 months, representing an annual rate of 60. Comparison with the cases of pneumonia and bronchogenic carcinoma admitted to Graduate Hospital during the same 18-month interval demonstrates that pulmonary embolism is indeed the most common pulmonary disorder encountered in this hospital.

The very sharp rise in frequency recorded at Graduate Hospital may in small measure reflect an actual increase in prevalence in an aging hospital population, but we believe the increase to be principally the result of better diagnosis. The fact that resident physicians and surgeons as well as the radiologic staff have become alert to the frequency of pulmonary embolism is in our opinion the major factor in the apparent rise.

PULMONARY EMBOLISM AT GRADUATE HOSPITAL

Clinical Material: The diagnosis of pulmonary embolism was made in 90 patients at the Graduate Hospital in the 18-month period between August 1, 1955, and January 31, 1957. This includes seven patients in whom the diagnosis was not suspected antemortem, and 83 patients in whom the diagnosis was made on clinical evidence. The majority of the patients were examined and diagnosed by one or both of the authors. During the period of this study there were 10,130 adult admissions to the 350-bed Graduate Hospital, comprising 5,912 surgical and 4,218 medical admissions. There is no obstetrical department. There were a total of 9,088 surgical operations, including those on children, and a total of 221 necropsies.

It is believed that through the coöperation of the entire resident staff we were notified of all patients in whom pulmonary embolism was demonstrated or suspected. Since many of the patients were private patients, and since in many instances the diagnosis was not made at the time of occurrence of the

embolic episode but was made retrospectively after all the evidence was obtained, clinical and laboratory data are not complete in all cases.

Diagnostic Criteria: The clinical diagnosis of pulmonary embolism was based on generally accepted criteria,[2, 16, 18] i.e., by finding compatible symptoms and signs and at least one of the following: (1) evidence of thrombophlebitis of the deep veins, (2) characteristic findings in the roentgenogram of the chest, and (3) characteristic electrocardiographic abnormalities. Since the diagnosis of pulmonary embolism cannot be proved by any laboratory test, but remains essentially a clinical one, it is difficult to convince the skeptical of the diagnosis. Those who demand absolute proof before the diagnosis is accepted and treatment instituted may only too often be provided with the only absolute proof, namely, the autopsy. Since a healthy amount of skepticism is not alien to the authors, the accuracy of our clinical diagnoses has been checked by analyzing the necropsy findings of patients in whom an antemortem diagnosis of pulmonary embolism had been made by the authors. There were 10 such patients. Pulmonary infarcts were found by the pathologist in seven cases. In two cases autopsied several weeks after the embolism, findings were equivocal. In one case, most typical clinically, the embolism could not be demonstrated at necropsy; the embolus may have disintegrated and liquefied, or the diagnosis may have been in error. On the whole, these findings indicate that our clinical diagnoses were soundly based.

Incidence of Embolism: The 90 cases represent an attack rate of 0.09% among the 10,130 adults admitted during this interval. The rate among admissions to the medical service was 1.2%, and among those admitted to surgical services, 0.6%.

The rate noted among medical patients is higher than that observed in the few comparable American studies (table 1), but is not so high as the rate of 2.5% reported by Short.[4] The rate of 0.6 per hundred surgical admissions is comparable to that generally observed in such studies.

Thirty-three instances of embolism occurred after surgery; four patients died of massive embolism, giving a rate of postoperative fatal embolism of 0.04%. This is a much lower incidence than that reported in most Ameri-

TABLE 3

Mortality Among 90 Patients with Pulmonary Embolism
Graduate Hospital, 1955–1957

Outcome	Number of Patients	Per Cent
Recovered	69	76.6
Died	21	23.4
Number with necropsy	17	
Embolism occluding major vessel responsible for death	9	
Embolism occluding moderate sized vessel, contributing to death	7	
Embolism, incidental to other causes of death	5	

can hospitals, and approaches the rate of 0.03% reported by Bauer [12] in Sweden. The attack rate of nonfatal embolism in postoperative patients at Graduate Hospital was 0.32%; the ratio of diagnoses of nonfatal to fatal embolism was 8:1.

Nine instances of massive embolism were encountered in the 221 necropsies performed during this period. This represents a frequency of 4.1% of autopsied cases. In addition to two postoperative deaths, two occurred in patients admitted to the hospital with phlebitis, one occurred in a patient awaiting urologic surgery, and four represented the terminal event in patients with carcinoma. Eight other patients had moderate or small infarcts. The total with evidence of embolism or infarction at necropsy was 7.7%.

Of the 90 patients with pulmonary embolism, 69 (76.6%) recovered from their illnesses and 21 (23.4%) died within the period of study. The significance of pulmonary embolism in the causation of death is indicated in table 3. When these 21 cases are analyzed in detail, it is considered clinically that pulmonary embolism was merely contributory to the deaths of seven patients, and was an incidental finding in five others. In nine instances death is attributed entirely to embolism, since no other cause of death was evident. Four were postoperative patients in whom thrombo-embolic disease was not suspected, and who were not receiving treatment for thrombo-embolism. Two patients admitted to the hospital for treatment of thrombophlebitis of the legs died within 30 minutes and six hours of admission, respectively, before effective treatment could be instituted. Another patient admitted with thrombophlebitis died within 48 hours after admission despite vigorous anticoagulant treatment. One was a patient awaiting urologic surgery, and one was convalescent from the nephrotic syndrome. The seven cases in which pulmonary embolism was classified as a contributory cause of death include three with extensive myocardial infarction complicated by pulmonary embolism, one with advanced rheumatic heart disease, one with ascending polyneuritis, one with severe hepatitis, and one with extensive body burns. The frequency with which pulmonary embolism caused or contributed to death in this series of patients again emphasizes the importance of thrombo-embolic disease and the need for further study of the diagnosis and treatment of this dangerous condition.

Factors Predisposing to Thrombo-embolism

In an effort to improve the accuracy of detection of pulmonary embolism, a detailed analysis will be presented of the syndromes, symptoms, signs, and radiographic, electrocardiographic and laboratory findings in this series of 90 cases. First, however, it will be useful to review certain epidemiologic observations in these patients. Knowledge of the presence of predisposing factors is in many instances of prime importance in leading to the suspicion of pulmonary embolism.

Pulmonary embolism was preceded by surgery in slightly more than a third of cases at Graduate Hospital (table 4). Trauma resulting in skeletal-muscular injury appeared to be a factor in six instances. Twenty per cent of patients with embolism had been hospitalized with medical illnesses. More than a third of the patients suffered pulmonary embolism outside of the hospital, and it was symptoms of embolism or those of heart failure precipitated by embolism which led to hospitalization.

Mayo Clinic studies [17] showed that postoperative embolism was somewhat more common in the presence of obesity, anemia, cardiac disease, varicose veins, previous thrombophlebitis, severe infections and carcinoma. In a third of their patients, however, none of these predisposing factors was present. Embolism was most common from the eighth to the fourteenth day following surgery, but a quarter of cases occurred in the first week and an equal number after the second week. The risk of embolism was proportional to the severity of the surgical procedure; the ratio was lowest following surgery for hernia and chronic appendicitis, and highest after gastrectomy, cholecystectomy, splenectomy and hysterectomy.

Thromboplebitis may originate at the site of contusions and fractures. A common type of traumatic phlebitis encountered in hospital practice is that due to repeated venipuncture, to prolonged insertion of polythene tubes in veins, and to the intravenous administration of many commonly used diagnostic and therapeutic agents, such as hypertonic glucose and saline solutions, bromsulfalein and mercurial diuretics.

Among the medical diseases which predispose to thrombo-embolism are heart disease, polycythemia vera, anemia and other blood dyscrasias, and carcinoma. The notable frequency of thrombosis in patients with congestive failure or paralyses is indicative of the importance of stasis. Thrombosis and embolism, especially when recurrent despite anticoagulant therapy, have been reported to be an early sign of occult visceral carcinoma. However, a recent study by Anlyan and associates [23] casts doubt on this observation, and suggests that the alleged association has been based on impressions derived from isolated cases rather than on adequate statistical observations. Although it is possible that there is a more than coincidental relationship, in our experience it has not been rewarding to institute extensive diagnostic procedures in search of malignancy solely because of thrombophlebitis.

TABLE 4

Predisposing Influences in Patients with Pulmonary Embolism
Graduate Hospital, 1955–1957

Predisposing Factor	Number of Patients	Per Cent
Surgery	33	36.7
Trauma	6	6.7
Medical illness	18	19.9
Patients admitted with pulmonary embolism	33	36.7

TABLE 5

Age Distribution of Patients with Pulmonary Embolism Compared to Adult Population Graduate Hospital, 1955–1957

Age Group	Patients with Embolism	Percentage of Total	Distribution of Sample of Hospital Population, Per Cent
21–30	3	3.3	9.5
31–40	11	12.2	16.5
41–50	13	14.5	18.4
51–60	25	27.7	18.4
61–70	22	24.5	23.9
71–80	13	14.5	10.3
81–90	3	3.3	2.9
	90	100.0	100.0

The importance of age as a factor favoring the development of thromboembolism has been repeatedly emphasized,[11, 17] and it has been noted that 85% of cases of pulmonary embolism occur in persons over the age of 40. It will be seen in table 5, however, that 74% of all Graduate Hospital admissions exceeded that age. The rise in incidence of pulmonary embolism with age has perhaps been exaggerated. In any event, pulmonary embolism is not uncommon in persons under 40, and one should be as alert to the possibility of embolism in the young as in the aged.

THE VARIETY OF CLINICAL MANIFESTATIONS

The wide variety of manifestations of pulmonary embolism is not generally appreciated. The typical clinical picture of chest pain in a patient with frank venous thrombosis occurred in but 20% of our cases. Less well known are other syndromes, with which every internist should be acquainted, for some occur with considerable frequency (table 6).

Predominantly respiratory syndromes were most common. These were encountered in 39 patients, 43.3% of the total number with pulmonary embolism. The resemblance to pneumonia is often close, both clinically and on roentgenologic study. Since pneumonia is almost invariably preceded by upper respiratory tract infection, the absence of such illness should lead to suspicion of embolism. Recurrent episodes involving one lung and then the other are relatively infrequently due to pneumonia; this pattern is characteristic of embolism.

Pulmonary embolism is frequently the cause of "dry" pleurisy, i.e., a pleural friction rub with negative chest roentgenogram. This should not be attributed to pneumonia if the chest x-ray is negative, and tuberculosis is rarely if ever manifested in this fashion. Pleurodynia due to Coxsackie virus infection also requires consideration, especially if multiple cases of chest pain are encountered.

Pleural effusion of considerable size may be produced by infarcts. The

TABLE 6

Frequency of Pulmonary, Cardiac, Abdominal and Neurologic Syndromes in Patients with Pulmonary Embolism

Graduate Hospital, 1955–1957

	Number of Patients, Per Cent
Manifestations suggestive of	
Pneumonia	18
Dry pleurisy	10
Pleurisy with effusion	7
Lung abscess	2
Tuberculosis	1
Lung cancer	1
Pulmonary syndromes	39 (43.3%)
Myocardial infarction	22
Coronary insufficiency	4
Postinfarction syndrome	3
Congestive failure	3
Cor pulmonale	1
Cardiac syndromes	33 (36.7%)
Acute surgical abdomen	2
Subdiaphragmatic abscess	2
Primary hepatic disease	2
Abdominal syndromes	6 (6.7%)
Syncope	2
Convulsions	1
Hemiplegia	1
Neurologic syndromes	4 (4.4%)
Other manifestations	8 (8.9%)

cell count and cytologic study usually show numerous red blood cells, and approximately equal numbers of granulocytes and lymphocytes. The effusion is grossly bloody in half of the cases, usually in those with congestive failure. Effusion may continue for several weeks and lead to considerable concern over the possibility of tuberculosis or neoplasm. If in such cases biopsies of the parietal pleura reveal neither tuberculosis nor neoplasm, embolism is a likely cause.

Pulmonary tuberculosis may also be simulated by infarction. One patient thought to have tuberculosis because of hemoptysis, a history of contact with a tuberculous wife, and a right subclavicular infiltration, proved to have embolism originating in an axillary vein.

Infarcts may assume a rounded form roentgenologically, and whether symptoms of chest pain and hemoptysis are present or absent, there may be serious concern over pulmonary neoplasm. Thoracotomy has not infrequently been performed because of such findings, and merely an organizing infarct found in the resected specimen.[24]

The usual pulmonary infarct is uninfected, and secondary infection is uncommon. Lung abscess occasionally develops, however, and since this

type of abscess usually extends to the pleura, empyema may result.[25, 26] Such pyogenic complications are quite rare at present, perhaps because of the widespread use of antibiotics. It is possible that embolism may have been more commonly responsible than was realized for the abscesses and empyemas of the era before chemotherapy was available.

Cardiac Syndromes: Symptoms and signs suggestive of cardiac disease were the dominant manifestation in 33 patients. The association of congestive failure and embolism is reciprocal. Heart failure and the resulting immobilization of the patient are important predisposing influences toward venous thrombosis; and pulmonary embolism is commonly responsible either for sudden precipitation of heart failure or for its persistence in spite of treatment.

Acute myocardial infarction was the disorder most commonly confused with pulmonary embolism in the patients studied at the Graduate Hospital. Again, these disorders are intimately related.

Not only may the two resemble each other, but pulmonary embolism is also not infrequently a sequel of myocardial infarction. On the other hand, severe pulmonary embolism may be complicated, as a consequence of pulmonary hypertension and systemic hypotension, by subendocardial myocardial infarction. Electrocardiographic study may not suffice to distinguish these various complications, and determination of serum transaminase levels may be especially useful in these circumstances.

Angina Pectoris and Coronary Insufficiency: These diagnoses are often mistakenly applied to instances of recurrent chest or precordial pain which occur in ambulatory patients and which prove to be due to multiple small emboli. These patients usually have mild chronic thrombophlebitis, and exhibit only minimal roentgenologic and electrocardiographic changes.

Chronic Cor Pulmonale: Patients may have recurrent showers of small emboli with slight or no pain, with eventual obstruction of enough of the pulmonary arterial bed to result in pulmonary hypertension. Owen and his associates [27] reported 12 cases of chronic cor pulmonale due to insidious and unrecognized emboli.

Neurologic Syndromes

Syncope: Transient loss of consciousness is a not infrequent accompaniment of hypotension, chest pain and dyspnea in severe embolism. Syncope occasionally occurs as the result of embolism in the absence of these other symptoms. Two patients in the present series exhibited syncope without pain, and Hussey and Katz [25] have recorded a fatal case of this type.

Cerebral Vascular Insufficiency: Hemiplegia and convulsive phenomena may result from diminished cardiac output following pulmonary embolism, especially when this occurs in elderly patients with advanced cerebral arteriosclerosis. Two patients in this study represented instances of this syndrome.

Abdominal Syndromes

Severe upper abdominal pain with marked muscle guarding simulating true rigidity may be a manifestation of pulmonary infarction, and intra-abdominal disease was suspected on admission in six patients. The abdominal symptoms presumably result from referred pain due to irritation of the lateral portion of the right leaf of the diaphragm, or to distention of the liver capsule in patients with acute congestive failure due to pulmonary embolism. A diagnosis of cholecystitis is occasionally made, and laparotomy has been performed in such circumstances; the cause for the symptoms and normal abdominal findings may not be discovered until postoperative chest x-ray examination discloses evidence of infarction.

Symptoms and Signs

Pulmonary embolism should be included among the diagnostic possibilities whenever one of the syndromes enumerated in table 5 is encountered. Following is a detailed consideration of the individual symptoms, signs and laboratory features which were encountered in the 90 patients under study. Familiarity with these manifestations should greatly increase the accuracy of recognition of pulmonary embolism and infarction.

Not all of the classic symptoms, signs or laboratory findings occur in all patients with pulmonary embolism, and no single finding constitutes a sine qua non for the diagnosis of pulmonary embolism (tables 7 and 8). Hemoptysis and pleural friction rub are the two clinical findings which are usually regarded as most characteristic of pulmonary embolism, and which many physicians still consider indispensable to the diagnosis; both were relatively uncommon in this series (28.9 and 24.4%, respectively), and in Short's series ("less than 40" and 22.6%, respectively). Far more common were nonspecific symptoms and signs such as fever (78.9%), dyspnea (46.7%), pleuritic chest pains (56.7%), pulmonary râles (63.3%), tachycardia (58.9%), and tachypnea (44.4%). Obviously many of these manifestations are, in part at least, the result of such underlying diseases as congestive heart failure, trauma and infection. Nevertheless, these figures indicate the relative frequency of the various manifestations of pulmonary embolism encountered in hospital practice.

Fever, usually low grade, was observed in 78.9% of patients, and persisted despite the use of antibiotics in many cases. A response to anticoagulant therapy was often noted. Nine patients had temperatures of 103° F. and above; five of these went on to a fatal termination.

Anginal pain, sometimes prolonged—that is, precordial pain not related to respiration, presumably reflecting underlying coronary artery disease—was described in 27.8% of patients. In most instances of anginal pain, pleuritic pains were also noted. It is important that the history of both types of pain be elicited, since the pleuritic pains which may be overshadowed

TABLE 7

Frequency of Symptoms in 90 Patients with Pulmonary Embolism
Graduate Hospital, 1955–1957

Symptom	Number with Symptom	Per Cent
Chest pain	65	72.2
Pleuritic	51	56.7
Anginal	25	27.8
Dyspnea	42	46.7
Abdominal pain	11	12.2
Syncope without pain	2	2.2

by the more severe precordial oppression may provide the lead to the correct diagnosis. The pleuritic pain of embolism, it should be noted, is often agonizingly severe, especially in younger and more sthenic patients. The pain as a rule is more severe than the pain experienced in pneumonia.

Friction rubs are often faint, of short duration, and localized to small areas. Hence frequent and careful auscultation of the chest is required to elicit this important sign. Râles are frequently described, usually localized to the area of pleuritic pain. In most instances these fine râles are actually of pleural origin, but they are often misinterpreted as indicating congestive failure or pneumonitis.

Hypotension following pulmonary embolism was encountered in a total of 25 patients. Two patients had no pain, and syncope was the dominant symptom.

Congestive heart failure was present in a smaller percentage of our patients (25.6%) than reported in many other series.[4, 20] That congestive heart failure predisposes to pulmonary embolism and, in turn, may be precipitated by or made refractory to routine measures by the superimposition of pulmonary emboli has been stressed by others.[4]

Tachycardia and tachypnea out of proportion to the patient's fever and pulmonary congestion should always suggest pulmonary embolism. However, it should be emphasized that tachycardia and tachypnea may be absent:

TABLE 8

Frequency of Signs in 90 Patients with Pulmonary Embolism
Graduate Hospital, 1955–1957

Sign	Number with Symptom	Per Cent
Fever	71	78.9
Râles	57	63.3
Tachycardia	53	58.9
Tachypnea	40	44.4
Hemoptysis	26	28.9
Hypotension	23	25.6
Congestive failure	23	25.6
Friction rub	22	24.4
Rib tenderness	16	17.8
Cyanosis	7	7.8
Jaundice	2	2.2

a normal heart rate was frequently noted when pulmonary embolism developed in fully digitalized patients.

Cyanosis was observed in only 7.7% of our patients, usually during the acute stage of massive pulmonary embolism.

Exquisite local chest tenderness proved to be a valuable diagnostic aid. This sign [29] may be elicited in pneumonia patients who have unusually severe pleuritic pain, but it occurs far more frequently with pulmonary embolism. It is apparently produced by severe spasm of the intercostal muscles secondary to the inflammation of the pleura. When due to pulmonary emboli, it has in our experience resulted from emboli with little or no roentgenologic evidence of infarction. When due to pneumonia, the consolidation is usually quite extensive and readily detected by clinical or radiologic examination.

Clinical jaundice attributed to the effects of pulmonary embolism was encountered only twice in our series. (Jaundice also occurred in five patients with primary hepatic and biliary tract disease.) A third case had subclinical jaundice with slightly elevated serum bilirubin. All of these patients had repeated large pulmonary emboli and right heart failure. The explanation for this relatively low incidence of jaundice as compared to some other series [4, 28] may be the many instances of minor embolism and the relatively few instances of severe congestive failure among our patients. It has been suggested that the hyperbilirubinemia associated with pulmonary embolism is due to lysis of the infarct, i.e., hemolysis, but in all three patients with hyperbilirubinemia the direct-reacting fraction of bilirubin was significantly elevated. Bromsulfalein retention and alkaline phosphatase activity were significantly elevated in one patient; hence hepatic dysfunction rather than hemolysis appears to be the major factor in the icterus that follows infarction.

Abdominal pains were a prominent symptom in 11 patients (12.2%), and constituted the presenting complaint in six. Laparotomy was averted in all but one instance by detection of pulmonary abnormalities by physical or radiologic examination.

Clinical evidence of phlebitis (table 9) was found in 55 patients (61.1%), involving the lower extremities in 53 and the upper extremities

TABLE 9

Relationship of Venous Thrombosis to Pulmonary Embolism
Graduate Hospital, 1955–1957

	Number of Patients
Phlebitis in lower extremities	58
Phlebitis in upper extremities	2
Phlebitis, probably in pelvis	4
Emboli probably of cardiac origin	12
Inadequate examination prior to sudden death	6
Source of emboli unknown	13
	95

TABLE 10

Principal Electrocardiographic Abnormalities in 75 Patients with Pulmonary Embolism

Graduate Hospital, 1955–1957

Abnormality	Number of Patients	Per Cent
Acute cor pulmonale	5	6.7
Coronary insufficiency pattern	18	24.0
Minor positional changes	12	16.0
T wave inversions in right precordial leads	10	13.3
Other abnormalities	8	10.7
No abnormalities	22	29.3

in two patients. Four additional patients with pulmonary embolism had undergone recent pelvic surgery, and it is probable that the source of their emboli was in the pelvic veins. Repeated careful examinations of the extremities from foot to groin and from wrist to axilla for evidence of tenderness along the course of deep veins are necessary in every patient. Thrombophlebitis is extremely common with modern use or abuse of intravenous therapy and, when mild, is often disregarded. Such forms of phlebitis, whether in the upper or lower extremities, occasionally represent an unsuspected source of emboli.[29, 30] However, in spite of meticulous examination, evidence of peripheral thrombophlebitis may not be demonstrable. In 12 of the 55 patients with thrombophlebitis signs of the latter were not present at the time of the embolism but appeared later, sometimes after a week.

The frequency of delayed phlebitis or absence of signs of phlebitis is best explained by the theory [17] that a thrombus is most likely to break loose and form an embolus when it is of recent origin and has not yet resulted in the establishment of an inflammatory process in the surrounding wall of the vein. Only the inflammatory process (phlebitis) is detectable clinically. If the entire thrombus becomes detached and none remains behind to cause an irritative inflammation, no signs of phlebitis may ever result. If part of the thrombus remains, or if another thrombus forms in the same or another vein, the findings of phlebitis may belatedly appear. The important practical implication is that it does not suffice to examine the extremities once, at the time of the suspected embolic episode, but that daily examination for at least a week is mandatory. Furthermore, the absence of signs of thrombophlebitis, while casting some doubt on the diagnosis of thromboembolic disease, by no means rules it out. It should be recognized also that the prompt diagnosis of pulmonary embolism and early institution of anticoagulant therapy will diminish the frequency of appearance of late phlebitis.

Unexplained apprehension has been said to be a frequent premonitory sign of pulmonary embolism.[32] We have encountered apprehension not infrequently following an embolism, as one would in any patient in respiratory distress or with chest pains. In only one patient of the series,

however, did unexplained apprehension appear to precede pulmonary embolism.

The charts of the seven patients in whom the diagnosis of pulmonary embolism was not made until autopsy were reviewed. No symptoms or signs of thrombo-embolic disease were recorded in four patients. In three cases suggestive evidence was described but not recognized as indicative of thrombo-embolism.

Radiologic Findings: Chest roentgenograms were obtained within the week following the embolic episode in 67 patients. In 55.2% of the cases the radiologic reader suggested pulmonary embolism among other possibilities on the basis of the x-ray findings and the clinical history provided on the routine x-ray request form. In an additional 28.4% of cases, significant radiologic findings were present but pulmonary embolism was not among the diagnoses suggested by the radiologist. It should not be inferred that pulmonary embolism and infarction result in pathognomonic x-ray densities. While certain configurations are suggestive of pulmonary embolism and infarction, these diagnoses cannot be made from roentgenographic appearances alone. Furthermore, it is important to realize that the chest roentgenogram may be entirely normal following pulmonary embolism. This is true of major emboli which do not produce infarctions, and of minor emboli which may or may not produce small infarctions at the lung periphery. Some patients may have showers of emboli for years and eventually develop

TABLE 11

Significant Electrocardiographic Abnormalities in 75 Patients with Pulmonary Embolism

Type of Abnormality	Number of Patients	Per Cent
A. Positional changes		
1. Acute cor pulmonale	5	6.7
2. Chronic cor pulmonale	2	2.7
3. Minor positional changes		
Right axis shift	12	16.0
Clockwise rotation	22	29.3
Appearance of R in AVR	18	24.0
Vertical shift	8	10.7
Appearance of S_1	8	10.7
Appearance of Q in III and/or AVF	5	6.7
B. Nonpositional abnormalities		
1. Coronary insufficiency	18	24.0
2. ST-Segment elevations		
In III and AVF	3	4.0
In right precordial leads	6	8.0
3. T-wave inversions in right precordial leads	18	24.0
4. Disturbances of rhythm and conduction		
Transient atrial fibrillation	8	10.7
Transient atrial tachycardia	1	1.3
Sinus tachycardia (in association with other findings)	25	33.3
Transient premature contractions	10	13.3
Right bundle branch block	7	9.3

TABLE 12

Association of Some Nonspecific Electrocardiographic Abnormalities with Positional Changes in Patients with Pulmonary Embolism

Graduate Hospital, 1955–57

Type of Abnormality	Number of Patients	Number with Positional Changes	Per Cent
Coronary insufficiency	18	12	67
T-wave inversions in right precordial leads	18	9	50
Sinus tachycardia	43	25	58
Atrial fibrillation	8	4	50

cor pulmonale [27] without x-ray shadows indicative of infarction. Hampton and Castleman [2] in their fundamental study described a syndrome of incomplete infarction in which hazy densities are visible radiologically for only 24 to 48 hours after embolism. On the other hand, infarction may not be visible during the first 48 hours after embolism and may be missed unless a chest roentgenogram is obtained later.

A detailed analysis of the roentgenologic findings following embolism is in preparation. It is noteworthy that in 13 instances, clinical diagnoses of embolism were not supported by the radiologist, in six cases because the roentgenogram was normal. In seven instances, abnormal densities were present which were regarded by the radiologist as indicative of some other disease. When these films were reviewed for the purposes of the present study, however, it was evident that the changes were entirely consistent with embolism.

It is also of interest that the radiologist called attention to the possibility of pulmonary embolism in 10 patients in whom this possibility had not been suspected clinically.

Electrocardiographic Findings: Seventy-five of the 90 patients in our series had at least two electrocardiograms taken, one shortly after the embolism, another taken either before the embolic episode or several days later serving as control. Significant changes were encountered in 70.7% of these patients (table 10). "*Significant* changes" does not mean pathognomonic changes, which are rarely if ever produced by pulmonary embolism, but rather transient and characteristic abnormalities, whose demonstration in a patient with an appropriate clinical picture supports the diagnosis of embolism. In table 11 are listed all encountered electrocardiographic abnormalities and their incidence. The infrequency with which the classic pattern of acute cor pulmonale was encountered—less than in any other reported series—may again be due to the fact that our series includes many cases of minor emboli which are unlikely to produce a complete acute cor pulmonale pattern. The latter, when observed in a patient with a normal control tracing, indicates acute right ventricular strain due to sudden im-

TABLE 13

Serum Glutamic Oxalacetic Transaminase Levels in 49 Patients with Pulmonary Embolism

Graduate Hospital, 1955–1957

SGO-T Level	Number of Patients	Per Cent
Consistently normal	32	65.3
Above 40 units	17	34.7

pairment of the outflow of blood from the right ventricle. This is almost invariably due to pulmonary embolism, although other pulmonary diseases may occasionally produce acute cor pulmonale.[33] The most important electrocardiographic changes produced by pulmonary embolism and reflected in the acute cor pulmonale pattern are the effects on the electrocardiographic position of the heart. Since positional changes are not ordinarily produced by myocardial infarction, there should be no confusion between acute cor pulmonale and inferior wall myocardial infarction. On the other hand, the appearance of Q waves in Lead AVF is not a reliable sign of myocardial infarction, since it has been reported by others [34] and observed again by us to occur also as part of the acute cor pulmonale pattern as the result of pulmonary embolism.

The pattern of chronic cor pulmonale was encountered in only two of our patients. However, serial tracings in both of these patients revealed transient positional changes, possibly due to recurrent emboli.

While the full picture of acute cor pulmonale was infrequently encountered, "partial" acute cor pulmonale patterns were commonly observed. These consisted of one or more of the components of the full acute cor pulmonale pattern. Smaller emboli obviously have a lesser impact on circulatory dynamics and result in lesser degrees of positional changes of the heart, reflected by one or more of the following: shift of electrical axis to the right; clockwise rotation, manifested by the appearance of S waves over the left precordial leads and R waves in Lead AVR; vertical shifts in the electrical position; the appearance of S waves in Lead I and of Q waves in Leads III and AVF. The occurrence of any of these changes alone was considered to be of some significance, but in most instances two or more of the above changes occurred together (table 12). Three patients with pulmonary embolism manifested no electrocardiographic changes other than the appearance of R waves in AVR, and one patient manifested only transient T wave inversion in Leads III and AVF. Of all the purely positional changes, clockwise rotation (29.3%), with or without R in AVR (24.0%), was encountered most frequently.

As emphasized especially by Dack and associates,[35] the electrocardiographic findings of "coronary insufficiency," manifested by ST segment depressions and T wave inversions over left ventricular leads, may appear following pulmonary embolism. This is seen especially in patients with

TABLE 14

Occurrence of Other Diseases in 17 Patients with Pulmonary Embolism and Elevated Serum Glutamic Oxalacetic Transaminase

Graduate Hospital, 1955–1957

	Number of Patients
Hepatic or biliary disease	7
Myocardial infarction	6
Musculoskeletal injuries	3
No other disease	1

underlying coronary artery disease. Twenty-four per cent of our patients manifested this pattern. What has not, however, been heretofore emphasized is the frequent association of positional changes with those of coronary insufficiency in patients with pulmonary embolism. Of 18 patients with the coronary insufficiency pattern, 12 had such positional changes. The combination is far more suggestive of pulmonary embolism than is the mere appearance of ST-T changes, differing in no way from the findings in coronary insufficiency of other causes.

Intermittent right bundle branch block, incomplete in all but one instance, was seen in seven (9.3%) of our patients. The appearance of P pulmonale waves was noted in one patient.

A more important, yet frequently misinterpreted electrocardiographic finding in pulmonary embolism is the transient inversion of T waves over the right precordial leads. This was noted in 18 (24%) of our patients. These findings may be mistakenly considered as diagnostic of anteroseptal infarction. However, the fleeting nature of these changes, the lack of true coving in many instances and the frequent association with positional changes distinguish the T wave inversions of pulmonary embolism from those of myocardial infarction, in most instances. When the differentiation cannot be made on the basis of electrocardiographic findings alone, the clinical findings and the serum GO-T level (vide infra) will usually help to make the correct diagnosis. The inversion of T waves over the right precordial leads has been studied especially by Sodi-Pallares and associates [34] and has been attributed to right ventricular "strain" and ischemia. Dack and Grishman [36] include these with positional changes as part of the acute cor pulmonale pattern.

Various tachycardias and abnormal rhythms may be precipitated by pulmonary embolism. Because of their nonspecific nature they are of comparatively minor importance in diagnosis, unless they too are associated with suggestive positional changes. The 25 patients listed in table 11 as exhibiting sinus tachycardia did have associated positional changes.

Three patients (4.0%) manifested ST segment elevations in Leads III and AVF, and six patients (8%) manifested some degree of transient ST segment elevation in right precordial "window leads." The latter observation has not, to our knowledge, previously been reported in association with

pulmonary embolism. It was brought to our attention by Dr. David Finkelstein, of the Department of Cardiology of the Graduate Hospital. ST segment elevation in electrocardiographic "window leads," i.e., leads in which an R wave is absent, usually as the result of previous myocardial infarction, and which therefore transmit intracavitary potentials, has been described in association with angina, coronary insufficiency and a positive exercise test.[37, 38] Pulmonary embolism produces these changes, probably through the same mechanism, i.e., by producing coronary insufficiency and subendocardial ischemia registered in cavity or "window leads" as ST-segment elevations.

Serum Glutamic Oxalacetic Transaminase (SGO-T) in Pulmonary Embolism

We have previously reported studies of 26 patients in whom serum glutamic oxalacetic transaminase (SGO-T) levels were determined following pulmonary embolism, and had concluded that significant elevations did not occur after uncomplicated pulmonary embolism.[39] Of the 90 patients in this series, 49 (54.4%) had a total of 126 SGO-T determinations within one to nine days of the date of the diagnosed pulmonary embolism. Of these 49 patients, 32 (65.3%) had consistently normal SGO-T levels, and 17 (34.7%) had one or more elevated SGO-T levels (above 40 units/ml.) (table 13). An analysis of these 17 cases seemed important in an effort to find the cause of the SGO-T elevations. Table 14 lists associated diseases which may have been responsible for the SGO-T elevations. It is evident that in all but one case an adequate explanation for the SGO-T elevation was present. Thus, our preliminary conclusion—that uncomplicated pulmonary embolism is not associated with significant elevations of SGO-T—appears substantiated by additional experience. This is in agreement with the experimental findings of Agress and his associates,[40] and the clinical findings of LaDue and his associates.[41] Ostrow and his co-workers [25] listed pulmonary embolism among the conditions frequently associated with elevations of the SGO-T. This is apparently the result of their failure to distinguish uncomplicated pulmonary emboli from those associated with jaundice.

In our experience, the determination of the level of SGO-T has frequently been helpful in the differential diagnosis of myocardial infarction and cases of pulmonary embolism which simulate myocardial infarction. The determination of SGO-T levels in such cases is of value, moreover, because a normal value 24 to 48 hours after the onset of pain excludes myocardial infarction more speedily than this can be accomplished with other tests.

In cases of unequivocal pulmonary embolism associated with shock, an elevation of the SGO-T level is suggestive of complicating subendocardial myocardial infarction. Three patients showed this sequence. In one who required nor-epinephrine infusions for five days to maintain blood pressure,

the electrocardiogram showed only transient ST segment depression, yet a peak SGO-T level of 406 units was obtained.

It may be superfluous to state that an elevated level of SGO-T should not be regarded as evidence against pulmonary embolism, for embolism may be present in association with myocardial, hepatic or muscular lesions which are responsible for the transaminase elevation.

Discussion

It is believed that the experience which we have reported is typical of that encountered in general hospitals. The occurrence of pulmonary embolism in 1.2% of medical admissions and 0.6% of surgical admissions corresponds quite well to observations made in some other clinical studies, and does not appear excessive when it is considered that the frequency of pulmonary embolism ranges from 10 to 25% in necropsy studies.

Our intensive effort to see all hospital patients with this diagnosis has had the effect of excluding distorted impressions due to overemphasis on cardiac disease or pulmonary disorders. It seems reasonable to consider that approximately 45% of pulmonary emboli present as pulmonary problems, and approximately 35% as cardiovascular problems; in other cases, symptoms may suggest abdominal or neurologic disorders.

We have emphasized the observation that pulmonary embolism is now the most common disease of the lungs encountered in general hospitals; appreciation of this fact should ensure serious consideration of this diagnosis in every acute cardiorespiratory illness. The possibility of pulmonary embolism must be excluded in all patients thought to have pneumonia, pleurisy, myocardial infarction, angina, syncope, and other neurologic and abdominal crises. Physicians must become familiar with the varied clinical manifestations of nonfatal pulmonary embolism if subsequent, perhaps fatal, embolism is to be averted. The diagnosis can be made with considerable accuracy if attention is given to the history and physical examination, including careful study of the extremities, and if there is informed interpretation of the electrocardiogram and chest x-ray films. Determination of serum (SGO-T) transaminase levels is helpful in the differentiation of pulmonary embolism and myocardial infarction.

It is evident from other studies [46] that nonfatal pulmonary embolism is often unrecognized. While the incidence of fatal embolism following surgery is quite similar from one hospital to another, there is variation among hospitals in the frequency with which sublethal embolism is diagnosed. In some hospitals the diagnosis of nonfatal embolism is made less often than is the diagnosis of fatal embolism; in other hospitals, where there is special interest in the detection of this disease, the frequency of nonfatal embolism is reported to be three to nine times that of fatal embolism. If in any hospital the nonfatal instances do not considerably outnumber the fatal cases it must be assumed that the hospital staff is not adequately informed concern-

ing the diagnosis of this disease. It is noteworthy that the hospitals with the highest ratio of nonfatal to fatal embolism—9:1—are those whose postoperative mortality from pulmonary embolism is lowest. This experience indicates the practical value of early diagnosis and treatment: we believe it is no coincidence that hospitals which treat many instances of nonfatal embolism encounter fewer instances of fatal embolism.

Effective therapy for this disease is available. Antiembolic therapy may be medical (heparin intravenously or subcutaneously, Dicumarol, or other coumarins or phenindiones) or surgical (vena cava ligation, bilateral femoral vein ligation). Each method has advantages and disadvantages, which will not be enumerated here, but use of any of these methods is greatly to be preferred to inaction. Observations at the Mayo Clinic [7] before the discovery of anticoagulants indicated that if a patient had a postoperative embolism and survived there was a 30.5% chance of another embolism, and an 18.3% chance of fatal embolism in the same convalescence. The 29 postoperative patients in the present study who had nonfatal embolism and were given anticoagulant therapy had no further emboli and, without exception, survived.

Unfortunately, the problem of pulmonary embolism will not be solved entirely by improved diagnosis. Most instances of massive pulmonary embolism give forewarning by minor emboli or by signs of phlebitis that can be detected by the alert clinician. Occasionally, however, fatal embolism occurs without warning and, rarely, fatal embolism occurs in a patient who is receiving what appears to be adequate therapy. Nor have prophylactic efforts been wholly successful. Neither bilateral femoral vein interruption nor administration of anticoagulants routinely to postoperative patients has proved consistently effective. Early ambulation has had many benefits, but it has not materially reduced the frequency of thrombo-embolism.[42] This may be due in some instances to the fact that early ambulation has meant merely early sitting, a posture calculated to increase rather than diminish stasis in the leg veins. Wilkins and Stanton [43] applied elastic stockings to all hospital patients in an effort to increase the linear velocity of blood flow in the deep veins by decreasing their caliber. A significant reduction in the occurrence of pulmonary embolism was achieved by this method, and it would appear worthy of wide use in hospital patients. Attention is called again, however, to the large number of patients with thrombo-embolism in whom this method is not applicable, namely, the patients who develop venous thrombosis prior to hospitalization, often as the result of trivial trauma.[44, 45] More than a third of our patients had thrombo-embolism before hospital admission.

The need for improved methods of diagnosis, prevention and treatment of pulmonary embolism is evident. An essential first step is the recognition of the prevalence of this disorder, and utilization of presently available technics for diagnosis.

Summary

1. Pulmonary embolism has become the most common disease of the lungs encountered in general hospitals. Ninety instances were recognized in an 18-month period at the Graduate Hospital, outnumbering pneumonia and bronchogenic carcinoma.

2. Embolism was preceded by surgery in 33 patients, and by musculoskeletal trauma in six instances. Embolism occurred in 18 patients hospitalized with medical illnesses. In 33 instances, embolism occurred prior to admission to the hospital.

3. Respiratory symptoms predominated in 39 instances, cardiovascular in 33 instances. Abdominal symptoms were most prominent in six patients, and central nervous system manifestations in four. The disease most often requiring differentiation from embolism was acute myocardial infarction.

4. Chest roentgenograms were important in the diagnosis of pulmonary embolism. There was no pathognomonic configuration, but as the Radiologic Department became more alert to the characteristic features of infarction, this possibility was suspected in 55.2% of patients with pulmonary embolism who were x-rayed.

5. Electrocardiographic abnormalities were detected in 70% of cases adequately examined. Typical cor pulmonale patterns were observed in seven patients, transient positional changes in 28, and coronary insufficiency patterns in 18. The high frequency of transient electrocardiographic changes observed is remarkable, since the episodes of embolism were so often of moderate or slight severity.

6. Serum glutamic oxalacetic transaminase determinations were made in 49 patients. Levels were consistently normal in 32 patients, while elevations were noted in 17 patients. In all but one of these, associated hepatic, myocardial or musculoskeletal disease accounted for the elevation. In the absence of these complicating factors, transaminase determinations are valuable in differentiation of pulmonary and myocardial infarction.

7. Pulmonary embolism will be more commonly recognized when it is appreciated as being the most common lung disease now encountered in general hospitals, when this diagnosis is given first consideration in a wide variety of syndromes, and when informed use is made of chest roentgenograms, electrocardiograms, and serum transaminase determinations.

Summario in Interlingua

Embolismo pulmonar es un condition commun que frequentemente escappa al recognition, mesmo in centros de instruction medical. Le majoritate del medicos es familiar con le classic aspectos clinic e laboratorial del condition ben que illos se incontra, de facto, infrequentemente. Es signalate le varietate de syndromes clinic que es producite per embolismo pulmonar. Illo pote manifestar se como problema thoracic, con pleuritis e effusion, hemorrhagia, abscesso, o empyema, o illo pote esser misprendite pro pneumonia postoperatori, neoplasma pulmonar, o tuberculose.

Embolismo pulmonar pote presentar se como problema cardiac, con manifestationes simile a illos de infarcimento myocardial o de angina. Illo pote sequer infarcimento myocardial e simular continuate activitate coronari. Illo pote precipitar disfallimento congestive o esser causate per illo, o il pote occurrer que illo es primo recognoscite quando chronic corde pulmonal se ha disveloppate. Embolismo pulmonar pote presentar se como problema gastroenterologic, con symptomas que simula acute abdomine chirurgic o hepatitis. Embolismo pulmonar pote apparer como un morbo neurologic, con symptomas que suggere un accidente cerebrovascular.

Le presente studio monstra que embolismo pulmonar ha devenite le plus commun morbo del pulmones incontrate in hospitales general. Novanta casos esseva recognoscite al "Graduate Hospital" intra un periodo de 18 menses, i.e. un numero excedente le numero de casos de pneumonia e de carcinoma bronchogene. Embolismo esseva precedite per chirurgia in 33 patientes e per trauma musculoskeletic in sex. Embolismo occurreva in 18 patientes hospitalisate con morbos medical. In 33 casos, embolismo occurreva ante le admission al hospital. Symptomas respiratori predominava in 39 casos, symptomas cardiovascular in 33. Symptomas abdominal esseva le plus prominente in sex patientes, manifestationes de systema nervose central in quatro. Le morbo que le plus frequentemente requireva differentiation ab embolismo esseva acute infarcimento myocardial.

Roentgenogrammas thoracic esseva importante in le diagnose de embolismo pulmonar. Le configurationes roentgenographic non esseva pathognomonic, sed in tanto que le Departimento Radiologic deveniva plus conscie del aspectos characteristic de infarcimento, iste possibilitate esseva suspicite in 55,2 pro cento del patientes con embolismo pulmonar ab qui roentgenogrammas habeva essite obtenite. Anormalitates electrocardiographic esseva detegite in 70 pro cento del casos adequatemente examinate. Typic configurationes de corde pulmonal esseva observate in septe patientes, transiente alterationes positional in 28, e configurationes de insufficientia coronari in 18. Le alte incidentia de transiente alterationes electrocardiographic es remarcabile proque le episodios de embolismo esseva multo frequentemente de moderate o leve grados de severitate. Determinationes de transaminase glutamic-oxalacetic del sero esseva effectuate in 49 patientes. Le nivellos esseva uniformemente normal in 32 casos, durante que elevationes esseva notate in 17. In omne iste 17 casos, con un exception, le elevation esseva explicabile per associate morbos hepatic, myocardial, o musculoskeletic. In le absentia de iste factores complicatori, determinationes de transaminase es de valor in le differentiation inter infarcimento pulmonar e infarcimento myocardial.

Embolismo pulmonar va esser recognoscite plus communmente si tosto que illo es appreciate como le plus commun morbo pulmonar nunc incontrate in hospitales general, si tosto que iste diagnose es prominentemente prendite in consideration in un extense varietate de syndromes, e si tosto que roentgenogrammas thoracic, electrocardiogrammas, e determinationes de transaminase seral es expertemente utilisate.

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