Pulmonary Embolism and Infarction

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Pulmonary embolic disease has not received the attention it deserves. It is our most common serious pulmonary disorder; it causes more deaths than traffic accidents, or cancer of the lung or pneumonia. It is the immediate cause of death of 2 to 6 per cent of persons coming to necropsy in general hospitals, and in 20 to 25 per cent of those who die in hospitals for the care of chronic disease. It is contributory to the cause of death in many more patients.

One of every 500 to 1000 surgical patients will die of pulmonary embolism, yet this complication causes more deaths among medical than surgical patients. Particularly susceptible to this accident are postoperative patients and those receiving post-traumatic care, those who have chronic disease, particularly heart and malignant disease, those bedridden from any cause and those who previously have had thrombo-embolic disease.

Although physicians have not been apathetic toward the problem, a concerted attack on this catastrophic disease has not been forthcoming. Such an effort should be made soon.

No doubt improved diagnosis, early ambulation, venous ligation and anticoagulation procedures have proved helpful against embolic disease, yet the frequency of occurrence of embolism continues to increase because our people live longer and we are inclined to operate upon more older people than ever before. Pulmonary embolism is rare in a patient less than 20 years old and unusual in one younger than 40 years old. The incidence in the two sexes is equal.

Every practitioner, regardless of his type of practice, should be alert to the possibility of embolic disease, aware of the insidious nature of warning signs, and ready to administer treatment which may prove lifesaving.

Sources of Emboli

In about 90 per cent of cases, the sources of pulmonary emboli are the veins of the lower extremities, pelvis and abdomen. Sometimes the veins of the head, neck and arms or the valves or wall of the heart are the sites of origin. Uncommonly, the responsible embolic material may be malignant tumor cells such as hypernephroma, emboli, fat or bone marrow mobilized by trauma (recently reported in 30 per cent of patients receiving external cardiac massage), amniotic fluid released during parturition, parasites, air introduced by the intravenous administration of medications or oil introduced during lymphangiography.

Causes of Thrombo-embolism

Venous Thrombosis—This type of thrombosis is caused by local injury, either inflammatory or traumatic, by slowing of the blood flow or by an increased clotting tendency of the blood.

Local injury to the vein may be caused by intravenous needles and medication, trauma, surgical procedures and localized
infection. Although the foregoing are frequent causes of thrombosis and thrombophlebitis, it is not so common for thrombi thus produced to shed emboli because the inflammatory reaction usually binds the associated thrombi to the vascular wall. If the formation of thrombi extends well beyond the inflamed segment, emboli may result. Most emboli originate in the larger and deeper veins; no significant number arise from superficial veins.

Slowing of venous blood flow results from immobility, rest in bed, pressure on the extremity and congestive heart failure. Stasis, rather than inflammation or increased coagulability, probably is responsible for most instances of embolism in medical patients and in those who have undergone childbirth or operation or have sustained injury.

Increased coagulability of the blood would seem to be a logical cause of thrombosis, but the direct relationship of this factor is difficult to measure by available blood tests. Increased coagulability may well be a factor in the postoperative genesis of thrombophlebitis and in the development of polycythemia vera or dehydration with hemoconcentration.

In many cases it may be best, perhaps, to consider thrombophlebitis and pulmonary embolism as idiopathic until the true cause is known. It is well known that thrombophlebitis—and therefore pulmonary embolism—is often associated with infectious disease, malignant disease, heart disease, blood dyscrasia, pregnancy, ischemia of an extremity and various other conditions, but the reason for this association is generally obscure.

*Intracardiac Thrombosis*—Thrombosis within the heart is caused by slowing of the blood flow (auricular fibrillation, congestive heart failure or eddies in a dilated chamber), or by damage to the endocardium (myocardial infarction or bacterial endocarditis). Yet, even in patients with heart disease, embolism results more often from venous than from intracardiac thrombosis.

*Pulmonary-Artery Thrombosis*—Primary thrombosis of the pulmonary artery is rare, but it does occur.

**Pathologic Aspects**

Emboli in the pulmonary arteries will proceed as far as their size permits. In about half of the cases in which death ensues, the bifurcation of the main pulmonary artery is seen to contain large coiled emboli (Fig. 1); in a fourth both the right and left pulmonary arteries are obstructed. Among the rest, the occlusion involves a main pulmonary or lobar artery or multiple smaller vessels.
The problem of micro-embolization is now receiving more attention than formerly.

Large emboli often are present in the right atrium and ventricle; when they are, they may seriously alter hemodynamics. In the presence of right ventricular failure, right atrial pressure may become greater than left. Right-to-left shunt then can take place through a patent foramen ovale, previously held closed by the greater left atrial pressure; and paradoxic embolism to the systemic circulation may result.

To cause death, obstruction of more than half—perhaps 85 per cent—of the pulmonary arterial circulation appears necessary. Occlusion of a single major branch seldom causes death in persons younger than 50 years. Therefore, minor embolism may not be dangerous unless it forewarns of more serious trouble to follow. But factors in addition to occlusion of the pulmonary artery appear to be necessary. For example, death does not occur when a main pulmonary artery is occluded during a surgical operation, whereas it may if the vessel is obstructed by emboli. The cause of death in these instances is thought by some to be pulmonocoronary reflex, and by others to be the consequence of released serotonin which produces pulmonary vasoconstriction. The entire problem of reactive vasoconstriction requires clarification.

The occurrence of embolism without infarction brings about little change in tissues of the lungs themselves. It does, however, cause dilatation of the pulmonary arteries, enlargement of the right ventricle and auricle, and the changes of congestive heart failure. Death from pulmonary embolism without infarction is common; nearly always it is associated with embolization of the large vessels of the lung.

Pulmonary infarction is present in only 50 to 60 per cent of cases of pulmonary embolism. Because of the double blood supply to pulmonary tissue (meaning the bronchial artery, as well as the pulmonary artery) and anastomosis within the pulmonary circulation itself, infarction is not likely to occur unless circulatory disease of the...
heart or lung compromises the bronchial, as well as the pulmonary circulation.

Infarcts of the lung are circumscribed, dark-red, consolidated lesions, frequently multiple (Fig. 2). Microscopic examination shows the capillaries to be congested and the alveoli to be packed with blood cells. Hemorrhage is caused by congestion or necrosis of vessels, and the site may be the bronchial or pulmonary arteries. Infrequently, necrosis with cavitation is seen, but necrosis seldom is as marked in pulmonary infarction as it is when infarction of other tissues develops. As a rule, the infarct gradually resolves and heals with fibrosis, leaving little recognizable residual change.

Infarction may be incomplete and reversible. Areas of consolidation may develop, but may resolve before breakdown of tissue occurs, leaving little or no permanent residual damage.

**Physiologic Considerations**

Acute pulmonary embolism results in three major disturbances of gaseous exchange: (1) arterial oxygen desaturation, which commonly is associated with embolism but uncommonly with the disorders often confused with embolism; (2) hyperventilation; and (3) differences in the carbon dioxide composition of arterial blood and end-expiration air. Normally, carbon dioxide pressure is the same in the arterial blood as it is in alveolar air. When pulmonary embolism involves a significant extent of lung, however, the carbon dioxide pressure of alveolar air (determined in end-expiration air) may be somewhat less than that of the arterial blood. Use of this observation as a diagnostic test has been advocated by Robin and others. According to Robin, the test applies only to instances of massive pulmonary embolism, and should be used as a screening rather than as a diagnostic test. Only major differences between the carbon dioxide pressure of arterial blood and end-tidal air are indicative of pulmonary embolism. If these differences are small, it can be concluded that surgical embolectomy is not indicated. It is well to bear in mind the fact that conditions other than pulmonary embolism will cause differences in the carbon dioxide pressure of arterial blood and end-tidal air.

When acute obstruction of a major portion of the pulmonary arterial system occurs, pulmonary hypertension develops in the pulmonary artery proximal to the site of obstruction and in the right ventricle. This leads to right ventricular dilatation and failure. Reduced cardiac output in turn leads to systemic hypotension.

Little information is available as to the ultimate effect of pulmonary embolic disease on the function of the lungs. It is not often that comparative studies of function, before and after embolization, are obtained, and when they are, they may be distorted by the concomitant presence of associated disease of the lungs or heart. It is well known that serious permanent impairment, with or without pulmonary hypertension, may develop, but the frequency of occurrence and the degree of damage to the lung necessary to cause it are not well known.

**Clinical Considerations**

Less than half the instances of fatal pulmonary embolism proved at necropsy are recognized before death. This is a humiliating statistic.

**Thrombophlebitis**—On many occasions, recognition of thrombophlebitis is the clue to the diagnosis of pulmonary embolism. Unfortunately, when the source of the pulmonary embolus is a vein of the leg, thrombophlebitis can be detected in only half of the cases, and in perhaps half of these it does not appear until after the embolism has developed.

Low-grade fever, especially after surgical operation, is often the only symptom. Pain and tenderness of the leg muscles, tenderness and induration of the vein, swelling of the leg and foot (unilateral or unequally bilateral) and the presence of Homans' sign (not diagnostic) are to be looked for.

Multiple episodes of thrombophlebitis can occur, but apparently each episode is
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self-limited; whether chronic thrombophlebitis in fact exists is doubtful. When venous thrombosis develops, the clot is organized and as a rule is adherent to the wall of the vein within eight to ten days; occasionally this span is longer. Therefore, the time during which emboli may be shed from an attack of thrombophlebitis probably is limited. Thrombophlebitis and phlebothrombosis probably are different phases of the same entity, and there is nothing to be gained by distinguishing between them.

Embolism Without Infarction—When rest in bed is prolonged, pulmonary embolism may occur at any time, even after several weeks of rest. Embolism which follows injury or operation develops during the second week in about half these cases; in a fourth of the cases, embolism materializes before that week and in another fourth it arises after that week. It is unusual for embolism to occur before the fourth day, but it can happen.

Often embolism produces no symptoms or signs whatever. The first indication of difficulty may accompany exertion, such as getting out of bed, straining or change of position. The patient may experience apprehension, and dyspnea, cyanosis, tachycardia, cough, fever, syncope or convulsions may develop. When the main trunk of the pulmonary artery is obstructed or primary branches are similarly closed, 20 per cent of the patients experience substernal or precordial pain which may extend to the shoulders and neck, as in myocardial infarction. When these large emboli cause an increase of pulmonary arterial pressure and acute cor pulmonale, the patient may have severe dyspnea (hyperventilation) cyanosis, circulatory collapse with a decrease in blood pressure, pallor, sweating, tachycardia, gallop rhythm or arrhythmias and weakness of heart sounds. The pulmonic second sound may be louder than the aortic second sound. Venous pressure increases and the jugular veins become distended. When right-heart failure develops, the liver soon becomes enlarged, tender and painful, and edema is noted. Rarely, pericardial friction rub is heard over the distended pulmonary artery; it may be confused with the pericardial rub of myocardial infarction.

Of patients with massive fatal pulmonary embolism, nearly half die within 15 minutes and two-thirds die within two hours, allowing little opportunity for lifesaving treatment. Often they die without any warning. It is clear that prevention will be the most effective weapon against this catastrophe.

Infarction—Infarction occurs in only half of the instances of pulmonary embolism recognized clinically. Infarction itself causes cough, hemoptysis (in less than 25 per cent of cases), pleuritic pain (in less than 50 per cent), pleural friction sound (in less than 25 per cent), elevation of the diaphragm, and—over the region of the infarction—suppression of breath sounds and impairment of the percussion note. Involvement of the diaphragmatic pleura produces abdominal pain and rigidity which may suggest an acute abdominal condition. Occasionally, icterus is associated with pulmonary infarction, further complicating the pattern. Icterus is rare in cardiac patients who do not have pulmonary infarction. The development of bloody pleural fluid in small or large amounts is to be expected with infarction. It is unfortunate that little more than half of the patients with infarction will display some of these helpful diagnostic clinical signs or symptoms.

Laboratory Aids to Diagnosis

Roentgenology—The roentgenologic examination of patients with pulmonary embolism and infarction may be difficult because so often they are bedfast and unable to cooperate. In most patients with pulmonary embolism and in some with infarction, no roentgenographic signs at all will be found, and at best the changes seen merely suggest rather than confirm the diagnosis.

The signs produced by embolism without infarction usually are those caused by
Pulmonary hypertension: enlargement of a major pulmonary artery or its larger branches (Fig. 3), and enlargement of the right ventricle and its outflow tract. The sign of Westermark's oligemia or decreased vascularity of the affected portions of lung—is infrequently seen and is difficult to recognize. Not often, but occasionally, it is possible to see the shadow of the embolus itself within the hilar or larger pulmonary vessels. Rarely, evidence of pulmonary edema, congestion or pleural effusion resulting from congestive failure can be discerned.

Pulmonary infarction generally produces more definite roentgenologic signs, as a rule after a delay of a few hours or days. The involved segment of infarcted lung always extends to the peripheral or interlobar pleural surface, and the broadest diameter usually is at the pleural surface. The appearance may suggest obstructive pneumonitis or atelectasis, and linear shadows of plate-like atelectasis are common. At a given time, the density may resemble a tumor (Fig. 4a and b). Sometimes necrosis and cavitation occur (Fig. 5a and b).

Incomplete infarction is a well-established and common lesion which results in congestion, but does not progress to necrosis. It is reversible and, therefore, clears without residual damage. It can be seen in the roentgenogram if the plate is taken
at the proper time, but rapid and complete clearing is to be expected.

Pleural effusion is a common accompaniment of pulmonary infarction, and when the amount of fluid is large, the process can be seen clearly in the roentgenogram. When the quantity of fluid is small, examination with the patient in the lateral decubitus position may be necessary to demonstrate it.

Ventilation of the lung may be restricted; if so, it will cause the diaphragm to remain in a high position and will limit its excursion and produce areas of plate-like atelectasis in the base of the adjacent lung (Fig. 3).

One of the characteristics of pulmonary embolic disease is the changing nature of the lesions. Serial roentgenograms made at frequent intervals are very helpful in arriving at the diagnosis and in charting the progress of the disease (Fig. 4a and b).

The most nearly definitive diagnostic method thus far available is pulmonary angiography (Fig. 6), and it is being used with increasing frequency, efficiency and safety. Because of certain dangers, the procedure should not be used indiscriminately, but it is very helpful in selected patients, particularly those under consideration for pulmonary embolectomy. It must also be remembered that conditions other than embolism, such as focal emphysema, may cause confusing vascular defects in the angiogram. Angiography not only helps to confirm the diagnosis of pulmonary embolism, but also defines the extent and location of the emboli, important information if operation is to be performed.

After maximal healing of infarction has taken place—a matter of days or weeks—some residual roentgenographic abnormalities may remain. These are areas of fibrosis or linear shadows, pleural thickening, obliteration of the costophrenic angle or an irregular or flattened diaphragm.

Radioisotope Scanning—Recently Wagner and associates have developed a practical method of determining areas of relative avascularity of the lung by means of radioisotope scanning. They use a new radiopharmaceutical preparation, macroaggregated human albumin labeled with radiodine (I\(^{131}\)) or preferably radioactive chromium (Cr\(^{51}\)), which is injected intravenously.

The particle size of the albumin is from 10 to 50 micra, large enough to cause transient micro-embolization of the pulmonary vessels, permitting visualization by radioisotope scanning technics. Failure of a region of the lung to concentrate the radio-

![Figure 5a: Massive infarction of the left lung. b. Necrosis of infarct with cavitation.](image)
active material indicates reduced or absent blood flow to that area (Fig. 7), but it does not indicate the cause, which may be conditions other than embolism. The dose of the material is small and the danger from radioactivity is negligible. There have been almost no reactions to the material, according to Wagner and co-workers. The information obtained is not diagnostic of embolism, does not localize the embolus and is not a substitute for angiography prior to embolectomy, but it is a helpful screening procedure.

Studies of Blood and Pleural Fluid—Leukocytosis and increase of the rate of sedimentation of erythrocytes are the only hematologic abnormalities noted consistently in the presence of pulmonary embolism. Occasionally, the value for serum bilirubin is increased.

The rate for serum glutamic-oxaloacetic transaminase (SGOT) remains normal—a feature which is helpful in distinguishing pulmonary embolism from myocardial infarction. Wacker and associates have described a triad for diagnosis of pulmonary embolism and infarction: serial measurements of serum lactic dehydrogenase in which values increase during the first four days, elevated values for serum bilirubin and normal values for SGOT. This combination strongly favors the diagnosis of pulmonary embolism and infarction, and helps to distinguish them from myocardial infarction, according to these authors. This method is receiving increasing attention and may well provide corroborative information helpful to the diagnosis, if the limitations of the test are kept in mind. An elevated value for serum lactic dehydrogenase in the presence of a normal rate for SGOT probably is significant, even without an increase in serum bilirubin.

Pleural fluid, ordinarily bloody and having the characteristics of an exudate, is a usual development of pulmonary infarction. Pleural fluid resulting from congestive failure, however, is likely to be clear and to have the characteristics of a transudate.

Electrocardiography—Pulmonary infarction produces no electrocardiographic change. Some electrocardiographic abnormalities may be found in two-thirds of the instances of embolism, but tracings which show characteristic changes of definite help in the diagnosis are exceptions. They are obtained in perhaps 5 to 10 per cent of cases, and are caused by acute right ventricular stress. Limb leads show sinus tachycardia, a constant S wave in Lead I, a frequent Q wave in Lead III, inversion of
T₃, flattening or slight inversion of T₂ and rather low voltage. Occasionally, P₂ is tall and sharp. In chest leads the T wave is inverted frequently in V₁ to V₅, sometimes in V₄, and occasionally in V₅; and the RS pattern is brought around as far as V₆ or V₂. Transient right bundle-branch block occurs in about 15 per cent of cases. In the presence of pulmonary embolism, the electrocardiogram may suggest infarction of the posterior wall of the myocardium, rather than acute cor pulmonale. The electrocardiographic changes can be expected to return to normal within days or weeks; the manner in which they evolve and regress are important in the diagnosis. The findings may be confused by pre-existing electrocardiographic changes caused by heart disease, so common among patients in whom pulmonary embolism develops. Comparison of a current electrocardiogram with previous electrocardiograms may therefore be of great value. A normal electrocardiographic pattern is more consistent with pulmonary embolism than with myocardial infarction.

With due regard to its limitations, the electrocardiogram may prove helpful in the diagnosis because it is readily available and because it may show changes early in the course of the disease, before other conclusive signs develop.

**Differential Diagnosis**

Few disturbances can be so tragic or deceive the experienced physician so often as pulmonary embolism. It must be distinguished from myocardial infarction, pneumonia, atelectasis, pleurisy from whatever cause, abscess of the lung, chronic pulmonary infection, cancer of the lung and pleura, subphrenic abscess, acute surgical abdomen, syncope, hemorrhage with shock, cerebrovascular accident, convulsive disorder, disease of the liver with icterus, dissecting aneurysm of the aorta and other conditions.

More attention is being given to the problem of subclinical embolization of the smaller pulmonary vessels, so-called "microembolization." Lodged in sufficient degree, these emboli can cause obstruction of the pulmonary circulation, leading to secondary pulmonary hypertension, the only manifestation of which may be unexplained dyspnea. The condition of patients thus afflicted may be diagnosed erroneously as emphysema, heart disease or psychoneurosis. Extensive investigation, perhaps including cardiac catheterization, may be necessary for arrival at the correct diagnosis. The possibility that pulmonary embolism may be present should be kept in mind when patients are seen who have dyspnea of obscure cause.

Associated thrombophlebitis with a history of previous thrombophlebitis or pulmonary embolism lends strong support for the diagnosis of pulmonary embolism.

Although the diagnosis of pulmonary embolism and infarction often can be made with reasonable certainty by the use of all diagnostic aids, thus far absolute confirmation depends upon examination of tissue removed at operation or necropsy. It is hoped that better diagnostic methods will be forthcoming soon.

**Figure 7:** Same case as in Fig. 3: radioisotope scanning pattern shows decreased radioactivity over right lung caused by reduced blood flow produced by embolism of right pulmonary artery.
CAUSE OF DEATH, MORTALITY RATES AND PROGNOSIS

Death from embolism usually is the result of circulatory collapse brought about by right ventricular strain and failure. Generally, the terminal results of massive embolism are ventricular fibrillation and acute cardiac arrest. Asphyxia sometimes is a secondary cause of death, but is rarely the primary cause. Coronary insufficiency caused by lowered blood pressure is believed by some to be a major contributory cause of death.

It is difficult to determine the mortality rate produced by all cases of pulmonary embolism because in a large number the condition is mild and hence is not recognized. Among patients with clinically recognized embolism the mortality rate is perhaps between 10 and 30 per cent if anticoagulant therapy is not employed. The prognosis becomes markedly improved when effective anticoagulant therapy can be administered. It is extremely grave for patients who have acute cor pulmonale and in whom hypotension develops.

In a series reported by Barker and Priestley, 75 per cent of the patients who survived an attack of pulmonary embolism or infarction experienced subsequent episodes of embolism (fatal in 19 per cent). Of those patients who had had clinical thrombophlebitis without embolism, 11 per cent had subsequent thrombophlebitis and 16 per cent had subsequent pulmonary embolism (fatal in 6 per cent). Patients with a history of previous thrombophlebitis or pulmonary embolism are in a hazardous situation because of the frequent occurrence of fatal pulmonary embolism in this category.

TREATMENT OF THROMBO-EMBOLISM

Prevention—Prevention of embolism is directed toward the prevention of thrombophlebitis. The patient's legs should be elevated and pressure on the veins of the legs should be avoided. Furthermore, such veins should not be used as sites for the intravenous injection of medication and fluids.

Early ambulation or at least early leg motion (not just early sitting) should be encouraged. Elastic bandages should be applied to the legs, and—in selected cases—anticoagulant therapy should be instituted. These measures are distinctly helpful, but unfortunately are not completely successful.

In the postoperative period, prophylactic anticoagulant therapy is indicated for those who have had thrombophlebitis or pulmonary embolism and for older patients who have heart disease or trauma to the extremities or who are obese. When the diagnosis of pulmonary embolism or thrombophlebitis has been made, or even when it is strongly suspected, active anticoagulant therapy should be started at once.

Ligation of veins is not done now as often as formerly. It is now used only if pulmonary embolism recurs despite adequate anticoagulant therapy or if the use of anticoagulants is contraindicated, as it is for potential victims of bleeding. Ligation now is employed less frequently than it used to be because anticoagulant therapy is so effective. In most cases, if ligation is to be effective, the inferior vena cava should be ligated, and the venous insufficiency thereafter can be very distressing to the patient. To avoid this disturbance, methods of filtering the blood in the inferior vena cava without obstructing its flow have been devised, such as the plication method of Spencer and co-workers 8 and the vena caval filter method of Deweese and Hunter. 9 Inferior vena caval and selected femoral ligations have a definite role and should be employed when they are indicated.

Active Therapy.—As soon as the diagnosis of thrombophlebitis or pulmonary embolism has been made or even strongly suspected, anticoagulant therapy should begin. It should be continued until the patient is symptom-free and is fully ambulatory. Generally, such results require ten days to three weeks. Should there be a recurrence, long-term anticoagulant therapy is indicated. Hemoptysis arising from pulmonary embolism does not contraindicate the use of anticoagulants.
When the decision for anticoagulant therapy is made at the onset, 50 mg. of heparin is administered every four hours. At the same time, use of one of the coumarin compounds (200 to 300 mg. ofbishydroxycoumarin [Dicumarol], or 35 to 40 mg. of warfarin sodium). When the prothrombin time reaches the desired therapeutic value, usually after 24 to 48 hours, heparin therapy is stopped, but coumarin therapy is continued with carefully controlled dosage. Adequate anticoagulant therapy will save a large majority of those patients who otherwise would die from pulmonary embolism.

_Treatment of Cor Pulmonale._—During the acute phase of cor pulmonale, therapy should be considered urgent. For the relief of pain and apprehension, morphine or meperidine (Demerol hydrochloride) is effective and is urgently needed. Oxygen relieves hypoxia; it can be given by mask or tent. Shock may be treated by the intravenous administration of medicaments such as phenylephrine (Neo-Synephrine hydrochloride), 50 to 100 mg., in a 5 per cent solution of dextrose in water, or levaterenol bitartrate (Levophed bitartrate), 4 ml. of a 0.2 per cent solution diluted to 1000 ml. in 5 per cent dextrose in water. Aminophylline, 250 to 500 mg. administered in 500 ml. of a 5 per cent solution of dextrose in water, helps to relieve dyspnea. Rapid digitalization is indicated if heart failure is present.

The intravenous administration of papaverine, with or without atropine, was highly recommended in the past, but is now considered of unproved value.

Pulmonary embolectomy currently is receiving more attention, and a few successful operations have been reported. This procedure should be considered only for patients who have massive embolism likely to cause death unless it is relieved. Very few patients will exhibit the necessary indications for embolectomy. Pulmonary embolectomy of course should be performed only in centers equipped for thoracic surgery, including cardiopulmonary by-pass procedures. With few exceptions, when embolectomy is indicated, ligation or filtration of the inferior vena cava also should be performed.

Improvements are being made in thrombolytic enzyme therapy, and ultimately this procedure may prove a boon to the treatment of pulmonary embolism. As yet, however, the incidence of unfavorable reactions, the cost, the difficulty in securing adequate laboratory control and the lack of proved effectiveness greatly limit the usefulness of thrombolytic enzyme therapy.

Because it is frequently impossible to make a definite diagnosis, it sometimes becomes necessary to recommend that a patient with a pulmonary complication be treated both with antibiotic agents for possible pneumonitis or subphrenic abscess and with anticoagulant agents for suspected embolism. The risk of properly controlled anticoagulant therapy is small and the benefit derived from the treatment of embolism is great. We should not hesitate to treat a patient when the diagnosis of pulmonary embolism is merely suspected.

**SUMMARY**

Pulmonary embolic disease is the most serious disturbance of the lungs, often striking without warning and causing death within a few minutes when least expected. The clinician should consider the possibility that pulmonary embolism is present in many instances of complications referable to the lungs, particularly among patients more than 40 years of age, patients who have sustained injury or undergone an operation, those bedridden from any cause, those who have chronic disease, especially heart disease and malignant conditions, but particularly among those with thrombophlebitis or a history of it and those who previously have had pulmonary embolism.

Even after using every means at his disposal, the clinician still may have difficulty in making the diagnosis correctly, and the over-all accuracy of diagnosis in cases in which the outcome ultimately proves fatal is perhaps no better than 50 per cent, an arresting fact.
Death may occur early, but treatment may often prove lifesaving. Therefore, early accurate diagnosis and early treatment are imperative. New methods of diagnosis and treatment present greater hope for the future.

RESUMÉ

La maladie embolique pulmonaire est la pneumopathie la plus sérieuse, frappant souvent sans avertissement, et causant la mort en quelques minutes lorsqu'elle est le moins attendue. Le clinicien devrait envisager la possibilité d'embolie pulmonaire dans de nombreux cas de complications attribuables aux poumons, en particulier chez les malades ayant plus de quarante ans, les malades qui ont eu un accident ou subi une opération, ceux qui sont confinés au lit pour une cause quelconque, ceux qui ont une maladie chronique, essentiellement cardiaque ou maligne, mais surtout chez ceux qui ont une thrombo-phlébite ou des antécédents de thrombo-phlébites, et ceux qui ont antérieurement eu des embolies pulmonaires.

Même après avoir utilisé tous les moyens à sa disposition, le clinicien peut encore avoir des difficultés en faisant le diagnostic correctement, et l'exactitude globale du diagnostic dans ces cas où l'évolution ultime est fatale, n'est sans doute pas supérieure à cinquante pour cent, un fait notable.

La mort peut survenir précocement, mais le traitement peut souvent sauver la vie du malade. C'est pourquoi un diagnostic précis et exact, et un traitement précoce, sont indispensables. Les nouvelles méthodes de diagnostic et de traitement font naître un grand espoir pour le futur.

ZUSAMMENFASSUNG

Die Erkrankung an einer Lungenembolie ist die wohl ernsthafteste Störung der Lungen, oft schlagartig Auftreten ohne Vorwarnung und innerhalb weniger Minuten zum Tode führend, wenn er am wenigsten erwartet wird. Der Kliniker muß die Möglichkeit in Erwägung ziehen, daß eine Lungenembolie in vielen Fällen vorliegt, bei denen auf die Lunge bezügliche Komplikationen vorkommen, besonders bei Patienten von mehr als 40 Jahren, Patienten, die ein Trauma erlitten haben oder eine Operation durchgemacht haben, Ferner solche, die aus irgendwelchen anderen Gründen bettlägerig sind, solche die chronische Erkrankungen haben, insbesondere Herzerkrankungen und bösartige Krankheitsformen, aber besonders solche mit einer Thrombophlebitis oder einer Vorgeschichte davon und solche, die schon zuvor einmal eine Lungenembolie gehabt haben.

Selbst nach dem Einsatz jedes ihm zur Verfügung stehenden Mittels wird der Kliniker Schwierigkeiten haben, eine korrekte Diagnose zu stellen und die Genauigkeit der Diagnose bei Fällen, bei denen sich der schleichende Verlauf als tödlich herausstellt, ist vielleicht höher als 50%-eine einrucksvolle Tatsache.

Der Tod kann früh eintreten, jedoch kann die Behandlung sich oft als lebensrettend erweisen. Daher muß man eine akkurate Diagnostik und eine Frühbehandlung fordern. Neue Methoden zur Diagnostik und Behandlung bieten eine größere Hoffnung für die Zukunft.

REFERENCES


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