PULMONARY EMBOLISM Small pulmonary emboli can occur with few symptoms and with no abnormal physical signs, and may be followed by larger fatal emboli in hours or days. The electrocardiogram usually is helpful in diagnosing large emboli. Early roentgenologic signs of pulmonary emboli, seen in 30–50 per cent of patients, include enlargement of the pulmonary arteries, abrupt termination of a pulmonary artery or branch, and diminished vascularity of the lung fields. Later roentgenologic signs are diaphragmatic elevation, decreased aeration, or infarction. Pulmonary angiography is the most reliable way of localizing emboli, but can be dangerous and deceptive and occasionally fails to localize small peripheral emboli. The lung scan can be affected by almost all pulmonary diseases, severe cardiac diseases, and some upper abdominal disorders. Individual lung scans *per se* are never diagnostic of pulmonary embolism. In a patient with a normal chest roentgenogram and an abnormal lung scan with several areas of diminished perfusion, clinical suspicion of a pulmonary embolus makes this diagnosis almost certain. If the chest roentgenogram suggests embolism, the lung scan usually shows the involved areas are larger than suspected. If both roentgenogram and lung scan are normal, pulmonary embolism is highly improbable. Repeated lung scanning can demonstrate the efficacy of anticoagulant or thrombolytic therapy or the occurrence of new "silent" emboli. Perfusion defects have disappeared in eight days, usually resolve in three to four weeks, and have persisted for seven months. In patients thought to have pulmonary embolism, the lung scan supported the diagnosis in 50 per cent, excluded it in 22 per cent, was not helpful in 18 per cent and suggested the presence of pulmonary hypertension in 10 per cent. (*Secker-Walker, R. H.: Scintillation Scanning of Lungs in Diagnosis of Pulmonary Embolism, Brit. Med. J. 1: 206 (April) 1968.*)

PULMONARY EMBOLISM Necropsy studies show that pulmonary embolism is much more frequent than suspected clinically in hospitalized patients but that it is usually not lethal. Massive thromboemboli released to the pulmonary circulation of anesthetized rabbits caused immediate death in only 15 per cent. Similar emboli released 30 minutes after intravenous injection of 5 μg/kg epinephrine were associated with a mortality rate of 60 per cent. After epinephrine, circulating platelet concentration fell markedly and degranulated platelets were found aggregated in increased amounts on the emboli. Pretreatment with heparin or methysergide, a serotonin antagonist, prevented this increase in mortality, suggesting that serotonin release from the aggregated platelets caused death by small-airway obstruction. The epinephrine presumably enhances platelet aggregation to the thrombi by increasing the amount of thrombin coating the fibrin of the emboli. This might explain the high mortality associated with emboli in subjects who have high catecholamine levels, such as those with myocardial infarction. (*Thomas, D. P., and others: Epinephrine Potentiation of Platelet Aggregation: Its Effect on Death from Experimental Pulmonary Embolism, J. Lab. Clin. Med. 71: 955 (June) 1968.*)

FAT EMBOLISM Arterial hypoxia, unrecognized and severe, regularly accompanies fat embolism. Prompt recognition of the hypoxia by measurement of the arterial blood gases and its correction by aggressive oxygen therapy is the most effective treatment for fat embolism. The disturbance may be the result of a diffusion barrier for oxygen caused by destruction of normal lung surfactant activity. Diffusion of carbon dioxide appears to be unaffected. (*Wertzberger, J. J., and Peltier, L. F.: Fat Embolism: The Importance of Arterial Hypoxia, Surgery 63: 626 (April) 1968.*)

AIR EMBOLISM The minimum LD_{100} for right atrial oxygen injection was established as 7.1 ml/kg in dogs. Right atrial injection of .27 to .4 mg/kg atropine 10 to 20 seconds after the oxygen protected five of six dogs from fatal embolism. Antifoam A used in a similar manner protected only one of six animals. (*Crockett, A. T. K., and others: Pathophysiology of Aeroembolism Following Intravenous Injection of Oxygen, Aerospace Med. 39: 407 (April) 1968.*)