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When the lungs are involved as part of a systemic disease the following are noteworthy: (1) a symmetrical distribution of lesions in both organs; (2) a vascular or perivascular pattern of abnormal lung markings, and (3) a tendency toward pleural implication. The first and second elements are related to the fact that the pulmonary involvement in most systemic diseases is either within or along blood vessels and/or lymphatics. The third is due to the fact that the pleura is richly supplied with blood vessels and lymphatics. Furthermore, the blood vessels supplying the pleura as well as the stroma of the lungs in man are the bronchial arteries, tributaries of the aorta and therefore of the systemic circulation.

The pulmonary changes giving rise to abnormal roentgenologic patterns are a composite of (1) "normal" bronchovascular markings which vary with the age, sex, occupation and body build of the individual; also the conditions under which the roentgen examination is made; (2) physiologic and pathologic changes within the air-containing and/or fluid-containing units of the lungs which largely determine the radiotranslucency of the affected parts, and (3) specific architectural alterations in the way of fibrosis, emphysema, consolidation or necrosis as well as associated reaction in pleura, lymph nodes and adjacent structures. In toto, the various elements mentioned endow the lights and shadows in the roentgenogram with certain qualitative and quantitative characteristics which immediately bring to mind certain possibilities to be considered in differential diagnosis.

A disease may manifest itself in the lungs in diverse manner and dissimilar diseases in comparable manner. The pulmonary changes may assume the configuration of diffuse fibrosis, small or large air cysts, miliary or nodular infiltrations, patchy or massive consolidations with or without an accompanying pleural effusion; more often there is a combination of the aforementioned. Yet these several patterns may be the result of a single or a limited number of agents. On the other hand, diffuse interstitial fibrosis may be the end stage of viral pneumonia, allergic pneumonia, sarcoidosis, scleroderma, chronic pulmonary congestion and other diseases. Cystic changes may be associated with pituitary disease, lipoid storage disease, tuberous sclerosis and other diverse conditions. At least fifty abnormal states may give rise to miliary or nodular foci. It would take us too far afield to enumerate the diseases which may give rise to pulmonary consolidation or pleural effusion. The totality of the picture depends on the type of tissue involved, the duration of the disease and the intensity of the pathological process. As the disease progresses, or becomes chronic and recrudescent, the causative agent plays an increasingly lesser role as far as determining the end result.

There are times when one has the impression that the morphological changes in the lungs, as depicted in the chest x-ray, are in keeping with the presence of a systemic disease, yet one may not be able to demonstrate organ or tissue involvement outside of the chest. This state of affairs is exemplified in instances of diffuse interstitual fibrosis, cystic lung and other obscure conditions currently being met with increasing frequency as larger segments of the population are examined roentgenologically. It may be speculated that in some the pulmonary changes represent the end stage of a disease at one time widepsread, and in others the lungs are momentarily the shock organs of a

systemic disease, the nature of which is obscure. We have learned by analogy that sarcoidosis was originally described as a dermatologic disorder; in time it was recognized as a systemic disease and is now often found in the lungs and regional lymph nodes without demonstrable involvement of other organs. Lupus erythematosus is going through a similar cycle and other diseases will probably follow suit.

For the moment we are concerned with the lung as a mirror of systemic disease, a subject which takes in a sizeable segment of internal medicine. Some of the conditions to be discussed are infrequent; several are rare. But it is well to bear in mind that a rarity once recognized often becomes commonplace. Furthermore, for every patient with a rare or bizarre condition discovered in specialty practice, many times the number pass through the offices of general practitioners and hospital wards with their disease either unrecognized or misdiagnosed. With the development of new techniques in diagnosis and treatment, diseases and syndromes are constantly being discovered, more often rediscovered, which have been with us for centuries and new ones are being created.

In the following pages a somewhat heterogeneous collection of diseases is discussed. They are presented in a sequence arranged primarily for orientation purposes, not as a classification in the generally accepted sense of the term. It is often impossible to draw sharp nosological distinctions between one group of diseases and another, especially so between diseases of the skin and allergic diseases or between the latter and disturbances of metabolism and certain blood dyscrasias. With advances in knowledge, there are constant regroupings of diseases. In fact, several of the conditions to be described cannot be fitted at present into any classification because they are too fluid to be pigeonholed into specific compartments. The discussion will be limited to a definition of terms and concise descriptions of the morphological and roentgenological findings. Other features will receive passing attention insofar as they pertain to the major thesis under consideration.

Diseases of Metabolism

1. Hypertrophic Pulmonary Osteoarthropathy (Acropachy)
2. Diabetes and Pulmonary Infections
3. Lipoid Storage Disease of the Lungs
4. Honeycomb Lungs
5. Azotemic Lungs
6. Cystic Fibrosis of the Pancreas and Pulmonary Infections
7. Amyloidosis of the Lungs
8. Metastatic Calcifications of the Lungs

Metabolic diseases may affect the lungs through structural damage of the organs and by providing conditions favoring infection. In several disorders the latter constitutes an almost inevitable accompaniment of the disease. In view of the profound derangements which take place in the vascular and internal secretory organs as well as in the reticuloendothelial system, the one chiefly concerned, it may be pertinent to dilate on several phases of the problem which have an indirect although important bearing on the major topic of the discussion. I refer specifically to the first two items listed.

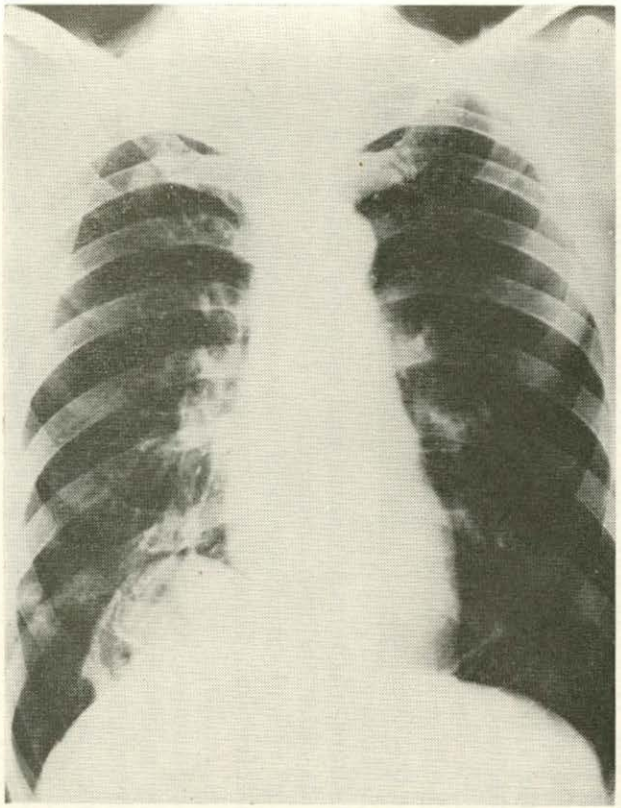
Hypertrophic Pulmonary Osteoarthropathy (Acropachy):

Clubbing of the fingers and toes, associated with variable degrees of non-pitting edema and a productive periostitis chiefly of the long bones of the extremities, are often encountered in patients with pulmonary abscess, bronchiectasis, hemangiomas and other developmental defects. It is generally agreed that ditigal clubbing is due to hypertrophy and hyperplasia of tissues caused by increased blood flow and nutrition of the affected parts. From the time Marie first described the condition in 1890, physicians have speculated on the possible relationship between the diseases mentioned and the development of clubbing. The fact that the clubbing may recede with healing of the pulmonary disease has been most intriguing.

Ray and Fisher noted that osteoarthropathy is much more often associated with peripherally situated malignant pulmonary neoplasms than with main bronchial tumors. My own observations, based on a review of the material at hand, are in keeping. The presence of unexplained osteoarthropathy calls for a careful roentgenologic survey of the patient in various planes to make certain that a neoplasm is not present behind the heart or is hidden in some other recess of the thoracic cage. Not only chronic pulmonary infections but also cirrhosis of the liver, ulcerative colitis, congenital heart disease and a number of other diseases may give rise to digital clubbing. The condition may be unilateral; on rare occasion, hereditary.

Possible causes mentioned in the development of pulmonary osteoarthropathy include anoxic, infectious, neuritic, toxic and endocrine factors. There is evidence that in some cases hypertrophic pulmonary osteoarthropathy is related to endocrine imbalance. Reason for this belief is furnished by a number of reported instances of osteoarthropathy occurring in patients with thyroid disease, a condition in which the pituitary gland plays an important role. Thomas and others have reported cases of osteoarthropathy occurring after subtotal thyroidectomy for exophthalmic goiter. Signs of osteoarthropathy appeared coincidentally with symptoms of postoperative hypothyroidism. After the institution of thyroid treatment there was noted a thinning of the subperiosteal bone as well as clinical improvement. Fried reported four cases of bronchiogenic carcinoma associated with advanced degrees of pulmonary osteoarthropathy. He found, in addition, signs of acromegaly as attested by the characteristic facies, tufting of the terminal phalanges of the fingers and toes, hirsutism, macroglassia and thickening of the cranial vault. In a case reported by Bloom, metastatic lesions were found in the pituitary from a primary carcinoma of the lung. There was hypertrophic pulmonary osteoarthropathy associated with acromegalic features. In line with these clinical observations it is noteworthy that recent experiments in rats have shown a close relationship between the pituitary and the development of tumors. In spite of many studies, the mechanism involved in the development of hypertrophic pulmonary osteoarthropathy appears to be too complex to be resolved by a single formula. The subject of clubbed fingers, which has engaged the attention of physicians from times immemorial, will probably continue to provoke erudite discussions for many years to come (Fig. 1).

Diabetes and Pulmonary Infections: Diabetes favors infection of the lungs as it does of the skin, kidneys and other parts of the body. The reason for the susceptibility of the diabetic to infection is not clear. For a time it was



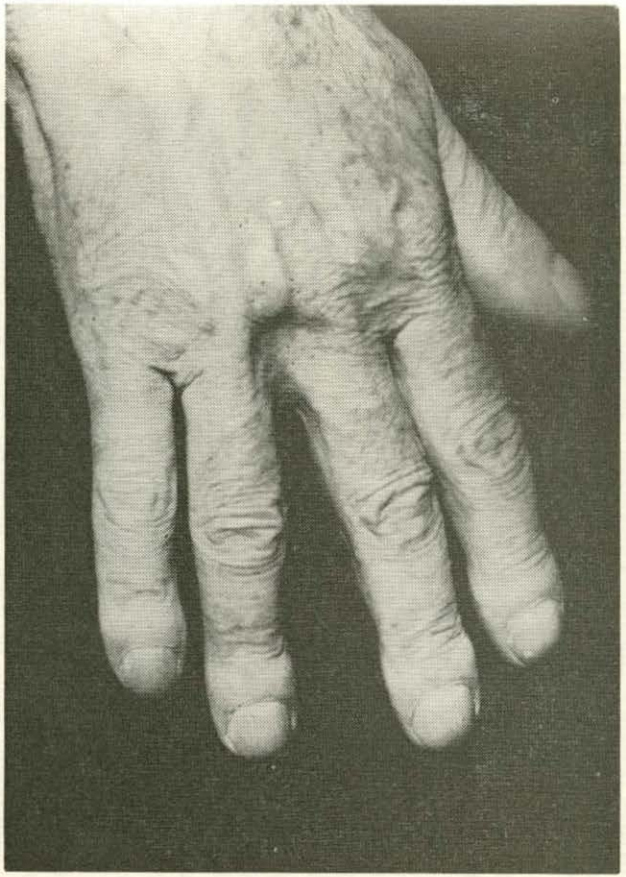


Figure 1.—Hypertrophic pulmonary osteoarthropathy in association with peripheral bronchiogenic carcinoma in a man of 62. A. Round, well circumscribed density in right lower lobe. (Lateral chest x-ray revealed it to lie posteriorly adjacent to the spine. At operation peripheral tumor found, histologically epidermoid carcinoma.) B. Clubbed fingers of same patient.

believed that the increased sugar content of the blood provides a medium favoring the growth of microorganisms. As is well known, the addition of glucose to culture media enhances the growth of the tubercle bacillus. Hyperglycemia, however, does not explain matters since pyogenic organisms do not grow better in vitro in a blood sugar medium than in a blood medium alone. But, if the blood is obtained from a patient in diabetic coma, certain bacteria do grow better. More weight is ascribed to faulty metabolism of the tissues. Additional factors which may favor infection in the diabetic are vitamin A and C deficiencies, pancreatic disease, endocrine disturbances and lowered resistance of the tissues resulting from vascular damage. In the uncontrolled diabetic, dehydration, acidosis, malnutrition, poor mouth hygiene and a greater tendency to aspiration pneumonia are important considerations.

Pulmonary infection in a diabetic is apt to be acute and massive, the involved tissues undergoing necrosis and suppuration in a relatively short time. In a diabetic tuberculosis is apt to be especially severe and of a caseocavitary type. Although the diabetic is usually of middle or advanced age, the tuberculosis is of a type more often seen in the young Negro and Puerto Rican. The disease has a predilection for the mid-and basal portions of the lungs, so-called "diabetic phthisis." The appearance of the roentgenogram may be sufficiently impressive to prompt the physician to examine the patient for a latent diabetes. Although much of the dread associated with pulmonary infections has been eliminated since the introduction of insulin and anti-microbial medication, the occurrence of a suppurative broncho-pheumonia, lung abscess fungous infection or tuberculosis remains a serious complication (Fig. 2).

Lipoid Storage Disease of the Lungs: This condition is characterized by the presence of large, pale, "foam" cells, usually containing lipid and involving chiefly the reticuloendothelial system. Depending on the specific lipid present in the cells, several forms or phases of the disease are recognized. (*1*) Gaucher's disease, in which the lipid is cerebroside, is characterized by enlargement of the spleen, skin pigmentation, hemorrhages and involvement of long bones, especially of the femur which often sustains spontaneous fracture. (*2*) Niemann-Pick disease, in which the lipid is phosphatide, is featured by enlargement of the liver and spleen, ascites, anemia, leukopenia, mental changes as well as bone involvement. (*3*) Hand-Christian-Schuller's disease, in which the lipid is cholesterol and its esters, is characterized by a bizarre triad consisting of expohthalmos, diabetes insipidus and map-like defects of bones. The condition affects older children and adults. (*4*) Letterer-Siwe's disease is a nonlipid form of reticuloendotheliosis of unknown cause. Another form appears to follow infection. The disease occurs exclusively in infants and young children. It is featured by hepatosplenomegaly, hemorrhagic tendencies, localized skeletal tumors, anemia, splenic enlargement and lymphadenopathy. (*5*) Eosinophilic granuloma of bone is a related condition characterized by solitary bone defects, often of the membranous bones of the skull. It has been found in association with the aforementioned.

Hodgson and co-workers found the average duration of active disease in 26 cases of reticuloendotheliosis to be slightly more than two years. The longest duration of active disease was six years. They noted that the prognosis was directly related to age; the younger the child, the worse the outlook. Following the introduction of antibiotics, the prognosis has somewhat im-

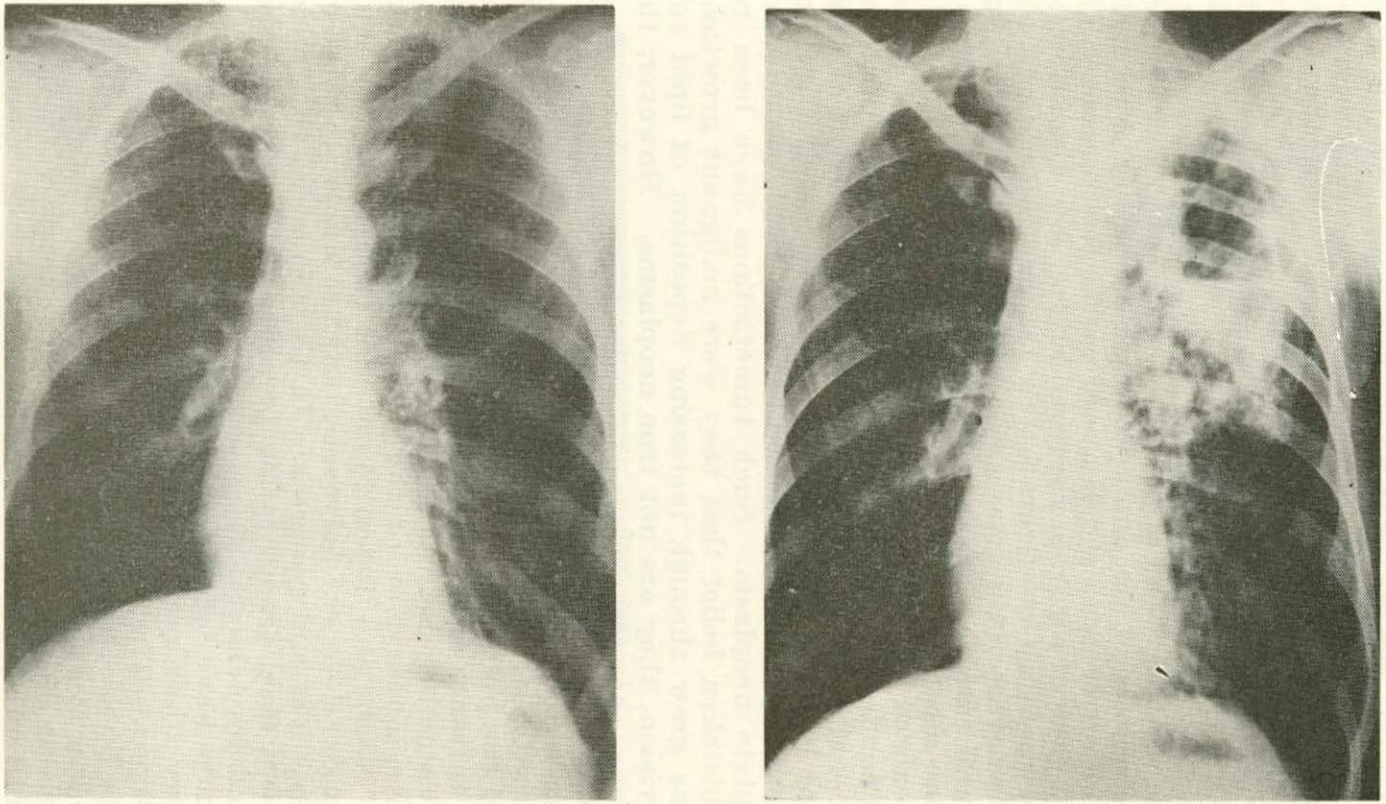


Figure 2.—Caseocavernous tuberculosis in a diabetic man of 63. A. Minimal infiltrations in apices; increased left hilar shadow and regional accentuation of bronchovascular markings. B. Six months later, large cavity showing fluid level occupying major portion of left upper lobe; scattered infiltrations in lower lobe. (Sputum contained acid fast bacilli).

proved. Bierman and co-workers reported ameliorative effects of antibiotics in identical twins, aged 9 months, with Letterer-Siwe's disease. The administration of a combination of antibiotics resulted in considerable clinical improvement with disappearance of hepatosplenomegaly, lymphadeopathy, regression of anemia and roentgenographic evidence of healing. Residual disease was still in evidence so that the final results could not be determined.

Lipid storage disease rarely involves the lungs without associated lesions in the skeleton. One type of pulmonary involvement consists of miliary or nodular disseminations, evenly distributed in both organs representing minute lipid deposits in the alveoli. The appearance of the film may simulate that seen in miliary tuberculosis of pneumonoconiosis. Another type is featured by innumerable small air cysts in a delicate or coarse reticulation. The cysts are formed by the escape of air from ruptured alveoli into the interstitial tissues. This type of roentgen configuration is the one most likely to arouse one's suspicion of the nature of the disease. As a result of the frequent presence of blebs on the pleural surface, spontaneous pneumothorax is common.

Occasionally one meets with diffuse fibrosis of the lungs of a delicate or coarse consistency caused by organization of lipid deposits in the parenchyma and proliferation of connective tissue. In such, the disease is apt to terminate in chronic cor pulmonale, as was recently observed in a patient at Montefiore Hospital. A similar instance was reported by Chester and Kugel from the same institution some years ago. Circumscribed lipid granulomas (xanthomas) may simulate neoplasm. Such tumefactions have been removed surgically in the mistaken belief that they were malignant growths. Inasmuch as xanthomas grow through increasing deposition of lipid substance rather than cell division, they are not true neoplasms. However, they may undergo sarcomatous changes (Fig. 3).

Honeycomb Lungs: The lungs may be the seat of numerous air cysts. The cystic changes may be part of a systemic disease or the condition may be limited to the respiratory tract. Unless the evidence is clear, it may be difficult to determine if the pulmonary disease is acquired or is due to faulty development. In an instance of the latter, seen by the writer, there were found at autopsy multiple small cysts in both lungs associated with a patent ductus arteriosus, congenital aneurysms of the pulmonary arteries and anomalous coronary arteries. Norris and Tyson found that the lesions and sequence of anatomic changes in development polycystic lung are similar to those observed in polycystic kidney, liver and pancreas. In their opinion, the fundamental lesion appears to be a focal segmentation preceded or followed by dilatation of small bronchi and bronchioles.

Honeycomb lungs have been found in association with hepatic disorders of infancy, pituitary disorders (diabetes insipidus), tuberous sclerosis and several other conditions. The most frequent systemic disease associated with honeycomb lungs is lipoid storage disease. In fact, Chester and Kugel, in their review of the literature on lipoidgranulomatosis (Hand-Christian-Schüller's disease) comprising 50 reported cases, detail so many variegated facets of this intriguing condition that it is not unlikely that most noninflammatory diffuse cystic lungs are of granulomatous origin of the same order. This impression finds support in the lucid descriptions of honeycomb lungs by Parkinson and his co-workers. In the acute phases of lipoid storage disease the na-

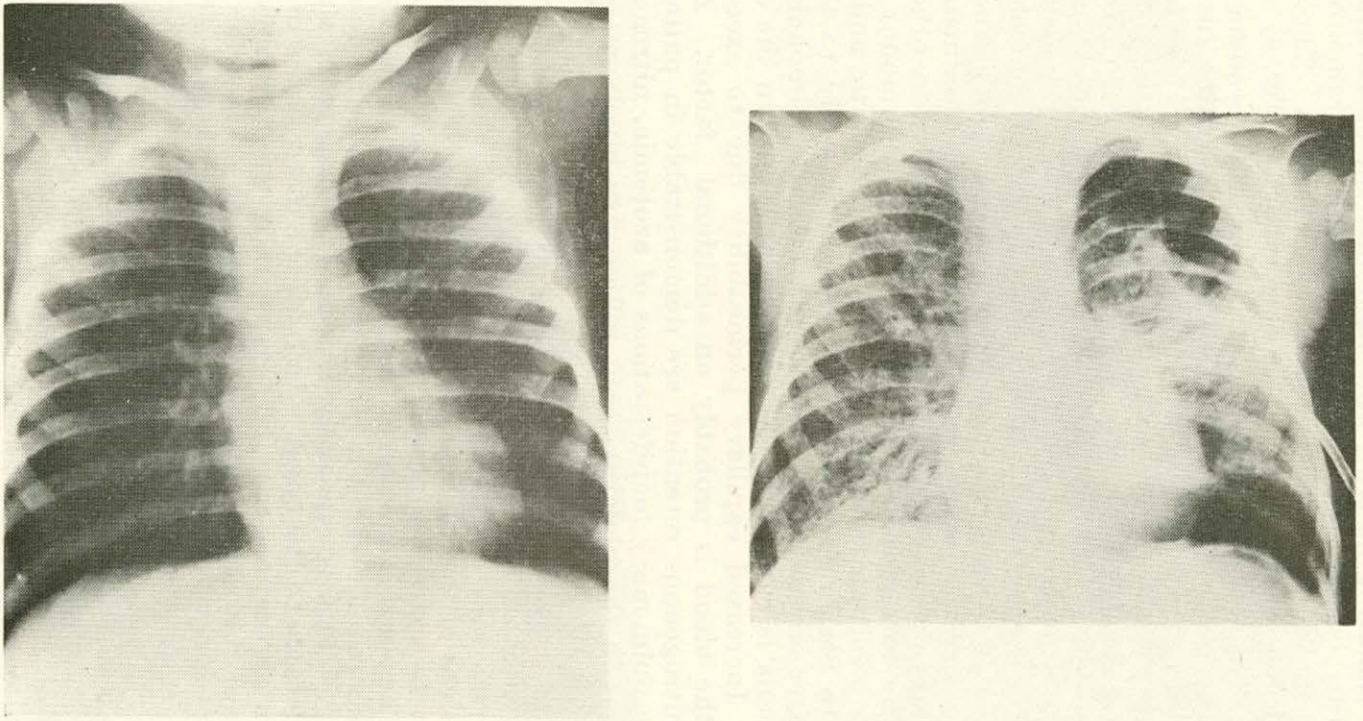


Figure 3.—Lipoid storage disease of the lungs in a child. A. Faint, fine stippling in upper lobes, especially left. B. Five months later, diffuse lace-like reticulations with minute cystic changes throughout right lung; pneumothorax (spontaneous) left with catheter in situ. (Purpuric eruption in skin, jaundice, generalized lymphadenopathy, enlarged liver and spleen. Autopsy showed, in addition, numberous blebs on surface of lungs; yellowish foci scattered in parenchyma; histologic picture in keeping with lipoid storage disease.)

ture of the defect is more readily ascertained; in the chronic, presumably cystic phases, the characteristic histiocytic response may not be demonstrable.

The lungs of the following patient, who was under the observation of the writer for several years, showed grossly the classical appearance of honeycomb lungs but they lacked distinguished histologic features. The roentgen appearance of cystic lungs is illustrated in the case report. (Fig. 4)

Azotemic Lungs: Toxic agents, including nitrogenous products in the blood, may damage the pulmonary vascular bed. The resulting filtration of fluid from capillaries into alveoli and interstitial tissues may give rise to edema. Inasmuch as pulmonary edema is apt to be transient and patients are often too ill to be examined roentgenologically, the condition is often overlooked. Until recently, the problem has been left largely to the curiosity of experimental physiologists and pathologists. With increasing use of bedside roentgenography, the subject is assuming a proportionate degree of clinical interest.

The basic features of acute pulmonary edema are the same whether the condition is caused by cardiac failure, excessive parenteral administration of fluids, or is present in association with nephritis and uremia or any other pathological condition. The mechanism involves an abnormal amount of fluid exchange in the lungs resulting from disturbances of hydrostatic and osmotic pressures, according to Starling's principles. The latter apply to the lesser as well as to the greater circulations (Paine et al). Acute pulmonary edema associated with azotemia is caused by left ventricular failure in the course of cardiorenal disease. Increased capillary permeability due to excess nitrogenous products in the blood is probably an additional factor. Although variable degrees of nitrogenous retention are demonstrable in patients with nephritis showing the pulmonary manifestations of azotemia, nitrogenous retention may be present in the blood without pulmonary involvement (Rendich et al).

The features of the azotemic lung which have aroused particular interest have been the roentgen changes. The chest x-ray reveals a butterfly distribution of fluffy, uneven, mottlings extending fanwise from the hilar regions into the adjacent portions of the lungs and obscuring the normal root shadows. The densities are apt to spare the periphery, the apices and very often the bases of the lungs. The conglomeration of irregular densities in the midcentral portion of both lungs surrounded by a fairly well illuminated zone of tissue may be quite striking. This is especially true in the absence of significant symptoms or signs referable to the chest. In fact, the discovery is often accidental. The roentgen appearance may be suggestive of the possible existence of azotemia and prompt the physician to investigate the patient for its presence. Initial acute seizures of pulmonary edema are reversible and, following treatment, the roentgenogram may resume a "normal" appearance. After recurring seizures of pulmonary edema and chronic pulmonary congestion, the densities are more nondescript, possibly as a result of associated pulmonary infarction. The densities tend to gravitate to lower portions of the lungs and variable amounts of free fluid usually appear in the pleural cavities. An enlarged heart is an almost inevitable accompaniment. (Fig. 5)

Cystic Fibrosis of the Pancreas and Pulmonary Infections: Since 1938 fibrocystic disease of the pancreas has become a popular subject of discussion in medical literature. In that year Dorothy Andersen, also Blackfan and May

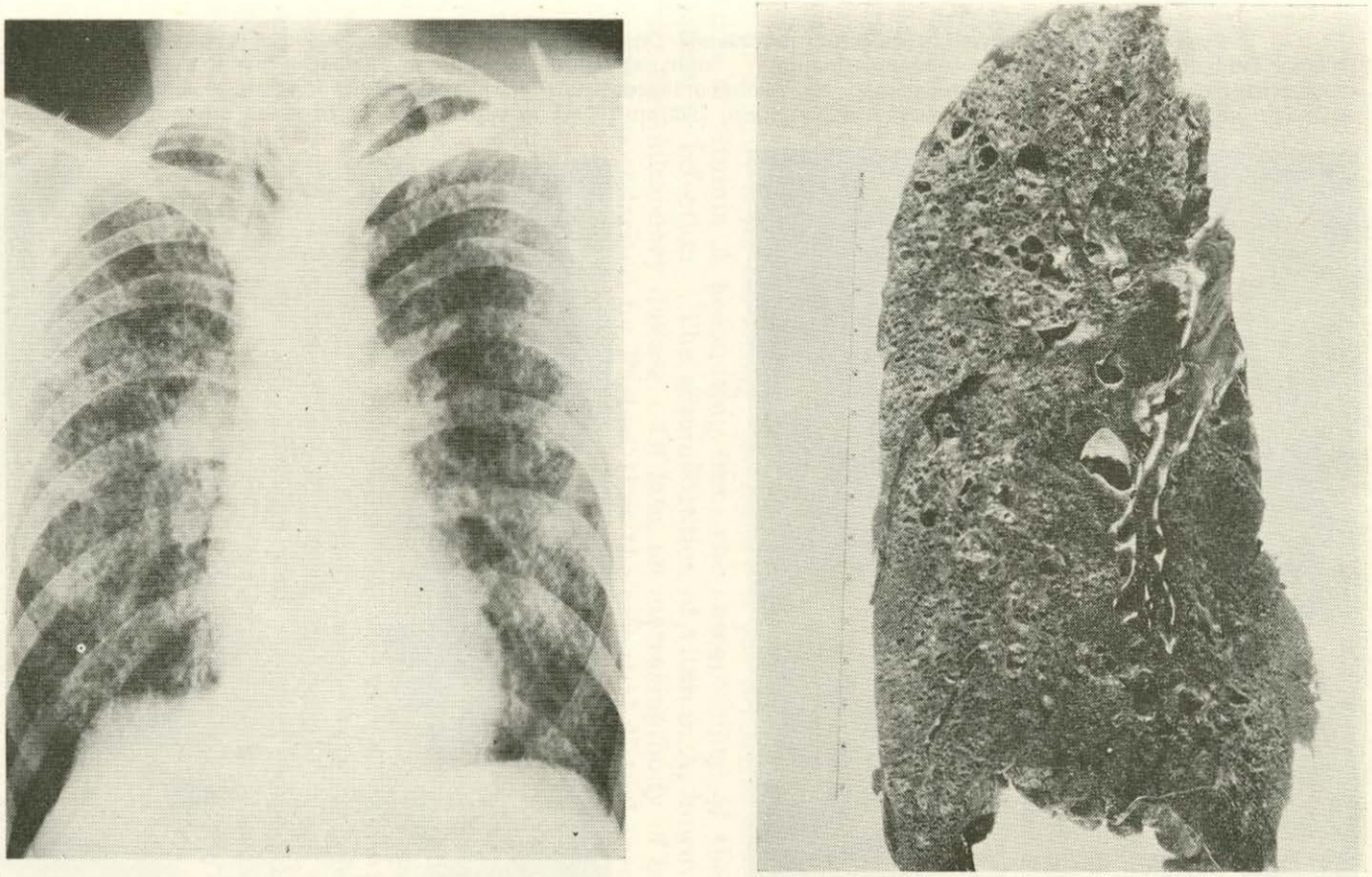


Figure 4.—Honeycomb lungs in a man of 37. A. Lace-like reticulations with minute air cysts throughout both lungs. (Seven years previously sustained bilateral pneumothorax; chest x-ray showed cystic changes of lesser degree). B. Specimen of lung shows diffuse honeycombing of organ. (Death caused by chronic cor pulmonale).

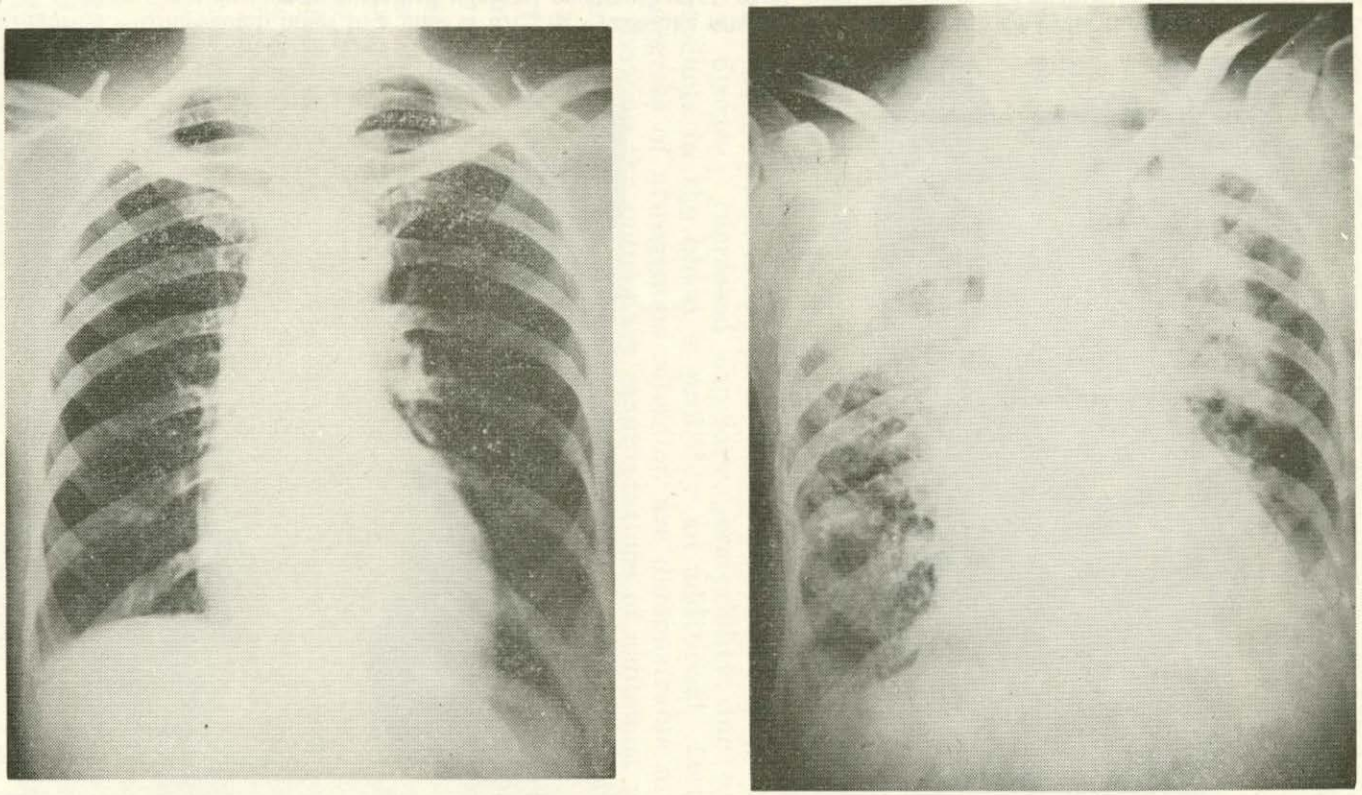


Figure 5.—Azotemic lungs in a woman of 45. A. Enlarged heart (hypertensive heart disease and chronic nephritis for a number of years); obscuration of right costophrenic sinus. B. Nine months later, ill defined, irregular densities in both lungs, especially of mid- and upper portions. (Clinical evidence of severe uremia; autopsy showed marked edema, hemmorhage, possibly uremic infarcts; contracted kidneys; hypertensive heart disease).

described the condition and brought it to the attention of the profession. The disease is usually present at birth and has a recessive but definitely familial tendency. Cystic fibrosis of the pancreas is now recognized as an important cause of death in infancy and early childhood.

Fibrocystic disease of the pancreas presents a clinical picture not unlike that met in celiac disease. The infant shows wasting, abdominal distention and eliminates fatty stools (steatorrhea) which are bulky and foul smelling. However, there are several noteworthy differences. In celiac disease no cause is demonstrable and improvement follows dietetic treatment. On the other hand, fully developed fibrocystic disease of the pancreas reveals histologically dilated acini and ducts which contain variable amounts of coagulated mucinous substance. Dietetic treatment is of no avail and the infant succumbs within a matter of months, or, at most, a few years. With the wider recognition of the disease and intensive use of antibacterial treatment, life may be prolonged.

Of particular interest, from the viewpoint of the present discussion, is the fact that fibrocystic disease of the pancreas is often associated with pulmonary infection to a degree that the latter is almost an integral part of the syndrome. In fact, the mother may bring the infant to the physician for treatment of the respiratory rather than the nutritional disorder. The reason for the association of cystic disease of the pancreas and pulmonary disease is not entirely clear. Andersen and her associates believe that the condition is the result of a nutritional deficiency, the failure of the infant to absorb adequate amounts of vitamin A accounting from the susceptibility of the respiratory epithelium to infection. The administration of vitamin A, however, does not improve the pulmonary disease. Of late, an increasing body of opinion holds to the view, popularized by Farber, that the essential disturbance is systemic in nature and is due to the production of an abnormal secretion of mucous glands in other organs as well as the pancreas.

The changes in the lungs, as in the pancreas, are due to obstruction of bronchi by viscid secretion. Obstruction of bronchi by plugs of mucus affect not only the small channels but often the larger bronchi and, at times, the trachea. This results in variable degrees of emphysema and atelectasis. Secondary infection, particularly with the hemolytic Staphylococcus aureus gives rise to suppuration. Early in the disease the chest x-ray may show few abnormalities although the infant may already have symptoms referable to the respiratory tract. After repeated infections, the lungs reveal accentuated perivascular and peribronchial markings and areas of consolidation. The disease is concentrated to the inner portions of the lungs. In time there is increasing diffuse fibrosis, emphysema, atelectasis and suppuration. The pulmonary manifestations of fibrocystic disease of the pancreas as revealed roentgenologically may be so striking that they may lead the physician to suspect the nature of the underlying disease (Fig. 6).

Amyloidosis of the Lungs: Atypical amyloid deposits are found on rare occasion in the heart, lungs, tongue, stomach, uterus, skin and other structures and tissues of the body. Atypical or primary amyloidosis differs from the secondary variety not only by the fact that the deposits are found in unusual sites, such as those mentioned, but also by the fact that the amyloid often fails to take on specific stains. Furthermore, the amyloid is apt to be deposited in nodular form and cause tumefactions of the affected organ, especially when the condition is associated with multiple myeloma. Atypical or pri-

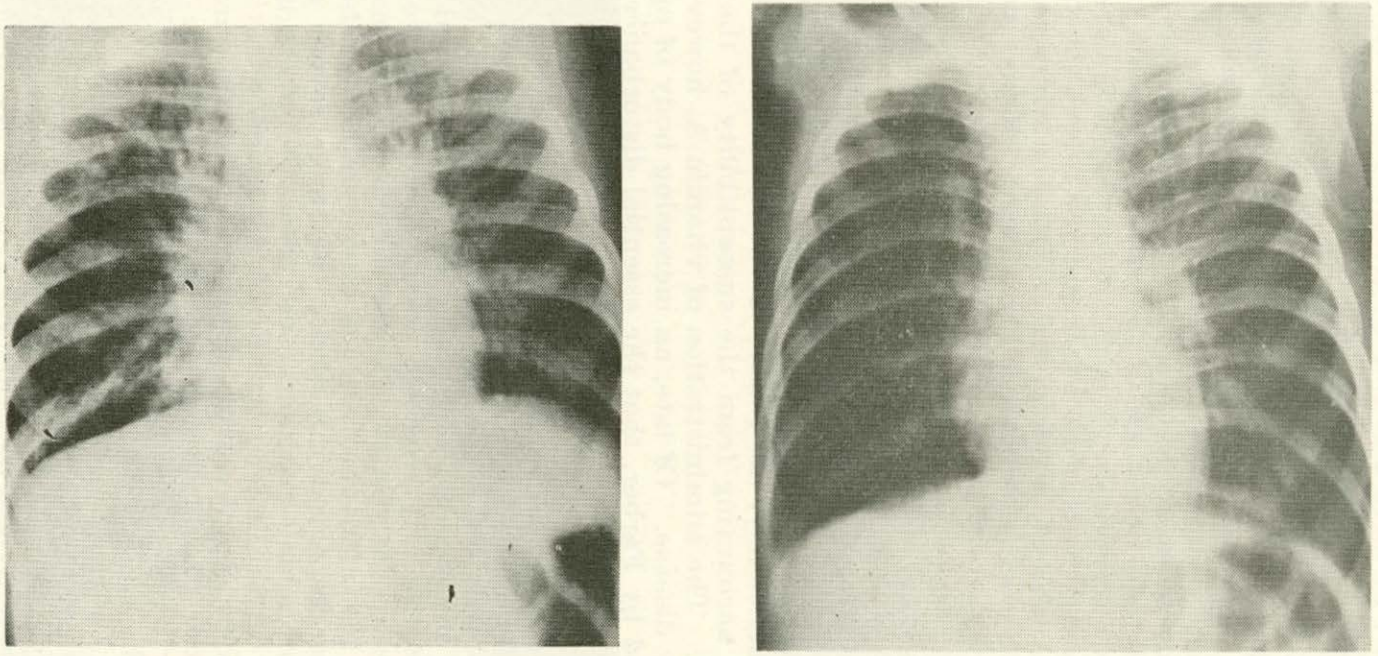


Figure 6.—Cystic fibrosis of the pancreas and pulmonary infection in a child. A. Increased bronchovascular markings in left upper lobe. (History of repeated respiratory infections and pneumonias since age of 7 months; cough, vomiting, lack of gain in weight; large, loose, yellowish, malodorous stools—pancreatic disease suspected. B. Four months later, considerable increase in linear markings in both lungs. (In spite of intensive antibacterial medication, expired at age of 26 months; autopsy showed cystic fibrosis of pancreas; diffuse fibrosis with bronchiectasis of lungs).

mary amyloidosis is not to be confused with the secondary variety encountered in the spleen, liver and kidneys as a by-product of chronic pulmonary tuberculosis, bronchiectasis, empyema and other suppurative diseases.

Amyloidosis may affect the lungs as a diffuse interstitial fibrosis spreading from the hili into both organs or as a localized process involving one of the major bronchi. An instance of the former, reported by Perla and Gross from the Montefiore Hospital, was diagnosed carcinoma; the autopsy disclosed amyloidosis. In another instance recently reported by Schottenfeld and co-workers, from the same institution, the disease was discovered accidentally. As in the preceding one, neoplasm was suspected but specimens removed bronchoscopically on repeated occasions showed amyloidosis. In most instances the disease involves the larynx, trachea or main bronchi; in a few, the lung parenchyma. Weismann, Clagett and McDonald reported a case of amyloid disease of the lung treated by pneumonectomy. The disease involved the right main bronchus, its branches and the corresponding branches of the pulmonary vessels at the root of the lung (Fig. 7).

Metastatic Calcifications of the Lungs: Metastatic calcification refers to a metabolic disturbance in which lime salts are deposited in previously unaffected tissues as a result of excess precipitation of calcium and phosphorus from the blood. A sharp distinction between metastatic calcification and dystrophic calcification, a by-product in the evolution of tuberculosis, histoplasmosis and other conditions, is not always possible. Metastatic calcification favors the lungs, kidneys and gastric mucosa, organs whose excretion of acid causes a more alkaline reaction in the tissue cells. This results in the precipitation of calcium and phosphorus from the plasma, these salts being less soluble because of the lower concentration of hydrogen ions in the tissues.

Directly concerned in the production of metastatic calcification are the effects of destructive lesions of bones, metastatic carcinoma, sarcoma, multiple myeloma, osteomyelitis; hypercalcemia in late stages of chronic renal insufficiency; demineralization of the skeleton caused by hyperparathyroidism in association with renal disease; osteoclasis produced by hypervitaminosis D_2, and prolonged ingestion of milk and alkali in long standing peptic ulcer, as well as local factors. A number of cases are on record of vitamin D_2 intoxication caused by overdosage with fortified cod liver oil or ultra violet irradiated ergosterol. Wilson and co-workers refer to approximately 120 reported cases of intoxication with vitamin D_2. In some, the metastatic calcifications resulted from treatment of arthritis with high doses of vitamin D_2. One such instance is presently at the Montefiore Hospital. The patient, a physician, had been medicating himself for about four years with large amounts of vitamin D for progressive arthritis. X-rays of various parts of the body reveal, in addition to hypertrophic arthritis, numerous calcific deposits in soft tissues most pronounced around joints, also in the abdomen and kidney regions.

The lungs, when involved, contain white chalky material which, on histological examination, are found chiefly in association with the arterial vascular system, also in the walls and capillaries of alveoli, bronchioles and bronchi. The chest x-ray shows a nondescript and variegated appearance. In most cases there are no demonstrable changes roentgenologically although the histologic appearance may be quite striking. In the cases described, as in the one illustrating this condition, the chest x-rays showed scattered irregular opacities distributed in lungs and lymph nodes (Fig. 8).

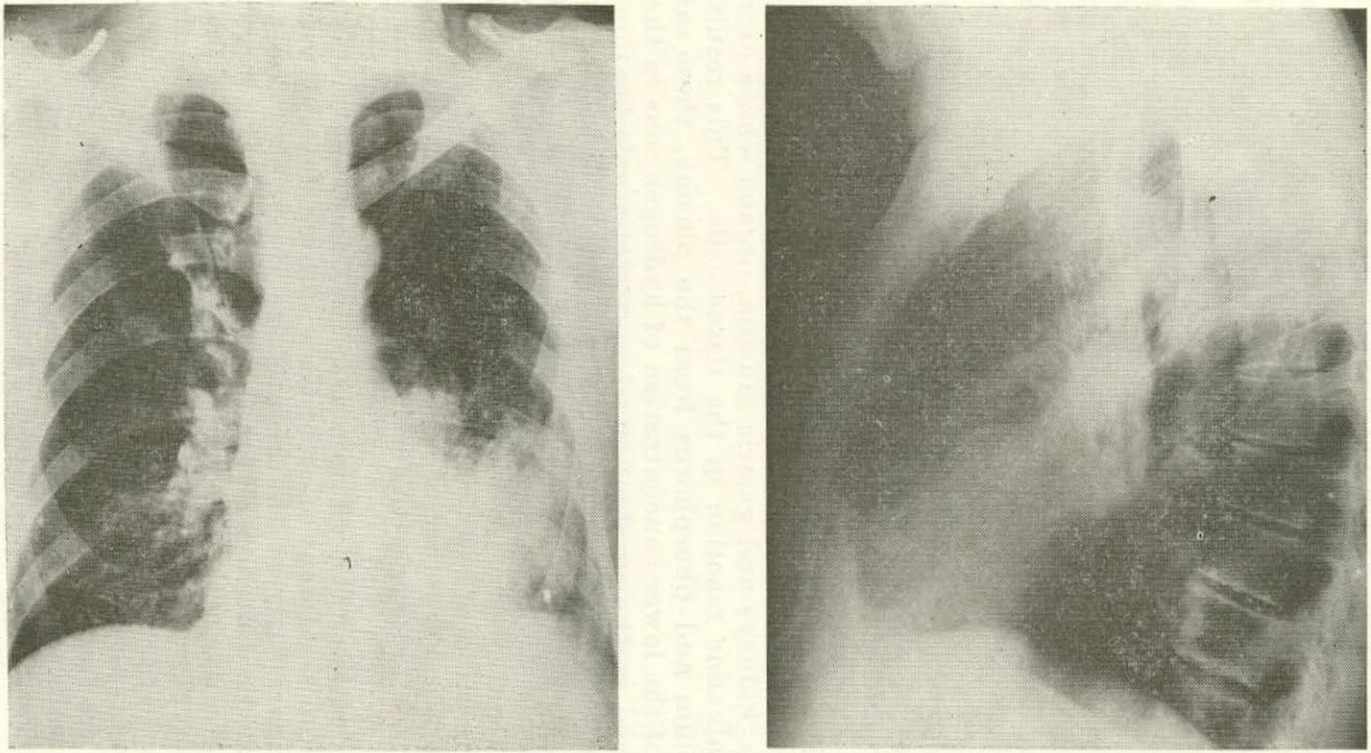


Figure 7.—Amyloidosis of the lungs in a man of 43. A. Irregular density in left lower lung field which in the left lateral projection B, appears to be localized chiefly at the hilum and in the lingula; infiltrations in left lower lobe; increased hilar and bronchovascular markings in right. (History 5 years duration; bouts of pneumonia, excessive cough and expectoration, dyspnea, hemoptysis. Specimens obtained bronchoscopically on repeated occasions revealed atypical amyloid deposits).

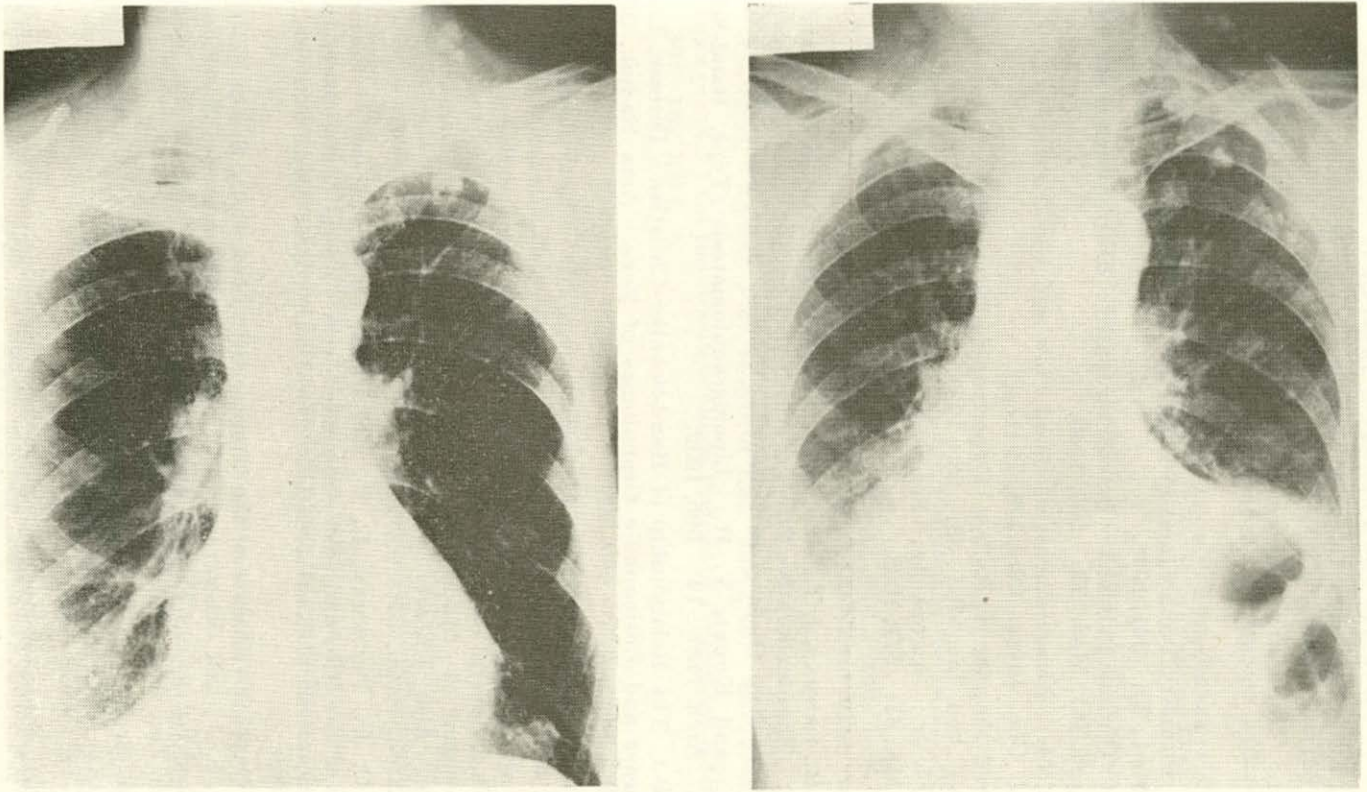


Figure 8.—Metastatic calcifications of the lungs from carcinoma of the breast in a woman of 54. A. Irregular, calcific in left upper lobe and left supraclavicular region. (Left radical mastectomy 4 years previously; histologic examination revealed undifferentiated carcinoma of breast and axillary lymph nodes with necrosis and calcium deposition; biopsy of supraclavicular lymph node also showed calcifying carcinoma). B. Three years later (7 years after operation), X-ray shows increase in number of calcific deposits, several being faintly seen in right lung and right supraclavicular region; pathological fracture second rib, left, posteriorly; upward displacement of left leaf of diaphragm due to phrenic nerve involvement.

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Lipoid Storage Disease of the Lungs

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Abstracts From Current Literature

DISSEMINATED ASPERGILLOSIS AND MONILIASIS ASSOCIATED WITH AGRANULOCYTOSIS AND ANTIBIOTIC THERAPY. Rankin N. E.: British Medical Journal I: 918-919, April 25, 1953.

A FATAL case of generalized aspergillosis and moniliasis is described. A window cleaner developed a staphylococcal abscess in the thigh and was treated with sulphonamides. Following surgical evacuation of the pus a pyaemia resulted with lung lesions. He was treated with penicillin and chloramphenicol. Total dosage of chloramphenicol (chloromycetin in this country) was 30 grams. An agranulocytosis developed and despite vigorous treatment the patient died. Necropsy tissue sections revealed two different fungi, Aspergillus fumigatus and Candida albicans.

MONILIA PERITONITIS. P. C. Reynell, E. A. Martin, A. W. Beard: Ibid,

A gastro-intestinal case with fever developed an active carditis and was treated with penicillin. Response was poor and he was given 100 mg. of A.C.T.H. and 2 gm. of aureomycin daily. He developed a peritonitis and from the aspirated fluid Candida albicans was cultured. Intraperitoneal gentian violet and intravenous terramycin failed to prevent a fatal outcome.

(It is too early to state if the above articles are typical of a state of affairs that will become more common or not. Aspergillosis and moniliasis are usually localized infections and dissemination has been considered a rarity. However, wide spectrum antibiotics and multiple antibiotic therapy will suppress the growth of susceptible bacteria and these may be replaced by fungi which can in certain circumstances become pathogenic and produce disseminated fatal disease.

Powerful remedies bring with them fresh complications and dangers. More reports are required before the extent of this danger can be assessed.)

G. A. BLACK

TREATMENT OF DELIRIUM, PSYCHOSIS, AND COMA DUE TO DRUGS. Jonathan Gould Lancet I: 570, 573 Mar. 21, 1953.

The standard treatment of barbiturate poisoning is discussed. The disordered brain symptoms—delirium, coma and psychosis—are thought to be related mainly to abnormal glucose metabolism. And it is known that in the chemical reactions involved the important substances necessary are oxygen, glucose, ascorbic acid, aneurine hydrochloride, and nicotinic acid.

The author has used large dosages of the above mentioned components of the vitamin-B complex group, ascorbic acid, and glucose intravenously. The preparation is administered by rapid intravenous drip and repeated as indicated. Favourable results were obtained in barbiturate poisoning, post-operative psychosis, in delirium due to drugs, and in acute alcholic psychosis.

(The author seems to have modified and greatly extended the technique that was evolved several years ago in the treatment of delirium tremens of alcoholic origin.)

G. A. BLACK

APRESOLINE IN THE TREATMENT OF HYPERTENSION: Hafkenschiel, J. H., Lindauer, M. A.: Circulation 7: 52-58, January, 1953.

These investigators from the University of Pennsylvania followed 40 patients on a low salt diet and apresoline over a period ranging from one year minimum to two years maximum with particular emphasis on patients who might be expected to do poorly with sympathectomy. In the less severe hypertensives, diastolic blood pressures fell from 120 mm. or higher to below 110 mm. at least twice during the period of study in 15 out of 26 mild cases but in only 5 out of 14 moderately severe to severe cases. Other evidences of improvement occurred in only 7 of the 40 patients.

Side reactions were common, 15 of the cases suffering headaches, 7 palpitations and 5 nausea and vomiting.

It would appear from this article that those cases who are suitable for surgery also benefit from apresoline while those who are not, do not.

LEA C. STEEVES

A NEW EPIDEMIC DISEASE: Pearson, J. S.; Phlebodynia, Circulation 7: 370-372, March, 1953.

The author reports on 22 cases seen in the fall and winter of '50-'51 in the nursing staff of his hospital, complaining of incapacitating pain in one or both lower extremities. The only finding was exquisite tenderness along the course of the superficial veins with cord like thickening and slight swelling locally. Systemic signs were not unusual, consisting of headache, gastrointestinal upsets, lower abdominal pain, low grade fever. Epidemiological studies and detailed laboratory examinations were non-contributory, including biopsies of the veins and bacteriological studies of the biopsy samples. The biopsies revealed "subacute phlebitis limited strictly to vasovasorum", no therapeutic benefit derived from any of the antibiotics or from ACTH. Bed rest and elevation of the legs was followed by symptomatic relief but recurrence developed in several cases and overall morbidity was prolonged.

LEA C. STEEVES

Technical Procedure

THE TUBERCULIN TEST

THE tuberculin reaction is an acute, local, specific inflammation resulting from the injection or application of tuberculin. Few procedures employed in the control of tuberculosis are of greater value than the tuberculin test. As an index of the extent of infection in communities, it is of the utmost epidemiological value. It locates sources of contagion and provides a guide for case finding and family and group supervision. Its importance in the diagnosis of tuberculosis rises constantly as the rate of infection in the population decreases and as the total number of cases of active disease declines. Whereas its value in clinical diagnosis once was exclusive rather than definitive in character, the tuberculin test is becoming a more positive indication of significant infection. Finally, with the expansion of BCG vaccination programs and the importance of determining the relation of artificial infection with an organism of low virulence to the development of individual resistance, the tuberculin test has become of special significance as an indication of the "take" of the vaccine.

Obviously, once BCG has been administered, the tuberculin test loses its specific value as an indicator of natural infection.

A reaction to certain specified amounts of tuberculin is an indication of tuberculosis infection. With few exceptions, definite sensitivity to tuberculin once acquired persists through life. This sensitivity may vary in intensity and may temporarily decrease or disappear in the course of high fever, exanthematous disease, miliary tuberculosis, and the last stages of pulmonary tuberculosis. A very small percentage of persons may fail to react to tuberculin even after infection resulting from exposure to cases of open tuberculosis or after the administration of vaccine prepared from living or dead tubercle bacilli. The tuberculin test remains negative in a high percentage of cases of sarcoidosis.

At the present time Purified Protein Derivative (PPD) and Old Tuberculin (OT) are widely used. Purified Protein Derivative (PPD) is the tuberculin of choice and is recommended as the standard for comparative studies. It is prepared by growing tubercle bacilli on a synthetic medium of known composition. The tuberculin protein produced in the medium is then obtained by precipitation with ammonium sulfate at neutrality and further purification. Old Tuberculin is prepared by concentrating by heat the glycerine broth medium upon which the tubercle bacillus has grown.

Technique

The method of choice in administering tuberculin and the one recommended for accuracy and for all general purposes is the intracutaneous (Mantoux) test. Occasionally, however, special circumstances may make other methods of administration preferable. In the following paragraphs the intracutaneous method is described as the standard procedure. A description is also given, however, of the scarification (Pirquet) test and of the Vollmer patch test, which is frequently employed when there is objection to the use of a needle. Neither of these tests is considered as accurate as the intracutaneous test, in which a measured amount of tuberculin is introduced into the skin.

Intracutaneous or Mantoux Test. The intracutaneous tuberculin test is best carried out by injecting the desired concentration of PPD or Old Tuberculin into the cleansed skin of the forearm. This injection is made with a short (half inch), sharply beveled 24-or-25-gauge platinum or steel needle and a tuberculin syringe. When the injection is properly made, a wheal should appear immediately at the site of injection.

The use of a single dose of 0.0001 mg. of Purified Protein Derivative (PPD) or 0.1 mg. of Old Tuberculin is recommended for case-finding programs. This dose is recommended since persons showing roentgenographically demonstrable lesions characteristic of tuberculosis are, in the vast majority of instances, highly sensitive to PPD or Old Tuberculin. A dose of 0.00002 mg. of PPD or 0.01 mg. of Old Tuberculin is recommended for the initial dose for those living in areas having a high morbidity or mortality from tuberculosis, for those with a history of severe reactions following previous administration of tuberculin, for those with a history of intimate contact with persons with clinically manifest tuberculosis, and for persons, with extrapulmonary forms of this disease.

If PPD is used, a standard product, which is available on the market, is dissolved in the requisite amount of the diluent supplied so that each 0.1 ml. of the dilution will contain 0.0001 mg. PPD. To prepare the dose of 0.1 m.g. of Old Tuberculin recommended for case-finding purposes, 0.1 ml. of concentrated Old Tuberculin is added to 9.9 ml. of sterile physiological salt solution, thus making a dilution of 1 : 100. One milliliter of the 1 :100 dilution of Old Tuberculin is transferred to 9.0 ml. of sterile physiological salt solution, thus making a dilution of 1 : 1,000. Each 0.1 ml. of this dilution will contain 0.1 mg. of Old Tuberculin. Purified Protein Derivative and the diluted Old Tuberculin are best prepared fresh. Solutions more than a few days old, should not be used. When not in use diluted solutions should be kept in a refrigerator.

For the present, with certain exceptions as noted, the use of a single dose is recommended. Further studies on the specificity of the larger doses of both PPD and Old Tuberculin are being made.

The intracutaneous tuberculin test (Mantoux) with either PPD or Old Tuberculin should be read 48 or 72 hours after the injection. Readings should be made in a good light with the arm slightly flexed. Response to injection is classified as positive, negative, or doubtful. Reactions may be classified arbitrarily as one, two, three, or four plus depending upon the extent of induration measured at its widest diameter. A reaction showing some definite induration more than 5 mm. and not exceeding 10 mm. in diameter is recorded as a one plus (+) reaction. A two plus (++) reaction is an area of induration measuring from 10 to 20 mm. in diameter. A three plus (+++) reaction is characterized by marked redness and induration exceeding 20 mm. in diameter. A four plus (++++) reaction consists of severe induration and an area of necrosis. A reaction with a trace of induration measuring 5 mm. or less in diameter is rated as doubtful. Redness without associated induration does not constitute a reaction.

Syringes which have been used for the administration of PPD or Old Tuberculin should not be used for the administration of preparations such

as coccidionidin, histoplasmin, or other diagnostic reagents since PPD and Old Tuberculin are difficult to remove from the syringes.

Scarification or Pirquet Test. This procedure is carried out by applying a drop of undiluted Old Tuberculin to the cleansed skin, preferably over the region of the deltoid muscle, and scarifying the skin through the drop of tuberculin by means of a sterile needle or small borer. Care should be taken that the scarification does not cause bleeding. The tuberculin should be permitted to remain *in situ* for about 20 to 30 minutes before the excess is removed. The results of the test are read 48 to 72 hours later and are interpreted in the same manner as the intracutaneous test. If there is no tuberculin reaction by this method, it is advised that it be followed by the administration of 0.0001 of PPD or 0.1 mg. of Old Tuberculin, using the intracutaneous method.

Patch or Percutaneous Test of Vollmer. The patch test is carried out by applying a prepared patch to the skin in the region of the sternum or between the scapulae. The patch is composed of two squares impregnated with concentrated tuberculin prepared from cultures grown on a synthetic medium and one control square impregnated with the concentrated culture medium. The skin is first cleansed with acetone and the patch applied and permitted to remain *in situ* for 48 hours. The reaction is read 48 hours after the removal of the patch. A reaction appears as an inflamed area of varying intensity. In those highly sensative to tuberculin, it is advisable to remove the patch as soon as the persons complains of irritation since a severe local reaction with blistering may occur. If the patch test is negative, 0.0001 mg. PPD or 0.1 mg. of Old Tuberculin should be injected.

Of the procedures here described, the intracutaneous method is the most sensitive and permits the administration of graduated and accurate amounts, thus making it possible to detect cases with a low threshold of sensitivity to tuberculin. The general use of the Pirquet and the Patch Test is not recommended since they are not as efficient as the Mantoux Test.

MUST WE CHANGE OUR CODE OF ETHICS?
Interim Report of the Public Relations Committee

H. G. Grant, M.D.,
Secretary, The Medical Society of Nova Scotia,
Dalhousie Public Health Clinic,
University Avenue,
Halifax, N. S.

Dear Doctor Grant:

At this time the Public Relations Committee appointed at the last Annual Meeting feel that there is reason and need for an interim report to the Executive on the work that has been done to date. Following direction from the Executive at its December meeting when $300.00 was appropriated to provide for articles in the daily press of public relations interest we retained the services of a local newspaperman to write articles on timely topics, namely medical licensing and medical education. The material for these articles has been provided by members of our Society and before going to press the final copy is being edited and approved by the special committee appointed for that purpose. We expect that the first two articles should be appearing in the month of July; two more similar releases are planned for the current year, probably in the early Fall.

Our Committee has been in more or less constant touch with the parent Committee on Public Relations of the Canadian Society, and we have on file considerable material indicating the desire by that Committee to see certain changes made in our Code of Ethics, particularly those dealing with our relations with the press and radio. The parent committee stresses in particular the need for improved press relations and suggested the releasing of suitable articles in the press throughout the country attempting in a small way to clear up many misconceptions relating to the practise of medicine. The Committee has forwarded us a draft on the proposed radio-press relations code which our Committee plans to study with a view to making any recommendations to the Annual Meeting in the Fall. We feel that the need of suggested changes in our Code of Ethics may prove controversial and we plan to make a thorough study of the matter and report further in the Bulletin any suggested changes prior to the presentation of the Committee's report at the Annual Meeting.

Respectfully submitted,
FRED J. BARTON,
Chairman, Public Relations Committee.

PHYSICIAN WANTED

Physician needed for a Mining Company in the Gaspe Peninsula. Good salary—House provided. Apply to Secretary.

LOCUM TENENS WANTED

Doctor J. Cameron MacDonald of Freeport, Nova Scotia, wishes a Locum Tenens for two weeks, on or about June 26th to August 10th. Attractive financial arrangements can be made.

Society Meetings

ANTIGONISH-GUYSBOROUGH MEDICAL SOCIETY

Dr. H. G. Grant,
Secretary,
The Medical Society of Nova Scotia,
Halifax, N. S.

Dear Doctor Grant:

The present officers of the Antigonish-Guysborough Medical Society are:
President—Dr. T. B. Murphy, Antigonish.
Vice-President—Dr. G. L. Silver, Sherbrooke.
Secretary-Treasurer—Dr. J. E. MacDonell, Antigonish.
Executive—Dr. Alden Hansen, Canso, Dr. Rolf Sers, Goldboro, Dr. T. W. Gorman, Antigonish.
Representative to The Medical Society of Nova Scotia—Dr. J. A. MacCormick, Antigonish.
These officers were elected at the Annual Meeting of November 30, 1952.

Yours truly,
(Sgd.) J. E. MACDONELL, M.D.,
Secretary-Treasurer,
Antigonish-Guysborough Medical Society.

CAPE BRETON MEDICAL SOCIETY

Dr. H. G. Grant,
Secretary, Nova Scotia Medical Society,
Dalhousie Public Health Centre,
Halifax, N. S.

Dear Doctor Grant:

At the Annual Meeting of our Society held in Sydney on May 6th, the following slate of officers were elected for the year 1953-54.

President—Dr. H. J. Martin, Sydney Mines.
Vice-President—Dr. A. W. Ormiston, Sydney.
Secretary—Dr, H. R. Corbett, Sydney.
Treasurer—Dr. Carmen D'Intino, Sydney. Re-elected.
Executive Members—The Medical Society of Nova Scotia, Dr. Harold Devereux, Sydney, Dr. J. A. MacDonald, Glace Bay.
Executive, Cape Breton County Medical Society—Dr. Ray Ross, Sydney. Dr. W. T. McKeough, Sydney Mines, Dr. Malcolm Chisholm, New Waterford.

Would you be good enough to pass this list on to Dr. Margaret Gosse, for the Bulletin?

Sincerely,
H. R. CORBETT, M.D.,
Secretary.

THE PICTOU COUNTY MEDICAL SOCIETY

Dr. H. G. Grant,
Secretary-Treasurer,
Nova Scotia Medical Society.
Dear Dr. Grant:

At our annual meeting of May 6th the following slate of officers was elected.

President—Dr. C. E. Stuart, New Glasgow.
Vice-President—Dr. S. D. Dunn, Pictou.
Secretary-Treasurer—Dr. H. A. Locke, New Glasgow.
Executive Member—Dr. C. G. Harries, New Glasgow.

The meeting was held at the Braeside Inn, Pictou, with guest speaker, Doctor Denis Howell, who spoke on "The Insulted Skin". Following this a lobster dinner was enjoyed.

Yours very truly,
(Sgd.) H. A. LOCKE, M.D.