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150 to 115 pounds over a period of several years. Past medical and family history were negative.

Physical examination revealed an emphysematous type of chest with a good deal of emaciation. His systolic blood pressure was 100 with a diastolic pressure of 70. There was marked supraclavicular and infraclavicular retraction with dullness over the upper third of both lungs and increased resonance below. Breath sounds were greatly diminished in the upper third on both sides. Coarse moist râles and musical râles were present over the lower two-thirds of the chest.

The roentgenogram showed dense shadows in the upper half of both lungs, suggesting consolidation; in the third and fourth intercostal spaces there were fine and moderately coarse mottlings. The heart was enlarged to the right. Both diaphragmatic leaves were depressed.

Sputum examinations were repeatedly negative for tubercle bacilli and fungi. A blood count showed nothing of significance. Urine contained a faint trace of albumin. Kahn precipitant test was one plus.

The temperature was never elevated and pulse rate averaged 80 while at rest in bed. His condition remained unchanged from the time of admission on June 22, until Sept. 13, 1931, when he began complaining of severe pain in the left chest with dyspnea. He perspired profusely and the temperature became subnormal. He grew gradually worse and died 12 hours after the onset of pain.

At necropsy the lungs were removed with little difficulty. The upper lobes of both lungs and about half of the right middle lobe were retracted and had the consistency of a solid rubber ball. The lower lobes appeared well aërated. On section, both upper lobes were solidly filled with carbon particles. The adjacent lobes contained scattered deposits and were emphysematous with many blebs on the outer surface. The heart walls were thin and dilated and the coronary vessels revealed a great deal of sclerosis. A very thorough examination both macroscopically and on section failed to reveal any evidence of tuberculosis or other chronic infection. A few fibrous and calcified tubercles could be demonstrated in one of the peribronchial lymph nodes.

The cause of death was apparently right heart failure caused by fibrosis and emphysema of the compensatory and presenile type.

This patient was unusual in our experience because of a third degree anthracosis with dense deposits of carbon dust practically limited the upper half of the lungs. The findings before death were such that it was difficult to make a definite clinical diagnosis. Though we were not satisfied that the patient's symptoms were due to tuberculous disease, pulmonary tuberculosis could not be entirely ruled out before necropsy. The reason for the unusual distribution of the anthracotic material remains unexplained. The cause of the fibrosis is also open to discussion. Some authorities maintain that it is the direct result of the irritation of the dust; others believe that it has a primary infectious basis.

Tuberculosis League Hospital.

PULMONARY ASBESTOSIS*

BURGESS GORDON, JR., M.D., PHILADELPHIA

Although workers have been employed for many years in mining asbestos, it was not until recently that this occupation was recognized as an industrial hazard. It appears that Murray, in 1900, published the first report on the essential features of pulmonary fibrosis with spicules of asbestos. This apparently was not widely recognized until 1924 when Cooke published in England his findings of an extensive fibrotic process distinct from so-called tuberculous foci in a patient who worked with asbestos for 20 years. In 1927, he characterized the deposits in the lung as iron-containing fragments. Page and Wood reported necropsy findings similar to those of Cooke, but without evidence of complicating tuberculosis. An interesting case of asbestosis was reported by Soper in 1930. The patient, except for short periods, had been an asbestos worker for 13 years. The onset of symptoms was in 1928 when he complained of dyspnea, malaise, and loss of weight. Blood streaked sputum occurred in 1929; later, burning pain in the chest; whitish expectoration in the mornings; and marked shortness of breath. The skin was cyanotic but there was no clubbing of the fingers. The roentgenograms showed diffuse shadows over the lower two-thirds of the chest. Dyspnea was a striking phenomenon. Mills also described a case in 1930 with death occurring about 17 years after exposure to asbestos dust. Lynch and Smith reported a case in 1931 in which the essential clinical features were a history of work for 14½ years (1911-1925) in an asbestos plant; the onset of cough and expectoration occurred in 1914-1915, dyspnea and pain in the lower part of the chest in 1918. The clinical findings were not unlike those reported by Soper. Necropsy showed hyaline fibrosis of the lungs, bronchiectasis, and passive congestion of the viscera. Death was due apparently to slow cardiac failure. Lynch and Smith collected a series of 172 cases of pulmonary asbestosis. The majority of these occurred in England. In 27 instances asbestos bodies were found in the sputum, in lung juice by puncture, or at necropsy. In 3 necropsies the disease was complicated by pulmonary tubercu-

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losis, 3 by lobar pneumonia, and 3 by bronchopneumonia. So far as determined there are only 4 records in the literature of necropsy on uncomplicated pulmonary asbestosis. The following case is reported because of the rapidly growing interest in the condition.

The patient, male, single, aged 54, entered the Department of Diseases of the Chest, Jefferson Hospital, on Sept. 3, 1930, complaining of weakness, loss of weight, pain in the chest and dyspnea. He had been a janitor and worker in various factories for many years. His family history was irrelevant. He had scarlet and typhoid fevers in childhood. There were no important contacts with tuberculosis and up to his present illness, he had been a strong, hard working man. The onset of his present illness was in 1929 when fatigue, nonproductive cough, and pain in the chest occurred. These symptoms were aggravated by deep breathing and were most severe in the winter. He entered the Philadelphia General Hospital in February, 1930, and remained for 11 weeks. He returned to work somewhat improved but soon weakness and shortness of breath increased and there was a loss of 11.5 kg. in weight. In May, he was placed in the out-door clinic of the Chest Department of the Jefferson Hospital. He improved during the summer but in October his symptoms were marked and he was admitted to the ward.

In the physical examination he was described as emaciated but well developed. The chest was emphysematous in type and expansion was diminished, especially on the left side. The percussion note was impaired at the right apex and below the clavicle on the left side. The breath sounds were harsh throughout, especially at the right apex. Coarse rales were heard practically over the entire two lungs. Amphoric breathing and whispering pectoriloquy were present over the right apex. The roentgenograms showed a process not unlike tuberculosis involving the right apex. This was fibrotic in type and with cavity formation. The interlobar pleura on the right side was thickened and the pulmonary markings in the right lower lobe were increased. In the middle and lower regions of the left lung the shadows, according to Dr. John T. Farrell, Jr., "resembled dust changes rather than bacterial invasion." The laboratory findings were essentially negative except a red blood cell count of 2,800,000 and hemoglobin of 50 per cent; there was an occasional trace of albumin in the urine. The sputum was mucopurulent in type. No tubercle bacilli were found. His temperature and pulse were normal except during the first 3 days after entry when there was a slight increase. He gained 4.2 kg. in weight. He left the hospital against advice, symptomatically improved, on Feb. 23, 1931. A diagnosis of pneumonoconiosis and pulmonary tuberculosis was made. The former was considered because of signs of diffuse change in the lungs, especially in the hilus areas. The question of pulmonary tuberculosis was raised because of apical involvement and cavity formation.

Two weeks following discharge he experienced loss of appetite and insomnia. His previous symptoms returned and he reëntered the hospital on March 7, 1931. There was no essential change in the findings except increased sputum, weakness, and dyspnea. His temperature increased to 100° to 101° F., the dyspnea became extremely severe and he died following a stupor of 16 hours, 26 days after reëntry.

Necropsy showed essentially normal organs, except for the following: Atheromatous nodules and patches were present at the base of the aortic valves and in the left coronary artery. On the parietal pleurae were discrete, small, gray nodules of firm consistency and on the left side there were fibrous adhesions. In the upper half of the left upper lobe was a large cavity, honeycomb in type. The margins of this were not limited by fibrosis and it appeared the excavation had progressed without the usual formation of a fibrous wall. Throughout the remainder of the lung there were areas of brown and black pigment and the tissues were firm in consistency. In the intervening parts the lung was red, firm, and somewhat bloody. Crepitation was practically absent. In the middle of the right lung, on the outer surface, were two fibrous lesions. The left kidney was small, contracted, and firm.

In the histologic examination, the caseous lesions in the right lung had the appearance of lymphatic and epitheloid cell infiltration. There was extensive necrosis but no giant cells and very little fibrous change was present. The histology of the larger pulmonary lesions resembled diffuse fibrosis. In many places the alveolar walls were thickened, in others the alveoli had disappeared in the maze of fibroid tissue. The bronchi showed chronic catarrhal inflammation and there were areas of pneumonia. Tubercle bacilli were not found in smears taken from these tissues. The arteries showed obliterative endo-arteritis. The most striking features were the asbestos bodies in the lung juice and alveoli. These measured 10 to 100 microns, were golden brown in color, and appeared as a series of regular disks not unlike red blood cells in roulette formation. The response to stains was interesting in that hemotoxylin, eosin, or Gram's stain had no effect but the prussian blue reaction for iron was pronounced.

In considering these data it appears that the most striking clinical features were the absence of toxic phenomena and the gradual onset of respiratory manifestations with aggravation in the winter time which decreased in severity during the summer and following rest. Dyspnea was far greater than that noted usually in patients with more extensive chronic pulmonary change. In the physical examination certain signs suggested tuberculosis but the type of rales heard throughout and the variations in breath sounds were characteristic of asthma, dust inhalation, and nonspecific pulmonary infections. The roentgenograms were not altogether helpful because of two processes, i. e., cavity formation and diffuse shadows.

The necropsy diagnosis of asbestosis and atypical pulmonary tuberculosis, the former not being suspected before death, emphasizes the importance of the occupational history in the study of pulmonary disease. (The fact that the patient was employed in an asbestos mine was not discovered until after necropsy because the patient and relatives regarded this apparently as only an incident in a long list of occupations.) In doubtful cases the technic for sputum study reported by Simson and Strachan should be used. This is as follows: Two series of films are made from the sputum. The first slides contain films of thick mucoid sputum. These are dried in a paraffin oven at 54° C., fixed, and saturated with mercuric chloride solution, one set being stained with hemotoxylin and eosin to determine the presence of various organisms, the other is

stained for tubercle bacilli. The second series is made from the centrifuged deposit after digestion with antiformin. The fact that asbestos bodies were discovered in 48 out of 50 asbestos workers emphasizes the value of this procedure.

1832 Spruce Street.

SPONTANEOUS RUPTURE OF THE ESOPHAGUS

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Spontaneous rupture of the esophagus includes those cases of sudden rupture in which there are no discernible signs of previous disease of the organ. The condition occurs most frequently in adult males, who have been addicted to the excessive use of alcohol. Twenty-four of the 28 cases collected by Whipham were in males, and 17 of them were more than 30 years of age. Most of the esophageal ruptures reported, occurred during the act of vomiting or retching. A few followed a fall or other external violence. In all cases, the rupture occurred in the posterior or posterolateral wall of the esophagus immediately above the diaphragm.

Spontaneous rupture in a healthy organ has been denied by most observers. Williams and Boyd found the surrounding tissue denuded of mucous membrane, and infiltrated with mononuclear and polymorphonuclear leukocytes, which they attributed to a preëxisting inflammation. Zenker and Ziemssen ascribe the rupture to an intravitam esophagomalacia caused by retained acid and pepsin. Beneke believes that in some patients excessive acidity and low resistance in the tissues predispose to softening of the wall. McWeeney described a condition of inflammatory softening with round cell infiltration at the site of rupture and supposes this to be the predisposing cause.

The condition is of interest not only because of its uncertain etiology and rarity, but also on account of the marked resemblance of the cases in history, signs, and symptoms. Such a case, without the history of recent alcoholism, is herewith reported.

A white male, aged 46, was admitted to the Jefferson Hospital, September, 1928, complaining of intense pain under the lower half of the sternum and the precordium. His family history and previous medical history were unimportant, except for rheumatic fever at the age of 12, and several attacks of tonsillitis. He used tobacco moderately. He drank considerable liquor between the ages of 25 and 35, but during the past 10 years, he has taken only an occasional drink. There is no history of difficulty in swallowing and his digestion had always been good except for an occasional attack of heartburn, which he attributed to chewing tobacco.

The onset of his present illness occurred while lifting a 50-pound bundle. He felt a sudden, severe pain in the chest, became very weak, and had to sit on the floor. He vomited a small amount of blood, but was not nauseated. The pain was dull, nonradiating, and constant. He continually called for water and drank freely, but did not continue to vomit.

The patient, a well nourished adult, was suffering intensely. His skin was ashen gray, cold, and clammy. The chest was an emphysematous type. The respirations were rapid (35 per minute) and shallow. Expansion was diminished but equal. The percussion note was hyperresonant throughout. The breath sounds were distant on the right side and absent over the lower half of the left side. The heart sounds were rapid, regular, and weak. The heart dullness was completely obliterated. The abdomen was scaphoid, symmetrical, and tympanitic on percussion. The muscles over the upper half of the abdomen were rigid. There was exquisite tenderness in the epigastrium upon light pressure.

The blood examination showed 4,750,000 red blood cells, 12,000 white cells, and 85 per cent hemoglobin, with a normal differential count.

The urine was normal.

The patient's condition became progressively worse. Five hours after admission his temperature was 96.5° F., pulse rate 160, and respirations 50 per minute. A half grain of morphine had afforded him very little relief from the pain. At this time subcutaneous emphysema was seen in the left supraclavicular fossa, and 2 hours later it had extended over the neck and face. Death occurred 9 hours after the onset of symptoms.

Necropsy was performed 14 hours after death. The following is an abstract of the report.

On opening the chest there was a large amount of interstitial emphysema in the mediastinum. The left pleura contained air and about a liter of greenish-yellow fluid with fat and food particles. There was a laceration of the left pleura near the vertebral column and just above the diaphragm. The surrounding tissues were dark and edematous.

The esophageal wall and mucosa were of normal thickness. There was a longitudinal rupture 5½ cm. in length in the left side of the lower end of the esophagus, with a slight inflammatory reaction and some extravasation of blood in the margins of the rupture. No ulceration of the mucosa or thickening of the wall was present to indicate a previous lesion.

Upon histologic examination of the sections from the ruptured margin of the esophagus, there was present a slight inflammatory reaction with polymorphonuclear cell infiltration. There was no evidence of a chronic inflammatory lesion. The inflammatory reaction present could easily be accounted for by the reaction of the tissues following a traumatic rupture and the irritation by the gastric secretions.

Chemical examination of the fluid found in the left pleura showed blood and hydrochloric acid.

Our studies fail to reveal any predisposing cause for this rupture. I believe the act of lifting the comparatively light bundle was not the sole cause, although it is evident that it was a contributing factor.

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