When preserved in liquor formaldehydi, the constriction of the duodenum presented the appearance of an obstructing fold of the duodenal wall rather than of a narrow tube, 15 mm. long, as it had appeared before fixation. Grossly and microscopically the constriction was covered with normal mucous membrane, and careful search failed to reveal any scar tissue or other evidence of old ulceration. The condition was therefore considered to be congenital in origin, the type of occlusion corresponding to that represented in Figure 1 A. The possibility of the condition being caused by an old ulcer which had disappeared without leaving any trace of itself was, of course, considered; but this is quite improbable, and is all the more unlikely in view of the rarity of duodenal ulcer in early youth.

In 302 cases of ulcer of the duodenum reported by Moynihan,¹⁶ there were but three occurring under 20, his youngest patient being aged 17. In the case here reported, symptoms of stenosis had already developed by the age of 20.

Why symptoms due to congenital duodenal stenosis should appear only after several years of life and then grow progressively worse is difficult to understand. It is possible, however, that in the process of growth the constricted portion may not have increased in diameter in proportion to the rest of the duodenum, so that the *relative* degree of obstruction was increased. Then, too, as the obstruction began to affect the patient's nourishment, dilatation and ptosis of the stomach were undoubtedly increased and, in turn, tended to aggravate the stasis, thus producing a vicious cycle.

It is of interest to compare the history of this case with those of five cases reported by Harris¹⁷ of constriction of the duodenum by what he considered to be a congenital fold of peritoneum derived from the lower border of the embryonic ventral mesogastrium. This fold extended from the gallbladder across to the mesocolon in such a way as to constrict the duodenum at the second portion. The duodenal wall itself was normal, the lumen of normal size, and the condition was relieved in each case by dividing the constricting fold. The histories of his cases were similar to that of our case—characterized by their chronicity, symptoms beginning in youth or early adult life and growing progressively worse, with periods of improvement but not of complete freedom from distress. They presented rather indefinite epigastric distress, often with a few attacks of quite sharp pain. They differed, however, from the case here reported in that vomiting was not a characteristic symptom. The point of interest is that, if Harris is right in considering the condition to be congenital, his cases show the possibility of congenital constrictions of the duodenum causing symptoms which do not begin until after several years of life.

SUMMARY

1. Congenital duodenal stenosis is a rare condition, almost invariably fatal in infancy.

2. A case is reported in an adult, characterized by a history beginning in adolescence before the usual ulcer age and growing progressively worse, the symptoms and findings indicating benign pyloric stenosis.

16. Moynihan: *Ulcer of the Duodenum*, Ed. 2, 1912, pp. 317 and 428.
17. Harris, M. L.: *Constrictions of the Duodenum*, *THE JOURNAL A. M. A.*, April 18, 1914, p. 1211.

Influence of Digestion.—A light supper, a good night's sleep and a fine morning have often made a hero of the same man who, by indigestion, a restless night and a rainy morning would have proved a coward.—Chesterfield.

PNEUMOTHORAX OF UNCERTAIN ETIOLOGY*

ARTHUR H. TERRY, JR., M.D.

NEW YORK

Pneumothorax is said to be spontaneous or natural when there is no evident external or internal cause. There must be a cause, but in many cases it is difficult to find and impossible to prove. The report of the following case very well illustrates these points:

History.—The patient is a man, aged 25, who has been under my observation for three years with several routine physical examinations. Nothing abnormal has been detected in heart or lungs. He was always considered a healthy, active young man. His father died of heart trouble several years ago; his mother is living and well. He has no brothers or sisters. There has been no tuberculosis or cancer in the family.

His habits are good. He has never had syphilis. He smokes a pipe four or five times daily and takes an occasional glass of beer. There has been no recent change in weight or mode of living. He has had a few colds lasting only a few days, and also some very slight digestive complaints. There is no history of infectious diseases. There has been no chronic cough, loss of weight, continued fever, night sweats or suspicion of tuberculosis. The patient complained one year ago of some pain and tenderness directly over the ensiform cartilage. This lasted three weeks unassociated with cough or fever, and was diagnosed as a strain.

Present Illness.—On the day previous to examination the patient took his usual cold shower, and without any unusual muscular or respiratory effort felt something "give way" or "grow loose" in his episternal region, inside of him. This gave him a queer indescribable sensation as of something having gone wrong. It caught him to draw a deep breath, and full inspiration was attended by a short, dry, expiratory cough. He went to bed directly, but had to lie perfectly quiet, any change in position being uncomfortable. If he attempted to rise it caught him in his back like a lumbago. He did not consider himself seriously ill, and did not feel sick; yet he believed that something had gone wrong inside of him. There was no dyspnea, nausea, chill, fever or sweat, and the patient was comfortable when quiet.

Physical Examination.—The temperature was 98.6; pulse, 80; respiration, 20. Inspection revealed a well developed young man, not sick and yet worried, somewhat pale and anxious. There was no dyspnea or cyanosis when he remained quiet, but rather marked dyspnea on the slightest exertion. The left chest was fuller than the right, the intercostal spaces bulging rather than concave, and no expansion. The right chest was normal in shape and respiratory excursion. There was no visible apex impulse to the left of the sternum but some cardiac impulse in the substernal notch. The apex impulse was not palpable. Fremitus was absent over the entire left chest, and normal over the right chest. There was no expansion of the left chest, and 1 inch expansion of the right chest. Percussion revealed a dull tympany from the apex to the base of the left chest, with no area of cardiac dullness. The right chest was normally resonant, with cardiac dullness extending 2 inches to the right of the sternum in the fourth intercostal space. Breath sounds were inaudible around the base of the left chest. About the middle of the left chest there was amphoric breathing with a metallic echo. Coin sound through the left chest gave a typical metallic click. There were no râles. Whispered voice over the entire left chest had the same amphoric character as breath sounds. The right chest was normal. There were no succussion and no metallic tinkle. Roentgenoscopy revealed the left chest full of air, the lung being displaced upward and inward, and the heart displaced slightly to the right. This slight displacement of the heart to the right did not accord with the entire absence of cardiac dullness to the left of the sternum.

* Read before the Washington Heights Medical Society, April, 1915.

Subsequent History.—One week later: Temperature, pulse and respiration have always remained normal. The patient has felt daily improved. Pain on sitting up has become less noticeable. Cough on full inspiration is less marked. Physical examination has showed continuation of air in left chest, though amphoric sounds in upper chest have become fainter and fainter. A few subcrepitant râles have been heard from time to time in the fourth and fifth intercostal spaces just outside the nipple line. These entirely cleared. There is still no appreciable expansion of the left chest, and cardiac dullness remains to the right.

Two weeks later: Temperature, pulse and respiration have remained normal throughout the week. Left chest has regained an expansion of one-half inch. Breath sounds have gradually returned to the left upper chest anteriorly and posteriorly, but are still faint with bronchovesicular character below. Cardiac dullness is now elicited 3 inches to the left of the midsternal line in the fifth intercostal space, and heart sounds are now audible to the left of the sternum instead of on the right, as previously.

Three weeks later: Patient is feeling about normal. Roentgen and physical signs show normal chest except for a very small amount of air in left costophrenic chink.

Four weeks later: Lungs are absolutely clear. Heart is normally situated. Patient is feeling fine. There is no dyspnea or cough.

Five weeks later: Patient is normal.

Six months later: Patient is normal.

COMMENT

The etiology of pneumothorax in this case must have been one of the following conditions:

1. Generation of air within the pleural chink.
2. Entrance of air from below the diaphragm or from the esophagus.
3. Entrance of air through the chest wall.
4. Entrance of air through the lung.

In order to be generated within, there must be air-forming organisms present, in which case there would be some fluid and some infection. Physical signs and roentgenoscopy show no fluid, and symptoms show no infection. The sudden onset of symptoms would also be against infection.

Air from below the diaphragm or a hernia would show in the roentgenogram and not heal quickly. Esophageal lesion would be detected in the same manner.

Air through the chest wall would necessitate some injury such as a stab wound. This case shows none.

There remains as the causative factor air from the lung. There must, then, have been a break in the lung surface. This might have been caused by injury, deformity, neoplasm or inflammation. Deformity or a neoplasm would have persisted. Injury to the surface of the lung would have been due to so great a difference between intrapulmonary and intrapleural pressure as to cause rupture. This would not have been sufficient to rupture the lung unless there were some structural weakness brought about by some inflammatory surface lesion. Such a lesion might have been tuberculous or nontuberculous. Against tuberculosis are the following:

No history of tuberculosis in the family, no previous history of tuberculosis in the patient, no subsequent history of tuberculosis in the patient, no symptom or localized sign of tuberculosis, no sign in the roentgenogram, and the acute condition of the case. This, therefore, leaves as the cause some inflammatory lesion of the lung surface, nontuberculous probably, very small with pin point rupture and prompt closure.

MECHANICS

The lungs are suspended within a noncollapsible chest wall. They tend to collapse because of their elasticity. This collapsing force equals about 4 mm. of mercury at the end of expiration and about 8 mm. of mercury at the end of inspiration; on forced inspiration it may fall to 30 mm. of mercury. So long as the glottis is open, allowing free communication with the outside air, there is no greater variation of pres-

sure within the pleural spaces; when the glottis is closed, as in coughing or sneezing, then intrathoracic pressure is increased; also when the glottis is closed during inspiration, then intrathoracic pressure is lessened. That the normal lung will stand considerable variation in pressure may be demonstrated by forcibly inflating the excised lungs of a dog with a pair of hand bellows.

CONCLUSION

On taking his cold shower the patient may have suddenly taken a deep breath, thus lowering the pressure within the pleural space, rupturing a small point of the lung surface. This may also have been brought about by sudden coughing or sneezing. Inasmuch as the variations in pressure are not sufficient to rupture a normal lung, there was probably a small focus of nontuberculous softening on the surface of the lung. While there was prompt healing of the communication between the intrapleural and intrapulmonary air, it does not seem correct to term the case spontaneous pneumothorax, any more than that a fire of unknown origin should properly be called spontaneous combustion.

411 Convent Avenue.

A CASE OF PHLEBITIS MIGRANS *

CARL A. HEDBLUM, A.M., M.D., SHANGHAI, CHINA

A married woman of 26 was in her fourth pregnancy in five years. The first two babies had died of marasmus; the third was living and well. The husband was in good health. There had been no known tuberculosis or cancer in the family. Except for measles and scarlet fever in childhood, and malaria five years before, the patient had never been ill. General physical examination showed normal findings excepting for rather marked anemia.

The patient did not menstruate between the last two pregnancies. She presented herself for admission one week before the onset of labor because the "waters had broken." There had been only a little dribble, but it continued in small amounts until labor set in.

At admission the temperature was 99.2. Labor pains had begun three hours before. The abdominal tumor corresponded to that of an average sized baby in cephalic presentation. Only vaginal examination was made seven hours after onset of labor. The cervix was found to be three fourths dilated, and the vertex presenting in the occipitolevo-anterior position. The labor progressed without incident, and a 7¼ pound baby was born at the end of the twelfth hour by normal mechanism. No laceration of the perineum occurred. The placenta came away after ten contractions. The uterus was atonic, and in spite of ergot and massage there was rather more than the average amount of bleeding, but the pulse did not go above 90.

The temperature remained between 99 and 100.2 for the first ten days of an otherwise uneventful convalescence. On the eleventh day the patient had a drenching night sweat, and there was a sharp rise of temperature to 104, pulse 110. The breasts were normal, the fundus well down and not tender. The white count was 17,000, hemoglobin 60 per cent. There was marked tenderness and some induration over the course of the right saphenous vein, and the leg soon became swollen and edematous. Treatment consisted in elevation, use of the ice bag, absolute rest, etc. After three days there was a marked drop in temperature and pulse, and although the tenderness and swelling extended progressively down to the foot, there was a gradual but steady improvement until the beginning of the fourth week. At this time the temperature again rose sharply to 104.5, pulse 135, and marked tenderness, induration and swelling developed in the right thigh. The patient was nauseated a great deal during the next two

* From the Hospital of the Harvard Medical School of China.