words, pus. Besides these there were places here and there scattered through the lungs, most marked in the lower lobes, which were areas grayish-red to dark brownish red, resistant, and which could very well be called bronchopneumonia. So that in these lungs we found in the background influenza and, added to that, bronchopneumonia and abscesses,—a very striking and interesting picture.

DR. CABOT: An empyema?

DR. RICHARDSON: If you wish to call it so.

DR. CABOT: Everybody is right!

DR. RICHARDSON: The circulatory system generally was negative.

The liver weighed 1565 grams, was pale brownish red with engorged vessels. The spleen was plump and dark brown red, not particularly soft, yet there was in this case definite infection.

DR. CABOT: How big was the biggest abscess that you remember seeing?

DR. RICHARDSON: I did not put the measurements down, but we will say the largest was 7 cm. across. Some rested up underneath the pleura and over them where they came to the surface we found exudate. Then there were some along the bases with exudate and some fluid.

DR. CABOT: Does that represent the whole consolidated area, or just the purulent portion?

DR. RICHARDSON: That is the whole. We presume it started as a bronchopneumonia.

DR. CABOT: The abscesses came as a complication and termination of pneumonia. You spoke of this all through as "*influenza.*" You do not mean by that to suggest any adherence to the bacillus of Pfeiffer?

DR. RICHARDSON: No; I do not follow any one in that. I do not know what the cause of influenza is. They find the so-called influenza bacillus, but we do not bow down to it.

DR. CABOT: I think the majority of physicians today pay no attention to the bacillus of Pfeiffer. Pfeiffer himself has said that it does not amount to much. But influenza is a definite entity. Post mortem the distinguishing thing is the presence of a lot of blood in the trachea and bronchi and everywhere. In our hospital abroad we saw many of these people spit blood. More people had nosebleed and spit blood in our influenza epidemic than in any disease that I ever saw. Is there any record of cultures post mortem?

DR. RICHARDSON: For some reason none were made, or they were contaminated.

DR. CABOT: There has been an enormous amount of work on the bacillus of influenza since the war. Everybody thought they had it. We thought so in our unit. But nobody knows it today.

DR. E. L. OLIVER: Is that diphtheritic membrane common in influenza?

DR. RICHARDSON: Not so that we put it down as a diagnosis. I do not know whether there was any question of diphtheria in this man.

DR. SAMPSON: No, there was no question. There was nothing in the throat.

DR. RICHARDSON: Do not mistake this term "diphtheritic." It simply means a membrane of degenerate necrotic material and fibrin. It may be associated with diphtheria or with cholecystitis or cystitis, etc.

DR. CABOT: It is a bad term. We should get rid of it.

DR. MERRILL: May I speak of the apparent discrepancy in the pathological picture as Dr. Richardson sees it and the x-ray plate? This man was very sick in the Emergency Ward, as I understand it, and the x-ray examination was made before he was admitted to the house, on the 26th. He died three days later, and the findings are separated by that interval. Of course this was a very sick man with a very rapidly acute infectious process, and the picture can change very considerably in that length of time. We find more marked conditions in the lung and a small amount of fluid in the pleural cavities. 150 c.c. would be almost invisible anyway, and at the time that plate was taken probably that did not exist.

DR. FOSTER: May I ask about the blood vessels in this sort of lung?

DR. RICHARDSON: They are engorged.

DR. FOSTER: But not thrombosed. One of the students asked me the other day about the occurrence of primary infections in the lungs and the occurrence of endocarditis. The blood gets directly back to the heart,—why are not the valves involved? They are in the other direction often.

DR. RICHARDSON: When we have an infection, especially with the staphylococcus, the septic pneumonias,—that begins as an infarct because the bacillus plugs up the vessels. But here there is no particular evidence except in some of the areas of bronchopneumonia, where they were a little resistant. Those vessels might be plugged by the infection, but there was no visible thrombosis.

A PHYSICIAN: You believe that the staphylococcus was a secondary infection? It is inconceivable that a primary staphylococcus could give a picture like this.

DR. RICHARDSON: It would not be very much unlike this except that it would not have the influenza picture. That added element shifts the whole thing.

CASE 9432

AN American schoolgirl of thirteen, entered March 9, 1910.

F. H. Her mother died of tuberculosis two years after the birth of the patient.

P. H. She had a few sore throats with removal of adenoids several years ago. Three years ago she had a bad attack of chorea. She had always been very nervous.

P. I. Three weeks before admission she began to have twitching, which March 8 became much worse and in the evening very violent. For two days she had had slight cough.

P. E. A well-nourished girl unable to sit in a chair, screaming at times, with violent choreiform movements of all the muscles. At times she had to be held in bed. Left tonsil enlarged. Apex impulse of the *heart* best felt in the fifth space in the anterior axillary line 6 cm. outside the nipple, 13 cm. from midsternum. No enlargement to the right. Action rapid. A faint systolic murmur at the apex transmitted to the axilla. At the base sounds of an entirely different quality. In the pulmonic area a late systolic murmur with a loud sharp pulmonic second greatly accentuated and louder than the aortic second, but not definitely made out because of the activity of the patient. Pulses of fair volume and tension. *Lungs, abdomen, pupils* and *reflexes* normal. *Genitals* and *extremities* not recorded. Skin of back and exposed areas reddened and roughened. A skin consultant pronounced the condition irritation due to constant movements.

T. 99.8°-105°; continuous general rise after March 10. *P.* 119-151. *R.* 29-58. *Urine.* ℥ 9-29. Sp. gr. 1.012-1.026. Cloudy at all of four examinations, the slightest possible trace of albumin at the last, slight acetone at two. *Blood.* Hgb. 75 per cent., leucocytes 17,000, polynuclears 85 per cent.

The patient continued to be very violent and was not quieted except for periods of half an hour to an hour by any of the sedatives used until large doses of veronal made her sleep most of the time for thirty-six hours. After this the motions were easily controlled by morphia. March 16 the heart seemed larger than at entrance. During the next two days the jerks increased, and the patient became quite cyanotic and had a good deal of cough and pain in the chest. A pleural rub developed over the left chest and also a to-and-fro cardiac rub loudest over the area of relative dullness and hardly heard over the area of flatness. March 19 there was some dullness in the middle of the left back, with a moderate number of fine respiratory crackles and a dry rub over the right back. The heart apex impulse became more forcible and localized, now in the fifth space as far out as the anterior axillary line. The pulmonic second sound seemed louder.

March 19 the patient became suddenly worse and died.

Discussion

BY DR. RICHARD C. CABOT

NOTES ON THE HISTORY

We know that this girl died at the age of thirteen and that her illness lasted only a little more than a month. What acute fatal diseases are most common at this age? Of course, the exanthemata are the commonest, but not ordinarily fatal. Tuberculous meningitis is common and fatal. Other forms of tuberculosis at this age are practically never fatal within a month. All the neoplasms except brain tumor are very rare. The affections of the ductless glands and the different forms of anaphylaxis which often show themselves at this age represent chronic diseases and are therefore out of consideration. Accidents, especially automobile accidents, which are terribly common in children, deserve a word of mention but no consideration. Heart disease of the rheumatic type and the variety of nephritis often due to the same organism are moderately common, representing one or two per cent., perhaps, of the population of this age, but as a rule they do not kill so early. Certainly then the commonest menace to life characterized by a brief illness at this age is some form of infection. That is inevitably in our minds as we begin the study of the case.

It may be assumed that the patient was exposed to tuberculosis during the illness of her mother. Nothing is said of a removal of the child from the mother's presence. But in the past history there is nothing to suggest tuberculosis in this patient. On the other hand, there is definite evidence suggesting streptococcus infection both in the form of sore throats and of chorea, and in the present illness it is obvious that this latter disease is the main cause of her sufferings. Hysteria might cause such violent twitching. But if the history is correct it would show other manifestations of the same disease previous to three weeks ago. Organic brain disease with incoördination and twitching shows itself much earlier, if it occurs at all at this age. The various intoxications and industrial poisons which may cause tremor and perhaps twitching are hardly to be considered, as there is no mention of any such exposure.

NOTES ON THE PHYSICAL EXAMINATION

In the physical examination there seems no reason to doubt that the movements of the patient are due to chorea. As would be expected in so severe an attack, the heart is evidently involved, the most important evidence of its disease being enlargement. The systolic murmurs heard at the apex and at the base might well be present in any infectious disease, without leading us to assume any endocarditis. In this particular disease, however, endocarditis is so

common that we may picture to ourselves a collection of minute reddish granulations dotted along the mitral valve a few millimeters from its free edge, with possibly the same appearances on one of the other valves, though the mitral is much the most often affected. The loudness of the second pulmonic sound at her age is very difficult to settle as pathologic.

Otherwise the physical examination shows nothing of importance, though the leucocytosis and the rise of temperature go to confirm the idea of a streptococcus endocarditis.

The treatment of a case of this kind offers great difficulties. If one gives enough sedative to control the violent movements, one upsets in a dangerous way the normal functions of the body. If one does not give this sedative the patient may die of exhaustion, even when endocarditis is absent or plays a minor part. In the present case apparently the motions were controlled more easily than is always possible.

During the patient's stay in the hospital there developed additional physical signs pointing to acute pericarditis and acute pleuritis, and also to some pulmonary lesion, as indicated by dullness and fine crackling râles. This pulmonary condition may be a bronchopneumonia due to the streptococcus or the pneumococcus, or possibly to the development of a tuberculosis started through contagion from the mother but hitherto latent.

DIFFERENTIAL DIAGNOSIS

Our impression is that this patient died not so much of exhaustion as of infection, and as we have evidences of infection in the pleura, lung, pericardium, and probably in the heart, it is not surprising that the patient was overwhelmed.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Acute chorea.
Acute endocarditis.
Acute pericarditis.
Mitral regurgitation.
Dry pleurisy.
Bronchopneumonia (?)

DR. RICHARD C. CABOT'S DIAGNOSIS

Streptococcus infection.
Chorea.
Acute endocarditis.
Acute pericarditis (?)
Acute pleuritis (?)
Bronchopneumonia or tuberculosis of the left lung.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesions*

(Acute chorea.)
Fibrous and verrucose endocarditis of the aortic valve.
Verrucose endocarditis of the mitral valve.
Serofibrinous pericarditis.

2. *Secondary or terminal lesions*

Hypertrophy and dilatation of the heart.
Chronic passive congestion, general.
Hydrothorax, double.
Tuberculosis of the upper lobe of the left lung.
Status lymphaticus.

3. *Historical landmarks*

Foci of obsolete tuberculosis in the bronchial lymphatic glands and lungs.
Chronic pleuritis.
Chronic appendicitis.

DR. RICHARDSON: We were not permitted to examine the head.

The appendix showed a well-marked stage of chronic appendicitis.

Each pleural cavity was half full of thin, pale fluid.

The right lung was bound down generally by old adhesions, and the left lung presented a few scattered old adhesions, some in the region of the apex. The trachea and bronchi contained a moderate amount of dirty yellowish mucopurulent material.

The thymus gland was still present and weighed 12 grams. The bronchial and mediastinal glands, generally, were more or less enlarged and pigmented, and three of the bronchial glands on section showed small fibrocalcareous masses.

The tissue of the right lung generally was rather leathery, dark salmon red, and yielded a moderate amount of dark red, frothy fluid. In the substance of the middle portion of the upper lobe there was a small fibrocalcareous mass situated just beneath the pleura, which was slightly retracted over it. In this lung elsewhere there was no good evidence of tuberculosis of any sort. The upper two-thirds of the upper lobe of the left lung was resistant and lumpy to the touch. In the substance of the upper part of this lobe there was a small cavity 2 cm. in diameter lined with a grayish yellow membrane 1 mm. thick and contained a small amount of caseo-purulent material. At one point in the wall of the cavity a small bronchus opened into it. The lung tissue about the cavity showed fibrosis and induration, and within it were several small fibrocaseous and fibrocalcareous masses, and in their region were numerous mi-

nute to small discrete and confluent tubercles. These tubercles extended pretty well down into the substance of the middle portion of the lobe. Just beneath the pleura on the upper part of the anterior lobe there was a very small fibrocalcareous nodule. The tissue of this lung elsewhere was generally leathery, dark red, and yielded a moderate amount of dark reddish, frothy fluid.

The pericardium contained a considerable amount of pale, cloudy fluid supporting fibrinous shreds and masses. The vascular and parietal layers were coated with a thin layer of membranous fibrinous material which weakly bound the two layers together. Here and there on the pericardium there were discrete and confluent dark reddish hemorrhagic spots and areas.

The heart weighed 257 grams, enlarged. The myocardium was negative. The cavities showed some enlargement. The valve circumferences were: mitral 9 cm., aortic 6 cm., tricuspid 10½ cm., pulmonary 6½ cm. The mitral curtain showed scattered along its free margin an irregular row of reddish coxcomb-like granulations. The curtain elsewhere showed but little if any fibrous thickening. The aortic cusps presented a moderate amount of diffuse fibrous thickening which slightly contracted and deformed at least two of the cusps. Along the free margin of the cusps there was an irregular band of grayish granulations. The other valves were not remarkable. The coronaries were free and negative. The aorta, however, showed in a few places fibrous streaks and areas in an extent not to be expected at this age. The great branches were not remarkable.

The spleen showed a dark red, elastic tissue with prominent follicles.

The kidneys weighed 252 grams and showed passive congestion. The gastro-intestinal tract was negative except that the solitary and agminated follicles showed marked prominence.

The retroperitoneal and mesenteric lymphatic glands were moderately enlarged, up to 2 cm. in greatest dimension.

DR. CABOT: It is of interest to note that there was a fibrous and therefore chronic endocarditis on the aortic valve in addition to the acute process there and on the mitral. This means that at previous time, very possibly in the attack of chorea three years ago, the aortic valve was involved. The chronic pleuritis also gives us reason to believe that the tuberculous process had extended beyond the bronchial lymphatics at an earlier time. Indeed this seems to be proved by the presence of obsolete foci of tuberculosis in the lungs themselves.

One may speculate a little perhaps upon the question whether this old tuberculosis, presumably acquired in her second year, was lighted up by the attack of chorea, or whether the chorea itself represents an infection made possible by diminished resistance, itself a result of tuberculosis. I remember another case with exactly this association of an acute fatal chorea and acute pulmonary tuberculosis in a patient of this age, when pulmonary tuberculosis is relatively rare. It would be interesting to collect cases of this association and see if one could trace the order of development so as to determine whether the chorea determines the outbreak of tuberculosis, or *vice versa*.

What relation, if any, has the condition of status lymphaticus found here at necropsy to the rest of the lesions? We have ordinarily supposed, on rather insufficient evidence, I think, that the presence of status lymphaticus makes any other disease or injury, such as operative insult, much more serious. But one needs to trace further the relation between this mysterious condition and the two infections which were present in this case.

Since no organisms were demonstrated in this case except those of tuberculosis in the lung, it might be argued that the lesions in the heart and pericardium were really manifestations of tuberculosis. Against that we can merely say that from their morphology it is reasonable to suppose that they represent another infection, since we often see such lesions in connection with streptococcus disease, and do not so far as I know ever see them demonstrated to be of tuberculous origin. That is, we never find tubercle bacilli in the vegetations on the valves in such case. Until such are demonstrated we may well refuse to believe that tuberculosis is ever a cause of endocarditis.

CASE 9433

AN American barber of sixty-two entered July 21, 1923, complaining of pain in the stomach.

F. H. Unimportant.

P. H. He had always been well and strong except for gonorrhea at twenty-two and tape worms at thirty-two.

P. I. A year and a half before admission he began to have pain under the ensiform radiating to the back, steady, and "burning like fire." It occurred every day, sometimes in the middle of the forenoon or the middle of the afternoon, sometimes at night. Because of it he gave up working altogether two months and a half ago. It was severe enough to make him curl up or walk the floor. It was relieved by pressing his hand to his stomach, eating a bowl of corn flakes and milk, taking baking soda, which gave immediate relief for several hours, or applying Sloan's liniment or a hot towel. During the