Case Records
of the
Massachusetts General Hospital

EDITED BY RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.

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ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

CASE 9431

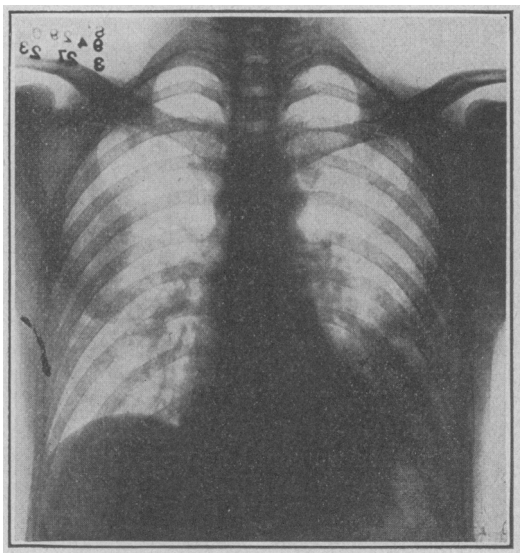
AN American student of twenty-four entered March 26, 1923, complaining of pain and dyspnea.

F. H. and P. H. Not recorded.

P. I. On the afternoon of March 23 the patient had a severe cold with moderate headache and backache. He felt quite weak. He stayed in bed until noon next day and remained in his room all day. March 25 he stayed in bed all day and took aspirin, ten grains every four hours. This gave considerable relief to the headache and backache. That afternoon he noted dry unproductive cough for the first time. He felt very weak and thought he had fever. About four o'clock he had a chill that made him shake considerably and hunched him up in a knot. At three o'clock on the day of admission he awoke with severe pain just below the ensiform associated with and aggravated by unproductive cough. Breathing was difficult, and deep breathing aggravated the pain. A physician applied adhesive plaster to his chest with great relief to the pain.

P. E. (Because of his condition the examination was incomplete and unsatisfactory.) A fairly well developed and nourished, acutely ill, highly nervous young man wrapped up in several blankets, shivering. Breathing rapid, shallow, labored, painful. Skin dry and hot. Very slight pyorrhea. Throat somewhat injected. Tonsils absent. Frequent paroxysms of racking cough which caused excruciating pain chiefly under the lower sternum, with production of small amounts of pinkish, thick, slightly purulent sputum. Apex impulse of the *heart* seen and felt in the fifth space 8 cm. to the left of the midsternum in the midclavicular line. No enlargement to percussion. Sounds rapid, regular, of good quality. Soft systolic murmur at the apex, not transmitted. *Lungs.* Expansion greater on the left. Diminished breathing at the right back from the lower third of the scapula down. No definite dullness or change in character of breath sounds made out. No râles or friction rub heard. *Abdomen* negative except for scar of hernia operation on the left side. *Genitals, extremities, pupils* and *reflexes* normal.

T. 103.1°-105.5°, rectal. *P.* Steadily rising, 92-145. *R.* 28-58. *Urine.* ℥ 24 on the one occasion recorded. Sp. gr. 1.032. No albumin or sugar, two or three leucocytes per high power field and a few hyalin casts. *Blood.* Hgb. 90%. Leucocytes 14,500-3,700. Polynuclears 79%.



9431

Area of dullness at left base occupying the costophrenic angle and obscuring the heart apex and diaphragm. The outline of the diaphragm below is suggested by the gastric gas bubble seen beneath it. Upper limit of area of dullness is indefinite and has not the characteristic position and outline of fluid. In the lung above it and diminishing upward are small areas of mottled dullness extending to the lung root, which is increased in size and density. On the right side scattered throughout the lower two-thirds of the lung field, most marked in the lower half, are numerous shadows of mottled dullness.

Two *blood cultures* negative. *X-ray.* (See illustration.) "The findings are probably due to a pathological process in the lung parenchyma, most marked in the left base. . . . Shadows in the remainder of the lung fields are suggestive of a scattered infectious condition throughout both lungs probably of the same nature as the denser area noted. There is no evidence of free fluid, but there may be a small amount of fluid in the left base."

The day after admission Dr. W. H. Smith noted, "Very ill. Dyspnea with pain on inspiration. No focus found for aureus infection. No costovertebral tenderness. No palpable kidney. Abdominal muscles held rigid, probably from pleural pain. Two small hyperemic papules, one on the left palm, the other on the right index finger. The systolic apical bruit appears harmless. . . . At the right base some dullness, suggesting fluid rather than consolidation. Serious outlook." That afternoon the house officer could make out no abnormal signs in the lungs. The patient was raising considerable amounts of sputum colored uniformly with

blood and becoming more and more purulent with each examination. March 28 the left back showed dullness, increased fremitus and voice, loud bronchial breathing and a very few râles from the lower third of the scapula down. The right back showed slight dullness, distant breathing and occasional medium râles. The sputum was much more purulent. A culture showed a profuse growth of staphylococcus aureus.

The condition the next morning was much the same as the day before. The patient gradually grew weaker, and that evening died.

Discussion

BY DR. RICHARD C. CABOT

NOTES ON THE HISTORY

Ordinarily if the pain is in the chest and we have the combination of pain and dyspnea we cannot help registering a guess that pneumonia is what we are dealing with. Here we have not enough to go on.

The record does not say anything about his being of a race we could not communicate with, so we may assume that his family and past history were not recorded because he was so sick.

As we read on it seems to me that our first guess is still in our minds and is somewhat reinforced by what we learn. The pain is in a rather queer place. We do not usually have epigastric pain in pneumonia, but we may have it. It is usually on one side, either in the iliac region or below the ribs. Still I do not see why it should not be in the epigastrium. On the other hand we are always liable to be fooled by a disease which starts off like an acute respiratory infection but is really a general infection. A gall-bladder trouble may start in this way. In retrospect we are then accustomed to say that we did not take a proper history and that there really must have been pain over the gall-bladder at the start. But I am convinced that gall-bladder disease, pancreatitis, and I suppose less common things like ulcer, begin as a general septicemia without any localization until several days after the onset.

The things to keep in mind here are pneumonia, which I think is the first guess, and then some sort of local peritonitis with general symptoms.

NOTES ON THE PHYSICAL EXAMINATION

1. I should say that that shivering, although we do see it with subdiaphragmatic lesions, is not so common as it is with disease above the diaphragm.

2. The apex beat is where it ought to be. This is the sort of murmur to which we pay no attention whatever.

3. The lung expansion is greater on the left, so we suspect the right.

This is just the sort of physical examination that makes us cautious in saying there is anything the matter in the chest, just the sort we get when the trouble is below the diaphragm and the liver is pushed up so that when we think we are percussing the base of the chest we are really percussing the liver. The absence of râles and of any change in the breath sounds—merely their diminution—makes us wonder whether this is not liver. On the other hand the earliest sign of pneumonia, especially in my experience the influenza type, is often this diminished breathing and nothing else. But there ought to be râles very soon if it is going to be pneumonia.

As we go on we have not a thing to confirm my guess of some local peritonitis. Everything that is at all definite points rather to the respiratory tract. Pneumonia is still my first guess.

Of course the second white count, if this is pneumonia, gives us a bad prognosis.

I will ask Dr. Merrill to take charge of the x-ray plate.

Dr. Merrill: Usually we associate an area of dullness of that character in one of the costophrenic angles, obscuring the anatomical markings, with the presence of a certain amount of fluid. That is our first guess. We could see a suggestion of gas in the stomach or perhaps in the splenic flexure, which suggests the position of the diaphragm. I see no mention of a fluoroscopic examination. Perhaps he was too sick. If that had been done we could have seen if there was any excursion of that side of the diaphragm. With a certain amount of fluid the characteristic outline is an oblique line rising from some point in the diaphragm, according to the amount of fluid, upward to the chest wall. While having a suggestive shape this does not have the typical upper border. "The extreme apices were relatively clear on both sides." That is a rather important differential point against the possibility of tuberculosis. Knowing as little as we do about many of these patients when they come to us, everything must be considered. We conclude that the process involves the lung itself and not the pleura alone. Of course in any acute infectious condition involving lung or pleura the presence of any small amount of fluid is possible. But the general picture here suggests to us that the lung is involved not only in the left base but also in other parts by a scattered process of some infectious nature. That is a picture not infrequently shown in the distribution of a purulent process, essentially numerous scattered purulent foci, oftentimes simulating a metastatic condition, often from a pyemic origin. Or it might here be the extension of a process of some purulent infection from below the diaphragm causing a metastatic suppurative condition in the lung. Of course there are several possibilities. If this

were an elderly person, and not a young man, we should have to consider metastatic malignancy, and there is the possibility of an unusual type of tuberculosis. But the upper lobes being so clear, the probability of a lighting up of a chronic phthisis would be out of the question.

DR. CABOT: Malignant disease, abscess, tuberculosis, pneumonia, are the four possibilities Dr. Merrill has mentioned. In tuberculosis the point he has made is the absence of trouble in the apices. We have been over a very large number of necropsies lately. We have not had a single case of phthisis which did not start in the apices or in the hilus; we have not had any case of primarily basal tuberculosis. So if the apices are free we have not got tuberculosis.

Should you say, Dr. Merrill, that if we were judging by radioscopic appearances alone this might be malignant disease?

DR. MERRILL: It might be.

DR. CABOT: Therefore we have to combine what we see with our other knowledge of the patient before we can judge about that. Should you say those might be little abscesses?

DR. MERRILL: They might be, with the strong probability that there might be abscesses in the base, not primary but a secondary infection.

DR. CABOT: In the lung rather than outside.

DR. MERRILL: Yes.

DR. CABOT: Of course we are always thinking of empyema as complicating pneumonia, if we are thinking of pneumonia at all. But you think if there is pus here it is inside rather than outside?

DR. MERRILL: Yes. We think that from the fact that both sides are somewhat involved. From the findings in the left base alone we could not say that it was pneumonia and not empyema.

DR. CABOT: Would you say that the involvement of the lobe is probably patchy rather than lobar?

DR. MERRILL: Yes.

DR. CABOT: We leave out malignant disease by the patient's age, tuberculosis by the site of the lesion. *Abscess.* In the vast majority of cases we have a cause, we have something that starts it,—ordinarily in this part of the world a tonsil operation. Then we have ordinarily pus in the sputum. It is said here to be slightly purulent. We do not see people die so fast. I never knew anybody to die at this rate with abscess of the lung as the main cause of death. Of course there might be *abscess plus something else.* But he has been sick three days in all. With this history it is almost inconceivable to me that this is abscess. So that so far as we go I should say he had pneumonia of the patchy type of both lungs, especially at the left base.

A HOUSE OFFICER: The sputum at first contained a lot of influenza bacilli and a few staphylococci. As it became more purulent the staphylococci went up until it became almost pure pus.

DR. CABOT: Dr. Smith was, of course, looking for some source of staphylococcus infection, of which a carbuncle is the ordinary example. We have nothing here. Dr. Smith considered the possibility that some subdiaphragmatic process had worked through. We must consider that before we get through. But so far I see nothing to suggest it. In the "small hyperemic papules" he is thinking of metastatic septic emboli.

So far the clinical examination has emphasized the right base and the fluoroscope the left, which is an interesting contrast. Was this case fluoroscoped in the ward?

A HOUSE OFFICER: I believe it was on the way up to the ward that the x-ray was taken, and he was too sick to fluoroscope. He was an emergency case.

DR. CABOT: Finally we get "a considerable amount of sputum." That is of importance. Now we are getting more emphasis on the pus, more than we had in the beginning.

DIFFERENTIAL DIAGNOSIS

This pus seems to me the first point we have to consider here. A person can die with purulent bronchitis on top of pneumonia. I have been struck here in the last few years with the frequency with which Dr. Richardson reports purulent bronchitis as the outstanding infection even when there is very little in the lung itself; certainly we get that associated with pneumonia. So we can explain the purulent sputa in that way. On the other hand it seems to me that those abundant purulent bronchial exudates are not so common at the age of this patient as in older people.

The diagnosis then seems to me between multiple abscesses of the lung, which this purulent sputum strongly suggests, and a number of foci of bronchopneumonia with a purulent bronchitis and with or without an empyema on the left. Sizable abscesses of the lung are generally right-sided. There is a very large preponderance of the right side among abscesses which are produced during operations, presumably because the course of the right bronchus is so much more direct than the left. Here of course we are not dealing with a postoperative case, so the location on the left side does not prejudice us against abscesses.

A HOUSE OFFICER: On the day before death there seemed to be a great deal more dullness on the left side.

DR. CABOT: Now we have all the facts, I think. There is nothing to make us consider the abdomen so far as I see. There is nothing to make us consider the kidneys, the nervous system, or the digestive tract. It comes down to the lungs and pleura, and in the lungs and pleura it is certainly either patchy pneumonia or multiple abscesses. I still incline towards

pneumonia as either the whole or a large part of this process. I am trying not to be influenced by anything Dr. Merrill or any one else has said which tends to push me towards abscess.

The chief question seems to me as between bronchopneumonia and abscess. But I have never seen abscess kill at this rate. I have never seen people die as quickly as this with abscess. With bronchopneumonia often.

DR. LINENTHAL: I do not believe he has any pus in the pleura.

DR. CABOT: I do not think I should feel clear about that.

DR. LINENTHAL: Would you get a white count of 3000 in empyema?

DR. CABOT: Yes, if he is going down to death. I have seen it in general peritonitis with quarts of pus.

A PHYSICIAN: What would be the condition you would expect here?

DR. CABOT: Patchy pneumonia,—I think Dr. Richardson generally calls it that rather than either broncho or lobar.

DR. RICHARDSON: Yes, or focal.

A PHYSICIAN: Isn't the predominance of staphylococci in the sputum rather unusual?

DR. CABOT: I do not know enough to answer that question.

DR. YOUNG: Did he have a positive blood culture of staphylococcus aureus?

A HOUSE OFFICER: He had two blood cultures and both were negative.

DR. CABOT: Staphylococcus aureus grows pretty well in the blood stream?

DR. YOUNG: It is a worse prognosis when it is found.

DR. CABOT: I had an idea that the aureus grew better in the blood than the streptococcus. We have a tremendous number of negative cultures here ante mortem when we have been pretty sure that the streptococcus was in the blood.

A PHYSICIAN: There was no reason to suspect the staphylococcus was metastatic?

DR. CABOT: No. There was no focus known from which we could get it, and primary staphylococcus process in the lung, so far as I know, is extremely rare.

A HOUSE OFFICER: There was a sputum culture which came out aureus.

CLINICAL DIAGNOSIS

Bronchopneumonia.
Septicemia, staphylococcus.

DR. RICHARD C. CABOT'S DIAGNOSIS

Bronchopneumonia.

ANATOMICAL DIAGNOSIS

1. *Primary Fatal Lesions*

Pneumonic influenza.
Bronchopneumonia with abscess formation and hemorrhages.
Diphtheritic tracheitis and bronchitis.

2. *Secondary or Terminal Lesion*

Fibrinopurulent pleuritis.

3. *Historical Landmarks*

Slight chronic pleuritis.

DR. RICHARDSON: The conjunctivae were clear, but the skin of the face and trunk was a little sallow.

DR. CABOT: Were there any petechiae on the skin anywhere to suggest sepsis?

DR. RICHARDSON: It merely was sallow. The head was frankly negative. Rigor mortis was well marked, although there was well marked infection in the case. The peritoneal cavity, appendix, esophagus, stomach and intestines were frankly negative, except that the mucosa of the small intestine showed streaks and areas of reddening. These not infrequently accompany infections.

DR. CABOT: There was actual hemorrhage?

DR. RICHARDSON: Yes. In each pleural cavity there was about 150 c.c. of thin purulent fluid. The lungs here and there were coated with fibrinopurulent exudate. There was what was apparently thymus gland. But here again one has to be very careful, because frequently what appears to be thymus gland tissue turns out to be fat tissue with no thymic elements left whatsoever. So although he was twenty-four years of age and had a thymus gland weighing thirteen grams he had apparently no thymus tissue at all.

The trachea and bronchi and even the small branches showed blackish-red, thick, velvety mucosae and on top of that a thin layer of fibrinous dirty grayish material—in other words the character of pneumonic influenza and added to that a so-called diphtheritic membrane. The bronchial glands were slightly enlarged, blackish-red and bloody. All through, these tissues above the diaphragm were bloody.

The apices of the lungs were frankly negative. The tissue of the lungs generally ran from pale purple-red to blackish-red and was saturated with blackish-red fluid. That is the picture in pneumonic influenza. In places the tissue is almost gelatinous; it sinks in water, and yet is not consolidated tissue. Here and there were smaller and larger resistant areas more or less soft and necrotic, and in places the center material was frankly purulent, semi-fluid,—in other

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.