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HEMORRHAGIC PNEUMONITIS*†

ROENTGEN RAY STUDIES DURING THE RECENT INFLUENZA EPIDEMIC AT THE WALTER REED GENERAL HOSPITAL, TAKOMA PARK, D. C., BEGINNING OCTOBER 1, 1918

BY JOHN HUNTER SELBY

Major, M. C., U. S. A.

Chief of Roentgen Ray Section, Walter Reed General Hospital

DEFINITION.—An acute localized toxic inflammation of the cells lining the alveoli, bronchioles, and their capillaries, producing a frank pernicious hemorrhage into the air passages.

Etiology.—It is undetermined what rôle, if any, the influenza bacillus plays in the production of the specific toxin that destroys the integrity of the cells lining the alveoli and bronchioles; but there is strong presumptive evidence to support the theory that the particular toxin which would seem to have a selective action upon these cells is produced locally by organisms which proliferate within air passages in the immediate vicinity of the lesions. This conception of the process is not offered to apply to any other lesion than hemorrhagic pneumonitis.

Occurrence.—The pathological literature on influenza epidemics antedating the present is said to contain no authentic description which conforms to the gross or microscopic appearance exhibited by these lungs, and similarly, no roentgen ray appearances such as we have observed have been described.

Hemorrhagic pneumonitis was recognized by the method of serial daily roentgenographs with the standard U. S. A. bedside roentgen ray machine, and was found to be present to a varying extent in 386 of the 470 patients referred. All of these, in one form or another, presented clinical evidence of pulmonary complication. The series included 3400 films. Observation on this condition, which we have called hemorrhagic pneumonitis and which we have learned to recognize as a definite roentgenographic entity, was subsequently confirmed by autopsy protocols.

Judging from the descriptions of the autopsy findings on these influenza cases that have come to our attention during the last few months, it seems that pulmonary hemorrhage is a conspicuous factor during this epidemic in all parts of the country. However, the degree of hemorrhagic involvement and also the percentage of hemorrhagic pneumonitis, as compared with streptococcic pneumonia and other variously described pneumonias, have varied considerably in different localities.

Pathology.—The microscopic findings

* Read at the Midwinter Meeting of Roentgenologists, Atlantic City, N. J., January 25, 1919

† Authority to publish granted by the Board of Publication, Surgeon General's Office, Washington, D. G.

have been variously described, some observers laying emphasis upon confluent bronchopneumonia, others describing bronchiolitis associated with more or less hemorrhage.

Captain M. W. Lyon, pathologist to the Walter Reed General Hospital, describes the typical lung appearance as follows:

"The external appearance of a freshly removed lung presents a slaty purple color. It feels firm and airless. Along the lower and anterior edges where there are air-containing and emphysematous lobules there is a sharp line of demarcation between the slaty portion and the air-containing portion, which is pinkish gray. In some cases there may be found air-containing islands in the slaty purple solid portions. In lungs which are incompletely involved, there may be found slaty purple islands in the midst of a pinkish gray field. Beneath the pleura, over the solidified portion, in many places, there are seen red hemorrhagic spots varying in size from less than a millimeter up to 3 millimeters in diameter. These spots have a tendency to coalesce and form large red areas.

"On sectioning the lung, using a sharp knife, without exerting any pressure upon the lung, the cut sections drip great quantities of blood. The cut surfaces do not appear mottled, but are homogeneous, without roughened areas or nodules. The general appearance strongly resembles a large fresh clot of blood."

We encountered a number of cases where several lobes would show a hemorrhagic pneumonitis, while another lobe presented a typical gray hepatization. The recognition of lobar pneumonia associated with hemorrhagic pneumonitis was possible in several cases by means of serial roentgen ray films made prior to death.

Other coexisting intrathoracic complications were encountered in order of frequency as follows:

1. Interlobar pleuritis.
2. Pleurisy with effusion.
3. Mediastinal adenitis.
4. Cardiac enlargement.
5. Empyema.
6. Pericardial effusion.
7. Lobar pneumonia.
8. Diffuse mottling.
9. Plastic exudate.
10. Mediastinal empyema.

Mode of Onset.—Hemorrhagic pneumonitis usually began coincidently with, or soon after, the incidence of a secondary rise in temperature,* occurring from one to five days after the patient had run what was apparently a typical influenza course. Less frequently it appeared on the fourth or fifth day after the initial onset of influenza, where the temperature curve was atypical, in that the characteristic fall did not occur. In a few instances, it was detected the day after the patient went to bed.

Characteristic Roentgen Ray Appearance.—In the earliest stage demonstrable by the roentgenogram hemorrhagic pneumonitis is recognized as a faint filmy haze opposite the level of the lower angle of the scapula. The mesial portion of haze is partially obscured by the outer portion of the normal hilum shadow. This hazy area enlarges in all directions and frequently it is observed that the adjacent portions of the upper and lower lobe are involved simultaneously. The process may advance so rapidly as to include the greater portion of all lobes in the same side of the chest within forty-eight hours. In fulminating cases, all five lobes may become blood-logged and death ensue within forty-eight hours. Hemorrhagic pneumonitis invariably began as a unilateral process, and in 82 per cent of our cases the left lung was primarily involved. It was never seen to begin in any peripheral portion of a lobe, but invariably appeared in the region of the lung roots. It was also noted that the involvement never began simultaneously in two or more widely separated areas, but it was found to spread from the original site.

* This observation was noted early in the epidemic by Lieut. J. Harkavy and personally communicated to me. This enabled us to detect a larger number of early cases than otherwise would have been possible.

Later it may develop around the roots of a lobe on the opposite side. The peripheral portions were the last to fill with blood. The true apex and the lower borders of the lower lobes were never involved. We discovered this characteristic absence of apical costophrenic involvement early in the epidemic and requested a check by autopsy. The autopsy confirmed this, so that we were enabled to utilize the sign in detecting pleuritic involvement. In other words, we found that in every case where the roentgenogram showed a haziness or a total density over the apex or costophrenic angle, it was indicative of a pleuritic complication or lobar pneumonia. In the vast majority of cases we found pleural effusion responsible.

Course.—The hemorrhage showed a tendency to disappear within three days where the area of involvement did not reach a greater diameter than a silver dollar. In moderately advanced cases, e.g., where the invasion was arrested when the involvement included no more than the lower proximal half of the upper left lobe and the adjacent proximal portion of the lower left lobe, the condition would slowly but progressively fade away, usually disappearing by the twelfth day unless complicated by some other intrathoracic condition. If in such a case there had been a progressive clearing demonstrated over a period of several days followed by an interruption, a complication usually appeared soon thereafter, the most frequent one being a pleural effusion. This often changed into empyema.

During the height of the epidemic the cases which showed extensive involvement of all five lobes usually proved fatal regardless of whether the involvement developed slowly or rapidly. Later in the epidemic, the virulence diminished and the percentage of fatalities in extensive involvements became conspicuously less. In some cases the process of absorption of the hemorrhage was noted in one lobe while a complication would develop in the region of another lobe.

Prognosis.—Soon after these daily roentgen ray observations were instituted it was found that the cases presented certain definite characteristics by which a prognosis could be offered with much accuracy. The second or third film usually sufficed to base our predictions on. Our prognosis was made utterly regardless of the clinical data. We based it upon the rate, direction and extent of the spread, interpreted in the experience furnished by the earlier cases.

The rapidity with which the hemorrhage spread to include the lobes opposite the original hemorrhage bore a direct relation to the gravity of the case. We did not observe a fatal case in which the hemorrhage remained a unilateral condition, even though all lobes on the affected side became extensively involved.

Ordinarily if the patient survived seven days after the onset of the hemorrhagic pneumonitis, the prognosis was favorable. However, the convalescence in many cases was interrupted by one or more of the previously enumerated intrathoracic complications. Empyema was the most frequent to occur after the seventh day in hemorrhagic pneumonitis.

In our series, where the roentgen ray observations were possible early in the course of the disease, we erred in two cases where a grave prognosis had been rendered. In one, all five lobes were involved and a bilateral empyema developed subsequently. This case was interesting in that an entirely different organism was recovered from the right pleura than from the left. In the other case, the convalescence was complicated and prolonged.

Theory of Invasion.—The serial radiographic studies upon the trachea, bronchi, bronchioles, alveoli and their capillaries, interpreted in the light of the autopsy findings, support the conception that the causative organism, whatever it be, gains access through the respiratory tract. It lodges and proliferates in the lower portion of the trachea or in a main bronchus, whence the organisms are distributed by contiguity, reaching first the bronchi and

alveoli in the immediate vicinity. These individual colonies elaborate the toxin which produces local destructive change in the cells lining the bronchi, the alveoli, and also the capillary walls, the result being a frank pernicious hemorrhage. The spread is perhaps augmented by the flow or inhalation of the infected bloody exudate into other portions of the respiratory tract. The process seems to develop by spread in contiguity from the original focus in the main bronchus to the alveoli.

The serial radiographs proved that the hemorrhage invariably commenced in the central portion of a lobe nearest the hilum. Later it spread in all directions simultaneously. It is plausible to assume that when the hemorrhage becomes extensive enough all approaches to the peripheral portions of the lobe are likely to be blocked off by the presence of the rapidly accumulated blood in the distributing bronchi. If this is true, the air circulation is retarded and the organisms cannot be transported either by air or by the flow of exuded blood into the more distant alveoli. Autopsy consistently confirmed the conspicuous absence of hemorrhagic pneumonitis in the true apex and in the costophrenic borders of the lower lobes. It was also noted that the alveoli situated nearest the pleura are relatively free from hemorrhage.

This absence of hemorrhage at the apex and in the costophrenic borders, together with the delayed involvement of the peripheral portions of the lobes, strengthens the theory that the hemorrhage is due to the toxins elaborated locally. It is also obvious that the hemorrhage cannot be explained on the assumption of a state of hypostatic congestion because the autopsy findings failed to confirm hypostasis in the most dependent portions of the lower lobes. Thus it is obvious that the condition is in no way a septicemia.

We found it advisable to divide our 470 cases into the following groups:

(*a*) Hemorrhagic pneumonitis uncomplicated. These presented the typical hazy spreading shadow produced by the increased condensation. No discrete mottling was present, nor was there any conspicuous enlargement of the mediastinal glands. No pleuritic complications were present.

(*b*) Hemorrhagic pneumonitis associated with a conspicuous enlargement of the mediastinal glands, but without discrete mottling.

(*c*) Hemorrhagic pneumonitis associated with discrete mottling, but without conspicuous enlargement of the mediastinal glands.

(*d*) Hemorrhagic pneumonitis associated with conspicuous enlargement of the mediastinal glands and with a definite discrete mottling over the pulmonary area.

(*e*) Hemorrhagic pneumonitis complicated by some form of pleural involvement, pericardial involvement and mediastinal empyema.

(*f*) Adenitis unassociated with hemorrhagic pneumonitis, discrete mottling, or any other form of pulmonary or pleural pathology discernible by roentgen ray.

(*g*) Adenitis and fibrosis unassociated with other intrathoracic pathology discernible by roentgen ray.

(*h*) Adenitis without hemorrhagic pneumonitis or discrete mottling but associated with some other intrathoracic pathology.

(*i*) Discrete mottling of the pulmonary area uncomplicated by hemorrhagic pneumonitis, enlargement of the mediastinal glands, or any other intrathoracic pathology discernible by roentgen ray.

(*j*) Discrete mottling associated with conspicuous enlargement of the mediastinal glands unassociated with any other intrathoracic pathology discernible by roentgen ray.

(*k*) Mottling unassociated with hemorrhagic pneumonitis and enlargement of the mediastinal glands, but associated with some other intrathoracic pathology.

(*l*) Hemorrhagic pneumonitis associated with pulmonary tuberculosis.

(*m*) Hemorrhagic pneumonitis associated with lobar pneumonia.

(*n*) Lobar pneumonia uncomplicated by hemorrhagic pneumonitis.

(*o*) Cases in which there was definite clinical evidence of pulmonary pathology but in which the serial roentgenographs failed to confirm the presence of a pulmonary lesion.

Roentgen Ray No. 5622. Group (e). Case illustrating hemorrhagic pneumonitis of all lobes associated with pleural effusion (bilateral). This was one of the early cases which formed the basis for the investigation.

FIG. 1. Made in autopsy room. Note homogeneous density in all 3 lobes on right and both on left. Note relative clearness of both apices. The costophrenic angles are obscured by what proved to be fluid. Compare this with Fig. 2 made of thoracic viscera after removal in toto.

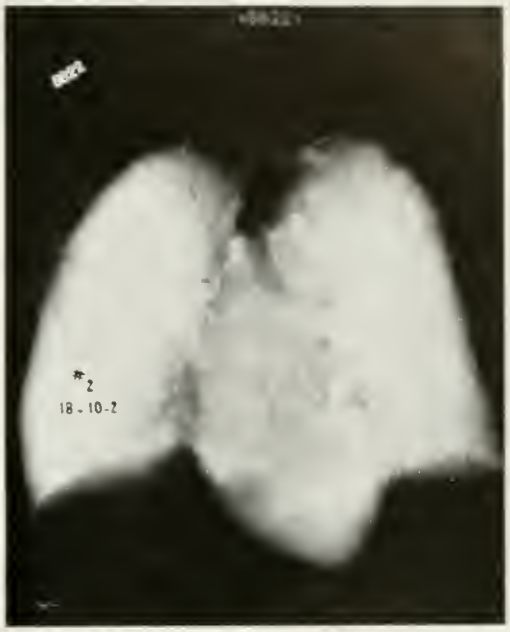

FIG. 2. The true apices are clear. The costophrenic portions of the lower lobes are relatively clear. All five lobes present a homogeneous density, which is not of such a high degree as is characteristic of lobar pneumonia.

Naturally the cases presenting a discrete mottling offered the greatest difficulty in interpretation, for we did not have preliminary plates by which an estimate might have been made of the previously existing chronic lesions; such as peribronchial adenitis, chronic parenchymatous, tubercular changes, etc. However, we were soon enabled to identify the mottling by autopsy. It was found to be due to discrete interstitial lesions, commonly held to be streptococcic in origin. In our cases the sum total of mottled types was so small as to be conspicuous.

The total number of patients studied roentgenographically was 470. Of these 82 per cent, or 386, presented hemorrhagic pneumonitis complicated or uncomplicated by one or more of the intrathoracic conditions already noted. Of the remaining eighty-four patients, thirty-three failed to show any lung pathology by the roentgenogram, although clinically there was evidence of so-called bronchopneumonia, seven were Group (*g*) (mediastinal adenitis), thirteen were Group (*i*) (discrete mottling uncomplicated); the remaining thirty-one could not be classified, as the presence of fluid completely obscured the original pathology.

Of the cases which came under roentgen ray observation early enough, the hemorrhagic process was found to appear primarily in the left lung in 82 per cent. This is a striking fact, but no less remarkable than the observation that in our ninety-one cases in which there was unmistakable evidence, the initial invasion appeared in the lower left lobe eighty-five times to six in the upper left lobe.

Group (*a*) includes a few cases where autopsy revealed a small amount of pleural fluid, which roentgenographically was not present twenty-four hours before death. Presumably this developed as a terminal process. The importance of serial roentgenograms in influenza has been amply proven in this clinic, chiefly in detecting and iden-

Roentgen Ray No. 5770 Group (*a*)

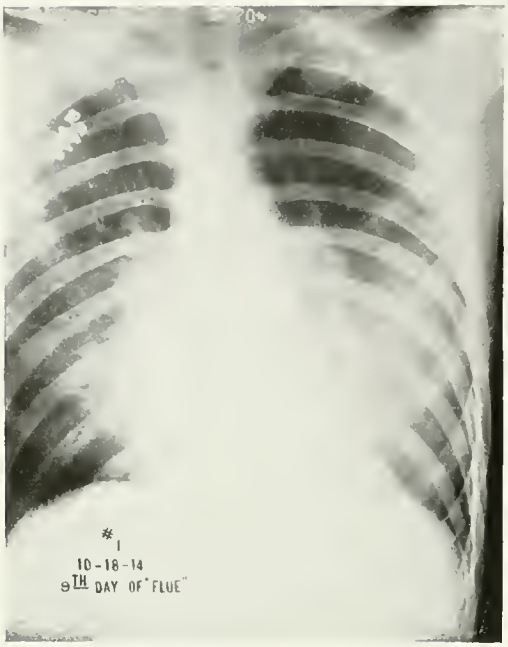

FIG. 1. The initial film reveals the presence of hemorrhagic pneumonitis in the adjacent portions of the upper and lower left lobes and also in the adjacent portions of the lower and middle right lobes. Note that the density is most intense at the hilum and fades out toward the periphery. Note the clearness of the costophrenic angles on the right and left sides.

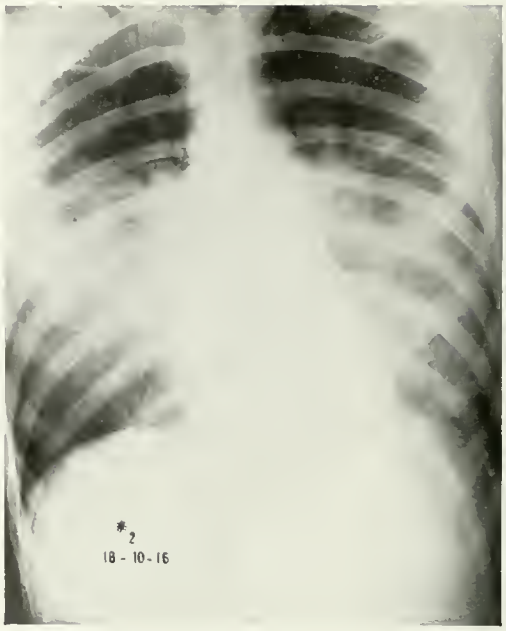

FIG. 2. Compare with Fig. 1 and note the extent of spread in 48 hours.

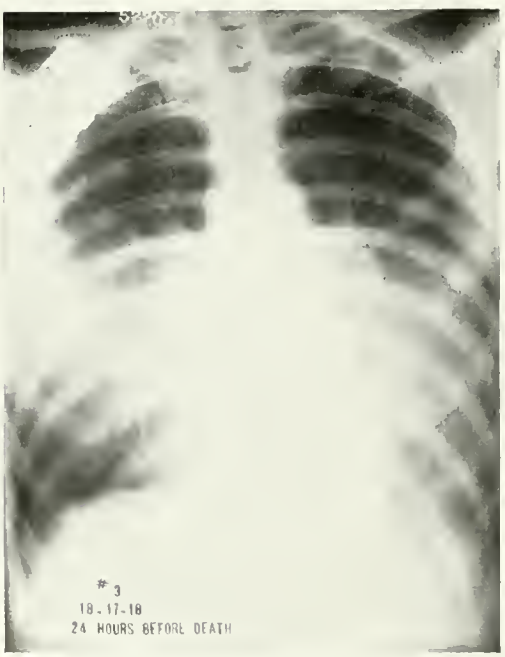

FIG. 3. The patient was too ill to hold his breath and died within twenty-four hours after this examination.

tifying pulmonary complications in the early stages.

We had no deaths from Group (*f*) or (*g*) and so far we have not seen a single instance of fatal influenza unassociated with an intrathoracic complication.

Anticipating possible criticism of this summary on the ground that the clinical, roentgen ray and laboratory findings have not been presented in concerted form, it may be stated that the clinical, roentgen ray and postmortem findings have coincided to a remarkable degree. This will be substantiated by the full report which is under preparation for the Surgeon General and for the medical history of the war.

These roentgenographic studies were stimulated by the extraordinary autopsy findings in the early cases. Authorization was promptly obtained from the Commanding Officer, Colonel E. R. Schreiner, who with Chief of the Laboratory Service, Lt. Col. Nichols, and the Chief of the Medical Service, Major B. M. Randolph, enabled us to bring this to a successful conclusion. We are especially indebted to Captain

Roentgen Ray No. 5765 Group (*e*). A fatal case.

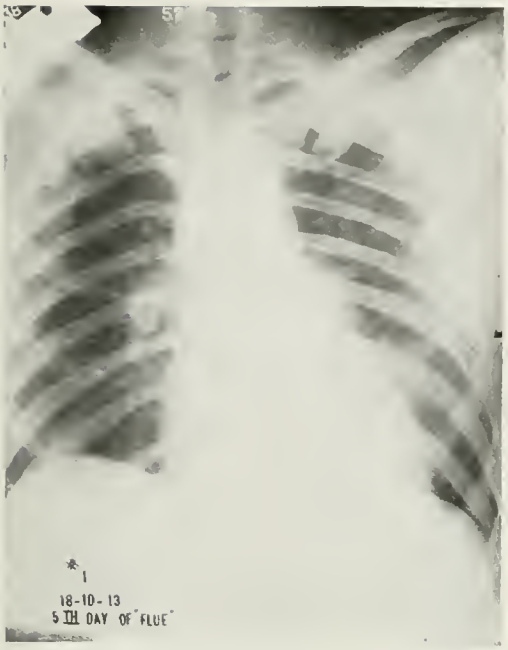

FIG. 1. Shows an early appearance of hemorrhagic pneumonitis in lower left lobe. Note area of increased density projecting from hilum. Compare with Fig. 2, taken twenty-four hours later, and note great increase on 18-10-14. Note the clearness of the apices and the costophrenic angles.

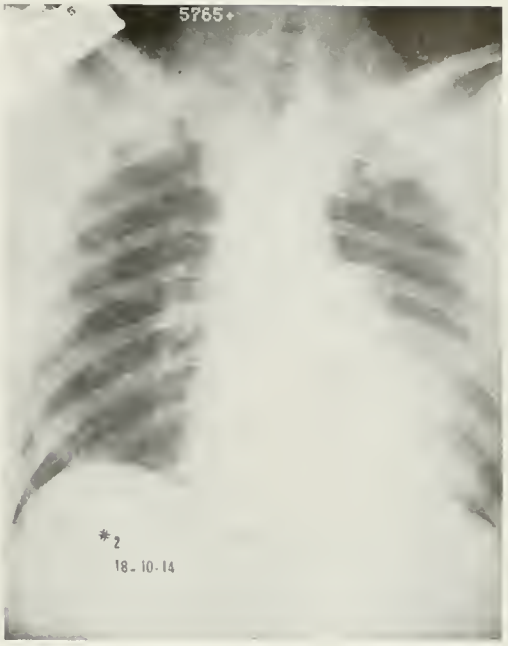

FIG. 2.

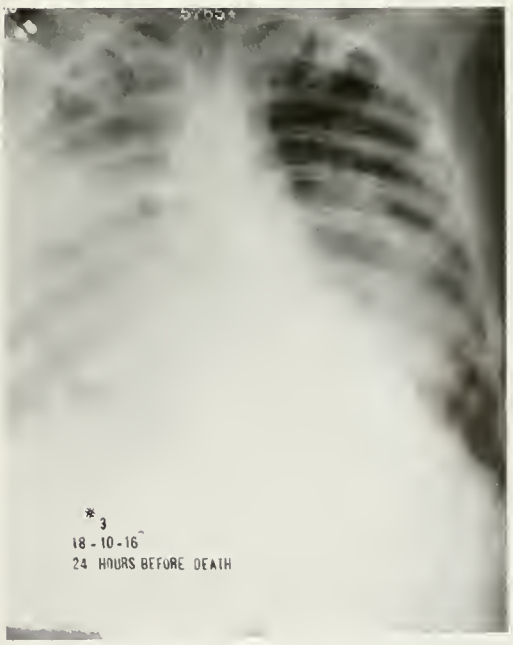

FIG. 3. On 18-10-16 shows extensive involvement of lower and middle right lobes, lower portion of upper right and adjacent portions of upper and lower left lobes. The patient was too ill to hold his breath but Fig. 3 illustrates clearness of costophrenic angles and true apex. The patient died twenty-four hours after Fig. 3 was taken.

M. W. Lyon, Lt. J. Harkavy and Lt. Adolphus Rood for their hearty cooperation. We cannot close without expressing our thanks to each and every member of the Roentgen Ray Section, all of whom volunteered to serve in the infected wards and who unsolicited cancelled their engagements that they might devote day and night to the investigation. Lt. Jamie C. Thompson was most successful in organizing the bedside squads and Lt. J. H. Hirsh was equally untiring in furthering this work.

To Major H. E. Ashbury, Roentgenologist, Army Medical School, we are indebted for his invaluable aid in placing at our disposal a sufficient number of roentgen ray technicians to form five bedside squads. The prompt cooperation of Lt. Col. George C. Johnston, Chief of the X-Ray Division of the Surgeon General's Office, enabled us to secure the necessary apparatus in the emergency.

Roentgen Ray No. 5629. Group (*e*). Demonstrating an extensive involvement by hemorrhagic pneumonitis which developed double empyema with recovery.

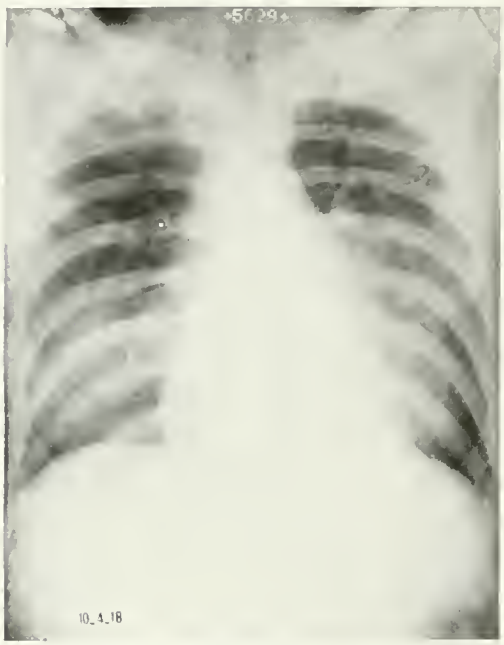

FIG. 1. Extensive hemorrhagic pneumonitis in lower right lobe, lower left lobe, and adjacent portions of upper left lobe.

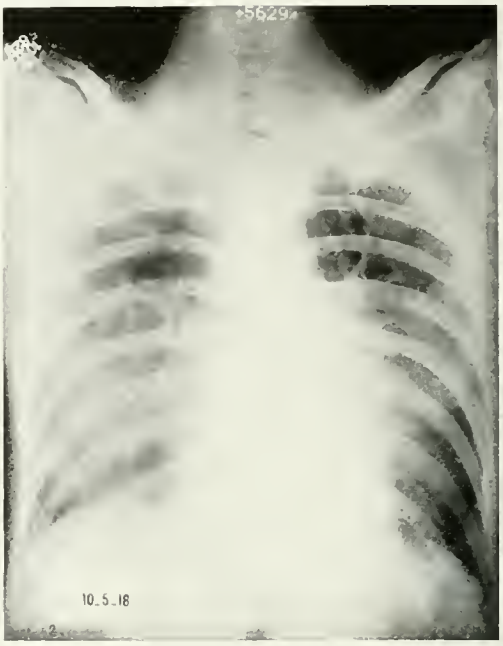

FIG. 2. Compare with Fig. 1 and note extent of spread in twenty-four hours. Both costophrenic angles remain clear.

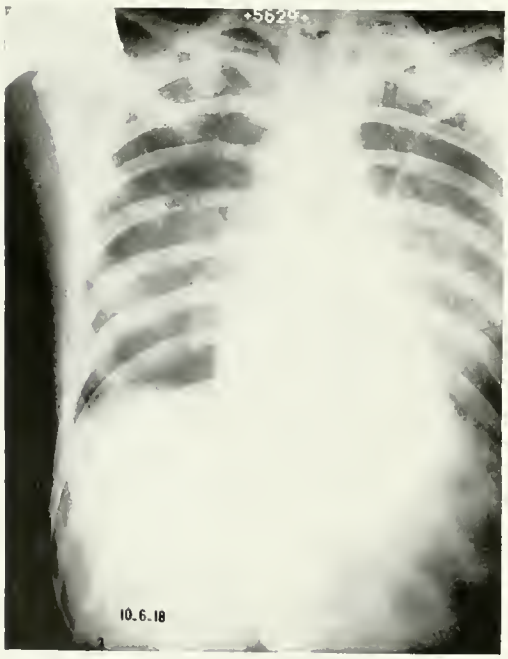

FIG. 3. The right and left chests show great improvement since No. 2.

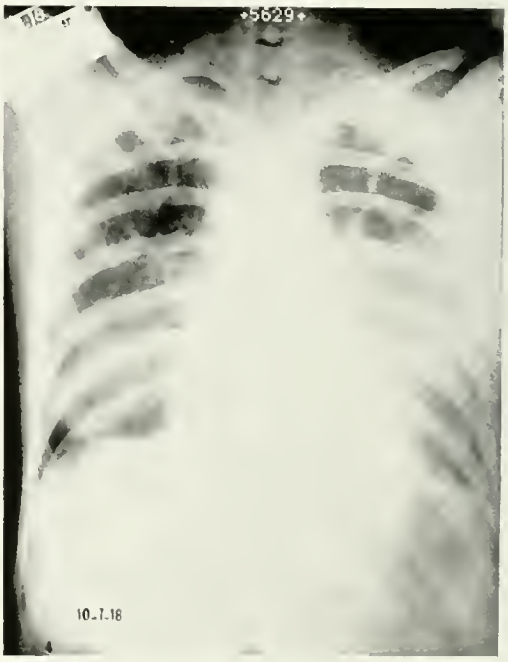

FIG. 4. Compare with Figs. 2 and 3. The hemorrhagic pneumonitis has again spread in left chest and density has increased in right chest.

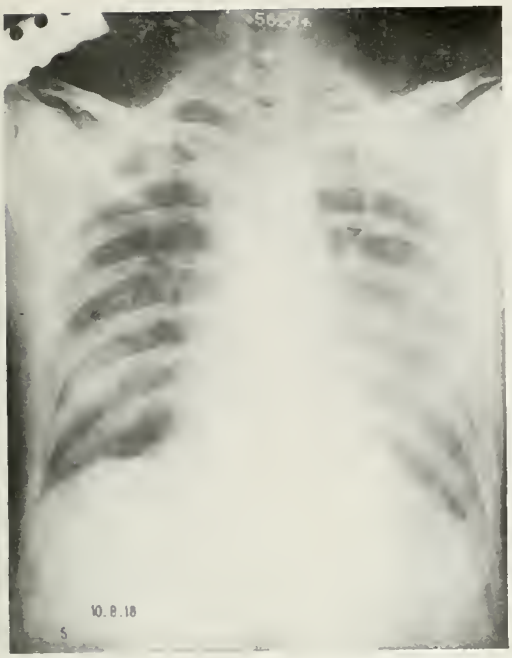

FIG. 5. Interlobar pleuritis has developed between upper and middle right lobes indicated by arrow. (Compare this with Figs. 6 and 7 for confirmation.) Lung tissue in both chests appears more mottled than in any of preceding films. Left costophrenic angle is cloudy.

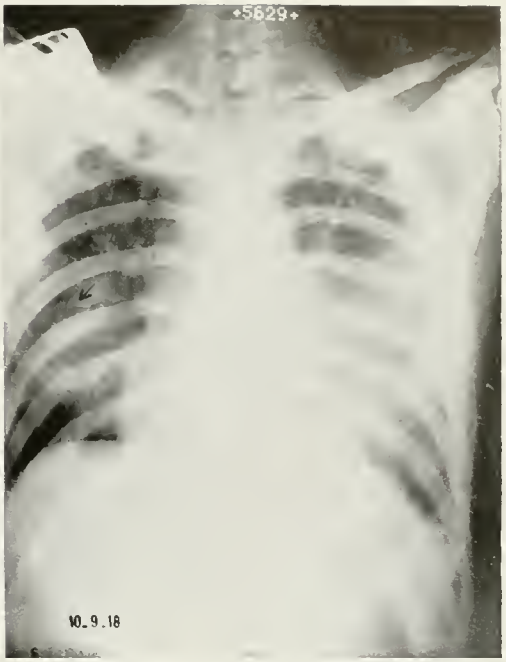

FIG. 6. Very little change since Fig. 5.

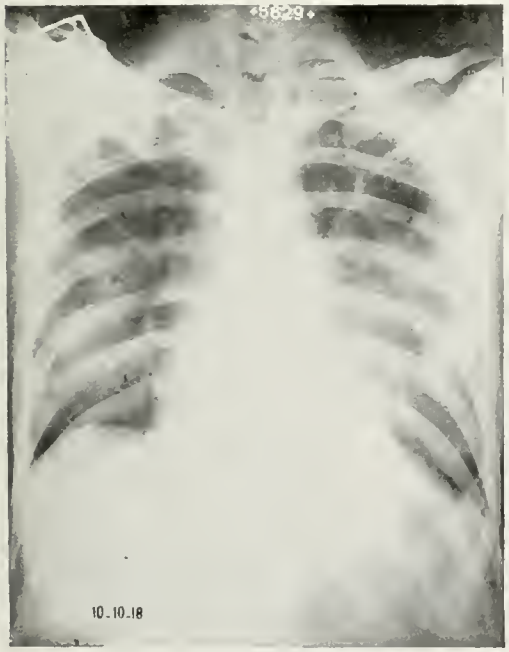

FIG. 7. Very little change.

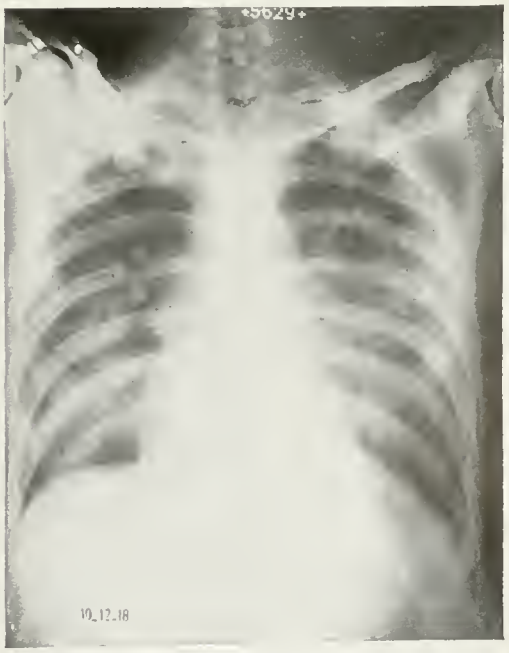

FIG. 9. Very little change since No. 7.

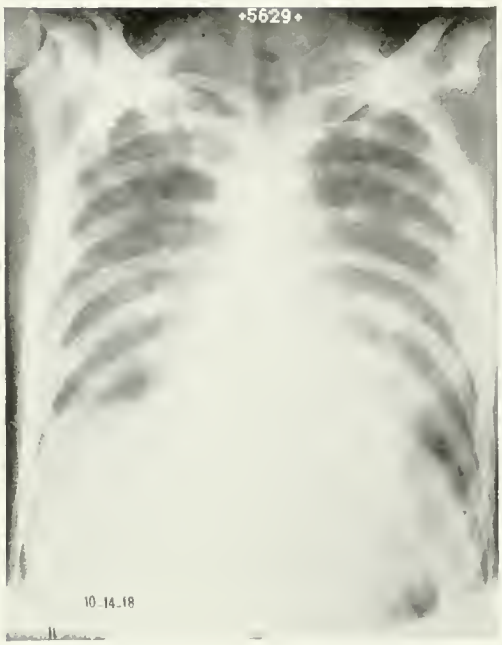

FIG. 11. Practically no change since Fig. 9.

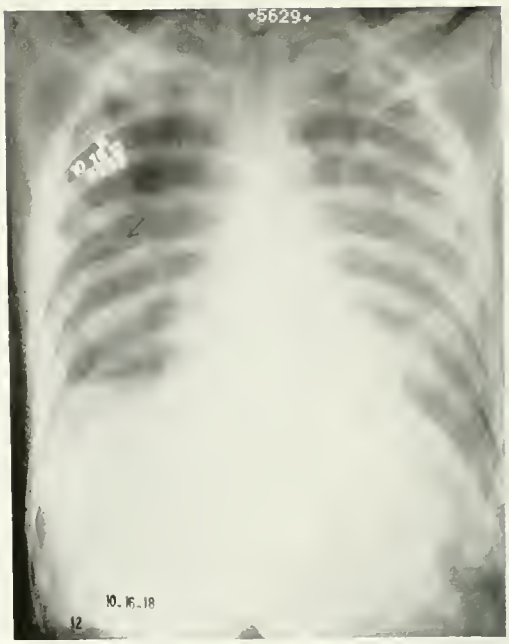

FIG. 12. Left chest is more hazy, including costophrenic angle, than in Fig. 11. Right chest shows pleural effusion. Interlobar pleuritis is conspicuous.

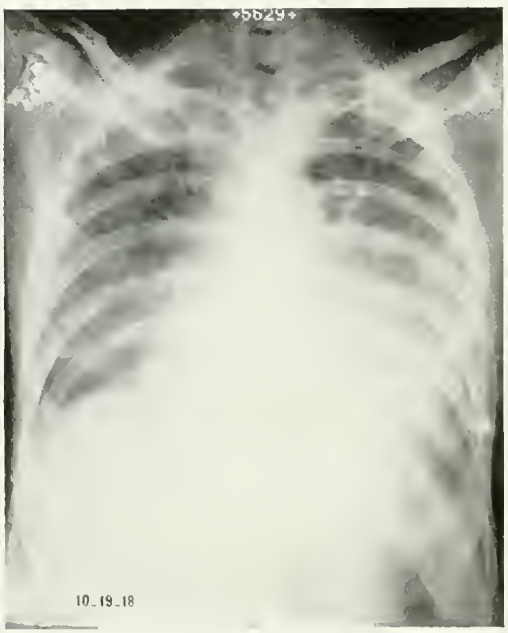

FIG. 15. The right chest remains hazy. Density of the left chest is more marked than in the preceding plate. Fluid is present.

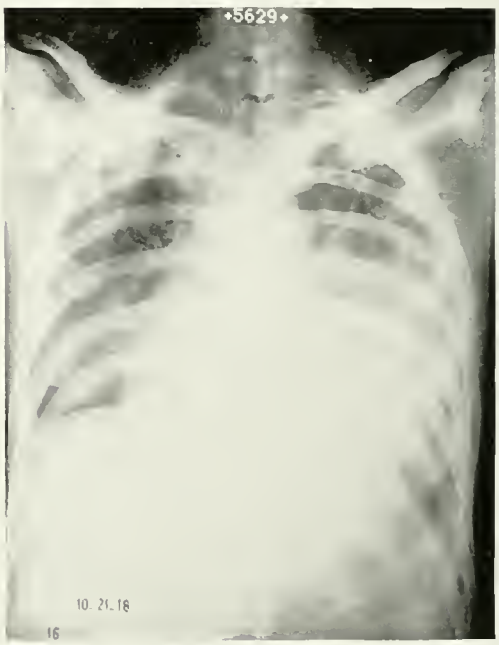

FIG. 16. No appreciable change as compared with Fig. 15.

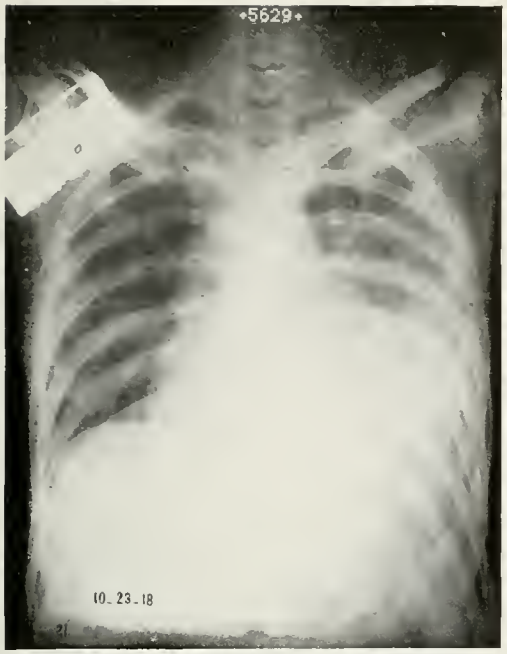

FIG. 21. Left chest remains dense from second rib to diaphragm.

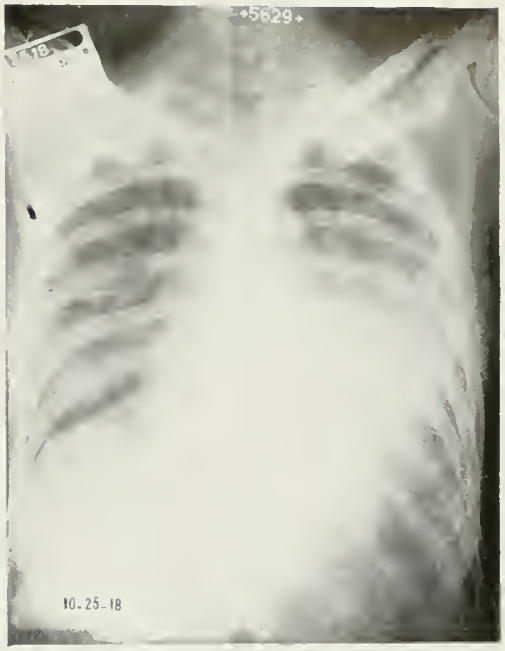

FIG. 22. Lower right chest has developed an increased density since Fig. 21.

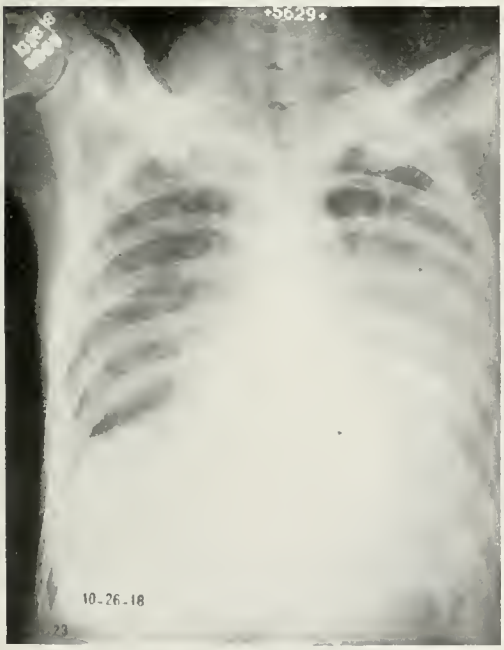

FIG. 23. No change as compared with Fig. 22.

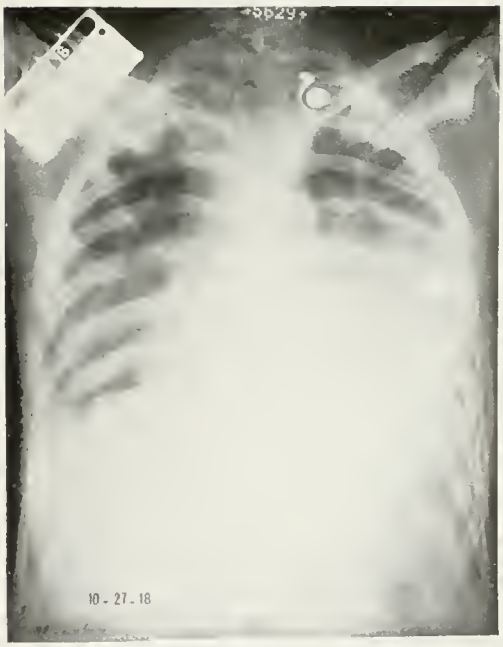

FIG. 24. No change as compared with Fig. 23. Empyema has developed in the lower left chest. Two days after this film was made 1000 c.c. of pus was aspirated.

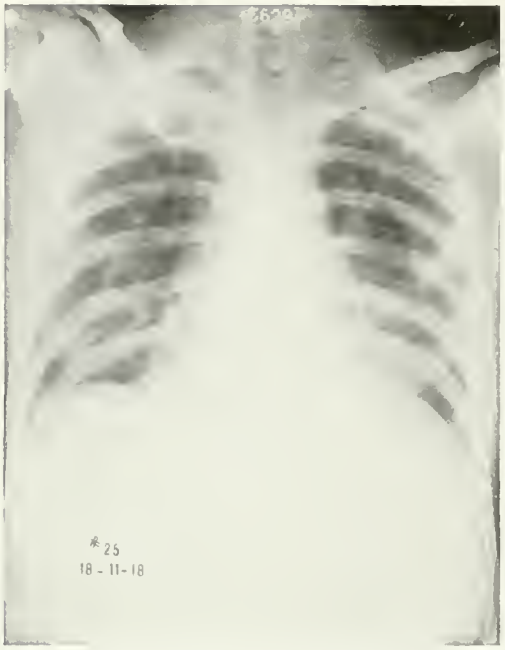

FIG. 25. This film was made nineteen days after Fig. 24. During this time, the empyema was drained. The interlobar pleuritis has almost entirely disappeared and both chests are much clearer.

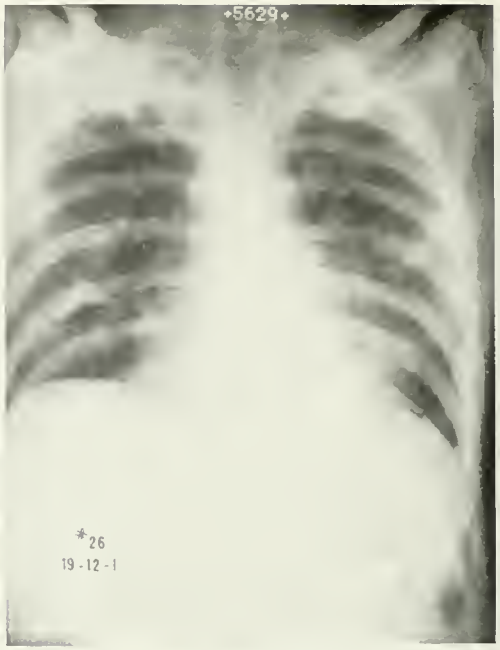

FIG. 26. Continued improvement.

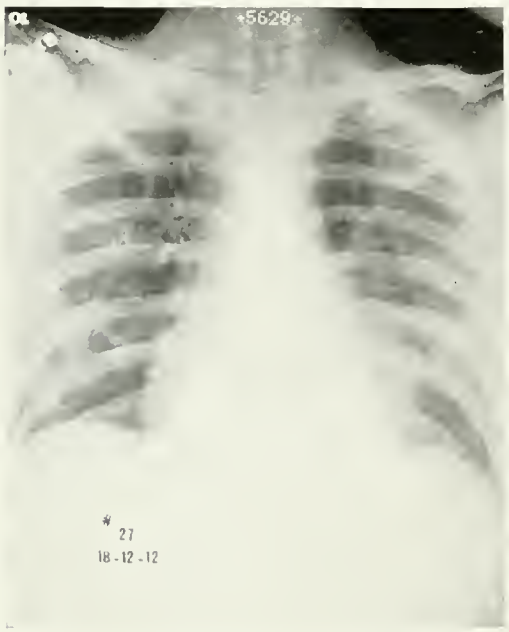

FIG. 27. Both chests are clearing up.

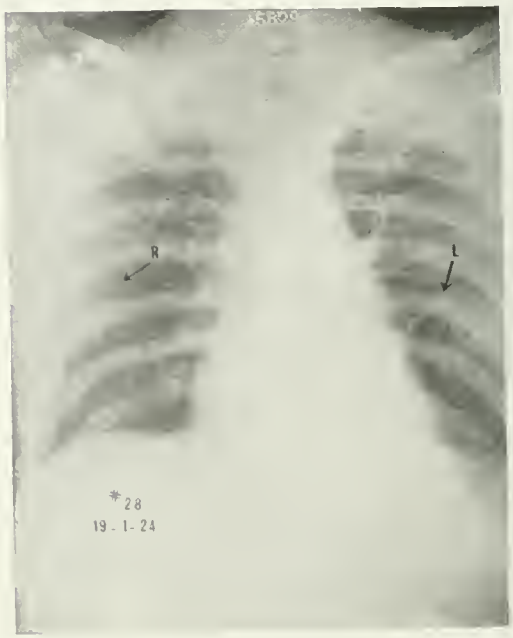

FIG. 28. The right chest is almost clear. The arrow (R) indicates a faint trace of the interlobar pleuritis. Arrow (L) indicates an adhesion developed by the empyema.

NOTE: This case was referred to in the paper as one in which an error in prognosis was made. On the date of Fig. 8, the indications were that it would prove fatal.

Roentgen Ray No. 6882. Group (*e*). This series presents a case in which the lungs remained uninvolved for seven days and then developed hemorrhagic pneumonitis, complicated by pleural effusion, interlobar pleuritis, and empyema, which was confirmed by autopsy.

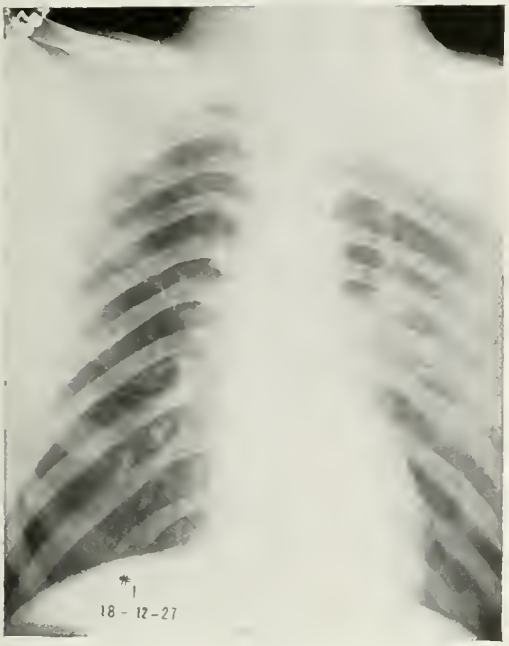

FIG. 1. No evidence of any pulmonary involvement.

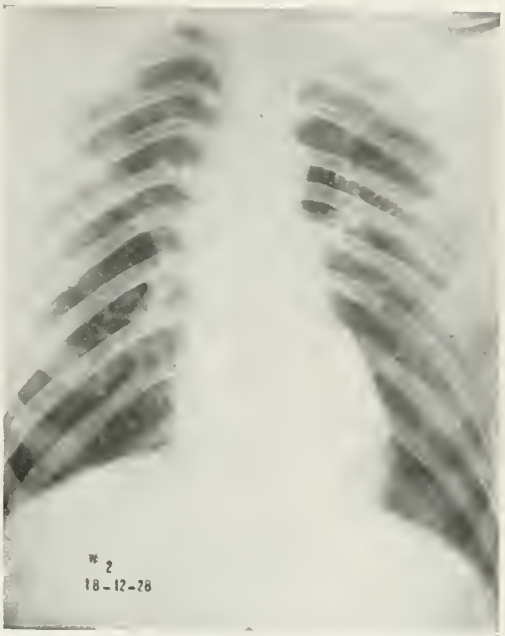

FIG. 2. No evidence of any pulmonary involvement.

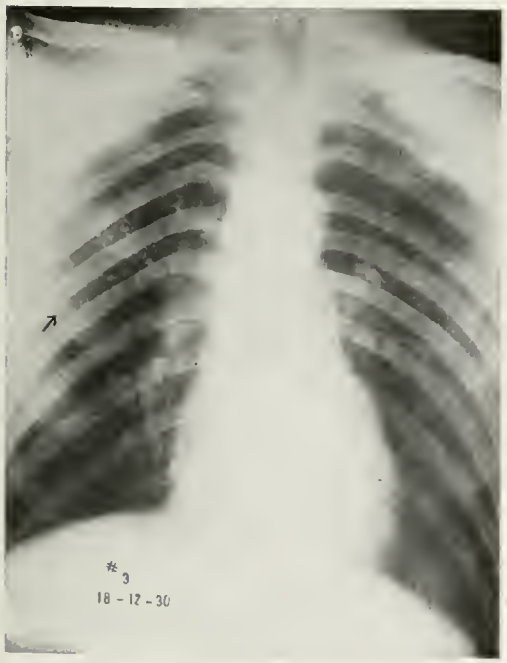

FIG. 3. No evidence of any pulmonary involvement. Shows normal lungs.

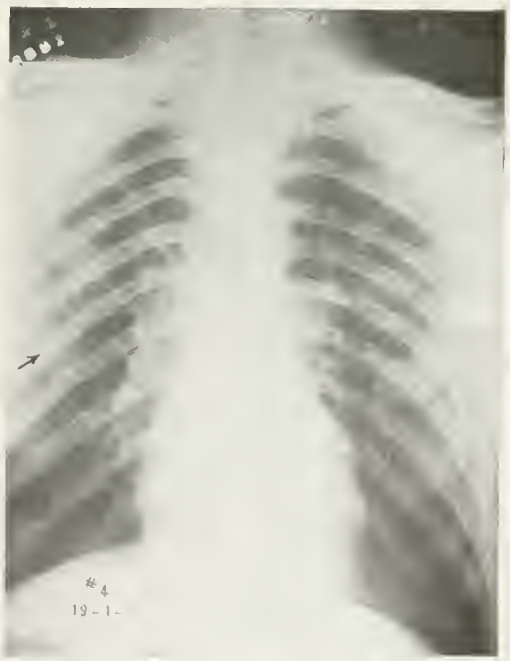

FIG. 4. Shows a beginning interlobar pleuritis betwee upper and middle lobes on right, associated with slight haziness of middle right lobe.

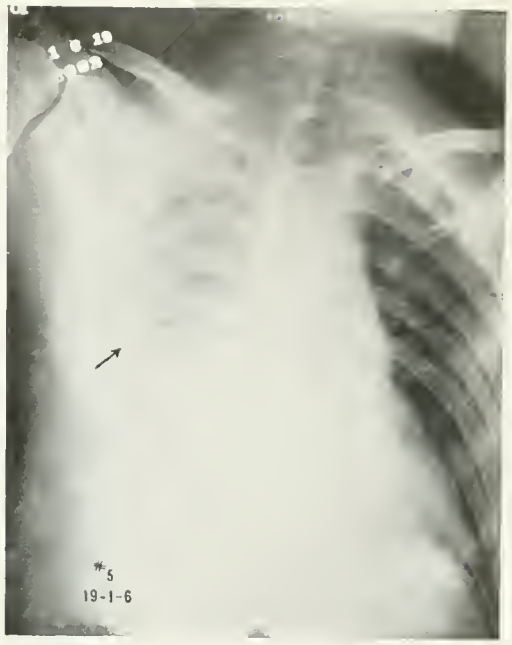

FIG. 5. Four days later, shows extensive involvement of all three lobes on right, costophrenic angle, and apex obscured by fluid. Interlobar pleuritis well developed, indicated by arrow. Lower left lobe shows involvement to less degree than on right. Heart silhouette greatly enlarged and contour changed compared with Fig. 4.

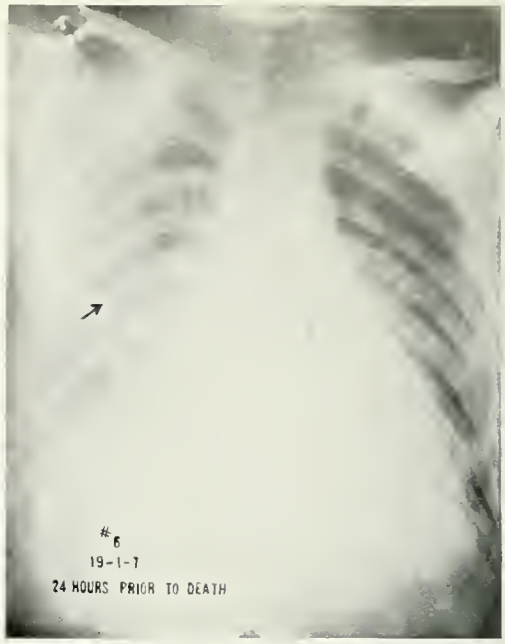

FIG. 6. The right chest is slightly clearer than in Fig. 5 500 c.c. of fluid have been removed since Fig. 5 was made. Lower left lobe is more extensively involved. The patient was too ill for further examination and died twenty-four hours after this film was made

Roentgen Ray No. 6859. Group (*d*). This series of films illustrates a case of hemorrhagic pneumonitis associated with adenitis and mottling. It also presents the complication empyema of the mediastinum and empyema of the lower left pleura.

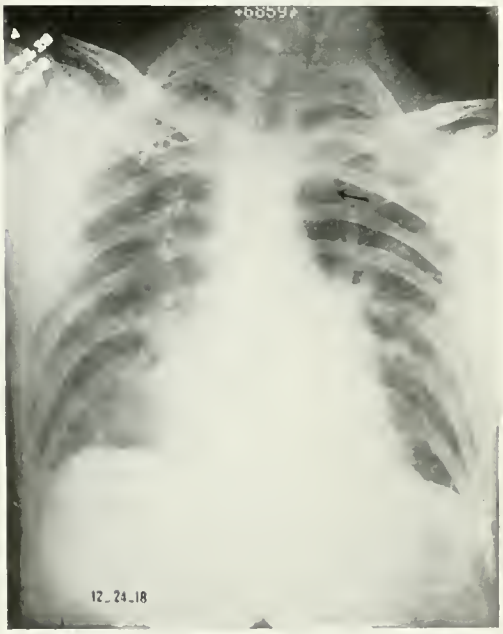

FIG. 1. Shows hemorrhagic pneumonitis of lower right, diffuse mottling of upper and middle right lobes, diffuse mottling of lower and slight mottling of upper left lobe. Costophrenic angle on left is obscured. Mediastinal glands are enlarged. Arrow points to area of abnormal density in mediastinum, which subsequent films prove was a developing mediastinal abscess.

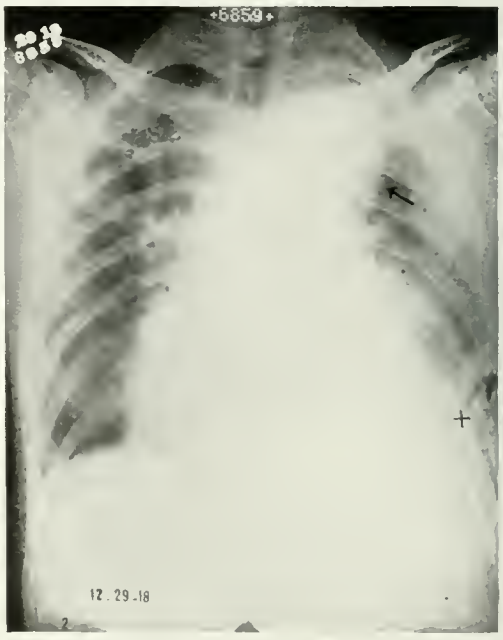

FIG. 2. The arrow points to the large mediastinal abscess. The cross indicates the presence of empyema of the lower left chest. The heart silhouette is greatly enlarged, suggesting pericardial effusion.

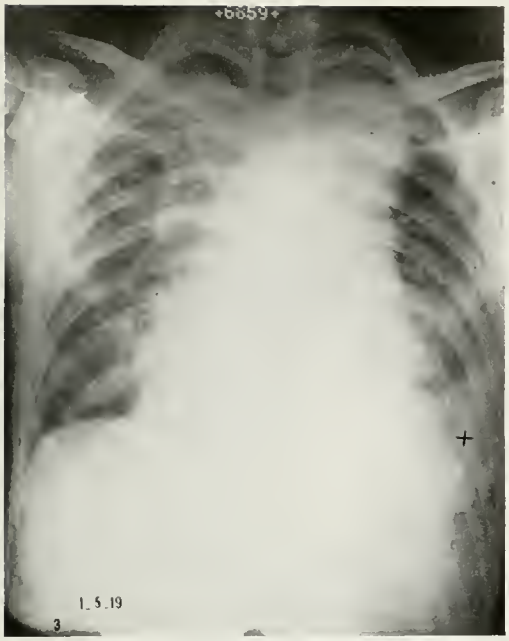

FIG. 3. Compare with Fig. 1 and note the discrete mottling which has developed since that date. The mediastinal empyema is unchanged in appearance. The empyema is more extensive than in Fig. 2. Compare this with Fig. 4, made after aspiration of the mediastinum and of the lower left chest.

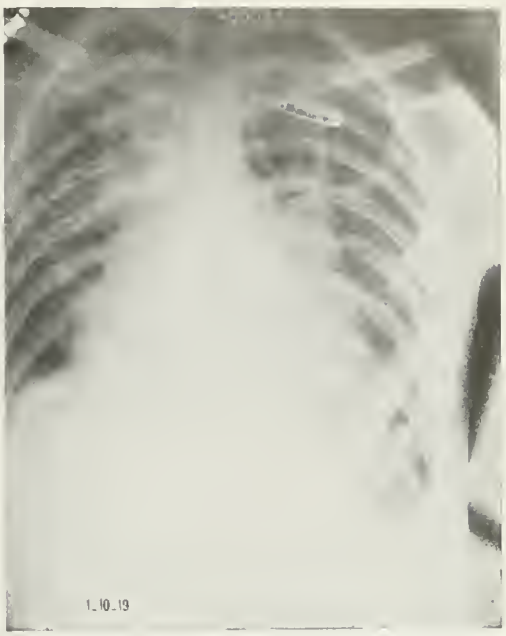

FIG. 4. The safety pin holds the drainage tube in the left mediastinum. The empyema has been completely evacuated from the lower left chest. The lungs are much clearer than in preceding figures.

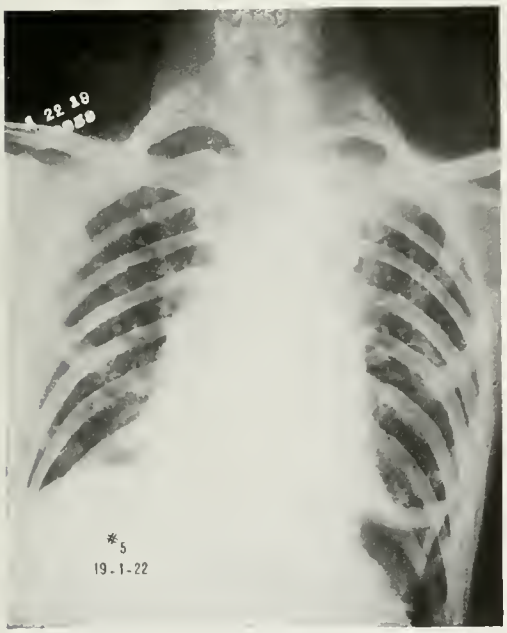

FIG. 5. The lungs are clearing rapidly, adequate draining is being maintained and the chest presents a greatly improved appearance.

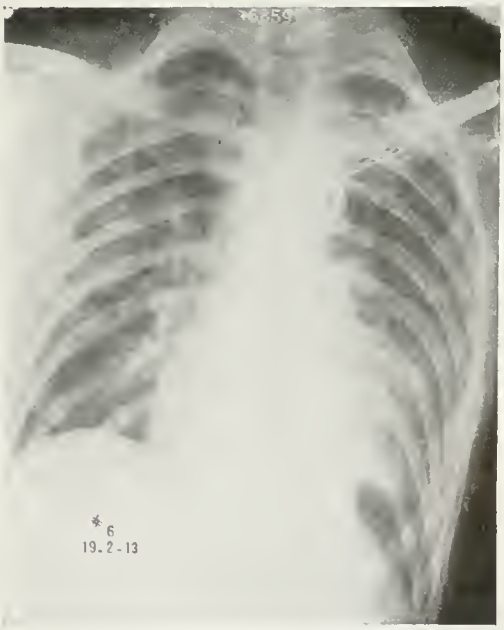

FIG. 6. The mediastinum is practically clear. Note the drainage tube with the wire insert. The safety pin holds a large caliber drainage tube in the lower left chest.