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A STUDY OF EIGHTY CASES OF EMPYEMA AT CAMP UPTON *

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Even a casual observation of the cases of empyema which have appeared in the Base Hospital at Camp Upton during the past winter and spring shows that we are dealing for the most part with an altogether different type of this disease from that to which we are accustomed. The very early appearance of this complication in the course of pneumonia, its unusual bacteriology and the very high mortality which accompanies it, even under favorable conditions, are the most striking and important variations. Closer study indicates a different train of symptoms, certain alterations in physical signs and a modified pathologic picture; while operative procedures, which, when resorted to in ordinary empyemas give almost uniformly good results, have in this epidemic been attended by so high a death rate as to cause both physician and surgeon to question seriously their advisability, value and type.

In view of these deviations from the usual in this frequent cantonment infection, a close study of all cases was imperative. In this problem it was at once apparent that closely associated laboratory and clinical methods must be constantly applied, and a large portion of the work of the medical and laboratory staffs of this hospital has been devoted during the past winter, to this insistent problem. This study has been a conjoint clinical and laboratory one, far more than is usually the case, for it has been found absolutely necessary that the laboratory staff follow the clinical aspects of the cases, even to the point of studying the physical signs, and a proper clinical comprehension of the individual case has been found inseparably connected with the work in the laboratory.

Analysis of the clinical picture and the laboratory findings soon showed us that we were dealing with a specific and virulent infection

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TABLE 1.—DATA OF EMPYEMA CASES

No.	Type of Pneumonia	Onset of Empyema	Type of Organism		Blood Culture	Treatment	Termination	Post Mortem Findings
			Sputum	Pleural				
1	Sec. infl.	4/10-4/16	Pn. II	Pn. II		Aspiration	Died 4/17/18	
2	Primary	1/ 9-1/14	Pn. I	Pn. I		Resection	Recovering	
3	Sec. infl.	12/15-1/ 1	Pn. I	Pn. I		Resection		
4	Sec. infl.	12/18-1/ 4	Pn. I	Pn. I	Neg.	Resection	Duty 2/19/18	
5	Sec. pleur.	3/15-3/22	Pn. II	Pn. II		Resection	Convalescent	
6	Primary	3/22	Pn. II	Pn. II		No record	Died 3/26/18	Lobar pn., L. U. and L. R. U., emp. lt.
7		2/ 1-2/22	Pn. II	Pn. II	Pn. II	Thoracotomy	Duty	
8	Sec. infl.	3/ 7-4/ 8	Pn. IV	Pn. IV		Resection	Prognosis (?)	
9	Primary	3/26-3/30	Pn. IV	Pn. IV		No record	Died 4/3/18	Lobar pn., rt. bilat., emp. sac.
10	Sec. mitral stenosis	1/4	Pn. IV	Pn. IV	Pn. IV	No record	Died 1/10/18	Lobar pn., L. L. and Rt. U., emp. lt., veg. endocarditis, infarcts in lt. lung
11	Sec. infl.	12/25-1/ 1	Pn. IV	Pn. IV		No record	Duty 1/3/18	
12	Sec. bron.	3/18-4/ 2	Pn. IV	Pn. IV		Resection	Prognosis (?)	
13	Primary	3/13-4/ 6	Pn. IV	Pn. IV		Resection	Prognosis (?)	
14	Primary		Pn. II	Pn. IV		Resection	Convalescent	
15	Primary	4/ 6-4/14	Pn. IV	Pn. IV		Aspiration	Died 4/14/18	
16	Primary	2/ 7-2/21	Strep. haem.	Strep. haem.	Neg.	Resection		Bronchopn., emp. sac. rt., abscesses in rt. lung, fibrinopur. pericarditis
17	Primary	3/12-3/13	Strep. haem.	Strep. haem.	Neg.	Resection	Died 3/23/18	
18	Primary	3/18-3/30	Strep. haem.	Strep. haem.	Neg.	Resection	Convalescent	Bronchopn., emp. rt. sac.
19	Primary	3/ 3-4/ 4	Strep. haem.	Strep. haem.		Aspiration	Died 4/12/18	
20	Primary	3/13-4/ 3	Strep. haem.	Strep. haem.		Resection	Convalescent	
21	Primary	3/30-4/ 1	Strep. haem.	Strep. haem.		Aspiration	Died 4/2/18	Bronchopn., emp. lt. L. L., acute fibrino. pericarditis
22	Primary	3/12-3/21	Strep. haem.	Strep. haem.		Resection	Convalescent	
23	Sec. bron.	3/25-3/31	Strep. haem.	Strep. haem.		Resection	Prognosis (?)	
24	Primary	4/ 5-4/ 9	Strep. haem.	Strep. haem.		Aspiration	Died 4/10/18	Bronchopn., lt. emp. lt. f., fibrinous pericarditis
25	Primary	3/29-4/ 2	Strep. haem.	Strep. haem.	Neg.	No record	Died 4/3/18	Bronchopn., (interest.) emp. lt.
26	Sec. bron.	3/ 9-3/20	Strep. haem.	Strep. haem.		Resection	Convalescent	

27	Primary	3/21-4/ 9	Strep. haem.	Strep. haem.		Resection	Convalescent	Lob. pn. rt. lobe, emp. rt. sac., emp. lt.
28	Sec. measles	3/26-4/ 3	Strep. haem.	Strep. haem.		Aspiration	Died 4/11/18	
29	Sec. bron.	3/16-3/17	Strep. haem.	Strep. haem.		Resection	Died 3/18/18	
30	Sec. bron., pharyngitis, G. measles	1/ 7-1/17	Strep. haem.	Strep. haem.		Resection	Convalescent	
31	Primary	3/ 4-3/ 5	Strep. haem.	Strep. haem.		Resection	Died 3/5/18	Lobar pn. left lung, emp. lt., central pn. rt. lung
32	Sec. infl.	3/ 5-3/ 9	Strep. haem.	Strep. haem.		Resection	Died 3/10/18	
33	Sec. measles	3/24-4/ 7	Strep. haem.	Strep. haem.		Resection	Convalescent	
34	Primary	3/6	Strep. haem.	Strep. haem.		No record	Died 3/8/18	
35	Primary	12/25-1/ 6	Strep. haem.	Strep. haem.		Aspiration	Died 1/17/18	Lobar pn. L. L. L., emp. lt., mult. abscesses of lung
36	Primary	3/ 1-3/ 3	Strep. haem.	Strep. haem.		Resection	Died 3/3/18	Bronchopn. lt. (interst.) emp. lt., fibrinopur. pericarditis
37	Primary	3/11-3/15	Strep. haem.	Strep. haem.		Resection	Died 3/19/18	Lobar pn. R. L. L., abscess of lung, emp. rt.
38	Primary	2/18-2/20	Strep. haem.	Strep. haem.	Neg.	Resection	Died 3/27/18	
39	Sec. infl.	3/ 7-3/10	Strep. haem.	Strep. haem.	Strep. haem.	Resection	Convalescent	Lobar pn. L. L. L., emp. lt., pneumothorax lt. infarcts of spleen
40	Sec. mumps	2/11-2/23	Strep. haem.	Strep. haem.	Neg.	Resection	Convalescent	
41	Sec. infl.	1/28-2/11	Strep. haem.	Strep. haem.	Neg.	Resection	Died 2/24/18	Bronchopn. R. L. L., lobar pn., L. U. L., emp. rt.
42	Sec. infl.	3/ 5-3/ 5	Strep. haem. (?)	Strep. haem. (?)		Resection	Died 3/11/18	
43	Primary	2/ 4-2/ 9	Strep. haem. (?)	Strep. haem. (?)		Resection	Died 3/12/18	
44	Sec. mumps	3/ 7-3/ 7	Strep. haem. (?)	Strep. haem. (?)		Resection	Died 3/9/18	Bronchopn. rt. lung (interst.) type, fibrinopur. pericarditis, emp. rt.
45	Primary	3/ 8-3/ 8	Strep. haem. (?)	Strep. haem. (?)		Resection	Died 3/10/18	Lobar pn. R. L. L., emp. rt. sac., fibrinopur. pericarditis
46	Sec. pharyn.		Strep. haem. (?)	Strep. haem. (?)			Died	Bronchopn. biat., emp. biat. sac.
47	Primary	4/14-4/16	Strep. haem.	Strep. haem.		Aspiration	Died	Sarcoma testes and lung, bronchopn., emp. lt., acute general peritonitis
48	Sec. tonsil	4/ 5-4/ 6	Strep. haem. (?)	Strep. haem. (?)			Died	Bronchopn. biat., emp. lt. sac., fibrinopur. pericarditis
49	Primary	4/17-4/20	Strep. haem.	Strep. haem.	Neg.	Aspiration	Died 4/21/18	
50	Primary	4/17-4/21	Strep. haem. (?)	Strep. haem. (?)		No record	Convalescent	
51						Resection	Duty	
52	Primary	1/ 1-1/20	Strep. virid.	Strep. virid.		Incision	Died 3/30/18	Bronchopn. rt. lung, lobar pn. L. L. L., emp. lt. sac., acute strep. meningitis
53	Primary	3/15	Strep. virid.	Strep. virid.	Strep. virid.	No record	Died 1/14/18	Bronchopn. (interst.) rt. L. L., emp. rt. sac., fibrinopur. pericarditis
54	Primary	1/ 8-1/ 4	Strep. virid.	Strep. virid.		No record	Died 2/3/18	Bronchopn. (interst.), emp. rt. sac., fibrinopur. pericarditis
55	Primary	1/12-1/24	Strep. virid.	Strep. virid.	Neg.	No record	Died 2/3/18	Bronchopn. (interst.), emp. rt. sac., fibrinopur. pericarditis

TABLE 1.—DATA OF EMPYEMA CASES—Continued

No.	Type of Pneumonia	Onset of Empyema	Type of Organism		Blood Culture	Treatment	Termination	Post Mortem Findings
			Sputum	Pleural				
56	Primary	12/15	Pn. II	Strep. virid.		No record	Died 12/29/17	Bronchopn. and lobar pn. (bilat. interst.), emp. lt., mult. abscesses of lungs
57	Sec. inf.		Pn. II	Strep. haem.		No record	Died 2/11/18	
58	Sec. inf.	1/ 8-2/14	Pn. II	Strep. haem.		Resection	Prognosis (?)	Bronchopn. and lobar pn. (interst.), emp. saec. rt., fibrinopur. pericarditis, mult. abscesses of rt. lung
59	Sec. tonsil., bronch. (?)	1/21-2/17	Pn. III	Strep. haem.		Resection	Recovering	
60	Sec. inf., pharyn. Primary	1/18-1/26	Pn. IV	Strep. haem.		Resection	Died 1/28/18	
61	Primary	12/22-1/20	Pn. IV	Staph. aureus.	Neg.	Resection	Died 2/11/18	
62	Sec. bron.	12/21-12/29	Pn. IV	Strep. virid.		No record	Died 12/21/17	
63	Primary	1/22-2/18	Pn. IV	Strep. haem.	Neg.	Resection	Convalescent	Lobar pn. lt. lung and rt. L., emp. rt.
64	Primary	3/4	Pn. IV	Strep. haem.	Neg.	Resection	Died 3/13/18	
65	Sec. mumps	1/26-2/19	Pn. IV	Strep. haem.		Resection	Convalescent	General millary T.B., lobar pn. lt. lung, emp. lt.
66	Sec. measles	12/6-1/27	Pn. I	Sterile		Resection	Convalescent	
67	Sec. infl.	3/27-8/30	Pn. I	Sterile		Aspiration	Died 4/1/18	
68	Sec. pleurisy	2/ 6-2/20	Pn. II	Sterile	Neg.	Resection	Died 3/15/18	
69	Primary	2/26	Pn. II	Sterile	Neg.	No record	Died 3/3/18	
70	Sec. measles	12/18-1/ 3	Pn. III	Sterile	Neg.	Resection	Died 3/5/18	General millary T.B., lobar pn. lt. lung, emp. lt.
71	Primary	1/30-2/25	Pn. IV	Sterile		No record	Duty 4/7/18	
72	Primary	3/30-4/ 9	Pn. IV	Sterile		No record	Died 1/21/18	
73	Primary	1/ 3-1/25	Pn. IV	Sterile		Resection	Duty	
74	Sec. bron.	3/ 7-3/11	Pn. IV	Sterile		Aspiration	Convalescent	
75	Primary	3/21-3/23	Pn. IV	Sterile		Aspiration	Died 4/19/18	Died 3/3/18 Duty 4/22/18
76	Sec. bron.	3/11	Strep. haem.	Sterile		No record	Convalescent	
77	Primary	4/ 8-4/15	Strep. haem.	Sterile		No record	Convalescent	
78	Sec. scarlet	3/30	Strep. haem.	Sterile		No record	Convalescent	
79	Sec. bron.	1/ 7-1/14	Strep. virid.	Sterile		Resection	Died 3/3/18	
80	Sec. inf.	3/12-3/17	Strep. virid.	Sterile	Neg.	Resection	Duty 4/22/18	

by an organism which is rarely encountered in civil infections of the lung and pleura. In most of these cases the organism in question — the so-called *Streptococcus haemolyticus* — has been the causative agent, not only of the empyema, but of the pneumonia associated with it. The relation between streptococcus pneumonia and the incidence of empyema is well shown by a comparison of the different types of pneumonia which have been encountered at Camp Upton.

During the period from Oct. 7, 1917, to April 20, 1918, 300 cases of pneumonia were admitted to the Base Hospital at Camp Upton. Of the 300 pneumonias, 283 had careful bacteriologic examinations of the sputum; 136, or almost half, were found to be of streptococcus origin, and 137 were caused by the pneumococcus. There were 49 cases of streptococcus empyema, showing an incidence of empyema among the streptococcus pneumonias of 36 per cent. On the other hand, there were only 15 cases of pneumococcus empyema, or an incidence of empyema among the pneumococcus cases of 11 per cent. Finally, there were 15 cases of sterile empyema about equally divided between streptococcus and pneumococcus pneumonia. Altogether, our records show for this period, 80 cases of empyema, and these 80 cases form the basis for this study.

Before proceeding to the discussion of empyema proper it might be well to refer briefly to the relation of measles to this disease. In some of the other camps a large percentage of the empyemas, especially those of streptococcus origin, have developed as a complication of post-measles pneumonia. Such has not been the case at Camp Upton. In the first place, a comparatively small number of our measles patients have developed pneumonia — thirty out of 164 cases, with a mortality list of but two. Furthermore, only four cases of empyema (Cases 28, 33, 66 and 70) have developed among these thirty pneumonias, and of these only two patients died. By referring to Table 1, however, it will be seen that a considerable number of the empyemas developed in pneumonias which followed influenza, tonsillitis and other mild infections. We are quite certain, however, that streptococcus empyema may develop from a primary streptococcus pneumonia. Two of the pneumonia cases which followed mumps (Cases 40 and 44) were complicated by empyema, both of these being streptococcus infections.

In this study special attention has been devoted to the bacteriologic examination of the cases. In most instances direct specimens of the patients' pleural exudate were collected as soon as the diagnosis was made, and taken to the laboratory at once for examination. The bacteriology of the sputum was carried out as follows: A fresh specimen of sputum was thoroughly washed with sterile saline and smears made for the gram stain. The sputum was then emulsified and 0.5 c.c. added to a tube of Avery's blood broth, which was incubated in the

water bath at 37 C. for five hours. At the end of this time smears were made and if a pure culture was found agglutination tests were made at once in the usual manner. Under all conditions, however, subcultures were made from broth or plain agar plates and colonies were picked from these on the following day for further study. In many cases the blood broth method was controlled by injections of the sputum into the mouse and identification of the organism was determined from the peritoneal exudate or the heart's blood. In a few instances cultures of the sputum were made directly on blood agar plates. The results obtained by the various methods have been surprisingly consistent.

TABLE 2.—CLASSIFICATION OF EIGHTY EMPYEMAS

	Cases	Deaths
I. <i>Pneumococcus</i> in both sputum and pleural exudate:		
Type I in both.....	3	0
Type II in both.....	4	2
Type IV in both.....	6	2
Type II in sputum; Type IV in pleural exudate.....	1	0
Sputum not examined; Type IV in pleural exudate.....	1 (?)	1
	15	5 (33%)
II. <i>Streptococcus</i> in both sputum and pleural exudate:		
Strept. hemol. in both.....	29	16
Sputum not examined; strept. hemol. in pleural exudate.....	7 (?)	6
Strept. virid. in both.....	4	3
	40	25 (60%)
III. Mixed infections:		
Pn. Type II in sputum; strept. vir. in pleural exudate..	1	1
Pn. Type II in sputum; strept. haem. in pleural exudate	2	1
Pn. Type III in sputum; strept. haem. in pleural exudate	1	0
Pn. Type IV in sputum; strept. haem. in pleural exudate	4	2
Pn. Type IV in sputum; strept. vir. in pleural exudate..	1	1
Pn. Type IV in sputum; staph. aur. in pleural exudate.	1	1
	10	6 (60%)
IV. Sterile empyemas:		
Pn. Type I sputum.....	2	1
Pn. Type II sputum.....	2	2
Pn. Type III sputum.....	1	1
Pn. Type IV sputum.....	5	1
<i>Streptococcus haemolyticus</i>	3	1
<i>Streptococcus viridans</i>	2	1
	15	7 (50%)

CLASSIFICATION OF EMPYEMAS

By referring to Table 2 it will be seen that our eighty cases of empyema naturally fall into three groups: (1) pneumococcus empyemas; (2) streptococcus empyemas; (3) sterile empyemas. These three groups will be discussed separately.

1. *Pneumococcus Empyemas*.—In the present series of eighty cases there were only fifteen that showed the pneumococcus from culture of the pleural exudate. These pneumococcus empyemas differed in no essential respect from those seen in civil practice. Without exception

they developed in cases of lobar pneumonia in which the pneumococcus had been previously isolated from the sputum. This type of empyema usually comes on late in the course of the disease when the patient is convalescent. There is a sudden rise in temperature and a corresponding increase in pulse rate. The patient, however, may not be very ill. Frank signs of fluid are usually present and on exploring the chest over this area cloudy or creamy pus is withdrawn which on examination shows many pus cells and capsulated pneumococci.

Pneumococci of Types I, II and IV are all represented in this group, and in all except one case the same kind of pneumococcus was found in both sputum and pleural exudate. In this case (Case 14) pneumococcus Type II, atypical, was isolated from the sputum, and pneumococcus Type IV from the pleural fluid. Of this group of fifteen cases, five patients died, a mortality of 33 per cent. One patient (Case 10) developed a pneumococcus endocarditis and septicemia. One of the Type II cases (Case 7) on blood culture showed twenty-four colonies per cubic centimeter of blood, but the patient made an excellent recovery after rib resection and drainage. This patient showed a curious metastatic focus, a perithyroid abscess. In the fatal cases of this group the men all died early in the disease apparently from the intensity and extent of the pneumonia rather than from the concomitant empyema.

2. *Streptococcus Empyemas*.—By far the largest number of our empyema cases were streptococcus infections, and it is this group that constitutes the most interesting phase of the problem. For convenience of discussion it will be well to divide this group into three subgroups.

- (a). *Streptococcus haemolyticus* empyema.
- (b). *Streptococcus viridans* empyemas.
- (c). *Streptococcus* empyemas following pneumococcus pneumonia.

(a). *Streptococcus Haemolyticus Empyemas*: In this group there were twenty-nine cases in which *Streptococcus haemolyticus* was recovered in pure culture from the empyema fluid and in which bacteriologic examination of the sputum showed the same organism. There were seven other cases in which the hemolytic streptococcus was recovered from the pleural exudate, but in which for some unavoidable reason the sputum was not examined. In these seven cases, however, the clinical course and physical signs strongly indicated a streptococcus pneumonia, so they probably belong to this group. These thirty-six cases constitute a fairly definite and characteristic picture. The patient who often gives a history of some recent infection of the upper respiratory tract is admitted to the hospital complain-

ing of cough, fever, and occasionally pain in the side. The onset, as a rule, is gradual, though it may be very sudden. Many cases are admitted with a diagnosis of influenza or bronchitis. The cough is usually dry in character and is often entirely absent, a feature which makes it difficult or impossible to collect a satisfactory specimen of sputum. The sputum itself is rather characteristic. It is usually of a white or yellowish color, quite viscid and mucopurulent. The rusty or hemorrhagic sputum of lobar pneumonia is rarely seen. The fever usually runs an irregular course, and considerable febrile periods succeeded by rapid oscillations, as in an active sepsis, appear at the onset. The pulse is comparatively slow in this early stage of the pneumonia, but is in proportion to the temperature. The respiratory rate is, however, proportionately high throughout. Pain in the side is a very inconstant symptom and when present is an additional cause of the inspiratory distress. Inspiratory pain is not usually long continued and few patients have required strapping or opiates because of it.

Physical Signs Before Onset of Empyema.—The physical signs before the onset of empyema are often indefinite. On percussion, areas of dulness may or may not be present at first. Moist crackling râles are nearly always to be heard, at times confused with the pleuritic friction rub, but typical bronchial voice and breathing are rather unusual signs at the onset of the pneumonia. More often the voice and breath sounds are faint. Furthermore, one must say that there are few distinctive signs or symptoms at the onset of the pneumonia which serve to differentiate cases which subsequently develop empyema from those that do not.

Physical Signs After Onset of Empyema.—In several instances patients have entered the hospital under the regimental diagnosis of empyema. Careful investigation of the history has, however, in all these instances shown a story of several days of indisposition and probable fever. Patients who have entered the hospital usually because of prostration and a slight febricula and before any physical signs indicative of either pneumonia or empyema appeared, have often developed under observation surprisingly early empyemas. Thus our group of streptococcus cases has shown a positive diagnosis of empyema possible in an average of 5.4 days after admission. When one takes into consideration the fact that three of these patients developed their pneumonia in the hospital as a secondary process two or three weeks after admission, the very early onset of streptococcus empyema is impressively demonstrated. Suffice it then to conclude that this empyema develops with surprising rapidity and often before a conclusive diagnosis of pneumonia, in addition to pleurisy, could be

made except by the assistance of the roentgen ray. It is therefore seen that these empyemas are imposed on, and do not merely succeed, the active stages of pneumonia. The prognostic significance of this fact must be apparent to all.

In our early experience with the disease we were surprised, in three instances, to find empyema at the postmortem where simple lobar pneumonia had been expected. In two of these cases the physical signs should certainly have given the diagnosis, and mere indolence on the part of the ward surgeon accounted for this failure. Careful analysis of the charts in these three cases, however, presented nothing of a symptomatic character which would lead any one unsophisticated in this epidemic to suspect the development of an empyema, for the picture was typically and solely that of a pneumonia. Nor was this finding limited to our early cases alone, for not seldom, even after we were keenly alive to the frequency of empyema, the needle and the roentgen ray have detected rapidly developing empyemas which were entirely unmarked by alterations in the clinical picture, and with strikingly few modifications in the physical signs.

One of the most constant and suggestive signs has been the appearance of profuse sweats, comparable in degree and in exhaustion to those of acute rheumatic fever. The entire surface of the body, more notably the head and the neck, are frequently beaded with large drops of sweat. These signs are unaccompanied by a chill, which is found to be a rather unusual symptom at any stage of these cases, and one which never appears but at the period of onset. Under onset we have mentioned the marked exhaustion which typifies these cases. This, however, is in no way characteristic of the empyema, since it may be also strikingly manifest in the simple streptococcus pneumonia. It is usually more pronounced when a purulent exudate is forming, or has formed, than when the pneumonia is uncomplicated. Where the exhaustion is very striking we have learned to be very constantly on guard and to apply the needle with even more diligence than ordinary, though not infrequently with negative results. Acceleration of the respiration is another suggestive sign, though of course in recognized pneumonia it can be considered as of only relative value; but there can be no doubt that as the pus begins to form there is a very perceptible increase in the respiratory rate, above that which may be attributed to the pneumonia alone, and with increase in rate is also to be found increased dyspnea and respiratory distress. There is nothing characteristic in the temperature and pulse curve in the early stage that suggests the onset of empyema. Of course, where it develops later in the pneumonia, when pulse rate and temperature are falling, an ascent in both appears. It rarely assumes a septic form, however.

In ordinary pleurisy and particularly in a process which so largely, as in these cases, involves the bases and diaphragmatic pleura, one is accustomed to consider abdominal rigidity a sign of considerable value, especially when, as is usually the case in this epidemic, it is monolateral at the onset. This sign has been strikingly absent in the cases which we present in this study. In but four of the cases do we recall this indication as demonstrable. It was often entirely absent even in instances in which we were able to demonstrate readily the fact that the diaphragmatic pleura was involved. Head zones of hyperesthesia is another frequent sign that one finds in cases of pleurisy of whatever origin, but which has not been present in our cases. Another negative sign which has impressed us very much has been the very general absence of or diminution in degree of the stabbing pain which is so generally complained of in the onset of pleurisy. This has not been frequent in our cases, even where the onset of pleural effusion seemed to take place synchronously with or but slightly later than the first pneumonic symptoms or signs. On the contrary, we have all been impressed with the frequency with which skin tenderness (not hyperesthesia) has been evident on even ordinarily vigorous percussion over the involved area. Not seldom this tenderness, which is a deep and not a superficial one, has largely determined us in selecting a point for needling.

Our study of the physical signs in general as regards this group of unusual cases has been disappointing and often the findings most difficult of explanation. As a result of great disparity between positive physical signs and lesions, our ward surgeon has been instructed to needle or fluoroscope even when all signs point away from the existence of pleural effusion. We both feel that we have "unlearned" far more physical signs of pleural fluid than we have perfected. All observers have been struck with the great frequency of skodaic resonance, and many times it has been most typical over areas from which the needle gave abundant fluid. Postmortem and roentgen-ray study of this sign has taught us to consider it as due in many instances to early pulmonary compression of more than the usual degree, because of the fact that the pleura in these cases was but little thickened, and the adjacent pulmonary tissue but little involved by consolidation. That this sign may be transmitted through a considerable amount of superjacent fluid has been demonstrated to us numerous times, and over areas where no transmission of abdominal tympany could be assumed. Probably for the same reason, breath sounds and vocal and tactile fremitus have been transmitted through massive exudates with great and deceptive distinctness. We have been forced to exaggerate alterations in these signs, or to totally ignore their appar-

ent significance. Doubtless the more limpid condition and less cell concentration of the fluid as compared with that of the antebellum conception of empyema has also something to do with the much modified physical signs. Beyond question the single physical sign which we have come to consider as of most value in the detection of these effusions is alteration in percussion. A high pitched note or the flattening of the customary resonance is a sufficient indication for a tap in these cases, and if the tap be a dry one a repetition is justified.

In six instances distinct and unmistakable pneumothorax has developed before the cases have been tapped, and in quite frequent instances minor degrees of this phenomenon appeared. In none of them could the usual explanatory causes be found present. There had been no possible injury to the thorax; no bacteria capable of causing gas formation were found in the fluid, and no possible cause of pulmonary laceration could be made out clinically, or in the three instances that came to necropsy. The amphoric splash and metallic tinkle of mingled fluid and air has come and gone in several typical cases also without a reasonable explanation. One hypothesis for these curious and surprisingly early manifestations is that they may be due to rupture of minute pulmonary abscesses, which thus allow the escape of air into the pleural cavity. Microscopic study lends some support to this theory.

In a discussion of the pathologic anatomy of the disease mention will be made of the frequent development of pleuropericarditis, and the very early appearance of this lesion is quite as remarkable as the appearance of the empyema itself. Usually when this process is present it becomes very early apparent from a primary pleuropericardial friction rub which is commonly so distinct as to become unmistakable. It diminishes, of course, as the effusion develops. As a natural sequence of this complication a pure pericarditis appears also with considerable frequency. Postmortem evidence shows that this first involves the parietal layer of the chamber, the visceral layer of the membrane only becoming involved later. Under our intensive method of study of the cases, the complication has only rarely escaped detection clinically. The amount of fluid which may thus accumulate in the sac may be very large — as much as 900 c.c., for example. The character of this fluid is precisely like that seen in the pleural cavities. First, it has a typical clear to turbid urine appearance, later on it becomes highly fibrinous, with the characteristic deposit of fibrin on both layers of the membrane and the ultimate transformation into pus of a clinical type. The bacteriology of the pericardial exudate coincides with that of the pleural fluid.

There are no signs or symptoms that appear in the pericardial complication which differ from those customarily seen in this condition. In two instances the pericardial sac was drained, once early in the course of the complication, once late after the fluid had become a thick and gelatinous pus. Both patients recovered.

Study of the total and differential white cell count has been very disappointing as a diagnostic measure. With the acknowledged existence of the pneumonia there is nothing shown suggestive of a developing exudate, nor of the fact that this fluid is of a highly septic character.

The blood pressure is that usually seen in pneumonias. Of course in fatal cases the pulse pressure becomes very much diminished as death approaches, and with it the usual tachycardia and cardiac arrhythmias which are common to the immediate premortem status.

The urine provides no data of an unusual character and nothing of a diagnostic value.

The roentgen ray has been used in almost all cases, in diagnosis; not so frequently for the demonstration of fluid as to indicate the extent and nature of the pulmonary process. In most cases so far as the demonstration of empyema is concerned the roentgen ray, particularly stereoscopic plates, have been most useful. It is much more necessary for the location of pulmonary disease. In several instances it has been misleading, indicating no fluid where the needle showed it to be present and less frequently the opposite fault has been found. The fluoroscope has not always been employed as a diagnostic routine for the reason that most of our patients have been so gravely ill that they could not be set up. It was soon found that satisfactory plates could only be obtained when the patient was raised up on a rack and kept in this position for a short period until gravity had caused the fluid to occupy a dependant part of the thorax. For a most satisfactory method of accomplishing this we are much indebted to our colleagues at Camp Devens, who first demonstrated their excellent practice to us. Our experience has shown us, however, that one may not depend absolutely on the roentgen ray in the diagnosis of empyema. In other instances it has misled us where the fluid was encapsulated at the left internal and posterior angle of the thorax, and its shadow obscured by that of the heart. The use of the roentgen ray is indispensable in the diagnosis of empyema, but it will not alone keep the lazy clinician out of trouble with the pathologist.

Frequent needlings have been most certain and more satisfactory in our hands. Where the puncture was to be made for diagnosis only, a fine hypodermic needle from 3 to 4 cm. in length attached to an ordinary 5 c.c. Luer syringe is used. The fluid in most questionable

instances is so limpid that it can be very readily obtained in this manner in sufficient quantity for bacterial and histologic diagnosis, and the pain caused by this needling is so slight that the patients do not object to frequent taps. We ordinarily do our aspirations with the Potain aspirator, but sometimes when the amount to be aspirated is small, an ordinary 20 c.c. glass syringe is used. We cannot commend the use of the needle too highly. It is indispensable in diagnosis.

Character of the empyema fluid: The pleural exudate when removed is usually light yellow, often with a greenish tinge, and in the early stages only slightly cloudy. Later in the disease it may take on a distinctly purulent character. Smears from the fluid show pus cells in large numbers, and large round or slightly flattened cocci arranged in short chains of from two to ten organisms. We have observed that the bacteria are much more numerous in the streptococcic empyema than in the pneumococcic empyemas. Cultures from the exudate on blood agar plates show, after twenty-four hours' incubation, many small, elevated, opaque, colonies of a pearl-gray color, each surrounded by a sharply defined zone of hemolysis. Subcultures in blood broth produce diffuse clouding of the medium, with a heavy sediment in the bottom of the tube. Rapid destruction of the blood cells takes place and the blood broth takes on a brilliant, claret-red color. Smears from the blood broth show chains of cocci similar in all respects to those seen in the smears from the pleural exudate.

The mortality rate in this group was very high, our experience in this respect corresponding to that met by clinicians in other camps. Of the thirty-six cases, twenty-two patients died, a mortality rate of 61 per cent.

(b). *Streptococcus Viridans Empyemas*.—In this group of cases cultures from both the sputum and the pleural exudate showed a streptococcus of the nonhemolyzing variety. There were found four cases of this type (Nos. 52-55). Clinically, these cases have differed in no respect from the *Streptococcus hemolyticus* cases. All four of these were cases of bronchial pneumonia, and the symptoms and signs of empyema were similar in every way to those of *Streptococcus hemolyticus* empyema. Three out of the four patients died.

(c). *Streptococcus Empyemas Following Pneumococcus Pneumonias*.—This very interesting group of mixed infections consists of nine cases (Nos. 56-65) and corroborates the findings of Cole and McCallum at Fort Sam Houston. In these cases the pneumococcus was cultivated from the sputum, but cultures from the pleural exudate gave streptococcus, either hemolyticus or viridans. Pneumococci of Type II, III and IV are represented in this group of mixed infections. There is also a case (Case 61) which we include in this group which

gave pneumococcus, Type IV, in the sputum, but *Staphylococcus aureus* in the exudate. It is possible, however, that in this case the staphylococcus infection developed after operation and drainage had been performed. The majority of these cases were frank lobar pneumonias. There were two cases diagnosed bronchopneumonia, and it may have been that in these two cases mouth pneumococci confused the bacteriologic picture. This was also a highly fatal group, six out of ten patients dying. Clinically, the empyema in this group possessed all the characteristics of the pure streptococcus type.

Summarizing these three subgroups, there were forty-nine streptococcus empyemas with thirty deaths, a mortality rate of 61 per cent.

3. *Sterile Empyemas*.—In addition to the sixty-five empyemas already mentioned, we have included in our study fifteen cases (Nos. 66-80) in which fluid of a purulent or semipurulent nature was removed from the pleural cavity, but from which cultures were sterile. In several of these cases gram-positive cocci were seen in smears from the fluid. They usually showed, however, some sign of disintegration and could not be cultivated even on blood medium. Ten of these empyemas were associated with pneumococcus pneumonias of the lobar type, and five patients died. Five cases were associated with streptococcus pneumonia and two of these patients died.

Blood Cultures.—Blood cultures were made on twenty of our empyema cases, with only three positive results (Nos. 7, 10 and 39). Two of these were pneumococcus cases and one *Streptococcus haemolyticus*. In the early part of the year, blood cultures were taken on every case of pneumonia, but when it was found that the results were almost universally negative this procedure was discontinued as a part of the routine examination. The clinical picture might lead one to suspect that this disease was one phase of a general septicemia. The character of the temperature curve, the pulse rate, the profound prostration, the rapid course of the disease and the high mortality all support this impression. The rare occurrence, however, of metastatic foci points strongly against this theory. Strangely enough, two out of the three patients in whom the blood culture was positive made uneventful recoveries.

PATHOGENESIS OF EMPYEMA

All of our cases of empyema have been associated with pneumonia, but in many of the streptococcus cases the pneumonic process has been relatively slight, as verified by physical signs, the roentgen ray and necropsies. Cases of *Streptococcus haemolyticus* empyema have been reported from other camps in which no evidence of con-

comitant pneumonia could be obtained, and such a phenomena seems possible in consideration of our own experience. It is quite certain that in many cases the empyema has far outweighed the pneumonia in clinical importance. It is probable, however, that in the majority of cases streptococcus empyema develops from a bronchopneumonia, the infectious organism being conveyed to the pleura either by the way of the lymphatics or by the direct extension of one of the small abscesses so frequently seen, through the pleural coat. The severity of the pneumonia may be a factor in the development of the empyema, but this is a difficult point to settle, since the onset of the empyema early in the course of the disease masks the real character of the pulmonary condition. It may be stated, however, that streptococcus empyema has usually appeared in those cases of pneumonia that have seemed to be severe infections.

The question if ward infections occur in this disease, that is, patients suffering from pneumonia of whatever type are likely to contract a streptococcus empyema from their associates in the ward or from streptococcus carriers, receives some confirmatory evidence from the fact that our group of cases includes nine instances in which a pneumococcus infection of the lung was followed by a streptococcus empyema. On the other hand, we have seen nothing clinically that would seem to substantiate this hypothesis, and since we have adopted careful methods of isolation of cases in the wards by means of screens and punctilious gowning, capping and veiling of attendants, nurses, physicians and convalescent patients, no change in the incidence of empyema has been evident. It is interesting to note that our camp was entirely free from streptococcus empyema until we received a large contingent of colored troops from another encampment said to have suffered much from this condition. This complication is no longer limited to "ultrabrunettes" with us at Camp Upton.

The likelihood of an autogenous infection from streptococci already in the mucous tract of patients before they contract the pneumonia seems to us a much more probable factor. This has received suggestive corroboration at Camp Upton from a series of cultures made during an epidemic of streptococcus pharyngitis in the convalescence of which a considerable number of streptococcus pneumonias developed.

PATHOLOGY

Altogether, twenty-seven of the empyema cases came to necropsy. Three of these (Cases 6, 9 and 10) were pneumococcus empyemas, one gave sterile cultures and the remaining twenty-three all showed streptococci in smears and cultures.

The three pneumococcus cases were associated with typical lobar pneumonia, with consolidation in the side involved. The fluid in all three of these cases was frankly purulent, and in one case there were pockets of pus between the lobes of the lung. One case was associated with acute vegetative endocarditis of pneumococcus origin. In all three cases the pleura was covered with a thick layer of fibrin, which could be readily removed from the underlying membrane.

The one post mortem case (Case 70) of empyema from which cultures during life had been sterile showed at necropsy pulmonary tuberculosis and tuberculous pleuritis.

The remaining group of twenty-three cases all showed streptococci in pleural exudate. Three of these cases (Cases 56, 62 and 64) belonged to the mixed group of infections. They had been classified from sputum examination as pneumococcus infection of the lungs. The cultures from the pleural exudate gave in two cases a non-hemolyzing type of streptococcus, and one case the hemolytic streptococcus. At necropsy all showed typical lobar pneumonia, but in addition, patches of bronchopneumonia were present in those portions not involved in the lobar process. In three other cases (Cases 53, 54 and 55) bacteriologic examination of both sputum and pleural exudate gave the nonhemolyzing or green streptococcus. All three of these cases showed a patchy type of consolidation and were classed as bronchopneumonias. The empyema in this group was of the sacculated type and the fluid was cloudy, yellow and full of flecks of fibrin. In two cases (Cases 54 and 55) which showed purulent pericarditis, the process was apparently an extension by direct contiguity from pleura to pericardium, for the pleura adjacent to the pericardium was extensively involved.

The third subgroup in this division is that in which the process in the lungs and the pleura was all of *Streptococcus haemolyticus* origin. There were seventeen of these cases that came to necropsy. Eleven were diagnosed as bronchopneumonia and six as lobar pneumonia. The empyema in this group was described as sacculated in half the cases; in the other cases the fluid was lying free in the pleural cavity. The fluid was sometimes cloudy and amber-colored and sometimes distinctly purulent. In the former group there were often a number of large, entirely separate pockets which did not communicate with one another. One such pocket was frequently found between the pericardial sac and the anterior flap of the left lung which lies over it. This probably accounted for the frequency of pericarditis in this group.

The lung on the affected side in these cases is covered with a fibrinous coat which varies in thickness with the age of the empyema. It is closely adherent to the pleura. The lungs in these streptococcus cases

have nearly all shown the changes described by Cole and McCallum as interstitial bronchial pneumonia. The lung is atelectatic, due to the compression caused by the fluid. On section the bronchioles stand out with remarkable distinctness and are usually filled with pus. The walls of the bronchioles appear thicker than normal and there is an over-growth of fibrous tissue throughout the organ which accentuates the fibrous trabeculae. The thickening about the bronchioles may be so marked as to give them the appearance of small tubercles or miliary abscesses. Even in those cases in which a part or all of a lobe is consolidated, these changes have been observed in other parts of the lungs.

Microscopic sections of the lungs in these cases show areas of bronchopneumonia around the bronchioles and an extensive infiltration of small round cells about the bronchioles and blood vessels. There is a marked tendency toward organization of the exudate in the alveoli, which consists of unusually dense fibrin, lymphocytes and desquamated epithelium.

The pleural covering has been converted into a thick coat of granulation tissue, on the surface of which there is a layer of fibrin and leukocytes. Small miliary abscesses have been observed in cases, sometimes occupying the site of a bronchiole and at other times situated so as to displace a number of the alveoli. In one case these minute abscesses were located about the small blood vessels.

OTHER PATHOLOGIC CONDITIONS ASSOCIATED WITH EMPYEMA

The only other pathologic condition which occurred with any frequency was suppurative pericarditis. This was found altogether in nine cases, eight of which were in the streptococcus series and one in the cases of mixed infections. In one other case a fibrinous pericarditis was found, and in this latter case it was easy to understand why the pericarditis accompanies the empyema so frequently. That portion of the parietal pericardium which lay in immediate contact with the left pleura was intensely reddened and covered with a fresh deposit of fibrin. The infection had apparently extended into the pericardium by direct contiguity, probably by way of the lymphatics. In quite a number of these infections of the pericardium a pocket of pus was found between the pericardial sac and the adjacent portion of the left lung.

Abscess of the lung was the next most common pathologic finding and was noted in five cases. These abscesses were usually small and multiple, but in one case there was a single large abscess located in the center of the consolidated area.

Infarcts were found in two cases (10 and 38), once in a pneumococcus empyema and once in a *Streptococcus hemolyticus* case.

Acute vegetative endocarditis was noted in one of the pneumococcus cases (Case 10), while acute general peritonitis (Case 46) and acute suppurative meningitis (Case 53) were each observed once, and each in connection with a streptococcus infection.

Bilateral empyema was noted in only two cases, once in a pneumococcus case and once in a streptococcus case.

It will be seen from this summary that in spite of the virulence of this infection it was usually confined to the lungs and pleura, with occasional involvement of the pericardium. This would seem to indicate, as already noted, that the disease is not essentially a septicemia.

TREATMENT

It has not been our original intention to attempt to consider this phase of the subject extensively in this paper. Perhaps our mortality rate in the streptococcus pneumonias should suffice as an excuse for this reticence on our part, but we do feel it necessary at least to outline what in our experience should not be done in the hope that it may be of assistance to others who are dealing with these same problems. At the outset we feel ourselves forced to say that the unfortunate interference of lay military commanders in the matter of necropsies must be held responsible to some extent for failures in treatment and the much longer and costlier method of forcing us to solve this problem by experiment rather than from the more exact knowledge which the postmortem alone gives to serious inquiring clinicians.

It is of course obvious that since most cases of streptococcus empyema develop very early in the course of pneumonia, it cannot do in case operative measures are decided on to use general anesthetics, nor does it seem wise to trust the after-treatment of such cases to surgeons alone. Entire cooperation must exist between the medical and surgical departments, and these patients, even though operated on, should be still considered as properly under medical charge.

In ordinary empyema of pneumococcus type we think there can be but little question that in the larger number of cases diagnosis should be followed by early operation. This older idea has been confirmed in our minds by such cases of this class as have occurred in this epidemic. As in the usual run of cases, operation has in most instances been followed by prompt benefit and reasonably early recovery. The results have been decidedly different in our cases of streptococcus empyema, as also in those so-called sterile empyemas which we believe are likewise of streptococcic origin.

In eleven of our twenty-two fatal cases in the *Streptococcus haemolyticus* group the men have been operated on, most of them in the relatively early stages of the empyema, and of the fifteen men who have recovered or are still in the hospital, but eight had a resection done. Study of this recovery list, however, has apparently shown that those patients operated on early have, almost without exception, died, while those in whom the operation has been postponed until the pus became thick and gelatinous (frequent aspirations in the meantime having been made), recovered.

As a result of our study of this group we have naturally arrived at the practice of withholding our cases from the surgeon until later in the course of the disease, when the pus has become thick with fibrin and cells.

We have felt that our better success since this method had been adopted has depended in part on the fact that when the pleural cavities are drained through the active stage of the pneumonia the rapid collapse of the diseased lung on the operated side has thrown an added strain on the unprepared heart, which has been a dominant factor in the cause of post operative death. Furthermore, when the waiting policy is pursued, not only may the pneumonia improve and the heart adjust itself to the strain thrown on it, but it has been our observation that the pleura becomes so thickened as a result of the exudate that the subsequent collapse of the lung following thoracotomy is less marked.

We may summarize our present procedure briefly as follows, but while our results are now better than formerly, we at the same time acknowledge that they are very far from satisfactory.

The case is carried as an ordinary case of pneumonia. As soon as the fluid is discovered (and with the persistent use of the needle and roentgen ray this is usually very early determined) the aspirated diagnostic specimen is examined by smear and culture. If the exudate is clear, notwithstanding the presence of streptococci, it is allowed to remain in the chest until it is apparent that the fluid is embarrassing respiration, in which case it is aspirated by the Potain aspirator. This aspiration is repeated as frequently as seems indicated, and careful watch is maintained on alterations in the character of the fluid. When it becomes thick and viscid, the case is deemed to have reached the stage for surgical intervention.

Meantime, of course, the general symptomatic treatment of pneumonia has been followed. As a rule, digitalis is given freely in the early stages and camphor, epinephrin, caffein, etc., are given as symptomatically indicated. The usual attention is given to elimination, to abundant aeration, and in suitable cases the patients are placed out of doors exposed to the sun and air. Temperature is controlled in part,

at least, by hydrotherapy and aërotherapy, and where much pain is suffered or when the patient coughs incessantly or becomes excitable, codein or morphin is used as indicated.

We are most anxious to attempt treatment of these cases by the use of an antistreptococcus serum, and steps in this direction are now being taken. We feel that until some protective mechanism is discovered we shall continue to find these cases most unfortunate problems in military medicine.

A marked improvement in results has followed the surgical treatment of these cases of hemolytic streptococcus empyema since they have been placed exclusively under the care of a single surgeon (Captain Perry), who has made a special study of this condition, and particularly of its surgical management.

SUMMARY

A highly virulent type of empyema has been prevalent at Camp Upton during the winter and spring months of 1918.

Eighty cases have been observed in a series of 300 pneumonias.

Fifteen of these were caused by the pneumococcus; fifteen others were sterile; the remainder of the cases were caused by a streptococcus, usually of the hemolytic type.

In this series of cases empyema has been constantly associated with pneumonia of either the bronchial or lobar type. Usually a bronchopneumonia has accompanied a streptococcus empyema, and a lobar pneumonia the pneumococcus empyemas.

In the great majority of cases the organism which has been found to be the causative agent in the pneumonia has also been isolated in pure culture from the pleural exudate. In nine cases, however, a pneumococcus pneumonia was associated with a streptococcus empyema.

The onset of the pneumonia and the empyema is apparently simultaneous in some cases.

This epidemic of empyema has been, in large part, independent of measles and other similar infections.

The clinical symptoms and the physical signs differ from those of classic empyema.

Diagnosis is frequently difficult by reason of the equivocal physical signs and the indefinite symptomatic picture. Diagnostic aspirations are the most certain means of discovery of the condition in its early stage. The roentgen ray stands second in diagnostic importance. Of the physical signs, alterations in percussion seem to be the most helpful.

The interstitial type of bronchopneumonia, recently described by Cole and MacCallum, is usually found associated with streptococcus empyema.

Fibrinopurulent pericarditis is the most frequent complication. Pyopneumothorax has also been observed in a number of cases.

The mortality rate is high and the prognosis is serious.

Medical treatment is essentially expectant.

The most efficacious method of surgical treatment and the correct time for its application are yet to be determined.