

It is not possible, at present writing, to make any definite statement as to the proportion of cases of lobar pneumonia in which foregoing pleurisy occurs. On theoretical grounds, however, we can readily conceive of primary pneumococcic infection of the pleura with subsequent involvement of the lung texture from extension. Perhaps the majority of instances belonging to this class are met with in the secondary form of lobar pneumonia, or that which arises in the course of the acute infections and certain chronic diseases, such as, chronic nephritis, organic heart affections, and the like.

To distinguish the cases of pleurisy under consideration from those presenting identical signs, but without any tendency to merge with ensuing pneumonic features (simple pleurisy), is often difficult in the extreme. An initial chill may occur in cases in which pleural involvement, followed by lobar pneumonia, arises in apparently healthy individuals; this should invite attention to the possibility of its having been a pneumococcic infection from the beginning. On the other hand, the rigor may be absent in such cases. An examination of the blood usually reveals a leukocytosis, which often develops rapidly, in the hybrid under discussion. Again, the systemic prostration is greater and the temperature range higher than in non-pneumococcic pleurisies. In suspects, it is well to attempt to demonstrate the pneumococcus in blood cultures, if no pleural exudate be obtainable, but for this purpose large quantities of blood (not less than 15 c.c.) are required.

A bacteriological study of the exudate when present is invariably to be advised and encouraged, and it is the only reliable means of drawing an etiological distinction between the recognized varieties. It is to be recollected also that pneumococcic pleurisy may occur quite independently of any involvement of the lung texture; but the cases falling under this head are rare, and occur oftenest among children with symptoms resembling those of pneumonia, although the physical signs are those of pleural effusion. It is possible, as some contend, that patches of consolidation, of larger or smaller size, are undiscovered; hence these would be examples of pneumonia complicated or cloaked, as it were, by a pleural effusion.

**INCIDENCE OF SEROFIBRINOUS AND PURULENT VARIETIES.** The relative proportion of cases in which the exudate is purulent or non-purulent when pleurisy complicates pneumonia is not definitely known, although there are at hand a considerable number of figures bearing upon the question. Fraley,<sup>2</sup> in 52 cases of pleurisy occurring in connection with 500 cases of lobar pneumonia, found that only 4 were of the serofibrinous variety, while 48 were empyemas. On the other hand, Norris,<sup>3</sup> in an analysis of 500 cases of croupous pneumonia, noted pleurisy as a complication in 18, of which, 12 were of the serofibrinous form and 6 were empyemas. In dealing with

<sup>2</sup> AMER. JOUR. MED. SCI., May, 1907

<sup>3</sup> Ibid., January, June, 1901, p. 684.

the question of pleural effusions in children as related to lobar pneumonia, two points deserve especial notice: First, as compared with adults, purulent effusions from all causes are more frequent relatively than serous, and secondly, during early childhood not less than 75 per cent. of empyemas from all causes are due to the pneumococcus according to the figures of Netter, Blaker, Bythell, and others.

It has been pointed out repeatedly that pleural effusions in children are generally purulent from the beginning, whereas in adults empyemas are more apt to originate from a serofibrinous exudate. Says Emanuel,<sup>4</sup> pertinently: "If we are going to compare an empyema of a child with one in an adult, we must take pains to compare empyemas which are alike bacteriologically." At present, however, it is an accepted fact that practically all pleurisies found in connection with pneumonia are due to the pneumococcus.

A consideration of available statistics for all periods of life by Musser and Norris,<sup>5</sup> leads to the inference that empyemas, if we include metapneumonic cases, are of common occurrence, but by no means so frequent as serofibrinous effusions in connection with lobar pneumonia. These observers found that of 24,511 cases of pneumonia, pleural effusion occurred in 1535, or 6.26 per cent. At a later date, Cameron<sup>6</sup> analyzed 217 cases of primary pneumococcal pneumonia, personally observed, of which 65 were over twenty years of age and 120 under that age (32 not mentioned). Of these, 15 were complicated with empyema—only 7 of the latter patients being over ten years of age, and 8 under. "Of 1331 cases treated in Guy's Hospital, empyema developed in 45, or 3.3 per cent."

On the other hand, in 974 autopsy records in pneumonia, pleural effusions were encountered in 415 or 41.58 per cent. Again, of 973 necropsies in pneumonia cases, 50, or 5.1 per cent., were empyemas. Norris autopsied 127 pneumonia cases, and found pleuritis in 59, or 46.4 per cent., as follows: Fibrinous, 23; serous, 5; fibrous, 11; and purulent, 20. Kerr, in 173 pneumonia autopsies, obtained results that differed markedly from the above, namely, acute fibrinous, 74; acute serofibrinous, 78; and acute purulent (empyemas), only 6. Obviously, the results from postmortem examinations show a larger number of cases of serofibrinous and fibrinous pleurisy proportionately than antemortem observations, for the reason that the condition is of too light a grade in many instances to be detected during life by means of the physical signs. On the other hand, the antemortem and postmortem observations in cases of empyema do not seem to present the same discrepancy.

**BACTERIOLOGY.** It is to be recollected that the gravity of the condition depends so largely upon the particular species of bacteria

<sup>4</sup> Reports of the Society for the Study of Disease in Children, 1906, vi, 75.

<sup>5</sup> Osler's Modern Medicine, 1907, II, 597.

<sup>6</sup> Australasian Medical Gazette, Sydney, November 20, 1908.

<sup>7</sup> Matheny, Surgical Complications of Pneumonia, with Special Reference to Empyema, Penna. Med. Jour., March, 1909.

present in the given case of pleuritis as to make the question of its bacteriological cause one of the utmost practical importance. That the pneumococcus has great power of invasion of the pleural cavity from the lung is an accepted fact, but when this extension of bacterial activity takes place in cases in which empyema arises as a sequel of pneumonia is still unknown. The pneumococcus is often found in a state of purity in pleurisy complicating pneumonia, but it may also occur mixed with the streptococcus, the staphylococcus, the tubercle bacillus, and rarely with other organisms.

As before stated, the pneumococcus is quite generally the bacteriological factor in pleuritis associated with pneumonia, whether they be serofibrinous or purulent. While the bacteriological work that has been done in empyemas secondary to pneumonia has given results which may be regarded as definitely conclusive, the same is not true of serofibrinous effusion. To determine the bacterial agent present in the latter exudate is confessedly difficult, but fortunately it may be fairly assumed that almost invariably the pneumococcus is found in pure culture. As in the case of pneumococcic empyemas, however, so here, other microorganisms, especially the streptococcus, or the staphylococcus, may rarely be found mixed with it. In cases of pneumonia in which the streptococcus occurs in the pleural cavity, it also occupies the lungs in association with the pneumococcus, and may excite pulmonary abscess. The foregoing facts concerning the bacteriology of pleurisy found complicating pneumonia must be borne in remembrance in view of their practical bearings upon its prognosis and treatment.

**DIAGNOSIS OF PLEURITIS, THE COMPLICATION IN PNEUMONIA.** The diagnosis of associated dry or plastic pleurisy, the commonest variety, can be definitely recognized by the friction sound, except in the stage of complete solidification of the lung when it is missing owing to the absence of any expansive motion of the chest. Both in the first and third stages of these cases, pathologically speaking, the friction rub, however, is generally detectable, if care be exercised in the examination. We must recognize the well-established pathological truth that a certain, although often mild, grade of pleurisy is present in practically every case of pneumonia. Moreover, friction sounds may be detectable at any stage of pneumonia in cases in which the lung is imperfectly solidified. A serofibrinous exudate occurring in connection with pneumonia is usually recognizable by means of the physical signs, which, however, are modified by the consolidated lung texture. (Physical signs of empyema, *vide infra*.)

As to the time of occurrence of the complicating pleurisy, there is wide discrepancy of opinion among authors and writers, some contending that it more often develops during the course of the fastigium than as a sequel, while others affirm the contrary view.

In cases of pneumonia with complicating serous pleurisy, the crisis followed by resolution generally occurs at the customary period of the disease, and the effusion may then disappear with extraordinary

rapidity. A case in point has recently fallen under my care at the Medico-Chirurgical Hospital, in a female, aged twenty-six years, and this experience has not been an uncommon one.

In a second group of cases, the physical signs of effusion may be either well defined or more or less indefinite, but the fever is protracted beyond the usual time for the occurrence of the crisis. Recent experience, has shown me that such a temperature chart is of even greater value in the determination of associated pleuritis than is generally supposed. The curve remains irregularly elevated, but does not present the septic type, unless the pleural infection be caused by the streptococcus. That an intermittent form of fever curve may rarely be observed, due to mixed infection, must be granted. A gradual fall of temperature in pneumonia in lieu of the more common crisis is oftener due to associated pleuritis than to any other single cause.

A rising leukocytosis with the onset of a secondary pleurisy has been noted by A. Lambert and R. M. Daley,<sup>\*</sup> in one case the count varying from 19,600 to 25,400. Granting that further observations of the leukocyte count will confirm this finding, it must be recollected that extension of the pathological changes of the pneumonic process, and complications other than serofibrinous pleurisy, may likewise cause a sudden rise of leukocytosis. It may be said at present that this symptom is not sufficiently established to be of more than corroborative value as a diagnostic factor in pneumonia with complicating serous effusion.

Movable dullness or flatness, that most characteristic sign of a liquid exudate, should not be investigated for, since to place the patient ill of pneumonia in the sitting posture during the attack may be attended with distinctly harmful effects. A sign of high value in the diagnosis of complicating pleurisy is absence of vocal tactile fremitus. It is well to resort to the exploring needle in a dubious case after noting the physical signs, and it is my custom to employ for the purpose a hypodermic syringe having a needle somewhat longer and of slightly larger caliber than that ordinarily used in subcutaneous medication. In serofibrinous effusion a larger needle is rarely required, although it is not safe to exclude pleurisy if fluid fails to be obtained on so-called needling. It is the part of wisdom to resort to a full-sized exploring needle in highly suspicious cases, if occasion demands. Here I may be allowed to state my firm belief, based on personal observation, that shock, even of the slightest degree of intensity, is badly borne by pneumonia patients, and it is for this reason that the smallest needle that will suffice is to be preferred.

Although most serofibrinous pleurisies are pneumococcal, a careful bacteriological and cytological examination of the exudate is nevertheless to be advised. Thus, as is well known, if the withdrawn

<sup>\*</sup> Leukocytosis in Pneumonia, St. Paul Med. Jour., 1902, p. 849.

fluid should contain a preponderance of small mononuclear cells or lymphocytes, the pleurisy is probably tuberculous in nature. Here may be pointed out the fact that concomitant infection of the pleura with both the tubercle bacillus and the pneumococcus may occur in rare instances, and the tuberculous infection is more commonly primary than secondary in such cases. This opinion is confirmed by that of Bythell, who has reported 4 cases in which "latent tubercle bacilli were stirred into activity by the pneumococcus infection."

In every case, therefore, it is important definitely to determine the presence or absence of pathogenic activity, due to organisms other than the pneumococcus, even though it be confessedly difficult to determine the bacterial agent by staining films after centrifugalization, by cultural methods, or, finally, by inoculation experiments.

Again, serofibrinous pleurisy may develop as a sequel, *e. g.*, one, two, or more days after the occurrence of the critical decline of fever. These postcritical serous pleurisies, if pneumococcal in origin, usually run a mild course with a moderately elevated temperature, the curve presenting no particular type. A pneumococcal serofibrinous pleurisy, however, may though rarely, pursue a severe course; when caused by the streptococcus, the pleurisy is generally accompanied by septic phenomena with a serious clinical aspect. Phagocytosis of larger or smaller amount may be present, particularly in the latter variety. In these residual or postpneumonic serous pleurisies, cough of an unproductive, irritative, useless nature is apt to occur and is aggravated by movement. Movable dullness may be elicited in these cases with safety to the patient. If, however, doubt surround the diagnosis, a needle should be introduced. The hypodermic syringe may not answer our purpose here, as was true of 2 of my cases, in which the effusion was decidedly fibrinoplastic in nature.

In serofibrinous pleurisy, as in empyema, investigation must embrace the entire chest, and it is to be especially recollected that pleuritis may attack the non-pneumonic side—a most serious condition if it develop prior to the occurrence of the crisis, and one that must be promptly met. Finally, while it is quite generally possible to recognize the existence of fluid exudate in the pleural sacs when present in any appreciable amount by noting carefully the physical signs, it is impossible definitely to determine whether it is a serofibrinous or purulent effusion without resorting to an exploring needle. I may add that he who entertains the belief that the distinction between pus and serum can be drawn in any other manner is only deceiving himself.

**DIAGNOSIS OF EMPYEMA OCCURRING AS A COMPLICATION AND AS A SEQUEL OF PNEUMONIA.** As before stated, almost all of the empyemas that develop during and after pneumonia are due to the pneumococcus pure, although double infection is not unknown.

The condition in any case may, as Bythell<sup>10</sup> points out, extend its operations beyond its original seat of infection, thus possessing a dangerous power. Concerning the time of development of empyema in connection with lobar pneumonia, that is, whether before the crisis as a complication, or after that pivotal event as a sequel, opposite opinions are held. The medical profession is greatly in need of reliable statistics bearing upon this question. With the kind aid of my associate, Dr. H. Leon Jameson, I have been able to collect from the literature of the past two decades 218 cases, of which, only 66 could be classed as true complications, while 152 occurred as sequels.

Reporter and reference.	Empyema pre- ceding crisis (complication)	Empyema following crisis(sequel).
Flagler, Amer. Lancet, 1890, xiv, 363 . . . . .	..	1
Begg, Cleveland Med. Gas., 1890-91, vi, 308 . . . . .	..	1
Drummond, Brit. Med. Jour., 1891, ii, 113 . . . . .	2	
Holt, Proc. New York Path. Soc., 1892, p. 90 . . . . .	1	
Bureau, France Med., 1892, xxxix, 177 . . . . .		1
Carr, Trans. Clin. Soc., London, 1892-93, xxvi, 29 . . . . .	1	
Russel, Brit. Med. Jour., May 6, 1893, p. 949 . . . . .	..	1
Bartelling, Trans. Indiana Med. Soc., 1893, xlv, 163 . . . . .	..	4
Crandall, International Clinics, 1893, vii, 3d ser., 167 . . . . .	1	1
Sutherland, Lancet, 1894, i, 1441 . . . . .		2
Fox, Lancet, 1894, ii, 394 . . . . .	..	1
White and Pearce, Guy's Hosp. Reports, 1894, ii, 13 . . . . .	5	9
Coulpaud, Middlesex Hosp. Rep., 1894, p. 65 . . . . .	1	
Hughes, Univ. Med. Mag., vii, 1894-95, p. 131 . . . . .	1	3
Withington, Boston Med. and Surg. Jour., 1895, i, 5 . . . . .	3	6
Lockwood, Medical News, 1897, lxxi, 818 . . . . .	..	1
MacKinnon, Brit. Med. Jour., 1897, ii, 1057 . . . . .	2	2
Shelden, Wisconsin Med. Recorder, September 1898, 191 . . . . .	1	
Buchanan, Liverpool Med.-Chir. Jour., 1898, xviii, 305 . . . . .	2	
Seun, Jour. Amer. Med. Assoc., 1898, xxxi, 1497 . . . . .	..	5
Blaker, Brit. Med. Jour., 1903, i, 1200 . . . . .	9	53
Rowley, Brit. Med. Jour., 1903, i, 1203 . . . . .	1	1
Oechsner, New Orleans Med. and Surg. Jour., 1903-04, lvi, 98 . . . . .	1	2
Borland, Glasgow Med. Jour., 1904, lxi, 53 . . . . .	..	2
Donald, Detroit Med. Jour., 1904-05, iv, 300 . . . . .	..	2
Heiman, Mt. Sinai Hospital Reports, New York, 1905, iv, 125 . . . . .	..	1
Leys, Amer. Jour. Med. Sci., 1906, cxxxii, 8 . . . . .	..	4
Hall, International Clinics, 1906, iv, 15th Series, 48 . . . . .	5	12
Landeen, Jour. Minn. Med. Ass., 1906, xxvi, 244 . . . . .	2	1
Smith, Med. Bulletin, 1906, xxviii, 375 . . . . .	..	1
Talley, Jour. Amer. Med. Assoc., xlvii, 730 . . . . .	1	4
Allen, New York Med. Jour., May 26, 1906, p. 1077 . . . . .	..	1
Vickery, Boston Med. and Surg. Jour., 1907, civi, 501 . . . . .	..	3
Abeken, U. S. Naval Bulletin, 1907, i, 161 . . . . .	..	1
Craig, Brit. Med. Jour., 1907, ii, 710 . . . . .	1	
Bramwell, Clinical Studies, Edinburgh, 1907-08, vi, 379 . . . . .	..	1
Parsons, Canada Lancet, 1907-08, xli, 592 . . . . .	..	1
Dunn, Virginia Med. Semi-Monthly, Sept. 25, 1908, p. 266 . . . . .	..	1
MacDonald, Maritime Medical News, 1908, xx, 174 . . . . .	1	3
Burrus, Atlanta Jour. Rec. Med., 1908-09, x, 374 . . . . .	..	2
McCrae, Personal Communication . . . . .	25	4
Hall, Trans. Amer. Clin. Assoc., 1909, xxv, 182 . . . . .	..	8
Bannerman, Brit. Med. Jour., 1909, ii, 201 . . . . .	..	1
Lake, Trans. New Hampshire Med. Soc., 1910, x, 192 . . . . .	..	2
	66	152

An examination of the literature also revealed the fact that most writers who have reported cases of empyema in association with pneumonia have spoken of it as being a sequel in the majority of instances, at least. On the other hand, Thomas McCrae and A. Lambert contend that a preponderance of the cases arise before the crisis, and are, therefore, to be regarded as complications. A considerable number of instances were eliminated from consideration because of the fact that the time of onset could not be accurately determined from the accounts given. The selection of case reports was influenced largely by the temperature curve; thus, those in which the crisis occurred at or about the usual period followed by a fresh rise of fever anywhere from one to several days or more thereafter, coupled with the simultaneous appearance of the characteristic physical signs, were classed as sequels of the disease. Again, all instances that developed before the time for the crisis (and often prevented its occurrence) were regarded as complications, and so reported in the accompanying table.

The general features of pneumococcic empyema, whether it develops as a complication or a sequel, are usually more or less indefinite. In some cases, however, in which either a fistulous connection with a bronchus, or an empyema necessitatis, is established, chills, an exacerbating temperature curve, and sweats may occur.

*Empyema, the complication*, usually causes the temperature to fall by lysis, and an irregular fever curve may persist for weeks together in cases that go unrecognized and in those inappropriately treated. The examiner is, as a rule, enabled to recognize the presence of free purulent effusion by means of the physical signs.

The solidified state of the underlying lung, however, tends to modify certain of the more characteristic signs as compared with those noted in cases in which the lung is healthy at the time of onset of the pleurisy. The tracheal sounds, therefore, are readily conveyed through the bronchi, and even through the fluid with slight change unless the effusion be copious in amount. Vocal resonance is also decreased to a less extent than in cases in which the lung is retracted or compressed by an exudate arising independently of lobar pneumonia; and the breath sounds, while faint or even inaudible at the extreme base, are apt to be more bronchial at lower levels than in primary empyema. The note to percussion, however, is flat with board-like resistance. In children, vocal fremitus is of little value as a diagnostic aid, and the breath sounds, except at the base posteriorly, are apt to be distinctly bronchial in quality over the effusion, and associated with augmented vocal resonance. As stated in connection with serofibrinous fluid, one must use the exploring needle, which in these cases should not be too small.

The diagnosis of *empyema occurring as a sequel* of pneumonia is free from difficulty, as a rule, if one does not neglect to note care-

fully the physical signs in any given case in which, following the usual crisis, irregular fever returns, the prostration increases, and there occurs a marked rise of the leukocyte count. These show the existence of pleural effusion, which invariably calls for an immediate exploratory puncture, followed by a bacteriological study of the withdrawn exudate. It should be further emphasized that the empyemas, which originate during the progress of a pneumonia, and in which the exudate is free in the pleural cavity, do not require exploratory operation for their recognition, as a rule, although it may become necessary to needle occasional obscure cases for purposes of diagnosis.

In the diagnosis of a *localized* (encapsulated) *empyema*, or one engrafted upon a pleura previously the seat of disease, many points of difficulty are presented. These small collections of pus rarely cause any noticeable bulging of the chest wall. A restricted expansive movement over a circumscribed area, especially during forced respiration, is an early and significant sign. Palpation will confirm inspection relative to the expansile motion of the chest, and will elicit diminished, or even absence of, tactile fremitus over the fluid, provided that the examiner uses his finger tips; but to detect this sign he may be obliged to cover every square inch of thoracic surface. Should the abscess, however, occupy a position between the lobes with more or less consolidation lying anteriorly, tactile fremitus may be increased rather than decreased, although this particular relationship of pathological conditions rarely obtains.

A most valuable sign, emphasized by Musser,<sup>11</sup> is localized tenderness to firm pressure over the interspace corresponding to the seat of the abscess. There may be also subjective pain; this is generally but not always referred to the circumscribed area of tenderness. Thus, it may be felt below the costal margin as in one of my cases, or anteriorly, at about the point where the intercostal nerves emerge from the deeper to the more superficial anatomical structures. When the localized infection implicates the diaphragm, the pain may be referred to the scapular region. On percussion, a flat note is usually obtained co-extensive with the abscess, but movable dullness is generally absent in these encapsulated empyemas. Neither does my personal experience confirm that of other observers, who have noted Skodaic resonance immediately over the loculated pus collection; but quite commonly this note is present over the surrounding lung texture when not the seat of consolidation.

Auscultation reveals either diminished or absent breath sounds, depending upon the size of the abscess. Thus in one of my cases, which was also seen by my surgical colleague, Dr. W. L. Rodman, a slight diminution in the intensity of the vesicular element of the breath sounds was the only auscultatory phenomenon. In this



instance the abscess proved to be small, and after evacuating the pus recovery ensued promptly. When the abscess is situated between the lobes of the lungs (interlobar) near to their margins, a pleural friction sound may be audible in the earlier stages, at least, and tends, when coupled with the other signs, to localize the seat of the collection.

These small loculated empyemas are rarely recognizable by means of the physical signs alone, but in the presence of the general and local features of infection, particularly irregular fever, and the history of an antecedent pneumonia, we are justified in according to them a strong corroborative value, and they will often indicate the necessity for (and the site of) prompt surgical intervention.

The x-rays are of considerable value in detecting small or larger collection of pus in the pleural cavities, since they produce a denser shadow than that of a resolving pneumonic consolidation, which is so commonly a concomitant condition. Its circumscribed character will serve to distinguish an abscess from mere thickening of the pleura. It must be confessed, however, that the results of radiography are not always definite, and they must, therefore, not of themselves be regarded as conclusive. The results of x-ray studies, however, often aid us in deciding in favor of further explorations.

It is always difficult and sometimes impossible to differentiate an encapsulated empyema following pneumonia (abscess of the pleura) from pulmonary abscess without communication with a bronchus. This is particularly true if the purulent pleurisy be of the interlobar variety, and it must be recollected that abscess of the lung may be an associated condition. In pulmonary abscess, when it communicates with a bronchus, the sputum contains elastic fibers and shreds of lung tissue, both of which can be readily demonstrated. If the condition develop during the attack of pneumonia, the sputum changes in color appearance; it may become yellow, indicating its purulent character, grass green, or "hemorrhagic or blackish" (Leyden). The sputum emits an "offensively sweet" or even fetid odor. The signs of cavity formation develop if the abscess communicate with a bronchus, and these may be readily confirmed by means of the x-rays.

Exploratory puncture should be resorted to without delay in cases in which the special local and general features and physical signs are indefinite, but the failure to find pus in this manner must not lead us to exclude loculated empyema. The causes of a negative result are several. In the first place, the purulent collection may be small and environed by thick, dense adhesions, hence difficult to locate. Again, the instrument may be imperfect, or of too small a caliber. I am of the opinion that, notwithstanding failure to prove the presence of pus by puncture in cases in which the history, signs,

and symptoms leave little room for doubting the existence of an accumulation, an exploratory operation should be undertaken.

The physician, no less than the surgeon, must realize the dangers, both to the lung and general condition of the patient, of such collections, if allowed to remain, unless nature should be so fortunate as to be able to establish a fistulous connection with a bronchus or bring about rupture externally. On the other hand, the general condition of the patient may be such as to prohibit a safe operation, or render delay advisable for a time, but timely use of the method is to be advised in all instances in which distinct contra-indications do not exist. Indeed, to permit a circumscribed pleurisy following pneumonia to drag on with a view to waiting until spontaneous rupture occurs is inexcusable. Exploratory operation will not only clear the diagnosis, but also accomplish the cure in the immense majority of the cases.

Here I should not fail to point out that a concomitant non-loculated empyema (one developing in the course of the pneumonic process) is more benign than so-called metapneumonic empyema, which begins after the crisis, often in greatly debilitated subjects.

Exploratory puncture and timely surgical intervention will prevent shrinking of the lung followed by thoracic deformity in metapneumonic empyema. On the other hand, when the condition is too conservatively treated, or goes unrecognized, most cases terminate fatally after a long-continued fever, with chills and gradual exhaustion. The cases that rupture through the diaphragm, setting up subphrenic abscess, eventually die, if not recognized and treated surgically.

**TREATMENT.** It is manifestly true that the treatment of pleuritis developing before the crisis in pneumonia may require different management from that appropriate in cases arising after the occurrence of that pivotal event. When a serous exudate supervenes in the course of the pneumonic process, an expectant method of treatment, as before intimated, is to be advocated, first, because puncture followed by aspiration is badly borne in pneumonia cases, and secondly, for the reason that if the crisis is survived by the patient, the exudate quite generally is absorbed with surprising rapidity. If, however, the serous effusion does not promptly disappear following the crisis, recovery is to be hastened by puncture, which in my experience has been found to be successful. Again, the removal of a part of a massive effusion, which has displaced the heart and other adjacent viscera, is rarely necessary during the fastigium, and favors spontaneous resorption. Such cases, however, may rarely demand more radical operative measures for their cure later on (after the crisis), and these should not be too long delayed.

On the other hand, a complicating serous pleurisy may prevent the occurrence of the crisis in lobar pneumonia, fever of an irregular type continuing indefinitely, or the temperature dropping by lysis.

In such cases, if the effusion be copious and remain undiminished in amount, puncture should be undertaken early, and repeated if necessary.

Before leaving this aspect of my subject—the treatment of serofibrinous exudate in the course of pneumonia—I wish to emphasize the point that my own empiric results from aspiration in this disease prior to the time of the crisis or the beginning of resolution of the hepatized lung in seriously ill patients have been uniformly unfavorable, although I am ready to admit that other observers may deduce indications for its employment either by scientific reasoning or from experience.

*Medical Measures.* Remedial measures intended to control pneumococcic inflammations of serous membranes are of small value, and it is of the utmost importance to avoid the use of drugs that act as depressants in all pneumonia cases. The remedies that exercise a favorable effect in serous pleuritis in general are opium, quinine, and the salicylates. In pneumococcic pleurisy, the salicylates are to be eschewed owing to their depressing action. Opium may be resorted to during the early stage of the primary disease, pneumonia, to relieve the pains of a complicating pleuritis, but it should not be employed during the advanced periods, when the bronchi contain secretory products, since it dries these, thus favoring their accumulation rather than their removal. It is seldom necessary to continue this remedy after the second day. I have had considerable experience of the use of quinine in serofibrinous pleuritis complicating pneumonia, and feel strongly that it possesses a limited value, both as a supportive agent and in mitigating the serous inflammation. It is my custom to administer the drug in 4-grain doses, in capsule, followed by a few drops of dilute hydrochloric acid to insure its prompt solution, three or four times daily.

*Local measures* may prove serviceable in selected cases. Counter-irritation by means of sinapisms is to be advised in the milder forms of associated pleuritis that may accompany pneumonia, and the same is true of cold applied in the form of an ice bag or ice-water bag. If the pain be severe, we may follow the excellent suggestion of Roberts, and keep the inflamed structures at complete rest by mechanical fixation of the side affected. "For this purpose, strips of adhesive plaster must be firmly and evenly applied to the chest; they should be removed during the stage of effusion."

The treatment of an *empyema*, whether a complication or sequel of pneumonia, is surgical. If the condition develop on the side of the pneumonia from the third to the seventh day of the illness, it is well to postpone exploratory operation or resection of a rib until resolution of the lung has progressed considerably, when, however, free and permanent drainage should be promptly established. On the other hand, if the exudate be massive, and, as a consequence, the adjacent organs, particularly the heart, be markedly displaced, then aspiration

must be immediately performed, to be followed by a more radical operation at an appropriate period of the affection. It is to be recollected, however, that even a slight degree of shock is ill-borne prior to the time of occurrence of the crisis in pneumonia, and it is for this reason that operation should be delayed until early convalescence, except under the conditions previously mentioned.

It is not my desire to discuss at length the question of the advantages of mere incision and drainage as compared with those of the Eslander operation or complete resection. I have, however, time and again, witnessed a speedy termination in recovery, both from mere aspiration of the thorax, and incision and drainage without resection of a rib in empyema, occurring as a sequel of pneumonia, most commonly in young children. On the other hand, it is the safer rule to resort to rib resection in cases of pneumococcic empyema. These patients demand an abundance of fresh air, and hence should, if practicable, be placed out of doors, or, at all events, in a highly ventilated apartment. They also demand vigorous feeding with highly nutritious forms of nourishment.

Of medicines, personal observation and experience of the use of the tincture of the chloride of iron in somewhat massive doses enables me to speak confidently of its apparent efficacy in some cases of empyema, at least.

---

## THE USE OF THE SOY BEAN AS A FOOD IN DIABETES.

BY JULIUS FRIEDENWALD, M.D.,

PROFESSOR OF DISEASES OF THE STOMACH IN THE COLLEGE OF PHYSICIANS AND SURGEONS,  
BALTIMORE,

AND

JOHN RUHRÄH, M.D.,

PROFESSOR OF DISEASES OF CHILDREN AND THERAPEUTICS IN THE COLLEGE OF PHYSICIANS  
AND SURGEONS, BALTIMORE.

THE soy bean (*Glycine hispida*), sometimes incorrectly called the soja bean, is an annual leguminous plant which originally grew in a wild state from Cochin China to the south of Japan and to Java. It has been cultivated from the very earliest times in Japan and in China, long before the time of Confucius. Notwithstanding its extensive use in China and Japan the bean has spread but slowly to other lands. It gradually found its way into India and was brought to Europe somewhat over one hundred years ago where it was grown in the botanic gardens chiefly for show. From Egasse<sup>1</sup> we learn that Koempfer, who traveled extensively through the East,

<sup>1</sup> Bulletin général de thérapeutique, 1838, cxv, 433.

**GYNECOLOGY.**

UNDER THE CHARGE OF  
JOHN G. CLARK, M.D.

Clinical and Experimental Studies Concerning the Curative Action of Laparotomy in Peritoneal Tuberculosis . . . . .	919
A Method of Anastomotic Repair of the Divided Ureter . . . . .	920
Contribution to the Operative Treatment of Benign and Malignant Tumors of the Ovary . . . . .	920
Periodic Intermenstrual Pain . . . . .	920
Late Recurrence in Carcinoma of the Uterus . . . . .	921

**DERMATOLOGY.**

UNDER THE CHARGE OF  
LOUIS A. DUHRING, M.D.,  
AND  
MILTON B. HARTZELL, M.D.

Sarcomatosis Kaposi, with Special Reference to Its Visceral Localizations . . . . .	922
A Peculiar Case of Keloid Following Injections of Camphor Oil . . . . .	922
A Histological Study of Parapsoriasis . . . . .	923
Arsenical Keratosis and Arsenical Cancer . . . . .	923

**PATHOLOGY AND BACTERIOLOGY.**

UNDER THE CHARGE OF  
WARFIELD T. LONGCOPE, M.D.

The Internal and External Secretion of the Organism in Health and Disease in the Light of Vital Staining . . . . .	924
The Alterations of Metabolism and Pathology of the Blood in Chronic Lead Poisoning . . . . .	925

**HYGIENE AND PUBLIC HEALTH.**

UNDER THE CHARGE OF  
VICTOR C. VAUGHAN M.D.,

Experimental Infection of Fowls with Anthrax by Feeding . . . . .	925
Tuberculosis Among Indians . . . . .	926

THE  
AMERICAN JOURNAL  
OF THE MEDICAL SCIENCES.

DECEMBER, 1910.

ORIGINAL ARTICLES.

**PLEURISY AS A COMPLICATION AND A SEQUEL OF LOBAR  
PNEUMONIA: ITS DIAGNOSIS AND TREATMENT.<sup>1</sup>**

BY JAMES M. ANDERS, M.D., LL.D.,

PROFESSOR OF MEDICINE AND CLINICAL MEDICINE IN THE MEDICO-CHIRURGICAL COLLEGE,  
PHILADELPHIA.

It is universally conceded that pleurisy is the commonest complication of the pneumonic process. The precise definition of the condition, however, is difficult. In my view, pleurisy should be regarded as a complication only when it co-exists either as a severe form of the dry plastic variety or is accompanied by sufficient effusion to be easily demonstrable by means of the physical signs. On the contrary, the slight dry pleurisy, which is ordinarily associated with pneumonia and disappears promptly with the occurrence of the crisis, should be looked upon as a part of the pathological picture of the disease.

Apart from the commoner forms of complicating pleuritis, which arise either during or following the active stages of the pneumonic process, and yield either serum or pus, there are instances of pleurisy, principally of the plastic variety, that clearly precede the development of a pneumococcal infection of the lung (pneumonia). This class of cases is one that has not received the measure of consideration on the part of medical writers that it seems to me to merit.

In the course of my observations of pneumonia with an atypical onset, clinical indications of dry or plastic pleurisy alone have been demonstrable by means of the physical signs during one, two, or more days, in some cases, at least. In rare instances, a moderate amount of either serofibrinous or purulent exudate may be present for a similar, variable, period of time prior to the appearance of the succeeding pneumonic features.

<sup>1</sup> Read at a meeting of the Ohio State Medical Association, Toledo, Ohio, May 12, 1910, and in a modified form at a meeting of the York County Medical Society, Pennsylvania, February 3, 1910, and the Delaware County Medical Society, Pennsylvania, July 7, 1910.