The aetiology, immediate and remote prognosis of primary pleurisy with serous effusion / by C.E. Hedges.

[Reprinted from St. Bartholomew's Hospital Reports, Vol. XXXVI.]

THE ÆTIOLOGY, IMMEDIATE AND REMOTE PROGNOSIS OF PRIMARY PLEURISY WITH SEROUS EFFUSION.[1]

BY

C. E. HEDGES, M.D. CANTAB.

It has always been a point of the greatest interest to me, and must be to others who have worked in the wards of a large general hospital, as to the fate of those patients who have been admitted with pleurisy with effusion, and, as far as one can find out, with nothing whatever else ailing them. How often does one see during the treatment that the effusion is evacuated or absorbed, and the patient, often much debilitated, enters upon a period of convalescence. Now examine that same chest after all the physical signs diagnostic of fluid have disappeared, and one finds only too often the signs of grave mischief wrought by the slow inflammatory process, "impairment of resonance," due to the presence of false membranes, or to the collapsed condition of the lung, or to both combined; "deficient expansion" of the affected side, obscuring or "alteration of the pitch of the vesicular murmur," and perhaps "friction" persisting at some point. One might almost say that, except in a few cases, and those chiefly in children, a patient admitted with the physical signs of pleurisy with effusion never leaves the hospital without some abnormal physical signs still persisting, denoting a more or less serious impairment of the function of the contents of that half of the chest in which the effusion took place. If on the left side of the chest, the inflammatory process may have spread to the pericardium and caused pericarditis with effusion, and subsequently an adherent pericardium with all its results, as I well remember in the case of a boy of 13 years of age admitted into the City Road Chest Hospital. This is for-

[1] A Thesis for the degree of Doctor of Medicine of the University of Cambridge.

tunately a rare occurrence; and even if the inflammation does extend to the fibrous covering of the pericardium, as denoted by a pleuro-pericardial friction sound, it progresses no further. We may find on examination the total or partial collapse of the lung, with corresponding falling in of the chest wall of the same side, and perhaps the lung bound down by firm adhesions, and thus for ever restricting the expansion of that lung. In spite of all this, the patient is discharged as cured, and is thus registered in the hospital statistics at the end of the year. It was first impressed upon me during my term of office as house-physician under Sir Lauder Brunton at St. Bartholomew's Hospital that these patients would well repay one for further observation and watching through several years, and that one could arrive at more correct statistics with regard to the prognosis of the so-called "idiopathic pleurisy" or *pleuritis a frigore;* in fact, it would be well to follow all cases of pleurisy with effusion whether the abnormal physical signs have disappeared or not. I have often heard Dr. Gee state, when going round the ward, that you never know what lies at the bottom of a pleural effusion; and I have often heard it stated that it is "much less grave to have an attack of pneumonia than to have a pleurisy with effusion," and this in spite of the fact that the annual death mortality from pneumonia is stated at 1089 per million, and that of pleurisy as only 42 per million. This dictum which I have just quoted shows the necessity of considering the prognosis of pleurisy with effusion from an immediate and remote point of view, *i.e.* one must consider the mortality not only due to the pleuritic effusion itself, but also due to the complications which follow it, or to the *causa causans* of the pleurisy, and with which it must be intimately connected. The point was also all the more impressed upon me by the report of a lecture by Dr. Kingston Fowler in the *Clinical Journal* of February 3, 1897, tending to show that all, or nearly all, these cases terminated sooner or later in the development of tubercle in some part of the body.

One knows that in all cases of phthisis in some part of their course pleurisy always arises as a complication, and probably acts as a conservative element, in that it must check the formation of pneumothorax by, as it were, gluing the two surfaces of the pleura together, and thus closing, or partially closing, the potential cavity of the pleura. It is still a debated point as to whether even these cases are really due to the action of the tubercle primarily, or simply to the spreading of inflammation from the periphery of the lung in

contact with it; these pleuritic adhesions have over and over again been stained for tubercle, and in only a very few cases has a positive result been obtained.

In all post-mortems in cases of phthisis, the lung is bound down to the chest wall by firm adhesions. It is not, however, to these cases that I refer, but rather to those cases in which pleurisy precedes all other manifestations of tubercular disease. The above cases are almost always without effusion, although, of course, this may occur either hæmorrhagic, purulent, or serous in form.

With this end in view, I have collected all the cases of pleurisy with effusion which have been admitted into St. Bartholomew's Hospital or the City Road Chest Hospital from the year 1890 to 1896 inclusive, and have visited them at their homes where possible, or have been in communication with them where they lived at too great a distance. All these cases, when in hospital, showed no signs of phthisis, and, except for the pleurisy with effusion, were in fair health. In case of death, where possible, I have sought out the post-mortem record, where one has occurred, and in other cases have seen the registrar's register of the causes of death. Further than this one cannot go. Unless one has undertaken the search for patients in London who have been lost sight of for several years, and although the address on admission to hospital was to hand and correct, one cannot realise the difficulty of tracing these poor people, who change and change from lodgings to lodgings, from shelter to shelter, altering their names for reasons best known to themselves, and, in the case of females, they marry and thus adopt their husband's name.

After this part of the subject is finished, I propose to inoculate a series of guinea-pigs with the serum from the pleural cavity of patients suffering from pleurisy with effusion, and in which no sign of phthisis exists, and to demonstrate the presence or absence of tubercular or other infection after a lapse of six weeks. Should these lead to any positive results, I intend to trace these patients through a series of years and watch what becomes of them, whether those who give a positive result die of tuberculosis and *vice versa*.

I also intend to draw up a table of the relative frequency per month during the years 1890–96 inclusive of pneumonia and pleurisy with effusion, to see if any trace can be found of a relation between the two diseases; they will be obtained from the records in the case books of St. Bartholomew's Hospital.

History of Pleuritis.

Although in the pre-Hippocratic period mention is made of pleuritis, meaning thereby a pain in the side or "stitch," accompanied by fever, yet this disease was so often confused with pneumonia and other lung affections, that one is in doubt whether Hippocrates or Galen, coming later, in A.D. 130–200, recognised a primary affection of the pleura. Pleuritis was first described and definitely placed under the heading of inflammation of serous membranes by Pinel in 1745, and has since that time been looked upon as a separate disease. In a book of the Aphorisms of Stollius, published in 1790, is the first mention of the relation of phthisis to pleurisy: "Pleuritis latens est sæpe chronica, non raro hæreditaria, tumque in phthisim terminanda." This addition to our knowledge was soon followed by Lænnec in 1818, "De l'Auscultation Mediate," with the means of diagnosing pleurisy by physical signs; and Pinel and Lænnec practically laid the foundation for the gross pathology of the subject.

Lænnec puts as the commonest causes of pleurisy:—

1. Inclemency of winter and long-continued exposure to cold after violent exercise.
2. Metastasis of gout and rheumatism.
3. Mechanical injury.

As predisposing causes:—Slender frame, narrowness of chest, and above all tubercle of the lungs. And in conclusion he says:—"I would remark of pleurisy as of pneumonia, that the occasion and predisposing causes are frequently hidden from us, or at least seem insufficient to account for the attack." He also puts immoderate use of spirits as a predisposing cause.

Pleuritis being thus recognised as a definite disease, naturally the next step was an investigation as to its causation.

Before proceeding with the history of the disease, it will be as well to give some classification of the subject of pleuritis, so as to point out clearly the type to which I refer.

The following is the classification given by Fagge:—

1. "Idiopathic," rheumatic, or pleuritis *a frigore*.
2. Secondary pleurisy, *cf.* (*a*) from injury, (*b*) during the course of some specific disease, *cf.* enteric fever, acute rheumatism, syphilis, pneumonia, phthisis, the exanthemata, &c.

It is solely with regard to cases coming under the first category that I refer in this treatise, and with the discovery of the relation of micro-organisms to disease the word "idiopathic" has long ceased to be used. By the term "rheumatic," I don't mean that class of pleurisy which comes on in the course of

acute rheumatism, and which is without doubt a pleuritis set up by the same organism which causes the effusion into the joint and the vegetations on the valves of the heart, but rather to a form of pleurisy which is supposed to be predisposed to by a chronic condition of rheumatism closely allied to rheumatoid arthritis and gout, in the same way that this inherited dyscrasia predisposes to the inflammation of mucous membranes, &c., *cf.* tonsillitis and Dupuytren's contraction of the palmar fascia, which is probably nothing more nor less than a chronic inflammation of the palmar fascia in a gouty subject.

The following case will describe the type to which I refer :—

E. R. B., 35, mineral-water traveller, admitted to Rahere Ward on April 11, 1896, complaining of cough and pain down the right side.

History of present condition.—About five weeks ago was quite well ; got wet through several times ; pain gradually developed in right front and axilla, of a cutting nature ; pain worse on cough and deep breath ; diarrhœa ; no history of pneumonia ; pain still continues, but is not so bad ; still cough.

Family history.—Father died of "paralysis ;" mother died of old age ; one brother died at 41—"clot of blood under the heart." No history of phthisis, gout, or rheumatism.

P.I.—Always healthy ; never subject to cough.

P.C.—Patient looks a strong healthy man ; good colour ; tongue clean. P. 108, reg. ; fair volume and tension. T. 101.5.° B.O. Never hæmoptysis. Slight loss of flesh and sweats at night. Urine natural.

Chest.—Except showing the signs of a small right pleural effusion, natural ; the fluid gradually became absorbed, and the temp. fell to normal and remained so. All cough and sweating ceased. Discharged after five weeks with signs of thickened pleura and collapse of base of right lung.

The investigation of the cause of this primary pleuritis has chiefly been carried on by the French.

Pinel in 1818 makes the statement that in chronic pleurisy the lung often becomes tuberculous and like that in phthisis of the first degree.

Broussais a few years previously stated that pleurisy with effusion was the cause of the development of tubercle in the lung, and that the condition of the lung was nothing more than a process of inflammation set up by the irritation of the presence of fluid in its immediate neighbourhood.

Bouillaud in 1837 upheld this doctrine, saying that it is not as a sequence of pure and simple pleurisies that tubercle of the

lung develops, but rather to pleurisies followed or preceded by a catarrh which passes on to a chronic state.

Louis, on the other hand, upheld the doctrine of Lænnec, and even goes farther, and states that all pleurisies are tubercular, and in a great number of cases hasten the fatal termination of phthisis.

Legroux in 1850 not only denied the relation between tubercle and pleuritic effusion, but went so far as to say that the production of the fluid was a process by means of which nature closed up tubercular cavities, and by condensing the tissue of the lung rendered it less liable to the formation of tubercles.

In 1854 *Engster* (Erlangen):[1]—"Idiopathic primary pleurisy is considered as either due to a chill or to some epidemic influence, and appears to admit also of the presence of some specific organised poison, and not merely due to cold." He states that out of 163 cases only 71 offered any explanation of the onset.

In 1868 *Trousseau*:[2]—"The presence of pleurisy was often followed by tubercular disease through keeping up a congested state of the lungs."

In 1866 *Peters*:—"By lowering the resisting power of the organism."

Thus there were up to 1868 two doctrines as to the nature of the relation between tubercle and pleurisy with effusion.

1. Broussais and his school regarding tubercles as only an inflammatory product, tubercular disease as only a local inflammation, chronic to start with or arising from an acute inflammatory state of the lung or pleura.

2. Lænnec, on the contrary, states that pleurisy, even when it precedes phthisis, is only a manifestation or an effect produced by the presence of tubercles and can hasten the progress of tubercular disease.

From this survey it becomes evident that these authors, from Stoll downwards, had observed pleurisy in cases which presented no other signs of tubercular disease, and which ultimately died of pulmonary tuberculosis.

Such also was the state of affairs when Koch in 1880 made known his great discovery of the specific organisms concerned in the causation of tubercular disease in general.

Naturally a fresh impetus was given to the subject, and then commenced the search for the tubercle bacillus in the exudate, both by means of staining reagents and by cultivation on artificial media, neither of which was attended by any positive value, and it was not until 1884 that Chauffard and Gombault,

[1] "Ætiologie und Therapie der primären Pleuritis."
[2] "Clin. Med.," 1868, iii. 244 *et seq.*

working in Pasteur's laboratory, succeeded in inoculating guinea-pigs with the effusion from cases of primary pleurisy, and demonstrating the presence of the tubercle bacillus in 50 per cent. of their cases. They experimented with twenty animals, and ten, after about six weeks, died of tubercular disease; they also inoculated guinea-pigs with the effusion from undoubted cases of tubercular disease without producing tuberculosis, thus showing the sparseness of the bacilli in the effusion. The animals were inoculated with the effusion, whether serous, hæmorrhagic, or purulent, and they do not state with which kind of effusion the animals which died of tubercular disease were injected.

In 1882 *Fiedler*[1] collected 92 patients with pleurisy complicated with effusion: 28 are dead of phthisis; 66 discharged with or strongly suspected of phthisis; 21 appeared well after two years; 82 per cent. suspected to be tubercular.

Ehrlich in 1882 was the first to describe micro-organisms in pleural effusions; he describes them as being ovoid in shape, and sometimes grouped in twos and fours; in the first case probably staphylococci and in the latter streptococci; they occurred in cases of pleurisy with effusion during the progress of puerperal septicæmia.

Fränkel also demonstrated the presence of the "diplococcus pneumoniæ" in pleural effusions, and said it was of good prognostic value.

Sée (*G.*)[2]:—Pleurisy, which as a rule was set down to cold, was nothing but a pleurisy due to the presence of tubercles whose nature had been misunderstood.

Landouzy, 1886[3]:—"I do not deny cold as a cause of pleurisy, but that it is so must be exceedingly rare."

One of the most important pieces of evidence of the relation of tubercle to pleural effusion is that furnished by *Kelsch* and *Vaillard*.[4] They studied 16 cases of pleurisy with effusion, in most cases serous, in vigorous men, who died suddenly or rapidly during the course of convalescence of the malady. In every case there were present tubercles on the pleura, often very difficult to find, and tucked away between the lobes of the lung, and in some cases only to be discovered microscopically. They naturally exclude all cases of secondary pleurisy. In the same paper there is an analysis of 113 cases of acute pleurisy, of which nearly 82 per cent. proved to be tubercular.

[1] "Ueber die Punction der Pleurahöhle und Herzbeutels."
[2] "Des Maladies Simples des Poumons," 1886, pp. 441 and 518.
[3] "Revue de Médicine," p. 614.
[4] "Archives de Physiologie Normale et Pathologique," 1886, ii. p. 162.

In 1888, *Gilbert* and *Lion*[1] were unable to grow the bacilli on artificial culture media, and Ehrlich and Fränzel have scarcely ever been able to demonstrate the presence of tubercle in the effusion by staining methods.

In 1891, *Netter* produced almost identical results with Chauffard and Gombault.

In the same year *Koch* discovered tuberculine, supposed to be an antitoxin for tubercle. This, however, proved, after an extensive trial, to be of no value therapeutically, but as a means of diagnosis to be of assistance, as only 8.5 per cent. of healthy people reacted to it. *Netter* showed that of 15 cases of pleurisy with serous effusion, 13 (87 per cent.) reacted. *Osler* in his Shattuck lecture in 1893 cites a case where the reliance on this point led to a mistaken diagnosis, the patient ultimately dying of carcinoma of the lung. He also says that a sterile effusion is regarded as a point in favour of its tubercular nature.

Barrs, of Leeds, in 1890 collected 62 cases of primary pleurisy, out of which, six years afterwards, 22 (35.5 per cent.) were dead of phthisis or other tubercular lesion. In his list of cases the average duration of life in the cases which died was $2\frac{1}{2}$ years.

Bowditch, of America, in 1889,[2] collected 90 cases of all forms of pleurisy except those with suspicions of lung mischief. Of these, 44 were dead, 23 died of phthisis, 1 still living but suffering from phthisis, 21 died within five years or less, 2 died within eighteen years.

Lancereaux[3]:—Pleurisies should be classed amongst the infectious maladies, and exposure to cold is only occasionally an exciting cause, while the action of the infecting agent escapes us.

In 1891, *Landouzy* says that 98 per cent. of pleurisies put down to cold are due to tubercle, and every individual who cannot furnish an infection—dyscrasia or traumatism—to account for his pleurisy is tubercular, whether he be vigorous, young, fat, or muscular.

Recently bacteriological examination has thrown more light upon pleural effusions, and the following varieties have been described:—

1. Pneumococcus pleurisy.
2. Streptococcus ,,
3. Saprogenic ,,
4. Tubercular ,,
5. Staphylococcus ,,

---

[1] "Annales de l'Institut Pasteur."
[2] "Med. News," p. 63.
[3] "Bulletin de l'Académie de Médicine," 1892, p. 758.

Other organisms, *cf.* Eberth's bacillus of enteric fever, are capable of exciting inflammation of the serous membrane.

In 1895, *Straus*[1] says:—"Tubercle is the most common cause of pleurisy under all its anatomical forms, whether it be serous, hæmorrhagic, or purulent."

After the foregoing history of facts I now propose to bring in evidence the statistics I have gained from the 130 cases of primary pleurisy with serous effusion which I have collected either from the records of St. Bartholomew's Hospital or of the Royal Hospital for Diseases of the Chest. I may now state, once and for all, that, except in one case, and that I selected for a particular reason, none of them were suspected of having tuberculosis of the lungs. These cases I have traced either to their death, or have visited and seen them when they lived in or within reasonable distance of London, or, when too far away, have been in communication with them.

FIRST TABLE.

Year.	Cases.	Deaths.	Deaths from Phthisis or Other Tubercular Lesion.	Alive with Phthisis.	(?) Phthisis.	Total Tuberculosis.	Deaths from Other Causes.	
							No.	Disease.
1890 . .	10	5	3	...	2	4	1	Carcinoma of jaw
							1	Tumour of liver
1891 . .	17	6	6	...	1	7	...	.....
1892 . .	12	3	3	2	1	6	...	.....
1893 . .	21	6	4	8	1	13	1	Child-birth
							1	Chronic interstitial nephritis
1894 . .	23	6	5	1	..	6	1	Malignant disease of lung
1895 . .	29	6	5	5	1	11	1	Morbus cordis
1896 . .	18	3	3	3	3	9	...	.....
Total .	130	35	29	19	9	56	6	.....

Of those attacked in 1890, by 1898 40 per cent. are dead from or have signs of tubercle

"	"	1891	"	41	"	"	"	"
"	"	1892	"	50	"	"	"	"
"	"	1893	"	60	"	"	"	"
"	"	1894	"	26	"	"	"	"
"	"	1895	"	38	"	"	"	"
"	"	1896	"	50	"	"	"	"

From the above it is clear that of patients who were attacked between 1890–96 inclusive, 43.6 per cent. had either died of

[1] "La Tubercle et son Bacille."

phthisis or other tubercular lesion, or presented signs of phthisis.

Table showing Percentages of Death from Tubercle.

Of those attacked in 1890, 30 per cent. were dead by 1898.
" " 1891, 35 " " "
" " 1892, 25 " " "
" " 1893, 19 " " "
" " 1894, 22 " " "
" " 1895, 17 " " "
" " 1896, 17 " " "

or, in other words, 24 per cent. per annum died of some tubercular lesion.

Taking the whole series of years into consideration the average duration of life of those that died was exactly *three years after attack.*

Sex.—Out of 130 cases, 97 were males and 33 females, *i.e.* 75 per cent. of the cases were males.

Age when Attacked.

1–5	3 cases	=	2.3 per cent.	before 5
5 10	12 "	=	9.2 "	between 5 and 10
10–20	20 "	=	17.0 "	" 10 " 20
20–30	40 "	=	31.0 "	" 20 " 30
30–40	33 "	=	26.0 "	" 30 " 40
40–50	15 "	=	12.0 "	" 40 " 50
50–60	7 "	=	4·5 "	" 50 " 60

74 per cent. occur between the ages of 10 and 40.

Occupation.—All my cases are of necessity drawn from the working and poorer classes, and consequently most of them follow occupations in which they are much exposed to the weather. One cannot classify their occupations, as they are too numerous; no special class of case is particularly picked out.

Side of Chest Affected.

In 61 cases the left side was affected
" 67 " right " "
" 2 " both sides were affected

Age of Attack of Patients who died of Tuberculosis.

1–10	0	or	None died before the age of 10
11–20	3	"	10 per cent. between 10 and 30
21–30	2	"	8 " " 20 " 30
31–40	15	"	54 " " 30 " 40
41–50	5	"	18 " " 40 " 50
51–60	3	"	10 " " 50 " 60

Only 18 per cent. of the cases died before the age of 30.
82 " " " after " 30.

Family History of Phthisis, Rheumatism, or Gout.

Phthisis = 34 cases, or 24 per cent. of total cases
Acute rheumatism = 14 „ 11 „ „
35 per cent. of the cases gave a family history of phthisis, gout, or rheumatism.

Family History of Phthisis in Cases Developing or Dead of Tubercle.

Phthisis . . . . 13 cases = 23 per cent.
Acute rheumatism or gout 4 „ = 7 „
30 per cent. gave a family history of phthisis, gout, or rheumatism.

In every case the fluid cleared up before patient was discharged from the hospital.

Paracentesis Thoracis.

67 cases (52 per cent.) out of the total 130 were tapped.
34 „ (61 „) which developed tubercle.

Of the 67 cases aspirated, 60 yielded serous fluid.
„ „ „ 7 „ blood-stained fluid.
Four of the cases which yielded blood-stained fluid became tubercular, and two of these were dead before 1898.

Cases which Died of Tubercle after Tapping.

64 per cent. were aspirated.
10 „ were explored alone.
One case of malignant disease of the lung yielded sero-fibrinous fluid.

Not in a single case did a serous effusion become purulent after paracentesis was performed.

Clear Bill of Health as regards Affections of the Chest since leaving Hospital.

Females . . 10 cases, or 30 per cent.
Males . . . 26 „ „ 26 „

The above 36 cases are the only ones out of a total of 130 which one could say were well and strong; even those cases which were not subject to tubercle, except the above, have suffered from chronic cough, hæmoptysis, loss of flesh and pain in the chest, and other symptoms suggestive of pulmonary disease.

The point which interested me most was the type of the patient who had been subject to pleurisy with effusion; they were mostly very anæmic, spare, delicate-looking people, with flat chests and prominent clavicles. It was very rare to find a strong healthy man of good colour. One might say that when

once a patient has been the subject of pleurisy with effusion, he will in all probability become the subject of chronic chest symptoms, whether it be catarrh, shortness of breath, hæmoptysis, or tubercle; and again and again have the patients in whom I could find no tubercular mischief told me that "they don't seem the same since they had the pleurisy; could not do hard work on account of weakness and shortness of breath."

In very many of the cases one got a definite history of chronic excess in alcohol. This probably acts either by lowering the resisting power of the organism or by inducing acts of indiscretion, *cf.* exposure to cold when under its influence. It is a very common thing in the post-mortem rooms to see in cases of cirrhosis hepatis the lung bound down by adhesions to the chest wall, and the end of cases of chronic alcoholism is often hurried on by tuberculosis pulmonum.

Another point which proved interesting was that if a patient who had been treated for pleurisy with effusion returned to the hospital, it was never for that malady, but for some tubercular lesion.

From this historical survey and from my own cases one can see that there must be some very close connection between tubercle and pleurisy with effusion, and that connection must come under one of the following headings.

1. The so-called idiopathic pleurisy is only an early manifestation of tubercle.

2. That it is the point of attack for tubercle in an individual already predisposed to it.

3. That in certain cases it creates this predisposition by lowering the resisting power of the organism, and establishes the *locus minoris resistentiæ* for the entrance of tubercle.

In all probability the whole three factors come into play, but there can be no doubt, after the facts I have brought out earlier in this paper, that tubercle is a common and perhaps the commonest cause of primary pleurisy.

These cases of primary pleurisy may be divided under two headings: (1) insidious, (2) acute, from their mode of onset; and yet there can be no arbitrary division between the two classes of cases, as they insensibly merge one into the other, and one finds great difficulty in thus classifying these border cases.

Attempts have been made to prove that the acute form ushered in by headache, malaise, shiverings, and rise of temperature, are less likely to prove tubercular than those with a more insidious onset. This point is not, however, borne out by my statistics; acute and chronic alike have an equal tendency to

prove tubercular. The progress of the more chronic, insidious form, nevertheless, more resembles the progress of tubercular inflammation from—

(*a.*) Its insidious onset. (*b.*) Absence of marked inflammatory process. (*c.*) Its tendency to increase indefinitely. In all these respects it bears a close analogy to tubercular peritonitis.

The cases of acute pleurisy usually admit of a double factor: (1.) Predisposition of the subject to external causes; (2.) the presence of tubercle, and the two combined, *cf.* tubercle and exposure to severe atmospheric conditions, may give rise to an attack of acute pleurisy.

There can be no such disease as pleurisy due to cold alone (*a frigore*); cold cannot of itself set up inflammation; the only part it can play in the causation of disease is that it can lower the resisting power of the organism.

Many authors have urged the plea that, as so many cases of pleurisy with effusion ultimately recover and show no further manifestation of tubercle, these cases cannot have been tubercular to start with. Tubercle is capable of arrest in its progress and cure. How often has one seen in the post-mortem rooms in the apices of the lungs or elsewhere, old tubercular foci which have become converted into fibrous tissue, calcified or otherwise rendered innocuous.

Then look at cases of tubercular peritonitis, white swelling of the knee and other joints. Under proper treatment the disease is arrested and the tubercle bacillus is not necessarily disseminated throughout the body.

It must also be borne in mind that the progress of tubercle of serous membranes is far more favourable than when the disease attacks the other organs, and thus the inference that in many cases of primary tubercular pleurisy the disease is arrested at the time and never reappears is therefore justified. Earlier in this treatise I stated that the cases were those in which no suspicion of phthisis existed except in one instance, and that was admitted, as when in hospital there were signs of phthisis at one apex as well as the effusion; tubercle bacilli were found in the sputum; and yet, in spite of this, five years afterwards the patient is in good health with no signs or symptoms of pulmonary or other tubercular lesion. Both the patient's father and mother died of phthisis. It must also be borne in mind, as Dr. Klein has shown in his report on bovine tuberculosis, that all tubercle does not show the same power of infectivity. The tubercle bacilli which attack cattle are much more powerful than those which, as a rule, attack man; and in

the same way as there is gradation in the infectivity of bovine tubercle bacilli, so also there may be in the case of those which attack the human race. One must also consider not only the bacilli but the personal equation.

Other authors have urged against the tubercular theory of pleurisy with effusion that the bacilli are so rarely found in the effusion. For my own part, I do not see why one should expect to find them. The bacilli attack the serous lining of the pleura and set up inflammation, the result of which is a cell proliferation on the surface of the pleura, and the bacilli are, as it were, imprisoned amidst this neoplasm, whether it be in the giant-cells or in the fibrous tissue around. Surely the exudation is only the effect of the slow inflammatory process, and in it one would expect only to find the toxins and antitoxins generated or called into play by the action of the bacilli, and which tend to their ultimate destruction. It also seems to me, seeing that this is so, that the value of one positive result is of so much more value that that of many negative results; and when one considers that all the inoculations were performed upon guinea-pigs, animals which upon the slightest interference tend to die, so susceptible of any disease whatever that even the scratch of a pin will often kill them and show nothing post-mortem to account for death, can you wonder that such susceptible animals often die from the operation alone previous to the possible formation of any tubercles? I have also pointed out earlier that even when the case was known to be tubercular that inoculation gave negative results.

The following conclusions appear to be justified:—

1. That in a very large proportion of cases of acute pleurisy with serous exudation, no results are obtained from the bacteriological examination of the exudation.

2. Tubercle bacilli may not be found in cases which are undoubtedly of that nature.

3. The inoculation of fluid from a case of tubercular pleurisy into susceptible animals may not be followed by tuberculosis.

4. That nevertheless the bacillus of tubercle is the causative agent in a very large proportion of the cases, and that is true whether acute or insidious.

5. That the prolonged observation of 130 cases of primary pleurisy with effusion through seven years proves that over 40 per cent. became tubercular.

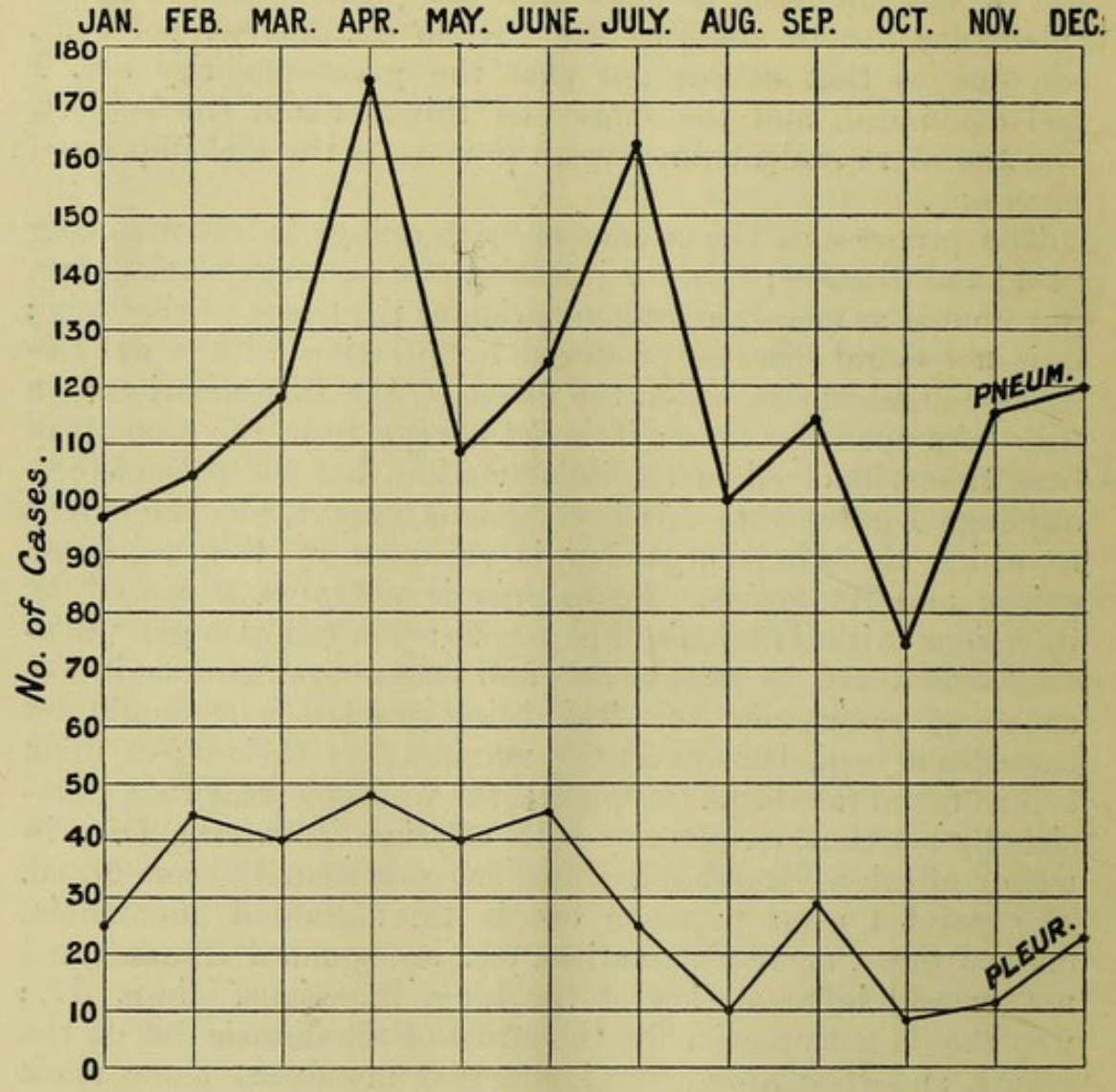
6. That by the use of bacteriological methods of diagnosis it is possible in some cases to determine the nature of a sero-fibrinous effusion.

7. That tubercle is capable of arrest and cure.

I would not wish to state that all primary pleurisies are tubercular, nor to state, as the French do, that 75 per cent. are due to that cause; but that the great majority are, I feel convinced, and the more one thinks about the subject, and the more patients one sees, the more is the fact impressed upon one.

The progress of the science of bacteriology is fast removing "regional diseases;" as, for instance, one no longer looks upon pneumonia as merely an inflammation of the tissue of the lung; it is a general disease, produced by infection with a definite specific organism in which the stress of the inflammation as a rule falls upon the lung; it is not always from the amount of lung tissue involved in the inflammation that the patient dies, although I grant that this is of serious import, but the extent to which the whole organism is poisoned by this organised poison and its toxins. Pneumonia is not always caused by infections with Fränkel's diplococcus; you can also get pneumonia produced by streptococci and other organisms, as in the course of septicæmia, &c. This being granted, why should not these same organisms exert the maximum of their effect upon serous membranes? *cf.* the pleura, for we know that they occasionally act thus, *cf.* pleurisy with effusion in enteric fever, in which effusion Eberth's bacillus has occasionally been found. The point I want to make out is this: granted pneumonia, enteric fever, acute rheumatism, &c., are general diseases, and not merely inflammation of the lung, intestines, joints, &c.; why should not occasionally the stress of the disease fall on the pleura, and by calling attention to that membrane alone mask the point of infection?

One knows how often, when a patient is admitted to hospital, the diagnosis between a small patch of pleuro-pneumonia and acute pleurisy is occasionally very difficult to make, and even impossible, the patch of consolidated lung being too small to give any physical signs, and unless one gets a crisis or rusty sputum the diagnosis becomes impossible. I believe that often those cases are secondary to a small patch of pneumonia; they may proceed to the stage of effusion, which may be serous or purulent, the latter being perhaps most common after pneumonia, especially in children. On the other hand, there is no reason why the organisms should not first attack the pleura. I have collected all the cases of pleuro-pneumonia and pleurisy with effusion which were admitted to hospital between the years 1890–96 inclusive, to see if there could be traced any relation between the monthly incidence of the two diseases.



The above table refers to 1430 cases of croupous pneumonia and 320 cases of pleurisy with effusion. The date of onset has been taken and not that of admission.

From the above table it is clear that the maximum number of cases of both diseases occur in the same month, viz., April, and also that the minimum number of cases occurs in the same month, viz., October. The average number of cases in both diseases diminishes after August, but whereas pneumonia presents two maxima—one in April and the other in July—that of pleurisy presents a fairly constant maximum from February to June.

Pneumococci have been found in the exudation of primary pleurisies with no history of pneumonia, and I don't think there can be any doubt that many cases of pleurisy with effusion are due to infection with the diplococcus pneumoniæ, very often secondary to pneumonia, but at times primary.

Another point which impressed itself upon me was the rapidity with which some effusions cleared up under salicylates after previous treatment with iodides and diuretics had failed, and these not coming on in the course of acute rheumatism.

Thue, of Christiania,[1] examined microscopically and attempted to obtain cultures in 30 cases of sero-fibrinous pleurisy. In 20 the results were negative; the 10 positive results were as follows:—

Bacillus tuberculosis	1
Streptococcus	1
Cocci of doubtful nature	2
Staphylococcus aureus and albus . .	1
,, albus alone . . .	3
,, ,, which was followed by tuberculosis on inoculation . . .	1
Micrococcus cereus	1
	10

In three cases pneumococci, but in all the effusion became purulent.

With regard to the prognosis of primary pleurisy with effusion, it must be considered under two headings:—(1.) Immediate; (2.) Remote.

1. The immediate prognosis is very good; the fluid will gradually become absorbed in most cases, or can be syphoned off.

Out of a total of 320 cases of pleurisy with effusion, 6.1 per cent. died before leaving hospital, and this was in the case of all pleural effusions. One might say that, except in very large pleural effusions, there is no immediate danger of death. Occasionally one hears of a patient with a large pleural effusion suddenly dying, and various theories as to its causation have been suggested, *cf.* kinking of the inferior vena cava from the pressure of the fluid or the twisting of the heart. *Osler* has, however, shown this to be false. There is absolutely no danger in tapping a chest, provided that ordinary antiseptic precautions are taken. I have always allowed the fluid to syphon off, and can conceive of no more useful or safer apparatus than that invented by Dr. Garratt. I have no doubt that half the cases of bloody effusions which used to be obtained were due to aspiration under too great a vacuum.

In 67 of my cases, clear fluid was obtained in 60 cases and blood-stained fluid in 7 cases; 4 of the latter became tubercular.

[1] "Bulletin des Hôpitaux," 1895, p. 439.

I have never seen any harm come from aspiration. There exists no relation between paracentesis thoracis and the incidence of tubercle. It is very rare for any patient to become collapsed during its performance, although I have drawn off seven pints from the chest of an old man of sixty at one time; a moderate amount of cough is no indication for it to be stopped. It is as well, when performing the operation on a highly sensitive patient, to give a whiff of gas until the needle is inserted.

On the other hand, one sees very many effusions which will not clear up immediately do so even after a small amount of fluid has been withdrawn; this is said to act by relieving the tension inside the pleura.

Collapsed lungs become "carnified" by the too long collapse caused by the pressure of the effusion, and incapable of future expansion. Fluid, once diagnosed, as a rule, ought not to be allowed to remain more than fourteen days unless it shows signs of absorption.

I have seen two very rare sequelæ after paracentesis thoracis, viz.:—(1st) Thrombosis of hepatic vein, and (2nd) of the axillary vein. In the first case the progress of the case was only delayed for a short time; in the second case the patient ultimately died of thrombosis of the superior vena cava. In the case of thrombosis of the hepatic vein, it occurred after paracentesis and washing out the chest with a solution of boracic acid, gr. iv. ad 3i.

Occasionally in primary pleurisies with effusion the fluid will not clear up in spite of repeated tappings, and Dr. West has in such cases resected a rib or two; the effusion has become converted into an empyema, and was treated as such and ultimately cured. In one case under Sir Lauder Brunton, the one referred to above who had thrombosis of the hepatic vein, the chest was washed out with boracic acid after the effusion was evacuated, and it did not recur. The immediate prognosis then is very good.

1. With regard to the remote prognosis, one sees that at least 40 per cent. of the cases develop phthisis or other tubercular lesion within six years.
2. The average duration of life of those who develop phthisis is three years.
3. The longer one lives after the attack the less is one likely to develop tubercular lesions.
4. A patient having had pleurisy with effusion is not, or ought not, to be considered as a good life for insurance until five years after the expiration of the attack.
5. Acute onset with rapid absorption of the fluid is more

favourable than a chronic insidious case, although both may develop tubercle.

6. Females are less likely to die of phthisis than males.
7. Family history of tubercle does not help much in the prognosis.
8. The prognosis is much better in cases where the patient can furnish a possible method of infection.
9. The side of the chest which has been attacked by the pleurisy usually presents the most advanced stage of phthisis.
10. Patients with pleural effusion addicted to excess of alcohol often die of phthisis.
11. Most of the patients attacked who ultimately died were between the age of thirty and fifty. None died before the age of ten.
12. Rapid loss of weight whilst under treatment is a bad sign.
13. Continued hectic temperature which does not fall to normal before discharge is bad, although even this is not always a bad prognostic sign.

I should like to urge that in any case where there exists no history of infection and the progress of the case is somewhat tardy, the pleural cavity should be drained and washed out with some mild antiseptic, *cf.* boracic acid, gr. iv. ad 3i., used at the temperature of the body; this to me seems the only rational treatment for such cases, and no possible harm can come of it if done with all due care.

In conclusion, I should like to urge that all cases of primary pleurisy when complicated with effusion should be treated as if they were due to infection with tubercle, and, if possible, after the effusion has cleared up, they should be sent away to some warm climate, *cf.* the South of France, the Cape, Bloemfontein, or the South of England.

I have not had time to complete the experiments I had begun upon the infectivity of pleural effusion upon guinea-pigs. I have only had one death up to the present, having inoculated seven animals. I am greatly indebted to Dr. Klein and Dr. Andrewes for their kindness in performing the inoculations for me, and for other assistance during the progress of this thesis.

1. Animal weighing 573 grammes. Intra-peritoneal injection of 3 c.c. clear serous fluid under all antiseptic precautions on February 26, 1898. Animal gradually lost weight in spite of good appetite. One week before death began to be dull and crouch about the corner of its cage. Died March

22, 1898; weight, 420 grammes, *i.e.* a loss of 153 grammes in four weeks.

P.M.—Great emaciation; no fat. No free peritoneal fluid, and except that the peritoneum was somewhat injected, nothing abnormal was found; all the viscera natural. Sections were cut of two somewhat large mesenteric glands and stained for tubercle, with no positive result. Cultures were made from the blood of the heart on serum-agar with no result. No cause of death found.

I propose later to go on with these experiments, and see if one can get anything tangible to work on.

In conclusion, I have to thank the Medical Staff of St. Bartholomew's Hospital for their kindness in allowing me the use of their cases, and especially Dr. Klein and Dr. Andrewes for their kindness in helping me in the bacteriological work. I have also to thank the Medical Superintendents of the Hackney, Marylebone, Homerton, Shoreditch, and East Dulwich Infirmaries for their kindness in looking up the post-mortem notes of patients admitted into the various infirmaries.

"Watch carefully every case of pleurisy with effusion, for you are never certain as to what may lie at the bottom of it."—*Dr. Gee.*

REFERENCES.

PINEL, 1745, Ziemssen's Cyclopædia (trans.), 1876, vol. iv. p. 589.
STOLLIUS, 1790, Aphorismi.
LÆNNEC, 1818, De l'Auscultation Médiate.
BROUSSAIS, Histoire des Phlegmasies ou Inflammation Chronique, 1808.
BOUILLAUD, 1837, Journal Hebdomadaire des Sciences Médicales.
LOUIS, 1825, Recherches Anatomico-Pathologiques sur la Phthisie.
ENGSTER, 1854, Ætiologie und Therapie der Primären Pleuritis.
TROUSSEAU, 1868, Clin. Med., 1868, iii. p. 244.
PETER, 1866, De la Tuberculisation en Général.
WILLIAMS, 1871, Pulmonary Consumption.
CHAUFFARD et GOMBAULT, 1884, Des Hôpitaux de Paris, p. 309.
FIEDLER, 1882, Uber die Punction der Pleurahöhle und Herzbeutels.
EHRLICH, P., 1882, Beiträge zur Ætiologie und Histologie Pleuritischer Exsudate.
SEÉ, G., 1886, p. 441 et 518, Des Malades Simples du Poumon.
KELSCH et VAILLARD, 1886, Arch. de Physiologie Norm. et Patholog., t. ii. p. 162.
GILBERT et LION, 1888, Annales de l'Institut Pasteur.
NETTER, 1891, Bulletin des Hôpitaux, p. 176.
BARRS of Leeds, 1890, Brit. Med. Jour., p. 1059.
BOWDITCH, 1889, Med. News, p. 63.
LANCEREAUX, Bulletin de l'Academie de Méd., 1892, p. 758.
LANDOUZY, 1891, Revue de Méd., p. 611.
JOANNY, 1891, Prognostique Eloigné de la Pleuresie.
STRAUS, 1895, La Tubercle et son Bacille.
FOWLER, 1897, Clin. Jour., p. 226 *et seq.*
THUE, Bulletin des Hôpitaux, 1895, p. 439.
OSLER, Shattuck Lecture, 1893.

TABLE OF CASES.

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1890. 1.	H. O.	33	M.	Ship's Steward.	R.	None given.	Malaria in Burmah 15 months ago.	Malaria in Burmah 15 months ago; attack every three weeks. Three weeks ago sharp pain in R. side; chilly; sweated profusely.	Nov. 1890.	Healthy-looking man; signs of fairly large R. pleural effusion up to third rib in front; cleared up before discharge. Temp., except for four days when it reached E. 100°, normal. Urine 1020, o/a o/s; contains much urea. Paracentesis 0iii. ℥vii.; somewhat turbid serum; coagulum found; sp. gr. 1022.	Alive and well at present time. Parents refuse address.	None.	Alive.
2.	G. B.	33	F.	Housework.	L.	No history of phthisis or rheumatism.	Twelve months slight cough.	Pain in L. side four or five months.	Oct. 1890.	Fairly healthy-looking; signs of fairly large L. pleural effusion; crackling râles at R. apex; fingers not clubbed; râles cleared up before discharge. Temp. up to 100 and 99° at night until just before discharge, when normal. Urine natural. Paracentesis, ℥iii. blood-stained fluid.	Died June 1891 at Highgate Infirmary of phthisis.	Phth.	Died June 1891.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1890. 3.	A. C.	57	M.	Waiter.	L.	Father died insane. M.? No history of hæmoptysis or phthisis.	Moderate drinker. No history of gout, rheumatism, or cough.	Insidious onset with cough and shortness of breath.	April 1890.	Somewhat wasted, with signs of fairly large pleural effusion up to third rib; double friction L. base. Left hospital with chest clear on R. side; weak breathing and impaired note at L. base. Temp. at first up to 102° at night, 99° on discharge. Urine 1025, o/a o/s. Paracentesis, 3 pints clear serous fluid, rapidly coagulated.	Died at St. Bartholomew's Hospital of phthisis.	Phth.	Died.
4.	R. W. M.	43	M.	Brewer's servant.	L.	Father died of inflammation of lungs; one brother died of bronchitis.	...	Six months ago whilst at work rigors and pain in L. side; in Temperance Hospital tapped 3 times; (1) 0iii. clear serous fluid; (2) 0ii. g. clear serous fluid; (3) ℥ix. clear serous fluid. Since then not able to do much work, but kept at it till last week. Lately breathing short and pain L. side.	Aug. 1890.	Fairly nourished; looks rather ill; signs of small L. pleural effusion up to L. angle of scapula; no adventitious sounds; cleared up. Temp. always normal. Urine 1012, o/a o/s. No paracentesis.	Slight cough off and on since; never hæmoptysis. Says he is neither gaining nor losing flesh. Occasional night-sweats. Is very short of breath. Chest poorly covered, with much flattening of L. side, and definite expansion of this side at apex and base. Vocal vibrations present, but diminished at base. Note against L. side. Definite vesical murmur and voice sounds. Delicate looking. Nothing very definite. Influenza three weeks ago. Occasional creaks at L. apex, with harsh wavy breathing.	Phth. ?	Alive.

5.	T. M.	55	M.	Labourer.	L.	One brother died of phthisis; one daughter of phthisis, aged 14.	Always healthy before; no winter cough; rheumatism, but never laid up; always temperate.	Jan. 1890, influenza. Cough since, with loss of flesh for past three weeks. Pain in side.	Jan. 1890.	Rather wasted; finger-ends bulbous; signs of small L. pleural effusion; some scattered fine râles to be heard all over L. lung. Temp. up to 101° E. during first half of stay, afterwards normal; gained 2 lbs. No paracentesis.	Died of tumour on liver and exhaustion, Dec. 1893.	None.	Died Dec. 1893.
6.	R. D.	13	F.	School.	R.	No history of phthisis or rheumatism.	Diphtheria in Homerton, followed by scarlet fever years ago.	Acute onset with pain and cough, then shortness of breath and shivering.	Dec. 1890.	Healthy-looking child; signs of largish pleural effusion up to clavicle. On leaving, dulness with weak breathing sounds; no moist sounds below L. angle of R. scapula; no signs of fluid. Temp. on admission 101°, very unsteady afterwards, and going up to 99° at night on discharge. Gained 4 lbs. Urine natural. Paracentesis, ℥xxviii. clear serous fluid, sp. gr. 1018.	Quite well since leaving hospital. Now suffering from chlorosis. No cough, and chest natural.	None.	Alive.
7.	T. H.	24	M.	Boot salesman.	R.	No history of phthisis or acute rheumatism.	Always healthy; no potus.	One month ago pneumonia; 10 days ago cough and pain in R. side; cold shivers.	Dec. 1890.	Signs of small R. pleural effusion to L. angle of scapula, which gradually absorbed, leaving friction at base. Self-discharged in five days. Temp. up to 101° E. Urine 1014, natural. Paracentesis, ℥v. clear serous fluid.	Quite well since leaving St. Bartholomew's; able to do heavy work; occasionally slight cough. Fractured R. arm four months ago. Chest natural.	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1890. 8.	D. T.	30	M.	Letter-sorter.	R.	Good.	Never ill.	Pain R. side of chest three weeks; worse lately, with loss of breath; sweating at night.	Jan. 1890.	Healthy-looking man; signs of a fairly large pleural effusion up to spine of scapula behind and second rib in front. Discharged with some impairment of note, marked friction in axilla and crackling over front of chest. Temp. 99.6° on admission, afterwards subnormal. Urine 1030, o/a o/s. No paracentesis.	Quite well except for slight pain and cough on and off since leaving hospital. One slight attack of hæmoptysis with sputum. Nothing abnormal detected in chest.	None.	Alive.
9.	A. M.	47	M.	Blacksmith.	R.	No history of phthisis.	Much abroad; never ague or dysentery; generally good health; no rheumatism or gout; potus.	Acute onset, with pain, fever, and vomiting.	Aug. 1890.	Strong-looking man; signs of small pleural effusion at R. base with friction, which cleared up. Temp. always normal. Urine, no albumen. No paracentesis.	Died in Whitechapel Infirmary of carcinoma of jaw.	None.	Died.
10.	J. B.	35	F.	Charwoman.	L.	One sister died of phthisis; no history of rheumatism.	No previous illness; much exposed to sudden changes of extreme temperatures.	Acute onset, with cough, pain in R. side, and dyspnœa.	July 1890.	Pale, unhealthy woman; signs of large L. pleural effusion, almost filling chest; fluid cleared up, but left the side much flattened with collapse of lung. Temp. up to 99° E., normal M. Urine 1022, o/a o/s. Paracentesis, ℥xvi. blood-stained serous fluid.	Died two years ago; cause unknown. Cannot trace any further.	?	Died 1896.

1891. 1.	B. W.	53	F.	Fruit-hawker.	L.	One brother died of phthisis.	Gout three times; rheumatic fever 14 years ago; always strong.	Cough six months, influenza six weeks ago; pain in L. side four weeks; wasting six months; slight hæmoptysis three weeks ago; slight night sweats all along.	June 1891.	Ill-nourished; signs of fair-sized L. pleural effusion. Temp. occasionally up to 99° at night, and even on discharge gained 8 lbs. Urine? Paracentesis, ℥iv. clear serous fluid; ℥i. clear serous fluid.	Alive and well two years ago. Cannot trace further.	?	Alive two years ago.
2.	F.L.H.	32	F.	Housework.	L.	No history of phthisis, rheumatism, or gout.	Always healthy before this.	Chronic onset, with slight pain for eight weeks, gradually getting worse, preceded by short dry cough during pregnancy.	May 1891.	Signs of large pleural effusion filling L. side of chest. Left hospital with some slight dulness and dimin. vesicular murmur at L. base. Temp. on admission 103°; gradually fell to normal before discharge. Urine 1032, acid; trace albumen; much mucus. Paracentesis, ℥xxvii. clear serous fluid, somewhat viscid.	Died in Lucas Ward, St. Bartholomew's Hospital, September 22, 1897, of tubercular disease of hip. No post-mortem.	Tubercular disease of hip.	Died Sept. 1897.
3.	W. H.	4	M.	School.	R.	Father died of hæmoptysis 1891.	None given.	Acute onset after exposure, with pain in R. side and shivering.	Nov. 1891.	Signs of small R. pleural effusion. Left hospital with some dulness at R. base. No signs of fluid. Temp. still occasionally goes up to 99° E.; up to 102° E. on admission. Gained 1 lb. Urine natural. Puncture with needle; clear serous fluid.	Died Nov. 4, 1897, in Matthew Ward, St. Bartholomew's Hospital, of phthisis and profuse hæmoptysis.	Phth.	Died.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1891. 4.	T. W. B.	34	M.	Painter.	L.	No history of phthisis.	None given.	None given.	?	Not wasted much. Signs small L. pleural effusion; developed signs of pneumo-thorax; dry bronchitic signs over R. lung on discharge. Temp. always up to 102° at night; 99° morning. Exploratory puncture; clear serous fluid.	Died 1891, of phthisis, in City Road Chest Hospital.	Phth.	Died.
5.	T. M.	33	M.	Porter in meat-market.	R.	Uneventful.	Never pleurisy before; always good health previously; potus.	Quite well till 14 days ago, then cough followed by pain in R. side; never severe.	May 1891.	Strong-looking man, healthy complexion; signs of large pleural effusion filling R. side of chest, which entirely cleared up, leaving only impaired note at R. base with definite vesicular murmur. Temp. 100° on admission, fell to normal and remained so. Urine 1024, natural. Paracentesis, 0ii. clear serous fluid.	Died in St. Bartholomew's Hospital, 1895, of phthisis and pleural effusion.	Phth.	Died.
6.	H. P.	23	M.	Stone-polisher.	R.	No history of phthisis.	Always strong and healthy till three months ago; influenza, bronchitis, and inflammation of lungs, never well since.	Lately pain in R. side with loss of flesh.	May 1891.	Signs of large R. pleural effusion. Only in hospital one day, self-discharged. Temp. subnormal. Paracentesis, no fluid obtained.	Alive at Christmas 1897 and looking well. Cannot find patient.	?	Alive.

7.	M. C.	28	F.	Housework.	R.	Gout, mother. Father died cause? No history of phthisis.	Always healthy; never any cough before; smallpox when infant.	Sore throat three weeks with pain all over; shivering and pain in R. side eight days.	March 1891.	Well-nourished; signs of fairly large R. pleural effusion up to 4th rib and spine of scapula. On discharge, no signs of fluid; note somewhat impaired, and weak breathing in axilla and base. Temp. 101.5° on admission; soon fell to normal. Urine 1010, o/a o/s. No paracentesis.	Has been quite well since leaving hospital in 1891. No chest symptoms. Nothing abnormal detected in lungs.	None.	Alive.
8.	I. S.	10	F.	School.	L.	One sister died consumptive bowels. No definite history of phthisis.	Always delicate, and has lost flesh; measles, pertussis, and varicella.	Cough some weeks, then developed pain in L. side and shortness of breath.	July 1891.	Fair, delicate, large-eyed child; signs of large L. pleural effusion filling whole of L. chest. Discharged with all signs cleared up. Temp. on admission 101°, gradually fell to normal. Paracentesis, ℥xxvi. s/s. rather dark yellow serous fluid, 1025 neutral, much albumen.	Has been quite well since leaving hospital in 1891. Healthy girl. Nothing abnormal detected in lungs.	None.	Alive.
9.	W.C.	6	M.	School.	L.	None given.	Scarlet fever 1890. Always healthy.	Three days ago quite well, then pain in chest, cough, and shortness of breath.	Sept. 1891.	Well-nourished; signs of small L. pleural effusion, which cleared up after tapping. Temp. at first 102° E., gradually fell to normal. Urine 1015, o/a o/s. Paracentesis, ℥ix. clear serum with much fibrinous element.	Alive and well. Living at Daventry, Northampton. (Not examined.)	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1891. 10.	T. W. H.	21	M.	Draper's assistant.	R.	Mother has rheumatism; one sister suffered from acute rheumatism. No history of phthisis.	Scarlet fever at 8. No history of pleurisy or hæmoptysis.	Acute onset, with shivering, nausea, and headache; sharp pain R. side; loss of flesh.	March 1891.	Weakly-looking; signs of large pleural effusion filling up R. chest. Left hospital with flattening and defective expansion R. side, dull at base, with definite vesicular murmur and vocal vibrations somewhat diminished. Temp. on admission 103°, very irregular for three weeks, going up to 100° at night. Normal before discharge; gained 12 lbs. in six weeks. Urine 1020, natural. Paracentesis, 0iii. clear serous fluid.	No cough; paracentesis once; very healthy-looking man; chest natural.	None.	Alive.
11.	H. R.	48	M.	Storekeeper.	R.	Father frozen to death; gout. Mother died "broken blood-vessel." No other history of phthisis.	No illness since quite young; winter cough; never rheumatism or gout.	Quite well till three weeks ago. Wet through. Pain in R. side and shivering.	July 1891.	Strong well-nourished man; signs of large pleural effusion up to R. clavicle and to spine of scapula. Discharged with friction at R. base and signs of small effusion. Temp. always raised at night, about 100°, occasionally up to 101°. Lost 12 lbs. in hospital. Urine 1016, o/a o/s. Chest explored; clear serous fluid.	Died November 1891; tuberculosis pulmonia (certificate).	Phthisis.	Died Nov. 1891.

12.	C. G.	17	M.	Cabinet-maker.	R.	None given.	Never very strong; nothing serious till present time.	Two weeks ago pains and aches all over, headache and sweating, followed by stabbing pain in R. side; cough and fever in about a week.	June 1891.	Signs of small R. pleural effusion. Temp. 100° on admission, afterwards normal. Urine 1020, o/a o/s. No paracentesis.	Alive and well; occasional pain in R. side on cough. Nothing abnormal detected in chest.	None.	Alive.
13.	E. M.	7	M.	School.	R.	One aunt died phthisis; most of family "suffer from chest." One brother died peritonitis; one brother spinal caries.	Pertussis, varicella, measles; never scarlet fever.	Three weeks ago cough and pain. Temp. 102.5°; three months previously a similar attack.	Sept. 1891.	Signs of small R. pleural effusion to scapula; signs cleared up before discharge. Temp. on admission, 102°, rapidly fell to normal. Well-nourished boy. Urine 1014, o/a o/s. No paracentesis.	Quite well since leaving hospital, except for slight cough. One brother just died peritonitis, tubercle(?); one brother spinal caries. Chest nil.	None.	Alive.
14.	F. P.	24	M.	Waterside labourer.	R.	Father alive, mother died of fit; no history of phthisis.	Cold and cough for past two months, otherwise quite well.	Insidious onset; cough and shortness of breath for two months.	Dec. 1891.	Well-nourished; signs of a large R. pleural effusion reaching up to clavicle. Temp. normal except on one or two occasions at night, when 99.2°. Urine 1020, acid, o/a o/s. Paracentesis, 0iv. s/s. clear serous fluid, which afterwards clotted.	Died in Guy's Hospital, February 28, 1897, of pulmonary tuberculosis.	Phthisis.	Died.
15.	H. W.	27	F.	Office-cleaner.	L.	Father died lockjaw; mother has rheumatism; no history of phthisis.	No illness except measles as a child.	Sudden onset with pain in L. side.	May 1891.	Well-nourished; signs of large pleural effusion, almost filling L. chest. Temp. 103° on admission; kept up to 99.5° at night for three weeks, then fell to normal. Urine 1030; trace albumen; urates. No paracentesis.	Alive and well since leaving St. Bartholomew's. Lungs natural.	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1891. 16.	W. W.	22	M.	Agricultural labourer.	R.	None given.	Has suffered with erysipelas, otherwise healthy.	Onset with shortness of breath; no history of hydatid; no pain.	July 1891.	Healthy-looking; signs of fair-sized R. pleural effusion to 4th rib and middle scapula. Discharged with dulness at R. base, and definite vesicular murmur, with a few râles above. No fluid. Temp. on admission 100° E., 98° M.; 103° after second paracentesis, afterwards normal. Urine 1008, trace albumen, o/s. Paracentesis 0ii. s/s. clear serous fluid; 0ii. s/s. clear serous fluid.	Alive and well; not ailed since leaving hospital. Living at Bishops Stortford. (Not examined.)	None.	Alive.
17.	S.A.P.	18	F.	Laundress.	L.	None given of phthisis.	Good health.	Quite well till three weeks ago. Bronchial catarrh followed by pain in L. side.	April 1891. July 1892.	Flushed; signs of small L. pleural effusion, which cleared up completely. Temp. normal. Urine 1022, acid, o/a o/s. No paracentesis.	Alive and well; very well since leaving hospital, except in 1897, abscess in R. breast. Healthy-looking woman. No symptoms pointing to phthisis. Chest poorly covered, otherwise normal. Married since leaving hospital.	None.	Alive.

1892. 1.	A.P.S.	36	M.	Book-binder.	L.	No history of phthisis.	Never ill before.	Ailing since Christmas 1891, six months ago. Influenza two months ago, and pain recurred with shortness of breath.	July 1892.	Fairly well nourished; signs of largish L. pleural effusion with cardio-pulmonary friction; dulness up to 3rd L. rib and whole of L. back. On discharge still friction and definite vesicular murmur on L. side. Temp. at first 101° E., gradually fell to normal. Lost 2½ lbs. Urine 1022, o/a o/s. No paracentesis.	No cough after leaving hospital; slight hæmoptysis mixed with sputum on one or two occasions. Expansion good all over; no signs of fluid in L. axilla, and at L. base vesicular murmur diminished and note slightly impaired. Some falling in of L. side of chest at base (?).	Phth.	Alive.
2.	S. H.	29	F.	Housework.	L.	One brother died of hæmoptysis; one sister died of "rapid consumption;" father died of abscess on liver.	Rheumatic fever in 1892; never quite well since.	Onset with pain and cough for three months, then exacerbation with vomiting, headache, and increased pain.	Dec. 1892.	Pale thin woman; signs of large L. pleural effusion filling L. chest; 1st sound at apex not pure and sharp; no definite vesicular murmur; absorbed whilst in hospital; no fluid on discharge; collapse and thickened pleura; no moist sounds at either apex. Temp. 100.5° for five days, after which normal. Gained 4 lbs. in hospital. Urine natural. Paracentesis, 0iv. s/s. clear coagulable serum.	Advanced phthisis; moriturus; signs of cavity at R. apex; very much wasted.	Phth.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1892. 3.	S. C.	41	M.	Traveller.	L.	No history of phthisis, rheumatism, or gout.	Pleuritic effusion; potus.	.....	Feb. 1892.	L. pleurisy with effusion. Died. Post-mortem, tubercle of tracheal, bronchial, and mesenteric glands. Paracentesis, 0ii., fluid character (?).	Died in Mark Ward, Feb. 1, 1898. Tubercular peritonitis. No tubercle discovered in lungs or pleura. R. pleural effusion after influenza.	Tubercular peritonitis.	Died Feb. 1898.
4.	E. K.	9	F.	Child.	L.	Nothing in notes.	Nothing in notes.	Measles nine months ago, not well since; fourteen days ago feverish, pain in L. side, vomiting.	Jan. 1892.	Delicate, pale-faced child, with signs of small L. pleural effusion and bronchitis. On discharge, note slightly impaired at L. base with definite breathing sounds. No moist sounds. Temp. normal. Urine 1010, acid, o/a o/s. No paracentesis.	Quite well since leaving hospital except for occasional pain in L. side of chest; no cough. Healthy-looking girl. Nothing abnormal detected in chest.	None.	Alive.
5.	S. M.	21	F.	House-work.	R.	No history of phthisis, rheumatism, or gout.	Always well.	Fairly acute onset with headache and pain in R. side.	June 1892.	Fairly healthy-looking girl; cracked-pot sounds at R. apex (?). Signs of fairly large pleural effusion R. side; pecty. with bronchial breathing at R. apex and in R. axilla. Signs much improved before discharge, and no signs of fluid. Physical signs at R. apex cleared up before leaving. Temp. always subnormal. Urine 1010, acid, o/a. o/s. No paracentesis.	Quite well since leaving hospital; no cough and no pain; chest natural. Strong healthy-looking woman.	None.	Alive.

6.	A.O.C.	36	M.	Carpenter.	R.	Good.	Always strong and healthy.	Well till three weeks ago, then caught cold after exposure; cough and pain in side developed.	Feb. 1892.	Well nourished and healthy looking. Signs of small pleural effusion. On discharge, still much impaired note at R. base behind; loud friction behind and in R. axilla; no pain. Temp. M. 98.8°, E. 100°. Up to 99° E. on discharge. Weight unaltered. Urine natural. No paracentesis.	Alive and well, living in Kent. No symptoms of phthisis. (Not examined.)	None.	Alive.
7.	R. B.	31	M.	Greengrocer.	L.	Good.	Never laid up before.	Insidious onset with shortness of breath and cough.	Oct. 1892.	Thin face, lips blue; signs of large L. pleural effusion; apex to base dull. Discharged with signs of collapse of L. lung; no fluid. Temp. at first up to E. 101°, M. 99°. Normal for one month before discharge. Gained 13 lbs. whilst in hospital. Urine 1012, natural. Paracentesis, 0iii. s/s. clear serous fluid; 0ivgs. clear serous fluid; 0ii. clear serous fluid.	Died Feb. 1895; tubercular disease of lungs.	Phthisis.	Died Feb. 1895.
8.	M. W.	34	F.	Cook.	L.	Father alive and well; mother died of apoplexy; one sister has gout; paternal uncle and aunt died of phthisis.	Never any serious illness; ague when a child.	Insidious onset for several months with malaise and fleeting pain in R. side of chest. Nil definite.	Nov. 1892.	Fairly well nourished woman; signs of small L. pleural effusion, with friction over the scapula. Discharged with slight pain at L. base. Fluid cleared up. Temp. normal after 1st week, when it reached 100° at night. Urine 1020, trace of albumen, o/s. No paracentesis.	Living at Guildford. Well, except suffering from general debility.	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1892. 9.	H. R.	20	M.	Plasterer.	R.	Very good.	Inflammation of lungs only illness; sober.	Insidious onset with slight pain in back and chest; cough.	Feb. 1892.	Healthy-looking lad; signs of fair-sized pleural effusion up to 4th rib in front. Discharged with friction at R. back below scapula. Temp. E. 100°, M. 98.6°; normal before discharge. Gained 10 lbs. Urine natural. No paracentesis.	Died in Champion Hill Infirmary, East Dulwich, of phthisis, Aug. 30, 1897.	Phth.	Died. Aug. 1897.
10.	A. B.	16	M.	Cabinet-maker.	L.	Father had pleurisy and rheumatism; no history of phthisis.	Always good health; pleurisy L. side some weeks ago.	Very acute onset; headache, cough, and pain.	March 1895.	Small L. pleural effusion; not much sweating. Temp. always normal. Urine natural. No paracentesis.	Ailing off and on since leaving St. Bartholomew's; cough and night sweats; losing flesh. Christmas 1897, rheumatic pleurisy; signs of consolidation and breaking down at R. apex; phthisis.	Phth.	Alive.
11.	H. H.	22	M.	?	L.	Very good.	Always healthy except slight cough past two years.	Acute onset with pain and cough.	Oct. 1892.	Absolute dulness L. back and front; signs of large pleural effusion filling L. chest; fluid cleared up after tapping twice; chest expands well, and vesicular murmur all over. Temp. on admission 100°, afterwards normal. Urine natural. In hospital five weeks. Paracentesis, ℥lxviii. clear serous fluid; ℥xciv. clear serous fluid.	Well; living at Norwich.	None.	Alive.

12.	A. T. H.	35	M.	Warehouseman.	R.	Nothing important.	Always good health.	Eight weeks ago influenza; four weeks ago pain in R. side on sudden movement; not disturbed by cough.	April 1892.	Healthy-looking man; signs of small R. pleural effusion. Temp. 99.8° on admission, afterwards normal. Urine 1022, acid o/a o/s. No paracentesis.	Alive and well; occasional pain in L. side when very tired. Chest natural.	None.	Alive.
1893. 1.	F. P. L.	52	M.	Porter.	R.	Father died of potus. No history of phthisis or rheumatism.	Thirty years ago had acute rheumatism; 12 years ago in Abernethy Ward with hydrocele; always healthy.	Quite well till eight days ago, then pain in R. side and shortness of breath. Wet three or four times 14 days ago.	March 1893.	Rather spare man; signs of large R. pleural effusion up to 3rd rib in front, from mid-scapula to base behind. Signs of fluid cleared up after tapping. Left hospital with falling-in of ribs. Temp. after tapping came down to normal. Urine 1026, o/a o/s., urates; occasional faint trace of albumen. Paracentesis, 0iii. clear serous fluid.	Died in Golden Lane Infirmary, 1897, of pulmonary tuberculosis.	Phth.	Died.
2.	J. A.	52	M.	Engineer (fitter).	R.	No history of phthisis.	Acute rheumatism seven years ago; gonorrhœa at 17; moderate ale-drinker.	...	Christmas 1893.	Debility; pulmonary catarrh; small serous effusion at R. base. Temp. very variable, from 99.5° in M. to 98° E.; normal before discharge. Urine 1010, natural. Paracentesis, ℥vi. clear straw-coloured fluid.	Advanced phthisis; moriturus; cavitation at both apices. No signs of fluid.	Phth.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1893. 3.	A. G.	27	M.	Porter.	L.	Mother died of dropsy and morbus cordis; one uncle has phthisis.	Small-pox in infancy; always well; past 12 months vomit after breakfast; 1 pint beer per day.	Nine days ago stabbing pain in L. side; no shivering.	Oct. 1893.	Healthy-looking, strong man; signs of small L. pleural effusion with occasional crepitations at L. apex; this increased and went as high as 3rd L. rib. Cleared up after tapping. Temp. 99.5° on admission, afterwards normal. Urine 1026, o/a o/s. Paracentesis, 0ii. ℥v. clear serous fluid.	Strong, healthy-looking man. Nothing abnormal detected in chest except slightly impaired note with defective vocal vibrations and vesicular murmur at L. base.	None.	Alive.
4.	J. B.	27	M.	Window-cleaner.	L.	One child died at St. Bartholomew's Hospital in 1895 of pleurisy and pneumonia.	First attack 5 years ago.	...	? 1893.	Signs of fluid at L. base, afterwards friction developed. Tubercle bacilli found in sputum. No paracentesis.	Well-marked phthisis with cavitation at L. apex and ? some fluid at L. base. Laryngitis, ? tubercular. No ulcers seen. Much reddening and injection of arytenoids with swelling.	Phth.	Alive.

5.	J. H.	29	M.	Boot-laster.	R.	No history of phthisis.	Three years ago had cough; sputum a little blood-stained; no loss of flesh; otherwise healthy.	Three weeks ago pain in R. side of chest; seven days dyspnœa.	Aug. 1893.	Poorly nourished, looks ill. Nothing abnormal discovered at apices. Signs of a fairly large R. pleural effusion up to spine of scapula behind. Left hospital with signs of thickened pleura. Temp. on admission 100°, fell to normal on 3rd day and remained so. Urine 1020, acid, o/a o/s. Paracentesis, ℥xxxii. clear serous fluid.	Cough since discharge. Hæmoptysis on four occasions, once profuse. Father just dead of phthisis. Much loss of flesh with profuse sweatings. Signs of consolidation at L. apex, and many sharp crackles. Defective expansion L. side, with note impaired L. apex. Exaggerated breathing sounds with prolonged expiration. Soft, shiny, silky skin.	Phth.	Alive.
6.	H. S.	30	M.	Stable-man.	R.	Nil.	Healthy. Never pleurisy before. Not subject to cough. Never hæmoptysis.	Acute onset with rigors and loss of appetite. No cough or pain in chest.	Nov. 1895.	Somewhat wasted. Signs of fairly large R. pleural effusion, with friction at R. apex. Dull over whole R. back, and from second rib in front. Left hospital with signs of collapsed R. lung, with friction at base. Temp. on admission 102.5°, gradually fell to normal; gained 5 lbs. Urine 1020, acid o/a o/s. No paracentesis.	Since leaving St. Bartholomew's has been able to do his work. Cough on and off since, especially in morning, with mucous expectoration. Never blood. No sweating. Short of breath. Very thin and ribs prominent. Much flattening of R. side of chest, especially at apex. Defective expansion R. side with exaggerated vocal vibrations. Vesicular murmur. Harsh at apex, and expiration prolonged, almost bronchial in character. Many crepitations after cough and note at R. apex impaired. Increased vocal vibrations at R. base, with occasional creak on deep inspiration, and defective vesicular murmur to scapula. Phthisis.	Phth.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1893. 7.	F. D.	8	M.	Schoolboy.	R.	One brother died of diphtheria. No history of phthisis.	Nil.	Acute onset.	Oct. 1893.	Signs of fairly large pleural effusion up to 4th rib. Temp. often up to 100° at night. Normal before leaving hospital. Urine 1017, natural. Paracentesis, 0i. blood-stained fluid.	Since being in hospital was under care of Mr. Howard Marsh with tubercular disease of ankle. No cough nor hæmoptysis. Chest natural.	Tubercle of ankle.	Alive.
8.	E. R.	28	F.	Housework.	L.	Mother died of phthisis.	Never ill before, but subject to winter cough.	Ten weeks ago pneumonia and pleurisy; now pain in side and cough.	May 1893.	Anæmic. Signs of fairly large pleural effusion up to 4th rib in front and mid-scapula behind. Gradual improvement till discharged with no fluid. Temp. always subnormal. Gained 4 lbs. Urine natural. No paracentesis.	Died at childbirth from cardiac failure, Sept. 1894.	None.	Died Sept. 1894.
9.	E. R.	48	F.	Housework.	R. and L.	None given.	Acute rheumatism 30 years ago.	...	?	Signs of pleural effusion, double (?); chronic interstitial nephritis, with dilated heart. Temp. normal. Urine thick with albumen. No paracentesis.	Died September 1, 1893. Interstitial nephritis and dilated heart, pleural effusion and "gelatinous" peritonitis.	None.	Died Sept. 1893.

10.	J. H.	19	M.	Piano-forte-maker.	R. and L.	Father suffers from rheumatism; brother from lung disease.	Not subject to cough. Temperate.	One month ago pain in ankles; rheumatism (?). Ten days ago pain, chiefly precordial; cough and dyspnœa; no haemoptysis. Two days ago vomited.	July 1893.	Small double pleural effusion; pericardial friction A.B., 1 inch outside L.N.L. Temp. on admission up to 103°, gradually fell to normal in eight days. All signs clearing up on discharge. Urine 1024, o/a o/s. Paracentesis: 1. ℥vi. clear serous fluid; 2. ℥xxvi. turbid serous fluid.	Alive and well since leaving St. Bartholomew's Hospital. Doing hard work. (Not examined.)	None.	Alive.
11.	J. M.	46	M.	Iron-planer.	R.	None given.	Nothing given.	Quite well 15 days ago, then seized with acute pain in R. side; cough; delirious; no rigors.	July 1893.	R. side of chest full of fluid, and heart apex displaced to sixth space outside axillary line; double aortic murmur; pulse jerky; mitral regurgitant murmur; effusion cleared up. Temp. 101° on admission, almost always up to 99° at night during stay in hospital. Urine 1026, acid; occasionally slight trace of albumen, o/s. Paracentesis, 0iii. s/s. blood-stained serum, which coagulated on standing.	Died in Victoria Park Chest Hospital, Feb. 1897. Tuberculosis pulmonalis, morbus cordis, and exhaustion. No post-mortem.	Phth.	Died Feb. 1897.
12.	D. M.	42	M.	Labourer.	R.	Unknown.	Winter cough and gout; potus.	In usual health until eight days ago, then cold shivers and pain in R. side of chest.	May 1893.	Sunburnt, flushed face; signs of fairly large R. pleural effusion up to mid-scapular region. All cleared up rapidly before leaving hospital. Temp. for first 11 days up to 100° at night, then fell to 99° at night, at which it remained until the last week, when it dropped to normal. Urine 1014, acid, o/a o/s. No paracentesis.	Cough since discharge, with progressive loss of flesh; never haemoptysis; advanced phthisis. Signs of large cavity at R. apex, with softening around; early infiltration of L. apex; whole of R. lung involved; no fluid. Heavy drinker.	Phth.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1893. 13.	B. P.	11	M.	School.	L.	None given.	None given.	None given.	?	Signs of a fairly large L. pleural effusion up to apex in front and supra-spinous fossa behind; friction developed after effusion cleared up. Temp. 101° at night for first week, afterwards normal. Gained 4 lbs. Urine 1014, o/a o/s. No paracentesis.	Quite well since leaving hospital. No cough or pain in side; no symptoms of phthisis. Chest, except poorly covered, natural.	None.	Alive.
14.	G. W. T.	6	M.	School.	R.	Two uncles died of phthisis. Mother and father "weak chests."	Always delicate; weak on his chest and subject to cough. Measles at two.	Twelve days' pain in R. side of chest, with loss of appetite. Previous to this, slight cough.	Oct. 1893.	No enlarged glands in neck. Signs of a fairly large pleural effusion reaching up to 3rd rib in front and mid-scapular region behind. Left hospital with signs of thickened pleura and falling-in of R. side of chest. Temp. occasionally reached 99° at night. Paracentesis, ℥xv. turbid serous fluid, sp. gr. 1020, altogether nearly solid with albumen and contained leucocytes and red blood corpuscles.	Occasional slight cough since discharge. Enlarged cervical glands; cause (?). Feels quite well. No loss of flesh. No symptoms of phthisis. Chest very poorly covered. Nothing abnormal detected in chest except occasional creak at R. apex, which disappears on cough, and signs of thickened pleura at R. base with contraction of R. chest. Nothing definite.	None.	Alive.

15.	G. W. S.	8	M.	School.	R.	Grandmother died of phthisis. Father and mother suffer with chest.	Subject to cough since birth. Never spat up blood. Pleurisy in L. side five months ago.	Fourteen days ago stitch in R. side, headache and cough, vomiting.	Dec. 1893.	Anæmic thin child. Eyelashes long. Signs of a fairly large R. pleural effusion, which cleared up and left friction at R. base. Temp. always up to 100° at night. Gained 1 lb. Urine 1010, o/a o/s. No paracentesis.	Always cough since discharge; two years ago pleurisy; no effusion; always "tired and languid." Nothing abnormal detected in lungs. Chest very flat and not well covered. Tubercular-looking boy. Adenoids. Sweats at night. Never hæmoptysis.	Phth. (?)	Alive.
16.	E. K.	34	F.	Housework.	L.	Father and mother died of phthisis.	Winter cough 14 years ago; no hæmoptysis.	Acute onset with rigors and pains all over.	Sept. 1893.	Signs of small L. pleural effusion; diarrhœa. "Diagnosis — Pleurisy with effusion, phthisis?" Temp. 103° on admission, gradually sank to normal. Lost 3 lbs. in hospital. Urine natural. No paracentesis.	Always well since leaving hospital. Healthy-looking woman. Chest well covered, and nothing abnormal detected.	None.	Alive.
17.	J. H.	49	M.	Street-pavior.	L.	One sister died of bronchitis. No history of phthisis.	Never rheumatism or scarlet fever.	Three weeks ago sharp pain in L. side, then cough and shortness of breath.	Sept. 1893.	Fairly well-nourished man, good colour; signs of L. pleural effusion up to 2nd rib and supraspinous fossa; rhoncus and sibilus over R. lung. Left hospital with impaired note at L. base, with definite vesicular murmur and vocal vibrations. No crepitations or bronchial breathing. Temp. always up to 100° E., normal M. Gained 2 lbs. Urine 1015, o/a o/s. Paracentesis, ℥xxvi. serum.	Died in Bethnal Green Infirmary, September 1895, of phthisis. No post-mortem.	Phth.	Died.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1893. 18.	A. W. B.	7	M.	School.	L.	Not given.	Not given.	Not given.	?	Signs of large pleural effusion, displacing heart to R.; cleared up with definite expansion of L. base. Temp. always normal. Paracentesis, 0i. clear serum.	Cough for some time since leaving St. Bartholomew's Hospital; much loss of flesh. Delicate-looking lad, flushed, and bright blue eyes. Occasional attacks of pain in R. side; signs of consolidation at R. apex, with well-marked bronchial breathing and pecty. at R. supra-spinous fossa. Phthisis (?).	Phth.	Alive.
19.	J. S.	7	M.	School.	L.	Parents healthy; no history of phthisis.	Always healthy; no rheumatism, measles, or whooping cough.	Quite well till 11 days ago, when shivering, vomiting, and pain in L. side of chest.	April 1893.	Small L. pleural effusion, which gradually filled the whole of L. chest. Left hospital with falling in of L. chest and note much impaired on L. side, with definite vesicular murmur; no fluid. Temp. up to 102° E. first 10 days, after up to 99°. Weight stationary. Urine 1018, o/a. o/s.	Fairly healthy-looking child. Chest thinly covered. No cough, and apparently quite well. Nothing abnormal detected in chest.	None.	Alive.

20.	A. F.	23	F.	Housework.	R.	?	Always healthy.	Headache, pain in R. side of chest and dyspnœa seven days ago, with cough.	July 1893.	Seven months pregnant; well nourished; signs of fairly large R. pleural effusion up to mid-scapular region behind; fluid cleared up. Left hospital with impaired resonance, with weak breathing, sounds over lower half of R. back. Temp. normal for some days. Temp. on admission 100°; came down in five days. Urine 1020, o/a o/s. Paracentesis, 0i. greenish-yellow fluid, alkaline, 1016, very easily coagulated.	Quite well till Christmas 1897, then cough and shortness of breath. Has lost much flesh lately, and sweats much at nights. Occasionally slight hæmoptysis with sputum and pain in L. side of chest on cough. Crepitations at R. apex, front and back, with bronchial breathing and brchy. in R. supra-spinous fossa. Movements natural. Phthisis right apex.	Phth.	Alive.
21.	J. P.	35	F.	Housework.	R.	None given.	...	Two months ago pain in L. side, which persisted; has lost flesh; no pain now.	March 1893.	Signs of small R. pleural effusion, which afterwards filled whole of R. chest; friction developed; cleared up after paracentesis. Left hospital with dulness and definite vesicular murmur at R. base. Temp. never came down to normal except in morning; varied between 102° and 100° in evening. Urine 1020; trace of albumen; acid; urates. Paracentesis, ℥v. grains clear serous fluid.	Died at St. Bartholomew's Hospital in May 1896. Post-partum pyæmia. Post-mortem: Cavities both apices, with miliary tuberculosis to both bases. Both lungs adherent. Abscess in Douglas's pouch and L. broad ligament, with plugging of ovarian veins with clots.	Phth.	Died May 1896.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1894. 1.	C. B.	48	M.	Engineer.	R.	No history of phthisis.	Hæmoptysis 28 years ago; pleurisy 18 years ago.	...	July 1894.	Phthisis, with R. pleural effusion. Temp. always up to 101°–102° at night, 99° in morning. Urine 1020, acid, o/a o/s. Paracentesis, ℥xlii. serous fluid.	Since leaving hospital has been in Westminster and City Road Chest Hospitals. Is now in Brompton Hospital with advanced phthisis.	Phth.	Alive.
2.	H. T. T.	49	M.	Bookmarker.	L.	No history of phthisis or rheumatism.	In Luke Ward (St. Bartholomew's Hospital) in May with bronchitis. Acute rheumatism three times; potus.	...	May 1894.	Chest emphysematous; heart, no murmurs; Signs of fluid at L. base, with friction; some fine crepitations in front on both sides, chiefly on inspiration. Discharged with friction at L. base. Temp. very irregular, occasionally up to 99.5° at night. Urine 1018, o/a o/s. Needle inserted and ℥ii. clear serous fluid withdrawn.	Gradually dying of tubercular disease of spine and hip.	Phth.	Alive.

3.	E. P.	36	F.	Housework.	L.	One sister died of phthisis.	Married 16; 12 children dead, 3 living. As a rule healthy.	..	Jan. 1894.	Small L. pleural effusion. Temp. for first 14 days up to 100° at night, afterwards normal. Urine 1014, acid, natural. No paracentesis.	Fairly strong, healthy-looking woman, somewhat weakened by confinements; 15 children in 20 years; patient only 36. Suffers from cough. No signs or symptoms of phthisis. A few rhonci at both bases which clear up on cough. Vesicular murmur good all over. Air entry good; no fluid.	None.	Alive.
4.	S. S.	36	M.	Upholsterer.	L.	No history of phthisis. Father died, cause (?).	Winter cough. No previous similar attack. Variola, 1844. Temperate man.	Quite well till two weeks ago. Rigors, cough, and pain in back, with much perspiration.	July 1894.	Fairly well-nourished man. Physical signs of small L. pleural effusion with friction above it. Tubercle bacilli found in sputum. Gained 6 lbs. during stay in hospital. Temp. always raised at night, 99° or 99.2°. Urine 1018, natural. No paracentesis.	Died in Hackney Road Infirmary, 1895, of phthisis.	Phth.	Died.
5.	K. B.	30	F.	Laundry-work.	R.	No history of phthisis.	Healthy up to a year ago. No cough. Never hæmoptysis.	...	Nov. 1894.	Chronic pleurisy, with some effusion R. side. Temp. always sub-normal after first day, when it reached 100° at night. Urine 1008, o/a o/s. No paracentesis.	Except for occasional "catching" in chest on taking deep breath after taking cold, quite well. No symptoms or signs of phthisis. Chest natural.	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1894. 6.	A. T.	23	F.	Housework.	R.	Brother died of phthisis; father winter cough.	Never strong. Rheumatic fever four years ago.	...	Nov. 1894.	Thinly covered chest. Small pleural effusion R. base. Pneumonia (?). Temp. 103° at night for five days, afterwards fell to normal. No paracentesis.	Seven months pregnant. Well since leaving St. Bartholomew's Hospital. Note slightly impaired at R. base, with definite vesicular murmur and expansion. No fluid. Vocal vibrations equal all over.	None.	Alive.
7.	E. B.	19	F.	Jute weaver.	L.	Good. No history of phthisis.	Subject to winter cough. One confinement three months ago; previous to that, pain in L. side on and off.	...	Dec. 1894.	L. pleural effusion tapped —4″ pressure with variations 1½″. Respiratory movements converted into pneumothorax. Temp. up to 100° for four days after aspiration, otherwise normal. Urine 1028, acid, o/a o/s. Paracentesis, 0iv. ℥xii. turbid serous fluid.	Seven months pregnant. Quite well since leaving St. Bartholomew's Hospital.	None.	Alive.
8.	H. A.	18	M.	Paintbrush maker.	L.	Good. No history of phthisis, rheumatism, or gout.	Always healthy; never cough.	Acute onset with pain and cough.	June 1894.	Signs of small L. pleural effusion with friction at base of L. axilla. Temp. always normal. Urine 1026, natural. No paracentesis.	Alive and well. Living at 8 Magpie Road, Norwich, and now moved to Bristol. Writes is always in good health.	None.	Alive.

9.	J. H.	39	M.	Brick-layer.	R.	Nil.	Always good health. No cough.	Acute onset with pain and cough.	June 1894.	Physical signs of large pleural effusion up to 1st rib in front. Temp. irregular. On admission 103°; gradually fell to normal, and then occasionally went up to 100° at night. Paracentesis, 1st, 0ii. s/s. clear serous fluid; 2nd, 0ii. ℥xvi. clear serous fluid.	Except for slight attack of bronchitis has been well since leaving St. Bartholomew's Hospital. Potus. No symptoms of phthisis. Strong-looking man. Nothing abnormal detected in chest except signs of thickened pleura at R. base. No fluid.	None.	Alive.
10.	E. D.	22	F.	House-work.	R.	Good.	Never laid up before.	Acute onset; pain R. side on breathing.	June 1894.	Physical signs of large pleural effusion; whole of R. front dull, with absent vesicular murmur and vocal vibrations. Temp. gradually fell from an evening temp. of 102° and morning temp. of 99° to normal. Urine 1024, acid, natural. Chlorides not diminished. Paracentesis, ℥xvii. clear serous fluid.	Quite well since leaving hospital. No cough, and no symptoms of phthisis. Chest, nothing abnormal detected except that the note is slightly impaired at R. base, with deficient vesicular murmur. Vocal vibrations present and equal.	None.	Alive.
11.	J. H.	28	M.	Market-porter.	L.	Father died of rheumatic fever, otherwise good. No history of phthisis.	Nil. Strong and healthy; variola at two.	Acute onset with cough and shortness of breath.	Sept. 1894.	Physical signs of fairly large pleural effusion up to supra-spinous fossa behind; apex beat displaced to R.; discharged with loud friction under L. nipple. Temp. at first raised to 101°, gradually fell to normal. Urine 1024, acid, o/a o/s. Paracentesis, 0iv. s/s. clear serous fluid.	Quite well since leaving St. Bartholomew's Hospital. Not troubled with cough; no wasting; always well. Lives at Northampton. (Not examined.)	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1894. 12.	A. M. L.	23	F.	Hat-maker.	L.	Mother and sister died of phthisis since patient left hospital.	Healthy, except cough in winter. Loss of flesh four months.	November 12th, cold shivers, with pain in L. side of chest. Vomiting.	Nov. 1894.	Signs of large L. pleural effusion, dull apex-base; heart, apex beat displaced to R. and downwards. Temp. on admission varied between M. 102.5°, E. 100°; gradually fell to normal, though occasionally up to 99.5° at night. Urine 1020, natural. Paracentesis, 0ii. s/s. clear serous fluid.	Pain returned after leaving hospital, and occasional attacks of pain now when she "catches cold." Cough every winter. Slight loss of flesh. No hæmoptysis. No night sweats. Delicate-looking woman. Chest not well covered. Deficient vesicular murmur R. base. Nothing definite.	None.	Alive.
13.	C. C.	13	F.	School-girl.	R.	Good. No history of phthisis.	Scarlet fever at five; chickenpox and measles; acute rheumatism just before admission.	Sudden onset, with shivering and pain in R. side.	March 1894.	Signs of small effusion at R. base and mitral regurgitation. Temp. very variable for first three weeks, M. 100°; E. 98.6°; afterwards normal. Urine natural. No paracentesis.	Empyema in Lawrence Ward, R. side, 1895, since then quite well. Signs of mitral regurgitation. Impaired note at R. base with deficient vesicular murmur. Abscess in R. breast, discharging. No signs of phthisis or empyema.	None.	Alive.

14.	W. M.	14	M.	Schoolboy.	L.	Good. No history of phthisis, rheumatism, or gout.	Measles at five; subject to cough; otherwise healthy.	Insidious onset. Drowsy and apathetic for 16 days. No cough or pain complained of.	April 1894.	Large L. pleural effusion; dull below second rib; apex displaced to R. of sternum. Temp. on admission, M. 102.5°; E. 99.5°; for seven days, afterwards normal. Gained weight. Urine 1018, natural. Paracentesis, 0i. ℥xvii. clear serous fluid.	Since leaving St. Bartholomew's Hospital has been quite well. No cough. Chest flat, not well covered. Delicate-looking lad. Finger-nails much clubbed and incurved. Impd. note with deficient vesicular murmur at L. base.	None.	Alive.
15.	T. M.	40	M.	Labourer.	L.	Good. Father died of diphtheria (?).	In Poplar Hospital seven years ago with injury to spine; pleurisy L. side same year; otherwise good health.	Acute onset with severe pain in L. side and fever.	June 1894.	Fairly large L. pleural effusion; dulness up to third L. rib in front. Temp. for 10 days up to 100° at night, normal in morning; afterwards normal. Gained 5 lbs. during one month. Urine natural. No paracentesis.	Died of tubercle of lungs, November 1897.	Phth.	Died Nov. 1897.
16.	J. W.	38	M.	Slaughter-man.	L.	Father dead, cause unknown. Mother suffers from rheumatic fever. No history of phthisis.	Potus and gonorrhœa.	Acute onset with pain and cough.	Sept. 1894.	Signs of large L. pleural effusion, with displacement of heart's apex. Temp. always slightly raised at night, and occasionally went up to 101°. Urine 1009, o/a o/s. Paracentesis, 0iv. greenish, opalescent fluid; much fibrine; sp. gr. 1022. Alkaline; much albumen.	Cough with slight loss of flesh; no hæmoptysis. Chest L. side collapsed and very little air entering L. lung. Note impaired at L. base, with defective vesicular murmur and voice sounds. Vocal vibrations natural. No active signs. Collapsed lung.	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1894. 17.	A. C. A.	34	F.	Housework.	R.	Father dead, cancer stomach. Mother rheumatism. One brother meningitis after injuries.	Measles and whooping-cough as a child. When eight in Elizabeth Ward with hydatid cyst, which was tapped once, and afterwards laid open and drained. Two months ago in Elizabeth Ward for pain in R. side and jaundice.	Dyspeptic symptoms some months. Pain R. axilla and breast, 1894. Gradual loss of flesh.	July 1894.	Signs of small R. pleural effusion. Old hepatic irregularity, sequel of hydatid. Temp. varied between 100° and 99° during stay in hospital. Urine 1020, o/a o/s. No paracentesis.	Died of phthisis and exhaustion after childbirth, February 1897.	Phth.	Died Feb. 1897.
18	G. C.	32	M.	Labourer.	R.	Nothing important. No history of phthisis or rheumatism.	Enteric fever in India seven years ago; three years ago pleurisy and inflammation of lungs. No other illness. Twelve years in army.	Dull chronic onset, with dull aching pain in R. side.	Feb. 1894.	Signs of large pleural effusion reaching from 3rd rib in front. Temp. always subnormal. Lost 4 lbs. whilst in Bartholomew's. Urine natural. Paracentesis, 0ii. clear serous fluid.	Died suddenly of hæmoptysis, Christmas, 1895.	Phth.	Died Dec. 1895.

19.	W. H.	30	M.	Cabinet-maker.	R.	Mother died of phthisis, otherwise good.	Heavy drinker and "bad" from result of it in 1892.	Wet through, July 9th. July 10th, cold shivers. Vomiting, cough, and pain in side.	July 1894.	Signs of large R. pleural effusion, filling whole of R. chest. Temp. 100.4° on admission; gradually fell to normal. Urine 1020, acid. Paracentesis, 0iv. clear serous fluid.	Died of phthisis in Bethnal Green Infirmary, March 10, 1897.	Phth.	Died.
20.	B. N.	16	M.	Tailor.	L.	None given.	Could not be obtained, as he speaks neither English, French, nor German.	Acute onset with rigors and pain in L. chest.	April 1894.	Anæmic, looks ill. Signs of large pleural effusion. In hospital 8 weeks; left with L. chest much fallen in and signs of collapsed lung. Whilst in, attack of acute tonsillitis. Temp. much raised. For first five weeks up to 100° at night; after tonsillitis fell to normal. Urine 1022, o/a o/s. Exploratory puncture; clear serous fluid.	Quite well since leaving hospital. Anæmic, delicate-looking Jew. Some contraction and definite expansion of L. chest. No signs of fluid. Note slight against L. side in front and behind, with definite vesicular murmur. Nothing active discovered. Probably thickened adherent pleura with some collapse.	None.	Alive.
21.	J. H.	52	M.	General labourer.	L.	One sister died of dropsy; two brothers died of asthma. No history of phthisis.	L. pleurisy nine years ago, otherwise well.	Insidious onset with pain down sternum. Expectoration tinged with blood.	April 1894.	Moderately well-nourished man; looks ill. Signs on admission of small L. pleural effusion, which gradually filled whole of L. side of chest. No tubercle found in sputum. Self-discharged. Temp. on admission 100.5°; gradually fell to normal, but again rose to 100° before discharge. Urine 1020, o/a o/s. Exploratory puncture, clear serous fluid.	Died in Tunbridge Wells Infirmary of malignant disease of lung.	None.	Died.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1894. 22.	S. A. H.	22	F.	Housework.	R.	Very good. No history of phthisis or acute rheumatism.	Always good health; 6½ months' abortion.	Six months ago breath began to get short, with shivers and pain in R. side.	Jan. 1894.	Well-nourished. Signs of large R. pleural effusion filling R. chest. Tapped three times. Temp. always normal. Lost 4 lbs. Paracentesis, 1. ℥xxxii. gs. blood-stained serum. 2. ℥lii. blood-stained serum. 3. ℥xv. blood-stained serum.	Cough in winter. Under treatment Jan. 1895, cough and dyspnœa. Sciatica, 1896. No symptoms of phthisis except blood-stained sputum 1 week 3 years ago. Fairly healthy woman, well nourished, signs of collapse of lowest lobe of R. lung. Nothing active discovered. Some shortness of breath on going upstairs.	None.	Alive.
23.	W. D. J.	2	M.	Child.	L.	Good.	Measles seven weeks ago.	Acute onset four days ago, with drowsiness and cough.	Aug. 1894.	Signs of small pleural effusion with much bronchitis on both sides. Temp. 100° on admission; after 1st week subnormal. Urine 1012, o/a o/s. Exploratory puncture. No fluid found.	Since leaving hospital has been quite well. Healthy-looking little fellow. Nothing abnormal in chest.	None.	Alive.

1895.													
1.	H.T.S.	16	M.	Plumber.	L.	Father sciatica; sister pleurisy.	Always healthy.	Insidious onset.	April 1895.	Fairly well-developed lad. Signs of L. pleural effusion; no signs of phthisis. Temp. [occasionally up to 99°, otherwise normal. Urine 1022, natural.	Died of phthisis in Highgate Infirmary, 1896. Extensive cavities in both lungs.	Phth.	Died.
2.	F. W.	55	M.	Porter.	L.	Father died of gout; mother died of old age; no history of phthisis.	Cough 18 months with pain in L. side; whooping-cough when child; variola at seven. Many attacks of gout in left big toe. Potus. never anything wrong with chest before this.	No marked sudden onset.	Early in 1895.	Signs of large pleural effusion, with displacement of heart. Temp. always normal while in hospital. Re-admitted Sept. 21 with phthisis. Sept. 29, two pints turbid yellow effusion removed. Mucous rales at L. apex. Tubercle bacilli in sputum. On one occasion ℥xii. removed and ℥i. gs. Morton's fluid injected. After first day of second admission temperature varied between 98° in morning and 100.5° in evening. Paracentesis, 1st. ℥xliii. sp. gr. 1022, a few leucocytes; 2nd. ℥xlvi.; 3rd, ℥xii.	Died of pulmonary tuberculosis, 1896.	Phth.	Died 1896.
3.	H. C. H.	4	M.	Child.	R.	Mother winter cough.	Always delicate; bronchitis three times; last three mos. ago. Varicella a year ago.	...	June 1895.	Rickety child. Signs of large pleural effusion, reaching up to clavicle in front. Temp. before paracentesis, 101°–102°, afterwards came down to normal. Urine natural; ℥xi. clear serous fluid, solid with albumen.	Cough on and off since leaving hospital. No pain; doing well. Nothing abnormal detected in chest; very flattened. Rickets.	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1895. 4.	A. S.	18	M.	Blind-maker.	L.	Father suffers with rheumatism; uncle died with phthisis. Cousin in Chest Hospital with phthisis.	Two large abscesses lanced in neck four years ago. Influenza three years ago.	...	July 1895.	Left pleurisy with small effusion. Temp. varied between 102°-99° at first, morning and evening; in 10 days normal, and kept down. Urine natural. Left hospital with physical signs all cleared up. No paracentesis.	No symptoms since leaving hospital. Chest flattened. Note slightly impaired, with deficient vesicular murmur at L. base; occasionally a creak on deep inspiration.	None.	Alive.
5.	W. S.	38	M.	Labourer in oil-factory.	R.	Father and mother both dead; latter suffered from bad cough.	Always healthy till six months ago. No history of potus. Gonorrhœa several years ago. Eleven years in army, two years in Malta. No syphilis.	...	Dec. 1895.	Small effusion with friction at R. base. Temp. normal except on four occasions when it reached 99°. Urine 1016-1018. No albumen. No paracentesis.	No work since admission. Always cough and occasional hæmoptysis. Much loss of flesh and purulent expectoration. Advanced phthisis, most marked L. apex. Fluid L. base.	Phth.	Alive.

6.	J. P.	23	M.	Engine-cleaner.	R.	Good.	Never rheumatism. Enteric fever seven years ago; said to have suffered from pericarditis.	...	Nov. 1895.	Fairly well-nourished man; anæmic. Cough since November 1895; has spat up a little blood mixed with phlegm. In Brompton Hospital 14 weeks. R. pleural effusion persistent; washed out, and followed by plugging of portal vein (ascites). Temp. very variable; up to 102° occasionally at night during last three weeks; came down to normal in St. Bartholomew's Hospital. In West London Hospital, 1897. Empyema L. side (opened); discharged for three months. Unable to work for past two years. Paracentesis: 1. ℥xxv. clear serous fluid; 2. ℥xv. clear serous fluid; 3. 0ii. gs. clear opalescent serous fluid; 4. ℥xxxiv. clear serum; 5. chest tapped and R. pleura washed out with warm boracic lotion, gr. iv. ad ℥i.	Left chest much flattened. No active signs. Thickened pleura and collapsed L. lung, lower lobe.	None	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1895. 7.	W. F. M.	10	M.	Schoolboy.	R.	Nothing in notes.	Always healthy.	...	June 1895.	Small pleural effusion R. base with friction, which cleared up before leaving hospital. Temp. on admission and for a week varied between 100° and 98° morning and evening; afterwards normal. No paracentesis.	Quite well till just recently, when cough developed. Healthy-looking boy. Never hæmoptysis. Chest flat. Gaining flesh. Nothing abnormal detected in chest except some rhonchus at L. apex, which disappears on cough.	None.	Alive.
8.	E. A.	39	M.	Bootmaker.	R.	No history of phthisis, rheumatism, or gout.	...	... ...	May 1895.	Small R. pleural effusion, which cleared up before leaving hospital. Temp. varied between 102° and 103° on admission; came down to normal in 10 days; up again to 103.5° for two days, after which fell to normal. Urine 1009; no albumen, no sugar. No paracentesis.	Heavy drinker. Hæmoptysis three months ago. ? cirrhosis of liver. Nothing abnormal detected in chest.	None.	Alive.

9.	H. A. V.	21	M.	Printer.	R.	Mother died of phthisis. Father alive and well.	Always healthy; measles as a child.	...	May 1895.	Signs of fairly large pleural effusion, coming up to 4th rib in front; gradually diminished without tapping. Temp. except for first two days (99°), normal. Urine 1010, acid; trace of albumen; no sugar. No paracentesis.	Quite well since leaving St. Bartholomew's Hospital in 1895. Nothing abnormal detected in chest.	None.	Alive.
10.	S. S.	20	M.	Call-boy.	R.	Nothing of importance.	Influenza three mos. ago.	...	Feb. 1895.	Fairly large R. pleural effusion, reaching up to 4th rib in front. Temp. occasionally up to 100° at night, afterwards normal. Urine natural. No paracentesis.	No chest symptoms since leaving St. Bartholomew's Hospital. Is attending University College Hospital with pain in head (?) due to a fall from a bridge a few years ago. Tendency to melancholia. Nothing abnormal detected in lungs.	None.	Alive.
11.	J. C.	30	M.	Carman.	L.	No history of phthisis or acute rheumatism.	Winter cough, especially during fogs.	...	June 1895.	Large pleural effusion almost filling whole of L. chest. Temp. very irregular; generally up to 100° at night and subnormal in morning; normal last four days in hospital. Urine 1010, natural. No paracentesis.	Died May 1896, of pulmonary tuberculosis, at St. Mary's Infirmary, Highgate.	Phth.	Died May 1896.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1895. 12.	R. C.	29	M.	Pantryman.	R.	Mother died of phthisis; two brothers died of phthisis.	S. C. 10 years ago; gonorrhœa 10 years ago. Five years ago attended Chest Hospital with cough. Potus, beer, much rum during past week.	...	Jan. 1895.	Small R. pleural effusion. Discharged with signs cleared up. Temp. for first two days, 99.5°; afterwards normal. Urine 1020, o/a. o/s. No paracentesis.	Alive and well, living at Seaford. Has suffered with cataract since discharge.	None.	Alive.
13.	T. D.	13	M.	Clerk.	L.	One sister died of phthisis.	Pleurisy after injury to chest, rickets and scarlet fever. No acute rheumatism.	...	July 1895.	Physical signs of small pleural effusion L. base, ? mitral regurgitation. Temp. subnormal after first two days, when it was 100°. Cleared up before leaving hospital. Urine natural. No paracentesis.	Died Sept. 1895. Morbus cordis. Homerton Infirmary. No post-mortem.	None.	Died Sept. 1895.

14.	C. T. C.	38	M.	Mail-driver.	R.	Father died of phthisis, 1897; mother died of phthisis.	Gonorrhœa and rheumatism four years ago. Never hæmoptysis; no wasting; not subject to cough; always previously healthy.	...	Sept. 1895.	Signs of R. pleural effusion, filling whole pleura. Paracentesis on several occasions; clear serous fluid, becoming blood-stained later on.	Much improved by treatment. Nothing abnormal detected in chest except signs of collapse of lower lobe of R. lung and thickened pleura. Exaggerated compensatory bronchial sounds over L. lung. Occasionally creak at R. base on forced inspiration. ? stretching of adhesions. No fluid. Impaired note up to level of scapula.	None.	Alive.
15.	W. J. S.	20	M.	Fancy leather-work.	L.	No history of phthisis or acute rheumatism.	Ulcerated throat; at same time discharge from ears.	...	Feb. 1895.	L. pleural effusion up to 4th rib. Signs of consolidation L. apex. Tubercle bacilli found. Temp. on admission, 103.5°; gradually fell to normal for one day, after which varied between 100° and 98° in morning. 11 lbs. during stay in hospital. Urine 1015. No albumen. No paracentesis.	Had pneumonia January 19, 1896. Delicate-looking lad; constant slight cough. Signs of consolidation at L. apex in front and behind. Showers of moist sounds with inspiratory note and bronchial breathing. Night sweats. Never hæmoptysis.	Phth.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1895. 16.	W. F.	24	M.	Labourer in hot rooms.	L.	One sister died of phthisis. Father and mother healthy.	...	...	April 1895.	Small pleural effusion, reaching up to 6th rib in axilla and middle of scapula behind. Physical signs almost cleared up before leaving hospital. On admission temp. up to 103° at night for three nights; 99° in mornings; gradually came down to normal. Urine 1020, o/a o/s. No paracentesis.	Always felt well since leaving St. Bartholomew's. Chest natural.	None	Alive.
17.	J. S.	28	M.	Fireman.	R.	No history of phthisis or rheumatism. Father suffers from sciatica.	Influenza, 1894. Winter cough several years.	...	June 1895.	Fairly healthy-looking man. Stokes furnaces; constantly exposed to great heat and cold. Small R. pleural effusion (to nipple in mid-axilla, mid-scapula behind). Temp. for 1st week up to 101° at night, afterwards normal. Urine 1022, o/a o/s. No paracentesis.	Cough since leaving hospital, with progressive wasting with night-sweats; never hæmoptysis. Advanced phthisis. "Moriturus." R. side signs of cavitation and breaking down in both lungs.	Phth.	Alive.

18.	T. G.	9	M.	Schoolboy.	R.	Grandfather, grandmother, two uncles and aunt, all died of phthisis. Mother, hæmoptysis; has seven children with bronchitis.	Nothing of importance.	...	April 1895.	Small R. pleural effusion. Discharged and came back from Swanley looking well. Admitted Colston Ward, St. Bartholomew's Hospital, January 1896, and died February 12, 1896, of meningitis. Optic neuritis. No paracentesis.	Died, meningitis, February 1896. Post-mortem not allowed.	Meningitis, ? tubercular.	Died Feb. 1896.
19.	R. C.	16	F.	Schoolgirl.	R.	Good.	Never similar attack. No history of cold or exposure to damp. Always strong.	...	June 1895.	Fairly large pleural effusion at R. base, up to 3rd rib in front and mid-scapula behind. Temp. occasionally went up to 100° at night, otherwise normal. No paracentesis.	Slight cough since leaving hospital. No other symptoms of phthisis. Expansion slightly against L. base, with some rhonchus in L. interscapular region, not persistent.	None.	Alive.
20.	T. T.	48	M.	Stonemason.	L.	Father died of enteric fever. Mother dead; cause (?) No history of phthisis or rheumatism.	In army 23 years. Left in 1892. Egypt, Malta, Gibraltar, and Bermuda. No ague; denies pleurisy in 1874 and 1884(?). Side, and since then pain in L. side from time to time. No history of rheumatism. Has drunk a good deal.	...	May 1895.	Well-nourished, well-built man. Fairly large L. pleural effusion up to 3rd rib in front, and spine of scapula behind. Temp. very irregular; up to 102° at night on admission; gradually fell, but even on discharge up to 99.4° at night. Urine 1020, acid, o/a o/s. Paracentesis twice. Character of fluid (?).	Since discharge, winter cough; unable to work on account of weakness and shortness of breath. No loss of flesh. No symptoms other than above of phthisis. L. side of thorax absolutely immobile. Signs of complete collapse of L. lung from apex to base. L. side of chest much sunken in. No active signs.	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1895. 21.	M. N.	28	F.	Married.	L.	Father died of phthisis. Mother alive and well. No history of rheumatism.	Suffered six years ago from "inflammation of womb." Nothing else.	...	Jan. 1895.	Fairly healthy-looking woman. Small L. pleural effusion. Temp. irregular; 101° at night and 99° on morning of admission; later on came down somewhat; often up to 99° at night. Urine natural. No paracentesis.	Attending Royal Free Hospital since November 1895. R. lung normal. L. signs of cavitation both at base and apex, notably at base. Very few moist sounds anywhere. Not much cough. No fœtid expectoration. ? Tubercle.	Phth.	Alive.
22.	W. L.	20	M.	Carman.	L.	Uneventful.	Nil. Good health.	...	July 1895.	Pale, anæmic man. Small L. pleural effusion. Pericardial friction, which disappeared. Sweatings. Temp. always normal after first ten days, when it reached 105.5° at night. Urine 1014, natural. No paracentesis.	Quite well since leaving hospital. Chest natural.	None.	Alive.

23.	J. M.	26	M.	Steve-dore.	R.	Good. No history of phthisis.	No history of pleurisy or cough. Five years ago had a tumour removed from upper jaw, which recurred and was again removed ? nature. Poplar Hospital for fall on head three years ago. Acute rheumatism ten years ago.	...	July 1895.	Pleural effusion R. base. Nil at apices. Pre-systolic murmur at apex. Paracentesis, 0ii. clear straw-coloured fluid.	Quite well since leaving hospital. Chest natural.	None.	Alive.
24.	H. S.	44	M.	Upholsterer.	R.	Father died of Bright's disease. Maternal grandfather died of phthisis.	Potus. Measles and scarlet fever as a child. ? typhoid æt. 7.	Two months chronic insidious onset with malaise and evening vomit. No pain until four weeks ago. Fell into Thames.	April 1895.	Anæmic. Looks as if he had been ill for some time. Fairly large pleural effusion up to 5th rib in front, and spine of scapula behind. Temp. always normal. Urine 1016, acid; trace of albumen, 0/s. Needle withdrew clear serous fluid.	Was quite well till Christmas 1897, since then bronchitis three weeks. Always somewhat short of breath. No hæmoptysis. Mucous expectoration, chiefly in morning. Sweats at night. Putting on flesh. Anæmic. Note impaired at R. base, with defective vesicular murmur; vocal vibrations all over. No moist sounds. Heart irregular, with signs of hypertrophy. Urine 1010, acid; trace of albumen. ? chronic interstitial nephritis. Eyes natural; no optic atrophy.	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1895. 25.	G. A.	43	M.	Packing-case maker.	L.	Three children died of bronchitis. One brother phthisis (?).	Quite well except for cough for years. Always strong.	Christmas 1894, hoarseness. February 1895, influenza. May 1895, pain in L. side on deep breath.	April 1895.	Physical signs of fairly large pleural effusion, reaching up to 2nd rib in front and mid-scapula behind. A few soft crepitations at R. apex. No tubercle bacilli found in sputum. Left with no signs of disease. Temp. at first slightly raised to 101° at night, afterwards normal. Urine natural. Paracentesis. Clear serous fluid.	Continued cough since leaving hospital. Expectoration mucous; on one occasion blood-stained. Has lost flesh. No night sweats. Not able to work on account of shortness of breath. Signs of consolidation L. apex, with harsh breathing and crepitations and rhonchus; vocal sounds exaggerated, commencing R. side. Well-marked phthisis. No fluid.	Phth.	Alive.
26.	J. N.	24	M.	Confectioner.	R.	Both parents died of phthisis.	Similar symptoms to present three months ago. Got well under treatment. Three years ago palpitation of heart, gnawing pain in head. Four months ago slight hæmoptysis.	Quite well till three months ago, when he began to have pain in R. side of chest and felt ill. Hæmoptysis for two days.	March 1895.	Diagnosis: Tubercle R. apex. Small pleural effusion same side. Bacilli found in sputum. No paracentesis.	Slight cough since leaving hospital. No pain, no loss of flesh. Never hæmoptysis. Anæmic. Chest well covered. Nothing abnormal detected. No signs of phthisis.	? Phth.	Alive.

27.	W. E. N. D.	23	M.	Medical student.	...	Uneventful.	...	...	April 1895.	Physical signs of a small pleural effusion which cleared up. Temp. 101°–99°. Normal before discharge. Slight friction at R. base on discharge. No paracentesis.	Healthy - looking, robust, good colour. Doing his usual work, and apparently well. Not examined.	None.	Alive.
28.	W. M.	38	M.	Indoor servant.	L.	Father died of typhus; mother died of phthisis; one sister alive and well.	Always healthy. Never laid up.	Cough 6 or 7 weeks, then pain in L. side and headache. No history of chill.	Feb. 1895.	Sallow - looking man. Looks distressed and ill. Large L. pleural effusion up to 2nd rib in front. Left hospital with almost complete collapse of L. lung. Temp. on admission 103°; gradually fell to normal before discharge. Lost 4½ lbs. in hospital. Urine natural. Paracentesis, fluid, 0iv. Character (?).	Quite well except for pain in knees and hips. Able to do his work. Writes from Newcastle that he is in good health.	None.	Alive.
29.	J. R.	36	M.	Clerk (formerly in army).	R.	Not stated.	Not stated.	Not stated.	?	Well-nourished man, with signs of large pleural effusion filling R. chest. Aspirated twice. Fluid persistent. Temp. on admission 101°, and after first aspiration up to 103.4°. Afterwards gradually came down to normal. Urine 1010, natural. Paracentesis, 1st. 0iii. clear serum; 2nd. 0i. clear serum.	Died, February 1898, at East Dulwich Infirmary. Post-mortem—malignant disease of stomach, ascites, tubercle at R. apex.	Phth.	Died Feb. 1898.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1896. 1.	J. R.	45	M.	Labourer.	L.	Two brothers died with phthisis; one brother died of drink.	Always healthy. No history of cough.	Sudden onset, with pain, headache, and cough.	June 1896.	Healthy, strong man. Signs of large pleural effusion almost filling L. side of chest. No signs of phthisis. Paracentesis thoracis, 5½ pints clear serous fluid.	Died Oct. 1896, with tubercle of lungs and exhaustion.	Phth.	Died Oct. 1896.
2.	M. E. N.	38	F.	Housework.	L.	Mother died of phthisis. Otherwise good.	Deafness since 18. No discharge. Influenza two years ago. Cough one year, with loss of flesh. No sweats or hæmoptysis.	Insidious onset, with cough and slight pain, followed by dyspnœa.	April 1896.	Signs of fluid in L. pleura, which cleared up and left persistent friction at L. base. Temp. normal after first two days. No paracentesis.	Died March 1897, with pulmonary tuberculosis.	Phth.	Died March 1897.
3.	E. L. P.	24	M.	Medical student.	R.	Father pleurisy; brother pleurisy; mother died of pneumonia.	...	Insidious onset after exposure.	March 1896.	Ninety-seven ounces clear fluid, sp. gr. 1020; alkaline; thick with albumen; no blood.	Died at Brentwood Asylum(?). Tubercular peritonitis.	"Tubercular peritonitis."	Died Feb. 1897.

4.	W. D.	6	M.	Child.	L.	No history of phthisis.	Always bronchitis. Weaned at 14 months.	Onset with cough and pain in R. side of chest.	June 1896.	Signs of a small L. pleural effusion; a few râles over L. side of chest, with slight dulness at the L. apex. Temp. 102°–100° first week, after which 98°–99°. No paracentesis.	Continual cough since discharge. Delicate child, anæmic. Attending Children's Hospital for bronchitis. General bronchitis; no signs of consolidation; slight flattening at R. apex; No fluid.	Nothing definite. Phth. ?	Alive.
5.	K. R.	36	F.	Housework.	R.	No history of phthisis. One child died with bronchitis.	Winter 1895 bad cough, with much expectoration.	After confinement three months ago, cough and pain in R. side, which persisted till admission.	July 1896.	Physical signs of a small R. pleural effusion, which was absorbed. No paracentesis.	Patient now very anæmic, with constant cough; some loss of flesh with night sweats; persistent crepitations and flattening at R. apex; also signs of thickened pleura at R. base. No signs of fluid.	Phth. ?	Alive.
6.	H. S.	21	M.	Barman.	R.	Good. No phthisis.	Eleven years in army, two years in Malta. Gonorrhœa. No history of syphilis or drink.	Always healthy till six months ago, then first had pain in R. side with cough.	Nov. 1896.	Small effusion and friction at R. base. Urine 1016; no albumen. Temp. 99° on admission, and then came down to normal. Two drachms of blood-stained serum drawn off. Nothing afterwards.	Now is a healthy, strong man; good colour; no cough; nothing abnormal detected in chest.	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1896. 7.	E. D.	19	M.	Carpenter.	R.	No history of phthisis.	Always healthy. No illness.	Acute onset, with pain, cough, and headache.	Aug. 1896.	Fairly large pleural effusion; no signs of phthisis; discharged with thickened pleura at R. base. Temp. varied between 103°–100°, even on discharge. Paracentesis, 2 pints 5 oz. clear serous fluid.	Often has pain at seat of puncture; much cough and expectoration; no loss of flesh; sweats much at night; never hæmoptysis. Nothing abnormal detected except signs of thickened pleura at R. base.	Phth. ?	Alive.
8.	H. W.	30	M.	Cellarman, wine and spirits.	R.	Father died of phthisis. Otherwise good.	Pleurisy in 1885. Otherwise healthy.	Acute onset, with pain and cough with shiverings.	Feb. 1896.	Small pleural effusion at R. base. No paracentesis.	Pain in R. side since discharge. Nothing abnormal detected in chest.	None.	Alive.
9.	J. J.	3½	M.	School.	L.	Two relations on father's side died of phthisis.	For about a year has "suffered with his chest." No measles or whooping-cough.	Malaise for a week. Then pain in L. side and shortness of breath.	Aug. 1896.	Fairly well-nourished child. Rickets; signs of large effusion filling L. side of chest. Temp. at first reached 101°, afterwards normal. Paracentesis. One pint clear serous fluid.	Rickety child. Always short of breath. Nothing abnormal detected in chest except rhonchus at both bases; apices appear natural.	Bron.	Alive.

10.	W. E.	56	M.	Labourer.	L.	Good.	Never any other illness.	Acute onset with pain and cough. Pain came on suddenly at night.	May 1896.	Healthy strong man, with signs of a large pleural effusion, almost filling L. side of chest. No signs of phthisis. Paracentesis: 1st, 3 pints of greenish yellow serum; 2nd, 1½ do.	Since discharge gradual wasting and cough. Well-marked signs of phthisis at L. apex. Impaired note at L. base with deficient vesicular murmur. Night sweats profuse.	Phth.	Alive.
11.	W. E.	20	M.	Warehouseman.	R.	None of phthisis.	Influenza 1890. Subject to sore throats.	Acute onset.	Mar. 1896.	Large R. pleural effusion; pleuro-pericardial friction developed. Left hospital with signs of collapse of R. lung. Paracentesis: 1st, 3⅛ pints clear serum; 2nd, 3¾ pints do.; 3rd, nothing obtained.	Patient looks extremely well; some dyspnœa on exertion; has put on much flesh. Signs of collapse of lower two lobes of R. lung. Heart drawn over to R. side. No active signs.	None.	Alive.
12.	S. A. W.	8	M.	Child.	L.	None of chest complaints, gout, or rheumatism.	Always strong.	Acute onset.	April 1896.	Signs of pleurisy with effusion at L. base. Temp. always normal. No paracentesis.	Delicate, flat-chested child, poorly covered. Continual cough. Nothing abnormal detected except occasional crepitations at L. base, not persistent.	None.	Alive.

TABLE OF CASES—*continued.*

Case.	Name.	Age.	Sex.	Occupation.	Side Affected.	Family History.	Past History.	History of Present Condition.	Date of Onset.	Condition on Admission, with Character of Fluid.	Remarks, 1898.	Phthisis.	Result.
1896. 13.	A. E. M.	17	M.	Telegraph messenger.	R.	No history of phthisis.	Measles and rickets.	Acute onset with pain, headache, and cough.	Feb. 1896.	Small pleural effusion at R. base. No paracentesis.	Quite well since discharge; gaining flesh. No cough. Nothing abnormal detected in chest.	None.	Alive.
14.	E. D.	43	M.	Stick-mounter.	R.	Good. No history of phthisis.	Always healthy.	Acute onset.	July 1896.	Limited pneumothorax and small pleural effusion. Clear serous fluid on exploratory puncture.	Quite well since leaving hospital; gaining flesh. Tubercular? glands removed as a child. Nothing abnormal detected in lungs.	None.	Alive.
15.	H. B.	35	M.	Butcher.	R.	Father died insane. One brother gout.	...	...	Feb. 1896.	Paracentesis, 47 oz. clear serous fluid withdrawn.	Short of breath since discharge; losing flesh; much cough. Soft, glossy, shiny skin. Potus. Signs of phthisis at R. apex. Deficient expansion with impaired resonance and vesicular murmur at R. base. No signs of fluid.	Phth.	Alive.

16.	C. B.	30	F.	Housewife.	L.	Father died of acute rheumatism and dropsy. No history of phthisis.	Always well. No serious illness.	Acute onset with pain and cough. Afterwards shortness of breath.	Sept. 1896.	Signs of fairly large pleural effusion, which was tapped twice. 1st, 2 pints clear serous fluid; 2nd, 2 pints do. Temp. morning 99°, evening 101° for first week, afterwards normal.	Slight cough and pain in L. side since discharge; some loss of flesh; very anæmic. Night sweats and shortness of breath. Signs of phthisis at L. apex. Note impaired at R. base with deficient vesicular murmur.	Phth.	Alive.
17.	J. D.	5	M.	Child.	R.	Good.	Measles four months ago. Weaned 2½ years. Rickets.	Malaise one week, then cough and pain in R. side.	Aug. 1896.	Anæmic child, fairly well nourished. Signs of fluid in L. pleura. Cleared up before discharge. One oz. blood-stained fluid withdrawn.	Child always in good health. Chest natural, except flat. Rickets.	None.	Alive.
18.	J. R.	35	M.	Furniture porter.	R.	Father died of bronchitis. Mother died at change of life. No history of phthisis.	In bed two months with rupture of blood-vessel in L. side. Pleurisy and influenza.	Fourteen days ago cold shivers. Pain in R. side.	Jan. 1896.	Well-nourished, good colour. P. S. of large pleural effusion up to 3rd rib in front and spine of scapula behind. Temp. at first 101° at night, afterwards normal. Gained two stone. Urine natural.	Always slight cough since discharge, and attacks of fainting. Cannot do heavy work as is very short of breath. Signs of collapsed lung and thick pleura at R. base.	None.	Alive.