On the relations between 'idiopathic' pleurisy with effusion and tuberculosis : a thesis for the degree of Doctor of Medicine / by Ernest H. Cartwright.

ON THE RELATIONSHIP

BETWEEN

(7.)

"IDIOPATHIC" PLEURISY WITH EFFUSION & TUBERCULOSIS.

A THESIS

FOR THE DEGREE OF DOCTOR OF MEDICINE.

BY

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RELATIONSHIP BETWEEN "IDIOPATHIC" PLEURISY WITH EFFUSION AND TUBERCULOSIS.

Knowing that this, a question of considerable interest and of no little importance, is still a very debatable point, it occurred to me when I was House Physician at the City of London Chest Hospital, Victoria Park, that it might be worth while to collate what had been hitherto published on the subject and at the same time to try and follow up, as far as possible, those patients who had in past years been treated in that hospital for apparently primary pleurisy, with a view to ascertaining in what proportion of the cases tubercular disease had ensued.

The investigation of these cases, unfortunately, turned out to be less satisfactory, from a purely statistical point of view, than I had hoped; nevertheless, although the number I was able to collect is but small, I am tempted to bring them forward as being perhaps not altogether without interest, though I shall not pretend to deduce any results therefrom in the form of percentages.

Before dealing with them, however, it is necessary to refer briefly to the history of the subject, to consider what the

principal text-books have to say regarding it, and to give a *résumé* of the various papers which have from time to time been published treating of the matter.

With regard to what may be called the ancient history of the subject, there is but little that need be said.

As we all know, Hippocrates, and even older writers than he, spoke of *pleuritis*, meaning apparently by the term a disease the prominent features of which were pain, or "stitch," in the side accompanied by more or less fever.

Galen (A.D. 131–200) drew a distinction between pleuritis, and pneumonia and peripneumonia. Early writers also made a division of pleurisy into "*sicca*" and "*humida*." But for a long time there was considerable wavering of opinion as to whether or no pleuritis existed as a disease apart from a lung affection.

A French physician, Pinel (1745–1826), was, we are told by Fräntzel (1), the first to put pleuritis definitely among inflammations of serous membranes, and since his time it has been looked upon as an independent disease.

The possibility of drawing a distinction clinically between pleurisy and affections of the lungs was due to Laennec, who, by the publication, in 1819, of his work on auscultation (2), laid the foundation of our present diagnosis, pathology and treatment of pleurisy.

Pleurisy being recognised as a distinct affection, its various forms and their classification came to be studied, and (excluding pleurisy of traumatic origin) we find it broadly divided into:—

(i.) *Primary*, or *idiopathic;* and

(ii.) *Secondary*, that is to say, occurring as part of, or as the direct result of, diseases such as Bright's disease, tuberculosis, acute rheumatism, &c.

I must here note with regard to pleurisy occurring in cases of acute rheumatism, that although Dr. Bristowe includes it among the primary pleurisies (3), most authorities appear to

place it among the secondary. For the present I shall adopt the latter view.

It is the first of the above divisions that I am concerned with in this paper, and more particularly that form which is accompanied by serous effusion; but it will be also necessary to consider in connection therewith those pleurisies which occur secondarily to pulmonary tuberculosis.

By "idiopathic pleurisy" we mean that which comes on without any obvious or probable cause other than cold. Sometimes the history of exposure to cold or wet is very definite, but it may occur without any distinct knowledge of such exposure on the part of the patient, though he frequently ascribes the illness to his having "got a chill" or "caught cold."

In its most acute and typical form the illness commences suddenly with sharp, stabbing pains in the side, some fever marked by a succession of chills, severe pain on taking a full breath, and a short dry cough causing aggravation of the pain. Effusion of fluid rapidly takes place, the pain then diminishing somewhat; the breathing becomes more and more embarrassed; and by the end of a few days or a week the patient's condition demands prompt measures for relief. In such cases it is usual nowadays to perform thoracocentesis, after which as a rule recovery quickly ensues, and in a few weeks the patient appears completely restored to health; though, as Bristowe says, "even in favourable cases, it is usually a long time (it may be months), before friction wholly disappears; and even longer before resonance and respiratory sounds return to the base of the lung."

But the mode of onset is very far from uniform. Frequently it is marked only by slight pain, chilliness and malaise; and the patient, perhaps after a rest of a few days, may return to his work, "until in a week or two or more he is restored to health . . . . or increasing illness and difficulty of breathing make him consult a medical man, who may find his

chest full of fluid" (4). Such cases may be termed *subacute*, and, as will appear later, most of the cases at the Victoria Park Hospital are necessarily of this type.

We sometimes meet with cases where, in spite of a very acute invasion, the disease abates in the course of a day or two without effusion taking place at all. In fact, recovery may ensue at any stage; and all degrees of severity are found between the two extreme forms.

Now, the term *idiopathic* was by the older physicians applied to many diseases, and really signified that they occurred without any preceding lesion or pathological cause being discoverable by any of the means at the observer's disposal. With the modern advance of medical and scientific knowledge and improved means of investigation, such diseases are year by year becoming fewer in number.

Even in so-called "idiopathic" diseases, however, there must clearly be some determining cause, and we find that for many of them exposure to *cold* was accepted as a sufficient determining causative agency.

But by degrees—more especially as the germ theory of disease became generally known and established on a firm basis—it was seen that such a simple explanation was not altogether satisfactory. It was pointed out that whatever action cold may have as a predisposing cause, it would appear unlikely to be the essential cause—the *causa causans*—of the disease. This reasoning has been applied to idiopathic pleurisy, just as it has to other diseases—for instance, acute nephritis. Nevertheless, as a completely satisfactory essential cause has not yet been discovered, be it a germ or anything else, the "cold" theory has not been entirely abandoned, and the text-books still speak of pleurisy *a frigore*. Thus we read in Fagge (5) with regard to cold, ". . . . . one cannot be surprised that many physicians are reluctant to recognise its operation. But the clinical evidence of the direct dependence

of pleurisy upon cold . . . . cannot possibly be explained away." Again, Osler, while pointing out that modern views scarcely recognise cold as more than a predisposing agent, says, "We have not yet brought all the acute pleurisies into the category of microbic affections, and the fact remains that pleurisy does follow with great rapidity a sudden wetting or chill" (6). And Fräntzel holds that cold is a "cause of primary pleurisy, in spite of views widely spread that it is only theoretically a cause" (7). Fowler and Beale, on the other hand, somewhat guardedly say that "it is probable that exposure to cold and wet . . . . are not so commonly the cause of pleurisy as is generally supposed" (8).

Germain Sée, however, refuses altogether to admit cold as a *cause* of pleurisy. He will only admit that it favours its development by allowing the real cause, whatever that may be, to act more rapidly or more efficaciously. In his work on Simple Diseases of the Lungs he divides pleurisy into

(*a*) Pleurisy by propagation.
(*b*) ,, ,, infection.

Cases of so-called pleurisy *a frigore*, he would place some in group (*a*), some in (*b*).

Cold being thus discredited to a large extent as a cause of "idiopathic" pleurisy, the question naturally arises, what else can be the cause?

Various suggestions have been put forward: thus Lancereaux goes so far as to say (9) that "pleurisy should be classed among the infectious maladies, and exposure to cold is nothing but an occasional exciting cause, while the action of the infecting agent still escapes us." He and some other French observers believe that acute pleurisy is "a well defined cyclic malady . . . . and its evolution as constant as that of pneumonia or typhoid fever. It presents regular pathological changes after each seventh day . . . ." and so on.

Shurly ([10]) draws attention to the "Analogy between Acute Idiopathic Pleurisy and Acute Articular Rheumatism," and holds that at any rate many cases of the former are really part of an acute rheumatism, the other manifestations of which are so slight as to have escaped notice. He points out that this relationship was first noticed by Vallieux in 1854, but without attracting much attention; and in support of his view he quotes Koster ([11]), who expresses the opinion that the use of salicylates in the treatment of pleural effusion has not become as general as it should, and mentions 32 cases, of which 27 were "primary," in which under that treatment the effusion rapidly disappeared.

Others, including Aufrecht and Eichhorst, have advocated the same plan of treatment, though apparently without committing themselves to any decided expression of opinion with regard to the rheumatic nature of the pleurisy. ([12])

But the view that has been more generally advanced, and which during the last few years seems to have gained more and more credence, is that all cases of what had formerly been regarded as idiopathic pleurisy are in reality tubercular. This view was first put forward by certain continental physicians, and has recently gained adherents in England, but I do not think that one would gather from our English text-books that such a sweeping statement had ever been seriously entertained.*

The fact that there is frequently an undoubted and close connection between phthisis and pleurisy renders the decision of the question extremely difficult.

Thus it is well known that pleurisy often occurs in the course of, and secondarily to, pulmonary phthisis. Moreover, the first definite symptom of phthisis is not uncommonly an attack of pleurisy, though in the majority of those cases, while

* In the last edition (1891) of Fagge, the bare reference is given in a footnote to Dr. Barrs' paper (*v. infra*), but the subject is not discussed.

the pleurisy is the prominent and urgent symptom, signs of phthisis are unmistakably present also. Such pleurisies are usually dry, and, in conformity with the fact that phthisis almost invariably begins at the apices, located over the upper lobes. Still, sometimes we do undoubtedly get basal pleurisy, with effusion in connection with apical phthisis.

But pleurisy may also occur over the base of the lung as the direct result of that rare form of pulmonary phthisis which commences in the lower lobe. And, again, we sometimes have what is undoubtedly a tuberculous pleurisy, tubercles being evident post-mortem on the pleura, although no tubercular lesion of the lung itself can be found. These cases may arise by rupture into the pleural cavity of a softening tubercular bronchial gland; or sometimes it may be that the starting point is a minute tubercular focus in the lung but so near the surface that its real situation is not detected. It must not be forgotten that occasionally cases of tubercular pleurisy occur in which the tubercles, though plentifully scattered over the pleura, are concealed by deposited lymph, and so might escape the notice of a careless observer.

Then, too, we find cases of pleurisy apparently primary, coming on in hitherto healthy individuals (perhaps, too, after a definite exposure to cold), from which a seemingly good recovery is made, and yet in a very short time unmistakable phthisis supervenes. Here it is not unjustifiable to believe that tuberculosis was, after all, the primary affection. But in other cases the recovery may hold good for several, sometimes for many (even ten to fifteen or more) years, and yet after that lapse of time the patient is attacked by phthisis. Are we to consider that here too the original pleurisy was invariably tubercular? It is of course possible that it was so, and that the disease afterwards became dormant, only to light up in after years, as we know may happen with pulmonary tuberculosis. But seeing how rapidly the pleuritic attack may have

passed off and apparent recovery followed without the patient taking any prolonged rest, or special care of himself, it must, I think, be admitted that such a conclusion is not based on any very sure grounds.

That the connection between the two diseases was long ago noticed, and even resulted in some confusion, we have evidence from Stokes ([13]), who, writing of "pleuritis with effusion," points out that in some cases the patient falls into a hectic condition but little removed from health, when "the disease is almost always mistaken and treated as debility, consumption, &c.; and it too often happens that the neglect . . . . produces the affection for which it was first mistaken." Or he "may pass through the doubtful convalescence of Laennec, under which circumstances he runs the greatest risk of pulmonary consumption."

In 1864 Beau ([14]) noticed that it is not uncommon to see pleurisy occur in a subject who till then has presented no rational sign of phthisis, and to see it followed by the development of that disease.

Walshe ([15]) calls attention to the fact that pleurisy may occur where chronic disease of the lungs pre-existed, but he does not discuss the relation between pleurisy and phthisis beyond noticing that in phthisis we often get pleuritic attacks. Sir Andrew Clark (*Lancet*, 1885) also recognised phthisis as a not infrequent sequela of pleurisy.

"Idiopathic" pleurisy being so rarely fatal in itself, we have no certain means of discovering in any particular case whether or no tubercle is present. It might be thought that the tubercle bacillus were present it would be found in the fluid removed by aspiration, when that operation is performed, but experiments made with regard to this point have been inconclusive. Von Ziemssen inoculated dogs with pleuritic fluid, but without result. Prudden ([16]) making bacteriological examinations of the fluid obtained by an exploring syringe in

21 cases of sero-fibrinous pleurisy, was unable to discover the tubercle bacillus in any, although in three of his cases the pleurisy was secondary to pulmonary phthisis. In 24 cases of empyema he found bacteria in all, but the tubercle bacillus could only be demonstrated in one. He mentions, however, that Pansini had, by animal inoculations, demonstrated tubercle in 6 out of 15 pleuritic effusions. Fränkel, though he thinks that most pleural effusions are tubercular in origin, admits that it is very difficult to discover the bacillus in the fluids, and in some cases of undoubted tubercular origin he, Ehrlich, and others have unsuccessfully sought for the bacillus in the effusion.

Thorowgood ([51]) refers to some experiments of Levy ([52]), who in cases of tubercular pleural effusion failed to find the organism, although in effusions occurring in connection with pneumonia the pneumococcus was demonstrable.

Netter ([17]) injected 15 cases of serous pleural effusion with Koch's tuberculine and of these 13 (87 per cent.) reacted, but, as he rightly points out, we must remember that a certain proportion (8·5 per cent.) of healthy people have been found to react.* He mentions, however, some cases of pleurisy which reacted to tuberculine and in which, though there was no evidence of tuberculosis before, tubercle bacilli were subsequently detected in the sputum. Although he found that pleuritics reacted nearly as often as persons known to be tubercular, his results must be taken as far from decisive, especially as he does not give full details of the cases of pleurisy which were inoculated.

It is necessary that we should bear in mind that a simple pleurisy, not tubercular in itself, may be conceived so to affect the lung (perhaps the consequent thickening of the pleura im-

* Osler ([38]) cites a case where tuberculine was injected for diagnostic purposes in a case of enlarged glands; reaction followed, but nevertheless the case turned out to be cancer.

peding its action or interfering with its blood supply in some way), as to render it more susceptible to the invasion of the bacillus, which finds a nidus in the "crippled" lung that it would not in a normal one.

These considerations will suffice to show how exceedingly difficult it is to arrive at any decided or trustworthy settlement of the question. With our means of investigation, limited as they still are, all we can do is to multiply observations, always paying careful attention to details. It is well to deal only, as has been done by most previous observers, with effusive pleurisies, acute or subacute, for of that nature are the large majority, and the most typical, of what we are accustomed to look on as "idiopathic" cases.

As I have already said, the opinion that the cases we have been discussing are invariably tubercular, was first advocated in France and Germany. The most extreme views are those held by Landouzy, of Paris, who believes that nearly all pleurisies from "cold" are "the expression of an incipient pulmonary tuberculosis which may appear at any subsequent time, even after an interval of many years." He is supported by Joanney [18] and Mayor [19], and to some extent by Germain Sée [20], who quotes Fiedler as reporting that of 112 cases of pleurisy which were aspirated, 21 recovered, 25 died of phthisis, and 66 recovered from the pleurisy, but were found to have other tubercular lesions.

Chauvet [21] and Rühle [22] take more moderate views.

Gerhardt [23], writing in 1879 of pleural effusions, notices that they often precede phthisis, but he gives no sufficient details of cases.

According to Kelsch and Vaillard [24], pleurisy *a frigore* is always tuberculous, but their work is severely criticized by Blachez [25], who considers that many of the cases (16 in number) on which they based their results were not simple acute pleurisy at all, but had previous pulmonary lesions. He

firmly believes that pleurisy can develop in subjects absolutely healthy, and that the disease in these cases can be cured completely, leaving no trace other than a temporary hindrance to the expansion of the lung. "I have followed," he says, "cases for a number of years (some since 1866, when I had treated them by capillary puncture,) that leave me in no doubt on the point." Even purulent pleurisies he does not think are either necessarily or probably tubercular.

Strümpell ([26]), on the other hand, is of opinion that primary pleurisy occurs but very rarely, and that the larger part of all ordinary pleuritic effusions are tubercular. It is interesting to observe that in the American translation of Strümpell's book in 1887, Shattuck inserted an editorial note to the effect that American experience differed from German, for "there can be no doubt that with us the primary affection followed by lasting recovery, either absolute or relative, is common enough." In the following year, however, he writes that his views have undergone a change, for "facts seem to be pointing to the stand taken by some Continental writers that pleurisy is always due to tuberculosis" ([27]).

Dumin ([28]) thinks that most pleurisies are secondary to pulmonary phthisis which has not yet become evident by physical signs, though sometimes the tuberculosis may actually begin on the pleura, as in an instance reported by Delafield ([29]), where in a case of pleural effusion, tubercles were found post-mortem on the right pleura, and there only. In view of Northrup's theory (*v.* p. 18), one would like to have a definite account of the state of the bronchial glands in this case.

As the result of a careful study of pleurisies antedating phthisis, Westbrook ([30]) draws the conclusions that while pleurisy, apparently simple and completely recovered from, may be followed by phthisis even within a few months, nevertheless the pleurisy is here probably a predisposing cause, and not necessarily tubercular itself, and that where phthisis only

comes on many years after an attack of pleurisy, the latter "cannot but be primary and simple."

Blakiston is mentioned in Pepper's Handbook of Medicine as recording 53 cases, not one of which had become phthisical several years after recovery from pleurisy. And Flint gives 47 cases of which no more than three probably, only one certainly, were affected with phthisis subsequently.

I now come to what are, from a statistical point of view, the two most important contributions to the subject that I have found, one is by Dr. V. Y. Bowditch, an American (and to his paper I am indebted for several of the references quoted above); the other by Dr. Barrs, of Leeds.

Bowditch ([31]) dealt with all the cases of pleurisy that had occurred in his father's practice between the years 1849 to 1879, obtaining their after-history wherever it was possible. He took all (whether dry, or with effusion, serous or purulent), except those with suspicion of lung mischief. Out of a total of 90 cases, he found 44 were dead, of whom 23 had died of phthisis; one was still living but phthisical. Nearly all those who had succumbed to phthisis had died within a few years (five or less) of the pleurisy. Two did not die until 18 years afterwards, but he does not say when the disease first showed itself. As he points out, his results show that a large percentage recovered, and never had any recurrence nor subsequent pulmonary or other tubercular trouble; that, as in many cases, phthisis followed within a comparatively short time, special care should be taken during convalescence from pleurisy; but that we are not justified in giving such gloomy prognosis as we should have to give if we accept the extreme views of Landouzy and his followers. He also lays stress on the point that "the assertion that, because phthisis develops in subjects who several years before had pleurisy, the two diseases are necessarily dependent on one another, the intervening period having been one of robust health, is most unwarrantable."

It is, I think, to be regretted that he included cases of dry pleurisy and empyemata, and that having done so, he does not give more details as to the nature and duration of the pleuritic attacks.

Barrs ([32]) investigated the cases of pleural effusion, supposed to have arisen from cold, which were treated in the Leeds Infirmary between 1880 and 1884. Of 57 cases of serous effusion that he was able to trace, 32 had died; of these 3 died in hospital, 1 directly after leaving, and in the remaining 28 the average subsequent duration of life was 2½ years, the maximum being 5 years and the minimum 6 months. As to the cause of death he found it was:--

Phthisis	in 15 cases.
Other tubercular disease (1 acute tuberculosis)	in 3 cases.
Other causes (non-tubercular) ...	in 6 cases.
Unknown	in 8 cases.

With regard to empyemata, which he also investigated, he found the results markedly better, for of 38 whom he traced, 32 were well and only 6 dead, death being due to prolonged discharge in 2 cases, other causes in 2, unknown causes in the remaining 2.

He says that his views, like Shattuck's, have, as the result of experience, undergone a complete change during the last five years; he used to think "that simple pleural effusion was distinctly recoverable, easily treated, and of little danger, immediate or remote," but he now believes that "a very large proportion of the so-called idiopathic cases of pleurisy are really of a tuberculous nature."

Dr. Bramwell ([33]) reports 24 cases which he had aspirated, but the details are not sufficient to make them of any statistical value in the present connection, and, moreover, he seems to have included some effusions due to morbus cordis. He holds that when phthisis does not appear until two years after the

pleurisy, the latter "cannot in fairness be regarded as a cause of the phthisis."

Writing of pleurisy in children, Anders ([34]) gives five cases of pleural effusion which after the lapse of from four to ten years were perfectly well, and he thinks it would be "unjustifiable to attribute any manifestations of phthisis in the future to the original attack of pleurisy."

Dr. A. A. Smith ([35]) mentions a case of serous pleuritic effusion on the right side, which was aspirated twice, and apparently recovered, but six months later died of tubercular peritonitis, and at the autopsy a few tubercles were found on the pleural surface of the diaphragm on the left side, but nothing abnormal in the lungs or the right pleura. He aptly remarks that if so many pleurisies are tubercular, we must admit that tuberculosis of the pleura is more often recovered from than tuberculosis of the lungs.

Sears ([36]) gives an account of five cases of what seemed to be primary pleurisy followed by good recovery in which phthisis appeared after 1, 1, 5, 3, and 3 years respectively. He notices as a source of error in some published series of cases that no distinction is made between dry and effusive pleurisy. He quotes Coustan and Dubrulle ([37]) to the effect that no soldiers who have had pleurisy are fit for duty, and the majority die later of phthisis. He also throws out the suggestion that if pleural effusion, itself not in any way tubercular, occurs in persons who have a latent tubercular focus in a bronchial gland, the large amount of fluid passing through the glands during absorption may possibly dislodge the bacilli, and sweep them into the general circulation.

Osler ([38]) says that in private practice he has been "year by year increasingly impressed by the frequency with which the subjects of pleurisy with effusion subsequently become tuberculous." Still he thinks the modern tendency to consider all acute pleurisies as tubercular is "certainly unfounded" (in

which opinion he is supported by Parsons) ([39]); nor does he consider that hospital figures are by any means in favour of this view.

Again, Béchamp ([40]) does not believe that microbes are of so much importance in acute pleurisy as some would have it, and mentions that he himself had acute pleurisy at 30, and is now alive and healthy at the age of 76.

Oliver ([41]) maintains that exposure to cold may be a cause of pleurisy; but notes that those pleuritic effusions which have an insidious onset, and those that last months instead of weeks, are less likely to be permanently recovered from.

In his recent book on "Diseases of the Lungs," Dr. Douglas Powell writes ([42]), that though an attack of pleurisy favours the possible occurrence of phthisis later on, still if family predisposition be excluded and the attack be distinctly of an accidental character, complete and permanent recovery may be looked for. He does not commit himself to any more definite expression of opinion on the subject. He also gives some statistics, which appear, however, to be those compiled by Barrs.

Fowler ([43]) thinks that "it is highly probable that many cases of pleurisy, which are believed to be of idiopathic origin, are really tubercular."

In what is I believe the newest medical text-book ([44]), it is stated that "we cannot assign very great importance to the fact that a large proportion of the cases of acute pleurisy terminate in recovery, as opposed to the view that the affection is frequently tuberculous."

There is one other paper, by Northrup ([45]), that I will refer to, because it is to some extent, at any rate, suggestive. Speaking of the mode of invasion of the tubercle bacillus, he mentions experiments of Wyssokowicz ([46]), who found that the bacilli could enter through mucous membrane; that they did not grow at the seat of infection, but entered the lymphatics and became lodged in the glands. Their subsequent career

would appear to depend on the power of the tissues to withstand their tendency to grow. Loomis had taken the bronchial glands of persons dead from accident or non-tubercular acute disease, and found that in 8 out of 30 cases inoculation with them caused tuberculosis in animals. One was the case of a woman of 70, who had for a short time before her death been working in a phthisis ward, and her glands quickly caused general tuberculosis.

Northrup himself has often found tubercle bacilli in bronchial glands and nowhere else (*e.g.*, after death from diphtheria, &c.). He puts forward the theory that in cases of tubercular disease, wherever it manifests itself, whether in joints or elsewhere, the primary tubercular lesion is in the bronchial glands. He relates an instance of a child with tubercular disease of the wrist, but no other discoverable lesion, who died shortly after operation, and whose bronchial glands were found, as he had foretold, to be caseous.

If, as would seem probable, from the experiments here cited, so many apparently healthy people have a tubercular focus already present, it is conceivable that after all this may be the real cause of many pleurisies of which cold, though seemingly the essential, is really but the predisposing cause.

It will be observed that most of the recent papers have appeared in American journals; in fact, except the important contribution of Dr. Barrs, very little has been published in England on this subject.

I will now give an account of my own investigations into the cases of pleuritic effusion at Victoria Park. Owing to the letter system being in force at that Hospital, one does not meet there with cases of the more acute type of primary pleurisy. Nearly all that come there are of a subacute character, where either (the pain at the onset not being of very great intensity and the fluid not effused in excessive amount), the patients are content to lie up at home, relieved perhaps to some extent by

domestic remedies, until they can get a hospital letter; or else, as had happened in several of my cases, a medical man is called in to relieve the early acute symptoms, and afterwards, as soon as the severity of the pain abates, his services are for economical reasons dispensed with, and a letter obtained.

My first proceeding was to search the reports for those cases of pleurisy with effusion which might fairly be considered primary, that is to say, which had come on suddenly (whether definitely ascribed to cold or not) in apparently healthy individuals; in which there was no lesion discoverable to which the pleurisy might be supposed to be secondary, and no good reason to suspect a tubercular origin; and in which the patients after treatment had seemed to make a good recovery.

In deciding what period the inquiry was to cover, I had intended to begin with the year 1887, when the clinical reports of the Hospital were first collected and bound; and thence to go down to the end of the year 1890; the latter an arbitrary limit, but one which seemed not unreasonable, allowing as it did an interval of fully three years to have elapsed since the last of the patients was under treatment for the pleurisy.

However, when, after collecting the available cases from the reports, I proceeded to try and communicate with them, I found that, owing to the migratory character of the class of people from which hospital patients are drawn, a large number were impossible to trace. In fact, of the 1887 patients there was only one that I could hear of at all. For this and other reasons I was obliged to confine myself to the years 1888, 1889, and 1890.

During that period the possible cases available were:—

In 1888.—12,	of	whom	I	was able to trace 7
In 1889.—10	"	"	"	6
In 1890.—13	"	"	"	6
35				19

With regard to empyemata, although they are dealt with in this connection by some of the authors I have referred to, I did not include them in my own inquiry, for even when they appear to have commenced as "primary" pleurisies, the presence of pus and so of micro-organisms introduces side issues, and, moreover, nearly all such cases are operated on, and then by the prolonged drainage and retarded convalescence become unsuitable for the present purpose.

On the following pages will be found abstracts of the clinical reports of all the cases that I succeeded in tracing. Let me say here once for all that every report shows that the lungs, apices included, were carefully, and in most instances repeatedly, examined, and found free from any suspicious signs. I need not, therefore, especially note in each abstract that this was so.

To each case I have appended notes of the after-history. All those who are still living, I interviewed myself, and carefully examined.

Case I.

Emily W——, 21, servant. Admitted March 12th, 1888. (Dr. Smith).

Family history good.

Health good until five weeks ago. Then illness began suddenly with pain in both sides, feverishness and shortness of breath. Slight cough and frothy phlegm. Was treated by a doctor, but not getting better, came to the hospital.

On admission.—Much dyspnœa and cyanosis. Signs of fluid on right side as high as scapular spine.

Some blood-stained fluid obtained by exploring needle, but she was not aspirated.

March 26th.—Better. Breath-sounds heard at base.

April 9th.—Cough at night. No expectoration. Note impaired below inferior scapular angle.

April 26th.—Apparently well and went out.

Temperature was rather irregular for first week, but never above 101°. Lower next week. After March 31st normal.

After-history.—She has had good health since 1888, except for a bad cold in April, 1893, when she lost her voice. Was treated as out-patient at Victoria Park Hospital, and got quite well in a month. Used to have some shortness of breath on any especially violent exertion, but that has got gradually better. No cough.

Present condition.—Examination showed very slightly impaired expansion at right base; at extreme base breath-sounds, V.R., and V.F., slightly impaired, and occasionally on deep inspiration a pleuritic creak audible.

Case II.

William C——, 35, stoker in gas-works. Admitted May 3rd, 1888. (Dr. Smith).

Family history good.

Previous history.—Never ill except for an attack of pleurisy on right side seven years ago, when he says his chest was tapped. Was laid up eight weeks and then returned to work. Quite well till present illness.

Six weeks ago, having caught cold, had pain in right side, worse on coughing and deep inspiration. Breath short. Had to give up work.

On admission.—Dyspnœa on exertion. Signs of fluid at right base.

May 4th.—Aspirated. 80 ozs. of fluid withdrawn.

May 28th.—V.F. and breath-sounds good all over, but slightly impaired resonance at base. No pain. Feels well.

June 13th.—Went out.

Temperature.—99° first few evenings. Afterwards normal.

After-history and present condition.—Is a powerfully-built man; perhaps somewhat alcoholic. Weighs a stone heavier than in 1888. Since that time has been "rather liable to colds," and was ill for a week with influenza two years ago. Says his "breath is rather short on violent exertion, especially on lifting heavy weights." Otherwise health good.

Examination.—Lower right side of chest does not expand well, and on that side below level of inferior scapular angle breath-sounds almost inaudible, and V.F., and V.R., diminished. Liver slightly enlarged.

Case III.

Thomas S——, 32, gardener. Admitted May 11th, 1888. (Dr. Smith).

Family history good.

Has always had good health until present illness.

Six weeks ago he caught cold; had shivering attack, with pain in right side for three days. Breath short. Three weeks ago cough came on. No expectoration. A fortnight ago he was much better and was able to go out, but breath is still short.

On admission.—Looks healthy. Dyspnœa on exertion. Slight cough; no pain. Whole of right chest as high as clavicle dull. Diagnosis of pleural effusion; but he was not aspirated. Treated by blisters.

He gradually improved; signs of fluid disappeared and he was discharged on July 5th, feeling quite well, except for a slight "tightness in chest on taking deep breath."

Temperature never above normal.

Re-admitted November 3rd, 1890.—Health good since 1888 till 16 days ago, when pain came on in left side; this has continued since, with slight cough, a little expectoration in morning, and shortness of breath. Has lost flesh during the last fortnight.

On admission.—Left side of chest retracted; dull below level of third rib, with diminished breath-sounds, &c. Fluid diagnosed.

December 19th.—28 ozs. clear fluid aspirated from left pleural cavity. There is now a note, "? phthisis at right apex."

February 5th, 1891.—Has gradually improved and general condition now good. Temperature shows a nearly constant evening rise to 100° or less. Has gained 6 lbs. Nothing more definite made out with regard to right apex. Went out to-day.

Re-admitted December 15th, 1892.—Health good since his last admission until six weeks ago. Then pain came on in both sides, with cough and good deal of expectoration. Has lost flesh considerably during the last few weeks. Dyspnœa on exertion. Has never had any hæmoptysis.

Examination revealed "marked phthisis at both apices, especially left." Temperature very irregular. Tubercle bacilli

present in large quantities. Condition remained about the same and he went out on January 4th.

Soon after he got rapidly worse, and died in March, 1893.

Case IV.

Elizabeth M——, 35, married. Has three children living and healthy. Admitted June 4th, 1888. (Dr. Smith).

Family history.—Father, an uncle, and a sister died of phthisis. Has always had good health, except occasional colds. Was confined two months ago, and seemed to have made a good recovery. Was recently ("fortnight ago") attacked by pain "under right shoulder-blade;" worse on moving; breath became short. No cough or expectoration.

On admission.—Pale and thin; chest thinly covered. Fluid diagnosed at left base; and friction heard in left axilla. Not aspirated.

June 18th.—Appears well, except for some shortness of breath on exertion.

June 30th.—Went home.

After-history, &c.—Has been in good health since she left the hospital. No cough. Weight unchanged.

Present condition.—Healthy-looking and well-nourished. Very slightly impaired note and diminished breath sounds at right posterior base. No adventitious sounds. There is a slightly marked systolic murmur at apex of heart.

Case V.

Hugh S——, 35, printer. Admitted October 12th, 1888. (Dr. Smith).

Family history.—Father and mother both alive and healthy. No history of phthisis in their families. Two brothers and one sister of patient died of phthisis, all aged about 21.

Previous history.—Says he had pleurisy in left side five years ago. Recovered completely from the attack, and was well till six weeks ago, when shooting pains in right side suddenly came on. They became worse, and breath got short.

On admission.—Well nourished. Breath short. Pain in right side. Fulness and impaired movement of right chest. Fluid diagnosed. No aspiration.

November 5th.—Both sides now move equally. Breath sounds heard all over right side, though slightly weak at base.

November 12th.—Fluid apparently all gone.

December 13th.—Went out. Gained 10 lbs. while in hospital.

Re-admitted November 6th, 1890.—Slight cough came on soon after he left hospital in 1888. No expectoration till a few weeks ago; since then a good deal; never hæmoptysis. Emaciation and dyspnœa for the last two months, and considerable night-sweats.

Fingers slightly bulbous. Signs of phthisis at both apices, especially left. No abnormal signs at bases. Considerable dyspnœa.

November 25th.—Signs extended over nearly whole of left lung. Temperature irregular.

Died November 26th.

Autopsy showed both pleural cavities obliterated. Signs of old tubercle at both apices, and recent in middle and lower parts of both lungs.

Case VI.

James G——, 24, labourer. Admitted November 16th, 1888. (Dr. Heron).

Family history good.

Health good until three weeks ago, when he caught cold and had pain in right side; got a little better, but a week ago pain was worse, and shortness of breath came on.

On admission.—Signs of fluid on left side up to level of fourth rib in front and middle of scapula behind.

November 19th.—Aspirated. 30 ozs. dark-colored serum removed. He made somewhat slow progress at first, but gradually signs cleared up, and on February 4th the report says that his general condition is excellent, and the only abnormal signs in lungs are slightly impaired resonance and somewhat feeble breath sounds at extreme left base.

February 8th.—Went out convalescent.

Temperature.—103° first evening; then irregular, but never above 101°, until November 28th. Afterwards normal.

After-history.—He returned to work a fortnight after his discharge, and health has been good since.

Present condition.—Is a strong, healthy-looking man. Both sides of chest expand equally, and nothing abnormal can be detected in lungs.

Case VII.

Charles C——, 18, boot-clicker. Admitted December 12th, 1888. (Dr. Smith).

Family history good.

Patient always quite strong and healthy until this illness, which he thinks is the result of a cold. It commenced three weeks ago with sharp pains down back and front of chest, and he says he "felt chilly." Breath got gradually shorter.

On admission.—Well nourished. Dyspnœa. Cough. Pain all over chest. Chest slightly bulging on right and spaces flattened. Movements on right side almost nil. Apex beat $1\frac{3}{4}$ inch outside nipple line in 6th space. No murmur. Veins of right side of neck dilated. Whole of right chest dull; breath-sounds heard all over, but distant and bronchial in character; V.F. absent on right except at apex; V.R. bronchophonic on right side.

December 14th.—100 ozs. straw-coloured serum removed by aspiration.

December 17th.—Movement better. V.F. present, but weak below rib four. Dull below inferior scapular angle, and in front below fourth rib; and V.R. here is now diminished. Breath-sounds nearly normal.

Apex-beat in 5th space, nipple line. Pulmonary systolic murmur.

January 3rd.—Improving; no cough or expectoration.

January 4th.—Note still impaired at base. Breath and voice sounds, and V.F. slightly diminished. Friction at inferior angle of scapula. General condition good.

Temperature was 100° in evening for first five days. For the next week about 99·4°. After that never above normal.

Gained 6 lbs. in weight.

January 28th.—Went out.

He has had good health since he left the hospital. Has never had any shortness of breath nor cough.

Present condition.—Slight rickety flattening of lower ribs. Expansion of the two sides equal. Nothing abnormal can be detected. Heart normal.

Case VIII.

John B——, 41, blacksmith. Admitted March 7, 1889. (Dr. Smith).

Family history good.

Health good till a few weeks ago when illness began suddenly with pain in right side.

On admission.—Whole of right chest dull, and fluid diagnosed. Apex beat not felt but sounds best heard just outside nipple.

77 ozs. of serous fluid removed by aspiration.

Nothing suspicious detected in lungs. No cough while in hospital.

June 12th.—Went out convalescent.

Re-admitted December 30th, 1889.—Has been very well since June until a fortnight ago, when pain came on suddenly in right side. Has had a "dry" cough since then.

On admission.—Pain in right side on deep inspiration. At right base friction audible, and V.F. and V.R. diminished.

Diagnosis.—"? Fluid or thickened pleura." No signs of pulmonary phthisis.

Temperature was never above normal.

January 13th.—Went out apparently well.

Re-admitted March 13th, 1893.—Was quite well until twelve months ago when he caught cold. Since then has had several colds. Cough for last four months, with profuse expectoration. Emaciation for six weeks. No night sweats.

On admission.—Signs of cavitation at right apex. At right base impaired note; distant bronchial breathing; increased V.F. and V.R.

March 28th.—Signs increasing in extent at right apex. *Temperature* high and irregular.

April 5th.—Hoarseness and dysphagia. Weaker.

Disease rapidly advanced and he died on April 25th.

No P.M.

Case IX.

Stair W——, 25, draper. Admitted April 9th, 1889. (Dr. Thorowgood).

Family history.—No phthisis. Father and mother both alive and healthy, aged respectively 78 and 68.

Previous health always good until three weeks ago. He was then suddenly seized with pain in right side, which lasted three or four days. Had slight cough. For a week coughing or lifting anything caused pain in side. Shortness of breath getting gradually worse.

On admission.—Right side of chest dull up to third rib in front, and to spine of scapula behind, with breath sounds almost absent. Boxy note at apex. Fluid diagnosed.

April 26th.—40 ozs. clear fluid removed by aspiration.

May 11th.—Breath-sounds fair all over right chest, except quite at base, where they are somewhat feeble.

May 24th.—Good resonance all over. Breath sounds only slightly weak at extreme right base.

June 8th.—Went out apparently well.

Temperature in evening varied between 99° and 100° until April 28th. After that normal.

He appears to have continued well until 1891, when he became ill, and phthisis was diagnosed in right lung. In August, 1893, he had an attack of hæmoptysis, and died on September 2nd.

Case X.

Paul C——, 9, schoolboy. Admitted May 16th, 1889. (Dr. Ormerod).

Family history.—Father, and several members of his family, died of "heart disease." Mother healthy; her father and mother died of "consumption." Patient has a brother and a sister alive and healthy. None died of phthisis.

Has always been "delicate" and liable to colds and "bronchitis."

Present illness began with a cold, cough and pain in side, six weeks ago.

On admission.—Chest well formed. Signs of fluid at left base; dulness absolute below level of anterior axillary fold, and breath-sounds absent. Not aspirated.

May 27th.—Breath-sounds audible over whole of left side now. Still dull at back below inferior scapular angle.

June 4th.—Doing well. Signs as before.

June 27th.—Went out convalescent. Gained 4 lbs. Temperature: 99·5° first evening; 99° second evening; after that never above normal.

After-history.—Since he went out, has been quite well except for a week about a year ago, when he had some pain in left side. Says he sometimes notices his breath short on exertion. Never any cough. Works in bakehouse and so is subject to frequent changes of temperature. He is somewhat

pale. Is tall for his age; has "grown rapidly the last two years."

Present condition.—Chest somewhat emphysematous in shape. Movement equal on the sides. Nothing abnormal to be found except at extreme left base where breath-sounds, and V.R. appear very slightly diminished as compared with right side.

Case XI.

George P——, 42, labourer. Admitted August 7th, 1889. (Dr. Smith).

Family history good.

Always enjoyed good health till present illness.

On June 10th he got wet through and caught cold. Felt weight in chest and shortness of breath, with pain in left side. Has not been to work since. Pain got better, but breath worse. No cough. No emaciation.

On admission.—Much dyspnœa even when quiet. Pain in left side and between shoulders. Whole of left side of chest dull, with absence of breath-sounds and V.F., except at extreme apex. Compensatory breathing on right side.

Heart's impulse felt one inch below right nipple.

August 9th.—38 ozs. straw-coloured fluid removed by aspiration.

August 15th.—Left side resonant over upper half. Much better.

August 20.—Went out convalescent.

Temperature on first two evenings reached 100°. Otherwise normal.

Subsequent history.—Seemed quite well for a month. Then felt ill again, and was an in-patient at Brompton Hospital, where his chest was again tapped. For somewhat more than a month after leaving there he was fairly well, but then a cough gradually came on. Was unable to work. Did not seem to get any weaker or thinner until November, 1890, when he rapidly got worse. Was then told by a medical man that he had consumption. In January, 1891, he died.

Case XII.

Emily W——, 22, servant. Admitted September 6th, 1889. (Dr. Smith).

Family history good.

Patient was never ill until three weeks ago, when she caught cold, and cough came on, with some shortness of breath. Very little expectoration. Some pain in right side, but not severe.

On admission.—Impaired resonance over whole of right chest, except apex. Breath-sounds very feeble, and at base absent. Ægophony at inferior angle of scapula. Liver pushed down. Diagnosis, pleural effusion.

September 23rd.—Fluid is being absorbed. Friction sounds heard. Note still impaired, and breath-sounds weak on right side, especially at base.

October 24th.—Went out apparently well. Gained 10½ lbs.

Temperature.—Showed slight evening rise until December 15th, after which it was normal.

I find that she died of consumption in October, 1890, but I have been unable to obtain any details with regard to her health in the interval.

Case XIII.

John B——, 10, schoolboy. Admitted November 11th, 1889. (Dr. Harris).

Family history.—Father and mother both living and healthy. Some phthisis in father's family. None in mother's.

Patient's only previous illness was measles. Otherwise health very good. Was quite well till three weeks ago. Then had sharp pain in left side, which, after lasting on and off for a week, got better. A little cough. No expectoration. Breath became short.

On admission.—Dyspnœa on exertion. No cough. Chest well formed. Fulness on left side with deficient expansion. Left side impaired resonance (getting more marked towards base), below third rib and spine of scapula. Breath-sounds, V.F., and V.R. diminished over dull area. Fluid diagnosed. Heart's impulse in costal angle to left of xiphoid.

November 15th.—Five drachms clear serum obtained by exploring needle. Not aspirated.

November 22nd.—Dulness diminishing. Breath-sounds better.

November 28th.—Heart's impulse just below left nipple. Only dull now at extreme base.

December 13th.—Cardiac impulse normal. Resonance good everywhere. Air entry good except extreme base in axillary line.

January 3rd.—Feels quite well. No shortness of breath. Went out.

Temperature.—101° in evening for first four days. Then a slight evening rise (99°–100°) until December 3rd. After that normal.

Present condition.—Appears perfectly healthy. Has been quite free from cough. No shortness of breath.

Examination.—Expansion good and equal. Resonance good to extreme base. No adventitious sounds whatsoever.

Case XIV.

John A——, 36, labourer. Admitted February 8th, 1890. (Dr. Smith).

Family history good; but his wife died of consumption, after six months' illness, two years ago.

Previous health.—Had two attacks of "inflammation of lungs" when aged 15 and 16. English cholera nine years ago. No other illness, and health very good till after his wife's death, when, owing to worry and discomfort at home he got out of health. Nine months ago had occasional sharp pains in right side; this did not last long. No other symptoms till five weeks ago, when he caught cold; had severe pain in right side and had to give up work. Pain relieved by fomentations. Three days later cough came on and has continued since. Breath got very short.

On admission.—Expansion deficient at right base, where there are signs of fluid. Very slight cough; white frothy expectoration.

February 9th.—19 ozs. serous fluid aspirated from right chest.

February 14th.—Much better. Note only impaired at extreme base. Air enters fairly well. Some shortness of breath on exertion.

February 24th.—No shortness of breath now.

March 14th.—Went out convalescent.

Temperature.—Between 99° and 100° on first three evenings; afterwards normal.

After-history.—Since he left hospital he has never been ill except once for three days with a cold. He is a powerfully-built healthy-looking man, weighing 13½ stone. Has had no cough or shortness of breath.

Present condition.—Nothing abnormal can be discovered in chest except that at right base in axillary line a pleuritic creak is occasionally heard on very deep inspiration.

Case XV.

William C——, 11. Admitted March 27th, 1890. (Dr. Thorowgood).

Family history good.

Previous health good. Five weeks ago was seized with severe pain in left side, aggravated by cough and deep inspiration. [There is also a note of some indefinite pains in joints shortly before, but no details are given]. Pain got less, but is still present. Breath short on exertion.

On admission.—Left side dull below level of inferior scapular angle, with impaired movement, absence of breath-sounds, and diminished V.R. Diagnosis of fluid.

Was treated by Lin. Iodi, and the administration of Quinine and Pot. Iod., and later Iodide of Iron. Was not aspirated. He gradually improved; dulness became less marked, and air-entry better, and on

June 13th resonance was good to base, and air-entry only slightly inferior to right side.

June 20th.—Went out apparently well, having gained 5 lbs.

Temperature showed slight evening rise until March 31st; after that normal.

After-history.—Was well for some few months after leaving hospital, and then a cough came on, which has continued more or less ever since, but never troublesome. A little expectoration, which is said twice to have been streaked with blood about four months ago.

He is very anæmic. Rather small for his age. No clubbing of fingers. Fairly well nourished.

Examination.—Percussion note appears very slightly higher pitched below right clavicle. Above right clavicle a couple of doubtful râles were once heard on deep inspiration, but could not be detected again. In right supra-spinous fossa percussion note is slightly high-pitched. Opposite spine of scapula a doubtful râle was once heard. At extreme left base resonance is impaired, and breath-sounds and V.R. here slightly diminished.

Case XVI.

Joseph H——, 41, turner. Admitted June 7th, 1890. (Dr. Smith).

Family history good.

Had good health till February; then had influenza. Recovered. At end of April caught cold and had severe pain in left side; was in bed a fortnight. Pain still continues, and breath is short.

On admission.—Expansion deficient on left side. Signs of fluid at left base. Heart's impulse not felt. Sounds best heard in 4th left space, one inch internal to nipple line.

He gradually improved, but no aspiration was performed until August 3rd, when 10 ozs. of clear serous fluid were drawn off.

August 13th.—Appears well, and went out.

Temperature was never above normal.

After-history.—Has had excellent health since he went out; never any cold or cough, but he says that occasionally on hurrying or drawing a very deep breath he feels a slight pain in left side

Examination.—Strong, healthy-looking man. Nothing abnormal detected in chest except an occasional pleuritic creak at left base in axillary line on a very deep inspiration.

Case XVII.

Kate N——, 28. Admitted June 12th, 1890. (Dr. Thorowgood).

Family history.—Mother died of phthisis. No other case known in family. Patient has been married seven years, and has three children living. Husband healthy.

She always had good health till two months ago. Then, the time being one month after confinement, she caught cold and was in bed for two weeks; had pain in right side and shortness of breath. Slight cough then and since, especially in mornings; hardly any expectoration.

On admission.—Flattening and impaired movement at left base, and signs of fluid there. Occasional rhonchus heard over upper part of right side. Very little cough and expectoration. She was not aspirated.

June 28.—Much improved.

July 5th.—Cough entirely gone. Resonance better.

July 18th.—Entry of air fairly good. Some pleuritic friction heard.

July 31st.—Went out apparently well.

She has been very well since she left the hospital, except for occasional cold in head; never any cough.

Examination reveals nothing abnormal in chest.

Case XVIII.

John C——, 14, schoolboy. Admitted August 5th, 1890. (Dr. Beale).

Family history.—Maternal grandfather died of phthisis. No other case.

Good health previously except last January when he was laid up for two days with influenza. Got quite well afterwards. Three weeks ago was seized with stabbing pains in left side which lasted a week. Had slight cough which has continued since. Shortness of breath for the last ten days. Has lost flesh since illness began.

On admission.—Bulging of chest wall at left base. Dull below space three on left side; hyper-resonant above. Breath-sounds at base distant tubular. V.F. and V.R. weak.

Heart's impulse not felt. Sound best heard at ensiform. Cardiac dulness extends to within an inch of right nipple line.

August 5th.—22 ozs. clear fluid aspirated.

August 11th.—Aspiration repeated and 53 ozs. withdrawn. Afterwards apex-beat felt in normal position. After this he rapidly improved and on September 2nd was discharged convalescent.

Temperature.—Evening rise to about 100° until August 12th. After that normal.

After leaving the hospital he remained perfectly well for three months. He was then for some fault sentenced to imprisonment in Maidstone Goal. Soon after his committal, on November 28th, he complained of feeling ill, and had a dry cough at times. No physical signs could be discovered until March 24th, 1891, when temperature rose to 101·4° and fine crepitant râles were heard over both lungs. He got rapidly weaker, crepitations becoming coarser, and some dulness

appearing at right base. Sputum, at first white and frothy, became muco-purulent towards end. He got rapidly worse and died on April 3rd, 1891, of "acute tuberculosis." (For this report I am indebted to Dr. Ground, the medical officer of the Gaol).

Case XIX.

Walter S——, 20, wheelwright. Admitted October 18th, 1890. (Dr. Thorowgood).

Family history good.

Patient always had good health until three weeks ago. Then while at work was seized with pains in head and "cold shivers." Slight cough came on, which caused pain in left side. No expectoration. Considerable shortness of breath.

On admission.—Dyspnœa. Some cough. Movement impaired on left side of chest, and slight bulging at left base. Impaired resonance below level of rib four, with absence of breath-sounds, V.F., and V.R. Diagnosis, pleural effusion. Was not aspirated.

Apex beat not felt. Sounds best heard at usual situation of impulse. Slight epigastric pulsation.

October 31st.—Resonant nearly to base. Breath-sounds heard all over left side, but somewhat weak, and expansion still deficient. Some friction at inferior scapular angle.

November 8th.—No cough, and no shortness of breath even when going about. Breath-sounds on left only slightly weaker than on right, and resonance very fair all over left side. Apex beat normal.

November 20th.-- Signs all cleared up.

December 4th.—Discharged convalescent. Gained 3 lbs.

Temperature for first four weeks occasionally 99° in evening. Afterwards always normal.

He returned to work as soon as he left the hospital and has been quite well ever since. Never any cough except for a few days the winter before last. No shortness of breath on exertion.

Present condition.—Expansion seems slightly deficient at left base still. Behind resonance is a little impaired at extreme left base. Breath-sounds, V.R. and V.F. equal on the two sides. No adventitious sounds except on very deep inspiration, when a coarse creak can be heard at left base in mid-axillary line.

There are a few points in some of the above cases to which I would direct attention before proceeding.

Case I.—It is, I believe, generally taught that blood-stained fluid in the pleural cavity points strongly to tubercle; this may be so, but will not the accidental piercing of a small vessel often quite account for the presence of blood, especially in the specimen from a diagnostic puncture? In this case, certainly, there was no reason to suspect tubercle then or since, although the fluid found was blood-stained.

Case II.—This patient, it will be noticed, gave a history of pleurisy seven years previously, which he says was on the same side as his second attack. If that was so, the pleural cavity cannot have become occluded to any extent after the first attack. The nature of his work renders him peculiarly liable to sudden exposure to cold when heated.

Case V.—Although there was, during his stay in Victoria Park Hospital, no good reason (beyond the fact of two brothers and a sister having died of phthisis) to suspect tubercle, the result shows that the attack was probably of that nature. Perhaps the 'old tubercle at the apices' found post-mortem was connected with his former attack of pleurisy, and one wishes it were possible to know whether at that time it gave rise to any physical signs. His occupation was a notoriously unhealthy one. There was no history of 'catching cold.'

Case VIII.—Here again no suggestion of having 'caught cold.'

Case XI.—This case was apparently traced directly from a wetting, and yet phthisis followed so soon afterwards that it was most likely tubercular from the start.

Case XIV.—Although this case might well seem suspicious owing to the possibility of infection from his wife, it turned out well.

Case XV.—The question of rheumatic origin undoubtedly arises here, and yet the after-history and present condition suggest probable phthisis.

Case XVII.—Here it may be noted the pleurisy occurred not long after confinement.

The total number of these cases is far too small to form any basis for merely numerical statistics, but even were there enough for that purpose, I do not think such statistics, with regard to a matter like the present, where so many possibilities of uncertainty exist, are of any really great value.

In making my search through the hospital reports I noticed that a considerable number of cases failed to fulfil the conditions I had imposed, and so were rendered unavailable for my purpose on account of the diagnosis having been qualified by the addition (sometimes not until the patient had been in the hospital for some little time and had been repeatedly examined) of a note "? incipient phthisis at apex." For a specialist to put "?," the signs must needs have been very slightly marked indeed, and it can be well imagined that any less skilled or less careful ear would have passed them over altogether.

So that in determining what cases to include among the apparently tubercle-free pleurisies, the personal equation must enter to a considerable extent. Moreover, some cases will be found to be accompanied by circumstances which, according to the event, might be held to be either significant or meaningless. Therefore, in trying to form an opinion, we must not put too much faith in actual figures, but should rather be guided by a careful consideration, individually and collectively, of all the cases we can meet with; and in all cases of pleurisy the apices must be very thoroughly and repeatedly examined.

On the following page I have, for convenience sake, arranged the cases reported above in tabular form. From this it will be seen that of the 19 cases, ten were right-sided and nine left-sided.

Of the right-sided, 5 have done well, 5 ended in phthisis.

Of the left-sided, 6 have done well, 3 ended in phthisis.

(Trousseau, I may note, believes that nearly all right pleural effusions are tubercular, but Bowditch dissents from this view).

As regards family history (always somewhat uncertain), 14 cases were said to be quite free from phthisical taint, and of these, seven did well, seven badly. In only five was there a history of phthisis, and all but one of these did well.

Again, I find that nine cases were aspirated, while ten were not.

Of the former, 5 are well, while 4 have died of phthisis.

Of the latter, 6 are well, while 4 have died of phthisis.

Reviewing these cases, we must, I think, admit that on the whole the results are certainly better than we should expect on the supposition that tubercle was invariably present in the first instance.

SYNOPSIS OF CASES.

No. of Case.	Sex.	Age.	Family History.	Date of Pleurisy.	Side affected.	Aspirated or not.	Amount of Fluid.	Result up to present.	Remarks.
I.	F.	21	Good.	Mar., 1888	R.	Not aspirated.	—	Good.	Pleuritic creak audible.
II.	M.	35	Good.	May, 1888	R.	Aspirated.	80 ozs.	Good.	Diminished expansion and pleuritic creak.
III.	M.	32	Good.	May, 1888	R.	Not aspirated.	—	Died of phth. Mar. 1893	Was re-admitted in Nov., 1890, with L pleurisy, and was aspirated 20 ozs. Then "? phthisis at R apex"
IV.	F.	35	Phthisis.	June, 1888	L.	Not aspirated.	—	Good.	Pleuritic creak. Mitral systolic murmur.
V.	M.	35	Good.	Oct., 1888	R.	Not aspirated.	—	Died of phthisis, 1890.	
VI.	M.	24	Good.	Nov., 1888	R.	Aspirated.	30 ozs.	Good.	
VII.	M.	18	Good.	Dec., 1888	R.	Aspirated.	100 ozs.	Good.	
VIII.	M.	41	Good.	Mar., 1889	R.	Aspirated.	77 ozs.	Died of phthisis, 1893.	
IX.	M.	25	Good.	April, 1889	R.	Aspirated.	40 ozs.	Died of phthisis, 1893.	
X.	M.	9	Phthisis.	May, 1889	L.	Not aspirated.	—	Good.	Pleuritic creak.
XI.	M.	42	Good.	Aug., 1889	L.	Aspirated.	38 ozs.	Died of phthisis, 1891.	
XII.	F.	22	Good.	Sept., 1889	R.	Not aspirated.	—	Died of phthisis, 1890.	
XIII.	M.	10	Phthisis.	Nov., 1889	L.	[5 dms. by explor. syringe.]		Good.	
XIV.	M.	36	Good (wife phth)	Feb., 1890	R.	Aspirated.	19 ozs.	Good.	Pleuritic creak.
XV.	M.	11	Good.	Mar., 1890	L.	Not aspirated.	—	Incip. phth. R apex.	
XVI.	M.	41	Good.	June, 1890	L.	Aspirated.	10 ozs.	Good.	Pleuritic creak.
XVII.	F.	28	Phthisis.	June, 1890	L.	Not aspirated.	—	Good.	
XVIII.	M.	14	Phthisis.	Aug., 1890	L.	Aspirated.	22 ozs.	Died ac. tub., 1891.	
XIX.	M.	20	Good.	Oct., 1890	L.	Not aspirated.	—	Good.	Pleuritic creak.

After weighing carefully all the evidence I have been able to collect that bears on the subject, I am forced to the opinion that the case for the tubercle bacillus as the true cause of all, or nearly all, so-called idiopathic pleural effusions is by no means made out. When we find cases where phthisis manifests itself during treatment for pleurisy, or very shortly afterwards, no doubt it is justifiable in most instances to conclude that the pleurisy was tubercular, even though it may have seemed directly traceable to cold; but I cannot think that when patients have been, to all intents and purposes, in perfect health for some years (even if only three or four) after pleurisy, and then develop phthisis, we have any right to say that in all cases the effusion itself was in the first instance of a tuberculous origin.

At the same time, I do not mean to imply that recovery *necessarily* excludes tubercle. Indeed, some experiments of Chauffard and Gombault (47) tend to show that it cannot, for they record cases of pleural effusion which ended in "complete recovery," although in some of the cases inoculation of guinea pigs with the infusion had caused tuberculosis. (*See* also note on Case V., above).

Nevertheless, when we consider the amount of attention and prolonged rest that are indispensable for any marked success in the treatment of incipient pulmonary phthisis, and also recognize the fact that patients after pleural effusion not infrequently return to work within six or eight weeks, ceasing to take any further care of themselves, I think, if we supposed tubercle to be always present in the latter class of cases, we should have good cause for surprise at the number of apparently complete recoveries, and that the disease should become and remain latent for so long as it would then appear to do.

One cannot help being struck by the large number of pleuritics who do ultimately succumb to phthisis, but we must not forget that phthisis is a very widely-spread disease, and

that it is only natural to suppose that a lung crippled by a thickened pleura or other remains of a pleurisy, has its resisting power diminished.* That being so, our refusal to admit that pleurisy is invariably tubercular is no reason for relaxing our care during convalescence.

That Osler finds better results among hospital patients than in private practice (*v.* p. 16) is certainly curious. I doubt whether he would find it so in England, or at any rate in London.

But if we allow that pleural effusion occurs which is not tubercular, where are we to look for its cause? We cannot in the light of modern science accept cold alone as a satisfactory explanation. On the other hand, I do not see that it is necessary to assume with Lancereaux (*v.* p. 7) that pleurisy can occur as a special disease with a specific cause of its own.

Rheumatism, although its ultimate cause is still a matter of theory, we know follows after exposure to cold and wet. It manifests itself typically by serous effusions into synovial cavities; it is frequently accompanied by effusions into serous cavities; the severity of its manifestations vary enormously in degree. In Quain's Dictionary of Medicine ([48]) we are told that "Graves has described a completely latent form of acute rheumatism in which articular symptoms are entirely wanting, whilst the other symptoms may be of the usual character and follow the usual course"; while Goodhart ([49]) writes that, in children at any rate, "just as a pericarditis may be the only indication of rheumatism, so also may pleurisy or pleuro-pneumonia." It would seem, then, that the suggestion already mentioned that the so-called idiopathic pleural effusions are of a rheumatic nature, the joint affection being at a minimum and so escaping notice altogether, while the pleural affection is at a maximum, is well worthy of further consideration. The very

* Out of the ten hitherto lasting recoveries among my cases, traces of the former attack were still to be found on careful examination in seven.

fact that salicylates have been found to have such a marked effect in the treatment of these cases certainly lends support to this view (50). If it should be the correct one, Dr. Bristow's inclusion of rheumatic with "idiopathic" pleurisies is well-founded, but we should then rather be inclined to drop the term "idiopathic" altogether.

In Case IV. (*v. supra*) is it not possible that the mitral murmur now present may have had its origin in what was really a pleuritic manifestation of rheumatism?

That the subject is an important one there can be no doubt; for if it were ever to be considered proved that all those cases which we are in the habit of looking on as "idiopathic" are in reality tubercular, it would not only render the prognosis more grave, but might also make it necessary to seriously reconsider our treatment. The question would arise whether we ought not (in view of the good results that follow the operative treatment of empyemata and sometimes of tubercular peritonitis) to take more radical measures in the direction of operation and antiseptic irrigation, in place of resting satisfied with the less formidable procedure of aspiration. If, on the contrary, many of them are of a rheumatic nature, the salicylate treatment should be more generally adopted; but in all cases the diagnosis would have to be very carefully made, and due weight given to any suspicious attendant circumstances, such as a strongly phthisical history, previous cough, marked loss of weight, and so on.

It is evident that the question is not one to be decided hastily; our present knowledge is insufficient for us to form any really reliable opinion. Perhaps we may hope that eventually some means may be found of proving or disproving with tolerable certainty the presence of the tubercle bacillus in pleuritic effusions. Meantime, we must be content to go on accumulating observations, keeping the different views and possibilities always before us.

I cannot do better than conclude with another quotation from Bowditch. "Let us be on our guard," he says, "in accepting conclusions founded upon insufficient evidence, lest we run the risk of hindering what we most desire, namely, the recovery of our patients."

I have now the pleasure of expressing my thanks to those members of the Staff at the City of London Hospital who so kindly permitted me to make use of their cases, and also to Dr. Arnold Chaplin, who rendered me much valuable assistance in connection with the old reports.

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