in most cases it was observed repeatedly, and in 1 it was almost continuously present for 3 months; in 14 cases there was hæmatemesis without any mention of melæna, and in 3 melæna was present without hæmatemesis, thus showing the importance of a careful and continuous inspection of the motions in every suspected case of ulceration.

Complications.—In 15 cases the ulceration of the stomach was primary and uncomplicated, while in 20 there were complications. One patient was suffering from abdominal aneurysm; 1 was hemiplegic, 2 had bronchitis, and 1 of these had pleurisy in addition; 2 were scrofulous, 7 were anæmic, and 7 were chlorotic. I believe that several of those described in the reports as anæmic were really chlorotic, but, at any rate, the statistics show that, in women, chlorosis is a very common accompaniment of ulceration. This fact must be carefully kept in view, else the pain of the ulceration may be mistaken for neuralgia of the stomach, which is so frequently met with in chlorosis, all the more as these are the very cases which are most apt to terminate fatally by perforation.

Results.—Of the 35 cases 2 only remained for a week or two in hospital, and 1 insisted on leaving on the twenty-fifth day, being "much improved." One patient was "improved" when dismissed, 7 were "much improved," and 23 were cured. Only 1 patient, a male, died, as the result of hæmorrhage from the stomach and bowels, and on *post-mortem* examination an ulcer was found at the pylorus, which had perforated a large vessel. These results correspond pretty closely with the result of Brinton's observations, which show 1 death in 20 to 30 cases, males succumbing to the disease nearly four times as often as females.

Treatment.—Having recently directed your attention to the treatment of ulceration of the stomach in connection with cases in the wards, it is only necessary now to indicate briefly the lines which were followed in connection with the cases comprised in the foregoing tables. 1. The patients were always strictly confined to bed—a point which, I have reason to believe, is not always insisted upon, as not only does the ulceration heal more readily, but the danger of profuse hæmorrhage and perforation is lessened. 2. The dieting was generally the main point in the treatment. Hard, hot, stimulating, and irritating food was rigidly excluded: soft and fluid food, in small quantities, frequently repeated, being the rule; and, in the majority of cases, unless there was some contraindication, milk, in combination with a little potash or lime water, was prescribed. In some instances peptonised meat preparations were given, especially Carnrick's beef peptonoids, to the extent of a teaspoonful every four hours. The patient was directed to suck the powder, so as to mix it thoroughly with the saliva before swallowing it. In some instances a slight modification of Debove's meals was administered. This consists of 25 grammes of Carnrick's beef peptonoids, 1 gramme of burnt magnesia, 2 of prepared chalk, and 1 of saccharated lime. The gastric juice is thus neutralised, and no peptones are formed in the stomach. It was found, however, that three of these meals per day sometimes produced diarrhoea, so that latterly three half meals were prescribed. As to the quantity of soft or fluid food given at a time, two ounces was the usual allowance at first, but the quantity was diminished if pain or vomiting resulted, and, of course, the less food that was taken the oftener was it repeated. We always adhered strictly to the rule of gradually reducing the quantity administered at one time, until it produced neither pain nor vomiting; and if even a teaspoonful of milk disagreed, all food by the mouth was suspended for a time, and the patient was fed exclusively by the rectum, although he was allowed to suck a small piece of ice occasionally. The rectal feeding usually consisted of the introduction every four hours of a zymised milk or meat suppository, and on the alternate four hours an enema, consisting of 3 drachms of Carnrick's beef peptonoids mixed with 2 or 3 ounces of warm water, was given.

A cure often resulted from dieting and rest alone, but the bowels were regulated when necessary by enemata, or the injection of a drachm of glycerine into the rectum. In a good many cases Carlsbad salts (the artificial salts by preference when constipation was present), as recommended by Ziemssen, sometimes yielded excellent results. These are given on the principle that the most essential obstacle to the formation of granulations in the floor of the ulcer is the corrosive influence of the gastric juice. The salts neutralise the acids and check fermentation; they likewise discharge the fermenting liquids into the bowel, and act as an aperient. The dose is from 1 to 4 drachms dissolved in a pint of boiling water. This is allowed to cool, and when tepid is slowly

drunk to the extent of a quarter of pint every ten minutes until it is all swallowed, and is given in the morning.

During convalescence the diet was very slowly improved, and sometimes small doses of arsenic in the shape of Schieffelin's pills (gr. $\frac{1}{8}$ to gr. $\frac{1}{4}$), thrice daily, seemed to consolidate the cure.

In some cases, when pain was a prominent feature, and especially when it persisted when the stomach was empty, opiates in small doses, or a combination of hydrocyanic acid and bismuth, were administered. When hæmorrhage occurred to any extent rectal feeding was resorted to; although the patient was allowed to suck ice; ice was applied in an ice bag to the epigastrium, and morphine was given by subcutaneous injection, and occasionally ergotine. Finally complications were dealt with on general principles, according to their nature—chlorosis, the most frequent one, being treated with Schieffelin's Bland's pills.

REMARKS

ON THE TUBERCULOUS NATURE OF THE SO-CALLED SIMPLE PLEURITIC EFFUSION.¹

By ALFRED G. BARRS, M.D., M.R.C.P.,

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THE observations I am about to make are the outcome of an inquiry in which I have been engaged from time to time during the last year and a half for the purpose of ascertaining the remoter event in cases of pleuritic effusion.

I have taken from the hospital records, for the years 1880 to 1884 inclusive, the names and addresses of all the cases of simple pleuritic effusion, purulent or non-purulent, and have ascertained by such means as were at my disposal if they were still living, and if so, in what condition, and if they were dead I have, so far as possible, ascertained the cause of death, sometimes from the medical attendant, sometimes by an inspection of the death register of the district in which the patient had lived. No case has been inquired into in which there was any reasonable doubt as to the presence of fluid in the pleural cavity, or in which the effusion could be regarded as in any sense of secondary origin in the ordinary acceptation of the term. So that we have not to consider any case of pleuritic effusion which was secondary to any recognised pulmonary lesion, such as phthisis or acute pneumonia, both of which are, in my experience, extremely rarely complicated by fluid effusions into the pleural cavity, or secondary to cardiac or renal disease. We are, in short, concerned only with those cases which appear to be spontaneous, and which are usually said to arise from exposure to cold. The undertaking has been a somewhat laborious one, and it was originally my intention to have placed the results of it before the Section of Medicine of the British Medical Association at its meeting in Leeds in August last, but my duties in connection with that meeting made it impossible for me to do so.

I may state at once that the point I have had in view throughout has been to ascertain the relation of pleuritic effusion to pulmonary phthisis or other tuberculous manifestation, and so to a tuberculous pleuritis, for I see no reason to doubt that if a pleuritic effusion is followed by phthisis, the effusion itself was in the first instance of a tuberculous origin. I cannot better define my own position than by quoting a few short remarks made in a debate on this subject by Dr. F. C. Shattuck, of the Massachusetts General Hospital, in the early part of 1888: Dr. Shattuck² then said that "about three years ago he had written a note to Strümpell's *Textbook of Medicine* to the effect that the experience of physicians in America was not like that of the Germans, who, according to Strümpell, found pleurisy due to cold only in very rare instances. He had given as his reason for the statement that in America so many patients with pleurisy entirely recovered. Since then his views had undergone a marked change. Facts

¹ Read before the Leeds and West-Riding Medico-Chirurgical Society.

² *New York Medical Journal*, July 14th, 1888, p. 43.

seemed to be pointing to the stand taken by some Continental writers that pleurisy was always due to tuberculosis. We were in the habit of associating in our minds tuberculosis and an unfavourable or fatal prognosis, because formerly only tuberculosis pulmonalis had been recognised, and then only when going on to a fatal issue. But we had learned that local tuberculosis frequently existed, and was recovered from, whether in the pleura or elsewhere. It was true that the tubercle bacillus had not always been found in the pleuritic effusion or on the surfaces of the membranes, but that was only negative evidence, and could not be regarded as of weight when it was remembered that in the most favourable cases for finding the bacilli the search was tedious; nor was our inability to account for the arrival of the germs at the pleural cavity an argument of value against the microbic origin of the disease."

Dr. Shattuck has here defined my own present position in this question with complete precision, and it is from this position that I have set out upon my inquiry. Five years ago I should have said that ordinary simple pleuritic effusion was a distinctly recoverable condition, easily treated, and of little danger to life, immediate or remote; but during the last few years I have seen in the out-patient rooms more than a few cases of phthisis in persons who had previously been in the hospital for pleuritic effusion, and who were described in the hospital returns as "well."

At this point I should like to say, but without any intention of digressing into an abstract discussion, that I never have believed, and do not believe, that "cold" is an efficient cause of inflammation in any organ or structure of the body except such as those to which it can be directly and immediately applied, the skin and parts immediately beneath, and the earliest sections of the respiratory and digestive tracts, for example; and consequently I have never been able, to my own satisfaction, to state the cause of the so-called simple pleuritic effusion. While not going so far as those—Landouzy, for instance—who say that all cases of pleuritis which are not rheumatic, pyæmic, and so on, are of necessity tuberculous in origin, I am strongly inclined to agree with Dr. Shattuck that a very large proportion of the so-called "idiopathic" or "exposure" cases of pleurisy are really of a tuberculous nature.

I have never been quite able to understand why the pleura, in accordance with views very generally held until quite recent times, should be allowed to become inflamed under circumstances which are said to be innocuous to all other serous membranes in the body. No one, I imagine, really believes that exposure to cold is an efficient cause of peritonitis, meningitis, or synovitis. While we all readily recognise a primary tuberculosis of the peritoneum, the meninges and synovial membranes, there has always seemed to be a great reluctance to admit tuberculous pleuritis as a primary cause of pleuritic effusion. Dr. Douglas Powell,³ in opening the discussion on "Chronic Tuberculous Processes in Serous Membranes," at the meeting of the British Medical Association, 1889, said: "Tubercular disease of serous membranes is so rarely primary." "Tubercular pleurisy was by itself, as shown by *post-mortem* examination, a rare disease. Was it so rarely met with clinically? It was well known that pleurisy commonly preceded phthisis, but were not such cases of pleurisy in the first instance tubercular?"

In regard to the *post-mortem* evidence in favour of a primary tuberculous pleurisy, it is clear that such evidence must of necessity be scanty. The pleura itself is not a vital organ. Pleuritic effusion very rarely nowadays is allowed to be fatal in the only way in which it can be so, by a mechanical interference with the respiratory or cardiac movements; it therefore follows that in hospitals (and it is there where such evidence must be found if it is to be found at all) necropsies upon cases of simple pleuritic effusion are, comparatively speaking, extremely rare. In the Leeds Infirmary, for instance, there was one death in each of the years 1886, 1887, 1888 from pleurisy with effusion, while in these three years no fewer than 87 cases of pleuritic effusion were under treatment. The fact is that pleurisy with or without effusion cannot of itself be fatal under ordinary circumstances. It is only by directly involving some vital organ such as the lung, or by the induction of some general infective condition, that it can prove so.

In August, 1887, there came to the out-patient room a boy, aged 15, in whom the right lung was practically solid, and who a few months later died of phthisis, as I am informed by his medical attendant. The previous history of this case was as follows. In 1884 he was in the infirmary under Dr. Eddison's care from the

10th to the 19th of June. Two months before admission he had had a pain in the right side, said to have been due to his getting wet. He is described in the clinical report as a strong, healthy looking lad, with no apparent symptom of any illness; but his pulse was 132, and he had wasted a little. There were signs of fluid at the right base from a little above the angle of the scapula downwards.

The day after admission $7\frac{1}{2}$ ounces of fluid were drawn from the right chest (there is in this case, as in many others, a striking discrepancy between the physical signs and the quantity of fluid found which is worthy of attention). Seven days later the dullness is described as diminishing, the fremitus as fair, and the cough as practically gone, and on this day he left the hospital "well." This case is a striking example of what I believe occurs in a considerable proportion of cases of pleuritic effusion—a simple pleuritic effusion ending in pulmonary phthisis at the end of three years. An example still more impressive by reason of the necropsy, but belonging to another class of cases, occurred in this hospital in 1881, the chief details of which are as follows:—

A man, aged 43, was admitted under Dr. Clifford Allbutt's care on September 9th, 1881, and died on November 7th of the same year. The record is headed by myself (I was then house-physician): "Pleuritic effusion; repeated tappings; general tuberculosis; death." Five months before admission his illness commenced, not quite suddenly, with an "ordinary cold" and some shortness of breath. Ten weeks before admission he was seen by Dr. Clifford Allbutt, and the left chest was explored, there being signs of fluid, but with a negative result. Six weeks later exploration was again had recourse to with the same result. He wasted much and got steadily worse. On admission he looked very ill and was much wasted; pulse 124, respiration 36. The whole of the left chest was fixed and absolutely soundless, on percussion from apex to base with the other ordinary signs of fluid. The heart was perhaps displaced to the right, for its impulse could not be found in its usual situation. On September 12th the left chest was tapped, and 50 ounces of serum drawn off with immediate relief to the symptoms. On September 17th the chest was again tapped, and 23 ounces of serum drawn off, and again, on September 28th, 20 ounces of fluid were drawn off. Two days later his temperature rose in the morning for the first time, and for the next month he got steadily worse. At this time the left chest was explored, but no fluid found, although the signs were just as marked as on admission. On November 3rd great dyspnoea set in and ended with his death in a few days. At the necropsy the left pleural cavity contained about 40 ounces of serum and some masses of lymph. The surface of the lung, the left side of the pericardium, the left chest wall, and the left half of the diaphragm were covered with thick and fibrous feltings. The condition of the diaphragm was most interesting. The inflammatory material had infiltrated the muscle as a distinct band, and was here and there obviously caseous. The solid parts of the effusion were everywhere of a soft cheesy nature. The left bronchial glands were enlarged and caseous. The left lung, reduced to one-quarter its natural size, was almost airless, cedematous, and spleenised. Its substance was thickly studded with miliary tubercles, but there was not the least trace of an ordinary phthisis lesion. The right lung was also thickly studded with grey tubercles, and its pleura was acutely inflamed in patches. Both kidneys were also studded with grey tubercles. (I said a moment ago that practically pleuritic effusion could only prove fatal by setting up destructed processes in the lung or by a general acute infection. I am inclined to think, in the light of this case and some others which I have seen, that it is possible for there to be such changes in the pleura itself in the way of enormous production of solid effusion and the subsequent softening of that effusion to cause death by an ordinary wasting process such as in true phthisis pulmonalis.)

In commenting upon this case I should like to direct attention to the fact that in the lungs themselves only an acute tuberculosis was found, while the signs of pleuritic effusion had been present for at least six months before death, so that there could be no reasonable doubt as to the order of events, the pleural affection being long antecedent to any lung disease. Whether the caseous enlargement of the bronchial glands upon the same side had any causal relation to the pleuritic effusion is a question which has occurred to me. Was it a secondary or infective caseation, or was it the cause of the pleuritis in the same way that chronic tuberculous peritonitis is said to originate from caseous mesenteric glands? In my *post-mortem* room experience caseous

³ JOURNAL, December 14th, 1889, p. 1317.

enlargement of the bronchial glands is perhaps the commonest of chronic tuberculous lesions in young subjects. May it not be that in some instances this is the cause of the pleuritic effusions of later life by a simple spreading of a tuberculous inflammation to the pleural surface in the immediate vicinity of the diseased gland? The occurrence of acute pleuritis of the right lung is interesting, as showing, as I think it does, the association of acute inflammatory changes in the pleura with the presence of miliary tubercle in the same way in which it occurs in the meninges.

In October last I made the *post-mortem* examination of a very instructive case which had been under Dr. Churton's care in the hospital. A woman 29 years of age, of alcoholic habit, had been admitted suffering from hepatic cirrhosis, of which she died by means of coma in a few days. She had been in the infirmary from May 21st to July 17th, 1889, suffering from left pleuritic effusion. Six or seven years previous to that date she had had "pleurisy in the left side." The pleuritic effusion in May, 1889, had had a gradual onset, and the temperature curve throughout her stay in hospital had been of a remitting character. The chest was tapped on May 27th, and 43 ounces of fluid drawn off. Signs of fluid continued, but explorations in July failed to confirm them, and she left the hospital with persisting dullness in the chest. At the *post-mortem* examination, the liver weighed 92 ounces, and presented the ordinary appearances of cirrhosis. The left pleural cavity was practically obliterated by old adhesions, but where obliteration had not taken place numerous miliary and submiliary nodules were visible, clearly of a tuberculous nature. There were similar nodules on the right pleura. At the apex of the left lung there was a small recent caseating lesion with a central cavity. In the right lung there were numerous nodules and lumps of consolidation undergoing caseous change. There were also three large patches of tuberculous ulceration in the intestine and miliary lesions in the kidneys.

I have recited these cases in order to indicate the kind of anatomical evidence there is to hand of the close association of pleuritic effusion with tuberculous changes in the body. To establish tuberculous pleuritis as a primary disease, we should, I take it, require evidence of the following nature:—

1. The presence of characteristic organisms in the products of the inflammation or in the organs affected.
2. The demonstration in non-fatal cases of lesions generally recognised as tuberculous.
3. The demonstration after death of lesions of an admittedly tuberculous character.
4. A very frequent, not to say constant, association of the pleuritis with other tuberculous disorders.
5. A certain clinical march of the cases, stamping them as tuberculous.

As to the presence of the tubercle bacillus, there is very little positive evidence forthcoming; all observers are, I believe, agreed that it is exceedingly difficult to demonstrate its presence even in the most likely cases. Its absence in any case, I need scarcely say, could not be accepted as showing the non-tuberculous nature of the disease.⁴

In cases of tuberculous peritonitis, we can, after incision of the abdomen, see the nodular condition of the peritoneum. This kind of evidence is not available in the case of pleuritic effusion under our present methods of treatment. Estlander's operation has not, so far as I know, been at present applied to simple effusions. The evidence by *post-mortem* examination I have already dealt with. The association of pleuritic effusion with phthisis—taking the commonest tubercular lesion we have, or any other tuberculous disease—I will now proceed to deal with, in the light of the cases which have been the subject of inquiry.

There were found in the hospital records for the year 1880-84 inclusive 114 cases of pleuritic effusion conforming with the limitations I have previously laid down, and proper cases for inquiry. Of these, 40 were cases of empyema and 74 were cases of "pleurisy with effusion." To take the pleurisies with effusion first: Of the 74 cases, 32 are dead, 25 are living, and 17 could not be traced—that is, there had been a death-rate in the cases which could be traced of something like 57 per cent. In the 32 fatal cases the average age of the patients was 32.5 years, the maximum being 54 and the minimum 3. As to the duration of life after the onset of the disease, there died in hospital 3, died the day after leaving hospital 1. In the remaining 28 cases the average duration of life

was 2½ years, the maximum being 5 years and the minimum 6 months. The causes of death in the 32 cases were as follows:—

Known phthisis ...	...	...	14
Probable phthisis ...	...	...	1
Hip disease ...	...	...	1
Tubercular meningitis ...	...	...	1
Acute tuberculosis ...	...	...	1
"Pleurisy" ...	...	...	3
"Dropsy" ...	...	...	2
"Hydropericardium" ...	...	...	1
Unascertained causes ...	...	...	8
Total ...	...	...	32

So that 21 cases out of 32 died of the disease "pleurisy," or some recognised tuberculous condition, mainly phthisis pulmonalis.

I have not sufficient facts before me to enable me to state with any certainty what the precise position of the destroying lesion has been in regard to the pleuritic effusion. It has not always been in that part of the lung corresponding with the affected pleura. For example, in a case at present under my care, where the onset of the disease was so acute that the case was sent to the Municipal Hospital in July, 1887, as a case of enteric fever, there was pleuritic effusion at the right base, which was tapped. Shortly afterwards he was in the infirmary under Dr. Churton's care, when the right chest still being dull on percussion, an exploratory puncture was made, but no fluid found. After a short interval he came to my out-patients, spitting blood freely and frequently. The right chest was still dull, with signs of consolidation rather than of fluid. After attending for some months he was again admitted, under Dr. Churton's care, for pleuritic effusion on the left side, which was tapped, and the other day he came back to my out-patients with a very phthisical aspect and signs of consolidation at the right apex, and also, but to a less extent, at the right base.

I have made a careful examination of the details of the fatal and non-fatal cases, with a view to ascertaining, if possible, any features which distinguished the one class from the other, but I am in the end unable to say that there is anything in the clinical manifestations which could be relied upon for that purpose. Many of the fatal cases were just as acute in their march, just as readily relieved by tapping, and appeared, on leaving the hospital, to be in quite as promising a condition locally and generally as the cases still living. There was also the same proportion, or thereabouts, of "dry tapplings" and "negative explorations" and "expectant treatments" in the one set as in the other, so that I am unable to add anything to the information which is already to hand for the purpose of estimating the duration of life in any given case, beyond the important fact that 57 per cent. of the cases which could be traced are now dead, the duration of life over the series being not greater than two years and a half. If further experience should tend to confirm this, our prognosis in ordinary cases of pleuritic effusion will have to be more guarded, and perhaps our treatment more radical; it may be by free incision and antiseptic irrigation of the cavity, instead of by simple puncture, as at present.

In regard to the non-fatal cases, I will only at this time remark that their tuberculous nature is in no sense disproved to me by their recovery. The recoverability of tuberculous lesions fortunately requires no demonstration. In an important paper dealing with this subject, Dr. Vincent Bowditch⁵ has published the results of an investigation of the after-histories of the cases of pleurisy occurring in his father's practice from 1849 to 1879. He says: "In comparing the percentage mortality from phthisis in the cases from 1870 to 1879 inclusive, with that of the cases of the first decade, 1849 to 1859, we find the percentage 24 in the former as against 43½ per cent. in the latter, and against 47 per cent. in the second decade, from 1860 to 1869 inclusive." As will presently appear from my own figures, it is most important in considering Dr. Bowditch's statistics to bear in mind that "the cases are not classified so that 'dry pleurisies,' pleurisies with serous effusion, and empyemas are put all together." My own investigation shows very conclusively that the introduction of the empyema cases will considerably reduce the percentage mortality from phthisis, and so it may be that the death-rate from phthisis in Dr. Bowditch's cases is not materially less than in the Leeds cases.

On this occasion there is not much that I need say in regard to the empyema cases. The figures I am about to give go a long way

⁴ On the question of micro-organisms in pleuritic effusions, the reader may be referred to an important discussion reported in the *Berliner klin. Wochenschrift*, May 14th, 1888.

⁵ *Medical News*, July 20th, 1889, p. 63.

towards confirming me in the view that I have for a long time held, that empyema and simple pleuritic effusion have very little in common either in their clinical march, their pathology, or their ultimate event.

Of the 40 cases of empyema, 32 cases are well, 6 are dead, and of 2 there is no return. Of the 6 fatal cases, 1 died of chronic Bright's disease (not lardaceous), 1 died of croup when 4 years old, 2 died in hospital of "exhaustion from prolonged discharge," and in 2 cases the cause of death could not be ascertained.

My own conclusions, based upon grounds of which the preceding observations form only a part, are practically those appended by Mayor⁶ to an excellent thesis on the after-event in pleuritic effusion, namely:

1. "The majority of cases of simple idiopathic pleurisy, or of pleurisy from exposure to cold, conceal or reveal a tuberculous process."
2. "A careful study of the lung in the neighbourhood of the effusion is more important in regard to the prognosis in any given case than the study of the effusion itself."
3. "The immediate prognosis in cases of pleuritic effusion is, under present conditions of treatment, almost always favourable; the remoter prognosis, on the contrary, should always be most guarded."

THE TREATMENT OF PLEURISY WITH EFFUSION.¹

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THE subject I have chosen as that upon which to offer you some remarks this afternoon is one about which a very great deal has been and is being said and written. The question of the treatment of pleural effusion has received attention from both physicians and surgeons, and may be said to be placed upon the hypothetical boundary line which separates the province of medicine from that of surgery. The affection is, indeed, sufficiently common and sufficiently serious to make it of importance that every medical man, be he physician or be he surgeon, should have a definite idea as to the line of treatment to be adopted under the various circumstances in which pleural effusion occurs.

I do not propose to bring before you any exhaustive treatise upon this subject. This would be, in my opinion, undesirable even if possible. I shall rather confine my attention almost exclusively to my own experience, which a connection with a special chest hospital for many years has rendered somewhat extensive. I must warn you not to expect anything very new upon this, which may be called a well-worn subject.

Effusions into the pleural cavity, even in large amounts, are not by any means always diagnosed. First, because the physical signs of such effusions do not present themselves in orthodox form, supposing the chest is examined carefully; secondly, because it not infrequently happens that patients suffering from pleural effusion are so little inconvenienced, that they pursue their usual avocations, and consult a medical man for symptoms, it may be, of dyspepsia, which do not suggest an examination of the chest; and thirdly, because pleural effusion is very often secondary to some other affection within the chest, as, for example, to tubercle of the lung, cardiac disease, or aneurysm of the aorta, to which one's attention is more particularly directed.

Thus it happens that any increase in the dyspnoea, pain, or other symptom which the original affection had produced is often put down to an increase in the affection itself, and not to its real cause, namely, an intercurrent pleurisy. There can be no doubt in the minds of those accustomed to see many necropsies, that undiagnosed, and therefore unrelieved, pleural effusion is answerable for the deaths of many affected with chronic diseases. Such patients would, of course, have died from the primary affection after a varying time, but unless pleural effusion had happened, not just then. I may illustrate this by a reference to a *post-mortem* register nearest to hand. In a record of about seventy necropsies at the City of London Hospital for Diseases of the Chest, Victoria Park, I find that in nineteen the pleura was more or less affected; in thirteen cases, the fluid in one or both cavities

was very excessive; and in four cases, the record says that the pleural cavity was full of fluid. Certainly one would imagine that death might have been caused in four out of seventy cases, at the very least, by the intercurrent pleural affection, and not by the original disease. One other point with regard to the diagnosis of pleural effusion; it is, of course, best in all cases of doubt to insert the needle of a small syringe, and to withdraw a specimen of the fluid, if fluid there be, but sufficient stress has not been laid upon the necessity of employing a sufficiently long needle, nearly twice the length of that supplied with the ordinary morphine syringe, as without it, in cases of very thick-walled sacs, you may fail altogether in your attempt to reach the interior. I have quite lately seen a case in which one or two preliminary trials with an ordinary short needle failed to withdraw pus from a chest until a very long needle, about 5 or 6 inches, having been employed, the pus was reached.

Having arrived at the certain diagnosis of fluid within the pleural cavity, the question naturally arises: What is best to be done under the circumstances of the case? 1. Are we to leave the fluid to be absorbed by Nature's unaided efforts? This used to be the routine practice in a majority of cases.² It is certainly a fact that in some cases a pleural effusion disappears in a comparatively short time if left to itself, even if we regard as apocryphal those cases in which it has been stated that the largest effusions have been absorbed in a day or so. We have all, no doubt, watched cases in which absorption is taking place, noting the shrinkage of the area of fluid from day to day. It is by no means easy, it must be confessed, to understand how the fluid is absorbed—whether by the blood vessels or by lymphatics, or by both—more especially when we recollect that the surfaces of the pleura are usually covered by thick layers of fibrin, and that the blood and lymphatic vessels, thickened as they must be by this deposit, are in the condition most unfavourable for such a process. In the cases in which early absorption takes place one is tempted to imagine either that the fibrin has not been deposited upon the pleural surfaces or that the inflammation of the pleura is local and not general. It is believed by some that in many cases at any rate the upper part of the serous membrane is first inflamed; perhaps in some cases, therefore, the inflammation is localised to its primary seat. But, granting that effusions are sometimes absorbed by Nature's efforts alone, are we wait for this absorption, are we to try and help the absorption by drugs or other means, or are we to remove the fluid at once by operation? The practical summary of such questions, then, is, How long shall we leave an effusion in the chest before tapping it?

I think you will agree that it is quite impossible to lay down any arbitrary rule as to how long we are to leave an effusion before tapping it. My own plan of proceeding is as follows: If the preliminary aspiration with the small syringe has demonstrated the presence of pus, I do not delay the operation, if I can possibly help it, a single day; or again, if the physical signs reveal the presence of a large effusion of whatever nature, I recommend immediate operation. I think a patient with one side of his chest full of fluid is in considerable danger of death, and especially of death from syncope. With such a belief it behoves one to be careful not to delay the immediate and certain relief afforded by paracentesis thoracis.

If the fluid be not pus, and be moderate in amount, immediate operation is not necessary, and it is permissible to wait awhile and watch the case; in fact, to adopt the expectant treatment. As regards the use of drugs and other methods supposed to aid the absorption of the fluid, I confess I have but little faith in them. Diuretic, diaphoretic, and purgative drugs, for example, except in so much as they aid in maintaining the bodily functions in health, are, in my opinion, of little use. The exhibition of salts, the restriction of fluids in the diet, the strapping the side with mercury plaisters, and the like, although theoretically correct, have not proved, in my experience, of much practical value. I am inclined, however, to make an exception in favour of blisters and of the preparation of iodine externally, and of iodide of potassium internally. I think—I say I think—that I have seen good come of the use of these remedies in certain cases. But, at any rate, good and even generous diet—wine, cod-liver oil, maltine, preparations of iron—ought in all cases to be given, with the great object in view of improving the general health; with the same object, the patient should be allowed to get up part of the day, and, if the weather be

⁶ *De l'Avenir des Pleuritisques*. Thèse par Alois Mayor. Paris: 1887.

¹ Read at a meeting of the West Kent District of the South-Eastern Branch of the British Medical Association.

² From the table annexed I find, however, that operation of some kind was done in 24 out of 38 cases.