A CLINICAL LECTURE ON POLYORROMENITIS, OR COMBINED SEROUS INFLAMMATIONS.

BY FREDERICK TAYLOR, M.D., F.R.C.P.,
Senior Physician to Guy's Hospital.

CASE.

ADA E. S., aged 39, was admitted to Guy's Hospital on February 4th, 1900, for pain and swelling in the abdomen.

History.

She had had a slight attack of rheumatism twelve or thirteen years before, influenza eight years before, and again last Christmas. She had been married twelve years ago, and had had no children. She said on admission that she often suffered in the morning from retching, and brought up a little watery fluid, and had no appetite for breakfast. She had cramps at night, and suffered from flatulence and palpitation; she often lost her voice suddenly, and did not regain it for two or three days. She commonly drank beer during the day and spirits at night, and usually took a little alcohol to relieve the pain at the beginning of each menstrual period. For two or three weeks previous to admission she had a cough, with dark sputum, and lately a little blood was brought up. She also had a sore throat. A week ago (January 28th) she first noticed pain in the abdomen above the pubes on both sides, and extending thence all over the abdomen and round to the lumbar regions. On January 31st the abdomen swelled, so that on February 2nd she was unable to continue her work.

Condition on Admission.

She was well nourished, dark, wanting in colour, but without puffiness of the face or oedema of the legs. The abdomen was full, tense, bulging in the flanks, slightly tender, especially in the epigastrium. There was dulness in the flanks and above the pubes, changing with the position of the patient, but no fluctuation or thrill. The liver and spleen could not be felt, nor any tumour. The impulse of the heart could not be felt, the dulness covered the third costal cartilage, and a soft systolic murmur was heard at the apex and at the base. A few rales were heard at the left base. She expectorated a little frothy mucus. The urine showed a trace of albumen, and had a specific gravity of 1012.

Progress of Case.

The temperature during the next few days was 101.8°, the pulse 104, and the respirations 32. She complained of pain in the abdomen and in the loins. On February 9th a blood count gave red corpuscles 3,500,000, leucocytes 8,500, hæmoglobin 35 per cent. On February 11th a few fine rales were heard at the left apex, and on February 19th the left base behind was dull as high as the scapular spine, with diminished breath sounds, diminished vocal resonance, and absence of tactile vocal fremitus. Dulness was then noticed in front, and on February 23rd the left side of the chest was aspirated, and 76 ounces of a slightly reddish serum of specific gravity 1018 were drawn off. The condition of the abdomen remained very much the same, slightly increasing in bulk before the aspiration, and again diminishing.

On March 18th she was again aspirated, and 73 ounces of greenish-yellow fluid, of specific gravity 1020, were withdrawn. On March 19th the left side of the chest was resonant in its upper half, dull in the lower half. The breath sounds were rather faint at the base; there was no bronchial breathing, no egophony; tactile vocal fremitus was felt nearly to the base.

Temperature.

During the whole of this period of seven weeks from the date of admission the temperature has been oscillating above the normal level. The first week it ranged from 100° to 102°, the second week between 99° and 102°; in the third week the morning temperature fell on three occasions to 98°, the evening rising to 101° or 101.4°, and for the remaining four weeks it has been chiefly between 99° and 101°, with a morning drop to normal only on four separate occasions.

The patient whose case I have read above first, as you will see, came under us as a case of ascites, and the point to be determined was the nature of the origin of the peritoneal effusion. I cannot here enter into a full discussion of the causes of ascites and their differential diagnosis, but I will merely say that in the present instance the choice seemed to lie between tuberculous peritonitis and cirrhosis of the liver. In favour of the former was the fact that the temperature was raised; but, on the other hand, this was not strongly against cirrhosis of the liver, because it is well known that in cases of cirrhosis of the liver a long-continued fever, with daily oscillations of some extent, has occasionally been observed.

In favour also of cirrhosis were a few points in her history, as, for instance, her admission that she did take spirits on different occasions, and her occasional morning sickness, and inability to eat a good breakfast; but the general appearance of the patient did not support this. We were in this position, when it was discovered that she had some dulness at the base of the left chest.

Dulness at the base of the lung may indeed be the result of pressure upwards of peritoneal fluid, so as to force the diaphragm upwards and compress or displace the lower part of

the lung. But, on the one hand, such a procedure is much less likely to occur on the left side than the right, on account of the presence of the stomach in the former case; and, on the other hand, the amount of fluid in the abdomen was not sufficient—that is, the pressure of fluid in the abdomen was not enough to have explained such an occurrence, even if it had been on the right side.

When after a few days the dulness increased in an upward direction, it became obvious, or at least highly probable, that we had to deal with a liquid effusion into the pleura as well as into the peritoneum. Thereupon the diagnosis of tubercle became more probable, and that of cirrhosis was out of count as explanatory of the whole case, though it was conceivable that an ascites from cirrhosis might coexist with an independent pleural effusion arising in some other way.

There is, of course, yet another disease which would account for the simultaneous existence of pleural with peritoneal effusion, and that is heart disease causing passive effusions into both these cavities. In support of this we have a mention of a soft systolic murmur at both base and apex of the heart, and of the upward extension of the cardiac dulness. But the latter could be explained by displacement upwards as a result of the abdominal effusion, and there was no confirmatory evidence such as would be supplied by irregular and very rapid action of the heart, or by oedema of the legs and feet, or by cardiac distress, that the valvular apparatus of the heart was actually upset. It might indeed have been considered an objection to the view of its cardiac origin that the pleural effusion was unilateral, whereas heart disease commonly produces bilateral effects; but so far as the pleural cavities are concerned, the last formula does not hold good invariably, and a one-sided pleural effusion may certainly occur in connection with a valvular lesion.

Setting aside as still more deficient in supporting evidence, the possibility of a mediastinal tumour obstructing the veins in such a way as to produce this double passive effusion, we are thrown back upon a combination of inflammatory effusions of which, in a chronic or subacute form, tubercle is undoubtedly the most common cause.

This frequent association of the serous cavities in a common lesion is a fact which has long attracted attention, not only in the case of combined tuberculous peritonitis and pleurisy, but also in the case of some more acute diseases, such as acute rheumatism—which often invades the pericardium and pleura simultaneously—and pyæmia, which may attack every one of the four great serous sacs at the same time.

An interpolated note in the Italian edition of my *Manual of the Practice of Medicine*, which is now in course of publication, calls attention to this association, which has so far impressed Italian physicians with its clinical and pathological significance that they have given a name or names to this multiple inflammation of the serous cavities, and that several articles have been written upon this combined lesion, in which its causes have been classified, its significance indicated, and its course and terminations described.¹ The names are polyserositis and polyorrromenitis, of which the latter is to be preferred because it is derived from Greek words alone, and not from a Greek word and a Latin word, as polyserositis is. The word *ôppôs* in Greek means serum.

It is clear that we must guard ourselves from regarding this conception as a separate disease, and from thinking that we have got to the bottom of our case when we have given it this imposing name. It obviously only represents a common grouping of pathological conditions of sufficient frequency to require attention, and sufficiently constant in its pathological or etiological antecedents to make its study fruitful. We may compare the name with the name polyarthritis, by which we mean multiple inflammation of joints. Such polyarthritis occurs as a part of acute rheumatism and of osteoarthritis; it may be a result of pyæmia and of syphilis. When we meet with a polyarthritis we have to consider what are the diseases, what are the bacteria, or what the disease poisons which are known to produce it.

Similarly, when we meet with a polyorrromenitis we have to ask, what are the diseases, the bacteria, or the disease poisons which will cause it?

Polyorrromenitis may be acute or subacute or chronic, and the causes of these varieties are likely to be different. One cause of an acute simultaneous inflammation of the serous

cavities is the pneumococcus. It is a common experience to find that pericarditis occurs in the course of—and, as a rule, with a somewhat later onset than—croupous pneumonia; and as such croupous pneumonia is almost invariably accompanied by pleurisy, we have, then, two membranes, pericardium and pleura, inflamed together, and by a common cause. Similarly, pericarditis frequently complicates empyema, especially when the empyema is on the left side.

Another cause of a polyorrhomenitis is streptococcal or staphylococcal invasion. We see this in pyæmia and septicæmia. In the old days, when pyæmia was a common event in our surgical wards, and not infrequently imported accidentally, from the insignificance of the wound or lesion causing it, into the medical wards, we were constantly seeing cases of double purulent or sero-purulent pleurisy, with purulent pericarditis, and occasionally purulent peritonitis.

The following case, which died in the clinical ward only three weeks ago, is an instance of acute polyorrhomenitis involving all four serous cavities, and due, no doubt, to pyogenic organisms. The preceding erysipelas, a streptococcal disease, is of interest in this case:

An errand boy, aged 15, had an attack of rheumatism two years ago, another attack in the following winter, and a third attack in October, 1899. After this he had shortness of breath and swelling of the feet, and for two months before admission he was in bed with swelling of the abdomen, jaundice, wasting, and an erysipelas of the face, which cleared up in a few days. He was admitted on February 15th, having suffered for a week with diarrhoea and pains on the left side. On February 13th he had orthopnea, severe pain in the left side of the chest, swelling of the right hand and forearm, and a purpuric rash on the lower part of the chest and abdomen, and on the left wrist and forearm.

On admission, he was wasted, jaundiced, and very prostrate. The right forearm and hand, the upper arm and right side of the chest wall, and both legs and feet were cedematous; blotchy cutaneous hæmorrhages on the posterior surface of the left wrist, hand, and smaller petechiæ scattered over the lower part of the chest; a sore over the right olecranon process and a dark scab on the left. He was too ill to examine exhaustively, but he was found to have a feeble and diffused cardiac impulse, best felt 1 inch outside the nipple line, and a soft systolic murmur at the apex accompanying feeble cardiac sounds. A pleuritic rub was felt and heard at the lower part of the left chest in front and at the side, and some *rûles* at the left base. The abdomen was distended, tense, with conspicuous superficial veins, a thin smooth, shiny skin, and a somewhat protruding umbilicus. It was resonant in front, dull in the flanks, and a thrill could be obtained. The liver projected an inch and a half below the costal margin, and a peritoneal rub was felt on its anterior surface. The bowels were loose, and motions were passed in the bed. Pulse 120, respirations 32, temperature 98°. The urine contained bile pigment, but no albumen. He rapidly got worse, and died before 1 A.M. on the 17th.

Necropsy.

Recent pleurisy, with 40 ounces of fluid on the right side, and 20 on the left. Acute hæmorrhagic pericarditis, with 20 ounces of fluid. Recent vegetations on the aortic, tricuspid, and mitral valves. Acute peritonitis, with lymph over the surfaces of the liver and intestines, and 50 ounces of turbid reddish serum. Kidneys, stomach, and duodenum congested. The solid organs much congested. The liver weighed 1,140 grams; the spleen was small, weighing 82 grams. Posterior mediastinal tissues cedematous; anterior mediastinal glands enlarged and showing irregular caseous deposits, and glandular tissue stained with bile.

Then, as a cause of the acute form of the disease, we have rheumatic fever—or, we will say, the rheumatic poison or rheumatic germ—which, while it causes a polyarthrititis as its characteristic feature, causes in many cases a polyorrhomenitis—that is, simultaneous inflammation of the pericardium and the pleura.

In a communication to a provincial medical society, published afterwards in the *Lancet*,² I described cases of combined pleural and pericardial adhesions, which are often very obscure in regard to their symptoms and physical signs. Such cases include some of the cases of so-called mediastinitis, when the pleural or pericardial inflammation has extended to the connective tissue of the centre of the chest. But whether or not this last be the case, these cases are, of course, instances, of the later results of acute polyorrhomenitis, often having a rheumatic origin.

We must also mention the tubercle bacillus as a cause of acute polyorrhomenitis in a small number of cases. Though, as a rule, tuberculosis of the pleura and pericardium gives rise to somewhat chronic lesions, yet cases of acute inflammation not infrequently arise; and some cases of acute peritonitis in young people, which may look clinically like peritonitis from appendicitis, or even like intestinal obstruction, may prove on operation or after death, to be set up by tubercle. Only a few weeks ago a young woman died in the clinical ward under these circumstances.

But, after all, tubercle plays its most important part in rela-

tion to chronic and subacute inflammations of the serous membranes, and it is by far the most frequent cause of them. I have no doubt that many of you may have already seen such cases, and that a diligent search through our clinical or *post-mortem* records would readily supply us with a long list of them. Our Italian friends have submitted such cases in large numbers to analysis, and the following are some of the conclusions they have arrived at with regard to etiology, course, and development.

Polyorrhomenitis is more frequent in males than females, and the majority of the cases occur between the ages of 16 and 30. It is usual for one serous cavity to be invaded before the others, and in most instances it is the peritoneum which is first attacked. Here I may observe that the Italians are prepared to give this name, implying multiple inflammation, even to cases where only one serous membrane is involved, I suppose on the view that even in such a case the causative agent was the same, and secondary inflammations might just as well have arisen, as in undoubted cases of multiple serous inflammation.

In any case, the disease generally begins in one serous membrane and subsequently invades the others, and this invasion occurs according to the following three types, of which the first-mentioned is the most frequent, and the last-mentioned the least:

1. The peritoneum is first attacked, then the pleuræ, beginning generally with the right pleura.
2. The pleuræ, then the peritoneum.
3. The pleura of one side is first invaded, and then the opposite.
4. Pericarditis generally follows a pleurisy, and especially a left-sided pleurisy.
5. The inflammation may first involve one pleura, then the peritoneum, then the other pleura.

The interval between the invasion of the different sacs may be a few weeks, as in the case of our patient, or some months. The duration, too, is very variable, and the result may be recovery with adhesions, or it may be a fatal termination in phthisis. Attention is specially called to mild cases in children, involving perhaps the peritoneum alone, which cases often recover in a couple of months. The case I had a few years ago of a delicate, ill-fed boy, who had a mild fever with a moderate degree of ascites, who came into the hospital and went out well in eight or ten weeks, would no doubt be placed under this head. I was content to call it tuberculous peritonitis, and as already explained I should even now have scruples about classing it with polyorrhomenitis, because I have no evidence that more than one serous membrane was involved. The boy is now well, and has had no further developments in the direction of tubercle or serous inflammation. Many years ago, at the Evelina Hospital, a particularly healthy-looking and robust young nurse rapidly developed a pleural effusion in the left side. After two or three weeks her abdomen swelled, and all the evidences of a moderate ascites were present. She was warded in the hospital, and in a relatively short time both effusions cleared up, and she went away to the country apparently quite well.

I have no proof that this case was tuberculous; but what I wish to impress upon you is that the position taken up by some of those who have written upon this subject, and strongly maintained by Picchini,³ is that subacute and chronic polyorrhomenitis is practically always tuberculous. In the present case we have failed to obtain evidence of bacteriological research in favour of this. She has not provided any sputum for the discovery of bacilli, and, though the fluid from the pleura has been on each occasion inoculated into animals, the animals have died rather unaccountably a few days afterwards without presenting any tuberculous lesions. However, the strength of our belief of its tuberculous nature here lies in the constant fever, in the simultaneous effusions in the two cavities, and the impossibility of accounting for them by any cardiac, renal, or hepatic lesion.

While assenting to the general proposition that such cases are as a rule tuberculous, I am not prepared to exclude the possibility of an exception, bearing in mind that such a case as that I have already referred to,⁴ where dense and thick adhesions of the pleuræ, pericardium, and peritoneum were found after death, constituting, therefore, a polyorrhomenitis, or the results of one, without any single fact *post mortem* to

show that it was tuberculous, and nothing in the history to show that it had an acute origin.

The prognosis of polyorromenitis in general must depend very much on the cause. The prognosis of pneumococcal polyorromenitis is no doubt very much worse than the prognosis of a pneumococcal pneumonia, or a pneumococcal empyema or pleurisy; that is, the implication of the pericardium is a very unfavourable addition to the case, whether this be from the direct lesion of the heart itself, or from the increased virulence of the organism or organisms which might seem to be implied by its extension to another cavity.

Streptococcal and staphylococcal polyorromenitis is undoubtedly often fatal, and the case of the boy I have recorded just now may be taken as an instance of this. Rheumatic polyorromenitis is of much less serious prognosis. It is true that of the relatively small percentage of deaths in acute rheumatism, a good proportion are in cases where pericarditis and pleurisy coexist; but, on the other hand, of cases of the double inflammation a large proportion recover. But we must remember that it is those cases which may subsequently give rise to serious symptoms either in the form of adherent pericardium, or of combined adhesions of pleura and pericardium, with or without the addition of a mediastinitis.

Of the cases of subacute or chronic multiple inflammations which are attributed to tubercle the prognosis is no doubt more favourable. We know that ordinary uncomplicated tuberculous peritonitis constantly gets well; and the pleurisy which have been until recently regarded as idiopathic or catarrhal, and which are now by many regarded as tuberculous in all cases, also recover completely. So also we see recovery in cases where the serous cavities are associated in a common suffering. The case of the nurse is an instance, and there is at least a possibility that the case I have just described here may leave the hospital practically convalescent. This is not always the case; indeed, I should have thought the fatal cases would be in the majority.

Tuberculous peritonitis is probably fatal in more than half the cases; pleurisy is directly fatal in much less; but the associated lesion is not likely to be less fatal than the more fatal of the two operating alone. The addition of a tuberculous pericarditis probably renders death nearly certain.

But supposing recovery from the immediately inflammatory lesions, there remains the risk from permanent adhesions, whether in pleura, peritoneum, or possibly pericardium; and the risk of the development of tubercle in internal organs, especially the lungs or meninges, in the forms of acute miliary tuberculosis, phthisis, and acute tuberculous meningitis.

The figures given by Picchini are distinctly more favourable than I have here suggested. Out of his 50 cases, he states that there were 22 deaths, 7 cases improved, and 21 cures.

The treatment of the condition of polyorromenitis must be conducted on the lines of the treatment of the separate tuberculous lesions concerned. In suitable cases you would have recourse to perfect conditions of hygiene, to good food, and pure air with all the advantages that are so often seen to accrue from residence either at high levels, or at a bracing sea-coast. For our hospital patients there is no doubt that the residence in a well-ventilated, capacious, and airy ward, with regular and good food, and absence from all work and household cares, often constitutes such an improvement in the surroundings of the patient as to lead at once to a diminution in the activity of the tuberculous process. This at least we see constantly in the case of phthisical patients no less than in cases of tuberculous peritonitis.

Internally as drugs we have administered cod-liver oil, creosote, and iron, and many recommend no other drugs likely to antagonise the pernicious effect of the tubercle bacillus, arsenic, iodine, and mercury. As local measures we have aspirated the left chest, and have applied mercurial ointment to the surface of the abdomen. The former measure was justified partly as a means of diagnosis, partly as a therapeutic measure in view of the large quantity which seemed and was proved to be present. Mercurial inunctions in the treatment of tuberculous peritonitis have often seemed to be valuable, and their use has often been followed by practical cure, that is, by the subsidence of all the symptoms, and the return of the patient, often a child, to the ordinary

conditions of healthy life, though it must be allowed that peritoneal adhesions are very probably left behind in many cases. Laparotomy is a method of treatment which I will not now discuss, but which may become desirable, in the event of our present treatment not proving adequate.

P.S.—The tuberculous nature of the lesion was demonstrated by a laparotomy performed on August 7th. For some weeks previous to this a rather ill-defined area of solid resistance had been recognised in the abdomen below and to the right of the umbilicus. The abdomen was opened by a vertical incision. Between four and five pints of clear, yellow serum were discharged. Tubercles in great numbers were found in the peritoneum, and the subperitoneal tissue was greatly thickened. The patient died on November 10th. The lungs were found to be adherent to the parietes; at both apices were deposits of healed tubercle with surrounding emphysema and throughout both lungs were numerous tubercles of firm consistence like small glass beads with no sign of caseation. The stomach and intestines were matted together and completely adherent to the parietes by old fibrous adhesions, and masses of caseating tubercle lay in among them. The peritoneum was much thickened throughout. The intestines were in many places constricted by the adhesions, and there were no tuberculous ulcers. The liver and spleen had thickened capsules and were adherent to adjacent parts. There was no fluid in either pleural or peritoneal cavity.

REFERENCES.

- ¹ Corazza, Concato, Galvagni, Picchini, and others. ² *Lancet*, October 29th, 1898. ³ La Poliurromenite subacuta e lenta, in *Il Morgagni*, 1891. ⁴ *Lancet*, loc. cit.

THE HARVEIAN LECTURES

ON

PROGNOSIS AND TREATMENT IN PULMONARY TUBERCULOSIS.

Delivered before the Harveian Society of London, November, 1900.

By ROBERT MAGUIRE, M.D., F.R.C.P. LOND.,

Physician to the Brompton Hospital for Consumption and Diseases of the Chest; Physician to Out-patients, St. Mary's Hospital, Paddington.

[ABSTRACT.]

LECTURE III.

IN treatment we must still consider the pathology of the condition under discussion, and think of how we can:

- (a) Increase the normal resistance of the tissues to the invasion of the bacillus and its congeners;
- (b) Hinder the action of the bacillus by destroying its congeners, the staphylococcus pyogenes, and the pneumococcus;
- (c) Hinder the action of the bacillus tuberculosis by interfering somewhat with its own vitality; and
- (d) Attempt to kill the bacillus tuberculosis *in situ*.

(a) Increased resistance is obtained by augmenting the vital powers of the body by overfeeding, fresh air, and adjuvant measures. This has been done for many years and with much good effect. Nursing discipline is an important item in such treatment. The open-air treatment is of great value, but it is very necessary that it should be practised with more care and common sense than has hitherto been adopted. Individual cases require individual treatment, and not every case ought to be subjected to the routine of a sanatorium. The digestive powers and the capability of resisting cold require special attention, and without it much harm may be done.

Drugs are of use in the maintenance of general body health. Arsenic is very valuable, and in the form of sodium cacodylate I have used it by the mouth and also subcutaneously. In the latter form it is sometimes more convenient than the older preparation, and thus lends itself to the administration of a somewhat larger dose. Otherwise the cacodylate acts merely as a body stimulant, and is certainly not a cure for tuberculosis of the lungs.

Dr. Habershon has used glycogen subcutaneously, with the object of stimulating the phagocytic properties of the leucocytes. His observations are not yet completed. De Backer