A Clinical Lecture

ON

MULTIPLE SEROSITIS,

ILLUSTRATED BY

A CASE IN WHICH THE ABDOMEN WAS TAPPED SEVENTY TIMES.

DELIVERED AT GUY'S HOSPITAL,

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GENTLEMEN,—The patient about whom I am going to speak to-day was in Mary Ward some time ago. Her history is as follows:

I first saw her on March 25th, 1905, with an old student of this hospital, Dr. Horace Richardson. She was then 31 years of age, and had been married six years. She was quite sure she had never had any rheumatism. Three years before we saw her she had a six months premature confinement. Shortly after she noticed that her legs were swollen, and one month after this swelling of the abdomen was observed. Five months later, namely, in September, 1902, the abdomen was first tapped. From this time to when I first saw her in March, 1905, she had been partly under Dr. Richardson and partly in various hospitals in different parts of England. Diuretics had been given, mercury had been rubbed into the abdominal wall, but treatment had not prevented the collection of the abdominal fluid and the necessity of its withdrawal. When I saw her in March, 1905, she had been tapped thirty times, and the amount drawn off at each tapping had been from 28 to 36 pints. By March, 1905, it was necessary to withdraw the fluid every two or three weeks; the interval between the first and second tapping was eight months, and the intervals between each subsequent tapping had been gradually shortening until when I saw her on March 25th, 1905, she had been tapped seven days previously, and yet a good deal of fluid had re-collected. The liver could be felt to be smooth and quite firm; she had very considerable oedema of both lower extremities. This had been previously relieved by Spathey's tubes. There was no dilatation of the superficial veins, nor could we detect any disease of heart or lungs. She was admitted into Guy's Hospital three days after I saw her. Some of you may remember her; she was in No. 12 bed in Mary Ward from March 28th, 1905, to September 4th of that year. Soon after admission it became necessary to tap her again, and then 13 pints of fluid were withdrawn from the abdomen. In all, during her stay in the hospital, she was tapped ten times; the fluid always contained albumen; it was clear and pale yellow in colour, and it clotted readily. When the abdomen had been emptied we could feel the liver apparently enlarged to about three fingerbreadths below the ribs. The apparent enlargement was uniform, the organ was firm, and the edges thick and rounded. At times, if you listened over the liver you could hear a rub. She was always somewhat anaemic, but the diminution of the haemoglobin was equal to that of the red cells; the anaemia was of the secondary variety. Except for the fact that there was some fluid in the lower part of each pleural cavity, the lungs during her stay in the hospital were healthy, and so was the heart. There was no evidence of pericardial effusion or of an adherent pericardium. At no time during her stay here was the urine found to contain either albumen or sugar. We tried many diuretics, but they had no effect in checking the reaccumulation of the fluid in the abdomen. The diagnosis made was that she was probably an example of the condition called multiple serositis, or, in other words, that she had chronic peritonitis, perihepatitis, and chronic pleurisy, and we thought the vena cava was probably compressed by thickening of the peritoneum below the origin of the renal veins, because we had to explain the oedema of the feet although she had no albumen in her urine. When she left the hospital in September she again passed into the charge of Dr. Horace Richardson, and he wrote to me the other day, in answer to my letter of inquiry, saying that she died on March 5th, 1906. After she left here many more tapplings of the peritoneal cavity were necessary, and it was reckoned that by the time her death took place she had been tapped in the abdomen nearly seventy times. The total fluid withdrawn was nearly two thousand pints. Dr. Richardson says that at the end the secretion of urine was very scanty.

Here we have a very remarkable case, and I think we shall be profitably employing our time this afternoon if we discuss it. It is obvious that in order to try to make out what could have been the matter with this woman we must consider what are the different causes of chronic peritoneal effusion, because she had it to such a degree that she secreted 2,000 pints of fluid in her peritoneal cavity. She might have cirrhosis of the liver.

Ascites occurs in about 50 per cent. of all cases of cirrhosis of the liver, and although it is a very strong argument against her having cirrhosis of the liver that she was always a very temperate woman, yet you must not take that as proof positive against cirrhosis, for we know that cirrhosis of the liver has at least two causes. A very common one is over-indulgence in alcoholic beverages, but there is some other, at present unknown, cause acting to produce the variety of cirrhosis of the liver commonly called alcoholic. The evidence of that I can give you in a minute. In the first place, there are a certain number of people who die of cirrhosis of the liver, quite indistinguishable from drunkards' cirrhosis, and yet who have never taken alcohol to excess. There are many cases on record of children who certainly have not taken any alcohol and yet who have died of cirrhosis of the liver. There are a few cases also in which the same thing has happened to adults. Last week I made a *post-mortem* examination on a patient whom I know always to have been extremely temperate, but the cause of death was typical hob-nailed cirrhosis of the liver. Then there is the fact that cirrhosis of the liver is much commoner in England than in other parts of the world, although Englishmen are not more drunken than inhabitants of some other parts of the world. For instance, I believe a city which has the unenviable reputation of having much drunkenness is Glasgow, but cirrhosis of the liver is rare there. Sir William Gairdner wrote and told me so. It is exceptional in Aberdeen, where much whisky is consumed. It is not much seen in Germany, where beer is taken to an enormous extent. So the distribution of cirrhosis of the liver is not coextensive with the distribution of drinking alcohol to excess. Again, there seems no doubt that cirrhosis of the liver is much commoner among the poorer classes than among the well-to-do people, although many people of the middle and upper classes of society drink very considerably. Also cirrhosis is very rare in asylums and homes for inebriates; it almost appears that the liver is spared if the nervous system is permanently affected by alcoholic excess. You cannot give cirrhosis of the liver to animals by giving them excessive quantities of alcohol. So in whatever way you look at it, you must admit that so-called alcoholic cirrhosis of the liver has other causes than alcohol. There is no doubt that alcohol is a very potent cause, but mere over-indulgence in it falls to completely account for the disease. I mention these points to remind you that we must not argue that it is impossible for this woman to have cirrhosis of the liver because she was not a drunken person. It is possible she may have had cirrhosis and yet not have been a drinker, because cirrhosis of the liver has apparently two causes at least, which often operate together, but may operate singly. And alcohol is one of them. When one of the causes operates singly, it probably is one which we do not at present know. But this is not a lecture on cirrhosis of the liver, so I cannot go into the question with you this afternoon, although it is very interesting, as to why a person with cirrhosis of the liver should have ascites at all. But I might sum it up by saying that there are many very strong reasons for believing that cirrhosis of the liver is not the sole cause, by pressure on the portal vein, of the ascites that occurs in association with cirrhosis of the liver. It is much more probable that in cirrhosis of the liver the patient manufactures in some way, owing to the damage of the liver, a poison which circulates in his blood, and it is as harmful to him as the various poisons which circulate in the blood in granular kidney, and that one result of the circulation of this poison is that the patient gets ascites, and another result is swelling of the feet, and so forth. But I want you to remember that ascites associated with cirrhosis of the liver is not solely mechanically due to the pressure of the contracting fibrous tissue on the radicles of the portal vein in the liver.

Our patient, you will remember, was tapped many times, but unless there is associated with the cirrhosis some chronic peritonitis, you will never find a case of cirrhosis of the liver requiring anything like the number of tapplings that our patient required. For example, I went through some hospital cases. In 10 who had cirrhosis of the liver uncomplicated by chronic peritonitis, and who developed ascites, but the ascites was

not sufficient to need tapping from first noticing that the abdomen enlarged until the death of the patient, the average duration of the life of the patient was eight weeks. The same was true of 12 cases of uncomplicated cirrhosis of the liver which were tapped, for their average duration of life after swelling of the abdomen was first noticed was only eight weeks. In 4 out of the 12 cases death took place in less than a month after the abdomen was first noticed to be enlarged. And in only 2 of these 12 that required tapping was life prolonged beyond three months. Those figures clearly suggest to you that the onset of ascites in cirrhosis of the liver is in the majority of cases a terminal event, and means that the patient is not far off his end; so much so that it is exceptional for a patient whose ascites is due to uncomplicated cirrhosis of the liver to live long enough to require a second tapping. At the time those cases were collected they were much criticized; but some years after Dr. Campbell Thomson⁴ took 18 cases from the Middlesex Hospital records, and found that they in every respect supported the conclusions derived from the cases at Guy's Hospital—that is to say, that the supervention of ascites in uncomplicated cirrhosis means the beginning of the end. And quite recently Dr. A. Ramsbottom⁵ taking cases from the Manchester Royal Infirmary, begins his article by stating that he at first doubted the teaching that I have just given you, and so he collected his cases with the view of seeing whether it was right or wrong; therefore he approached the subject in an impartial spirit, and he concludes that in the great majority of cases of alcoholic cirrhosis of the liver in which the peritoneum was not thickened—that is to say, uncomplicated cases as shown *post-mortem*—ascites is a terminal event. This was the teaching of our forefathers; Austin Flint, Watson, Niemeyer, Fagge, Murchison, and others taught this view. That being so, it was clear that our patient probably had not uncomplicated cirrhosis of the liver, because she survived nearly 70 tapplings.

To take other causes of chronic peritoneal effusion you will suggest that she may have had renal disease, but we had her under observation six months and there was no albuminuria. You might suggest she had cardiac disease or pulmonary disease, but six months' observation failed to reveal any disease of the heart or pericardium, or to suggest any, and the only pulmonary trouble was some fluid in both pleural cavities, but not enough to cause sufficient back pressure to determine this 2,000 pints of fluid in the peritoneal cavity. When we used to discuss the case at the bedside, clerks sometimes suggested that she had malignant peritonitis; but she lived far too long for that, and hardly any case of malignant peritonitis would require seventy tapplings. But there is one case we must mention here. Perhaps some of you may have heard that a few years ago one of the celebrities of Guy's was a certain Mrs. S., whose abdomen was tapped 299 times in this hospital. She used to come in for a night and be tapped and go out next day. After the 299th tapping there was a *post-mortem* examination, fortunately for us, and it was found that the cause of this persistent collection of fluid in the abdomen was a papillomatous growth of both ovaries, with papillomatous nodules on the peritoneum. Dr. Pye-Smith recorded the case in the *Transactions of the Pathological Society*. But such a case as that, if not unique, is at all events so excessively rare that it hardly enters into a bedside discussion as to the probabilities of any case.

Some of you suggested at the bedside that this present patient might have tuberculous peritonitis as a cause of her repeated collections of fluid in the peritoneal cavity. But I have never seen any case of tuberculous peritonitis require 70 tapplings, and after she had been tapped we could never detect at the bedside any masses of tuberculous deposits in the omentum or peritoneum, such as we should have expected to find, and she never had any evidence of tubercle in any other part of her body, nor were there any general symptoms pointing to this disease, so I think we may put that on one side. We are driven, then, to believe that she had what is called simple chronic peritonitis. It is usual in medicine to call a thing simple when we do not understand it, and nobody knows what is the cause of some cases of chronic peritonitis—often called simple or idiopathic peritonitis.

I will tell you something about that condition, and try to picture to you such cases.

There are some patients who suffer from chronic peritonitis, but the peritonitis is not due to tubercle or growth, as can be shown by a most careful *post-mortem* examination. In such cases, when we look at the peritoneum in the *post-mortem* room, we find it thick and opaque. Like any chronically inflamed structure, it puckers, and therefore the great omentum is puckered up towards the colon, and lies across the abdomen like a transverse tumour, which can often be felt with the hand. In consequence of that there may be some difficulty in distinguishing it from the lower edge of the liver. In the same way the mesentery becomes puckered and shortened, so that the intestines are dragged back towards the spine, with the result that the collecting fluid flows over them and gives you a dull note at the umbilicus earlier than otherwise would be the case. Then, too, in other parts of the abdomen you may sometimes feel these thickened masses of peritoneum. And the way in which this thickened peritoneum will mat the bowels together is extraordinary. I have made more than one *post-mortem* examination in which I have been able to take out the whole of the bowels *en masse*, and they have been held together and kept open by this chronic peritonitis, and, except that the tubes were not parallel when I cut across this mass, it did not look unlike a cross-section of a tubular boiler. You will find it impossible to dissect the bowels out from such a mass. The wonder is how the intestinal contents get from one end of the bowel to the other, but they nearly always do, although, as Fagge showed, sometimes intestinal obstruction occurs. I had better now prove to you that such a condition exists. A sailor, aged 29 years, came into John Ward under my care on August 4th, 1887. He had been all over the world. Two years before admission he went to Rotherhithe Infirmary on account of what he says was rheumatic fever. During his convalescence he became dropsical. After being there seventeen months, and being frequently tapped, he went to St. Bartholomew's, where he was tapped, and where he remained two months; and shortly after leaving there he came to Guy's. Before he came into the ward he had been tapped thirty-four times. I have here a list of all the times he had been tapped. The average amount of fluid taken out at each tapping was 23 pints. He died soon after his admission to Guy's. We only tapped him once here, and six pints were taken away. The total amount of fluid withdrawn from him was 790 pints. At the *post-mortem* examination this state of affairs was found: The liver presented the usual condition of perihepatitis. It had a thick white covering, which easily peeled off; we will come back to this in a moment. There was old chronic peritonitis with matting of omentum into a thick mass; the mesentery was shortened, and there were other signs of chronic peritonitis such as I have described, but no evidence of tubercle or growth. There is a case, therefore, in which the *post-mortem* examination showed this form of chronic peritonitis, and during life the man's abdomen had to be tapped many times, although not so often as was necessary in the case of the woman the subject of this lecture.

This man I said was found at the *post-mortem* examination to have perihepatitis, and therefore this is a good opportunity to discuss that condition. Perihepatitis merely means an inflammation of the peritoneal capsule of the liver, and obviously it may be acute or chronic. The acute condition does not interest us this afternoon in connexion with our present case. It is very common; indeed one can hardly have a general peritonitis unless there is acute general perihepatitis. Sometimes there is local acute perihepatitis present over a hepatic abscess, and sometimes it is found following on a severe pleurisy of the right side, and sometimes in acute cases you can hear a rub when you listen over the liver. But usually you cannot, because the liver is so painful that every movement hurts, and the patient keeps that side still in breathing.

Next we come to the chronic perihepatitis. That is clearly either universal, in which case the whole liver is affected, or it is scattered in patches on the surface, and is then called local chronic perihepatitis. It occurs in a number of conditions which will suggest themselves to

you at once. For instance, a little here and there may be part merely of cancerous or tuberculous peritonitis. A cancerous nodule in the liver may have some local perihepatitis over it, and it is often met with round about a gall bladder which contains or has contained gall stones, so that the gall bladder becomes matted to the liver, or it may be seen when a gastric ulcer has been in contact with the liver, and patches of local perihepatitis are very commonly seen in the *post-mortem* room, when there has been much backward pressure from disease of the heart or lungs. Capsulitis of the spleen is frequently associated with this perihepatitis due to backward pressure. I do not think I need tell you more about local perihepatitis. Universal chronic perihepatitis is of much greater interest. In that condition the whole capsule of the liver becomes thick and opaque, because it is chronically inflamed. Here in this specimen from the museum is an early stage of the process; you see there is a thick layer forming all over the surface of the liver. The next specimen shows you a later stage—the whole liver has a thick white jacket, which has been compared with the ice which covers a wedding cake, and hence the condition is known in Germany as *Zuckergussleber*. This white jacket may be a $\frac{1}{4}$ in. thick. It very easily peels off the liver, and the surface of the liver underneath is smooth, and for some unexplained reason, as Fagge pointed out, the thick white jacket, by its contraction, may turn up the edge of the liver, so that its edge lies on the upper surface, bound down by the thick white jacket, and that gives to the liver a very broad rounded edge. In a case like that, when you feel the abdomen you feel this bent-up liver, and you do not feel the lower edge at all. Often you can see, on this thickened peritoneum over the liver, many little shallow pits, and I have brought this specimen to show you them. The thickened peritoneum consists of fibrous tissue which is undergoing hyaline degeneration in parts, and it is arranged in horizontal laminae. Frequently, as you would expect, there are considerable adhesions between the liver and the diaphragm, and that may explain the fact that when there is chronic inflammation of the pleura associated with this variety of chronic perihepatitis, it is usually the right pleura which is chiefly affected. The subjacent liver has often been erroneously said to be cirrhotic. That is undoubtedly incorrect. I have seen many cases and can testify to that. The specimen I last showed you is rather a rare one, because that liver is cirrhotic; but that is exceptional, as Fagge insisted. He pointed out that the liver under the thickened capsule is usually soft and fatty. It is interesting to remember that the capsule never exercises any pressure on the transverse fissure, so these patients are not jaundiced from pressure on the bile duct, and the portal vein is not compressed. The ascites which these people have is due to peritonitis which is associated with the chronic perihepatitis. The fundamental thing which you must remember is that universal chronic perihepatitis is merely part of a chronic peritonitis from which the patient is suffering. It has been exalted into a position which it hardly deserves. I can prove that to you from some cases taken from our own reports which I analysed.² I took indiscriminately 40 cases of perihepatitis, 18 were examples of partial perihepatitis, 22 were examples of the complete condition. Of the 18, 6 were examples of local perihepatitis due to tubercle or cancer. In the remaining 12, only 1 is stated to have had peritonitis, 8 are distinctly stated not to have had peritonitis. No mention is made of the peritoneum in the remaining 3. In 10 out of the 12 there was lung or heart disease which caused backward pressure. My point is that of the cases in which the perihepatitis was local and scattered about in patches, the *post-mortem* report in nearly all of them distinctly stated that there was no general peritonitis at all. They were cases of growth on tubercle with a little local perihepatitis, or examples of backward pressure from heart or lung disease, but not cases of chronic general peritonitis. Now take the 22 cases of universal perihepatitis like the specimen I pass round. In only 2 of them was it stated that there was no peritonitis. In 17 it is distinctly stated there was chronic peritonitis and in the remainder the peritoneum is not mentioned. The peritonitis was always of the chronic variety. To take one description: "The peritoneum was converted into thick tough layer, matting of the intestines together and covering them with a thick fibrous apron. The omentum was matted at its upper

part into a thick lump 2 in. broad below the stomach." Universal chronic perihepatitis is only part of a general chronic peritonitis not due to tubercle or growth. Partial perihepatitis has nothing to do with chronic peritonitis.

Often the form of chronic peritonitis which we are talking about is local in the peritoneum only. But there is a considerable tendency for serous membranes throughout the body to be affected together. Fagge used to teach that there was a group of cases in which the serous membranes throughout the body were liable to be inflamed at the same time. He did not know about pneumococci. But nowadays we should say most of the acute cases are pneumococcal infections of both the pleura and peritoneum, like the case of the woman now in 25 bed Miriam Ward. Some of both the chronic and acute cases are tuberculous. Some are streptococcal. Seven years ago Dr. Frederick Taylor³ gave a clinical lecture on these acute or subacute cases. But this afternoon our interest centres on a more chronic inflammation of the various serous membranes of the body, for wherever you see chronic peritonitis of the variety we believe to have been present in the woman the subject of this lecture, you have to remember that other serous membranes may also be affected.

This chronic affection of the various serous membranes has had many names given to it—polyorrhomenitis, multiple serositis, polyserositis, progressive serositis, and Concato's disease, after the Italian who described some cases. We have to consider whether our patient with chronic perihepatitis and chronic peritonitis was an example of this disease in which the peritoneum, pleurae, and pericardium are chronically inflamed and thickened, so that any of these serous membranes may become exactly like the thickened peritoneum which I have described to you as occurring in chronic universal peritonitis. The serous membrane may become centimetres thick; it has a dense opalescent appearance, and consists of several stratified layers. It has been inaptly compared to porcelain, and can always be peeled off the subjacent organs. No case should be regarded as an example of multiple serositis unless after careful examination neither tubercle nor growth can be discovered. To illustrate the disease, let me read you one of Dr. A. G. Nicholls's cases.¹ I have one here, that of a woman aged 48. On opening the thorax everything was found matted together, so that the thoracic and abdominal organs were removed *en masse*. The upper portions of both lungs were adherent to the wall of the chest, the lower lobes were each encased in a dense white, gristly-looking deposit, varying from 0.3 to 3 cm. thick, with firm adhesions to the diaphragm. In this deposit were large, irregular cystic spaces containing fluid. The lower parts of the lungs were also firmly adherent to the chest walls by means of this dense material. The external pericardium was converted into a hyaline-looking material of cartilaginous consistency, and of a semi-translucent appearance. The two layers of the pericardium were firmly adherent everywhere. The adhesions were so firm about the vessels that they had to be separated with a knife. The abdomen, which was enormously distended, contained 6,000 c.cm. of pale, straw-coloured fluid which clotted easily. The parietal peritoneum was markedly thickened by numerous adhesions. The two largest bands of adhesions were between the great omentum and the left ovary and the body of the uterus. The mesentery was shortened and thickened, and the intestines did not float in the contained fluid, but lay bunched up along the vertebral column. The thickening of the parietal peritoneum extended round the sides and over the lower surface of the liver. The thickened membrane had a tallowy appearance. The liver was small and the edges rounded. There were adhesions everywhere about it, and there was a great hyaline thickening of the capsule, more especially in the lower aspect. In consequence of this the gall bladder was scarcely recognizable. That is a very good account of the *post-mortem* appearances of a case in which the whole of the serous membranes of the body were affected with this chronic inflammatory disease called multiple serositis, which is not due to either tubercle or new growth. In this case no tubercle bacilli could be found anywhere.

Now, to tell you something about this chronic disease of many serous membranes. It appears that it may occur at any age. It is equally common in men and

in women. There is often a history of previous acute infectious disease, which strongly suggests that the serositis is bacterial. Out of 14 cases, in 7 the peritoneum and pericardium and both pleurae were all affected. In the remaining 7 one or more of these serous membranes, but not all, were affected. Perihepatitis is always present, and perisplenitis is nearly always present. Probably in the great majority of cases this disease begins in the peritoneum and the other serous membranes are affected later, but there appears to be a small group of cases in which the pericardium is first affected and the other serous membranes are involved later. When the pericardium is first affected it is very likely that indurative mediastinitis will be present, the connective tissue around the pericardium being thickened, so that the lungs are bound in one mass to the pericardium; the patients usually waste; the disease is virtually never associated with cirrhosis of the liver; the right pleura is more often affected than the left, which may be due to the fact that perihepatitis occurs early. The kidneys are often granular. As far as I know, the prognosis is very bad, but cases may last years. The woman who forms the subject of this lecture, you will remember, survived nearly seventy tapplings. There is a large amount of albumen in the ascitic fluid, which supports the suggestion I have made that the ascites is not due to pressure on the portal vein. The histology of the thickened membrane clearly shows that it is inflammatory; the exudate upon the surface of the serous membranes becomes organized. If you cut sections, you will see proliferation of the connective tissue of the capsules of the various organs and successive layers of fibrous tissue, the exudation having become organized. We know very little about the cause of this condition, but certainly a considerable number of the sufferers have chronic granular kidney, and it seems to bear some relation to specific fevers.

With regard to our case upon which I am lecturing, it was unfortunate that she died out of the hospital, and therefore that we had no *post-mortem* examination, but I brought her case forward because she certainly had chronic peritonitis with perihepatitis. We know that as the result of the tapping, and because we could feel the rounded liver. It may have been that she was an example of this disease as confined to the abdomen. But I would suggest to you that probably she was an example of genuine multiple serositis; for you will remember that she had fluid in both of her pleural cavities. You will notice that there is no boundary between cases of simple chronic peritonitis and those of multiple serositis, the difference depending merely upon the number of serous membranes affected, and it may well happen that a patient dies from simple chronic peritonitis, but if he had lived longer he would have become an example of multiple serositis.

The only thing which remains to be said is to try to find an explanation for the oedema of our patient's feet. If she had had associated pericardial trouble, it might have been easy to explain the oedema of the feet by backward pressure. But we had no evidence of that, nor had she albuminuria. So we suggested at the bedside, remembering the extreme degree of chronic peritonitis, that possibly the contraction of the thickened peritoneum compressed the vena cava and so led to oedema of the feet. But if it did so, the compression must have occurred below the renal veins, because there was no albuminuria.

With regard to the treatment of the condition, I am sorry to say we can do but little. Diuretics hardly seem to restrain the accumulation of the fluid, and as far as I know there is nothing which will do it. The only thing seems to be to remove the fluid frequently, as was done in this case. We never had to remove it from the pleural cavities; probably its withdrawal from the abdomen prevented a great accumulation in the chest.

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A PLEA FOR A NEGLECTED REMEDY.

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In these days of progress, when "modern methods of treatment" are so much belauded, and the home of every registered practitioner is flooded with a ceaseless stream of pamphlets acclaiming the virtues of new synthetic drugs (samples enclosed), it may be useful to spare a thought to some of the old remedies which are fast slipping out of mind and are in great danger of being forgotten. Of the new drugs, if past experience is to be trusted as a guide, few are likely to prove of permanent value. In comparatively recent times how many of such remedies have been placed with more or less flourish upon the market, but only to be laid quickly aside as disappointing if not altogether useless? Few have outlived the test of adequate trial. After a short experience of such failures one is disposed to cling more and more firmly to remedies which have stood the test of time and whose value was once universally acknowledged. Of these some have been neglected owing to our change of view with regard to the management of disease and the complete disuse of the antiphlogistic method of treatment. Fevers and inflammatory or catarrhal conditions, which used to be treated indiscriminately by measures directed to the reducing of a supposed "plethora"—by which term was understood either a general hyperaemia or a local determination of blood—are now managed upon a very different plan, and bleeding, together with the free internal use of the potassio-tartrate of antimony, has been abandoned in favour of ammonia and other drugs of similar action. But because antimony in full doses produces profound discomfort and depression it does not follow that the drug, if given in more moderate and prudent quantity, is not a remedy of the utmost value. In catarrhal states of mucous membrane antimony has never been dislodged from its eminence by later substitutes.

In cases of bronchial catarrh and bronchitis its neglect is the more to be deplored because there is no other drug of approximate value which can be given in its place—at any rate with equal advantage. On this subject—the treatment of bronchitis—I find the most erroneous views (as I think them) prevailing amongst the younger generation of medical practitioners. A young house-physician will order a patient who is suffering from a severe pulmonary catarrh a mixture containing carbonate of ammonia and other stimulating expectorants as a matter of course and in total disregard of the stage of the derangement or the character of the symptoms. But in the management of a bronchial catarrh each class of remedy has its own time for serviceable action and is useless or worse than useless if given out of its due season. The whole treatment of this derangement consists in unloading the congested vessels and setting up free secretion as a first and indispensable step before any attempt to reduce the amount of expectoration can be made. To give ammonia, squill, pargoric and other stimulating and antispasmodic drugs in the early stage of the catarrh is to make the cough harder and the chest tighter, and greatly to aggravate the discomfort of the patient, if not to produce worse ill-consequences. By such means I am convinced that what should have been a mild indisposition has often been aggravated into a serious illness by driving the catarrh further and further into the minuter tubes, and that in children a moderate bronchitis has not seldom been turned into a broncho-pneumonia. The use of these remedies should be reserved strictly for the later stages of the catarrh when the cough is perfectly loose from a free secretion of mucus. The earlier remedies have then finished their work, and the time has come for stimulants and astringents to take their place, and begin their task of bracing up the relaxed mucous membrane and guiding the complaint to a satisfactory issue. Antimony is not employed with any view of depressing the patient, and therefore it is advisable to prescribe it in small doses, frequently repeated, rather than in larger doses given at longer intervals. It not only acts more efficiently when used in this way, but its effect can be more easily noted, and the dose repeated more or less often according as it