flaccid. Dr. Macdonald and I made a stethoscopic examination of the heart, and finding no pulsation or cardiac sounds of any kind, I wrapped the corpse in a sheet and deposited it under the bed.

It being now noon, Dr. Macdonald left the house, and I extracted the placenta by Credè's method, and applied a binder.

Thinking I had had an anxious case in hand, I sat by my patient's bedside till 1 P.M., about which time I heard a rustling under the bed, and asked the patient if the house was infested by mice. She said, no. But, hearing a repetition of the sound, I became suspicious, and took leave to open up the sheet containing the wounded innocent, whereupon I found, at first, no signs of life, but just while again about to shroud it in its sheet I observed a quivering of the muscles, whereupon I immediately proceeded to artificial respiration and the alternate use of hot and cold water, &c.; these resuscitating measures being continued till 2·30 P.M., I was rewarded by being able to hand a living child over to the nurse, and after dressing and bandaging the lacerated limb I left. Both patients progressed favourably through the puerperal week.

At the end of fourteen days the mother called asking permission to proceed to the seaside. While there the baby contracted pneumonia and died; the mother was then observed by her relatives to become excited and very restless, and on medical examination was pronounced insane, and is now under care in an asylum in Perthshire.

NOTES OF A CASE OF ULCERATIVE ENDOCARDITIS.

BY J. CRAWFORD RENTON, M.B. ED.,
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JOSEPH COATS, M.D.

A. W., æt. 34, of average height, but pale and lightly built, came under observation on 13th March, 1880, when the following note was made:—Patient complains that two days ago he had a slight shivering, which was followed by headache, sickness, and vomiting, accompanied by pain over the region of

Fig 1

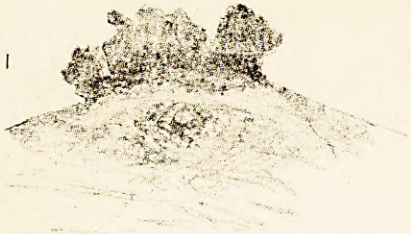

Fig 2

Fig. 9.

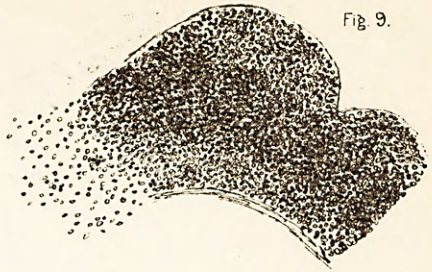

Fig 3.

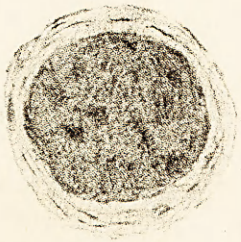

Fig 4.

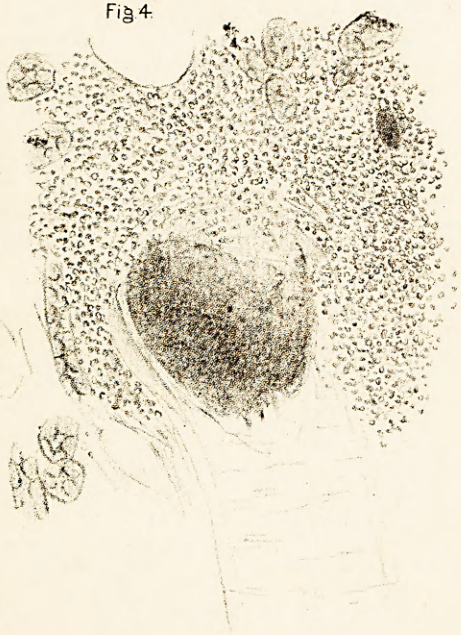

Fig 5

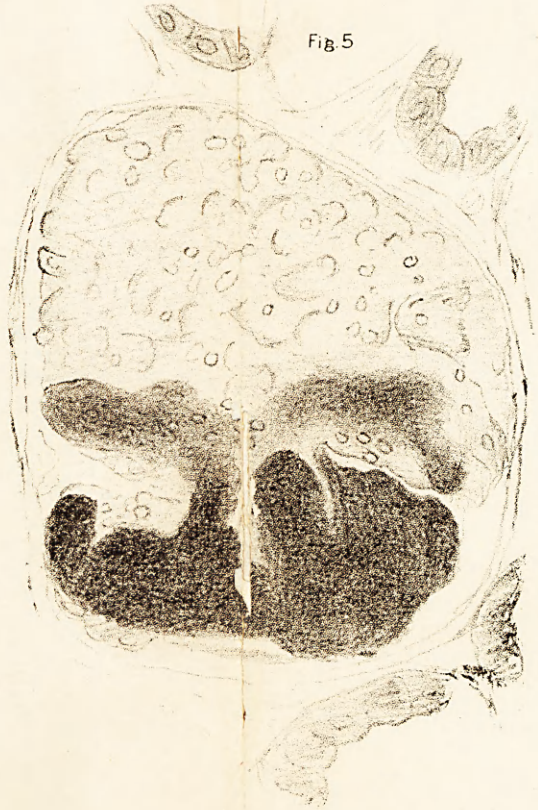

Fig 8

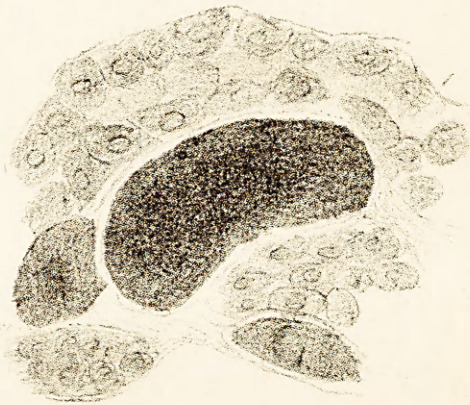

Fig 6

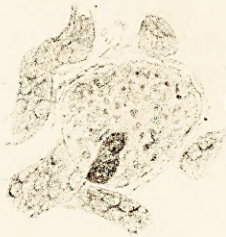

Fig 7

the liver. The vomiting is aggravated when any food is taken.

Previous History.—Five years ago patient had an attack of rheumatic fever which affected his heart, and since then he has been subject to rheumatic pains. Four weeks ago he had an attack of pain in the knees and ankles, which speedily subsided on the administration of salicylate of soda.

Family History.—Distinctly rheumatic.

Present Condition.—Pulse 120, temperature 102°. Tongue furred. Pain on pressure over the liver, but no increase of dulness. Bowels constipated. No cough or pain in the chest, the cardiac dulness is increased, especially in a downward direction and to the left, and on auscultation a loud mitral murmur is heard. This had been previously noted as a result of the rheumatic fever; the action of the heart is quite regular. Urine high coloured. Patient is advised to keep his bed, and to have linseed poultices applied over the liver; with iced milk or milk and potash water to drink, a simple pill to be given at bed time to move the bowels.

14th.—Patient has been very restless all night and wished to leave his bed. Pulse 120 regular, temperature 103°. Bowels have acted freely, and the vomiting has ceased. Milk is taken willingly.

Evening.—Pulse 120, temperature 104°. Over the chest and abdomen a number of hæmorrhagic spots were observed, and the nurse states that his nose bled freely. Two tablespoonfuls of brandy ordered to be given every three hours. Owing to a mistake a sample of the urine was only obtained to-day, and on microscopic examination was found to contain blood.

15th, Morning.—Has had a restless night. Pulse 120, temperature 103·5°. Tongue dry and brown; the purpuric spots have increased in number over the chest and abdomen, and the conjunctivæ are distinctly yellow, as also the body generally.

8 p.m.—Professor M'Call Anderson saw patient in consultation with me, and we found his pulse 130, and temperature 104°. After examining him carefully, Dr. Anderson coincided with me in thinking that our patient was suffering from some form of blood poisoning, its exact nature not being as yet apparent. The absence of any fresh cardiac symptoms, and the presence of slight abdominal tenderness led us to think that possibly enteric fever might be developing. We agreed to recommend a continuance of milk diet, with stimulants, and

sponging the surface of the body with tepid water to reduce the temperature.

16th.—Has had a restless night. Pulse 130, temperature 104°. Tongue dry and cracked. The yellowness of the skin is more apparent. The tenderness over the liver is more marked, and its dulness is decidedly increased. Has had one loose motion, pale in colour, and very fœtid. Urine redder in colour, and contains blood and tube casts. There are several large fresh hæmorrhagic spots on the feet, and some of those first observed seem to be increased in size. Patient continues to take nourishment well.

Evening.—Pulse 130, temperature 104°. Condition same as in morning.

17th.—After a very restless night patient became visibly weaker, and answered questions very slowly. Pulse weak and rapid, temperature 105°. Professor Anderson saw patient with me, and advised ice to be applied to the abdomen and head, and ten grains of quinine to be given internally. This had the effect of reducing the temperature to 102°, and rendering the patient more comfortable.

5·30.—Patient gradually became weaker, and died.

The *post-mortem* examination, which, being made in a private house, could not be perfectly complete, gave the following results:—The skin was of a generally yellow colour, with numerous red petechiæ on face, chest, and abdomen, and to a less extent on the feet. The posterior parts of the body had a generally dusky tint from decomposition, which extended forward some distance on the arms, neck, shoulders, and face.

On opening the chest, the pericardium was found adherent throughout. The heart, with pericardium attached, weighed 13½ ounces, being slightly enlarged. The heart being laid open in the usual way, the aortic and mitral valves were found to be festooned with soft and friable material. This was particularly manifest on the aortic valve, one of whose semilunar curtains (the right) had an extensive shaggy mass projecting from it. Where this mass was attached it could easily be seen that the valvular structure was disorganised, and, on removing the soft material, a circular gap was left in the curtain about a third of an inch in diameter. On the other curtains the deposition was mainly at the borders contiguous to that chiefly affected. In addition to these appearances of recent disease, the aortic curtains were generally thicker than normal, and there was also a localized thickening of the endocardium at the base of the curtains, forming a

band half-an-inch in breadth, and extending transversely for a distance of an inch and a half. This had the appearance of a dense tendinous layer, which, like the other white structures of the body, had a distinctly yellow colour. The mitral valve was also slightly thickened, and, when viewed from the auricle, it was seen to be fringed with soft vegetations just within the edges of the curtains.

The lungs were non-adherent, and simply presented œdema in their posterior parts.

The spleen was greatly enlarged, weighing over 14 ounces. Before opening it there were several hard places detected, which felt like infarctions; on cutting into these, fluid blood escaped abundantly, and the splenic tissue was seen to be broken down. There were one or two more solid infarctions with slight caseous metamorphosis.

Both kidneys were enlarged, weighing $6\frac{3}{4}$ and $7\frac{1}{4}$ ounces. In both there were two or three large wedge-shaped infarctions, which presented at some parts a yellow colour, as if from pus. In addition, the kidneys were beset by innumerable hæmorrhagic spots, each with a yellow point (pus) in the middle. These were mostly in the cortex, but were also present, to some extent, in the pyramids.

The liver was large and soft, but without any hæmorrhagic spots.

The intestines presented numerous blotches of hæmorrhage under the peritoneum, and in two or three places there were considerable tracts of intestine of a dark brown colour, which extended through the entire thickness of the gut, and was suggestive of gangrene.

On exposing the brain, considerable subarachnoid hæmorrhage was observed on the antero-lateral aspects, and in the brain substance there were a few small areas of softening, which, on section, presented a punctuated red appearance.

The heart and kidneys were examined, microscopically, in the fresh state. The exudation on the valves was found to be largely made up of micrococci, which gave the usual reactions. In the kidneys the yellow dots in the centres of the red spots were found to consist of masses of pus corpuscles, and surrounding the spots there was great hyperæmia of the vessels. It was also seen that micrococci were present in narrow tubes, but the consideration of their relations was deferred till the tissues were hardened. One of the petechial spots on the skin was also removed and hardened in spirit.

A portion of the mitral valve including the exudation, and portions of the kidneys, were hardened in alcohol and chromic

acid, and the following are the results of examination, illustrated by the lithographic plate.

In figures 1 and 2, sections of the mitral valve, with the lesion included, are shown; in figure 1, with a very low power, and in figure 2 with a moderate degree of magnification. It appears that, in the rough projections, two constituents are recognisable. On the surface there is a layer presenting merely a molecular structure, but really consisting, as appears when a high power is used, of fibrine mixed with a granular material. This granular material is in masses or colonies, and by comparison with the more definite aggregations of similar material in the kidneys is readily recognised to be masses of micrococci or bacteria. In fact, this superficial layer is made up of fibrine and minute organisms of the character, as commonly described, of micrococci or globular bacteria. Beneath this layer there are masses of round cells, as shown in figure 2, having very much the characters of granulation tissue, or any other inflammatory tissue. It is to be observed that the superficial layer consists of very soft material, portions of which are easily broken off, and that the layer represented in these figures is small as compared with what originally existed. On one of the curtains of the aortic valve especially, there were large masses of this soft material, which was found in the fresh state, as already mentioned, to be composed largely of masses of micrococci.

Turning now to the kidneys, there were here abundant evidences of embolism. Taking, first, the arteries, there were frequent examples of what is shown in figures 3 and 4. The calibre of the vessel is seen to be obstructed by a material essentially similar to that forming the superficial layer of the endocardiac lesion. We have not here merely micrococci, but a somewhat structureless material in addition, the micrococci being indicated by the darker areas seen especially in figure 3. It is clear that here there has been a direct transportation of material from the valvular structures to the kidney, and it was noticed that the actual plugging had frequently taken place at the bifurcation of an artery, this again being a usual occurrence in embolism. The dark spots and collections of pus, seen in the fresh state, were in connection with these embolisms. Figure 4 illustrates the condition of matters. The artery is shown for a short distance on the proximal side of the plug, and here its coats are distinctly distinguishable. But at the seat of plugging only the general lie of the coats can be distinguished, their constituents being no longer recognisable. In fact, the coats seem to have ceased

to exist as such. Around the artery there is an active inflammation, as evidenced by the enormous aggregation of round cells; these have infiltrated the coat of the artery, and are even penetrating into and partly displacing the plug, as appears at the upper part. The presence of the embolus has apparently led to destruction of the wall of the artery, and to an acute inflammation around it.

Besides the filling up of arteries, there was plugging of two other sets of vessels. In the first place, the Malpighian tufts were frequently the seat of masses of micrococci, as shown in figures 5, 6, and 7. The micrococci here appear to be unmixed with any other material, forming aggregations of granules which, together, produce an exceedingly dark opaque appearance. It is clear that the micrococci are in the vessels of the tufts, their whole arrangement indicating this. It often happens that just a single loop is filled, as in figures 6 and 7, and sometimes the afferent vessel is affected as well as one loop (fig. 7). Sometimes, as in figure 5 (much more highly magnified), there are a considerable number of loops involved. Occasionally, but rarely, a whole tuft is overwhelmed, and stands out as a dark mass in the midst of the more transparent tissue. Besides the Malpighian vessels there is still another set which are filled with micrococci, as shown in figures 8 and 9. There is never any great length of vessel involved, but here and there throughout the cortex of the kidney one meets with a little piece of vessel crammed with micrococci, the vessel being obviously distended by them. The micrococci here, as elsewhere, appear as minute granules, but sometimes with a very high power where the section was very thin, and had happened to lay open the vessel, an appearance such as that shown in figure 9 was presented. Here it is seen that there is not simply a mass of irregular granules, but the mass is composed of homogeneous and perfectly formed globular bodies. After careful examination we have come to the conclusion that the vessels concerned are capillary blood-vessels. In figure 8 the distended vessels are seen to lie between uriniferous tubules, and they are obviously capillaries. We were unable to find any evidence of the existence of micrococci in the tubules, although this has been described by others.

Sections were made of the piece of skin, including the petechial spot, but no embolic appearances were detected. The spleen was much too soft to make it possible to procure consistent sections. In bits removed by the scissors, nothing remarkable was found. Sections of the liver showed enlarge-

ment of the cells with cloudy swelling, and a very marked fatty degeneration, the usual appearances of an aggravated parenchymatous inflammation.

Having now described the appearances presented in this case, it remains to consider certain points in the pathology of the disease. It will be apparent, in the first place, that the diseased valvular structures of the heart supplied to the kidneys and elsewhere numerous emboli. These emboli were of a peculiarly irritative nature, not producing the usual results of the emboli of simple endocarditis, but giving rise, as we have seen, in the kidneys, to multiple abscesses. It is impossible to dissociate this peculiar character of the emboli from the existence in them, and in their source in the heart, of minute organisms—the micrococci. We shall discuss further on the relation of these organisms to the lesions in the kidney, but, in the first place, their relation to the endocarditis claims attention.

In the inflamed valvular structures of the heart, two abnormal constituents, as shown in figures 1 and especially 2, call for explanation. We have a superficial layer of material, consisting of fibrine and micrococci, and a deeper layer consisting of round cells, the usual products of inflammation. The condition differs from that seen in an ordinary acute endocarditis in the presence of the micrococci, and the question at once occurs—Are these organisms to be regarded as the cause of the inflammation, or can they be looked upon as secondary? This question must be considered entirely apart from that of the relation of these organisms to the lesions in the kidneys.

An important point in relation to this question is the fact that, as shown both by the clinical history and by the *post-mortem* examination, this patient had one or more previous attacks of rheumatic endocarditis. There was found, as the report bears, an unusual amount of cicatricial tissue, the result of a chronic endocarditis which had followed one or more acute attacks. The adherent pericardium points to a pericarditis, probably of the same date. The patient had gone through his attacks of acute endocarditis and pericarditis, but without any of those malignant manifestations which finally cut short his life. The presumption from this is that in the present illness the starting point was an ordinary acute endocarditis. The history of the fatal illness bears out this view. The attack of rheumatic fever is stated to have occurred four weeks before the onset of the malignant symptoms, and to have readily subsided under treatment. The same result is

reached by a consideration of the locality of the micrococci. They are found in masses on the surface of the inflamed structures, there being a tolerably distinct line of demarcation between the collections of inflammatory cells and the masses of micrococci. It is as if these had been deposited on the inflamed surface, perhaps adding to the inflammation by their influence.

At the same time, it should be said that although this inference may be drawn from the appearances presented, it is still not quite free from possible fallacy. The disease is here in a comparatively advanced stage of development, and it is very possible that at an early stage the relation of the micrococci to the inflammatory condition may have been much more direct and apparent. In this connection we have to refer to a recent communication by Professor Köster,* in which he asserts that micrococci are concerned in ordinary endocarditis, such as that met with in acute rheumatism. He finds in nearly all cases of acute endocarditis, even of the simplest forms, colonies of micrococci in the exudation covering the inflamed valvular structures, and he seems to assert that the ulcerative endocarditis is only an aggravated form of simple acute endocarditis. He believes that the micrococci circulating in the blood first lodge in the smaller blood-vessels of the valvular structures, forming minute embolisms there, and that the endocarditis is, in this sense, to be regarded as embolic. These views of Köster stand in need of confirmation, but taken along with the facts illustrated in our case, and with the fact that in several recorded cases of ordinary rheumatic endocarditis there have been secondary abscesses developed in the kidneys and elsewhere, it appears reasonable to draw closer the association between ordinary rheumatic endocarditis and the ulcerative form.

There is another relation of this disease which demands a passing notice. In puerperal fever and in pyæmia we sometimes encounter the conjunction of ulcerative endocarditis and secondary suppurations in the kidneys, &c. Eberth † has found in cases of pyæmia with this complication, minute organisms both in the affected endocardium and the secondary abscesses, and is inclined to regard the conditions here and in ulcerative endocarditis as identical, in so far as these structures are concerned. He also regards the organisms in this disease

* "Die embolische Endocarditis," von Professor K. Köster, in Bonn. Virchow's *Archiv.* Bd. lxxii, 1878. P. 257.

† "Ueber diphtherische Endocarditis," von C. J. Eberth. Virchow's *Archiv.* Bd. lvii, 1873. P. 228.

and in pyæmia, as the same as those occurring in diphtheria. According to his view there is in pyæmia a diphtheritic affection of the wound, the organisms penetrating some distance into the tissues, and reaching the circulating blood. In this view of the identity of these organisms with those of diphtheria, Eberth is opposed by certain weighty authorities. In this connection it will be remembered that ulcerative endocarditis is an occasional accompaniment of diphtheria, and when that is the case we may presume that the organisms which have to do with the primary local phenomena are the same in kind as those which find a lodgment in the endocardium. By some authors the relation of ulcerative endocarditis with diphtheritic processes is regarded as so close, that they designate the disease with the name diphtheritic endocarditis. In a case like the present, where the disease had not its origin in diphtheria or pyæmia, it is difficult to understand how the organisms have reached the endocardium. It is possible that such organisms are regularly absorbed from the intestinal tract under normal conditions, but are neutralised by the living tissues. In certain states of disease, however, the tissues may lose their power of destroying these organisms, and so their development is permitted.

The existence of these organisms in the affected endocardium explains most of the other phenomena observed. We have more particularly to consider the lesions in the kidneys. In this regard a very important difference is to be noticed between the conditions presented by the arteries as compared with the other vessels. The plugging of the arteries has in nearly every case given rise to an acute reactive inflammation, while no trace of inflammation could be discovered in connection with the other vessels, unless they were in the vicinity of an abscess. We may bring into relation with this the different constitution and circumstances of the plugs. In the case of the arteries the plug is composed of micrococci and broken down fibrine, in fact, the material is the result of processes which have been going on in the heart. In the Malpighian tufts and capillaries, the micrococci are pure, and they distend the vessels in such a way as to suggest that they have grown on the spot. It is probable that some organisms have attached themselves to the walls of the vessels, and have multiplied there to a very great extent. It is obvious that these organisms are more recent than those which have come direct from the heart. We shall afterwards see reason to believe that organisms exist abundantly in the blood in this disease, apart from massive embolism

from the heart, and it is probable that the plugs in the smaller vessels have their origin in these more isolated micrococci. The plugs in the arteries have frequently produced what appears to be a necrosis of the wall of the artery. Some have regarded this necrosis as the cause of the inflammation, the dead tissue irritating the surrounding structures, and the absence of inflammation in connection with the plugs of the smaller vessels seems to confirm this view. It is clear at any rate that the micrococci themselves are not a serious source of irritation. It is very possible, however, that their products may be so. The organisms in the smaller vessels being of recent growth, have perhaps not had time to produce sufficient irritating material, while the older ones from the heart have carried their products with them.

It is noted in the clinical history of the case, and in the *post-mortem* report, that the skin had a yellow colour. This jaundice was not of hepatic origin, as there was nothing in the liver indicating the reabsorption of bile. It had its origin in the decomposition of the blood corpuscles and the setting free of their colouring matter, the so-called hæmatogenous icterus. This decomposition of the blood corpuscles occurs also in pyæmia, and is to be related to the micrococci in the blood. It is to be expected that the altered blood will have some deleterious effect on certain of the tissues of the body, and it is a question how many of the lesions found are to be traced to this cause, and how many are embolic. The lesions in the spleen are distinctly embolic, although it was not possible in the soft state of the organ to determine the actual existence of plugs. The lesions of the intestine culminating sometimes in a condition approaching to gangrene, are also in all probability embolic. The petechiæ in the skin are possibly embolic, although we were unable to detect the actual plug in the one examined, but they may have their origin in the altered state of the blood. The softenings of the brain are embolic, although the subarachnoid hæmorrhage is more probably due to the decomposing blood. The enlargement of the liver is of the same nature as that seen in most acute diseases accompanied by high temperature, and is the result of irritation by the altered blood.

EXPLANATION OF PLATE.

Figure 1. General appearance of exudation on mitral valve, as seen with a very low power, about 12 diameters.

Figure 2. A portion of the lesion of the mitral valve, showing the two constituents, a superficial layer of fibrine and micrococci, and a deeper inflammatory layer, 90 diameters.

Figure 3. An artery plugged with micrococci and broken down fibrine. The darker spots are masses of micrococci, 90 diameters.

Figure 4. An artery plugged. At the upper part the wall is destroyed and partly infiltrated with inflammatory cells, which are in great abundance around, 90 diameters.

Figure 5. A Malpighian tuft, with a number of loops filled with micrococci, 300 diameters.

Figure 6. A Malpighian tuft, with a single loop filled, 90 diameters.

Figure 7. A Malpighian tuft, with the afferent vessel, and a single loop filled, 90 diameters.

Figure 8. Three portions of capillaries filled with micrococci, the centre one greatly distended, 300 diameters.

Figure 9. A capillary filled with micrococci, some of which are partially isolated, and seen to be composed of globular bodies, 650 diameters.

ON THE INHALATION OF CARBOLIC ACID IN DISEASES OF THE RESPIRATORY ORGANS.

BY ROBERT MUNRO, M.A., M.D., Kilmarnock.

THE recent publication of two articles in the *British Medical Journal*, one by Dr. J. B. Yeo (20th December, 1879), "On the Treatment of Tuberculosis based on the Theory of the Bacteric Origin of this Disease, as suggested and carried out by Dr. Max Schüller," and the other by Dr. Mackenzie, of Edinburgh (3rd January, 1880), "On the Antiseptic Treatment of Phthisis Pulmonalis," affords me an opportunity of bringing before the profession the result of my experience of a similar mode of treatment which I have practised more or less for the last ten years in various lung diseases. Ever since I commenced the study of medicine, the treatment of inflammatory phthisis, chronic bronchitis, asthma, gangrene of the lungs, &c., by the inhalation of vapours to act either as stimulating lotions, antiseptics, or antispasmotics, according to circumstances, appeared to me so rational that I have given more than ordinary attention to the subject. My first experiments were made with sulphurous acid, creosote, carbolic acid, tincture of benzoin, and various ethers; but, since 1870, I have exclusively adhered, in the suppurating stages of diseases of the respiratory organs, to the simple plan of inhaling the steam from a solution of carbolic acid and hot water.

As my object here is to draw attention to the beneficial effects of this mode of using carbolic acid, I lay aside all theoretical considerations as to the nature of tuberculosis and the mode of action of the acid, and merely state, from an