Two Clinical Lectures

ON

MALARIA AND THE MALARIAL PARASITE.

*Delivered at the Medical Graduates' College and
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MEDICINE.LECTURE II.¹*Delivered on Feb. 28th, 1900.*

GENTLEMEN,—The morbid anatomy of malaria is fairly constant, consisting principally of enlargement of the spleen, congestion of the liver and portal circulation, and pigmentation of the spleen, liver, brain, and many other viscera. The microscopic anatomy, too, is fairly constant. In sections of all the organs mentioned, in the vessels in every instance, and, in the case of the spleen and bone-marrow, in the parenchyma also, there is to be found a remarkable deposit of the peculiar pigment which is so characteristic of malaria. This pigmentation arises from (a) the pigment belonging to and contained in living parasites—those little aggregations of pigment which I described as a feature in all mature malaria parasites—and (b) from the same pigment included in leucocytes, in endothelium, and, in the case of the spleen and bone-marrow, in the cells of the parenchyma. If you study sections of brain from cases of fatal malarial fever you will often see the capillaries distinctly outlined by the pigmented malaria organisms which they contain. The whole of the lumen of the capillary network is occupied by corpuscles containing little pigment masses. By carefully focussing you will see that each pigment mass is included in a red blood-corpuscle. Sometimes about the centre of a corpuscle you will see the mass of black pigment and surrounding this a body which you may be able to resolve into a cluster of minute sporules. This crowding of the vessels of the brain with parasites affords ample explanation of many of the phenomena of that type of malaria known as pernicious cerebral malaria. In sections of liver you will see a similar arrangement—pigmented parasites, and, much more abundantly than in the case of the brain, masses of pigment in leucocytes and here and there in endothelium. In the case of the spleen in one of the preparations which I show this afternoon you can see, besides the small parasites, each with its little mass of melanin in the capillaries, and besides small pigment-laden splenic cells, huge cells filled apparently with large block-like masses as of charcoal. The bone-marrow specimen exhibits similar appearances. Like the spleen, it contains the malarial pigment outside as well as inside the vessels. In the preparation of mesentery also you can trace a delicate capillary dotted here and there with pigmented sporulating parasites. In chronic malarial conditions you have secondary changes, fibrotic and so forth. These, of course, are not peculiar to malarial disease.

In addition to the black pigment, particularly in those cases of malaria which are accompanied with extensive rapid destruction of blood-corpuscles, you will find another type of pigment—a yellow pigment. This pigment is present in all conditions, accompanied by acute blood destruction. It is not peculiar to malaria; it is found in the hæmolysis attending extensive burns, in poisoning by chlorate of potash or arseniuretted hydrogen, in paroxysmal hæmoglobinuria, and so forth. This pigment is deposited hæmoglobin.

Another and important feature in the pathological anatomy of malaria, and one which you will discover by a careful examination of the blood in the living subject, is anæmia—an intense and, in acute cases, generally a rapidly advancing anæmia. If you examine a patient from time to time after successive malarial paroxysms you will find that the drop in the number of blood-corpuscles per cubic millimetre is very great indeed. In the course of a severe untreated attack the patient is reduced to a condition of intense pallor with a

rapidity hardly equalled in any other disease. It is not so, of course, in every case, but in bad types you will find that this holds true. The cause of this excessive anæmia is not very obvious if we rely simply on examination of the blood from the finger for an explanation. If you place finger-blood from a case of malaria under the microscope you may find one or two parasites perhaps in each field. In many cases of severe malaria you will not find so many as that even; sometimes you will have to search through three, four, or five fields before you find one parasite.

How, you may ask, could so small a number of blood-destroying parasites account for anæmia so pronounced and so rapidly evolved? The reason is that the malaria parasite is not especially, or even ordinarily, a parasite of the peripheral circulation. It is a parasite of the blood of the central organs, as evidenced by the specimens shown this afternoon under the microscope. In a case of comatose pernicious malarial fever, with perhaps only three or four or five parasites in every field of finger-blood, you will find post mortem almost every blood-corpuscle in the capillaries of the brain carrying its own parasite. That is the reason why blood destruction is so prominent a feature in severe malarial attacks. The blood is not destroyed by the few straggling parasites seen in the peripheral circulation, but by the myriads of parasites in the blood of the viscera—the brain, the liver, the spleen, the bone-marrow, and so forth.

The behaviour of the leucocytes also is an interesting feature in the pathology of malaria. The polynuclear leucocytes decrease notably in number during the acute stages of malarial attacks; sometimes you may have a difficulty in finding them in the peripheral blood. The large mono-nuclear white cells increase as a rule and often contain malarial pigment. These large mononuclear corpuscles, as well as a certain number of the polynuclear corpuscles, carry the pigment to the spleen and there deposit their burden, which is taken up by the cells of this organ, producing the greater part of that enormous deposit of pigment so clearly illustrated by one of the sections under the microscope.

A peculiar feature clinically about malarial disease is its periodicity. This periodicity is accounted for by the periodicity and simultaneity of development of the parasites; they all seem to keep together as a swarm. At a particular time of the malarial clinical cycle you will find a certain form of parasite in the blood. Just before the explosion of the fever you will find a predominance of the rosette bodies; after the fever has subsided you will find the small form of the parasite as the predominant feature, and so on. Why should this be? Why should there be a conspiracy, as it were, among the different parasites all to mature at a certain time and all at the same stage? This simultaneity of maturation, so to speak, is the reason for the intermittence and regular periodicity of malarial fever. But what is the reason for the parasites observing this periodicity? The question is a very difficult one to answer. I believe the reason is twofold: first, the habit of the parasite to live a more or less definite number of hours; and, secondly, the diurnal rhythm in the periodicity of the physiological processes of the human body. At a certain time of the day in health the body temperature begins to rise and at a certain time it begins to fall; so with the tendency to sleep, to eat, to secrete, and to excrete particular substances there is a definite diurnal periodicity in the normal physiological cycle. This diurnal physiological periodicity, combined with the tendency of the parasite to live for a definite time impresses, I would suggest, the periodicity on the parasite of malaria which is so very characteristic of it and of the disease it produces.

So far as is known the lower animals are absolutely immune as regards the human forms of malarial disease. If you inject human malarial blood into a monkey there is no subsequent proliferation of the parasite and no fever. The same is the case with the dog or horse or other animals. Therefore in the lower animals there is complete immunity as regards the malarial parasite. We also know that certain human beings, very few it is true, enjoy a similar absolute immunity as regards this parasite. Some people coming into a malarial district contract the disease within a week or ten days of their arrival, others resist the disease for months, and a very few resist it permanently. That fact shows that *qua* the malarial parasite there is a varying degree of immunity in the same human body and in different individuals. I believe that this immunity possessed by the human body, like other physiological properties, has, as regards its amount a diurnal rhythm. At one time of the day the resistive power against a malaria parasite is greater

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than at another time of the day. I think that any malaria parasite which might enter or be set free in the blood when the immunising power of the body is at its height is likely to be destroyed by the resistive power. Should it happen that the power of resistance is at its minimum, then the parasite would survive, enter a blood-corpuscle, mature, and propagate its kind. In that way, it may be, the periodicity of malaria is caused—viz., by the combination of physiological conditions and the properties, so to speak, of the parasites.

We know when the new-comer arrives in a malarial country his first attack of malaria is usually a very severe one; it is usually either a continued or a remittent fever; that is to say, the fever does not exhibit in any marked way the definite periodicity which we are accustomed to associate with malaria. Apparently the parasites are permitted to enter the blood-corpuscles and mature at all times of the 24 hours. After a time, however, the body seems to become educated to a certain extent, so that by-and-by a definite periodicity is impressed upon the parasite and the continued fever after a time becomes remittent and by-and-by intermittent. I would interpret this by supposing that the body gradually acquires a certain degree of immunity and reaches at some time in the 24 hours the standard of absolute immunity and that in this way the parasite and the symptoms it gives rise to get a definite periodicity impressed upon them.

It is almost useless for me to attempt to describe the manifold clinical forms of malarial disease. We know that malarial fevers tend to assume a quotidian, a tertian, or a quartan type. We also know that in certain cases the quotidian becomes a remittent or continued fever. We also know that if we examine the blood in remittent or in continued malarial fevers we sometimes find in it the type of parasite characteristic of tertian, sometimes of quartan, sometimes of the æstivo-autumnal infection, and sometimes a mixture of two or even of all the types of parasites. At first it seems difficult to understand how a tertian or a quartan parasite could give rise to a quotidian fever. But such is the case and there can be no doubt that it can be explained. If you look at the blood of a patient with quotidian fever in very few instances will you find a quotidian parasite, which is amongst the rarest of malarial infections, if, indeed, it exists at all. You will find generally either a tertian, a quartan, or an æstivo-autumnal, which is also tertian in its periodicity. In the case of a quotidian fever arising from a tertian infection there are two generations of parasites, one which is half-matured and one which is practically matured. The half-matured parasites will produce the fever of to-morrow and of three days hence, the fully-matured will produce the fever of to-day and that of the day after to-morrow, and so on. A double quartan infection will produce a double quartan fever. You get fever to-day and to-morrow but no fever the day after to-morrow. A treble quartan infection, in the same way, will produce a quotidian fever. If the three forms of parasites concur you will have a corresponding mixture of clinical symptoms. You will easily understand how confused the resulting clinical manifestation will be from this fusion of forms, but with the microscope and the temperature chart you can unravel the clinical puzzle and read, so to speak, the writing in the blood.

Disregarding the ordinary intermittents, I will say a word about pernicious malarial fevers, a type which has been very much neglected by English writers and which is better understood by our French colleagues—at all events it is more dwelt on by them.

In the course of what appears to be an ordinary ague, and which we do not ordinarily associate with any particular danger, there is suddenly developed some alarming symptom which if it be not properly appreciated may prove fatal, and that in the course of a very few hours. These are called pernicious attacks (*accès pernacieux*), and are classified under two headings, one conveniently called "cerebral" and the other "algid," the latter being associated with adynamic conditions, the former with manifest implications of the central nervous system.

The most common type of pernicious attack is known as "pernicious comatose malaria." The patient in the course of what apparently is an ordinary malarial attack suddenly becomes comatose; he cannot be roused, he has stertorous breathing, and unless skilfully treated he may die. Occasionally he comes out of this condition and temporarily recovers, but possibly at the next paroxysm of the fever he will fall into the same condition and die. Often these sudden attacks of insensibility are put down to apoplexy and very

often a prognosis of hemiplegia is given. Fortunately for the patient this prognosis is sometimes upset by his coming out of the comatose condition without showing any evidence of hemiplegia.

Another type of pernicious cerebral fever, often mistaken for, and generally called, sunstroke, is hyperpyrexia. The patient has an attack of apparently ordinary ague, in the course of which, instead of his bursting out into perspiration and the symptoms subsiding when the thermometer reaches 105° F., the temperature may go on rising to 107° or 110° or even higher. Wild delirium merges into coma and death. Hyperpyrexial attacks are terrible things to witness. I have seen a good many and at first was prone to fall into the ordinary mistake of diagnosing heat-stroke. They occur very often during oppressive weather and are common in intemperate people. The diagnosis of heat-stroke, under the circumstances, is a somewhat natural one to make. On more careful consideration, particularly if the patient's blood is examined or a post-mortem examination is made, you will find, either from the presence of parasites or from pigmentation in the viscera, that the patient died not from heat-stroke but from malarial poisoning. Thus malarial coma and malarial hyperpyrexia are the most frequent and important of the cerebral pernicious types. But there are other forms: a not unusual one, particularly in children, is the epileptiform seizure.

In tropical countries it often happens that the first intimation of malarial infection in a child is an epileptiform seizure. Perhaps two days later there is a similar attack. You are inclined to give a gloomy prognosis; you tell the mother that the child is possibly an epileptic and you prescribe bromide. If you wait for 48 hours longer there is a third seizure and then if you use the microscope and the thermometer you will find that the child is not suffering from true epilepsy but from malarial fever. In adults also you sometimes have strange periodic seizures. One is a suddenly developed aphasia. I have seen this on more than one occasion. You are asked to visit a patient and you find him in bed exhibiting all the peculiarity of some form of aphasia, unable to speak, or using wrong words, misplacing them, and so on, and you are prone to diagnose grave cerebral disease. Occasionally the extrinsic muscles of the eyes are affected with some form of paresis. These cases are more common than is supposed but they are very frequently overlooked.

An explanation of these and similar conditions—the comatose cerebral attacks, the hyperpyrexial attacks, the epileptic seizures, the parietic conditions—is supplied by the brain sections shown under the microscope. You will see that the capillaries of the brain are crowded with malarial parasites. Should one particular centre of the brain be specially affected by the parasites, as, for instance, Broca's convolution, then you have a corresponding symptom. Should the heat-regulating centre—if there be such a centre—be selected then you may have hyperpyretic conditions. The algid attacks are somewhat rarer but they are equally important. It is necessary that one should be aware that such attacks do occur. A malarial fever very like cholera is not an unusual thing in certain districts. The patient, instead of getting an ordinary malarial attack, is suddenly seized with choleraic symptoms—vomiting, purging, rice-water stools, cramps, *voix cholérique*, and all the other signs of epidemic cholera. If you inquire into the history you may learn that he had malarial attacks perhaps only one or two days before. You examine the blood and you find malarial parasites, and if you treat the patient for malaria you will cure his cholera. It is well to recollect the possibility of such attacks in tropical countries. Dangerous syncopal attacks may occur in association with the symptom which we usually hail as heralding relief in a malarial paroxysm. Profuse perspiration occurs till the bedding is drenched; if the patient gets up and tries to stand he has, it may be, a fatal syncopal attack. Sometimes profuse sweating occurs at the very commencement of the fever paroxysm; the patient is cold and algid and the profuse sweatings may end perhaps in collapse. In other forms vomiting, dysenteric conditions, and so on occur. Indeed, the variety of clinical features exhibited in malarial infections is too great for me to fully discuss on this occasion.

A practitioner in tropical countries ought always to suspect that any sudden and inexplicable attack may be attributable to malaria. I would emphasise the word *may* because I do not wish it to be understood that such an attack *must* be malarial. We should not conclude, as is so often done, that every case of disease connected with the tropics which we

cannot diagnose is malaria; still, we ought to suspect it. There is one important type of what is believed to be malarial disease which has of late come into prominent notice, partly in consequence of the opening up of Africa and partly in consequence of the utterances of a well-known pioneer in pathology—Professor Koch. I refer to blackwater fever. This is a disease which, strange to say, has not been recognised till within the last 30 or 40 years. If you search the writings of the old Indian physicians you will find no mention of blackwater fever or of any corresponding disease. Some time in the "fifties" certain French physicians first drew attention to its occurrence in one island off the coast of Madagascar, and since that time the disease has been recognised in the West Indies, in South America, in the southern parts of the United States, and in the Malay Archipelago. It is known to be the most common cause of mortality among Europeans on the West Coast of Africa. It exhibits all degrees of intensity. A patient resident in one of the endemic districts who perhaps has had many attacks of ordinary malarial fever develops what at first he considers as one of his usual malarial attacks and he does not think much of it. Suddenly he is seized with severe rigor. (Strange to say, rigor, so prominent a symptom of agues and intermittent fevers in the temperate zones, is singularly rare in the tropical zones. In those countries in which malarial fever is a really dangerous item in the pathology rigor is not as a rule a prominent malarial symptom. In blackwater fever it is different in this respect—violent shivering is a usual initial symptom.) The patient may then be seized with the desire to pass urine; he is astonished to find that his urine is black. He goes on urinating frequently and the urine continues to have this peculiar colour. He looks at himself in the mirror and finds that his face has become as yellow as saffron. This condition is associated very generally with fever, vomiting of biliary material, pain about the loins and back, and severe hepatalgia and intense prostration. Often as the case advances the patient exhibits all the symptoms of sudden profuse hæmorrhage (doubtless associated with the great discharge of hæmoglobin), giddiness, sweating, sighing, restlessness, and syncope. In many cases fortunately the hæmoglobinuria—for such is the symptom—gradually disappears with the fever, the urine becoming less dark and after a time natural in colour, but the patient is left intensely acæmic and jaundiced and only slowly recovers. Very likely there is no relapse. In some cases, however, the symptoms do not subside, the hæmoglobinuria persists, and the patient may die from syncope or exhaustion; or, the hæmoglobinuria persisting, the urine gradually gets smaller in quantity and becomes thicker and almost glutinous, being reduced to two or three ounces in the 24 hours. It may be entirely suppressed and the patient may die with uræmic symptoms, or he may recover temporarily and the discharge of urine may be re-established, but in two or three days or weeks he may die with symptoms of acute nephritis. Blackwater fever is a terrible disease; the mortality from it is enormous, amounting in some epidemics to as much as 50 per cent., in others to 25 per cent. Occasionally the mortality is as low as 10 or 15 per cent. The disease, however, is always a dangerous one, and it is one that is almost sure sooner or later, if the patient remains in the endemic area, to recur. The liability to recurrence unfortunately does not cease when the patient has left the endemic area, for, as with other malarial disease (if it be a malarial disease), symptoms are liable to return or even to appear for the first time after the patient has returned to Europe. I have seen a good many cases in England. Only three weeks ago I saw a young Englishman die from this disease in London. I do not know how long the liability to recurrence persists after return to temperate climates, but certainly it persists for five months. I have known of such cases.

There are certain popular fallacies about the diagnosis of malaria which it is well to bear in mind. The principal of these is that everybody who has passed through the Suez Canal, or who has visited Rome, or has lived in a tropical country, is the subject of malarial infection. That is about the commonest and the most vicious fallacy among practitioners in this country. Fortunately many tropical lands are absolutely free, or relatively free, from malaria; it is only in certain places even in tropical countries that malaria exists. Do not, therefore, when a patient comes from a tropical country complaining of some more or less obscure symptoms, jump to the conclusion that those symptoms are the outcome

of a malarial infection. One sees curious mistakes in connexion with this popular fallacy. I recollect reading some time ago in one of the medical papers two cases of what was called "malarial neuritis." I was very much struck with the evidence for founding his diagnosis of malarial neuritis with which the writer appeared to be satisfied. One patient was a sailor, and doubtless in the course of his life as a sailor he had visited malarial countries—*ergo*, his neuritis was malarial; that was all the evidence for the diagnosis. The other case was very much the same, the patient being a sailor who at one time—many years ago—had had malaria; and that was all the evidence which the writer had for diagnosing the case as one of malarial neuritis. This is typical of the diagnosis one often finds of obscure disease in patients from tropical countries.

Few diseases are so easily diagnosed as malaria. We have three pathognomonic tests which, intelligently applied, are absolutely infallible. There is the clinical test of periodicity, but diurnal periodicity is of no value in the diagnosis of malaria. A periodicity of 24 hours is shared with malaria by many diseases, but it is otherwise with a periodicity of 48 hours or 72 hours. Only one pathological condition exhibits this feature and that is the malarial infection. Any disease exhibiting a tertian or quartan periodicity is certainly malarial. But I strongly dissuade you from putting any reliance upon quotidian periodicity in the diagnosis of malaria; it is most misleading and constantly gives rise to mistake. If a patient has well-marked rigor followed by pyrexia, and that by profuse perspiration, and if this occurs day after day, do not jump to the conclusion that the case must be malarial because of the periodicity and because the patient is from a tropical country. In diagnosis quotidian periodicity should be absolutely ignored. I have seen a patient with well-marked abscess of the liver, the patient himself being a medical man, and yet because he had rigor, pyrexia, and perspiration day after day he was sure that he was suffering from malarial fever, and he was taking quinine in consequence. Nearly all suppurative processes are attended by a fever with a quotidian periodicity more or less marked. Osler says that he never sees a case of abscess of liver that has not been drenched with quinine, the diagnosis of malaria having been made doubtless in consequence of the quotidian recurrent pyrexia. Many diseases—ulcerative endocarditis, surgical kidney, gallstones, syphilis, tubercle, almost every disease with febrile associations—may exhibit quotidian periodicity. But it is otherwise with tertian and quartan periodicity; these are both absolutely pathognomonic of malaria. Then we have the therapeutic test—the test by quinine. Quinine properly administered in a case of malarial disease in 48 hours is in my experience invariably followed by abatement of the symptoms. If you give a patient quinine for 72 hours and there is no abatement of fever you should revise your diagnosis; the patient is not suffering from malaria. There is no use in persisting with quinine for days and weeks on the supposition that the case is malaria. You can poison the malaria parasite with quinine as certainly as you can poison a man with arsenic.

It is not sufficient, however, to rely on the therapeutic test, because in many instances we have no time to wait for its application, as in grave cerebral attacks. In such cases you cannot wait to see the effect of quinine before making the diagnosis. By the time the diagnosis is established the patient might be dead. It is necessary to have some more rapid means of arriving at the conclusion of what is the matter with the patient. That is supplied by the microscope; it is an immediate and invaluable test for malaria. Practitioners in tropical countries fail in their duty to their patients if they do not acquaint themselves with the application of the microscope in the diagnosis of malaria. For the practitioner nowadays to undertake the care of life in tropical countries and to be ignorant of the use of the microscope in malarial diagnosis is positively culpable. I think that the practitioner who visits a tropical country and pretends to take care of patients there and who cannot use his microscope is like a physician in England who is ignorant of the use of the stethoscope. Of the two instruments by far the more valuable in tropical practice is the microscope. The microscopic test is easily applied. All that is required is a high-power lens, a $\frac{1}{2}$ objective, a fair illumination and two or three slides, some cover slips, and a patient. In five minutes you can tell whether the patient is suffering from malaria or not. Anyone who ignores

this simple method is not only foolish but I say he is also sinful.

One word in conclusion about the treatment of malaria. This resolves itself into one word, the use of quinine. We have in quinine really a specific (about the only real specific in the Pharmacopœia), and if we can properly apply the specific in 99 cases out of 100 we can save the patient from the effects of the parasite. It is one thing, however, to possess the specific and it is another to be able to apply it. In cases of violent remittent fever and grave cerebral attacks the patient may be vomiting and unable to absorb the drug, or he may be insensible and unable to swallow it. You must have recourse to other means and give five or ten grains of a soluble salt of quinine hypodermically or rather intra-muscularly. This may save the patient; he may die without it. At one time this method of using quinine got out of favour because in several instances the hypodermic injection had been followed by tetanus and the death of the patient. Now, tetanus is not caused by quinine; it is caused by the tetanus bacillus. Death from tetanus following on the hypodermic injection of quinine is caused by the culpable carelessness of the physician who, in ignorance of course, has used an injection containing the tetanus bacillus. The hypodermic injection of quinine, though painful and often objected to by the patient, is free from risk provided that ordinary precautions are taken to render the syringe, the skin, and the solution aseptic.

A certain number of patients appear to have peculiar sensibilities as regards the action of quinine. In some it gives rise to urticaria, in some to peculiar toxic effects, and therefore to some patients it cannot be given at all. In these cases methylene blue is of service. I have not much experience of its use; I have not the same faith in it as I have in quinine. Professor Koch is one authority for its use. It is given in doses of from three to five grains three or four times a day. Another and graver accusation against quinine is that it is apt to give rise to hæmoglobinuria. Professor Koch's utterances in regard to this have been somewhat misunderstood. Many people suppose that Koch said that quinine gave rise to—is the cause or virus, as it were, of—hæmoglobinuria. What he did say was that quinine under certain conditions brought on hæmoglobinuric symptoms, and what he said is in my opinion, true, only he has expressed himself somewhat vaguely. A man is responsible not for his words only but also for the effect which they produce, and Koch's words have had a most prejudicial effect in this matter. In consequence of what he has said quinine is looked upon with suspicion, if it be not absolutely tabooed, in some tropical countries. This is unfortunate, for even if quinine is a danger in certain cases, if it does tend to produce hæmoglobinuria in some cases, it is not a one-hundredth part so dangerous in this respect as the malaria parasite. Of the two evils the parasite is infinitely the greater one, and therefore the withholding of quinine from a patient in whom the parasite is circulating is more apt to be followed by blackwater symptoms than if the drug be given.

In cases of blackwater fever examine the blood and if you find in it the fever forms of the malarial parasite give quinine; if you do not find the parasite withhold the quinine. This is a good rule. It is a fortunate circumstance that hæmoglobinuria frequently kills the parasite in the circulation. If you look at preparations of blood made just before the appearance of hæmoglobinuric symptoms you will often find the malarial parasite; if you examine the blood of the same patient just after the appearance of the hæmoglobinuric symptoms you may find a very few parasites; and if you examine it a little later, say 12 hours after, you may fail to find any parasites at all. Apparently there is a process of natural cure caused by the parasite itself when it brings about hæmoglobinuria. That should encourage us to withhold quinine in many cases of blackwater fever, especially when the parasite cannot be detected by patient and competent observation with the microscope.

INSPECTION OF COWS TO DETECT TUBERCULOSIS.—The Dartford Urban Council, after communication with the urban district councils of Bexley, Erith, and Dartford Rural Council have jointly appointed for one year Mr. A. Edgar, F.R.C.V.S., on terms agreed upon, to undertake the duties of periodically inspecting cows in the respective districts under their control.

A Clinical Lecture

ON

MODERN METHODS OF AMPUTATING AT THE HIP-JOINT,

WITH A TABLE OF 15 CASES.

Delivered at the General Hospital, Birmingham, on March 1st, 1900,

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GENTLEMEN,—The patient here presented is rather an unusual case, for you will see that she has had both her lower limbs removed at the hip-joint. The clinical history is briefly this. Her age at present is 27 years and for some years she had suffered from tuberculous caries of the tarsal bones of the right foot, so that in October, 1895, it was necessary to perform a Symes's amputation at the ankle-joint. Some months later tuberculous osteitis manifested itself at the end of the tibia, and in October, 1896, Stephen Smith's amputation was performed at the knee joint. The stump never properly healed, and, becoming the site of another tuberculous deposit, she was re-admitted into hospital in May, 1897, and on the 29th of that month I removed the femur at the hip-joint. Healing was extremely protracted, and it was six months before the stump was sound. She was then lost sight of for some time, but she re-appeared at the hospital in April, 1899. The left foot and ankle were found to be much swollen and œdematous and there were signs of fluctuation on the outer side of the ankle-joint. On the lower third of the leg was a large tuberculous ulcer which had existed for three months; the knee-joint was filled with fluid and presented the usual symptoms of tuberculous disease. In the middle of the quadriceps extensor was a large caseating mass of the size of a clenched fist. The patient was emaciated and hectic. After a consultation with my colleague, Mr. Haslam, it was deemed desirable on April 20th to remove the left limb at the hip-joint. The healing of the stump was again protracted, but, as you now see, only two minute spots at the top of the external incision remain unhealed. The general health of the patient seems good, she has gained flesh and she takes her food and digests it well. At present there are no signs of any tuberculous deposit in any of the other joints of the body or in the internal organs.

I have now performed 15 amputations at the hip-joint upon 14 different individuals and herewith is presented in tabular form the sex and age of each patient, the disease for which the operation was undertaken, the plans adopted for restraining the hæmorrhage, the method of operating, and the results. On looking at the table you will see that in the series only two methods of operating have been practised. In the first anterior and posterior skin-flaps have been shaped from without inwards, and the muscles and vessels divided high up as close as possible to the pelvic bones by a circular incision. This method has been adopted in three cases (Cases 1, 2, and 9), each time with success, and theoretically it is the best method to be adopted when a malignant neoplasm has to be dealt with. Its drawbacks are that it undoubtedly increases somewhat the shock attendant upon the operation and the high division of the muscles necessitates the direct compression of the aorta for the restraint of hæmorrhage.

The second method is for practical purposes that known as the external racket operation and is really a combination of the vertical and circular incisions. The names of many eminent surgeons are associated with this plan of operation, notably, Furneaux Jordan, Lister, Esmarch, &c.; but I have preferred to speak of it as the external racket method as thereby the general principle of the operation is more likely to be impressed upon your minds than if it be associated with the name of any distinguished surgeon of the present or past generation. The precise steps will differ somewhat in a case in which the operation is undertaken for malignant disease from those taken when it is performed for the disease