

seen by Patrick Manson some time ago in London, by Marmorek of the Institut Pasteur in Paris, and were finally presented by Mense at the last Colonial Congress assembled in Berlin.<sup>8</sup> How can it be suggested that the bacteria so often isolated by us in pure culture may not be the same as the one seen in the sections and exudates, the identical morphology being given? and how can the germ isolated by Castellani from the same regions of the body where we obtained it during life and post mortem, not be the same we saw in our microscopical sections?

After all this there is not the slightest doubt that Castellani's bacterium is the one which we described and that his investigations are a complete confirmation of ours. This is the original and important fact. The priority in the question is relatively a secondary one, as no one can honestly and *sine ira* contest that it does not belong to us; but the works of this member of the English Commission, done quite independently of ours and in quite different and distant regions, do not in the least lose their merit or the important signification they bear for the clearing up of the etiology of sleeping sickness. It is, in fact, the first time that etiological researches of the African lethargy have been thoroughly verified and identical results arrived at.<sup>9</sup>

## ON A CASE OF ARTERIAL OCCLUSION AND GANGRENE.

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IN November, 1899, I was consulted by a retired officer of the army on account of pain in the left leg and foot. He was a spare, active man, in his fifty-third year, with a complexion ruddy from enlarged capillaries and with the skin of the hands also of a high colour. In India, when a young man, he suffered from malarial fever and dysentery and he also had dysentery while in Egypt in 1882. Of late years he had been liable to dyspepsia, and in 1898 he had tobacco amblyopia which afterwards improved. Some time in the summer of 1899, I think, I had attended him for painful redness and swelling affecting the ball of the left great toe and running up the dorsum of the foot between the first and second metatarsal bones. The pain which he complained of when I saw him in November was intermittent, occurring in paroxysms which often came on in the early hours of the morning. It ran down the front of the leg and along the dorsum of the foot and was felt in the ball of the big toe and the distal part of the foot. The foot was sometimes dark red and congested, when it felt hot, and sometimes it was pale and cold, conditions resembling the syncopal and asphyxial states of Raynaud's disease. Stiffness and some pain behind the knee-joint were also complained of. The heart, though perhaps a little weak, was perfectly normal and there were no indications of atheroma or arterio-sclerosis in any of the accessible arteries. The urine was free from albumin and sugar and in all respects normal. The lungs were healthy. The tongue was somewhat furred and there were some dyspeptic symptoms but the abdominal organs were otherwise healthy. The temperature was normal. Later, pulsation was observed to have ceased in the posterior tibial artery and the dorsal artery of the foot; the pains became more frequent and severe and the varying conditions of heat with dusky redness and coldness with pallor became more marked. Pains of a slighter degree were also felt in the right leg and down the arms, and the right foot also showed some signs of varying congestion and pallor, as also did the hands.

In the spring of 1900 he improved somewhat for a time, but about May he got worse again and a black spot formed on the under part of the distal phalanx of the great toe and another on the tip of the little toe. The extreme tip of the little toe gangrened and the process there made no further

advance but it slowly went on in the big toe till it was entirely black and mummified. A distinct line of demarcation, well beyond the joint, formed in, I think, the month of July, but it made one or two advances during the next month or two. In September there was a good deal of constitutional disturbance due, apparently, to septic absorption, the temperature rose, and he had a severe attack of aphthous stomatitis. At the beginning of October, as the line of demarcation was making no further advance, Mr. A. E. Hind removed the gangrened toe by cutting through the first phalanx where the separation had taken place down to the bone, just beyond the metatarso-phalangeal joint. After the operation the patient improved rapidly and in a month or two the wound had healed and he was able to move about again.

From the end of 1900 to the spring of 1902 the patient was in excellent health, though he had occasional pains in the legs. About the month of April he consulted me on account of occasional attacks of sudden and severe pain at the bottom of the sternum and over the præcordial region, seizures which had very much an anginal character. A little later he had pain running down the sciatic nerve on the right side, and some time in May he began to have sharp attacks of pain running down the front of the right leg, the paroxysms very frequently occurring in the early morning as they had done in the other leg. In June the right foot was frequently, indeed mostly, hot and congested, and sometimes cold and pale, and soon no pulsation could be felt in the posterior tibial or in the dorsalis artery. About the end of the month two black spots appeared on the sole of the foot, one on the heel and the other on the under surface of the little toe, just on the bend of the first joint. In July suppuration occurred at the seat of the black spot under the toe and some matter was let out by a small incision. In August unhealthy-looking ulceration began to extend from this point, gradually spreading two or three inches up the outer side of the foot, and the toe became purple and gangrene set in. By the middle of the month the toe and the ulcerated part of the foot were completely gangrened and a line of demarcation had begun to form; but the next toe was evidently becoming implicated. All this time the patient's condition remained good. The temperature was normal or only slightly raised sometimes. The pulse was good but occasionally quick and not very strong. The urine was normal.

About the middle of September the fourth toe was gangrenous and the line of demarcation was advancing onwards and upwards. The constitution was now showing the effects of the disease. The temperature was frequently raised, though not often above 100° F., and at the end of the month there was a rather sharp attack of aphthous inflammation of the mouth and throat, as during his last illness. There was also an aphthous ulcer on the conjunctiva of the left eye which gave rise to inflammation. During all this time the pain in the leg and foot was frequent, almost constant, and often very severe.

On Oct. 4th, as the line of demarcation seemed at last to be stationary, the gangrened parts were removed by Mr. Hind, who cut through the fourth metatarsal bone and disarticulated the fifth at its tarsal articulation. The raw surface left looked pale and bloodless. About three weeks later the second and third toes had become gangrenous and a line of demarcation had formed on the dorsum of the foot which included them, though it was not developed on the under surface. The great toe had also a dry gangrenous patch on the tip and the nail had been removed owing to suppuration in the matrix. It was then decided that the dead and dying parts should be removed, and Mr. Hind, assisted by Dr. J. R. Muir, R.N., separated them by disarticulating the remaining four metatarsal bones at their tarsal articulation and making a flap of the sound skin of the sole to cover the wound made at this operation and the still incompletely healed one left by the last.

For a day or two after the operation the temperature rose in the evening to 102° and there was some general constitutional disturbance, but great improvement of the general condition soon set in. The mouth, which was still sore, got better, the inflammation in the eye gradually subsided, and an ulcer on the back part of the heel which had given some trouble began to mend. A week or ten days after the operation a part of the flap which was stretched over the internal cuneiform bone sloughed and the bone protruded through the opening. The projecting part became necrosed and on Dec. 1st it was found to be loose and was easily

<sup>8</sup> Archiv für Schiffs- und Tropen-Hygiene, Band vii., 1.

<sup>9</sup> It is convenient to correct here a mistake in the English translation of our first report. It was said that we isolated constantly the diplococcus from the blood. This is absolutely incorrect. What we clearly confirmed was that in four cases examined in this point of view we only found it once. To-day we can add that we have had two positive results in seven cases. Castellani only isolated it once. We have here another point in which Castellani's researches agree with ours.

removed, leaving a vigorously granulating surface behind it. After the operation the attacks of pain still continued but they became less frequent and severe. There was also occasional pain in the left leg and foot and for a time the sole of the foot was sometimes congested and occasionally the foot was pallid. Pain was also complained of in the left arm and the fingers of both hands became bloodless now and then. By the end of January the wound had completely healed, though there was still some discharge coming from under the flap, and the patient was looking well and beginning to get about.

At about the middle of February, however, an apparent obstruction of the right femoral vein occurred. There was pain in the groin, the stump of the foot became swollen and purple, and there were paroxysms of severe pain in it. The whole leg was cedematous and the protruding bone in the stump became inflamed and bare. A fortnight later a similar attack took place on the left side and it was over a month before both legs had recovered and even then there were frequent pain and great tenderness in the stump of the right foot. The patient's health meanwhile improved greatly and by the middle of April the disease seemed to have entered upon another period of quiescence, though the foot still continued to give pain.

Though a description of the condition of the affected arteries cannot be given—fortunately for the patient—and though perhaps the possibility of slow-forming thrombus in atheromatous or sclerosed vessels cannot be quite excluded, this case has all the appearance of one of Friedländer's arteritis obliterans.

The heart was normal, the urine was free from albumin and sugar, and there was no evidence of atheroma or sclerosis in any of the accessible arteries. The obstruction could not have been caused by embolism, the lapse of time between the commencement of the symptoms and the occurrence of gangrene was too great. The arterial occlusion was evidently a gradual one. In this connexion the varying congestion and pallor of the feet are interesting, as they recall the symptoms in Raynaud's disease. This was most marked in the first attack; in the second attack the right foot was mostly congested and hot. The pains, from their severity and mode of occurrence, often led me to suspect neuritis, though there was perhaps not more wasting of the leg muscles than could be accounted for by disuse of the limbs and I did not notice any abnormalities of cutaneous sensation. The angoral attacks are also of interest when it is remembered that in specific cerebral arteritis—a typical obliterative arteritis—such attacks have been noticed and found to be due to affection of the coronary arteries. In this case, however, the attacks lasted for only a short time and did not recur, so they were probably of a purely neuralgic character. There were also pains in the arms, mostly the left arm, and the fingers were often dead and pale and the hands congested.

Friedländer<sup>1</sup> in his paper gives no clinical account of the disease; he confines himself to a description of its pathology and morbid anatomy. The morbid process begins by inflammatory cell growth in the inner coat which is thickened at the expense of the lumen of the artery. The other coats become affected and subsequently connective tissue formation sets in and the vessel is transformed into a fibrous cord. Complete obstruction is sometimes delayed by the continuance of the first, non-fibrous, stage in which the artery may not be completely blocked by the yet soft granular tissue, or occlusion may be hastened by the formation of thrombi in the partially obstructed vessel. The veins may also be affected. Friedländer says that obliterative arteritis is not so often primary as secondary to disease of the surrounding tissues.

Von Winiwarter<sup>2</sup> relates a case closely resembling that of my patient. The affected foot was amputated about four inches above the malleoli and he gives a most careful and detailed account of his examination of the parts that had been removed, pointing out that the morbid changes in the vessels were those previously described by Friedländer as arteritis obliterans. The veins were affected as well as the arteries and he also found that the posterior tibial nerve and its branches were involved and nearly doubled in thickness.

Dr. Prioleau in his Thèse (Paris, 1887) on "Rétrécissement Généralisé des Artères" does not seem to differentiate obliterative arteritis from arterio-sclerosis, but amongst his

cases are some which resemble that of my patient and he describes morbid appearances similar to those recorded by Friedländer and von Winiwarter.

Dr. F. W. Mott has an excellent article on obliterative arteritis in vol. vi. of "Allbutt's System of Medicine," and both Dr. Prioleau and Dr. Mott give a full list of references, only one or two of which, however, I have had the opportunity of consulting.

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## A FURTHER NOTE ON THE THERAPEUTICS OF A 10 PER CENT. SOLUTION OF SODIUM CINNAMATE IN GLYCERINE INJECTED SUBCUTANEOUSLY.

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IN the preliminary note published in THE LANCET of July 12th, 1902, p. 66, the groundwork of the treatment was briefly mentioned. Surprise has been expressed that a drug could profoundly alter the conditions of two diseases which are apparently so dissimilar in every respect as cancer and tuberculosis. However, there are points connected with them which appear to lead in the direction of the thought that, whatever dissimilarity there may be in their external manifestations, the conditions which precede the alterations in the structure of the cells which are affected are alike in type and that the general conditions of health in patients diseased either in the one or in the other way are equally alike in type. The difference in the alteration in structure is in detail, profound as that difference is. The difference in the general conditions of health is equally in detail, but not so fundamentally. In both diseases there are conditions intimately connected with the cells attacked which precede the conditions of actual disease and in both the results of the disease are manifested by the condition known as cachexia, varying in details even amongst those smitten by the taint of the two diseases. It is necessary, therefore, in the case of both diseases to consider very carefully the conditions affecting the cells themselves before they are attacked by the one or the other disease. Every cell, however low in the scale of organisation, has certain properties of its own, functional as well as structural. These are properties which may be designated as inherited properties in the case of some, acquired properties in the case of others. It is impossible to state that in cells affected by these diseases there is any evidence to show that both properties do not exist, and, indeed, it may be stated quite definitely that in the absence of inherited properties those acquired are only with difficulty obtained. If this be accepted there can be little difficulty in finding the reason for the many and great differences not only in the structural alterations noted in both diseases but also in the progress and clinical history of them. Varied as are the properties inherited by cells much more varied are their external conditions and surroundings. The variations which can be produced and perpetuated in many living cells are well known not only to the scientist but also to the stockbreeder and the gardener, although the latter know them in bulk, the former in sample. With the differentiation of the bacillus of tubercle and the demonstration of its connexion with the causation of the structural alterations in the cells which it attacked an important phase of knowledge was undoubtedly opened and so far as the improvement in the treatment of the disease goes there has resulted something. However, it never appeared to me that there was reality in that improvement. Under various treatments and under no treatment at all many patients become well—that is to say, that the masses of cells which had been attacked by the disease became cured in the manner adopted by nature.

The main lines upon which treatment has been generally based have been the improvement of the general condition of health by providing to the blood circulating in the vessels an abundant supply of oxygen and of nourishing food. It has been difficult in most cases, if not all, to say whether the drugs usually administered had any good effect, and still more difficult to find a satisfactory physiological reason for

<sup>1</sup> Ueber Arteritis Obliterans, Centralblatt für die Medicinischen Wissenschaften, No. 4, 1876.

<sup>2</sup> Ueber eine eigenthümliche Form von Endarteritis und Endophlebitis mit Gangrän des Fusses, Archiv für Klinische Chirurgie, No. 1, Band xxiii., 1878.