ANTHRAX AND FATALISM.

BY

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ACCORDING to Bacon "the chief obstacles in the way of truth (or causes of error) are four—namely, the plea of an authority that does not deserve the name, long-standing habit, popular prejudice, and an ingrained ignorance that masquerades as so much knowledge." I quote this in connexion with the attitude of most medical men towards internal anthrax or pulmonary anthrax, commonly known as woolsorters' disease; and I wonder why an attitude of fatalism should be reserved for this particular microbic disease and why we should refuse to believe that it is governed by the same biological laws as govern other germ diseases. The prevalent attitude may be thus stated: "If a patient has got internal anthrax that patient must of necessity die; if he does not die, it is not internal anthrax." Giving evidence at an inquest at Shipley last autumn my friend and colleague, Dr. Eurich, Bacteriologist to the Bradford Anthrax Investigation Board, stated "there was not a single case of lung anthrax known to have been cured by injections of any kind."

To my mind in regard to this disease we are reasoning in a circle. We know in undoubted cases of internal anthrax—proved to be such both *ante mortem* and *post mortem*—that bacteriological examination of blood and pleural and pericardial effusions rarely discovers anthrax bacilli more than twenty-four to thirty-six hours before death. Therefore it follows that if these fluids had been examined, say, forty-eight to sixty hours before death the examination would have been negative, and had the patient from the time of that examination begun to improve and eventually recovered, then we should have been placed in the absurd position of maintaining that it could not have been anthrax because we did not find any anthrax bacilli; and (again arguing in a circle) if it had been anthrax the patient would have undoubtedly died. I am quite willing to admit that when the disease has advanced so far that examination of the blood and fluids prove the presence of anthrax bacilli then the patient is beyond hope of recovery and will die. But I hold that until that fatal stage is reached we are justified in making a diagnosis from clinical symptoms alone, just as we do in many other diseases probably microbial, in which the bacteriologist has not been able to help us even to the extent of finding and isolating the germ at any stage of these diseases.

If, then, we can diagnose internal anthrax by symptoms alone, before the fatal stage where the bacilli may be demonstrated, and apply suitable treatment, say by Sclavo's or other anti-anthrax serum, and so bring about recovery; it follows that, bacteriologically, we are then unable to *prove* that the disease was anthrax. To continue the reasoning a little further, all who have had considerable experience of anthrax know that external or cutaneous anthrax, if subjected to no treatment whatever, will by the natural resistance of the body in many cases undergo spontaneous cure. Why, then, may not the same thing occur if the area that is infected primarily is situated in the lung or alimentary tract? We say, "because these local lesions have very seldom been found *post mortem* in the lungs and intestines in cases that have died." But the reason why we have not found these internal local infections clearly is that many have recovered and that those who have died have died of a secondary general infection, and at not one *post-mortem* examination in twenty is any real attempt made to locate the original seat of inoculation. In most cases it would not be practicable to do so, all the pulmonary tissues being altered by the conditions due to the general infection.

During the last half-dozen years, while acting as medical adviser with regard to anthrax for twenty-four large woolcombing firms, forming about 90 per cent. of the woolcombing industry in Bradford and district, I have had the opportunity of examining hundreds of employees who have been off work through illness; many of these cases being suspicious, I have treated with Sclavo's serum with good results. The following notes of one of the best examples of the kind I have had were published in the

annual report of the Bradford Medico-Chirurgical Society in 1906:

T. H., aged 53, a finisher, working amongst brown Persian and other dangerous wools, began to feel ill on January 16th, in the evening, was seen by me on the morning of January 19th; was then complaining of "shivering," cough, dyspnoea, nausea, headache, and vertigo; pulse 74, regular but small; temperature, mouth 100.4°, rectum 103.0°; respirations 30. No external sores. Examination showed diminished respiration over whole of right lung. Heart sounds normal. As I considered the case distinctly suspicious of anthrax I gave 40 c.cm. of Sclavo's serum hypodermically under the skin of the abdomen. At 11.30 the same night, pulse 120; rectal temperature 103.4°; respirations 33. Breath sounds puerile over right apex in front. Patient refused more serum that day.

January 20th.—Pulse 104; temperature, rectum, 102.4°; respirations 26. Auscultation gave same results as before; 40 c.cm. more serum given. Bacteriological blood examination for anthrax bacilli gave negative results.

January 21st.—Pulse 85; temperature, rectum, 100°; respirations 21. Breath sounds over right apex not so harsh as before, but some small sibilant rhonchi present.

January 22nd.—12 noon: Pulse 96, weak; respirations 34; temperature, rectum, 102° F.; some lividity of dependent parts. Four inches of fluid in right chest; about 1 in. in left side (took some for bacteriological examination—absolutely sterile); skin cold and clammy. Gave intravenous injections in median basilic vein of 40 c.cm. serum. At 11 p.m.: Pulse 90; temperature 101.6° F.; respirations 38. Patient feels better and is a better colour and skin warmer.

January 23rd.—Pulse 100, regular; respirations 38; temperature, rectum, 100.4° F. Fluid in both pleural cavities increasing.

January 24th.—11 a.m.: Pulse 84, weak and irregular (heart beats 100); respirations 28, laboured; temperature, rectum 100°, mouth 95.6° F. Superficial cardiac dullness absent. Pleural effusion still increasing. Rhonchi over both apices in front. Cyanosis of hands and feet. Gave 30 c.cm. in loin. 10 p.m.: Pulse 102, regular and stronger; respirations 28; temperature, rectum, 100.6° F. Looks bright and feels better. Aspirated 42 oz. blood-stained fluid from right chest. (Bacteriological examination of this fluid negative.)

January 25th.—Pulse 105; temperature, rectum, 100.6°; respirations 34. Fluid still in both cavities. Took some from left side—free from blood; sterile. Gave 40 c.cm. more serum, hypodermically.

January 26th, 11 a.m.—Pulse 106, small, weak, and irregular; could only count by auscultating the heart. Temperature, rectum, 100.4°; respirations, 28. Hands and feet cyanosed. 40 c.cm. serum *by mouth*; 15 minims liq. strych. hypodermically. 5 p.m.: Pulse 100, very small; respirations 24; temperature, rectum, 99°; looks better; 15 minims liq. strych. hypodermically.

January 27th.—Pulse 90, much stronger; temperature, rectum, 99.2°; respirations 15, full and deep (was sleeping, first time for some days); cyanosis of hands and feet less marked. Still fluid in both sides of chest. Bacteriological examination of specimen from right side negative.

January 28th.—Pulse 95, quite regular and much fuller; respirations 22; temperature, rectum, 98.6°; slept two hours. Cyanosis almost gone. Slight diminution of effusion. For the next three days improvement was steady, except that fluid increased in right chest and diminished in left. On February 4th I aspirated 70 oz. from right side—blood-stained, sterile.

From that date recovery was uninterrupted.

The principal features of the case were cardiac feebleness, cyanosis, double pleural effusion, the usual prodromal symptoms, headache, vomiting, and rigors. I based my diagnosis on these symptoms occurring in a man engaged in handling highly dangerous wools, where such symptoms, in the absence of pleuritic friction or pain, pneumonia, tuberculosis, cardiac or renal disease, could not be well explained by anything else. The injections of Sclavo's serum were in nearly every instance followed by a fall of temperature and an increased feeling of comfort and well-being. In all 230 c.cm. of serum were given.

I write this in the hope of combating the feeling of fatalism which attends this disease, and of urging a more generous use of the anti-anthrax serum—to give it, in fact, a "sporting chance." I have used it in many cases, and have seen serum fever and skin eruptions in only a few. They occur about the ninth day, and are accompanied by joint pains.

Since writing the above, some months ago, my ideas have received support from the last report of the Bradford Anthrax Investigation Board. In that report the following passage occurs:

Last year it was pointed out that of the 13 patients of whom the length of employment was ascertained, 5 had handled scheduled materials for only six months or less. The figures this year are still more remarkable, 6 out of the 10 having been employed only for six months or less continuously amongst scheduled materials. It would seem as though length of employment amongst scheduled materials gave immunity

from anthrax to some workers, or at any rate made them less susceptible to an attack of this special form of blood-poisoning.

Now I know of only one way in which immunity against, or diminished susceptibility to, a specific contagion can be accidentally acquired—namely, by contracting the specific disease in question. If, therefore, the above extract has any meaning at all, it can only mean that these wool-workers acquire slight forms of anthrax which give them immunity; in other words, that the views I have been inculcating for the last half-dozen years are supported by the statistics of the incidence of anthrax.

Messrs. F. M. Rimmington and Son, Bridge Street, Bradford, keep a constant supply of Selavo's serum.

THE INFECTIVE GRANULE IN CERTAIN PROTOZOAL INFECTIONS, AS ILLUSTRATED BY THE SPIROCHAETOSIS OF SUDANESE FOWLS.

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PRELIMINARY NOTE.

At the first meeting of the Tropical Medicine Section of the British Medical Association in London last year I advanced the view that, in all probability, what might be called the "infective granule" would yet be found to play an important part in certain protozoal infections, and more especially in spirochaetosis and trypanosomiasis. I based this belief on the work of Leishman as regards the changes undergone by *Spirochaeta duttoni* in *Ornithodoros moubata*, and on the allied changes which I had found to occur in the Sudan fowl spirochaete when ingested by *Argas persicus*. I have been continuing the work on fowl spirochaetosis and have recently arrived at some most interesting and significant results, which may yet have considerable bearing on the view we must take of the pathology of this and other spirochaetal diseases, and possibly also on their treatment.

The full account of these later researches will be presented in the fourth report of these laboratories, which is now in the press, and is due to appear in the autumn of the present year; here I wish merely to place on record a few of the more salient features of the work.

It will perhaps be remembered that one found intracorporeal forms in this fowl spirochaetosis, and that, following Sambon, one had come to the conclusion that these endoglobular bodies represented a stage in the life-cycle of the spirochaete—constituted, in short, its stage of schizogony in the fowl. Sambon, however, who expressed this view from the study of a few slides I gave him, did not indicate how this red cell invasion occurred. For a long time I believed the spirochaetes themselves entered the red cells and broke up, or coiled up, within them to form these remarkable bodies. As the parasites can and do enter and leave the erythroblasts of the fowl, there was good ground for this supposition. Now, however, I know better.

By the use of the dark-field method, and more especially by practising liver puncture on chicks at the crisis or on chicks which have been given a sufficiently large dose of salvarsan, I have found that in the liver in particular, also in the spleen and lung, the spirochaetes undergo an astonishing change. They discharge from their periplastic sheaths spherical granules, and it is apparently these granules which enter the red cells, develop in them and complete a cycle of schizogony. The appearance is very remarkable. If a well-infected chick be given a dose of salvarsan, the peripheral blood is soon cleared, or nearly cleared, of spirochaetes. If then a drop of liver juice be examined by the dark-field method, it will be found swarming with spirochaetes and with highly refractile granules. The source of the latter is soon apparent, for attention will be directed to spirochaetes which are not moving in the usual way, but are in a state of violent contortion, or are, so to speak, shaking themselves to and fro. Indeed, I cannot give a more apt comparison than by likening their movements to those of dogs which have

been in water and are shaking themselves vigorously to dry their coats. The object of the spirochaetes, however, is to rid themselves of the bright, spherical granules which can be seen within them, and which may or may not be aggregations of the so-called chromatin core. These are forced along the periplastic sheath and suddenly discharged, so that they become free in the medium and dance hither and thither as tiny, solid, spherical, brilliantly white particles. In process of time the spirochaete loses its activity, becomes difficult to see, and eventually all that is left of it is the limp and lifeless sheath drifting aimlessly in the fluid and liable to be caught up and swept away by some still vigorous parasite. Such a sheath may still retain one or two of the granules which it has been unable to discharge.

As may be imagined, the process is most fascinating to watch, and my observations have been confirmed by Captain Fry and Mr. Buchanan, of these laboratories, and by Captain O'Farrell, R.A.M.C. I may also say that the first-named had previously seen a shedding-off of granules by trypanosomes in the peripheral blood of experimental animals, a phenomenon which he is now studying.

It is these spirochaete granules in the liver, spleen, and lung, and possibly also in other internal organs, which, I believe, invade the red cells. I think I have seen the penetration occur, but require to make further observations in order to be certain as to the mode of entry. Such a chain of events fully explains all the puzzling features which this intracorporeal infection has hitherto presented, and, moreover, brings it into line with the infective granules found in the ticks, for these very closely resemble those seen in liver juice films both when examined by the dark-field method and when stained by the Levaditi process. There are various other points, more especially as regards the peculiar staining reactions of these granules, into which I need not enter beyond saying that the fact that, when free, they do not appear to take on the Romanowsky stain may explain why they have not previously been noticed. The work is also not yet complete, as it is necessary to find out if the spirochaetes ingested by ticks behave in a similar manner and thereby produce the granules of Leishman.

I see that Jowett in South Africa has recently discovered what appears to be an identical form of fowl spirochaetosis, and I trust he will employ the dark-field method and endeavour by liver puncture and the use of salvarsan, for the purpose of creating an artificial crisis, to follow out the curious cycle I have indicated.

From these observations and others which will be fully detailed at a later date I have come to the conclusion that this fowl spirochaete must be classed as a specific entity, and I am proposing for it the name *Spirochaeta granulosa penetrans*, which, though lengthy, suitably indicates its more important peculiarities. At the same time it is quite possible—nay, even probable—that other pathogenic spirochaetes behave in a similar manner. I have found these granules to be resistant forms, and their presence in countless numbers in the tissues might explain part of the mechanism of relapse and the difficulty of curing completely some of the more chronic spirochaetal infections, as, for example, syphilis and yaws.

In conclusion, I must thank Professor Ehrlich for most kindly placing at my disposal an ample supply of his new and valuable remedy.

AN Anticholera Congress has recently been held at St. Petersburg, under the presidency of the Minister of the Interior.

WE are informed that over 100,000 copies of the leaflets on "clean milk" published by the National League for Physical Education and Improvement (4, Tavistock Square, London, W.C.) have been distributed within the last few months mainly by officials connected with health authorities, and also through national and local health societies. The leaflets specially addressed to the householder have been supplied to many education authorities for distribution to the children when lessons on domestic hygiene are being given and for the instruction of the teachers themselves. The league is no doubt well advised in trying to influence the rising generation, and we are glad to congratulate it on the evidence of the good progress made in its useful campaign.