apparently non-pathogenic, had later on acute dysentery with an amoeba to all appearance the very same.

Typical Cases.

There are few records at present of Malay cases, but the following from our case-book may show that other nationalities suggest that the same amoeba may be pathogenic at one time and apparently non-pathogenic at another.

1. S., Malay boy, aged 16 years, Sejjak. Admitted July 7th, 1914, for poisoned wound on leg. Amoebae found July 12th, 1914. No symptoms of dysentery. History showed that he had had acute dysentery when about 6 years old. He then had blood in stools, and became wasted. His mother had dysentery three months ago.

2. N. K., Tamil girl, about 11 years. Admitted February 27th, 1913, as a visitor with mother and younger brother, who had acute dysentery. She was not ill, had no diarrhoea, no colic, nor blood in stool. She had ankylostoma, mounds, and amoeba, which persisted till May 18th, 1913. Amoebae were found moving about at the examinations on forty-seven occasions, although twelve ½-grain injections of emetine were given, and salol, beta naphthol, quinine, and irrigation were tried. She was readmitted on July 3rd, 1914, with acute dysentery, severe colic, and blood in stools. Amoeba of the same species was found daily until the 10th, when she was discharged well after emetine and saline. When a carrier, I could not see any difference between her amoebae and those of her brother, nor did the amoeba she had at first differ from that she had when acutely attacked.

3. M., Tamil woman, about 20. Admitted December 16th, 1912. Diarrhoea, blood in stools, and acute colic. Amoeba found. Emetine and saline given. Discharged well December 26th, 1912. Readmitted January 23rd, 1913. Acute dysentery as before. Amoeba found daily up to February 18th, 1913. Discharged February 23rd, 1913. Again admitted July 3rd, 1914. Acute dysentery as before. Amoeba found. Discharged July 10th, 1914.

4. A., Tamil woman, about 25. Admitted January 31st, 1913, with bronchitis, but stools found to contain amoebae and active rotifers. No diarrhoea, nor blood in stools. Readmitted November 6th, 1913, with acute dysentery, severe abdominal pain. Amoeba found as before, also ankylostoma and whip-worm. After treatment with emetine and saline, amoebae ceased to appear on December 1st, 1913. Discharged December 3rd, 1913. Again admitted April 4th, 1914, with otorrhoea. Amoeba found, also ankylostoma. No diarrhoea nor other intestinal symptoms. Amoebae persisted till April 11th, 1914, when discharged. Admitted fourth time July 2nd, 1914, with acute dysentery, diarrhoea, severe colic, and rectitis. After emetine and saline treatment no amoeba found on July 10th, 1914. Discharged July 13th, 1914.

5. K., Javanese man, about 35, a coolie who had not been in hospital for seven years previously. He had cut his shin with his parang, and severe sepsis had set in, with a large abscess as the result. Admitted June 24th, 1914. Amoeba was found on June 26th, 1914. Patient had had no diarrhoea nor colic, but on July 7th, 1914, acute dysentery began. He had emetine twice, but refused further injections. Saline was given freely and he recovered, but amoebae were still found. Gingivitis and stomatitis became very acute, but, though very weak for many days, he is now gaining strength.

6. S., Tamil female. Admitted October 10th, 1913, with acute dysentery. Amoebae found till the 12th. Discharged October 17th, 1913. Readmitted on January 6th, 1914, with pleurisy. Amoebae found, no diarrhoea, no blood, no colic. Amoebae found up to date of discharge.

7. S., Javanese man. Arrived from Java May 30th, 1914. He looked very weak and thin, with prominent eyes and a slight cough. He had ankylostoma, round-worm, whip-worm, and Trichomonas. He absconded on June 21st, 1914, and was caught after wandering about without proper food or shelter for five days. He was sent from the estate to hospital on July 13th, 1914, with acute dysentery. Amoeba was found, also blood corpuscles and broken-down epithelial cells. He died on July 27th, 1914. The post-mortem examination showed that 11 ft. of the lower bowel and ileum were ulcerated. The lower part of the colon was soft and the mucous membrane appeared to be melting away. The serous coat was exposed in places. The lower ileum was much injected and ulcerated. The mesenteric glands were enlarged and yellow, almost caseous. There were purulent nodules in the lungs; the pleurae were not adherent. This case shows the ravages of dysentery when debility and starvation, with concurrent disease, have their effect.

8. M., Javanese woman. Admitted November 8th, 1914. She died in a few hours of acute dysentery. She had been delivered of a 7-months child the day before, and had had haemorrhage. Probably latent dysentery had pre-existed.

Conclusions.

(1) That amoebic dysentery is endemic among the Malays of the Sarawak River; that some immunity seems to exist against very severe attacks or epidemics, but that some educational and remedial campaign would be beneficial. (2) If Case 1 as a carrier had a non-pathogenic amoeba, why did he not get a fresh

infection from the acute attack of dysentery to which his mother had been subject? The suggestion is that both amoebae are alike. (3) If in Case 2 the girl carrier had a non-pathogenic amoeba, why not at once get infected with the pathogenic one of her brother? As no difference could be seen, the suggestion is that both amoebae were alike, one acting in a latent way till able to produce severe symptoms at a favourable time. (4) Case 4 leads to the conclusion that a person may have latent dysentery alternating with acute, and no difference be seen between the amoebae. (5) Case 5 would indicate that amoebiasis caused acute dysentery when cocci were in the blood and the resistance was low. (6) Case 6 indicates amoebiasis following acute dysentery, and the amoebae appeared the same as during the acute stage. (7) Cases 7 and 8 show the virulence of the amoeba, latent at first, after exposure, starvation, and tubercle in the one case, and after haemorrhage and childbirth in the other. (8) Although the picture seems dark, yet ordinary hygienic cleanliness, and care, with freedom from intimate association with natives, will usually protect Europeans from danger of death from amoebic dysentery.

THE "ELASTIC TISSUE" ENIGMA:

A SUGGESTION FOR ITS POST-MORTEM STUDY,
IN ARTERIES KEPT PERMANENTLY ELASTIC, AND IN
THE "ROOSEVELT ELASTIC LUNG."

BY

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I. Its Biotics.

THE natural history of the elastic fibre is a standing problem which the inferior elasticity of indiarubber does not help us to solve. We perceive how it is destroyed in the lung by overstrain in emphysema and by disuse in obesity, and how readily it is then disposed of by phagocytosis. The mystery is its surprising normal "endurance." This would almost seem to be a function of its mechanical exercise, even more than of its nutrition. Facts have been recorded which demonstrate a patchy local "regeneration." But of its "alimentation" we know nothing, except that the fibre can be stained. And yet, if we assume that it grows, like the length and breadth of our muscular and of our tendinous fibres, it should be, at any rate at that stage, receptive of nourishment. On the other hand, the following facts would suggest that, when full grown, its vital exchanges are of a minimal order; and its viability and function largely dependent upon motile and hygrometric factors, whilst apparently independent of direct innervation.

The work of Alexis Carrel has opened up a new era in surgery—that of transplantation—by demonstrating the persistence, for appreciable periods after somatic death, of some degree of "vegetative" cellular vitality in the simple tissues, rendering them capable of a revival of their nutritional activity when implanted into vigorous living tissues, and also the facultative prolongation by low temperatures of that suspended animation; nay, more, the persistence, after successful transplantation, of the functional capacity of some of the organs, when "exsomatized" *in vivo*.

The lung is hitherto the isolated instance of a dead organ capable of preservation for indefinite periods in a state of fitness for discharging its "passive" motile function almost as well as during life, thanks to the survival of some degree of elasticity in all its textures. It affords the unique example of one of our tissues, the elastic fibre, preserving indefinitely, without any vital nutrition, the fullness of its own simple function, which seems to be limited to passive movement. I might venture to suggest that this strange instance of a relative "artificial immortality of function" should be associated with the name of the distinguished physician, my lamented friend the late John West Roosevelt of New York, to whom we owe its demonstration.

The only published account of his method which I can trace is that which he gave to the New York Pathological Society, of which he was then president, at its meeting of December 10th, 1890. In his own words, "The lung exhibited was removed on January 11th, 1890. It retains very nearly the normal colour of a freshly removed lung.

It expands and contracts in an apparently normal manner. The vesicular murmur can be plainly heard in it, and percussion gives the same note as obtained from fresh lungs. The process will be seen to be a modification of the old glycerine-carbolic method, well known and often used to preserve anatomical specimens in an elastic condition. The modifications are important, however; and the results far better than any obtained in the old way. The idea of the method is, first, to fix the tissues by the chromic and acetic acid solution, then to introduce the glycerine-carbolic solution, and, finally, to get rid of this, and practically to make an 'oil-tanned' lung."

The composition of the three solutions and the mode of using them are then given. It does not appear that, at that time, he had prepared any specimen of both lungs, or that he subsequently exhibited, or published any description of, any such. That which he prepared (by his new method) to present to me at New York in the spring of 1892 may perhaps have been the first. These lungs were slightly pathological. They did not remain in efficient working much more than a year: partly, perhaps, owing to insufficient exercising. Meanwhile, in the early summer of 1892, I prepared, according to his latest directions, a perfectly normal and complete specimen of both lungs with trachea. This was a complete success. It was long used for demonstration at St. George's Hospital; and it still preserves its elasticity after the lapse of nearly twenty-three years. Unhappily, through a preventable accident, it has quite recently developed a bad "puncture" which now disables it, though it is still inflatable, for any complete and lasting inflation. That long record, and the fear lest, owing to his untimely death, the value of his latest method might otherwise be lost to science, make me anxious to publish those simple directions which enabled me to secure that success, without the help of any actual demonstration of his technique, or of any knowledge of the published details of his earlier method.

"Improved Method for Preparing Lungs in order to Preserve their Elasticity and Gross Anatomical Appearance.—The lungs must be removed intact, and the pulmonary blood vessels cut as close to the heart as possible. If it is intended to prepare both lungs the trachea should be cut as high as possible. If only one is prepared, cut the bronchus at its junction with the trachea. Insert and tie as large a cannula as possible in the bronchus or trachea, and in the pulmonary artery."

"1. Take of Müller's fluid 1,000 and of acetic acid 75. Fill the lungs through the air passages, and suspend them in solution for twenty-four or forty-eight hours; height of funnel not more than 3 ft. 2. Inject through the artery a mixture of glycerine 4, carbolic acid 1. 3. Dry as well as possible by suction applied to the air passages, then inject through the artery, under pressure of 5 or 6 ft. of water, cotton-seed oil 100, oil of origanum 10. Dry as well as possible, and keep in a crock."

II. Its Physiology.

The "Roosevelt elastic lung" is therefore remarkable for its durability as a whole; but much more so for that of its elastic fibre and of the elastic function in it. A striking contrast is displayed between the pathological history of the fibre in life and its artificial history under preservation in death. As shown by emphysema and by obesity, it is more prone than most living tissues to mechanical destruction by overuse or disuse. As shown by arterial degeneration, and by vascular syphilis, it is also exceedingly liable to destruction by microbes and by phagocytosis. This is doubtless correlated with an extremely low coefficient of nutritional activity and requirement; which is fully demonstrated by its endurance, when it is adequately protected against the scavengers of death.

We know little about it. But we do know that it is the last to die; and that it is also our *ultimum movens*, when, by a final vital recoil, the all-pervading "elastic skeleton of configuration" restores our normal lineaments after their muscular rigidity. That enigma, the last we have approached, might well hold our solution for another problem, not the least important of those yet to be solved—"the mechanics of our biomorphology." If, as it appears, the elastic fibre has in its mechanical keeping our individual polyorganic architecture, might it not conceivably be itself the builder; and, if so, the missing link between our physics and our vital energy? Adami's striking conception of "fibrogenesis" as a linear intraplasmatic

crystallization along each line of movement and of stress is suggestive of a great mechanical antithesis. Our "rigid" skeleton of support is mineralized through "rest"; our "pulsatile" elastic skeleton of organic architecture and nutrition would be the direct mechanical product from the individual. In vital "work," too, might possibly lie the true explanation for our chief clinical puzzle. The extreme diversity in our "normal" individual "sphygmopiesia" might be but an expression of the diversity in our "physical" or elastic coefficient, as the inviolable or irreducible individual factor in our vital "dynamic equation" between the nervo-muscular heart and the neuromyo-elastic artery.

The Croonian Lectures

ON

TRYPANOSOMES CAUSING DISEASE IN MAN AND DOMESTIC ANIMALS IN CENTRAL AFRICA.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS OF LONDON.

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LECTURE IV.

In this concluding lecture I intend to sketch briefly the outstanding features of the trypanosomes belonging to Groups B and C. As far as we are aware, none of the trypanosomes attack man. They are, therefore, of less interest to us than the two species already described.

GROUP B.—THE TRYPANOSOMA PECORUM GROUP.

1. TRYPANOSOMA PECORUM.

The first of this small group, which only consists of two species, is *Trypanosoma pecorum*. It is probably the most important trypanosome disease of domestic animals in Central Africa.

Morphology.

Fig. 1 shows its general appearance. It is the smallest of all the African pathogenic trypanosomes, varying from 9 to 18 microns in length, with an average of 14 microns.

Fig. 1.—*Trypanosoma pecorum*.

Its breadth averages 1.96. This species is monomorphic, and short and stout in form. The contents of the cell are generally homogeneous. The nucleus is oval in shape, and situated about the middle of the body. The micronucleus is small and round, and situated near to, but not at the posterior extremity, and often appears to project beyond the edge of the trypanosome. The undulating membrane is simple, but fairly well developed, and there is no free flagellum.

There ought to be no difficulty in recognizing this species from morphology alone. If one or two hundred are drawn and measured, the average length would separate it from its neighbour *Trypanosoma simiae*. Another small trypanosome, *Trypanosoma uniforme*, differs in shape and in having a free flagellum.

Animals Susceptible to *Trypanosoma pecorum*.

Next in regard to the animals attacked by this trypanosome. This is essentially a disease of the herds. Horses, donkeys, oxen, goats, sheep, and pigs all fall victims.

One peculiarity about *Trypanosoma pecorum* is that it readily loses its virulence for certain animals by passage through certain other animals. For example, if this species—which is usually more or less infective to the monkey, dog, and rat—lives for some time in the blood of