bellum (Fig. 3). The magnification, 500 diameters, is not sufficient to show these small round bodies distinctly. Under a high power, however, no differentiation of structure can be made out. It is therefore impossible at present to do more than record the fact of their existence.

In all parts of the central nervous system, as indeed in all the organs and tissues, diplococci (staining by Gram's method) could be found (Fig. 4).

By Marchi staining, numerous recently-degenerated fibres could be found in the medulla, spinal cord, and cortex (Fig. 5).

The microscopical examination, therefore, of the nervous system shows changes similar to those which have been met with by us in cases of the disease occurring in Africans.

Lymphatic Glands.—A large number of sections of these were examined, and by Gram's method every section showed large numbers of diplococci in foci or in the lymph channels and blood (Fig. 6).

Bone Marrow.—Showed diplococci and large numbers of large mononuclear leucocytes.

Lungs.—Congestion and pneumonia of the right lower lobe was found. Diplococci present.

Liver.—The cells exhibit fatty infiltration; there is some increase of fibrous tissue; the blood in the vessels shows haemolysis; excess of mononuclear leucocytes and diplococci. There is no malarial pigment.

Spleen.—Congestion of vessels, increase of fibrous stroma; diplococci; no malarial pigment.

Kidneys.—Vessels congested, no diplococci discovered; epithelium of uriniferous tubules exhibits first stage of coagulation necrosis, probably due to the toxic condition of the blood.

Heart.—Epicardium, a marked mononuclear infiltration around the vessels (Fig. 7). Early pericarditis: Myocardium, the muscle fibres exhibit brown atrophy and pigmentation, and there is a condition of recent haemorrhagic myocarditis due to infection; for in the haemorrhages are numerous colonies of diplococci, and in one section what appears to be a dead trypanosome (Fig. 8).

This is the only distinct evidence pointing to the existence of trypanosomes found in the large number of sections examined, but its form and size and its existence amid the blood corpuscles point undoubtedly to its nature, although it does not present the typical characters of the trypanosome seen in the film preparation (Fig. 1).

We have recorded the bare facts, and consider that this case is of especial interest, inasmuch as it shows that a European who has resided in a country where sleeping sickness exists, and who leaves that country with trypanosomes in the blood, may for a considerable time present no symptoms of sleeping sickness, and then develop them and die, the tissues showing changes similar to those met with in Africans. It is a strong argument in favour of the trypanosomes being the cause of the disease; but what part do the diplococci play? Have these appeared subsequent to the onset of the characteristic symptoms of the disease, and is there, therefore, a causal connexion, or is it merely a terminal infection?

We desire here to acknowledge the services of Mr. Charles Geary, Assistant to the Pathological Laboratory, Claybury, for the excellent photomicrographs of his preparations of the tissues.

ASYLUM DYSENTERY IN RELATION TO B. DYSENTERIAE.

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(From the Bacteriological Laboratories of Guy's Hospital.)

THE researches of Shiga, Kruse, Flexner, Strong, Rogers and others have resulted in the accumulation of a mass of evidence pointing to the correctness of the assumption that acute dysentery, wherever it occurs, is a disease due to the invasion of the human organism by a specific bacillus—*B. dysenteriae*; or, perhaps, in view of the uncertainty existing at present with regard to the relative importance of the Flexner and Shiga types, it would be more exact to say some one or other member of that group of organisms typified by *B. dysenteriae*. Flexner and other workers in this field, notably Vedder and Duval, have also demonstrated the presence of a similar bacillus in material obtained from cases of epidemic diarrhoea occurring in asylums for the insane in

America, thus supporting the contention of Mott, that the disease, is identical with the dysentery of tropical climes. So far as I am aware, however, the organism above referred to has not hitherto been isolated from cases of asylum dysentery in this country, and I therefore wish, to state briefly the positive results obtained from the bacteriological examination of material derived from such cases. My material was obtained from an epidemic of dysentery which occurred towards the end of last year in one of the London county asylums, and I now wish to express my thanks to Dr. Mott for his kindness in placing the said material at my disposal.

COURSE OF THE EPIDEMIC.

With the clinical aspect of the cases comprising this outbreak, and the means by which the disease was carried to wards other than that in which it first appeared, I am not at present concerned. Indeed many interesting points bearing on asylum dysentery and its bacteriology must be reserved for a detailed paper, which will appear in the forthcoming volume of the *Archives of Neurology*.

Briefly, the course of the epidemic was as follows: The male side of the asylum had been remarkably free from cases of diarrhoea and dysentery for some time prior to August, 1903. On the 20th of that month, however, two cases of dysentery were reported from a ward which I will call Ward A, and were followed by two more on the 24th; a fifth case occurred on the next day, and a sixth on the 28th of that month. No more patients were attacked in this ward, and within two months all six cases were convalescent.

Towards the end of September a similar outbreak, but of a more severe type, made its appearance in Ward B. Here eight patients were successively attacked, and of these three succumbed to the disease. In Ward C three patients contracted the disease, of whom one died. Isolated cases, in ones and twos, cropped up also in other wards at varying intervals during the months of September and October to the total number of eight, three of these being fatal.

TABLE I.—Ward Incidence of Cases of Dysentery.

Ward.	Patient's Initials.	Date of Onset of Disease.	Result.	Date.
A	G. E. R.	Aug. 20th, 1903	Recovery	Sept. 10th, 1903
	H. M. H.	" 20th, "	"	" 10th, "
	C. V.	" 24th, "	"	" 11th, "
	C. H.	" 24th, "	"	" 10th, "
	T. W. S.	" 25th, "	"	" 12th, "
	J. R. M.	" 28th, "	"	" 11th, "
B	J. F. S.*	Sept. 25th, "	Death	Nov. 30th, "
	T. H.*	Oct. 15th, "	"	" 7th, "
	J. A.*	" 16th, "	"	Oct. 24th, "
	S. H.*	" 17th, "	Recovery	Nov. 14th, "
	S. A. H.	" 19th, "	"	" 8th, "
	J. J.*	" 29th, "	"	" 27th, "
C	P. M.	Nov. 11th, "	"	" 26th, "
	A. A. S.	Oct. 16th, "	Death	Oct. 17th, "
	J. L.	" 18th, "	Recovery	Nov. 10th, "
	W. S.*	" 31st, "	"	" 26th, "
D	E. C.	" 19th, "	"	" 13th, "
E	B. B.*	" 17th, "	"	" 14th, "
F	A. D.	" 6th, "	"	Oct. 30th, "
	E. O.	" 10th, "	"	Nov. 1st, "
G	R. P. D.	" 6th, "	"	" 2nd, "
H	H. M.*	" 23rd, "	Death	Oct. 25th, "
J	J. C. S.*	" 14th, "	"	" 24th, "
	W. G. B.	" 15th, "	Recovery	Nov. 3rd, "

* Cases examined bacteriologically are indicated by an asterisk.

None of the cases in Ward A came under my observation, but of the remaining 18 cases I was supplied with specimens of the stools from 5 during life. Further specimens of faeces, scrapings of dysenteric ulcers, blood, and bile were collected at the *post-mortem* inspection from 5 of the 6 fatal cases (with all precautions against accidental contamination), and sent to me for examination.

METHODS: FAECES.

The method adopted in the examination of the faecal material and scrapings from dysenteric ulcers was simple in the extreme, the work of differentiation and isolation being facilitated by the employment of a special nutrient medium—a modified form of the "lakmus-laktose-nutrose-agar" of Drigalski and Conradi.* A platinum loop, reserved for this particular purpose, and capable of holding about 2 mg. of faeces, was employed to emulsify three or more loopfuls of the specimen in 10 c.cm. of sterile nutrient broth. Three

* A simplified method of preparing and standardizing this medium formed the subject of a recent communication by the present writer to the Pathological Society, January 19th, 1904.

loopfuls of this suspension were deposited on the surface of a nutrose agar "plate," and then spread evenly over its entire surface by means of a sterile L-shaped glass rod. Two, and sometimes three, other plates were then inoculated in series, and the whole set of plates incubated at 37° C. for twenty-four hours. At the end of this time such colonies as corresponded in their naked-eye appearances, blue coloration, and characteristic odour of fresh semen, with those produced by a typical *B. dysenteriae* (Shiga) were severally emulsified with sterile broth, and tested against a 1 in 100 and a 1 in 200 dilution of antidyenteric serum (for which I am indebted to Professor Kolle, of the Institute für Infektionskrankheiten, Berlin) both macroscopically and microscopically.

If a good agglutination reaction was obtained within half an hour the organism was grown upon agar, the subsequent growth plated, the organism then isolated and subcultivated in parallel series with an authentic *B. dysenteriae* (also obtained from Professor Kolle), and its identity or otherwise therewith determined.

BLOOD.

Specimens of blood, unfortunately, were available only from such patients as succumbed to the disease, and were collected at the *post-mortem* inspection—the bodies being kept in a cold chamber during the interval that elapsed between death and the necropsy—generally from the femoral vein, but occasionally from the heart direct. Some of the blood was employed to inoculate agar and broth tubes, and if after incubation at 37° C. growth supervened, that growth was plated on nutrose agar in a manner similar to that adopted for the suspensions of faeces, and the contained organisms isolated.

The remainder of the blood from each case was thoroughly centrifugalized, the separated serum pipetted off, and tested in varying dilutions against the strains of *B. dysenteriae* previously obtained from this epidemic, and also against the various "stock" strains of *B. dysenteriae*, which have been given me from time to time by Professors Flexner, Kolle, and others.

BILE.

Bile was also collected at each necropsy direct from the gall bladder, and cultivations were established from it on agar and in broth. Careful search was made in every case for the minute diplococcus recorded by Durham as occurring in similar cases, but without success. When growth was observed in the cultivations from the bile it was plated on nutrose agar and the contained organisms isolated and identified.

The result of these examinations is most conveniently given in the form of tables (Tables II and III), which in their arrangement correspond to the list of the cases comprising this epidemic given above:

TABLE II.—Results of Examination of Faeces obtained during Life.

Ward.	Patients' Initials.	Age.	Date of Onset of Disease.	Date of Receipt of Specimen.	Naked-eye Appearances of Faeces.	<i>B. Dysenteriae</i> Present or Absent.	Bacillus Isolated, Agglutinated by "Shiga" Serum in Dilution of
B.	J. F. S.	35	September 25th, 1903	November 2nd, 1903	Normal "formed"	Present	1 in 200
	S. H.	50	October 17th, "	October 29th, "	Typical dysenteric stool, semi-fluid, containing blood and mucus	"	1 in 200
C.	J. J.	37	" 29th, "	{ November 2nd, "	Resembling pea-soup	Absent	—
				{ " 5th, "			
C.	W. S.	24	" 31st, "	{ " 6th, "	Dark reddish-brown, semi-solid	Present	1 in 200
				{ " 9th, "	Pea-soupy	Not isolated	
E.	B. B.	38	" 17th, "	October 29th, "	Putty-like in consistence and colour	Present	1 in 150

TABLE III.—Results of Examinations of Post-Mortem Material.

Ward.	Patient's Initials.	Age.	Date of Onset of Disease.	Date of Death.	Date of Receipt of Specimens.	<i>B. Dysenteriae</i> Present or Absent.	Dilution of Blood Serum giving Positive Reaction against		Organisms Present in	
							"Shiga" Strain.	"S.H." Strain.	Blood.	Bile.
B.	J. F. S.	35	September 25th, 1903	November 30th, 1903	November 30th, 1903	Absent	—	—	Streptococcus pyogenes longus <i>B. coli communis</i> . <i>Nil.</i>	
	T. H.	65	October 15th, "	" 7th, "	" 9th, "	Present	1 in 200	1 in 200		
	J. A.	37	" 16th, "	October 24th, "	October 27th, "	Absent	—	—		
H.	H. M.	69	" 23rd, "	" 25th, "	" " "	Present	1 in 10	1 in 10	<i>Nil.</i>	
J.	J. C. S.	60	" 14th, "	" 24th, "	" " "	Absent	—	—	<i>B. coli communis</i> .	

From these tables it will be seen that *B. dysenteriae* was isolated from six of the cases examined. All these had, at some time or other during the course of the disease, been passing typically dysenteric stools—semi-fluid and plentifully mixed with slime, mucus, and blood—but with one exception all had passed this stage before I obtained specimens from them. The single exception (S. H.) afforded me a typical dysenteric stool, consisting chiefly of blood-stained mucus, which, when suspended in broth and plated on nutrose agar, yielded a practically pure cultivation of *B. dysenteriae*. This specimen was one of the earliest I received, and the *B. dysenteriae* from it was actually the first to be isolated and identified. It was strictly comparable in all its characters and reactions with the Shiga strains obtained from Berlin and Philadelphia; further, it was completely and almost immediately agglutinated, macroscopically and microscopically, by a 1 in 200 dilution of antidyenteric (Shiga) serum, and also by the serum from the patient T. H. in similar dilution.

The results obtained from the examination of *post-mortem* material were by no means so satisfactory, as the bacillus was isolated from two only of the five cases examined.

At the necropsy of the first patient (J. F. S.) from whom the bacillus had been isolated during the early stages of the disease, the dysenteric lesions of the intestine were found to be healing, so that presumably the organism, if not actually absent, was probably present in but very small numbers. In the third case (J. A.) the failure to isolate the bacillus must, I think, be attributed to the interval that elapsed between the necropsy and the commencement of the examination—an explanation that holds good also in the fifth case (J. C. S.).

The agglutination reactions, too, seem at first sight unsatisfactory, but I think it is quite possible that the general septicæmia which had attacked the patient J. F. S., and which was associated with the presence of large numbers of streptococci in the blood (the blood, too, showed marked hæmolytic changes), was sufficient to account for the negative reaction. The other cases may be explained on the assumption that agglutination is a reaction of immunity rather than a reaction of infection. My experience, too, of numerous other cases of asylum dysentery leads me to believe that a positive serum reaction is most likely to be obtained during the first fortnight of the disease. An alternative explanation in the case of the fourth (H. M.) would be that the interval between the onset of the attack and death—two days only—was too short to allow of the formation of any agglutinin.

CONCLUSIONS.

The number of cases included in this preliminary communication is obviously too few to admit of any generalization as to the etiology of asylum dysentery, but the following

statements seem to be quite warranted by the facts observed:

1. That a bacillus, identical with the *B. dysenteriae*, described by Shiga as the specific cause of acute dysentery in Japan, can be isolated from the stools of many cases of asylum dysentery.

2. That the blood serum of some of these cases of asylum dysentery possesses a specific agglutinative action when tested against *B. dysenteriae* isolated from the stools of other similar cases, and also against other strains of *B. dysenteriae* isolated from cases of dysentery in tropical countries.

On the other hand, the experience of an extended series of examinations of the stools of patients suffering from asylum dysentery will permit me to state:

3. That in order to detect the presence of *B. dysenteriae*, the stools must be examined when fresh.

4. That *post-mortem* material must be collected and examined as soon after death as possible, as after a few hours' delay, even in quite acute cases *B. coli* becomes the predominant organism in the intestinal canal.

5. That under these conditions (3 and 4) the isolation of *B. dysenteriae* from the stools of patients suffering from acute asylum dysentery is comparatively easy by the methods I have indicated—when those stools are, naked eye, typically dysenteric.

6. That in cases of chronic asylum dysentery *B. dysenteriae*, if present, is so outnumbered by *B. coli* and other intestinal saprophytes as to render its isolation a matter of extreme difficulty.

DIFFERENTIATION OF *B. TYPHOSUS* AND *B. COLI COMMUNIS* BY MEANS OF THE PHOTOGRAPHIC PLATE.

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It does not appear to have been recorded that the gelatino-bromide photographic plate is sensitive to direct application of the products of certain bacterial cultures in a very remarkable manner, and that, further, the effect on the plate is variable with the special nature of the bacteria to a degree which renders easily possible, in at least one important case, a discrimination between bacteria otherwise difficult to differentiate.

My observations having been mainly confined, so far, to the typhoid bacillus and the *B. coli communis*, I will refer at present to these only.

Cultures of these bacilli are made in broth, samples of the same broth being used for each culture. After, say, 24 hours a few drops of each culture are placed upon the sensitive surface of a photographic plate and spread so as to wet an area of any desired extent—say, 1 cm. or 2 cm. in diameter. (In general, 0.2 c.cm. of the culture has been allowed to spread by itself upon the plate, covering an area of about 1 square cm.) This is done in a red light sufficiently faint to avoid sensible fog upon the plate. An opaque cover—an empty developing dish will do—is now turned over the plate, and it is left thus screened for about 40 minutes.

The cover is now lifted and the superfluous fluid upon the wet areas drawn off with a pipette. No change is observed at this stage of the experiment. The plate is then developed in the usual manner, and during development it is best screened from light. It will now be found that the moistened areas develop with very different densities of silver deposit. The *coli* culture produces a full and marked reduction of the salt. There has progressed over the area wetted by this culture an action similar to the formation of a photogenic material in the film. But where the typhoid culture has acted the result is quite different. Only a very faint deposit is, in general, obtained. And if the plate has, in fact, been exposed sufficiently to the red light (either wilfully or incidentally) to produce a general fogging, it will be found that its effect is even inhibitive and its presence marked by an area almost free from reduced silver.

The following points are of interest, but can here only be briefly noted:

(a) The discriminative action is the same, with some variation in degrees of distinctness, when typhoid and *coli* cultures of the same age are compared at very different periods after inoculation. Thus, cultures of these two organisms in broth compared, after 4 hours' growth and after 33 days, react as above.

(b) The activity of each increases with time up to a certain

age which has not been accurately ascertained. This activity possibly varies with the physical conditions attending culture, but may be taken as increasing from shortly after inoculation to 4 days of growth.

(c) A *coli* culture of 24-hours age, when compared with a typhoid culture of any age (tested up to 33 days) gives the more marked reducing effect.

(d) After a few hours' growth the broths may be boiled and thus sterilized, and the differential effect upon the sensitive surface persists.

(e) This discriminative reaction has been obtained with four cultures of *coli* from different sources and with five typhoid cultures from different sources.

(f) It will be found that if the sensitive plate is wetted with tap water and also with sterile broth, and simultaneously applications of the cultures are made—four separate areas being thus prepared for comparison—the typhoid and sterile broth do not differ markedly in their actions upon the silver bromide, whereas the effect of the tap water has been intermediate between that of the *coli* and the typhoid. It is known that water is to some extent a sensitizer.

(g) The time of exposure of the plate to the application of the cultures has varied between 10 and 90 minutes, and the discriminative action of 24-hour cultures found to be best marked with an exposure of 45 minutes. But from at least 10 minutes exposure upwards the difference is obtained with any broths in which there is a visible growth.

(h) While in some of the earliest of these observations, and before I had gained experience as to the best methods to employ, a few cases occurred in which the distinctive difference was not well marked, in all my recent observations, twenty-four in number, I have unfailingly obtained the differential effect.

(i) Finally I have observed that if pools of well-grown *coli* and typhoid broth cultures are poured on a photographic plate and exposed to light, a reduction of silver is seen in a few minutes without development. After about 30 minutes the deposit due to the typhoid broth is distinctly darker than that due to the *coli* broth. If the pools be now poured off the sensitive plate the difference reaches its maximum, but as the gelatine dries the distinction is almost completely lost. Tap water has no visible effect, except the swelling of the wetted gelatine of the plate.

I am continuing the experiments, and am not without hopes of more extended applications of the phenomenon.

NOTE UPON THE CAUSATION OF CANCER.

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THE observations of Professor Farmer and others with regard to the mode of division of the nucleus in cancer cells render it possible to formulate a theory which seems to me to afford reasonable explanations of many of the difficult points in connexion with cancer.

Briefly stated, my view is that cancerous growth is caused by the degenerative reversion of epithelial cells to a germinal type, in association with a local irritant and in the presence of an abundant blood supply. The post-heterotypic multiplication of the cells having once commenced, as a result of the degenerative reversion to an ancestral type, the local conditions serve to continue and fix the type, with the result that the growth becomes practically a colony of unicellular parasites. These act as one would expect members of such a colony to act; they encroach upon the tissues of the host, preferring to multiply along the lymph channels both on account of the resistance being less in this direction than in any other, and also on account of the favourable nutritional environment there met with; or when they permeate the walls of a vein tend to be washed off from the colony as independent units, which, when they lodge in suitable situations, give rise to new colonies, namely, the metastatic growths.

Just as in animals we find an ascending and a descending stream running along parallel lines, so in cells, with regard to specialization, evolution may be downward as well as upward. Again, just as the individual in his development presents a brief epitome of the history of the race, so, to a certain extent, does the individual cell in its decay tend to approximate to an earlier type of cell similar to that from which it has been evolved, and with similar attributes and powers. However, the more specialized the cell the greater