ENDEMIC BACILLARY DYSENTERY.

By J. J. B. MARTIN, M.D.

SPORADIC bacillary dysentery is an accepted concomitant of lunacy, and mental hospitals, on statistical evidence, harbour more than 99% of the disease.* When an epidemic of dysentery occurs, the laboratory of the mental hospital is cluttered with fæcal specimens, but when the epidemic is resolved on a solitary "carrier" and a faulty drain the disease ceases to arouse further interest. Little effort is made to assess and control the endemic level. This the author has attempted to do, in a county mental hospital of, roughly, 1,000 beds.

At the outset I wish to acknowledge the great help given in the preparation of this paper by the Assistant Pathologist and Laboratory Technician, Mr. O. T. Moore, whose skill and ungrudging labour alone made its completion possible. My thanks are also due to Dr. P. W. Bedford, who instigated and supported the investigation and permitted the publication of this paper.

The following is a summary of the work done over a period of four years :

Fæces : bacteriological examination	2395
Sera : Widal	921
Water : bacteriological examination	221
Gall-bladder, post-mortem bacteriological examination	90
Intestine, post-mortem bacteriological examination	12
Typing, absorption tests	48
Blood : culture	22
Urine : culture	21
Blood : bactericidal test	22
Gastric juice : chemical examination	60
Dysentery vaccine prepared (approx.)	30 litres.

A. ASSESSMENT.

1. *The Extent of the Disease.*

Dysentery being regarded as a sanitary index, and therefore as a reflection of hospital administration, hospital records minimize the extent of the disease. The first "obituary table" of the hospital, published a hundred years ago, reveals that 10% of the total population died from dysentery in one year, and

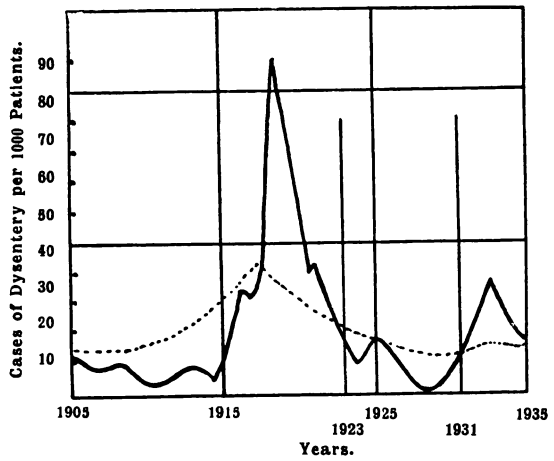
* *Incidence of dysentery in mental hospitals.*—Dawson (4) in 1921 stated that on statistics dysentery was only 100 times more common in mental hospitals than outside. When making his calculation he failed to deduct the registrations of dysentery given by mental hospitals from the total registrations given by the whole country, including mental hospitals. Actually dysentery is 400 times more common in mental hospitals than outside.

it is a fairly safe deduction that dysentery has persisted in the hospital ever since.

From 1905 onwards all cases of dysentery are purported to be tabulated in an official "register of dysentery and diarrhœa". From this the apparent incidence of the disease is plotted per 1,000 of population (Chart I). The dotted line represents the apparent incidence for all mental hospitals in England and Wales.

This graphical picture of the disease is far from accurate. From 1905 to 1923 the figures depend on arbitrary clinical diagnosis, and only epidemic groupings are shown. From 1923 (when a laboratory was established) until

CHART I.—*Incidence of Dysentery.*



——— = This Hospital
..... = Average of other Hospitals, England and Wales.

1931 the figures represent an opposite extreme: only those cases which were obvious clinically and which proved to be bacteriological positives are included. From 1931 to 1935 the figures represent an intensive effort to expose dysentery in every manifestation of the disease. Of the last 80 cases recorded, 29 (or almost 40%) were sub-clinical or symptomless, and the proportion of symptomless cases continues to rise. As an old nurse said, rather irritably: "There's far less dysentery, but far more fuss."

Serological incidence.—Even the figures of recent years represent only a fraction of the real extent of the disease. Below, two very graphic serological pictures are presented, side by side. On the left (Table A) are the agglutination figures of an average ward of 47 patients. Two cases of dysentery occurred in this ward; two further cases ("carriers") were found, and a routine bacteriological and serological examination was made of all cases. The examination extended over two months. On the right (Table B) is a

TABLE A.—47 *Female Chronic Patients.*

Agglutinins.					Fæces.
V.	W.	X.	Y.	Z.	
100	25	25	25	50	—
25	0	13	12	25	—
50	20	12	10	25	—
30	12	13	10	12	—
50	20	25	10	25	—
40	0	0	5	0	—
20	10	12	10	20	—
25	0	0	12	10	—
10	0	0	10	0	—
12	0	0	12	0	—
(25	0	0	8	12	±)
50	10	20	20	20	—
12	0	0	0	0	—
12	0	12	8	0	—
5	20	10	10	8	—
25	0	40	16	40	—
5	0	0	5	0	—
15	35	25	25	12	—
20	10	10	8	0	—
12	12	12	30	0	—
50	0	20	40	40	—
15	0	0	5	0	—
35	0	20	30	25	—
25	0	12	8	25	—
12	0	0	4	12	—
25	0	0	8	25	—
12	0	0	4	25	—
25	0	12	30	0	—
100	25	25	21	25	—
5	0	0	0	0	—
25	0	12	8	12	—
5	0	0	6	12	—
12	12	0	8	0	—
35	0	12	13	20	—
25	0	0	0	0	—
35	0	0	8	12	—
35	12	20	21	25	—
25	12	12	8	12	—
50	0	40	21	62	—
(25	0	20	21	12	+)
25	0	20	8	12	—
20	0	0	8	0	—
12	0	40	42	62	—
25	12	0	13	12	—
12	25	12	21	12	—
10	18	0	0	12	—
50	12	62	25	62	—
25	6	12	13	16	

TABLE B.—47 *Female New Admissions.*

Agglutinins.					Fæces.
0	0	0	0	4	—
0	8	0	0	4	—
0	8	0	10	4	—
0	0	0	0	0	—
0	0	0	0	0	—
16	0	0	0	0	—
0	0	0	4	0	—
0	0	0	0	0	—
0	0	0	0	0	—
0	0	0	0	0	—
0	0	0	0	0	—
25	0	0	0	0	—
0	0	0	0	0	—
0	8	0	0	0	—
0	0	0	0	0	—
0	0	0	4	0	—
0	16	20	8	25	—
6	0	0	0	0	—
12	0	0	0	0	—
5	0	0	0	0	—
5	0	0	0	0	—
5	0	0	0	0	—
12	0	0	0	0	—
5	0	0	0	0	—
12	12	16	12	12	—
12	25	16	0	12	—
5	0	0	0	0	—
12	0	0	0	0	—
20	0	12	16	12	—
6	0	10	8	12	—
12	12	0	8	12	—
3	0	0	0	0	—
50	12	25	8	12	—
12	0	0	8	0	—
50	25	12	8	0	—
5	0	0	8	0	—
12	0	0	4	0	—
12	0	0	4	0	—
2	0	0	4	0	—
5	0	0	0	0	—
5	0	0	0	0	—
25	12	0	0	0	—
12	12	12	8	12	—
12	0	0	4	0	—
12	0	0	8	0	—
12	0	0	6	0	—
35	0	0	8	12	—
9	3	3	3	3	

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comparative series of consecutive admissions to the hospital, taken about the same time. The agglutinations were performed against the standard Oxford suspensions. The readings were taken at four hours. The reduced titres are shown.

The contrast between the chronic patients, long resident in the hospital, and the new admissions is clear. The new admission, in the vast majority, is a serological negative. The chronic patient shows the full serological spectrum ; and in the average hospital ward, if active infection is momentarily confined to a small proportion of patients, over a period of years the Flexner antigen enters into 100%.

Under present hospital conditions the insane population makes an ideal breeding-ground for the disease. Dysentery is rarely imported into the hospital. Only one " carrier " has been found in the routine examination of over 500 new admissions. The source of infection is within the hospital walls, and the endemic level is never zero.

2. *The Causal Organism.*

In many endemic areas, and in some mental hospitals, the types of infecting organisms are extraordinarily numerous. In this hospital one particular species, *B. dysent.* Flexner, is responsible for 96% of dysentery, and the organism recovered is invariably of one sub-type, which may be designated Flexner Zx.

The cultural characters are largely as described in standard works. The colony has few distinguishing features. Growth is unaffected by mild changes in the H.I. concentration of the medium. It grows freely in bile, in whole blood or citrated blood, but less freely in serum or dilutions of serum. Rough colonies have not been obtained from original platings. The sugar reactions are constant : dulcite is never fermented. No organism giving the typical cultural reactions was found to be inagglutinable by specific sera or vice versa.

The toxicity of the germ appears to be constant. Typical dysenteric stools follow an excessive dose of the killed suspension, and in one subject who died of intercurrent disease a day after inoculation the intestine presented the appearance typical of the disease. On the other hand, the virulence of the organism, as measured by its power to infect, to cause disease, and to kill, shows a wide variation. Clinical variations in infectivity cannot be altogether explained in terms of the host and his environment, or in terms of dosage. A clean patient, in a ward with good hygienic conditions, excreting the germ only occasionally, and in small numbers, may precipitate a series of cases. A dirty patient, in bad hygienic conditions, may excrete large numbers of germs persistently, without infecting others. Similarly, while it is plain that rapidity of passage increases, and a high degree of specific immunity diminishes the severity of the individual case, this relationship is not constant.

Other organisms.—Morgan's No. 1 (28, 29, 30, 31, 32).—This organism was first described by Morgan in 1906. The cultural and biochemical characters are :

Glucose A → G, lactose O, mannite O, dulcite O, sacharose O, milk O or A → Alk., indole +, motility + or O.

We have found this organism in normal fæces and in solitary cases of diarrhœa in old and debilitated subjects. Although Shaw Bolton (1930) regards this organism as specific, or as so intimately associated with true dysentery as to be potentially specific, we have not found it related to any serious case of diarrhœa, or to any series of cases, nor have we found any association between it and true dysentery.

Douglas and Colebrook No. 8 (33, 34, 35, 36, 37).—This organism belongs to the *B. alkalescens* group. It was first described by Douglas and Colebrook in 1917, and labelled *B. dysenteriæ* Andrewes in 1918. The cultural and biochemical characters are :

Glucose A, lactose O, mannite A, dulcite A, saccharose O, milk Ac.-Alk. +, indole + ?, motility O.

It is distinguished from Flexner in the following respects :

(i) It is a late fermenter of dulcite. This feature is invariable, although fermentation may be very late (10 to 14 days).

(ii) Although perhaps at first faintly acid in milk, the ultimate reaction is strongly alkaline.

(iii) The production of indole is inconstant, whereas the production of indole by *B. Flexner* is always demonstrable by a suitable technique.

(iv) It is inagglutinable by Flexner sera and vice-versa.

We have found this organism :

(i) In the normal fæces of healthy patients.

(ii) Alternating with *B. Flexner* in subacute cases of dysentery ; fæcal specimens and sigmoidoscope swabs.

(iii) It was found more commonly in a ward where dysentery (Flexner) was active ; 14 out of 70 consecutive specimens.

(iv) In solitary cases of diarrhœa, where no (other) causal organism was found, particularly in old and debilitated patients.

We therefore had some reason to suspect the organism as pathogenic and its frequent association or alternation with Flexner at one time made us wonder whether it was not an inagglutinable variant of *B. Flexner*. The pathogenicity of the organisms seems to be very doubtful. It is rarely found in pure culture, like dysentery, from a loose stool. Its toxicity is slight ; the symptoms (a dangerous criterion, maybe) are never severe or lasting. The organism disappears rapidly from repeated specimens. It is not agglutinated by the patient's serum at any time. And finally, although patients excreting the organism have not been isolated or subjected to any special precautions, we have not known even two definitely related cases, such as we might expect to find if there were any potential epidemicity.

We have attempted to approximate Douglas and Colebrook No. 8 to Flexner in various ways. We attenuated Flexner by heat ; we grew Flexner in immune sera ; we educated it to strongly acid and strongly alkaline media ; we coaxed and cajoled it, but it refused to ferment dulcite. Nor would Douglas and Colebrook respond to the reverse form of education.

Our opinion is that the biochemical characters of Douglas and Colebrook No. 8 cover not one, but a group of allied organisms, often symbiotic with the Flexner group, which can be safely disregarded as primary causes of epidemic diarrhœa.

B. Sonne (38), although theoretically interesting as a comparatively new disease with a healthy exotoxin and a beautiful S—R variation, accounts for

only 4% of cases since it was first discovered in the hospital in 1929. It seems likely that the disease was introduced from the surrounding districts; Sonne is endemic in the villages near the hospital. Despite the liability to importation, Sonne shows no tendency to spread in the hospital population; and according to serological evidence it has not been responsible for any high proportion of disease in the past history of the hospital. The organism now holds few bacteriological mysteries. The cultural reactions are fairly distinctive; the agglutination of the suspect organism is clean-cut; the sera of suspected patients show rapid and distinct agglutination curves; and a serological positive, in contradistinction to *B. Flexner*, is sufficiently rare to have some value in diagnosis.

Path of invasion.—The path of invasion is a matter of conjecture. On the analogy of other organisms pathogenic to the bowel, such as *B. typhosus* or *B. ærtrycke*, the path of invasion would be indirect: absorption through the more permeable mucosa of the upper alimentary tract, bacteriæmia, excretion of the organism in the bile, and secondary infection of the lower bowel. In clinical fact a bacteriæmia is rarely demonstrable, and the organism has never been recovered from the bile. The usual assumption is, therefore, that the path of invasion is direct, by simple ingestion.

Distribution of lesions.—Whatever the path of invasion, the initial lesions of the acute case are not localized, but are distributed throughout the length of the colon and the lower third of the ileum. The chronic lesions are equally diffuse, but pus-pits and mucus cysts are more probable in the lower colon. The mesenteric glands draining the infected area may harbour the organism, but there is no focal residue in the gall-bladder. Urinary excretors are rare.

3. *Symptomatology.*

According to degree of symptoms, there are four distinct types of cases: symptomless, sub-clinical, subacute, and acute.

(i) *Symptomless.*—The organism may be excreted without signs or symptoms of the disease. The organism is ingested, passes through the body, and may be recovered from the fæces. The host is unaware of its presence, and the stool is normal, both to macroscopic and microscopic examination, with a complete absence of cellular mucus.

(ii) *Sub-clinical.*—

(*a*) The stool contains shreds of muco-pus.

(*b*) One loose stool, containing muco-pus, and, perhaps, blood.

(*c*) Mild, transient diarrhœa.

(iii) *Subacute.*—There is diarrhœa, lasting a few days, with slight malaise and slight pyrexia.

(iv) *Acute.*—This is the typical picture of the disease: a short incubation period, with malaise; a rapid pyrexia, sometimes preceded by rigor; usually vomiting; then diarrhœa, accompanied by griping pain and followed by

tenesmus. The stool contains blood, mucus, epithelial debris, inflammatory cells and organisms.

Chronicity.—There is a wide variation of chronicity, and the severity of symptoms is a bad gauge of the duration of the disease. An acute phase may last hours, or days, or weeks; a subacute ulcerative process drags on for months, and a retention cyst takes years to burrow to the surface. Ilitch quotes a case observed over a period of seven years, during which the organism was excreted only on 15 days out of 2557, i.e., one positive specimen to 170 negatives. This is, perhaps, a somewhat extreme example, but it emphasizes two vitally important points: first, that the excretion of the germ is essentially intermittent, and second, that this intermittent excretion may persist indefinitely.

The carrier state.—There are two distinct types of carrier.

The first is the *transient excretor*: the subject who harbours and excretes the organism for a very short period of time, without displaying any sign or symptom of disease. Of repeated consecutive fæcal specimens, only one is positive. The stool is formed. Blood and mucus are conspicuously absent. Sigmoidoscopy reveals a healthy gut. Such a type of carrier may be found among any infected group. He is an interesting phenomenon, suggesting the presence of many others of his kind, unsuspected and undiagnosed, in whom there is an exact balance between resistance and virulence. He will exist in large numbers during an epidemic, and he becomes commoner if the standard of immunity is raised by natural or artificial means and no measures are taken to minimize conveyance.

The second is the *chronic intermittent excretor*, who has a diseased gut, and excretes the germ intermittently, with minimal symptoms, over a period of years. There is no history of disease, and very often the only visible sign is the presence of cellular mucus in the stool. In the academic sense he is not a true carrier: he is a missed case. It is in him that endemic disease drags on, for 5, or 10, or 20 years, and no hospital can consider itself free from disease until he and all his kind have been completely eradicated.

4. *Conveyance.*

Viability of organism.—B. dysent. Flexner has a power of survival outside the body which is not generally recognized. An agar slope survives for months. The organism is easily killed by heat, but it will live for long periods in ice. Variations in humidity have little effect; it may live in sterile water for a month, and, although stated to favour a moist medium, it may survive drying for 10 weeks. Although the difficulty of recovering the organism from a stale specimen is notorious, it does not die quickly in fæces. According to personal experiment it survives in an impregnated fæces for 11 days;*

* A dysenteric exudate was impregnated with an autogenous culture. It was therefore much more heavily infected than a normal dysenteric stool, and the 12 days of viability represent a probable maximum time of survival, rather than the average time of survival.

according to the literature it has been recovered from dejecta on soiled linen after 30 days, from the garden soil after 49 days, and from the polluted earth of a military camp after one year. It is, however, very sensitive to light, being killed by exposure to bright sunlight after half-an-hour.

Habits of population.—In a mental hospital the path of the organism from anus to mouth is made peculiarly easy by the degraded habits of many of the patients. A group of 431 female patients was analysed thus :

	Number.	Percentage.
Definitely faulty in habit	86	20
Sometimes faulty in habit	75	17
Clean	270	63
Total	431	100

This same group of patients is distributed through ten different wards. Only two wards contain 100% of clean patients ; only those two wards have remained free from dysentery in the past 5 years. Of the last 80 cases of dysentery, 46 are of faulty habit.

Inadequate sanitary facilities.—Such a degraded population requires a very highly elaborated system of sanitation ; in the hospital to which these investigations refer this did not exist. The water-closets were inadequate in type and number ; there was a widespread use of chambers and old-fashioned commodes ; poor facilities were available for the disposal of soiled linen. Baths and running water were at a premium. The hospital structure is old-fashioned and not designed for cleanliness.

Overcrowding.—The hospital is often overcrowded. Not in theory. In theory each patient has his allotted quota of day-space (440 cub. ft.) and night-space (550 cub. ft.). This hospital has, theoretically, empty space for 200 extra beds. And yet at one time one might find a ward where, if all the patients sat down simultaneously, they were virtually touching each other, and where the beds, at night, were less than 1 ft. apart. The hospital at present accommodates 900 patients. Postulate an arbitrary maximum at a lower figure (800), and plot the curve of excess population against the dysentery index (Chart II). The similarity of the two graphs is too remarkable for sheer coincidence.

Subjective factors determining invasion.—A mental hospital population has little power of resistance to disease. The standard of the average patient is C3 ; dysentery picks out the C4 fraction. His food is deficient in protein value and in vitamins. The integrity of the mucous membrane of the alimentary tract is vitiated by a variety of causes : bad teeth ; imperfectly masticated food ; deficiency of secretions ; constipation and its corollary, excessive doses of salts and purgatives. 50% of patients show obvious signs of intestinal catarrh ; and the patient with feeble powers of general or focal resistance has

little chance of acquiring a specific immunity, however prevalent the disease. An attack of the disease does not preclude reinfection. There is a floating

CHART II.

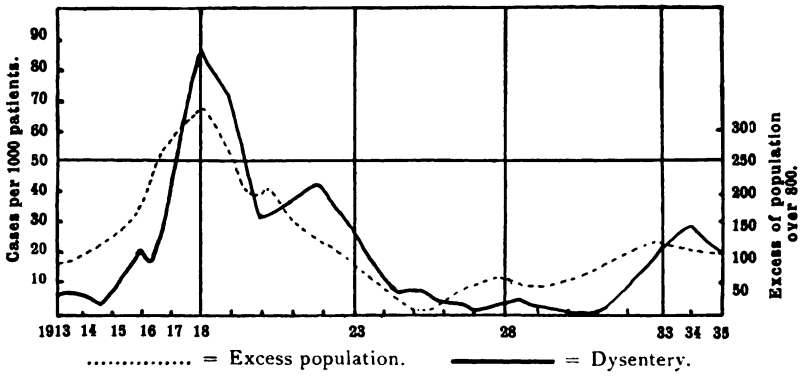
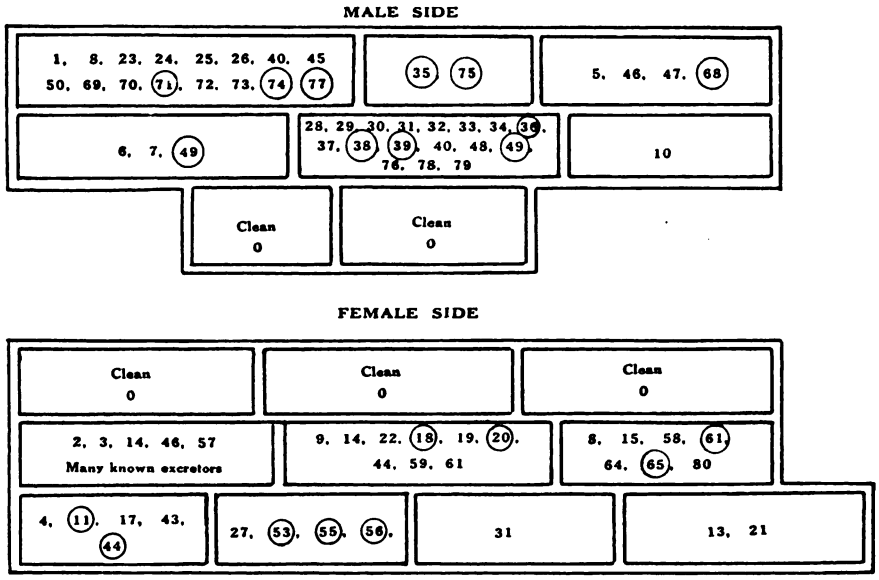


CHART III.



population of all grades of immunity, and a regular influx of new admissions—perfect soil for the disease.

The endemic nucleus.—The number of infectors is very much larger than the number of actual cases of dysentery would suggest. In Chart III is given

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a rough diagram of the hospital, divided into wards, and on this diagram a sequence of cases is plotted; symptomless excretors are marked with a ring.

These cases occurred consecutively over a time of only four years, following a period when the hospital was, supposedly, free from disease. Yet dysentery is widespread; more than two-thirds of the wards have been infected, and symptomless excretors have been discovered in 9 of the infected wards.

Spread.—The spread of the disease is at first slow, subterranean, and confined to the infected ward. But the segregation of the mental patient in his ward is far from absolute. Presently an infector is transferred, undiagnosed, to another ward, which in its turn becomes a focus of disease. Or worse, an infector is turned loose, as a worker, in the hospital. Active excretors have been found working in the hospital kitchen, the stores, the farm, and the garden.

Remote dissemination.—If active excretors are distributed all over the hospital, an epidemic is no longer a risk, it is a certainty. Sooner or later the organism will find its way into some common medium—water, or milk, or bread. With the outbreak of a large number of simultaneous cases, control becomes impossible. Diagnosis becomes guess-work. Complete isolation is impracticable. Treatment is imperfect. And when the epidemic subsides, practically the total hospital population is suspect of residual infection.

B. Control.

Effective control is possible only in the pre-epidemic phase of the disease process. Efficiency will depend on the ability of the hospital staff to diagnose the disease, to promote healing, to segregate infectors, to minimize conveyance, and to create in its population a power of resistance to disease.

1. *Diagnosis.*

Cases with symptoms.

There are two principles which should be inculcated *ad nauseam*:

(i) All fæces should come to the laboratory for examination if—

(*a*) The patient has diarrhœa, or—

(*b*) The stool contains blood or mucus, or both.

(ii) If a patient excretes blood or mucus or both in conjunction with any degree of pyrexia, the case should be considered as dysentery, and treated as such, until proved otherwise.

Cases without symptoms.

In the search for excretors without obvious clinical signs two alternative methods are available.

1. Routine examination case by case and ward by ward of the total population. This method is not advocated. Unlike typhoid, bacillary dysentery does not depend, for growth and spread, on an almost static number of "carriers". It is a much more active process, and, further, the symptomless excretor is much more difficult to diagnose.* To cover a hospital population of, say, 1,000 patients in any reasonable time would demand a laboratory of immense capacity, and an expenditure of material and labour totally incommensurate with the number of positive results obtained.

2. The alternative method is to wait for a case to display symptoms, and then examine contacts. This may at first sight appear to be a confession of failure; the attack on the disease seems indirect. The method is, however, extremely practical, and it has produced results in the immediate reduction of the disease which routine and indiscriminate examination *en masse* did not attain. There is a higher number of positive findings, and even if some of these come from transient excretors, there is a very high probability of the chronic infector being discovered and isolated.

Provocative vaccine.—To stimulate the latent excretor, a provocative vaccine may be used. The utility of such a vaccine was suggested in the course of routine inoculation of the hospital. Given an adequate dose, the latent excretor shows active signs of disease, and infected mucus is excreted in the fæces. Our practice is to give 25 millions intravenously.

Macroscopic examination of stools.—In the examination of contacts, it is almost useless to plate any stool not containing mucus. Whitehead and Kirkpatrick plated 5,000 stools not containing mucus to find 0·1% positive, and 100 stools containing mucus to find 75% positive—a ratio in proportionate infectivity of 750 to 1.

The whole stool should be exposed for examination. It should be passed naturally into a clean commode, drained of urine, transferred to a clean chamber, and covered. Thereafter the stool is spread on a flat dish. If mucus is apparent, several loopfuls are transferred to a tube containing glycero-saline and sent to the laboratory. If mucus is not apparent, a portion of the stool is macerated in sterile water, when shreds of mucus, if present, float out. Significant mucus (muco-pus) is recognizable on the surface of a formed stool. It is a yellow white, opaque, and more friable than simple mucus. Long, stringy mucus is usually negative.

* *Relation of "carrier" to acute cases in typhoid.*—We even doubt the jump from extreme chronicity (the carrier) in typhoid to the acute case. This hospital boasts carriers of typhoid and of paratyphoid B, and out of, roughly, a dozen cases of the disease we have only seen two text-book cases. Among the other ten we have seen and demonstrated:

(i) Typhoid fever without abdominal symptoms, a negative blood culture on the fourth day, and only one positive stool, on the tenth day.

(ii) Paratyphoid without abdominal symptoms, a negative blood culture on the third day, and only one positive stool, on the ninth day.

(iii) Paratyphoid of the pure bacteræmic type, blood positive, and stools consistently negative.

(iv) Paratyphoid with negative blood culture on the second day, no abdominal symptoms, a sore throat, a typical rash and temperature, and persistently negative fæces: the agglutination curve soared from 0 to 5400, clinching what seems, on paper, an absurd diagnosis.

Taking of specimens.—When the stool cannot be kept for macroscopic examination by a trained observer, a specimen outfit is employed by the nurse. This should be as simple as possible ; a metal spoon, preferably long to avoid soiling the nurse's hands, is embedded in a cork fitted into a wide, flat-bottomed tube containing enough glycero-saline to cover the specimen. The nurse is instructed to include mucus, or blood, if present, in the specimen.

Delay in plating.—It is essential that the specimen, whether taken by the bacteriologist or the nurse, should be fresh, particularly if, as usual, contaminated by urine. Little and Bernstein did comparative tests on a series of cases from acute cases, spreading one plate for each specimen within ½ hour of excretion, and another plate from each specimen after 3 hours ; after 3 hours the possibility of a positive finding was reduced by no less than 50%. Specimens should be timed as well as dated, and specimens more than an hour old should be discarded, if not previously transferred to a preserving medium.

Preservation and enrichment.—The preserving medium employed is glycero-saline, 10% glycerin in normal saline. This medium probably inhibits the normal overgrowth of coliform organisms, and allows the more fragile dysentery bacillus to survive.

Microscopic examination of fæces.—A portion of the mucus, or of the exudate of the acute case, is examined under the microscope for the presence of blood or pus. If pus cells are found, a bacterial process has been demonstrated, and further specimens are demanded from the case. Even if pus cells are not found, we proceed to plating ; if the plating is negative, and there are no suspicious symptoms, the examination of the case is temporarily closed.

Plating.—Plating is done on McConkey's medium, using the largest Petri dishes adaptable in quantity to a small incubator. If there is mucus, it is washed twice in sterile water, then emulsified in saline. A loopful of the emulsion should give an even spread of colonies.

Plates are examined with a lens after 24 hours ; on numerous occasions a solitary colony has been positive. All types of non-lactose fermenters are put in glucose. Where rapid identification is necessary, slide agglutination is useful, but it is unwise to give a negative finding because the N.L.F. refuses slide agglutination. The suspected organism is agglutinated against the standard sera, and no organism giving the typical sugar reactions in glucose, lactose and dulcite is considered inagglutinable until it has been grown in broth.

Agglutination.—The agglutination of suspected sera against the known organism has only a limited value in diagnosis.

(i) It is essential to use specific, not mixed suspensions, of uniform sensitivity, and a rigidly standardized technique.

(ii) A single estimation of agglutination titre has little practical value ; the highest titre may be found in the healthy contact, and a completely

negative titre in the actual case of the disease. In searching a widely infected group for active excretors a single estimation of each subject is completely valueless.

(iii) An agglutination curve, on the other hand, composed of two or more estimations performed under identical conditions is of practical value in diagnosis :

(*a*) A rising titre plus symptoms, even if bacteriologically negative, is tantamount to proof of the existence of the disease.

(*b*) If, in a case of the disease bacteriologically positive, the titre does not rise, there is a strong presumption that the disease has existed in the subject for a considerable length of time.

(*c*) The value of a curve is modified, but not destroyed, by protective immunization. In every suspected case specimens of blood should be taken at weekly intervals for 3 weeks.

Bactericidal tests.—These were performed according to Thojtta's technique. The serum to be tested was put up in dilutions of saline : $\frac{1}{5}$, $\frac{1}{10}$, $\frac{1}{20}$, and $\frac{1}{40}$. To each tube was added a drop of broth and 0·01 of a dilute emulsion of *B. dysent.* Flexner (one loopful to a standard emulsion of 100 c.c.). The tubes were incubated for 3 hours. 0·01 c.c. from each tube was plated on a 15 mm. Petri dish ; the dishes were incubated for 24 hours, and the colonies counted.

The control, without serum, was tubed, plated and incubated in the same manner.

The counts were proportionately reduced to make the control figure 100.

Results.

Dysentery Excretors.

	Agglutinins.	Dilutions. $\frac{1}{5}$	$\frac{1}{10}$	$\frac{1}{20}$	$\frac{1}{40}$	Control.
1	63 RT	5	5	6	47	100
2	63	5	6	6	87	100
3	8	6	11	57	91	100
4	0	9	11	28	79	100

Normal Subjects.

	Agglutinins.	Dilutions. $\frac{1}{5}$	$\frac{1}{10}$	$\frac{1}{20}$	$\frac{1}{40}$	Control.
1	13 RT	4	8	12	88	100
2	13 RT	14	16	57	109	100
3	0 RT	31	59	93	104	100
4	0 RT	58	104	110	94	100

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Comparative Tests in One Subject.

	Dilutions.				
	$\frac{1}{5}$	$\frac{1}{10}$	$\frac{1}{20}$	$\frac{1}{40}$	Control.
Serum	5	5	6	47	100
Defibrinated blood	36	48	100	100	100
Citrated blood	88	109	107	115	100
Serum heated at 55° C.	100	100	100	100	100
Serum absorbed with specific organism	100	100	100	100	100

For an improved technique and a discussion of the problems involved see Mackie, Van Rooyen and Finklestein, *Journ. Path. Bact.*, xxxix, 1934, p. 89 ; *idem*, *Journ. Hygiene*, xxxii, No. 4, p. 494.

Sigmoidoscopy.—In theory, direct examination of the diseased intestine with the sigmoidoscope should be an indispensable aid to diagnosis, particularly the diagnosis of chronic dysentery. According to Biggam the sigmoidoscope reduces the possibility of a missed diagnosis from 30% to 3%.

The signs to be looked for are four :

(i) Shallow ulcers, sharply defined, but without a raised margin. They are small, rarely larger than 1 cm., round or serpiginous, with a granulating base.

(ii) Umbilicated papules.

(iii) White granulations on a red and thickened mucosa.

(iv) Polypi.

H. J. Smyly states that these signs are present (to the sigmoidoscope) in 93% of cases of Flexner dysentery, and the diagnosis can be clinched by direct swabbing of the visible lesions.

In mental hospital practice results are not nearly so conclusive. Sigmoidoscopy takes a good deal of time and labour : the preparation of the mental patient is peculiarly difficult and he invariably requires a general anæsthetic. In the hands of a colleague (Dr. S. H—) a group of known or suspected excretors showed the following results :

Visible signs of disease, swab positive	5
,, ,, ,, ,, negative	19
No ,, ,, ,, ,, positive	1
,, ,, ,, ,, ,, negative	25
Total	50

The absence of visible signs of disease is obviously of greater importance than the presence of visible signs, and the chief uses of sigmoidoscopy seem to be these :

1. To establish a test of cure in recent cases when symptoms have subsided.

2. To distinguish between transient excretors and chronic cases without gross symptoms.

3. To eliminate the possibility of the disease in cases showing suspicious symptoms but persistently negative bacteriologically.

The complete diagnosis.—The complete demonstration of a case of bacillary dysentery would include five signs :

1. Positive symptoms.

2. The excretion of muco-pus.

3. The recovery of the organism from the fæces or from the sigmoidoscope swab.

4. Visible lesions.

5. Positive agglutination.

No single sign, of itself, proves the existence of the disease ; all signs are not equally demonstrable ; and immediate diagnosis is not a scientific certainty—it is a matter of probabilities, depending on the combined art of the nurse, the doctor, and the laboratory. If the nurse is negligent, the doctor is ignorant, and the pathologist is blind. The recognition of any one sign demands prompt isolation and treatment. The other signs may be elicited at leisure.

Registration of cases.—There is one essential of co-operation ; that an adequate record be kept of cases, of suspected cases, and of suspicious symptoms. The method of registration advised is threefold :

(i) A hospital register of infectious and potentially epidemic disease : of proved cases only.

(ii) A ward (and possibly individual) register of suspicious signs and symptoms.

(iii) An individual laboratory record for each patient.

2. *Treatment.*

Diet.—This, in the acute stages of the disease, should be minimal, with abundance of fluid. Glucose provides sufficient nourishment for the first few days. Milk and albumen water should both be avoided, but watery arrowroot or Benger's food may be added as symptoms subside. The diet of the chronic case should exclude foods with much roughage, but should have a high protein content to avoid excessive fermentation and a good vitamin value.

We gave an artificial preparation of vitamins A and D to the patients of an infected ward over a period of two months without appreciable effect in raising the general standard of health or diminishing infection.

On the other hand a glass of milk daily caused a general increase in weight and appeared to diminish infection.

Medicine.—The standard treatment is still the exhibition of some inert salt, usually sodium sulphate. The initial dose need not exceed ʒvj in the first day; this dosage should be gradually tailed off during the next week. The efficacy of the treatment is undermined unless drainage is counterbalanced by abundant fluid, given, if necessary, into the veins.

Specific.—If a potent serum specific to the sub-type of Flexner can be obtained it should be given to any patient severely ill. Such sera being expensive and difficult to obtain, in milder cases an identical antigenic effect can be procured by the exhibition of intravenous vaccine. The initial dose of five millions on the third or fourth day of the disease does not accentuate symptoms; on the seventh day the dose may be doubled and continued weekly over a period. In the chronic case a dose of 10 millions is always safe, and the dose may be worked up to 50 millions. No dose, unless designedly provocative, should accentuate symptoms. The ultimate effect of vaccine treatment, in the acute case, is to prevent chronicity.

Lavage.—No specific treatment, however potent, will break down the avascular fibrous barrier surrounding a very chronic lesion. The essential adjuvant is colonic irrigation, which is really a form of internal massage plus drainage. The solution used need not necessarily be irritating or antiseptic. It should be sterile, warm, and slightly hypertonic. Irrigation should be practised daily on all cases showing visible lesions to the sigmoidoscope until those lesions disappear.

Test of cure.—There is no single test of cure. From the communal aspect, no known case of dysentery should be released from isolation until these criteria are satisfied:

1. The absence of symptoms.
2. The absence of muco-pus from the fæces.
3. Negative sigmoidoscopy.
4. Negative bacteriological examination of sigmoidoscope swabs, and of mucus, if present in the fæces, and repeated negative examination of the fæces.

Our actual procedure is this: The selected patient is given a provocative dose of vaccine—20 millions intravenously—and put to bed for observation. The stool is examined daily for 5 days, macroscopically for mucus, microscopically for muco-pus, and bacteriologically. If these tests are negative the patient is sigmoidoscoped. If no lesion is seen, and the swabs are negative, he is considered as a potential cure, and discharged from isolation.

This procedure may seem somewhat lengthy and somewhat complicated; it requires a certain amount of labour—but it represents, we think, an irreducible minimum, even although the proportion of rejects is very high.

3. *Segregation.*

The ideal is the rigid division of the entire population into three classes, infectors, immunes and susceptibles, with as great a space as possible between the first and the last. In a mental hospital such a division is not feasible.

Infectors.—There must be an isolation hospital containing two wards.

(i) The first ward contains only known excretors.

(ii) The second ward contains suspected excretors of two types: (*a*) convalescent cases, and (*b*) cases displaying suspicious symptoms.

The second ward will be larger than the first. All patients showing signs and symptoms should be transferred immediately to this ward, and treated as infectious until proved otherwise. The practice of using a sideroom in a general ward cannot be too strongly condemned. The general ward has not the staff or equipment for proper disinfection and isolation, and the most lavish display of cresol and Jeyes' will not prevent spread of the disease. There is only one rule; isolate at once in the dysentery hospital.

Susceptibles.—(i) The bulk of new admissions must be regarded as highly susceptible. They should therefore be kept apart from the older hospital population. The admission hospital, if it exists, forms an admirable unit for such a purpose. If there is no admission hospital, one ward, or a group of wards, should be set apart for admissions.

(ii) Transfers from other mental hospitals should not enter the admission wards.

(iii) The fæces of all new admissions should be subjected to a bacteriological examination, preferably after a provocative dose of vaccine has been given.

Immunes.—Chronic mental patients constitute a heterogeneous group with diminished susceptibility. The spread of disease can be limited by the division of population into as many self-contained units as possible: the smaller the average ward, and the more independent, the less will be the risk of infection. Ward transfers should be infrequent.

4. *Minimizing Conveyance.*

The conveyance of dysentery in mental hospitals being contrived almost entirely by human beings, the aim of efficient control is to break, at as many points as possible, the carriage of fæces from anus to fingers and mouth.

(i) The first requisite of any mental hospital is lavatory accommodation adequate, in type and number, to the peculiar needs of its population.

(ii) Such a provision must be accompanied by the education of the patient in personal hygiene. He must be trained to regular habit. He must be persuaded to be clean. He must be compelled to wash his hands after using the lavatory, and before each meal. If, despite attempted education, the patient is persistently dirty or faulty, a rigid antiseptic nursing technique

should be evolved, ranging from the sponging of anus and hands of the patient who is merely careless, to the isolation of degraded patients in specially equipped wards.

(iii) The improvement in personal hygiene should be supported by a rigorous campaign to promote the general cleanliness of the hospital.

(iv) The activities of working patients should be severely restricted. No patient should handle food. No patient should perform sanitary duties. Only selected patients should work in the wards, and under scrupulous supervision.

(v) The hospital building must provide space, light and air.

5. *Measures to Increase Resistance.*

(*a*) *Care of the alimentary tract.*

Food.—The dietary of a mental hospital is largely governed by considerations of cost, and the recommendations of the Committee on Dietaries in mental hospitals have been largely forgotten. Without recapitulating these recommendations, three principles of dieting can be stressed: digestibility, a high protein content, and an adequate vitamin value.

Teeth.—Septic teeth must be eliminated, and where dentures are impossible the diet should be modified.

Constipation.—This should be treated primarily by the exhibition of fluid, adequate exercise, and the regulation of habit. Laxatives, where necessary, should be given daily: practically nothing is more inducive to chronic intestinal catarrh than the large weekly dose of salts.

(*b*) *Specific immunity.*

Choice of antigen.—Absolute specificity is somewhat hypothetical, at least as regards the Flexner bacillus, for even within the sub-groups there are further antigenic variants of a complex nature, governing toxicity and capacity for growth, and modifications dependent on the environment of the organism *in vivo* and *in vitro*. The antigen, therefore, should be prepared from as many strains as possible within the type. Our practice is to keep 6 to 12 strains; as a new strain comes in, we scrap the oldest tube. Further, it is logical to include a high proportion of strains taken from acute cases.

Vaccines are made every 4 months to avoid excessive subculturing. We have rarely to pass our stock through broth, as the supply of new cases has been, until lately, good. The organism is sown on agar, incubated for 48 hours, and washed off in saline.

Killing.—The method of killing the organism should, as far as possible, avoid alteration of the bacillary antigen. The detoxication of the Flexner bacillus, with its feebly soluble toxin, is unnecessary. The killing of the organism by chemical agents—phenol, ether, chloroform, formalin, and the various salts—holds no obvious advantages. The antigenic value, for

instance, of a formolized vaccine is slight. Nor can we perceive the virtues of "sensitized vaccines". The simplest method is killing by heat at the lowest possible temperature. The Flexner bacillus is easily killed by exposure to 50° C. for 1 hour if the emulsion is well mixed and not too dense. It should be tested for sterility by plating, using several large plates. Very occasionally a further brief exposure to heat is necessary. The emulsion is diluted to the required strength. Only 0·1 % phenol is added as preservative. No vaccine is kept more than 6 months. If mass inoculation is contemplated, if there are no laboratory animals, and the pathologist is tired of acting as such, it is wise to try the effect of a small dose on a few patients.

Combined vaccines.—If specific immunization is adopted for a variety of infectious diseases, or types of one disease, either each antigen may be injected separately, or they may be combined in a single dose. The latter course, from the patient's standpoint, is more humane, but offers certain difficulties.

(*a*) Certain mixtures of bacteria do not form a uniform emulsion, *e. g.* Sonne dysentery and Flexner dysentery.

(*b*) The combined dose must be appreciably greater, or the individual antigenic response appreciably less.

In this hospital we were faced with the problem of four concurrent diseases: Flexner dysentery, Sonne dysentery, typhoid and paratyphoid. Our procedure was this:

(*a*) We did not attempt immunization with Sonne, because, first, it was the least important disease, and second, the emulsion does not mix with others.

(*b*) We used a mixed emulsion of typhoid, paratyphoid and Flexner, varying the proportion of the individual components according to our estimate of the individual activity of the disease. At present this is 1, 2, 2; thus, in the intravenous dose of 20 millions there are 4 million typhoid, 8 million paratyphoid, and 8 million Flexner; in the subcutaneous dose of 2,000 millions the same proportions hold.

Method of Administration.

"*Inoculation.*"—It is usual and customary to administer vaccines subcutaneously or intramuscularly; so much so, in fact, that a clinical habit has become a clinical principle. Vaccines are modified in all sorts of queer ways to allow "inoculation"; and if a vaccine does not produce immunity when administered thus, it is considered to be void of protective power. To our mind the simple "inoculation" of any bacterial product is a poor and uncertain method of obtaining immunity. The antigen of any pathogenic organism is primarily a tissue poison. When "inoculated" a quantity of a very powerful irritant is concentrated in the immediate area of the point of injection. There is a prompt inflammatory (and very painful) response, one object of which is to localize the irritating particles of the damaged tissue. The absorption of antigen, therefore, is slight, delayed and uncertain. The local reticular cells

may produce a minimal quantity of antibody, but there is no guarantee that any sufficient stimulus is supplied to the remote and more numerous cells of the reticulo-endothelial system. There is no certainty of a generalized immunity, and before that immunity can exist, there must be a long period during which the amount of circulating antigen is too small to stimulate the reticulo-endothelial system, and merely sensitizes other tissues—leaves the body, in fact, more open to infection.

We have employed intramuscular injection on the female side of this hospital (500 patients) for the past 18 months. The heat-killed emulsion of Flexner produces less local disturbance, and less constitutional disturbance (except for some very severe reactions which are inexplicable except as individual idiosyncrasies) than a corresponding emulsion of *B. typhosus*. It is possible, therefore, to employ a larger initial dose. We give 1,000 million to a healthy subject of average weight; the dose is modified according to weight and general condition. A second dose of 2,000 million is given after 7 days, and again every 4 months.

1. The formation of antibodies is variable; there is a lag period of 10 days, a slow and uncertain rise, and a comparatively rapid fall, corresponding to a mild attack of the disease, which affords some, but not complete protection.

During the lag period the subject is more liable to infection; it is therefore bad policy to inoculate subcutaneously patients in an infected ward; by so doing we caused a mild epidemic.

It is probable that maximum protection is afforded just after the maximum rise; the saturation of the tissues with antibody is slight; and the protective effect transient. The injections of vaccine must therefore be frequent, and continued over a long period.

2. There has been a fall in the number of cases; in 18 months there have been only 3 cases, against an average expectation of 15 to 30.

3. Two cases were very mild; the last was very severe.

Oral and rectal vaccines.—In point of historical fact oral immunization preceded inoculation, although its revival in practice, accompanied by the theory of "local immunity", is of comparatively recent date. The theory of local immunity as propounded by its chief advocate is, briefly, that different organisms have an affinity or predilection for different tissues, and that by saturating the particular tissue with specific antigen the receptivity of that tissue is exhausted, and infection is thus made impossible. Specific immunization, therefore, should follow the probable route of infection. This route, in dysentery, is by absorption from the alimentary tract.

Our results have been disappointing. We selected two wards (134 patients) in which dysentery was active, and gave to each patient 3 doses of heat-killed vaccine, each of 100,000 million, according to the prescribed technique, on a fasting stomach and preceded by a dose of bile. This procedure was repeated after 3 months. There was no diminution in the number of cases, or in their

severity, either relative to the previous 6 months, or to other infected wards, not immunized.

When giving Flexner vaccine per os we tested the blood of six patients from the second day onwards without demonstrating any agglutinins, except those present in some patients before administration. A fortnight after administration the blood serum of one patient was highly bactericidal, and the residual agglutinins had disappeared :

14.iv.33 : Agglutinins 25.			
28.iv.33 : ,, 0.			
Bactericidal test :	Control	784	100
	Serum diluted $\frac{1}{10}$	47	6
	,, ,, $\frac{1}{5}$	22	3

Intravenous vaccine.—In theory at least the route of choice would appear to be the intravenous. The antigen is widely and immediately distributed throughout the blood-stream, and there is a certainty that a high proportion will be absorbed by the reticular cells of the spleen, liver and bone-marrow. The maximum response of antibody is obtained with the minimum dose of antigen. The response is prompt ; the concentration of antibody is maintained in the blood over a long period, and presumably the tissues are endowed with a corresponding degree of protection against the disease.

In practice intravenous inoculation is just as simple as subcutaneous inoculation. It takes little more time in execution ; it is less painful ; there is no focal reaction ; there is less risk of sepsis ; and the systemic reaction can be exactly calculated.

We have used this method on the male side of this hospital (330 patients) for the past year. An initial dose of 20 millions is followed in a week by a dose of 40 millions repeated every 6 months.

1. The formation of antibodies is constant. There is an almost immediate rise of agglutinins, rising to a figure 10 or 20 times greater than that produced by intramuscular inoculation, and lasting longer. The production of antibody is, in fact, much greater than that produced by the actual disease. It is possible that the peak may be above the optimum figure of antibody concentration.

2. There has been a fall in the number of cases. The apparent fall is slight—from an expectation of 15 to an actuality of 6. The real fall is probably far greater. Intravenous inoculation lights up any latent disease, and the majority of cases recorded are certainly not new ; they have a previous history of diarrhœa, and they occur immediately following inoculation. The real fall is, probably, from 15 to 2.

3. All the cases recorded were very mild.

We have also successfully experimented with other vaccines given intravenously. Autogenous intravenous vaccines yield good results, without any

focal or systemic reaction in staphylococcal furunculosis, and *B. coli* pyelitis. The complete absence of focal reaction is very notable. For instance, in a very advanced case of tuberculosis (lungs and kidneys) a millionth of tuberculin A.R. given subcutaneously produced a very marked focal reaction, with exacerbation of general symptoms; ten times this dose intravenously was without appreciable effect; after four injections the patient could tolerate a hundredth milligramme.

On the other hand, where there is an avascular and very chronic lesion, as in the cholecystitis of the typhoid carrier, intravenous vaccine sends the somatic agglutinins rocking, but does not touch the excretion of the germ.

Mass immunization.—Whichever method of administration is adopted, and whatever antigen is employed, it is essential that each member of the community be equally immunized. There must be no exceptions. If the patient is ill the dose is decreased or delayed, not omitted. The only danger of immunization is that it may not be sufficiently thorough, either for the individual or the mass. Half-hearted immunization will simply increase the ratio of undiagnosed cases, and render effective treatment and segregation impossible.

It is especially necessary to immunize the most susceptible fraction of the population, the new admissions. Abolish susceptibility, and the disease must die. The inoculation of the "older" population is relatively unimportant; they are, relatively, insusceptible.

Finally, in a recently infected ward, unless the active excretors have been diagnosed, the only safe protective agent is an immune serum; intravenous inoculation is relatively safe; subcutaneous inoculation should not be employed until the active excretors have been isolated.

The value of specific immunization.—The degree of protection afforded is considerable, both in minimizing the individual severity of the case and of the number of cases, but it is not complete. To take a very recent example. A ward of 50 degraded female patients remained free from dysentery for a year. During that year each patient received 4 intramuscular injections of vaccine, each of 2,000 million. A solitary case of dysentery occurred. The ward was searched for excretors, without result. As a further precaution, the patients were inoculated intravenously, resulting in an average titre of over 100 R.T. A month later there was another case of dysentery. Within two months there were six, until at last the infector—a patient excreting very intermittently—was discovered, and the disease was checked. Now the infected patients possessed, apparently, a surplus of specific antibody, and the type of the infecting organism was identical, according to cross-agglutination, with the organism from which the vaccine was prepared!

Conclusion: Efficient Control.

On paper, the scheme of control suggested may appear somewhat formidable. The practical administrator of a mental hospital, viewing dysentery as only one of many infective diseases, and infective disease, in bulk, as only a minor

part of the whole administrative problem, will refuse to be intimidated by the formidable picture of the disease which we have drawn: he adopts the time-honoured principle of expediency. If the incidence of unmistakable cases rises, he segregates one, and notifies the other. If, perchance, there is an epidemic (very mild), he remembers that dysentery is a sanitary index, digs out a bad drain, and sends his drinking-water for analysis. The analyst's report (as such reports often are) is non-committal and indefinite, so that the expedient administrator, when the second epidemic comes, consults a pathologist, who prepares a vaccine, and stabs various groups of patients. When the third epidemic comes, he builds his own laboratory, and dabbles mildly in bacteriological diagnosis. He is surprised to find so few positives; but in the fourth epidemic he discovers a real "carrier" and descends in wrath upon the wards, with stringent orders that dysentery bacilli shall be confined to their proper place. And when those recalcitrant little pests refuse to do anything of the sort, and proceed to a fifth outbreak of epidemic disease, the expedient administrator asks the taxpayer for a new hospital.

The moral of this fairy-tale is that mere expediency is not enough; only a deliberate and systematized method of control can hope to eliminate endemic bacillary dysentery from a mental hospital. No single measure will suffice. The laboratory, by itself, is quite unable to check the disease. There must be knowledge of the disease-process. There must be a deliberate assessment of the endemic level of the disease. There must be prompt and accurate diagnosis, and a reliable test of cure. In those matters the laboratory worker is indispensable, though not by any means infallible; he is, shall we say, a competent observer and critic of events. The practical attack on the disease is in the wards and in the person of the patient. Diagnosis without efficient segregation is useless. Immunization is only complementary to segregation. The root-problem is that of conveyance: how can the standard of personal hygiene throughout the hospital be raised to such a pitch that fæcal contamination is impossible?

Working from the narrow angle of the laboratory, we ourselves are afflicted by a sense of complete failure. After four years' work, we seem to know a little more about the disease, and the incidence figures seem to be falling. Someone has said: "If one can find an institution in which dysentery is at a lower ebb than in some other institution where the environmental conditions are better, it does not follow that what has been done in the second institution was futile. . . . The Mills of God grind slowly." They do.

Cases.

The total number of cases observed was 87. Eight of these came to the post-mortem table.

Only those cases are quoted which illustrate the text. Few cases are as

complete as we would like them to be, for reasons obvious to anyone with a knowledge of mental hospital routine.

The post-mortems are similarly disappointing ; bacteriological examination of material is vitiated by the fact that the post-mortem was usually delayed until 36 hours after death, by which time the only organism likely to be recovered, if it exists, is *B. typhosus* or *paratyphosus B* from the bile.

E.M—, male. *A Sonne carrier.*

Admitted 25.ix.33. Fæces positive to *B. Sonne* on admission. Serum agglutinated *Sonne* at 1/25. No symptoms of dysentery. Died of intercurrent disease 10.vii.35. No p.m.

H.S—, male. *Chronic dysentery ; undiagnosed transfer.*

Transferred from another mental hospital 25.i.25. On admission fæces negative ; serum agglutinated *Flexner* at 1/25. Passed blood and mucus. Fæces positive to *Flexner Z* 31.i.35, negative 2.i.35. Serum on 7.ii.35 and 14.ii.35. Continued to agglutinate *Flexner Z* at the same dilution. Diagnosis : Chronic dysentery ; not a recent case, and not infected in this hospital. Differentiation of chronicity only made possible by Widal.

O. S—, male. *Chronic dysentery ; working patient.*

Symptoms of acute dysentery 16.ii.32 ; fæces positive to *Flexner Z*. Periodically suffered thereafter from occasional diarrhœa ; 7 consecutive examinations of fæces negative.

A worker in the garden, who often distributed fruit and vegetables, and therefore suspect. Sigmoidoscope examination 26.vi.34 : Mucosa unhealthy, with typical granulations and one small, but definite ulcer. Sigmoid swab : Douglas and Colebrook No. 8.

27.iv.34 : Fæces negative ; mucus negative ; muco-pus gave *B. Flexner Z*. Since then 4 negative fæces. Blood-agglutination constant at 1/25. Diagnosis : A very intermittent excretor. Case illustrated : (1) The intermittent excretor ; (2) the danger of the working patient ; (3) symbiosis of *Flexner* and D. and C. No. 8 ; (4) value of the sigmoidoscope.

J. H—, male. *Chronic dysentery.*

Acute dysentery 1.viii.31 ; fæces positive to *Flexner Z*. Thereafter 4 negatives. Fæces positive again 19.v.32 ; thereafter 9 negatives. Fæces again positive 13.ix.33 ; thereafter 7 negatives. 12.v.34 : D. and C. No. 8 recovered from a loose stool ; thereafter 2 negatives. 11.ii.35 : Following an intravenous injection of vaccine, fæces positive to *Flexner*. Case illustrates : (1) Intermittent excretion ; (2) value of intravenous vaccine in lighting up latent disease ; (3) interruption of D. and C. No. 8 into a *Flexner* series.

E. B—, male. *Chronic dysentery.*

First positive with symptoms, 14.x.30, *B. Flexner* ; thereafter 4 negatives. Positive again 15.xii.33 ; thereafter 1 negative.

Sigmoidoscopy 24.ii.34 : Mucosa thickened, dark and congested. Several ulcers, one large. Floor of ulcer scraped ; 2 swabs yielded D. and C. No. 8.

On 5.iv.34 fæces contained mucus ; D. and C. No. 8 isolated. 16.iv.34 : Fæces negative. 18.iv.34 : Fæces again showed D. and C. No. 8. On 24.iv.34 the fæces (ward specimen) were negative, but mucus gave D. and C. No. 8. On 7.i.35, following intravenous vaccine, *Flexner* was again recovered. Blood-agglutination steady at 1/25. Case illustrates : (1) The subacute case ; (2) value of sigmoidoscopy ; (3) potential pathogenicity of D. and C. No. 8.

F. C. H—, female, æt. 63. *Reaction to vaccine.*

Routine inoculation with dysentery vaccine (2000 mill. heat killed *Flexner*) 19.iii.35. Died of coronary thrombosis 20.iii.35.

Post-mortem 21.iii.35: The intestine was congested, particularly in the lower ileum, and the mesenteric glands were swollen. There was considerable mucous exudate.

E. C—, female, æt. 80. *Acute lesions.*

Admitted 1923. No history of diarrhœa. Four negative fæces. Contracted acute dysentery 14.v.34. Fæces yielded *B. Flexner* in almost pure culture on 15.v.34 and 16.v.34. Died 28.v.34.

Post-mortem 30.v.34: The whole of the gut from stomach to rectum was inflamed, with numerous small ulcers in the small and large intestine of recent origin; there was considerable sloughing, hæmorrhage, and a muco-purulent exudate. The liver and spleen were enlarged, and there were inflamed mesenteric lymph-vessels and glands. Microscopic sections of the liver and spleen showed intense congestion, with focal necrosis. It is only fair to note that the only organism recovered at the post-mortem was *B. paratyphosus B* from the bile. The patient was a carrier of this organism, and the acute lesions were dysenteric.

M. S—, female, æt. 40. *Acute lesions.*

Suffering from large gluteal abscess. Contracted dysentery 27.v.34; fæces on that date gave almost pure culture of *B. Flexner*. Died 29.v.34.

Post-mortem twelve hours later: Mucous membrane of large intestine and of small intestine irregularly thickened, with patches of dusky congestion. The lower colon was least affected. The only definite ulcers were in the ileum; these were of recent origin. The mesenteric glands were swollen. The spleen was soft and pulpy. Bacteriological examination: The blood-stained exudate yielded *B. Flexner*; the bile was sterile.

A. F—, female. *Chronic dysentery.*

Found excreting *B. Flexner* without symptoms 11.ix.32. Died 18.xii.33.

Post-mortem 19.xii.33: There was slight congestion and catarrh of the ascending colon, and similar changes in the lower ileum, where the lymphoid aggregations were swollen; near the ileo-cæcal junction there were two small ulcers. The lymphoid aggregations of the lower ileum were swollen; near the ileo-cæcal junction there were two small indurated ulcers. There were a few enlarged mesenteric glands. Bacteriological examination of the bile, intestine and glands negative.

L. P. W—, female, æt. 58. *Chronic dysentery.*

Fæces positive, with symptoms, 8.viii.32. Symptoms recurred on various occasions, but fæces invariably (6 examinations) negative. Last negative 1.vi.34. Died 5.vi.35.

Post-mortem 6.vi.35: Pus-pits and mucous cysts in large intestine; small intestine inflamed and congested, and coated with a muco-purulent exudate. Bacteriological examination: Bile and intestine, no N.L.F.

E. F. B—, female, æt. 58. *Chronic dysentery.*

History of intermittent diarrhœa with excretion of mucus. Three negative fæces. 31.i.34: D. and C. No. 8. 7.ii.34: Found excreting *B. Flexner* without symptoms. Died 20.iv.34.

Post-mortem 22.iv.34: There were many pus-pits and a few mucous cysts in the descending colon. The small intestine was healthy. Bacteriological examination of gall-bladder negative.

M. A. K—, female. *Transient excretor.*

While resident in infected ward found excreting *Flexner* without signs or symptoms, 29.iii.34. Blood agglutination 1/250. No previous history of diarrhœa. Scrupulously clean. Negative sigmoidoscopy. Ten consecutive stools free from mucus. Fæces, after 29.iii.34, persistently negative.

E. H—, female, æt. 62. *Transient excretor.*

Found excreting *Flexner* without symptoms 24.vii.29. Thereafter fæces consistently negative (over 20 specimens). Labelled a carrier (our mistake) in 1932, when dysentery broke out in the ward where she was resident.

Post-mortem 3.iii.33: Intestine absolutely healthy. A transient excreter, falsely labelled a carrier. Gall-bladder and intestine: No. N.L.F.

A. R—, male. *Chronic dysentery. In hospital stores.*

Worked in main stores, handling food. A single loose stool observed by a vigilant night-nurse, and a specimen sent to the laboratory. Fæces positive to *B. Flexner* 15.xii.34. Blood agglutinations: 20.xii.34, 1/25; 2.i.35, 1/25. An intermittent excretor.

M. E. G—, female, æt. 82. *Subacute dysentery.*

Admitted 1.ii.33. Exposed to infection. History of diarrhœa, with blood and mucus. *B. Flexner* recovered from stool on 5.iv.34. Treated vigorously with sod. sulph. and died on 18.iv.34.

Post-mortem 19.iv.34: The whole large intestine and the terminal 5 in. of the ileum were intensely congested and œdematous. There were a few very chronic ulcers in the descending and some pus-pits in the pelvic colon. An acute exacerbation of a chronic case. Gall-bladder and intestine: No N.L.F.

R. D—, male, æt. 80. *Chronic dysentery with negative fæces.*

Acute dysentery with positive fæces 18.ix.33. Apparently cured. No further symptoms. Six negative fæces. Died 17.ix.35.

Post-mortem 18.ix.35: Granulations in ascending colon; small mucous cysts and two small polypi in sigmoid colon. Probably chronic dysentery.

A. S—, male. *Chronic dysentery. A farm worker.*

A more or less independent worker on the farm. Previous and very definite history of diarrhœa. A week following intravenous vaccine, on 18.iv.35, he had an attack of diarrhœa, with mucus in the stool; fæces positive to *B. Flexner*. No further investigation. Isolated for a while, but now again working on farm. Probably an intermittent excretor.

A. T. P—, male. *Combined infection.*

Sonne: 24.x.33. *Flexner*: 30.i.35. Excreted *Flexner* following intravenous vaccine. Probably a *Flexner* carrier.

M. L. S—, female. *Severe reaction to vaccine.*

A new admission. Collapsed two hours after a first injection of 1,000 millions. She produced a series of rigors; that same evening the temperature rose to 106·2° F. The patient was very ill for a fortnight. Certain possibilities can be dismissed: (1) There were no signs of sepsis and the blood-culture was sterile. (2) She was not the subject of dysentery; the fæces and Widal were negative on admission. (3) The needle did not enter a vein; the maximum agglutination titre was delayed and comparatively low.

References.

Amer. Journ. Dis. Child., No. 35, June, 1928.—*Annual Reports of Chief Medical Officer of Ministry of Health.*—Archer, G. L. T., *Journ. R.A.M.C.*, July, 1933, lxi.

Besredka, *Immunity in Infectious Diseases.*—Biggam and Arafa, *Trans. Roy. Soc. Trop. Med. and Hyg.*, August, 1930, xxiv, No. 2.—Boyd, J. S. H., *Journ. R.A.M.C.*, October, 1933, lix, No. 4.—Brearley and Clayton, *Lancet*, September 11, 1926.—Brooks, S. C., *Journ. Lab. and Clin. Med.*, xiv, No. 2.—Burnet, Mackie and Wood, *Med. Journ. Aust.*, December 5, 1931, ii, p. 714.

Cann and Navasquez, *Journ. Hygiene*, 1931, v, p. 31.—Castellani, *Lancet*, August 24, 1929; *Journ. Trop. Med. and Hyg.*, April 15, 1933.—Charles, J. A., *Journ. Roy. San. Inst.*, liii.—Charles and Warren, "Bacillary Dysentery in Great Britain", *Pub. Health*, 1931, v, p. 44; "Dysentery in North England (355 cases)", *Lancet*, 1929, ii, p. 626.—Compton, *Journ. Infec. Dis.*, November, 1932, li.—Cowen, J., and Mackie, T. J., *Journ. R.A.M.C.*, xxxii, No. 3.—Cunningham and Theodore, *Ind. Journ. Med. Res.*, October, 1924, xii, No. 2.

Dawson, W. S., "Ætiology of Bacillary Dysentery in Asylums", *Lancet*, July 30, 1921.

Edgworth, F. H., *Brit. Med. Journ.*, 1917, No. 2933, p. 362.

Finlay, Leonard, *Proc. Roy. Soc. Med.*, May, 1934, xxvii.—Fyfe, G. M., *Journ. Hygiene*, 1927, xxvii.

Gardner, "A System of Bacteriology in Relation to Medicine", Medical Research Council, *Bacillary Dysentery*.—Gettings, H. S., *Trans. Roy. Soc. Trop. Med. and Hyg.*, February, 1915, vii, No. 4.—Gordon and Carter, *Journ. Path.*, 1932, xxxv, p. 2.—Greenwood, "Preventive Aspects of Medicine", *Lancet*, Epidemiology.

Harris, S., *Journ. Amer. Med. Assoc.*, 1928, xci, p. 1452.—Harjas Raj, *Ind. Journ. Med. Res.*, January, 1924, xi, No. 3.—Harvey, E., *Lancet*, January 28, 1933.—Hood, A., *Journ. R.A.M.C.*, May, 1932, lviii.

Ilitch, Z., *C.R. Soc. Biol.*, 1933, p. 112.—*Ind. Journ. Med. Res.*, June, 1934, xi, No. 3.

Jaffe and Hassin, *Amer. Journ. Dis. Child.*, March, 1933, xlv.—Jameson and Parkinson, *Synopsis of Hygiene*, 1934, 4th ed.—Johns and Chalk, *Canad. Med. Journ.*, July 29, 1933.—*Journ. Lab. and Clin. Med.*, March, 1933, No. 18.—*Journ. Ment. Sci.*, January, 1931, p. 77.

League of Nations Epidemiological Report, April, 1933.—Little and Bornstein, *Ind. Journ. Med. Res.*, April, 1930, xvii, No. 4.—Lemann, L., *Journ. Amer. Med. Assoc.*, December 18, 1920.

Mummery, Lockhart, *Lancet*, June 25, 1921.—Mackie, Margot, and Burnet, F. M., *Med. Journ. Australia*, 1932, i, No. 22, Type T9.—Mackie, T. J., Journ. *Hygiene*, April 15, 1919, xvii, No. 1.—Macleod, G., *Pub. Health*, London, 1921, p. 34.—Manson-Bahr, P., *Journ. R.A.M.C.*, xxxiii, No. 2.—*Idem, Journ. Med.*, September, 1925, xxxiii, No. 9.—*Idem, Journ. R.A.M.C.*, August 13, xxxiii, No. 2.—*Idem, Proc. Roy. Soc. Med.*, May, 1934, xxvii.—Manfold, J. A., *Ind. Journ. Med. Res.*, January, 1928, xv, No. 3.—McLean and Marsh, *Lancet*, September 8, 1934, p. 545. *M.R.C. Special Report* No. 40, "Bacillary Dysentery in Institutions".—*Ibid.*, No. 40.—*Ibid.*, No. 20.—*Ibid.*, No. 14.—*Ibid.*, No. 53.—*Ibid.*, No. 7.—*Ibid.*, No. 6.

Perry, *Trans. Roy. Soc. Trop. Med. and Hyg.*, April, 1929, xxii, No. 6.—Pickworth, F. A., *Journ. Path. and Bact.*, 1927, xxx, p. 627.—Powell, A. T. W., *Brit. Med. Journ.*, August 2, 1930.—Pratt and Frew, *Glasgow Med. Journ.*, February, 1930.

Reports of Board of Control.—Report of Committee on Dietaries in Mental Hospitals, 1924.—Richards, Marion B., *Brit. Med. Journ.*, 1935, i, p. 99.—Rogers, Leonard, *Ind. Journ. Med. Res.*, 1913, i, No. 2.—*Idem, ibid*, October, 1913, i, No. 2.

Saisawa and Janabe, *Philippine Journ. of Med.*, 1926, xxx (3).—Smyly, H. J., *Trans. Roy. Soc. Med. and Hyg.*, xxiv, No. 1.—Spence, J. C., "Ward and Dormitory Infections", *Lancet*, December 9, 1933.—Stanley, *Journ. Amer. Med. Assoc.*, 1930, xciv, p. 12.—Stewart, R. M., *Lancet*, August 20, 1921.

Taylor, *Ind. Journ. Med. Res.*, 1930, xviii (1).—Thjotta and Waaler, Erik, *Journ. of Bact.*, October, 1932, xxiv.—Topley, *Principles of Immunity*.

Watts, R. C., *Ind. Journ. Med. Res.*, July, 1929.—Webster, L. T., *Journ. Exp. Med.*, 1930, lii.—Whitehead and Kirkpatrick, *Lancet*, August 3, 1918.—Willmore and Shearman, *Lancet*, August 17, 1918.—Willmore, *Proc. Roy. Soc. Med.*, December, 1928, xxii.—Wiseman, W. R., *Lancet*, April 16, 1927.

Zinsser and Jones, *Text-book of Path.*, 1934.