Colitis and the Normal Colon Bacilli. By HAROLD L. MOONEY, L.R.C.P.&S.Irel., D.P.H., Second Assistant Medical Officer, Nottingham City Mental Hospital, Mapperley Hill.

THIS infection, which is mostly met with in those past middle age, the neurotic, and especially in those with intestinal atony, is particularly prevalent in mental hospitals and causes a high mortality. Although many so-called cases of colitis occurring in these institutions were in reality bacillary dysentery of the Flexner, Shiga and allied types, particularly so in former years, when sanitation was more defective, yet the relation of the normal colon bacilli, as causative organisms, to this disease must not be overlooked, and with a view to studying this relationship a short account of the *B. coli* will here be given.

In 1885 Escherlick, while investigating the earliest appearance of bacteria in the intestinal tract of newly-born infants, isolated an organism which he named *Bacillus coli communis*, which is found in large numbers in the intestinal contents of healthy animals through life, also in sewage, in water, and in milk by contamination.

Later it was found on closer examination that there were many varieties of bacilli isolated from these sources, differing from Escherlick's original *B. coli communis* and from each other in their action to certain reagents and in their cultural characteristics.

An attempt was made in 1904 by an English committee to describe the type most frequently found in the intestine, and they described it as a small motile non-spore-bearing bacillus, never liquefying gelatin, Gram-negative, capable of growth at 37° C., and producing acid and gas in glucose and lactose. It was added by an American committee that the typical *B. coli* must also produce indol and reduce nitrates.

Great numbers of colon bacilli, although closely resembling the *B. coli communis*, prove to be different organisms on the application of further biological tests, and they may be divided into four groups, as described by McConky, and as in most cases the organisms have not yet been named, they may be differentiated as belonging to either group.

Group 1. Organisms having no action on saccharose and dulcite. Example: *B. acid lactici.*

Group 2. Organisms having no action on saccharose, but fermenting dulcite. Example: *B. coli communis.*

Group 3. Organisms fermenting both saccharose and dulcite. Example: *B. coli communior* (*B. Freidlander*).

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Group 4. (Divided into four sub-groups.) Organisms fermenting saccharose, but having no action on dulcite:

(*a*) Those which do not liquefy gelatin and react negatively to the Voges-Proskauer test.

(*b*) Those which do not liquefy gelatin, but give positive Voges Proskauer reaction. Example: *B. lactis aërogenes.*

(*c*) Gelatin-liquefying and positive Voges Proskauer reaction. Example: *B. cloacæ.*

(*d*) Gelatin-liquefying and production of yellow pigment.

There are many less common types, one of which, the *B. neapolitanus*, was the cause of an epidemic of choleraic dysentery in Naples.

In healthy human fæces 85 *per cent.* of the organisms known as *B. coli communis* conform to the tests of that organism while the remaining 15 *per cent.* are atypical (Houston), while in sewage, and stored sewage-contaminated water, the typical *B. coli communis* undergo a relative elimination and the other varieties increase.

While *B. coli* are normally saphrophytes, they are also facultatively pathogenic and pyogenic, and when they have produced a pathological condition the serum of the affected animal will agglutinate the particular causative type and sometimes allied strains, so that the relationship between any of the forementioned organisms and a pathological lesion may, as a rule, be ascertained by agglutination reactions.

There are, therefore, a great number of types differing slightly from one another, and the theory has been advanced that they are but pleomorphic forms. However, this question of mutation has been studied, and it has been proved that some types may change or lose some of their characteristics under certain circumstances, and the closer investigation of this subject will be of great interest, as it may explain the number of strains isolated.

The *B. coli*, when confined to the normal and healthy intestine, have a purely saprophyte action. They become more numerous in the elderly, also in neurotic subjects multiplication is much favoured by atonic conditions. The organism becomes enormously decreased in numbers after ingestion of sterile food. They act on the derivatives of decomposition and digestive products, forming such substances as phenol, cresol, phenyl acetic and phenyl proprionic acids, skatol, indol, fatty acids, sulphur compounds and ptomaines. In the putrefactive decomposition of proteins by *B. coli* other substances are produced, which have the action of increasing the blood-pressure when absorbed from the intestines; these substances are named "urohypertensine" "iso-amylamine" and "parahydroxy-phenyl-ethylamine." Many cases of hyperpiesia leading to compensatory arterio-sclerosis and other vascular changes owe their origin to the chronic absorption of these substances from the colon.

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The free growth of *B. coli* and their development of phenol and the various substances before-mentioned undoubtedly inhibits the growth or destroys the more pathogenic organisms, and it is considered by some authorities that they produce some specific bactericidal substance.

This inhibitory action may be shown by the fact that in cases of specific bacillary dysentery, if the examination of the fæces be delayed more than a few days the specific organism will be found to be absent, and, conversely, when many colonies of pathogenic organisms are isolated from the fæces there is a diminution or absence of the colon bacilli. This inhibitory influence may be observed in typhoid and paratyphoid fevers, bacillary dysentery and cholera, and it has been found that the *B. coli* isolated from typhoid stools are more virulent than those from normal fæces, so that the general symptoms of typhoid fever may be increased by absorption of its toxins and possibly some of the pathological changes of typhoid fever are really due to *B. coli*. However, the serum of typhoid convalescents which will agglutinate the typhoid bacillus has no effect on *B. coli*.

While the action of the *B. coli* is normally saphrophytic and possibly protective, yet the *B. coli* are most important pathogenic organisms, and when injected into the serous cavities of rabbits are said to produce a fibrinous inflammation, becoming purulent if the animal lives long enough. However, if the virulence of the organism be high death takes place before suppuration owing to a septicæmia. Injection into the peritoneal cavity of guinea-pigs may lead to death, or the formation of multiple abscesses, or death following cachexia. Intravenous injections of broth culture produce a septicæmia and scattered hæmorrhages in various organs.

B. coli infection is frequently the cause in the human subject of suppurative conditions, especially in connection with the intestine, appendix and urinary tract, particularly the pelvis of the kidney and bladder. Urinary infection is more common in the female and frequently more chronic in character.

The *B. coli* are also occasionally the cause of summer diarrhœa, of ulcerative and mucous colitis, infantile diarrhœa and some food poisonings, while one strain, the *B. coli communior*, if not identical with, is closely allied to, Freidlander's pneumobacillus, the causative agent of some cases of pneumonia and meningitis.

The great majority of asylum dysenteries are proved by bacteriological examination to be cases of colitis, *B. coli* types being only found, while only occasionally a true bacillary dysentery appears and *B. Flexner*, *Shiga* and allied types are isolated. Many true cases of bacillary dysentery are, however, frequently described as

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colitis owing to the inhibitory action of the *B. coli* in masking the more pathogenic organisms.

In this institution in numerous cases of colitis, presenting the usual clinical signs, *B. coli* types were the only organisms found, although the fæces and mucus were examined early and independently. It was further found that *B. coli* isolated from some of these cases were agglutinated in weak dilutions by the serum of patients recovering from this disease, but were not affected by that of healthy subjects.

It must be remembered that the *B. coli* tend to invade the organs and blood-stream after death, so caution is necessary before explaining these types to be the cause of a pathological condition.

In conclusion it appears that the *B. coli* types, while confined within the lumen of the intestine by a healthy and unbroken mucosa, are purely saphrophytic, acting upon the products of protein decomposition, producing hyperpiesic conditions in the adult, and perhaps acting as a protection from the more pathogenic organisms. On the other hand, when the mucosa of the intestine is once penetrated, in the case of the colon, probably through an erosion in the blind ends of the tubular glands, the organism becomes highly pathogenic, causing local inflammation, ulceration and hæmorrhage, and sometimes pathological changes in the various organs.

It would appear, then, in order to determine a means of prophylaxis and treatment of the main subject of these notes (colitis), we must find the most probable factors which lead to the penetration of the intestinal wall by these bacteria, so changing their nature from saphrophytic to pathogenic organisms, and they seem to be as follows: (1) An increase in number of the organisms brought about by constipation and atony, and possibly by an excessive carbohydrate diet, causing the swallowing of an increased amount of saliva, bringing the necessary oxygen to these aërobic bacilli. (2) An increase in virulence of the organisms by passage from patient to patient. (3) Weakening of the colon wall by catarrhal conditions brought about by errors in diet, "chills," and possibly by the chemical action of an excessive amount of the saphrophytic products, also by the presence of "bald spots" due to a former infection. (4) Direct trauma by undigested foods (haricot beans for example), foreign bodies, calculi, "intestinal sand," costive stools, etc.

If we accept the above conditions as the predisposing causes of colitis, the following measures should be adopted in the prophylactic treatment of this disease:

(1) Easily digested and nourishing food. The amount of fats in the dietary to be increased and carbohydrates relatively diminished. Expressed in terms of water-free foods, a daily dietary per average male patient of proteins 3 oz., fats 2 oz. and carbohydrates 13 oz. would

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appear to be well within the borderline of safety during winter months. (2) Warm clothing and an equable temperature to be maintained in the wards. (3) Regulation of the bowels. Atonic cases to have a special pill containing, for example, aloes, nux vomica, physostigmine and belladonna, to improve peristalsis by acting as a tonic to the unstriped muscle, by increasing reflex action and by depressing splanchnic inhibition. The property of aloes in particularly confining its action to the colon recommends its use. (4) Prevention of overcrowding and strict cleanliness. (5) Vaccines in recurrent cases.

Treatment of *B. coli* infections by vaccines, especially in cases of systemic infection, has been successful. An autogenous vaccine is preferable if all the strains have been isolated, but owing to the difficulty of this, a mixed stock vaccine would be as satisfactory, and should contain *B. coli communis*, *B. Friedlander*, *B. coli anaërogenes*, *B. acid lactici* and *B. lactes aërogenes*, the first-mentioned organism in the highest proportion.

As regards the oral administration of dead colon bacilli in capsule form little benefit can be expected, as it is an intestinal saphrophyte being introduced into its natural surroundings, though perhaps digested and modified by the gastric juice.

In cases of bacillary dysentery, typhoid and paratyphoid, the oral administration of living *B. coli* in keratin-coated capsules so as to escape the action of the gastric juice might have a beneficial effect on the disease on account of the inhibitory action of the colon bacilli before mentioned, but probably no more so than such an intestinal antiseptic as "salol," which produces the same disinfectant (namely "phenol") as the *B. coli* in free growth.

The experiment would be interesting but not without danger if we consider the previous statement regarding *B. coli* toxin absorption in typhoid fever.

Looking back at the forementioned probable predisposing causes of colitis, the following lines of treatment, once the disease is recognised, appear to be rational: (1) Soft, easily digested and sterile diet, milk to be well diluted and boiled at least twice, or proprietary dried milk powders might be substituted. It has been previously pointed out that there is an enormous diminution in number of the colon bacilli following a dietary made as sterile as possible. (2) Mag. sulph. one drachm *t.i.d.* By abstracting fluid from the intestinal vessels, a flow is established in the opposite direction to the pathogenic path of the *B. coli* and at the same time the organisms are regularly voided. Mag. sulph. is most conveniently administered in these amounts by making a solution of 3 oz. of the salt in a whiskey, wine, or other 25-oz. bottle, and giving 1-oz. doses. This salt, it must be remembered, is also the chemical antidote to the saphrophytic products

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cresol and phenol. A little acid sulph. arom. may be added with advantage. (3) Maintenance of warm temperature. (4) "Salol" as an antiseptic. (5) A vaccine as described to prevent systemic infection.

It must be remembered that it is most important before treating colitis on these lines to first ascertain by early examination of the fæces and mucous discharge in lactose bile agar medium, and with observation of the isolated organisms' fermentative characters on a few of the sugars, that the case is not one of bacillary dysentery.

In conclusion I would like to acknowledge the kind assistance of Dr. Brunton, the Medical Superintendent, in preparing these notes.

Clinical Notes and Cases.

Observations on the Treatment of Epilepsy with Borax and Bromide. By W. J. T. KIMBER, M.R.C.S.Eng., L.R.C.P.Lond., Senior Assistant Medical Officer, Herts County Mental Hospital, Hill End, St. Albans.

IN the treatment of epilepsy numerous drugs have been acclaimed from time to time by different observers as sovereign remedies, and with few exceptions each in its turn has been discarded as of little or no value. Borax (sodium biborate) has been advocated repeatedly, and results reported by some have been remarkably satisfactory, while others have not found it worth while to persist in its use. Can these wide divergencies of opinion be explained? It would seem that as regards borax an explanation can be found from an analysis of the following results.

In the cases under review the epilepsy was associated with either congenital or acquired mental disorder. They were not specially selected, apart from the fact that the fits were of frequent recurrence and did not show any marked periodic grouping. All were cases of major epilepsy, and in the majority the fits had been occurring for a number of years and had been unsuccessfully treated with bromides.

Treatment consisted of the administration of a draught containing ten grains each of borax, potassium bromide and sodium bromide in an ounce of water. This was given twice daily, after breakfast and at bedtime. The only other medicinal treatment given was an aperient as occasion required, while in a few cases 2 minims of liquor arseni-

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