

T H E S I S

For the Degree of Doctor in Medicine,
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Subject: DYSENTERY.

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DYSENTERY.

SYNONYMS. Greek, Δυσεντερία Latin, Tormina.
English, The bloody flux. German, Ruhr-Die rotha
Ruhr. French, Dysenterie - Tenesme - Flux de
sang. Italian, Dissentaria-Flusso. Spanish,
Disenteria, Camaras de sangue. Swedish, Roedsot.
Persian, شیکام رवेश (shekasteh del = broken heart)
or شیکام رवेश (shikam ravesh = the flowing of the
stomach).

DEFINITION.

As yet there has been no satisfactory definition of this disease evolved. Many attempts have been made with a varying degree of success. It is the old mistake of taking one of the most prominent symptoms and calling it the disease itself. Thus with this disease *δύς* = difficulty, and *έντερον* = the bowel. Anyone who has himself experienced, even in a slight attack of this disease, the sudden griping colicky pain followed by the imperative desire for defaecation, and then, the long continued straining before even a small quantity of mucus can be passed - giving momentary

relief only - will be able to appreciate and to at least a small extent sympathise with the man (whoever he may have been) who first called the disease by one of its most prominent and painful symptoms. One of the Persian names for dysentery further illustrates the natural tendency of the Ancients (when medical science was in its infancy) to resort to symptomatology in their designation of disease. They are struck more by the 'constant desire to go to stool' on the part of the patient, and by diarrhoea, and so call the disease, 'the flowing of the stomach or bowel', sometimes adding *بار خون* (bar khoon = with blood). Our old English term for the same disease, e.g., 'the bloody flux', corresponds exactly with the Persian synonym.

In these later days of more exact science, we are striving to find a more suitable (or more exact) designation of Dysentery, but at present with partial success.

Davidson's broad definition (1) covers a good deal of ground. "Dysentery is a clinical term connoting a complex of symptoms dependent on inflammatory, ulcerative and gangrenous lesions of the large intestine. It may be roughly defined as

a group of closely allied infective diseases, characterised by frequent mucous, bloody, or serous stools, by griping pains, (tormina) more or less straining (tenesmus) generally with retention of faeces."

This definition omits mention of any aetiological factor, neither does it include the so-called 'Climatic' dysentery - a catarrhal condition of the large intestine - which may, if prolonged, terminate in true dysentery.

In another article (2) the same writer speaking of the non-amoebic form of the disease says, "Our clinical conception of acute dysentery is that of an inflammatory disease of the large intestine, with or without fever, characterised by mucous, serous, or bloody stools, containing epithelium, débris, or sloughs of the bowel, accompanied by tormina and tenesmus." He qualifies this definition by pointing out that it includes too much - such as amoebic dysentery, toxic and secondary dysenteries, some local inflammatory processes due to obstructions and other mechanical causes, while excluding cases, which presenting themselves under the guise of diarrhoea, are nevertheless mild cases

of dysentery.

Osler (3) defines dysentery as "a clinical term embracing several varieties of intestinal flux - the acute forms characterised by pain, frequent passages of blood and mucus, the more chronic by diarrhoea alternating with constipation and a tendency to recurrence. Anatomically there is inflammation, and in the chronic cases ulceration, of the large bowel." His definition also omits mention of any aetiological factor, and curiously enough does not include the prominent symptom through which the disease first obtained its designation - namely tenesmus! Also, his definition might lead one to suppose that the ulceration of the large intestine was limited to the chronic forms of the disease - which is far from being the case.

A French author (4) says, "Le développement de la dysenterie est dans tous les cas subordonné à l'intervention d'un agent unique et spécifique" - that the disease is due to a specific microbe, hitherto undetermined. This is not so much a definition of the disease, as an attempt to solve the etiology of dysentery, a view, which if tenable,

would simplify matters considerably.

Others with better reason incline to the view that more than one pathogenetic agent is capable of producing acute forms of the disease (5). Davidson points out (2) "That it is quite certain that such specific agents do exist, but that until their presence be demonstrated they cannot be used as a basis of classification.

Ordinary micro-organisms of suppuration, putrefaction and sepsis play an important part in all forms of Dysentery. This fact renders it probable that any agent, specific or otherwise, whether parasitic, toxic or mechanical, which materially injures or destroys the vitality of the wall of the large intestine, will render the tissues more liable to be attacked by the pus, gangrene and sepsis exciting organisms which are present everywhere, and are ever ready to lay hold on partially devitalised tissue -- thus, a 'dysentery' in the etiological acceptance of the term, is set up."

That is to say, as in some diseases, e.g. tuberculosis -- Pneumonia, once given a suitable 'nidus' (which is generally obtained by lowering the vitality of the tissues) the specific microbe

finds a home wherein to rest, and to do its deadly work, even so with dysentery --**first** lessen the vitality of the intestinal wall, then it forms a suitable 'nidus' for all manner and kind of microbes.

Lafleur (6) gives a separate definition for Amoebic Dysentery, which is now held to be a distinct clinical type from the non-amoebic. He says, "Amoebic dysentery is a form of intestinal flux caused by a species of amoeba, characterised anatomically by ulceration of the large intestine, and clinically by a variable course often marked by periods of intermission and exacerbation; with a special tendency to chronicity and formation of abscess in the liver."

As we shall be discussing the subject of 'amoebic dysentery' more fully under the heading 'Bacteriology', I will here say no more than that Lafleur himself confesses (6) " That certain details in the pathological anatomy of amoebic dysentery can scarcely be explained without the co-operation of associated bacteria."

That is to say he believes that although the amoeba are the primary agents in 'amoebic dysentery' yet that they are not independent but depend

largely for assistance in their nefarious work upon the co-operation of bacteria. I would also add that abscess of the Liver may appear as a complication of non-amoebic dysentery, as well as of 'amoebic.'

In concluding these remarks upon the 'Definition' of Dysentery- I would re-iterate the fact that the term 'dysentery' is a purely symptomatic one, and must be taken in a clinical sense only. It is indelibly fixed in the mind of the non-medical world as the name, 'par excellence' for all severe diarrhoeas -- more especially if accompanied by the classical symptoms, colic and tenesmus, with passage of blood and mucus, but to the medical man it must always appear almost as indefinite as 'Cancer' or 'Fever'.

HISTORY. Perhaps before proceeding to discuss the etiology of dysentery, it would be better to give some slight sketch of the history of the disease as far as it is known. In Persia their knowledge of disease and how to treat it, is derived from the Arabians. They divide all disease into two classes, hot and cold, and have medicines to suit. They classify all European medicines as hot, and warn the people that if they drink our medicines, their stomachs and livers will shrivel up with the scorching heat of our drugs. Dysentery seems to have been known to them for many generations -- it is classified as a cold disease needing cold medicine, and water-melon is often freely given. As I have mentioned previously, the Persian synonyms of the disease are purely symptomatic. The Greek physicians were specially struck by the 'tenesmus' or awful straining before any stool could be passed in some forms of the disease, but the Persian or Arabian doctors were impressed by the colicky pain, and the frequent call to stool, and thus named the disease, 'the pain of the bowel' or, 'the flowing of the stomach'.

Dysentery was observed by Hippocrates,

mentioned by Herodotus, described first by Aretaeus and Celsus (37). Galen admits two kinds of dysentery -- hepatic and intestinal. Coelius Aurelianus defines dysentery as "Rheumatismus intestinorum cum ulcere." Celsus gives us our first pathologico-clinical description "Intus intestina exulcerantur -- ex hic cruor manat -- isque modo cum stercore, aliquo semper liquido, modo cum quibusdam quasi mucosis exernitur; interdum simul quaedam carnosae descendunt; frequens dejiciendi cupiditas, dolorque in ano est, cum eodem dolore exiguum aliquid emittitur, atque eo quoque tormentum intenditur."

Sydenham describes 3 forms:-

1. Gripes without stools, or dry colics accompanied sometimes with fever.
11. With frequent slimy motions attended with griping and usually with fever. This form he describes as a true bloody flux, even though there may be no passage of blood.
111. Along with the fever frequent bloody and mucus stools are passed, accompanied by griping and tenesmus.

Pringle says that "dysentery is a pestilential disease, an expression of the putrid decomposition

of the blood and humors under the influence of atmospheric vicissitudes, any fatigue, putrid miasmata, and contagion."

Zimmerman recognised two clinical forms of dysentery "the bilious and malignant."

Stoll had an idea that dysentery was allied to rheumatism, and said "Dysentery is a rheumatic fever, acknowledging the same material cause as that of rheumatism, that is, a serous matter, the humor of perspiration driven in by the cold." He said that he had never seen the disease develop except in persons exposed to cold when overheated.

The physicians of the 18th century overlooked anatomico-pathological lesions, paying special attention to the clinical part and nature of the disease. At that time there were two different and opposite conceptions of dysentery:-

I. A local disease of a fluxionary character and not contagious.

II. A general disease of a putrid character transmitted by contagion,

the former being the sporadic form of the disease, and the latter epidemic dysentery of armies on the march.

Cullen taking the second view defines dysentery as "pyrexia contagiosa, dejectiones, frequentes, mucosae, vel sanguinolentiae, retentis plerumque faecibus alvinis, termina-tenesmus."

At the beginning of the 19th century, the disease was regarded as a phlegmasia of the large intestine, characterised by different degrees of intensity.

Andral regarded fragments of mucous membrane in the stools as 'scales of concrete mucus' whatever he might mean by the term.

Chomel denied that there was any ulcerative lesions in dysentery.

Virchow defined the disease as a 'clinical syndrome' corresponding anatomically to a simple catarrh, or to a diphtheritic affection. His opinion was that "dysentery, as a rule, commences with a catarrh and only becomes diphtheritic under the influence of local causes, such as the contact of hardened faecal matter."

Trousseau alone remained faithful to the traditional view of the disease, referring frequently to intestinal ulceration and dividing epidemic dysentery into 4 clinical forms -- inflammatory,

rheumatic, bilious, and putrid.

Gely, Thomas, Masselot and Collin, the physicians who studied the regional epidemics of dysentery in France headed the re-action against prevailing ideas, and gave out their opinions even more strongly after completing their further study of the disease in Algeria and British India. Thomas as their spokesman says, "I recognised after carefully performing a considerable number of autopsies, that the opinions of the Ancients were very correct, whilst those of the Moderns are entirely wrong." Then he proceeds to admit as an undeniable fact that ulceration of the mucous membrane is an essential characteristic of the disease "just as the phylectenas are an essential character in erysipelas, the core in a faruncle, and pus in the phlegmonia of the cellular tissue."

Annesley also recognised the supreme importance of the intestinal lesions, and as regards the contagiousness of dysentery he admits its possibility "under rare circumstances."

In Algeria, Senegal and the Antilles, French physicians (Haspel, Canteloup, and Cambay) described the specific lesions of dysentery insisting upon

the presence of ulceration and false membranes, and also admitting that the lesions begin at the apex of the follicles. But at first these same observers went all wrong over the etiology of the disease ascribing it to cold, irregularity in diet, and especially as being a "partial manifestation of malaria."

Losch and Kartulis first suggested the microbic origin of dysentery. Councilman and Lafleur have lately been insisting upon a form of dysentery, supposed to be due to amoebae (vide Bacteriology).

Sodré (37) strongly criticises Lafleur's theory and says, "It would be more rational, if, while establishing relation of cause and effect between dysentery and the amoeba coli, if they, (i.e. Councilman and Lafleur) had at the same time proclaimed the specificity and unity of the disease, separating it from all forms of enteritis not proceeding from the same cause. To admit a dichotomy in the morbid process, to recognise the existence of non-amoebic as well as amoebic forms, seems to me absolutely unacceptable. Dysentery is one, and one only, whether considered from an etiological, clinical or anatomical point of view."

This is far too strong and dogmatic a criticism, dysentery in all its different forms, may be but one disease, or different forms of the disease may be caused by separate specific organisms. Even though Lafleur may be mistaken in his belief that amoebic dysentery is caused by the amoeba coli, yet (as I have pointed out under Bacteriology) there is abundant evidence to justify the clinical distinction between the two forms into non-amoebic and amoebic!

CLASSIFICATION of DYSENTERY.

In the Appendix to this Thesis, I have placed some of the classifications used by eminent writers on this subject, but in this Thesis, it will be more convenient to keep as closely as possible to the following:-

1. Non-amoebic dysentery.

A. Catarrhal form, sometimes known as Climatic dysentery.

B. Acute dysentery of which A. may be an initiatory stage, subdivided into two pathological forms:-

1. Purulo-gangrenous.
11. Fibrinous or Pseudo-diphtheritic.

C. Chronic dysentery.

11. Amoebic dysentery or Amoebic enteritis.

A third division used by some authors, viz., "Modified or Complicated Dysentery" seems to me to be unnecessary, as it would naturally fall to be considered under the more appropriate heading, "Complications of Dysentery."

I have also given the Catarrhal form a more prominent place than most writers, because I believe that in some countries, e.g., Persia, it is climatic, and really not a true Dysentery, although I am prepared to admit that in other cases, it seems to be the initiatory stage of Acute Dysentery. In Persia, the catarrhal form is the one form from which, almost without exception, Europeans just entering the country, suffer. It is, in fact, looked upon as a 'necessary evil' for the newcomer to endure. Sometimes it yields easily to simple treatment, at other times, it persists for six to twelve months, and then disappears entirely, and then the patient is said to be 'acclimatised.' I shall discuss it more fully later on.

As a synonym for 'Amoebic dysentery' I have used the term 'Amoebic enteritis' advisedly, avoiding designating it 'Tropical dysentery' because I agree with Dr. Lafleur (6) "That there certainly are cases of dysentery in tropical climates, in which no trace of the amoeba can be found.

BACTERIOLOGY.

I. Non-amoebic dysentery.

One, in the light of recent researches, would feel almost justified in terming this form of dysentery 'microbic'.

The intestinal canal, (more especially the large intestine) is the happy hunting ground of all manner of organisms, even in the healthy individual, and possibly many of them are not only non-pathogenic, but really beneficial and perhaps, absolutely necessary for health. Yet, it is amongst these same microbes that we expect to find the author (or authors) of this terrible disease of Dysentery which has killed off more people than all the wars that have ever been fought in the world's history.

About twelve years ago, the search for this colossal murderer of mankind seriously started. There is something humiliating in the thought of all microbic diseases, man in all his pride and strength brought to death's door, perhaps entering the portals thereof, through the agency of an organism, so minute as to require the high power of a microscope to be seen at all.

"In non-amoebic and amoebic dysentery alike

the common bacteria of suppuration are found in the stools, as indeed they are often found in the stools of healthy persons." (1).

Yet, these 'common bacteria' of suppuration, although they may not be primary agents, yet when once the mischief be started, they probably join in the destructive work with great vigour, and act as very prompt auxiliaries, in bringing about suppuration and destruction of tissue (necrosis).

A. 'Staphylococci.' All three varieties (viz., aureus, albus, and citreus) are found in dysenteric stools with equal frequency in both forms of dysentery (i.e. non-amoebic and amoebic). As far as is yet known, they are not specific, but as I said before, they probably bear no inconsiderable part in the destructive work when once the vitality of the tissues has been lowered by the action of the specific microbe.

B. Streptococci. Seem to play a more important part than the staphylococci. Experiments by Zancarol, Celli and Fiocca (7) shew that they may even be primary agents in some forms of dysentery. They succeeded in producing dysenteric lesions by administering by the mouth or rectum, pure cultures of

virulent streptococci derived from dysenteric stools. Zancarol (1) from these experiments believed that 'streptococci' were primary agents in the production of dysentery. Celli and Fiocca, however, while prosecuting their researches had found a bacillus which they believed to be 'specific', and so they relegated the 'streptococci' to the place of secondary agents with a very important function to perform, viz., to transform a harmless bacillus into one of the most virulent type, i.e. the *Bacillus Coli Communis* into the *Bacillus Coli Dysentericum*. How this thankless task is performed is as yet not known. Kruse and Pasquale (8) in the endemic dysentery of Egypt along with 'amoeba dysenterica' always found streptococci and other bacilli, which manifestly contributed to the evolution of the morbid process.

C.

Bacillus Coli Communis. This bacillus, or rather, a more virulent variety of it, or some closely allied organism, has been recognised as one of the most important pathogenic agents of Dysentery. Celli and Fiocca (7) have shewn that by itself it is capable of causing dysentery in animals, whether administered by mouth or rectum, and

that the toxins obtained from pure cultivations give rise to dysenteric symptoms and lesions, that the serum of dysenteric patients causes agglutination of the *Bacillus coli dysentericum*. They also have demonstrated that this bacillus, although capable by itself of giving rise to dysentery, is generally associated with streptococci, which exalt its virulence within the body and in faecally polluted soil. That is to say that they believe that the *Bacillus Coli Communis* with its virulence intensified by contact with, or action of the Streptococci becomes the ordinary specific microbe of dysentery which they have named the 'bacterium coli dysentericum.'

The *Bacillus Coli Communis* was one of the microbes constantly met with by Bertrand and Baucher in their researches (9), and was probably the bacillus found by Chantemesse and Widal (10) in the walls of the intestine -- mesenteric glands of one who had died of the disease, and in the stools of those suffering from dysentery.

Maggiora (11) found it in large numbers in every case he examined, and proved experimentally that it could produce dysenteric lesions in animals. Arnaud (according to Scheube (12) has also come to

the conclusion that this bacillus, with its virulence intensified by association with other microbes, or in some other manner, is the specific microbe of dysentery.

The verdict is almost unanimous, and the former harmless denizen of the large intestine, *Bacillus Coli Communis*, is now believed to really be the specific microbe of dysentery.

D. Bacillus Proteus. A small species of proteus has been occasionally found in the stools of dysenteric patients by Celli and Fiocca (7) which they found able to intensify the virulence of the bacillus coli communis (like the streptococci), and with which they have in some cases been able to produce dysenteric symptoms in animals.

E. Bacillus Pyocyaneus. Found abundantly in the stools of dysenteric patients in France, and less frequently in the chronic dysentery of warm climates by Bertrand and Baucher (9). Calmette (13) found this bacillus not only in the stools of dysenteric patients in Cochin China, but also in the blood, and ulcerations of the large intestines. He thought, that of all the microbes present in the stools, it alone was capable of reproducing

the lesions of dysentery in animals. He adds, however, that the pathogenetic effect of this microbe is strengthened by association with streptococci.

F. Shiga's bacillus. Shiga in 1899, working in Kitasato's Institute in Japan, isolated and cultivated a bacillus which is not found in healthy men or animals (14), but which is always present in dysenteric stools, in the lesions of the colon and rectum, and often in the mesenteric and retroperitoneal glands of those who died of dysentery, but never in the liver or spleen. His bacillus is believed to be identical with the bacillus coli dysentericum of Celli and Fiocca, as it has similar morphological characters.

In an epidemic of dysentery in the Lancaster County Asylum, reported by Gemmel (3), Goodliffe found an organism which had close affinities with Shiga's bacillus.

G. Ziegler's bacillus, (15). "In a local outbreak in Germany, he observed a small bacillus in the adenoid tissue of the mucosa within the lumen and under the epithelium of the crypts of Lieberkühn, and in the connective tissue, and the

increase of this microbe in the tissues was accompanied by inflammation, tissue necrosis and degeneration." Davidson (2) remarks, "Here we have an undoubted dysentery bacillus, but it has yet to be proved that it is the constant or common cause of epidemic dysentery." Perhaps, there is a special microbe for 'epidemic dysentery' and the bacillus coli dysentericum may be responsible only for the sporadic non-amoebic form.

H. Ogata's bacillus, (16). Ogata in 1892, found in the stools and ulcers of epidemic dysentery in Japan, a short bacillus, about a quarter of the length of the tubercle bacillus, with rounded ends and active movements, which formed greenish yellow colonies in plate cultures, and which produced dysentery when injected into the rectum of cats. It is uncertain whether this be the same bacillus that Ziegler found, or a variety of it. If it be the same, it strengthens my suggestion that possibly there may be a distinct specific bacillus for 'epidemic dysentery.'

I. Flexner's bacillus, (3). Flexner and Barker of the Johns Hopkins Commission for the study of Tropical diseases, found in the dysentery in the

Philippine Islands, a bacillus identical with Shiga's. It has also been found in cases of dysentery from Porto Rico. Flexner by subcutaneous inoculations of cultures has produced in rabbits a typical acute colitis. The organism agglutinates with the blood serum of cases of acute dysentery.

Flexner calls this bacillus the *bacillus dysenteriae*, and gives us the following note concerning it.

"*Bacillus dysenteriae*," Bacillus of average length of *bacillus typhosus*. Grows readily upon all culture media. Colonies upon gelatin, when fully developed, show a grape-leaf appearance. It does not liquefy gelatin. Sugars are not fermented and milk is not coagulated. The bacillus at first is slightly motile, but quickly loses its motility in artificial cultivation.

Flagella have not been demonstrated. Feeding animals on the bacilli, unless the intestinal tract is previously irritated with chemicals, has no effect. Feeding after irritation sets up colitis in cats and dogs. Intraperitoneal and subcutaneous injections into mice, rabbits and guinea-pigs are lethal. In the rabbit, subcutaneous injections

have, in some instances, given rise to extensive pseudo-membranous inflammation of the caecum."

J. Treille in Cochin-China has lately described a new organism the "Paramaecium Coli" which he suspects to be a cause of dysentery.

From this summary of our present bacteriological knowledge of non-amoebic dysentery, we are entitled to deduce the following conclusions and inferences:-

- I. That the normal healthy intestine has a great resisting power to disease since all the common bacteria of suppuration, i.e. Staphylococci (aureus, albus, and citreus), Streptococci, besides Bacillus Coli Communis, and a host of apparently non-pathogenic organisms as well as Amoeba may be found frequently in the stools of healthy persons.
- II. That it probably requires a lowered vitality of the walls of the large intestine even before the specific microbe of dysentery itself can start its destructive work.
- III. That this lowered vitality of the tissues may be brought about by a simple catarrhal condition of the bowel, by fatigue and other non-microbial causes so that the devitalised tissues then fall an easy victim to their microbial enemies.

- IV. Once given a weak spot, it is practically certain that by far the most important pathogenic agent of dysentery is the *Bacillus Coli Dysentericum*, which once known as the harmless *Bacillus Coli Communis*, now becomes virulent, and is probably identical with Shiga's bacillus.

- V. That *Streptococci* and a small species of proteus have the power of working this transformation in the character of the *Bacillus Coli Communis*.

- VI. That in addition to this special function, *Streptococci*, *Staphylococci*, and probably many other forms of bacteria 'lend a helping hand' in the destructive process going on in the bowel after it has been once started.

II. Amoebic dysentery.

By adopting this nomenclature, we do not commit ourselves to the definite statement that this form of dysentery is caused by the amoeba, for although some authorities have this belief, the result of investigations, especially in the form of injection experiments, are not conclusive enough to my mind to warrant any such dogmatic statement. But there is certainly sufficient evidence, as I shall endeavour to shew, to warrant our recognising 'amoebic dysentery' as a distinct form, upon purely clinical grounds.

The Amoeba. Lösch (17) of St. Petersburg in 1875 was the first to give an accurate description of an amoeboid organism which he found in the stools of a dysentery patient, but the organism seems to have been first described by Lambl (3) as long ago as 1859. Lösch's observation has been confirmed by various researches in different countries:—namely, in Europe by Hlava, Kartulis, Kovacs, Quincke and Mason; in Africa (Egypt) by Koch, Kartulis, Kruse, and Pasquale; in North America by Osler, Councilman and Lafleur (21), Musser, Dock and others; in Brazil by Lutz. It had long been

noticed that there was a special form of dysentery with a tendency to chronicity, an absence of the usual constipation and tenesmus, apt to run an irregular course with periods of intermissions and exacerbations, and the autopsies shewed that the chief lesions were in the caecum and ascending colon, while in Acute dysentery, the lower part of the large bowel and the rectum is generally the seat of the destructive changes.

After Lösch had found the amoeboid organism, subsequent reseaches discovered that in this special form of dysentery, the amoeboid organism was always present, in the stools, in the swollen submucous tissue of the bowel, in the ulcers themselves, and even in the lymphatics, shewing that they must at least be active agents in the dysenteric process.

Amoebae are met with in the stools of healthy people, and also in those suffering from Cholera, Typhoid Fever, and other inflammatory and ulcerative affections of the bowel. Schuberg (18) found amoebae in the loose stools of ten out of twenty healthy persons to whom he administered a purgative dose of Carlsbad salts, so that it seems as though any

simple irritation of the bowel will lead to their multiplication.

Amoebae seem to have a preference for warm climates; in some parts of Italy, Greece and Egypt, they are frequently present in the stools of healthy persons. This non-pathogenetic type of amoeba is called the Amoeba Coli, and is morphologically indistinguishable from the amoeba met with in dysentery, and which is known as the Amoeba Dysenteriae, although there seems to be a difference in their effects after inoculation into the lower animals.

According to Davidson (1) other species of amoebae, smaller and less readily recognised, are also frequently present both in the stools of healthy persons, and those suffering from various intestinal complaints--including dysentery--all of which seem to be harmless.

The question yet to be determined is, whether this amoeba dysenteriae is the direct cause of this special form of dysentery, or does it act indirectly being at first a harmless parasite of the normal intestine, which, like the bacillus coli communis is capable of becoming virulent?

The amoebae rarely contain leucocytes, but

often contain micrococci, bacilli, and their spores, also red blood corpuscles, hence it is contended by some that they have a phagocytic function, and that by engulfing and digesting the bacteria of dysentery, they convert what might have been an acute into a chronic process.

Morphology. The amoeba dysenteriae has been classified as a Protozoa in the class Rhizopoda. Lafleur (6) defines it as "a unicellular organism consisting of slightly differentiated masses of protoplasm, and under favourable circumstances, it exhibits spontaneous movements."

The amoeba dysenteriae is spherical (when at rest), has a faintly green colour, and may be easily distinguished from other cellular elements found in the faeces by its stronger refraction of light.

It varies in size from 6-35 μ more commonly between 12-26 μ .

Its border is well defined. The body consists of two portions:- I, An inner called the Entoplasma, which is of a darker colour and contains granules: II, An outer or Ectoplasma, which is homogeneous and looks something like finely ground glass, of a pale green tint. It has a nucleus

but it is very hard to detect or to stain (like the nuclei of all the Protozoa).

For staining purposes, portions of the dysenteric stools may be used, hardened in alcohol or Müller's fluid, then cut and stained like sections of tissue, the best stain being methylene blue.

The amoeba dysenteriae is mobile, and can either alter its shape, or it may actually progress, both phenomena being accomplished by means of Pseudopodia. The motion may be very slow and gradual, or at other times fast and jerky.

This mobility is affected adversely by cold and Sulphate of Quinine (solution of $\frac{1}{5000}$) in the latter case the movements are arrested immediately the solution is applied. Lafleur (16) gives the following rules to be observed for detecting active amoeba in dysenteric stools:-

- I. Warm bedpan to receive stools and keep the specimens at body temperature till after examination.
- II. Make your examination as quickly as possible before the stools get acid, for acidity kills the amoebae.
- III. In choosing your specimen take the portions of stools that usually contain the greatest

number of amoebae (e.g. when the faeces are soft and but slightly formed choose the small flakes of adherent mucus).

- IV. Keep the glass slides, cover glasses, and stage of microscope warm.

Kruse and Pasquale (19) describe the forms of degeneration preceding the actual destruction of the amoeba, namely a colloid and dropsical degeneration and a dissolution by separation of bud-like fragments of the body. That this third form is not a method of reproduction is quite certain because the nucleus remains undivided and unaltered.

Nothing as yet is known of nutrition, respiration and reproduction of amoeba. Red blood corpuscles having been often found in actively moving amoeba suggest that these are taken up as food by a process of inclusion, but the actual act has never been observed.

Quincke, Cunningham, and Grassi after giving calomel obtained resistant forms of the amoeba, and termed them "encysted amoebae."

Kruse and Pasquale (19) took dysenteric stools, froze them until presumably all the amoebae were dead, then after thawing injected them into cats producing thereby an amoebic enteritis. A similar

experiment with stools which had never contained amoeba produced negative results. These experiments strengthen the hypothesis that there are 'encysted amoebae' and this form may account for endemo-epidemic form of amoebic dysentery, which although rare, is still sometimes found in the Tropics.

Culture experiments. Have all proved unsuccessful, but in 1902 Celli and Fiocca (20) report having obtained pure cultures of the amoeba, and describe a life-cycle in two phases--the amoeba form and the cyst form. The amoeba multiply by division.

Inoculation experiments. The animals chiefly operated upon have been cats because they have been found most susceptible. Now it must be remembered that the cat is an animal in which even non-specific irritants may easily set up catarrhal and ulcerative inflammation of the large intestine, and as Dr. Davidson (1) says, "it has been too readily assumed in these researches that the inflammation set up in these various experiments has been dysenteric."

These 'experimental dysenteries' may thus be fallacious, due not so much to the amoeba dysenteriae, as to the fact that the injected fluid

acts as an irritant.

Three methods of inoculation may be used, (Lafleur (6)):-

- I. Per os, by feeding the animal with material containing the amoeba.
- II. By inoculating the small intestine (after a preliminary laparotomy).
- III. Per rectum, rectal injections with or without suture of the anal orifice.

The first method has never succeeded, probably because of the destructive action of the gastric juice on the amoeba.

The second method is fallacious, as handling the bowel, and the use of antiseptics during the operation, are in themselves sources of irritation to the bowel.

The third method has given positive results in the hands of several observers, especially Kruse and Pasquale (19).

They injected dysenteric stools or material from hepatic abscesses containing amoeba into the rectum of various animals for 24 to 28 hours.

In some cases, notably where motionless amoeba had been injected, no abnormal result followed.

In others blood-tinged mucus with actively motile amoebae appeared in the stools after the

second day, but there was no constitutional disturbance.

In a third series, with evacuations of a like character, the animals wasted and died after a variable number of days.

In both the second and third series post-mortem examinations shewed pathological changes in the large intestine.

Three inoculations with material from liver abscesses proved only to contain amoeba, were made with cats; in all three experimental dysentery was produced (Lafleur 6).

Source of Amoeba Dysenteriae. Unknown, its life history outside the body is still to be discovered. The mode of infection is probably by drinking impure water although Lafleur (6) mentions a case where he investigated the water himself, but found no organism resembling the amoeba.

Other organisms found in Amoebic Dysentery.
Kruse and Pasquale (19) out of 100 cultures from fresh dysenteric stools and the contents of liver abscesses, and (post-mortem) from several organs, these were found:-

Streptococci of various species in 50 per cent!!
Typhoid like bacilli - - - - in 25 " "

Bacillus Pyocyaneus and a bacillus resembling the pseudo-diphtheritic bacillus, and the *staphylococcus pyogenes*.

Post-mortem, the associated bacteria were always most numerous in the blood from the portal vein, and it has been suggested that they have been carried there by the amoebae.

None of these organisms were present so constantly, or in such numbers, as to suggest a specific relation to the dysenteric lesions. These cultures inoculated into cats gave negative results.

Lafleur (9) goes on to say that however "it must not be concluded that the bacteria found in association with the amoeba possess no pathogenetic properties whatever, but that they are not primary agents in this form of dysentery.

Kartulis (22) in nearly 500 observations, in every single case found the amoeba in the stools of dysenteric cases.

Kruse and Pasquale (19) in 40 out of 50 cases of the disease found the amoeba.

Councilman and Lafleur (21) in 13 out of 15, while in the remaining 2 cases the amoeba was found post-mortem.

Lafleur (6) remarks that in some cases the intestinal ulceration is latent, the motions being quite formed, perhaps with small flakes of mucus adherent to them, in which no amoeba can be found; in these cases the existence of dysentery is not suspected until an abscess of the liver occurs, in the pus of which got by means of an exploratory puncture, actively motile amoebae are found.

Dr. Davidson (1) in summing up the evidence for and against the theory of the amoeba dysenteriae being the specific factor in the causation of this form of Dysentery says:-

- I. "The numerous experiments of Kartulis, of Kruse and Pasquale, and others, prove beyond doubt that when dysenteric stools or pus from a liver abscess containing amoebae, are injected into the rectum of the cat, the amoebae multiply rapidly and induce a haemorrhagic and ulcerative inflammation of the bowel. Kruse and Pasquale succeeded in producing dysenteric symptoms by the pus of a liver abscess containing amoebae, but which was sterile as regards bacteria."

- II. On the other hand, as he goes on to point out "Casagrandi and Barbagallo got similar symptoms and lesions by the injection of hepatic pus containing neither amoebae nor bacteria." This rather strengthens the point that I have before emphasised, that

some of these "experimental dysenteries" are not true amoebic dysenteries, but (especially when cats are used for the experiments) dysenteries brought on by the irritation to the intestine caused by the inoculation of the fluid, perhaps they might be called "mechanical dysenteries."

III. "Zancarol has repeatedly produced not a dysentery only, but a dysentery complicated with liver abscess containing streptococci, by the injection of hepatic pus destitute of amoebae and sterile to culture." Here again it is evident either that the pus was not really sterile, or that the irritation caused by the injection caused the harmless streptococci present in the canal to become virulent, and that these had given rise to the dysentery and liver abscess. In other words another 'mechanical dysentery' where the chief aetiological factor was neither amoebae or bacteria, but simply irritation to the intestine caused by the injection, probably resulting in an intestinal catarrh, and thus affording a suitable 'nidus' for the streptococci.

IV. "It is still an open question whether the *Amoeba Dysenteriae* differs specifically, or at all, from the *Amoeba Coli*, and whether either of them can give rise to dysentery in the healthy bowel."

V. "In amoebic dysentery one always meets with the bacteria of ordinary dysentery,

especially streptococci and varieties of bacillus coli communis, so that in fact, it is a 'mixed infection'."

It has been proved that the amoebae disappear from dysenteric stools in the course of a few hours, probably due to the fact that the stools become acid after standing for some time. Schuberg (18) after his experiment before referred to (i.e. with Carlsbad salts in healthy people) came to the conclusion that "the amoeba is a normal and harmless parasite of the caecum and ascending colon; and that the reason for its non-appearance in ordinary faecal evacuations is the solidity and acid reaction of the contents of the lower bowel, which soon destroy it."

Deductions from the above experimental evidence.

1. "That the amoeba dysenteriae is the sole specific organism in amoebic dysentery." I am afraid that the verdict at present must be the convenient Scotch one 'not proven.' And to my mind, it is likely to remain so, until pure cultures of amoebae have been obtained from both healthy and dysenteric stools, and the experiments repeated with these.
2. 'That the amoeba coli is a normal and harmless parasite of the caecum and ascending colon,

but that once this part of the intestine has become devitalised, then by association with other organisms, especially the streptococci, it takes on a virulent form--the amoeba dysenteriae--and becomes the chief aetiological factor in causing the destructive lesions."

3. "That amoebic dysentery differs from ordinary dysentery merely in regard to the site of the lesions, and that this difference in site accounts for the clinical distinctions in symptomatology" noticeable in the two forms of the disease. When the lesions are in the rectum and lower part of the bowel, retention of faeces and straining are two of the most prominent symptoms. When the lesions are in the caecum and ascending colon, it is quite understandable why these two symptoms should be less marked or altogether absent. The chronicity and tendency to recurrence, two of the most marked distinctions of the amoebic form, might quite easily be accounted for by the more remote site of the lesions. Thus, in ordinary dysentery astringent injections can readily be applied and the whole lower bowel cleansed, but when the caecum and ascending colon are involved, this effective method of treatment becomes more difficult. Then the amoeba may be a normal and harmless (if not beneficial) parasite of the caecum and ascending colon. Its rare appearance in ordinary dysentery may be accounted for

by the proved fact that it cannot exist for long in an acid media, thus the retention of faeces in ordinary dysentery is probably unfavourable for its preservation. On the other hand its multiplication when the dysenteric processes take place in the caecum and ascending colon, may be for purposes of defence and not of attack, if it really does possess phagocytic properties, and would be comparable to leucocytosis in ordinary inflammations. This theory would also explain the great number of amoebae found round the ulcers in the forefront of the battle! The inoculation experiments are not conclusive, the experimental dysenteries obtained possibly being the result of irritation, and not of the amoeba.

Bacteria have often been found included in the amoeba, and some observers have concluded that the amoeba acts as 'carrying agent' transporting the bacteria to the liver through the Portal vein, but on the other hand, it may be that the amoeba is really engaged in its phagocytic work of destroying the bacteria.

If this theory be a correct one, the difference between the two forms of dysentery are not aetiological, but merely symptomatic due to the difference in site of the lesions. The exciting cause being, as in ordinary dysentery, any factor, mechanical or otherwise, having a depressant action upon the tissues of the bowel and the destructive lesions- being due to

microbes, especially streptococci, (the bacillus coli communis being more commonly found in the lower bowel). The reason that abscess of the liver is a more frequent complication of amoebic than of ordinary dysentery, may also be referable to the site of the lesions, the ascending colon being somewhat nearer the liver than the rectum.

So perhaps before long, it may be proved that we have been abusing our best friend, who is engaged in trying to preserve, not destroy, the life of its host, man.

I put forward this hypothesis with much diffidence, but personally, I shall feel inclined to put faith in it, until it has been conclusively proved that the amoeba is our inveterate foe, and not a friend in disguise.

ETIOLOGY.

This might well include the bacteriology of Dysentery which has just been discussed, but it is more convenient to treat them separately.

I. General conditions.

I. Geographical distribution. Dysentery in its sporadic, endemic, or epidemic form is a disease that at one time or another has been found everywhere, from Greenland in the North to Madagascar in the South. It has ravaged nearly every country in its epidemic form, and seems to flourish in the cold as well as in the heat.

In its endemic form, according to Dr. Davidson (23), its Northern limit is about 40° both in the Eastern and Western hemispheres, while its Southern limit in Africa is about 30° , and South America about 35° (vide maps). (*Appendix B*)

He also lays down the general principle that "Dysentery increases as we approach the Equator, although not constantly or uniformly," and in support of this statement he gives a number of statistics, that I have embodied in two maps. (vide Appendix B).

Another general rule seems to be that "high

temperature favours the development of dysentery, other factors go to determine the prevalence of the disease."

In India dysentery has always claimed a large number of victims; in the three large Presidencies of Bengal, Madras and Bombay, Madras easily takes first place for the general prevalence of the disease.

By 'going up to the hills' the general panacea for all manner of diseases in India, you do not altogether get rid of dysentery, although as you ascend, the disease becomes less prevalent.

Bryden, from statistics taken from soldiers in the Native Army (1867-76) gets the following results:-

<u>Percentage of deaths from</u>	
<u>Dysentery to the total mortality.</u>	
Bengal, in the hot plain.	= 25.3.
Gangetic Provinces.	= 16.9.
Meerut and Rohilkund.	= 12.0.
Punjab.	= 14.6.
Punjab frontier, (at an altitude of from 5 to 7,000 feet).	= 9.4.

Again Béranger Féraud (25) relates how in the year 1840 in Martinique, the troops had to be removed to an elevation of 1,200 mètres to escape Yellow Fever, but that the new station had to be abandoned four years later owing to their finding out

"that the dysentery of the hills was worse than the yellow fever of the plains."

I found myself that dysentery was prevalent in Kerman, Persia, although its altitude is 6,500 feet above sea-level, but I had to treat a far larger number of patients for the same disease in Ispahan (Persia) which is about 1,200 feet lower.

The following table bearing on the subject is interesting (1):-

In India ratio of admissions for	Per 1000.
Dysentery at stations less than 100 feet above sea-level.	= 41·9.
Under 500 feet.	= 32·5.
From 3,500-8,000.	= 18·7.
Above 8,000.	= 3·8.

There is no difficulty in understanding the prevalence of dysentery even at high altitudes. In Persia, for example, you broil in the sun all day long, then at sunset there is a big fall in the temperature, and the night is chilly, then if local conditions are favourable (or I should say unfavourable) you find yourself in for a dose of dysentery.

"No altitude affords security from dysentery unless it be sufficient to reduce the mean temperature of summer to that of temperate climates." (Davidson 2).

II. Influence of Season. In Persia dysentery is prevalent all the year round, but more especially during the spring and autumn months when there is the greatest difference between the day and night temperatures.

Out of the 705 accurately recorded epidemics collected by Hirsch (26):-

- 529 raged in summer, or in summer and autumn.
- 137 raged in autumn, or in autumn and winter.
- 14 raged in winter.
- 25 raged in spring, or in spring and summer.

In the Tropics, dysentery is usually most fatal between July and December, the highest death-rate being between October and December, and generally just as the temperature has begun to fall a little.

In Mauritius, according to Davidson, the month of greatest mortality from dysentery is August, although it is the coldest month of the year.

III. Fluctuations in Temperature and exposure to cold and wet and fatigue. Fluctuations in temperature are always dangerous even in temperate climates, but the risks incurred from being exposed to them in the Tropics are yet more serious. They used to be considered, (and I think not without reason) sufficient factors in themselves to cause

dysentery apart from specific infection. They certainly are still to be recognised as exciting causes, probably by their depressant action upon the tissues; so important indeed are they, that the specific organisms fail to give rise to the disease without their aid. In Persia variations in the temperature and exposure (which every would-be traveller in that country must be prepared to face), probably have a great deal to do with causing that catarrhal or climatic form of the disease to which I have previously referred. (vide Classification).

Pringle's historical illustration (27) is a good one--briefly it runs thus:-

"The army who had fought at Dettingen (June 1743) after marching all night, the next day had no tents and had to sleep on the battle-field exposed to heavy rain. The day following they marched to Hanau spent another night in a wet field without straw. In eight days after the battle 500 men were down with dysentery, and in a few weeks nearly half the men were ill or had recovered.

But a detachment of the same army remained with the king's baggage, and were camped about half a mile off the main army. These men had not lain wet or

been exposed to rain. This little camp almost entirely escaped infection, though the men breathed the same air, used the same victuals and drank the same water."

In this case it was clear that the exciting agents were cold, wet, and fatigue, a threefold link that possibly has nearly as much to do with the causation of dysentery as bacilli and amoebae! Anxiety and overwork are also predisposing agents in the etiology of dysentery.

IV. Soil. The geological formation of soil seems to have no influence upon the prevalence of dysentery, but its physical conditions play a far more important rôle. Limited epidemics seem to prefer water-logged soil, e.g., the lake districts of Sweden, the marshy provinces of Holland, in France Brittany, basin of the river Somme, and above all Morbihan (Kelsch and Kiener 1).

Soil contaminated with faecal matter, forms a most favourable 'nidus' for the disease. To this category belongs the outbreak in Cumberland Asylum, described by Clouston (28). In this instance the outbreak was traced to the emanations of sewage applied to the land at the distance of about 300 yards

from the ward where the dysentery occurred. Dysentery prevailed in the Wakefield Asylum in 1827-28; the whole sewage of the asylum was collected in cess-pools within a few feet of the wards. These cases suggest the idea that the disease may be air-borne.

Several cases have been reported from France by Kelsch and Kiener, in which the disease seems to have arisen from the drying up of ponds and lakes, thus exposing slime and mud to the action of the sun.

V. Impure water. This is a very fruitful source of the disease. One of the most necessary details in tropical life is to personally see to the boiling of your drinking water. The use of filters is not sufficient--nothing short of boiling your drinking water will ensure protection from water-borne diseases. When a year ago I was living in Yezd, Persia, we took every precaution possible to endeavour to get uncontaminated water for drinking, sending out daily about two miles where the water came straight from the mountains; yet recently I have heard that fifteen samples of Yezd water from different sources (including a sample obtained from the source that we used) had been sent to an expert in Bombay for analysis, and the whole fifteen had been

condemned as utterly unfit for drinking. Fortunately we always boiled the water, even when we thought it must be pure. It is to be little wondered at that dysentery and typhoid are so rife amongst Europeans abroad, for many of them will not take the trouble to use this simple precaution.

Manson (24), amongst other observers believes dysentery to be a water-borne disease, and certainly there is plenty of evidence to favour this view. Many instances are on record in which outbreaks of dysentery have been traced to a water supply contaminated with faecal impurities. One of the best known perhaps, being the outbreak in Metz, 1870, in one of the garrison regiments (29). The disease was confined to the one regiment, and the source of contamination proved to be their drinking water, drawn from wells contaminated with faecal matter filtering from latrines placed opposite and close to them. The wells were closed and the dysentery disappeared.

In 1881 (eleven years later) another regiment quartered there, used water from the same wells for drinking purposes, and the disease again broke out only to disappear again with the closure of the wells.

The outbreaks of dysentery in the Millbank

Penitentiary about 50 years ago was found to be due to faecally polluted water, derived from the Thames; a pure supply was provided and the disease disappeared.

The dysentery death-rate in the Navy has fallen from 12.7 in 1860 to 1.2 in 1880, coincidentally with the use of distilled water.

The awful loss of men in the late war in South Africa from dysentery might have been avoided if stringent rules had been enforced against their drinking unboiled water. Every man should have been given a daily supply of distilled or boiled water, and if men of any rank had been caught in the act of breaking these (unfortunately hypothetical) rules, they should have been severely punished. During a campaign it should be one of the chief duties of the commissariat department, under medical supervision, to see that the troops are kept well supplied with boiled or distilled water. There no doubt are many difficulties in the way of accomplishing this reform, but nothing should be allowed to prevent its becoming law. An officer once told me that the chief difficulty would be with the men themselves. After marching under a hot sun for hours, they become

almost delirious with thirst, and will drink from the first supply they meet with, whatever the penalty may be. Having been through similar experiences many times myself in the scorching sandy deserts of Persia I can sympathise with other men in the same circumstances; but, because a thing is natural, it does not follow that it is also right, and drinking un-boiled water in tropical climates is suicidal!! I would advocate lectures on ordinary simple principles of health to be held in each regiment by the medical officer in charge, for officers and men alike. They should be taught to drink as little as possible. I have been told that at the end of half a day's march in tropical climates, it is very easy to distinguish the seasoned soldier from the man just out from home, merely from an examination of their water-bottles. From the bottles of the men 'just out' every drop has disappeared, while some of the veterans have hardly drunk any, or very little of their supply.

It makes one very sad to think of the many homes made desolate during the South African war, solely from preventable causes.

Dysentery may be caused by the drinking of other water than that polluted with faecal discharges.

According to Hunter and Annesly (31) marshy and brackish water polluted with decaying animal matter will also cause the disease; likewise saline water or water containing irritant mineral substances; but this variety is more likely, I should think, to bring on diarrhoea and intestinal catarrh--not a true dysentery.

Thus unboiled water may cause dysentery either by being the vehicle for introducing the specific bacillus, or by bringing on a catarrh of the intestine, acting as an irritant, and thus predisposing the patient to the disease.

I should like to add that in Persia it is not to be wondered at, that the water is generally contaminated, the usual place for the village latrines being beside the village stream, which supplies drinking water to all the inhabitants of the place.

VI. Relation to Malaria. Dysentery and Malaria may occur in the same place, at the same time and even in the same individual, and the one disease seems to predispose in the same individual to the other, but they are perfectly distinct and separate diseases.

Manson (24) says that dysentery bears a close

resemblance to Malaria in geographical distribution and epidemiological points; thus it resembles Malaria in the following points:-

- I. Comparatively rare and mild in temperate climates (with exceptions).
- II. Endemic in certain spots where it appears in sporadic form, and at times in limited local epidemics it may extend beyond these endemic spots and involve more extensive tracts of country.
- III. Seems to like low-lying damp places.
- IV. In temperate climates seems to prefer late summer or early autumn.
- V. Tends to disappear coincidentally with the appearance of cultivation, drainage and improved sanitation.

In Persia, which possesses a high altitude and a dry climate, both malaria and dysentery are present. Some malarial patients of mine have developed dysentery as a complication, which yielded to quinine rather than to my usual treatment for dysentery.

In another case of a European lady in Persia suffering from a mild chronic dysentery extending over a year which resisted all treatment, examining her blood and finding there what looked like a malarial parasite (quartan) we put her upon quinine and she was apparently cured.

Davidson (23) on the other hand, believes that there is a kind of antagonism between the two diseases, if one comes, the other goes. He says that in the island of Guadeloupe, a town called Grande Terre was malarious but almost free from dysentery, while another town Basse Terre in the same island, was almost free from malaria, but suffered greatly from dysentery.

Again, Madras, a province which as we have seen suffers most of all the other Indian provinces from dysentery, suffers least from malaria.

As regards Madagascar dysentery was a very common and fatal disease there, before the appearance of malaria, and then it died out.

The island Rodrigues is free from malaria, but dysentery is very prevalent and fatal, (29.6 per cent of the total mortality).

Probably the most that can be said for the relation of malaria to dysentery is that a patient weakened by a malarial attack, if exposed to infection, is very likely to develop dysentery as a complication.

Haspel (2) records the fact that out of fifteen men arriving in Algeria from Rochefort, scarcely

convalescent from malarial fever, five died from dysentery within six months residence.

There are many non-malarious countries where dysentery is rife, but very few malarious countries where dysentery is unknown.

VII. War and famine. These factors go hand in hand with dysentery, and as a rule dysentery in its epidemic form.

Most of the statistics of this disease are compiled from Army medical reports, and therefore there is a fallacy that one must guard against, for they return simple catarrhs of the lower bowel arising from chill, indiscretion in diet, as true dysenteries.

War. 'Wherever there is a big war, there also is dysentery.'

In war dysentery three sets of factors come into play (1):-

- I. Conditions which predispose the system to the disease, e.g., fatigue, bad food, damp ground, overcrowding, etc.
- II. Conditions which more directly determine infection, e.g., faecal pollution of the soil and drinking water.
- III. Facilities for spread of the infection.

War dysentery is irrespective of climate or

season. It is also of a malignant type, with a great tendency to complications, and very fatal. It frequently assumes a contagious character.

During the Crimea (1854) it raged amongst our troops during the whole of a severe winter.

As regards the contagious nature of war dysentery Lombard (30) informs us that to whatever place the Russian troops returned after the end of the Crimean war, there dysentery broke out.

How to deal effectually with 'war dysentery' is a big problem. I have already suggested some measures, which if enforced, would most certainly be the means of saving many lives.

B.

Famine. Famine dysentery bears a close resemblance to war dysentery. It is equally fatal, subject to complications, and often of a contagious nature. Ireland and India suffer most from 'famine dysentery.'

In the Irish work-houses (1841-51), there were 50,519 deaths from dysentery, 20,507 from diarrhoea. (2).

In Naples during the famine of 1763, dysentery became epidemic.

India has suffered terribly during famine years

from dysentery; in 1897, a year of great misery and famine in the Central Provinces, the death-rate from dysentery and diarrhoea was more than four times that in ordinary years.

VIII. Food. Fruit, tainted meat, are predisposing agents to dysentery, acting as irritants to the intestinal canal.

II. Personal conditions.

I. Age. Dysentery attacks all ages alike; it is naturally more fatal in children, e.g., India 1878:-

	<u>Per 1000.</u>
Adults, dysenteric death-rate =	1.73.
Children, " " " =	3.84.

II. Race. Attacks all races alike, more fatal amongst the natives because they have less resisting powers, due to their manner of life and food.

III. Sexes. Nearly the same proportion in each sex fall victim to dysentery.

There is no acclimatisation for true dysentery. Perhaps for the first two years the European is more liable to contract the disease than for two or three years after, but after the fourth or fifth year the liability appears to increase rather than diminish.

While in what I term Climatic dysentery, after

the first six months or year in the country (speaking with reference to Persia only) the intestinal catarrh ceases often of its own accord, generally never to return.

ETIOLOGY of AMOEBIC DYSENTERY.

Very little as yet seems to have been ascertained as to the general aetiological factors of this disease, and what has been stated is chiefly negative.

Thus Lafleur (6) says "amoebic dysentery is more prevalent in warm regions. It is found specially in Egypt, and shews no sign of becoming epidemic even when there are endemic foci.

It is not contagious and overcrowding or insufficient food does not seem to predispose to the disease."

Observations upon the effect of soil and elevation have not been made.

The influence of water-supply is unknown.

Sex, age and occupation do not seem important factors.

MORBID ANATOMY. It will be many years yet before the pathological anatomy of dysentery in Persia will be able to be studied, and this for two reasons:-

- I. Persian dysentery is very amenable to treatment; during three years hospital practice in that country, I only lost one case, and he was collapsed at the time of

admission.

- II. Post-mortem examinations are impossible; if one was done, it would be at the risk of all the European lives in the country.

The pathological lesions of Dysentery are exactly what one would expect to find, given an inflammatory state of mucous membrane and the presence of suppurative organisms; as a result of their action one is hardly surprised to find the result to be ulceration, suppuration, necrosis and gangrene; while on the other hand if there be exudation into devitalised tissues, the natural result from the mechanical pressure would be necrosis of the injured tissue, ulceration from the raw surface left when the necrosed tissue sloughs away, and finally gangrene if the blood supply to the injured tissues is entirely cut off. One would be quite justified therefore in treating the morbid anatomy of dysentery under the heads indicated by the gross characters of the intestinal lesions, e.g., catarrhal, ulcerative and gangrenous dysentery.

But as this would be a somewhat cursory manner of dealing with the subject, I shall keep to the classification that I started with in this Thesis,

and describe the macroscopic and microscopic appearances of the different forms of dysentery under the appropriate heads.

I. Acute Dysentery.

I. Seat of Lesions. Dysentery is emphatically a disease of the large intestine, especially the Sigmoid flexure and Rectum, but also the lesions are found in the Caecum and ascending colon. The transverse colon often escapes altogether, but sometimes it is attacked by the disease, but not to the same extent as the parts above mentioned.

In the Cumberland Asylum outbreak (28), the lower part of the ileum was involved as well as the caecum and ascending colon, and it is interesting to remember that in this outbreak the disease was almost certainly air-borne.

The disease is generally limited by the ileo-caecal valve, and so strictly, that the upper part of the valve may shew signs of the disease while the lower portion remains healthy.

Yet Bleeker (31) records a case in which the disease involved the duodenum!

According to Dr. Davidson (2) the caecum is the starting point of that severe gangrenous form

of the disease met with in tropical malarious climates. This type prevailed during the Ashanti campaign of 1874 and the caecum was invariably found to be the seat of the disease, and was generally found distended with a blood-stained putrid fluid.

In other cases of this malignant form of dysentery, the whole tract from caecum to anus has been involved.

The starting point of the disease varies in different countries, and is probably not a matter of mere accident, and may depend upon the mode of infection.

I have wondered sometimes whether it is possible for the infection to take place "per anus", if so, it would explain many of the sporadic cases of dysentery amongst the natives--at least those of Persia. In Persia, when a native defaecates, after the passage of the stool, his method of cleansing his anus is by the application of water, which he carries with him in a native vessel. Now my hypothesis is this:- suppose a native whose tissues are in a state of depression from any of the predisposing causes, after the act of

defaecation, cleanses his anus with water, which is almost certain to be swarming with bacteria and very possibly also containing dysenteric discharges, is there at least not a possibility of his infecting himself? The liability of infection is increased by the fact that 50% of the Persians suffer badly from haemorrhoids (internal and external) for which they will not accept operative relief, and if they suffer at all from constipation or diarrhoea when straining at stool, not only do the internal haemorrhoids come down outside the anus, but also some of the rectal mucous membrane is exposed. If there be any truth in this supposition, it would explain the site of the lesions being so often in the rectum--at least amongst the Persians.

Béranger-Feraud (32) observed that in the Antilles, the disease as a rule started in the caecum and ascending colon, while in Cochin-China, it more often began in the rectum and sigmoid flexure.

II. General macroscopic appearances. If there has been perforation (which fortunately is a rare occurrence in dysentery) on opening the abdomen one finds faecal extravasation and general peritonitis. When a dysenteric ulcer does perforate, the

perforation usually occurs in the rectum or sigmoid flexure. The more ordinary course of events, when the ulcer has perforated through the muscular coat of the intestinal wall, is for a local peritonitis to be set up, leading to adhesions to another part of the intestine or to neighbouring viscera.

When there has been no perforation, the peritoneal coat of the large intestine may be healthy, hyperaemic or oedematous, or it may be coated with lymph, or dark patches, corresponding to the ulceration within, may be visible on the surface.

In the gangrenous form of dysentery, as I have said before, the caecum may be distended with fluid; otherwise it may look quite healthy--perhaps a little contracted, and its sacculation is less marked.

In rare cases the colon is hard and rigid to the touch resembling its condition in chronic dysentery; it is then due to the exudation that takes place in pseudo-diphtheritic dysentery.

A. Catarrhal dysentery. Ordinary catarrhal dysentery is rarely fatal, therefore we can only deduce its pathology from cases where the disease has proved fatal in one part of the canal, while in another it is only starting. The variety I have

named 'Climatic' may not be a true dysentery at all, for I believe even in very chronic cases, it only acts as a predisposing agent from its debilitating effect upon the patient's constitution. But it is quite certain that both the other forms of acute dysentery (i.e., the purulo-gangrenous and pseudo-diphtheritic) have to pass through an initiatory catarrhal stage, which to distinguish it from the Climatic form, I should feel inclined to term Inflammatory Catarrhal Dysentery.

In this form the chief lesions are streaks or patches of congestion of the mucous membrane, with tiny ecchymoses, the natural folds of the intestine being chiefly affected. (2). There is always a certain amount of thickening which distinguishes it from lesions found in acute diarrhoea. The solitary follicles may be enlarged, some abrasions of the mucosa may be visible, but generally there are no traces of real ulceration.

In a case described by Clouston (28) although there had been copious bloody evacuations during life, no trace of ulceration could be found at the autopsy. The woman had died after only two days' illness.

Microscopical examination. The epithelium is generally found to be shed; vessels are distended with blood, and there is an increase of white cells in the mucosa especially in the neighbourhood of blood-vessels.

In the crypts of Lieberkühn in mild cases the capillaries are engorged; in more severe cases there is a haemorrhagic or purulent exudative infiltration round and within the glands, but the glandular epithelium remains intact in this stage.

The solitary glands may be enlarged, the enlargement being visibly due to increase in the cellular elements of the follicles. If punctured a white secretion exudes containing chiefly small round cells.

Manson (24) describes the surface of the mucosa as being "softened, swollen, red, injected, perhaps eroded, and covered with a blood-streaked glairy mucus, which when found in the stools is so characteristic of dysentery."

In the more advanced condition of the inflammatory dysentery, the tissues around the follicles suppurate; the follicles deprived of their nutrition become necrosed and are extruded, leaving raw

surfaces which constitute the round ulcers, having for their base the submucosa.

There has been much discussion as to the 'primary lesion' in dysentery, some suggesting changes in the solitary follicles as being the primary lesion, but they probably play a passive rôle, becoming necrosed simply from having their nutrient supply cut off.

Others suggest that the 'primary lesion' is caused from the irritation set up by the specific organism causing an exudation to be thrown out on and into the mucosa, and from its pressure effects causing necrosis of the part, the necrosed mucosa being extruded and leaving a raw surface.

A third hypothesis brought forward is that the 'primary lesion' starts in the submucosa, forming small abscesses which protruding into the mucosa, cause pressure results, and burst through a pin-hole opening into the lumen of the gut, exuding a gummy pus; the ragged abscess cavity left is the ulcer which spreads rapidly in the submucosa. (24).

Davidson deprecates all this theorising over the primary lesion (23) and remarks that "granting that dysentery is always the result of a specific

cause, even granting (which is by no means proved) that it is in every instance the result of the same cause, it does not necessarily follow that the lesions produced must always be of the same nature, or be involved in a definite order of succession. Thus the dysenteric organism may produce very different lesions according to its intensity, and the tissues (mucosa or submucosa) primarily affected."

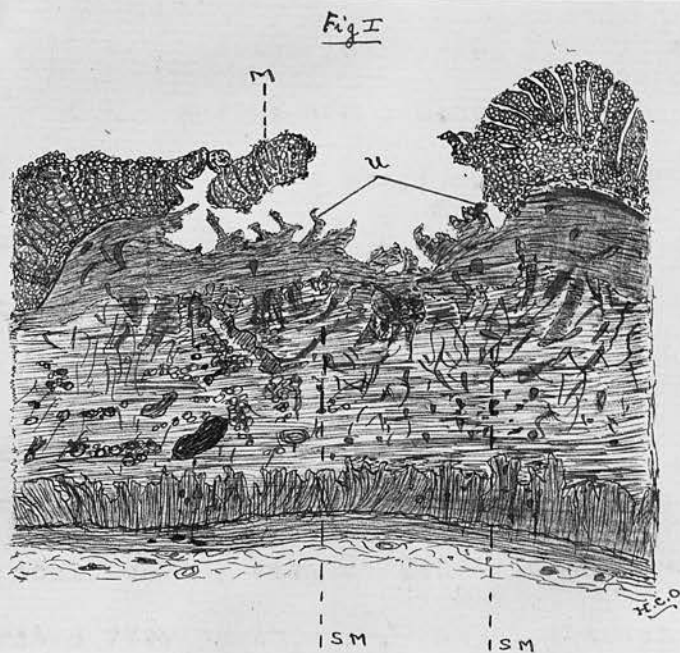
B. Purulo-gangrenous form. This nomenclature used by Dr. Davidson (2) expresses well the pathological lesions met with, but is really only a more advanced stage of A. The initial openings have been made and now all the suppurative organisms step in and make the most of their opportunity. Haemorrhagic extravasation and sero-purulent infiltration takes place into the sub-mucosa, causing it to become thickened; at the same time the muscular coat becomes oedematous. The affected patches are all colours--livid, dusky-red, yellowish-brown-- while the surface of the mucosa is seen to be irregular, the irregularity being due to localised sub-mucous collections of exudation. Then ensue the natural pressure results, necrosis and localised gangrene, the necrosed tissue forming sloughs of a black or

ashen colour adherent to, or partially detached from the sub-mucosa. If the gangrenous process extends, the whole of the mucosa and submucosa may become affected, and turned into a grayish, greenish or black sphacelated mass.

Extent of bowel affected varies from a small portion to the whole of the large intestine.

'Dysenteric ulcers.' If there be much necrosed or gangrenous tissue, no ulcers will be visible, but in less acute cases, various types of ulcers may be demonstrated varying in size, shape, character and amount of tissue involved. That most commonly found, is an ulcer oval in shape, transversely placed to axis of gut, which seems to originate from the transverse folds of membrane. Round, irregular or sinuous ulcers with ragged and undermined or raised, swollen, congested edges are also seen. Circular ulcers, varying in size from sixpence to a crown, with sharply cut edges may also be visible. These often coalesce. Ulcers with an irregular outline, and a soft shreddy base are probably the result of the discharge of sloughs.

See next page.



SECTION OF GANGRENOUS ULCER

- U. Shows the soft ragged base of Ulcer
- M. The shagreened, detached portion of mucosa
- SM. Thickened, oedematous, infiltrated submucosa

(From Davidson's "Hygiene of Warm Climates")

A dysenteric ulcer like a gastric one, may be superficial or deep, that is to say, it may only involve the mucous membrane, or it may go through all the intestinal coats and perforate the bowel; usually it goes no further than the serous lining.

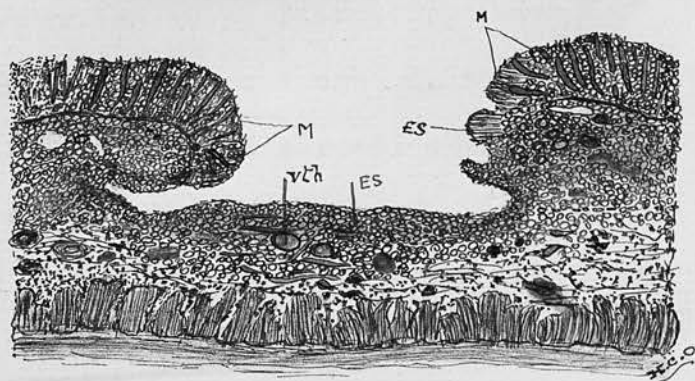
Microscopical examination. In advanced stages

little can be made out. In an earlier stage one simply sees an aggravation of the microscopical signs noted before in the inflammatory form; thus the solitary glands are now necrosed, and are visible ready for extrusion, if not already extruded, and their sites occupied by small ulcers. The crypts of Lieberkühn are bathed in pus, their epithelium detached, or their free ends destroyed; the glands themselves widely separated from each other or compressed. The submucosa is seen to be infiltrated with blood and pus; its connective tissue cells have disappeared. The muscular coat is seen to be infiltrated with pus and serum. (2).

Kelsch and Kiener (33) describe another form of ulcer in this form of dysentery which they say is the result of what they call the 'dry eschar.' These eschars appear on the mucous membrane both on and between the folds, and are really probably the same as the eminences before referred to, caused by a localised collection of exudation in the submucosa.

See next page.

Fig. II.



SECTION OF ULCER CAUSED BY THE DRY ESCHAR.

ES. Dry Eschar at base & Edges.

M. Underruned mucosa still retaining
the form of the elements of the tissue.

vth. Thrombosed vessel.

(From Davidson's "Hygiene of Warm climates")

The ulcers resulting from the disintegration of these eschars, involve the mucosa, but much more extensively the submucosa, and thus they have been termed the "ulcère en bouton de chemise" by the above authors.

Colin Chisholm (34) in his autopsies in the West Indies, along with gangrenous patches found tuberculous excrescences on the internal surface of the large

intestine resembling "very much the pustules of smallpox." I expect these were simply retained suppurative necrotic areas, which had never been extruded.

C. Fibrinous or Pseudo-diphtheritic form. (2).

This includes the form known previously as croupous dysentery. Dr. Davidson says that this designation includes, or rather assumes, three forms:-

- I. A croupo-fibrinous deposit on the free surface of the mucosa, which also involves to a greater or less extent, the mucosa itself.
- II. A fibrinous exudation in the mucosa, without any deposit on the free surface of the membrane.
- III. Exudation involving mucous and submucous coats.

If another title was wanted for this form of dysentery, one would feel inclined to suggest 'Exudative dysentery.'

In the first form the croupous deposit varies in colour, being grayish-white, greenish-yellow or rusty; and also in the extent of intestinal surface it covers. It brings about necrosis by mechanical pressure, and the tissue implicated is thrown off.

Viewed under the microscope the exudation is

seen to consist of amorphous or indistinctly fibrillated material, which penetrates the glands and compresses them or separates them widely. It' also invades the adenoid tissue of the mucosa.

In the second variety the exudation is non-croupous, but superficial and limited to the mucosa. Specks, streaks, or small patches of yellowish-brown or brick-red colour appear in the mucosa, generally on the summits of the folds. There is a general infiltration of the mucosa with white cells, granules and red blood corpuscles. The submucosa is thickened and also infiltrated with round cells. The bloodvessels of both coats are congested. The same old process takes place; necrosis of tissue which breaks down or is thrown off, leaving ulcers which tend to burrow under the mucous membrane and join neighbouring ulcers; the intervening tissue is destroyed by suppuration, or by being deprived of its blood supply.

The third variety is the most dangerous form of all. The Mucosa and submucosa involved at one and the same time. Bowel becomes rigid; may even feel semi-cartilaginous; the calibre of its lumen is often narrowed. The intestinal surface is uneven

and coloured yellow, green, red and black. It cuts like bacon, and the cut surface resembles somewhat the streaky appearance of that article of diet.

Microscopically much the same appearance is seen as in the second form. The exudation with pus and red corpuscles push their way between elements of normal tissue and the usual pressure results follow. The connective tissue of the submucosa is broken up, and gangrene ensues.

It is this third or pseudo-diphtheritic variety that has been so well described by Heubner (35) from observations made by himself and Virchow. He says that "on cross section the walls of the intestine are found exceedingly thick, but only two layers can be distinguished, the thicker one being the muscular coat, which is puckered inwards in many folds, and over it a stiff homogeneous tissue, partly yellowish and partly reddish, which offers considerable resistance to the knife. At places where furrows and cracks between the raised portions are situated, there is seen a small quantity of distorted tissue lying on the muscular coat, which is the remains of the mucous and submucous coats that have atrophied from pressure. . . . In this form gangrene is sure

to follow unless death occurs before this stage is reached."

As I have said before, it is rare in acute dysentery of the tropics to find the whole of the large intestine converted into a stiff, hard, solid tube. But that such cases are found is evident by the description of two morbid specimens exhibited before the London Medical Society by Fayrer (36).

One in which the gut is described as being diseased from the caecum to the anus--"it is swollen and rigid." Of the other specimen he says, "the whole intestine is contracted in calibre, abnormally firm, rigid and shortened."

When this rigidity and solidity of the gut is met with in cases of acute dysentery, it is due to exudation into the tissues of the intestinal wall.

II. Chronic Dysentery.

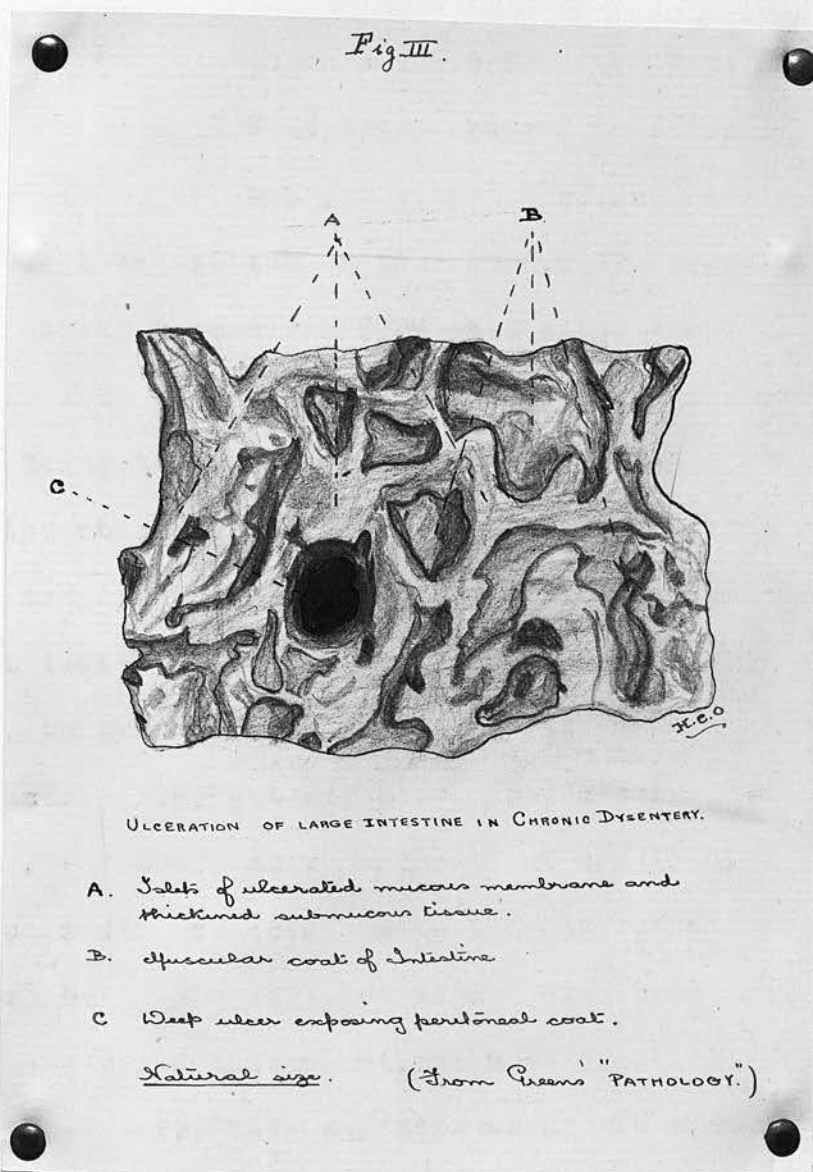
The determining factor between acute and chronic dysentery is unknown. An acute attack may become chronic, or the disease may be chronic from the outset. Some have suggested that it is the work of the much maligned amoebae, who by ingesting the suppurative bacteria, transform the acute attack into

dysentery of a chronic form, a dubious blessing, as chronic dysentery does not react so readily to treatment as the acute form.

The disease may become chronic when the vitality of the tissues is sufficiently lowered to allow inflammation to ensue, but yet retains sufficient vitality to resist the attack of the suppurative organisms, thus saving themselves from partial or complete death. The lesions visible in chronic dysentery are manifold many being the result of the acute attack. On opening the abdomen, adhesions to the neighbouring viscera are often visible. These adhesions are an attempt (often crowned with success) on the part of Nature, to guard against the perforation of a dysenteric ulcer. The gut may be visibly constricted in parts, the bowel above the site of constriction (which always coincides with the site of an healed ulcer) being generally dilated. The canal may be hard and rigid throughout, or more frequently at one part feel soft, thin and atrophied; in another doughy; in a third cartilaginous. (2).

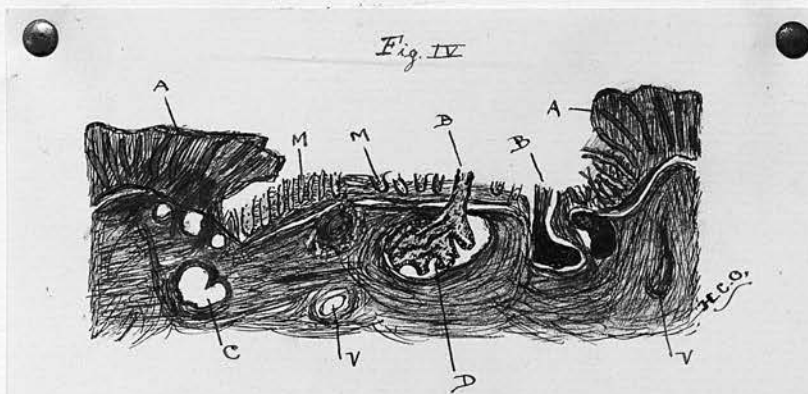
On the internal surface, the predominating lesions are ulceration and old cicatrices, the latter often pigmented. The ulcers may be few or many, large or

small, elevated or level with the mucosa, but thin edges are uniformly hard and indurated.



The general surface of the large intestine is slate-coloured, and the solitary glands, as a rule, are enlarged or pigmented.

Microscopically. Everywhere new tissue elements are visible; crypts of Lieberkühn are seen to be modified in form, being deformed, shortened, or having cystic dilatations, and being included in the solitary glands.



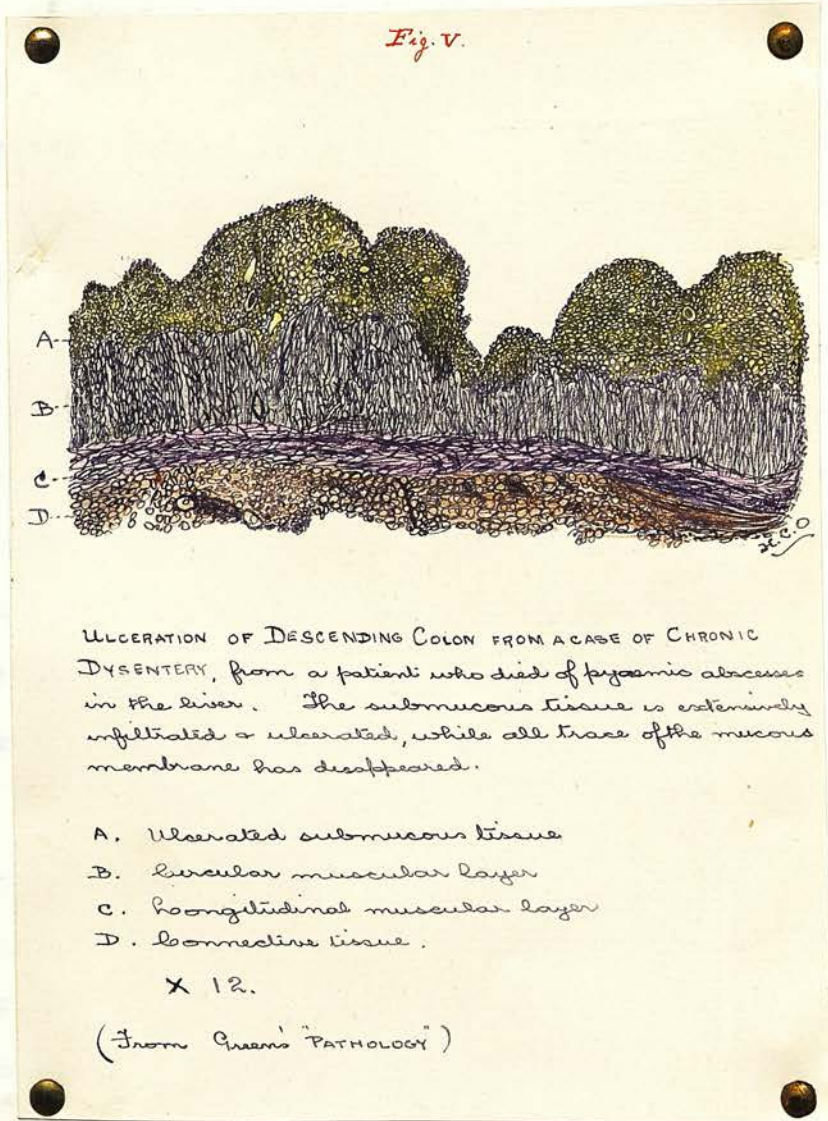
CHRONIC DYSENTERIC ULCER.

(MAG. 20. DIAMS.)

- A.A. Projecting portions of the mucous membrane with tubular glands.
- B. Masses of substance filled up with mucus, and cylindrical cells opening by a narrow orifice on the surface of the mucous membrane.
- C. Follicular depression visible to the naked eye & filled with mucus.
- D. Wall & contents of diverticula
- M.M. Tubular glands reduced to their cul-de-sacs situated in the ulcerated part of the mucous membrane
- V. Vessels.

(From Davidson's "Hygiene of Warm Climates")

The submucosa is thickened, fibrous or almost cartilaginous.



Cicatrization of dysenteric ulcers. (23).

The processes of repair are similar to those that take place in external wounds, but in ulcers of the

intestine they are rendered slower and infinitely more painful by peristalsis and the irritation caused by the passage of faeces over the sore. But in no case is there regeneration of lost granular tissue. If the loss of tissue is small, the cicatrix suffices to bring the surrounding mucous membrane over the sore, but by the contraction of this cicatricial tissue some puckering is left, which however, gets less in time, being replaced by pigmentation. If, on the other hand, the loss of tissue is extensive, process of repair is very slow, the mucosa is replaced by cicatricial tissue, which tending to project into the lumen of the gut, may cause obstruction, or distributed more uniformly round the gut, contracts, reducing its calibre.

III. Amoebic Dysentery.

The morbid anatomy of this form of dysentery is very similar to the lesions met with in other forms, although Lafleur goes very fully into the subject (6). The usual site of the lesions are in the caecum and ascending colon. The great distinctive pathological character, emphasised by Lafleur, is thickening of the bowel. This thickening is specially marked in the submucosa, and may be limited to it. "The

thickening consists of a general oedema and localised areas of thickening, which appear on the surface of the mucosa as nodules, and when incised are found to contain pale or grayish viscid material.

In a later stage, the mucosa over these nodules becomes necrosed from pressure, and is then cast off, leaving an ulcer which tends to spread by burrowing in the submucosa, and also by communicating with neighbouring ulcers." To my mind the whole process seems analogous to that already described and hardly seems worthy of separate notice. Thus the ulcers have ragged edges; bases sloughy, clean or granulating according to the stage in which they are found.

The microscopic appearance is the same as in Acute dysentery; engorgement of lymphatics and bloodvessels, small cell infiltration and oedematous thickening; the glandular structures are said to be only affected secondarily.

Amoebae are constantly seen to be present in the ulcers, generally confined to the submucosa; but, if the inflammation extends, they may also be observed in the mucous coat, the mucous membrane being generally free from them.

In the submucosa the amoebae are chiefly present in

the zone of oedema and small celled infiltration. The lymphatics and more rarely the veins also contain amoebae.

In the muscular coat the amoebae occupy exclusively the connective tissue of the intermuscular septa.

Lafleur says "wherever the amoebae are found the same changes are always to be observed in the tissues, namely oedema and infiltration with small round cells in front; while, in their rear, necrosis and liquefaction of the tissues."

Bacteria are often found with the amoeba, chiefly in the ulcers, and their presence is characterised by purulent infiltration. They are also sometimes seen within the amoebae.

The solitary glands are not generally affected.

The question remains, how do the amoebae get to the submucosa to start their foci? Lafleur suggests that their passage is through the interglandular substance.

It seems to me that the thickening of the mucosa, characteristic of amoebic dysentery, might well be analogous to the thickening that takes place in the tissues during any chronic inflammation, and might well be due to an attempt on the part of the amoebae

to stop the acute attack, and make the disease chronic, which as I remarked before, would be a blessing of very dubious value.

ASSOCIATED PATHOLOGY.

'Small intestine.' Often seen to be hyperaemic and injected, and in one outbreak already referred to (28), the ileum was observed to be actually involved. Bleeker (31) reports a case in which the 'duodenum' was also affected. Both the small intestine and the stomach are often found congested in cases of dysentery which have been complicated by malaria.

Liver. May be found normal, enlarged, or hyperaemic (23). May be the seat of solitary or multiple abscesses especially in amoebic dysentery, (19 per cent). In chronic dysentery the liver is generally found to be fatty.

Spleen. Should be found normal; if enlarged the dysentery has been complicated with malaria.

Heart and lungs. Generally found to be healthy; in chronic dysentery these organs are apt to become atrophied.

'Kidneys.' In acute dysentery they are generally found to be healthy; in chronic dysentery, one or other form of Bright's disease is commonly found.

Blood. In chronic dysentery, the number of red blood corpuscles is greatly reduced.

'Glands.' Of these the mesenteric naturally exhibit the most marked changes. In acute dysentery they become enlarged and hyperaemic; in the chronic variety they are found enlarged, pigmented, indurated, and sometimes contain small cheesy deposits.

In closing this branch of the subject, it is

interesting to note how in temperate climates, mental disease seems to make its subjects more susceptible to dysentery, several outbreaks having occurred in asylums, the lunatics being attacked whilst doctors and nurses, although exposed to the same infection, escaped free.

SYMPTOMATOLOGY.

I. Non-amoebic dysentery.

A. Catarrhal dysentery. For the present I shall leave the discussion of the symptoms of the catarrhal stage of simple acute dysentery as that falls to be discussed later under a different heading (vide Acute dysentery, first stage). Under the present heading I am going to give the symptoms of two forms of intestinal catarrh, which meet with but little consideration in text-books. The first form is probably not a true dysentery at all, although it may become so. I am going to term it "Climatic dysentery", and my remarks will be chiefly concerned with what I know of the disease after living three years in Persia. The second form is probably a true dysentery, but an abortive attack, and will be dismissed by me in a very few words under the heading "abortive dysentery."

I. Climatic dysentery in Persia. This is a diarrhoea with some dysenteric symptoms which greets the European new-comer, as a rule, when he lands on Persian soil, and it seems to make little difference whether he enters the country by the North via Russia and Caspian Sea, landing at Resht,

or from the South via the Persian Gulf and Bushire. It is certainly more apt to attack those who have left Europe for the first time, and thus may be looked upon as 'climatic', but probably not peculiar to Persia only. Amongst the chief exciting causes, change of climate is probably the most important. From the temperate climate of England with its damp moist atmosphere and ever present clouds, to the hot dry sultry Persian air, in the land of the sun, where clouds are a rarity much sought after, is a big transformation, and takes the body a little time to get accustomed to. Then in Persia the new-comer is subjected to sudden changes in temperature; as the sun sinks to rest in the west, the mountain tops blushing a rosy red in the afterglow, a chilliness as of desolation creeps into the desert air, and makes even the experienced traveller shudder and draw a cloak hastily around him; and that chill, that sudden drop in the evening temperature is greatly to be feared and well to be guarded against. It is the most fruitful source--amongst other things--of this dysentery, especially if the new-comer is travelling all night, as is the usual custom after winter is over. Then, not unlikely, the same man

who the previous evening was transported with delight at his first sight of a Persian sunset, will be in no fit state to enjoy the equally enthralling spectacle of the dawn breaking in the East, dispersing the dark shadows which seem to cling to the sides of the mountains as though praying for a little longer time to live, and heralding the approach and uprising of the sun in all its golden splendour. The unfortunate man will instead be in the throes of griping colicky pains, and will always have to be stopping the caravan or carriage in order to get relief, and will be only too glad to at last get to the end of his stage, and be able to lie down for a little.

Other predisposing causes are indiscretions of diet, e.g. eating too much fruit, exposure, fatigue. The bread also probably disagrees with the new-comer. It is made in varying sizes--in thin round flat slabs--baked in an open oven, the dough being first rolled flat, and then slapped on to the side of the oven. To the uneducated taste, it tastes something like thick blotting paper, but after a little while one commences to like it, especially when warm and fresh from the oven. But this bread

(which is brown) has a laxative action and may help to bring on this form of dysentery.

There is also undoubtedly a personal element amongst the predisposing causes, and that is the 'habit' of the individual before coming to the country. Thus if in England a man is constipated, he is not likely to suffer greatly from Climatic dysentery, at least in Persia; it is more likely that he will find (as I did myself) that the change of climate had cured the constipation, and that whereas he always had to be taking 'pills and potions' at home, that in Persia he has an evacuation once or twice a day naturally. If this be the case, as it most undoubtedly is, it follows that persons who are 'regular' in the homeland, will probably be troubled with diarrhoea in Persia, at least until they are acclimated, while those who in England suffer from 'looseness of the bowel' are not fit subjects for Persia, nor would I pass them for any tropical country.

The diarrhoea in this 'Climatic' dysentery may be only a 'bit of a nuisance', the bowels opening four or five times daily, and the patient suffering no great amount of pain or inconvenience, or it may become more serious, the motions increasing up to

twenty or thirty in the 24 hours, and the patient be-
-coming very weak and worn out. The 'stools' are
usually faeculent and copious, and lack the charac-
teristic mawkish odour of the 'dysenteric stool,'
but if the attack is a severe one, blood and mucus
may both make their appearance--the former in streaks.
There is occasionally severe colic, but more often
a griping colicky unresistible kind of pain seizes
the patient making him 'fly to stool' and leaves him
directly the 'stool' has been evacuated. As a rule
there is no 'tenesmus,' even in the severe cases,
and the whole catarrh may clear up in a few days un-
der very simple treatment, or if it persists the
disease tends to become chronic, the stools become
more watery, and the patient may become the subject
of real dysentery. Some people get acclimatised
(i.e. get rid of this climatic dysentery) very quick-
-ly; others are subject to the disease (with inter-
-missions and exacerbations) for weeks and even
months, but if it becomes chronic and persists over
the patient's first year in Persia, there is a seri-
ous risk of its becoming true dysentery, and the
sooner the patient is out of the country, the better
for him.

II. 'Abortive dysentery.' Perhaps hardly
deserves a place to itself, but having been a

sufferer from it myself, I am wishful to say a few words concerning it.

In Persia, at least during a time of any great strain of over-work or mental anxiety, one is apt to get obstinately constipated, with fleeting colicky pains which are relieved by going to stool, although all one manages to pass after a deal of straining is 'flatus'. The patient feels well, is able to do his work, and has rather a voracious appetite. The next day he is still constipated but after tremendous straining he manages to pass about a teaspoonful of gelatinous mucus, speckled with blood; or a table-spoonful of simple froth resembling the surface of bottled beer after it has been poured out. The patient still has fleeting colicky pains, and frequent calls to stool with similar unsatisfactory results. The second night he takes a good dose of calomel or castor oil, followed by 'salts' first thing on waking, passes a good motion and is cured. If however, unfortunately, the purgatives fail to touch this obstinate constipation, on the third or fourth day, in the evening, the patient feels a little nausea; he goes to bed and during the night wakes up suddenly feeling very sick--also a great

desire to go to stool. He gets up and passes a big motion, which may or may not contain a little mucus or some 'frothy abomination', and is violently sick, vomiting first the contents of the stomach and then bile. The vomiting is often very severe, and the resulting prostration great, but no more is seen of the dysentery. This always appeared to me an abort-ed dysentery, either yielding to the purgatives employed, or only yielding to the best physician of all--'Nature', the specific organisms of dysentery being perhaps killed by the action of the bile acids. Its tendency to appear at a time when a person suffers from 'brain-fag' is explainable on the grounds that very possibly the mental strain and anxiety act as a depressant upon the vitality of the tissues, and thus give the specific organism of dysentery a good opening to start work.

The treatment that I have found useful for these minor forms of dysentery will be indicated under the appropriate heading.

B. Acute dysentery. After a careful study of the subject, I cannot help thinking that the whole subject of the symptomatology of this disease would be greatly simplified if the different writers had

grouped the different symptoms under the headings "High and Low dysentery" or "Caecal and Rectal", because it seems to me that so many of the forms so minutely discussed would be 'understandable' if referred to their appropriate place under this classification. Thus under Caecal dysentery, owing to the site of the lesions, we expect certain modifications of ordinary symptoms, and we find them; also a distinctive character of the stools as opposed to Rectal dysentery, and we find them also! Also in studying the detailed description given by different authors (more especially Lafleur) of the symptoms peculiar to Amoebic dysentery, - I am afraid that I am heretical enough to believe that most of them are exactly similar to those found in cases of Acute dysentery where the lesions have afterwards been proved to be in the caecum and ascending colon. I have therefore drawn out two small tables, shewing the great similarity in some of the symptoms, first between Acute Caecal Dysentery and Acute Amoebic; secondly in ordinary 'Chronic Dysentery and the Chronic Amoebic form'. (vide Appendix to this Thesis C).

This probable difference in the site of the

prognostic conclusions can alone be drawn.

The history obtainable from individual cases varies. The attack may come on suddenly, especially if the dysentery be epidemic, and also in some endemic cases of warm climates where all the typical symptoms may be present from the first hour that the patient complains; in these cases the attack may be ushered in by a chill or rigor (1). More usually the patient will tell you that he suffered first for several days from obstinate constipation, or constipation alternating with diarrhoea. In yet other cases symptoms of indigestion accompanied the constipation, the patient felt 'out of sorts', not fit for his work, lost his appetite, felt some slight degree of nausea. All these premonitory symptoms may precede the development of the first or catarrhal stage of Acute dysentery.

I. Clinical symptoms of first stage, (Catarrhal). Patient complains of troublesome diarrhoea, preceded by severe colicky pain, and accompanied with a varying amount of tenesmus. At first the motions are loose and faeculent, then they become mixed with mucus and blood. If the lesions are situated in the Rectum and Sigmoid flexure, the tenesmus becomes

very marked, and after the passage of two or three faeculent motions, the character of the stools change. Now after very prolonged and severe straining the patient succeeds in passing from a teaspoonful to a tablespoonful of rose-tinted gelatinous mucus, which may sometimes be tinged with bile, the expulsion of which affords but little relief. The calls to stool are incessant; in comparatively mild cases 10 to 15 times in the 24 hours is a fair average; in severe cases as many times in one hour. The smaller the quantity of mucus passed the greater the straining. Very occasionally small hard masses of faeces (scybala) coated with slime are passed. If the dysenteric lesions are situated in the caecum and ascending colon, the straining is much less and may be absent altogether. The motions remain longer faeculent, are semi-formed, and more abundant, the calls to stool being less frequent, but they are preceded by gripes. In either form the ingestion of food or drink (especially if hot) provokes at once an urgent 'call to stool.'

During this stage there are very few constitutional symptoms, the patient becomes miserable, haggard, worn and irritable, but the temperature is

usually quite normal and the tongue clean. If however there is also malarial infection, the temperature may rise to 104° F. or even higher. Upon examining the abdomen, nothing very special can be made out, but there is generally a varying degree of tenderness or actual pain if pressure be exercised over the course of the large intestine. If the symptoms are not promptly arrested by adequate treatment, the disease goes on to the second or ulcerative stage.

II. Second stage, (ulcerative). The characters of the stools change, in 'rectal' dysentery they at once become watery, of a dark-red colour not unlike 'the washings of meat'; floating in them are seen the flesh-like lumps the 'raclure de boyaux' of the French physicians (2). Their odor is offensive, but characteristic, being unlike on the one hand the mawkish smell of the first stage, or on the other hand, the putrefactive or cadaveric odour belonging to gangrenous dysentery.

If the stools be allowed to stand, they deposit a sediment, which examined microscopically is found to consist of epithelium, blood corpuscles, pus cells, débris of tissue and sometimes small shreddy

sloughs.

The fluid part of the 'stool' is found to contain a varying amount of albumen. "The mean daily loss of albumen in a case of moderate severity estimated by Oesterlen as quoted by Woodward (38) at 50 or 60 grammes, which if correct, would account for the rapidly increasing debility of the patient."

The stools in 'caecal dysentery' go through an intermediate stage before becoming 'watery', after losing their faeculent character they generally become pale and frothy; one part of the same stool may be tinged bright yellow, another green and all mixed with blood and mucus. Over the caecum specially, there is a feeling of fulness and uneasiness, and there may be actual swelling, and pressure in the right iliac fossa giving rise to pain and tenderness. The stools may then become 'watery', of a chocolate reddish brown colour, and an extremely foetid smell.

In both forms the constitutional symptoms become more marked in this stage, slight evening rise in temperature may be noticed (generally not exceeding 100° F.;) pulse weak, fast and of low tension. Heart lesions are rarely found in dysentery, (but in an epidemic of dysentery occurring in Vincennes

during the summer of 1897, the cases were of a mild nature, but distinguished by a curious feature, namely out of 159 cases, 67 suffered from functional heart disease! The disease proved very infectious, 6 of the Hospital attendants being infected. The chief feature of cardiac disturbance was great irregularity. Post-mortem no organic lesion was found.)

The tongue is dry and may be glazed. Great thirst, loss of appetite, and there may be vomiting (especially in caecal form). Urine becomes scanty, may contain albumen and a few hyaline casts. The patient loses strength daily and becomes rapidly emaciated.

In the 'Rectal' form, the anus becomes inflamed, excoriated and the seat of a burning pain. At first the Sphincter ani is tightly contracted, but later it becomes relaxed, and prolapse of the bowel may occur from the severe straining. In this form also, 'bladder complications' are likely to ensue; strangury, dysuria and retraction of the testicles being the most prominent vesical symptoms. In the urine, bile acids may be found, and then the skin and conjunctivae take on a subicteric tint. Urea

and uric acid are in excess, the chlorides diminished. If treatment proves unsuccessful the disease proceeds to the third or gangrenous stage.

III. Gangrenous stage. Dr. Davidson (23) tells us that "gangrenous dysentery may start in two ways:-

A. With symptoms of simple form, e.g. colic, straining and scanty blood--stained mucus, being really the third stage of Acute dysentery, the symptoms from the onset having an intense character.

B. Insidiously as a diarrhoea; pain and straining not prominent. Stools copious, mixed with mucus and blood, sooner or later acquiring the characteristic cadaverous odour."

It seems to me possible that the explanation again lies in the indication of site, A being the final stage of the Rectal and B of Caecal forms of Acute dysentery.

A. The disease takes on a malignant character, 'the calls to stool' become incessant, and may amount to as many as 200 in the 24 hours. There may be troublesome vomiting from the outset. The stools become altered in character; they are thin, dark brown (not unlike an infusion of coffee) with sloughs ranging from scattered shreds of thin black

membrane, indicating gangrene of the mucosa, or thick black pus-infiltrated sloughs indicative of the destruction of both mucous and submucous coats. The sloughs commence to appear about the sixth or seventh day. There is often also a grumous sediment consisting of detritus derived from the necrosed tissue. The motions change daily in frequency, consistence and character. (For a complete enumeration and description of the sloughs passed, see Appendix D).

There is great prostration on the part of the patient. The odour of the stools is unbearably offensive, and well deserves the term 'cadaveric' so often applied to it. The patient may complain of a feeling of great heat along the tract of the colon, and the colon itself often feels doughy and thickened, and may be painful on pressure, although it is wise not to handle these parts much in this stage of the disease.

B. This form of gangrenous dysentery is probably identical with that described by Dr. Davidson (23) as having been rife during the Ashanti campaign in 1874, and which proved very fatal. In the autopsies, the caecum and ascending colon were

found to be the seats of the disease, and the former was found distended with blood-stained fluid, which was very foetid.

Babes has described the same form thinking it peculiar to Roumania.

Machiafava has described similar cases in Italy, and says that this form of dysentery is a "mixed infection of amoebae, bacillus coli, streptococci, staphylococci and other bacteria."

This form of gangrenous dysentery starts insidiously; stools are faeculent, copious, colic and straining not prominent. Gradually the stools lose their faeculent nature and become gelatinous and more frequent, though generally not exceeding 5 to 10 per diem. Frequently the stools are of a greenish-yellow colour and may contain clotted blood.

As the disease advances, the stools become more watery, of a reddish-brown colour, are extremely foetid and contain gangrenous slough. Vomiting may now appear, and be very severe even without there being any liver complication.

In both A and B after the gangrenous process begins, the pain gets less and not infrequently disappears, as the gangrene involves more and more

tissue. Troublesome hiccough may make its appearance before the end, and if the patient is in a state of collapse, it is a very ominous sign. Yet hiccough may persist for days in comparatively mild cases, without justifying a bad prognosis.

In these gangrenous forms of dysentery, the constitutional symptoms are necessarily severe, and the patient suffers from toxæmia. Death may ensue from asthenia, general peritonitis (if perforation has taken place) or collapse. Unfortunately the patient generally retains consciousness to the bitter end. During the last few days, the Sphincter ani becomes paralysed, and motions are passed involuntarily. Sometimes a big slough is seen extruded from the anus half-way, and the poor patient has not the strength to complete its expulsion.

The state of collapse is ushered in by the temperature becoming sub-normal, the patient's extremities getting icy cold, his skin becomes cold and clammy, his eyes are sunken, the pulse is fast, weak and thready, and he has no strength left to turn in his bed. The urine often becomes suppressed before death. "Sometimes before the end takes place, the patient seems to fall into a sort of

algid state with choleraic stools and muscular cramps; this is probably only an exaggerated form of collapse and not a real choleraic condition." (23).

Fayrer (36) speaks of a disease known as the "gangrenous colitis of Bengal," which seems to closely resemble gangrenous dysentery. He says "in this disease in the stools there is no branny mucus or sloughs, but a few days before death, a black or coffee coloured sphaclated object like an old sooty cobweb appears. Goodeve regarded this as sub-mucous areolar tissue separated after gangrene; death in these cases is inevitable. With these sloughs more or less blood is passed. The stools frequently resemble in colour and consistence 'cream mixed with blood' and they have a penetrating foetor. Post-mortem the large intestine is found lying like a dead snake in the abdomen."

These cases must be very acute, involving the whole of the large bowel almost at once.

Gangrenous dysenteric cases are not always fatal. Fayrer reports a case in which the patient passed a dark gray tubular slough 12 inches in length followed by another 3 inches long, and yet the patient recovered!

Microscopic examination of Stools in Acute

Dysentery. (2)

1st Stage. Composed of hyaline mucus, mixed with red and white corpuscles and epithelium from the bowel.

Later Stage. Consist of chiefly blood and pus, with sloughs and broken-down tissue, and also particles of undigested food. The micro-organisms already referred to may also be detected.

In connection with this, Sodré remarks (37):-

"Amoebae are almost always found in every variety of dysenteric stools, and in every period of the disease being usually most frequent in cases in which the lesions are most extensive." This is by far too dogmatic a statement, amoebae are often absent in dysenteric stools. In several of the hospitals during the South African war 1900-1902, in their reports concerning dysentery, they announced, "all the stools were searched most thoroughly and in not a single one of our dysenteric cases were we able to find the amoeba." (39).

Before leaving the symptomatology of Acute Dysentery I am appending a table shewing the changes in the stools of the 'Rectal' form as contrasted with the Caecal:-

Rectal form of Acute Dysentery.

- Stage I. Stools faeculent; very frequent; mawkish odour.
- " II. Rose-tinted gelatinous mucus; nauseating odour.
- " III. Stools watery; dark-red (meat washings) with special 'characteristic' odour; flesh-like lumps ('raclure de boyaux') floating about.
- " IV. Gangrenous; coffee coloured; thin membranous shreds, thick or thin black sloughs; odour cadaveric or putrefactive.

Caecal form of Acute Dysentery.

- Stage I. Stools faeculent, much less frequent than in 'Rectal' form.
- " II. Pale and frothy; bright yellow and green mixed with mucus and blood.
- " III. Watery, reddish-brown or chocolate coloured stools; extremely foetid.
- " IV. Copious reddish fluid with grumous deposit, mixed with shreds and sloughs; cadaveric odour.

It has been convenient to discuss Acute Dysentery in all its different stages, but this does not mean that the stages are clearly defined in every individual case.

II. Chronic dysentery.

'Diarrhoea alternating with constipation' is the most striking characteristic of chronic dysentery with exacerbations and remissions, the exacerbations lasting from 2 to 10 days. It may start insidiously with diarrhoea, which stops for a time and then recurs; after a varying interval mucus and blood are found in the stools, and the motions are passed with some straining, and preceded by colicky pains. After appropriate treatment, the patient is apparently cured, but looseness of the bowel remains which sometimes alternates with constipation. Then blood and mucus recur in the 'stools' and the chronic form of the disease is established.

More usually it is the outcome of an acute attack which neither proves fatal, nor is it entirely recovered from, but persists in a modified form; or in some other cases after the acute attack has apparently been cured, there appears to be a relapse and chronic dysentery is the outcome thereof.

During quiescence the stools may be well-formed and apparently healthy, but if carefully examined, very tiny flakes of mucus may be perceived upon the surface of the formed motion. As long as the stools

during quiescence exhibit these tiny flakes of mucus, there is always a likelihood, if not a certainty, that dysentery will reappear, exactly analogous to the fact that as long as one finds 'shreds' in the urine of a patient suffering from gonorrhoea, it is impossible to assure him that he is cured.

More frequently between the exacerbations the stools are thin, frothy, offensive, yellow, green, light-coloured, mixed occasionally with specks of blood and a little mucus. (Compare the character of faeces in Stage II Caecal form of Acute Dysentery). During exacerbations, the character of the stools change and resemble characters of stools in Stage III. Caecal form of Acute Dysentery, i.e., they are watery, of a reddish-brown colour, mixed with blood and mucus or pus, with foetid smell. Pain and straining are also present.

Chronic dysentery may go on, and on, and on--seven, ten, fifteen even twenty years, the patient living a life of absolute misery. He becomes anaemic; examination of his blood reveals a fall both in number of the red corpuscles and also in Haemoglobin; his tongue becomes red and glazed; he vomits occasionally; the pulse becomes accelerated and

weakened; his heart commences to fail; signs of backward pressure commence to appear, e.g., oedema of feet; congestion of stomach causes dyspeptic symptoms, e.g., fitful appetite, flatulence, undigested particles of food pass with the faeces. The patient then commences to complain of night sweats; gets rapidly weaker and thinner, and finally dies of Asthenia, if not carried off previously by some intercurrent affection.

The large bowel in chronic dysentery may become stenosed as the result of cicatrical contractions or its action may be impeded by adhesions; then the patient's abdomen becomes tumid and tender, great distress from feeling of distension, flatulent eructations, breath may become faecal, evacuations become scanty and are voided with difficulty, the patient's complexion takes on an earthy hue, and the patient rapidly loses strength and dies from exhaustion. (2).

III. Amoebic dysentery.

The resemblances in symptomatology between the acute and chronic forms of amoebic dysentery and the similar forms of caecal non-amoebic are so close,

that it would be mere 'tautology' to recapitulate them all again here. In the Appendix (C) will be found tables shewing this close resemblance. It is after all, what one would expect, remembering that in 'amoebic' dysentery, the caecum and ascending colon are primarily affected. It remains only for us here to consider the apparent differences between the two forms of the disease.

The onset may be so gradual, that the disease may not be suspected till abscess of liver supervenes; or, it may appear suddenly, the patient being seized with colicky pain and diarrhoea.

"It is characterised by an irregular course marked by seasons of intermissions and exacerbations, a tendency to chronicity, and the frequent occurrence of abscess of the liver." (6).

The periods of intermission vary from 1 day to 3 weeks; the periods of exacerbations resemble those of chronic dysentery, i.e., 1 to 10 days.

The disease has a great tendency to chronicity, (like all caecal dysenteries), rarely lasting less than three weeks, and 6 to 13 weeks being the ordinary range.

Like non-amoebic dysentery, the disease may be

acute or chronic. Davidson says (23), "A chronic state may be inferred when a moderate diarrhoea has continued for many weeks with or without intermissions, the stools being liquid, homogeneous, without blood and unaccompanied by pain."

The disease may have the same stages as the non-amoebic variety, that is Catarrhal, Ulcerative and Gangrenous, although they may not be well defined one from the other.

In the gangrenous stage the number of motions may amount to 40 in the 24 hours, but as the destructive lesions advance, the destruction of the muscular coat and general oedema of the bowel, brings about a gradual loss of expulsive power of the bowel resulting in the diminution of the number of stools passed.

All the remarks made upon the 'constitutional effects' in acute non-amoebic dysentery are also applicable to the amoebic form. Fever is rare, unless the liver is implicated. Sweating not only accompanies collapse, but also abscess of the liver, then it is of a paroxysmal nature, and useful as a diagnostic sign.

Anaemia is often present, especially in the

chronic amoebic form. Some of the amoebae are found crammed with red blood corpuscles, and these amoebae are much the most active. So Lafleur believes that they are really responsible for the anaemia. (6).

The pulse and respiration follow variations in temperature; as a general rule, the respiration increasing with the pulse, but in 'collapse' while the pulse runs away up to 140 and is thready and compressible, the respiration is infrequent and shallow, while in cases of complication with abscess of liver and right lung, the respiration is accelerated out of all proportion to the pulse rate.

In chronic cases the skin becomes dry and scurfy; often takes on a sallow or earthy hue, especially visible in the face. Lafleur declares that in many cases the skin of the face resembles 'chamois leather.'

Death may take place from asthenia, general peritonitis (following perforation), haemorrhage (from ulceration of intestinal bloodvessels), or from liver abscess.

The presence of the amoeba dysenteriae constantly in the stools is the distinctive feature of this disease.

In the bloody mucoid stools, the amoebae are present

in large numbers and are evenly distributed; later on in the course of the disease, they become even more numerous and are congregated in groups especially in the little gelatinous necrotic masses, to a less degree in the mucoid, and in quite small numbers in the fluid portions of the stools. In chronic amoebic dysentery they are not so numerous but are more evenly distributed; when the motion is soft and partly formed, most of the amoebae will then be found in the flakes of adherent mucus. Probably their distribution is affected chiefly by the chemical reaction of the media; they flourish best and are most motile in alkaline mediae, while they become motionless and die off rapidly when the reaction is acid.

Complications and special forms of Dysentery.

I. Abscess of Liver. Not a very common complication of dysentery in Persia. In a year's general practice in Kerman (near Persian Beloochistan) I only saw one case; during another year's work in Yezd, a city 250 miles S.W. of Kerman, I was called in to see another just before his death. During a third year spent in Ispahan, I admitted one case where the abscess had burst into the right lung.

Some weeks after operation he returned to his village refusing further treatment, but he had greatly improved in strength, but went out with the drainage tube still in the wound. We then lost sight of him entirely, and knowing what the native surgery is like, it is to be feared that he did not survive long. Before returning to England I spent another two months in Ispahan helping in the hospital work there, and in a period of six months they had tapped five cases of liver abscess using Cantlie's modification of Manson's trocar and cannula. I had the pleasure of doing one myself, and the new method is certainly a vast improvement over the old one. Of the five cases treated, all upon admission were in a desperate condition; two made excellent recoveries, while three died. The three who died seemed greatly relieved by the operation, but after two or three days' draining, the liver pus became replaced by almost pure bile, and they sank rapidly. We commenced to look upon the appearance of bile after the operation as a distinctly unfavourable sign.

The liver and bowel may be simultaneously affected, or signs of liver trouble may precede the dysentery. In the latter case, it would be doubtful

whether the hepatic abscess was of dysenteric origin, belonging probably to the supra-hepatic form of liver abscess. Sometimes during the course of the dysentery, the patient complains of pain in the right hypochondrium, which is increased on pressure; coincidentally there is a rather sudden diminution in the number of 'stools' passed in the 24 hours. There may be some slight rise of temperature, and patient feels 'out of sorts.' Upon examining the liver, the organ is found to be enlarged. In a few days other symptoms make their appearance--all characteristic of liver congestion, e.g., nausea, vomiting, jaundice. The vomiting may be caused by pressure exercised by the enlarged liver upon the diaphragm. In this form of hepatic abscess, which is the intra-hepatic variety, we are justified in attributing the cause to the action of organisms found in bowel infection (especially dysenteric) which are carried to the liver through the Portal vein. "It is quite certain that liver abscess can arise independently of dysentery (40)." In 855 cases of dysentery recorded by Buchanan--occurring in India--there was not a single liver abscess. (41).

The method or nature of infection has not yet

been satisfactorily demonstrated. As Cantlie says, "when there is suppuration in the neighbourhood of the Portal vein, it is not surprising that multiple abscesses in the liver should follow, but why there should be only a single localised abscess as a result of dysenteric infection is much more difficult to explain." The infection is probably caused by emboli obtained from the ulcerative gut, the infective material being carried by the Portal vein. But why should not an hepatic abscess follow the ulcerative lesions in the gut caused by Typhoid?

Dr. Neil Macleod suggests an explanation which will not explain away all the difficulties, but is worthy of consideration. He points out that in dysentery, the pus burrows chiefly beneath the mucous membrane and would thus be in closer contact with the radicles of the intestinal veins, than the more open and superficial ulcerations of Typhoid. But in native races dysentery is very common and liver abscess apparently very scarce. The question of why in some cases you get single, and in others, multiple abscesses, may simply be a matter of chance.

Cantlie concludes that "it is possible that the dysenteric ulcer is associated with a specific toxic

agency--bacterial, chemical, or embolic--which in the liver tends to establish a focus or 'foci' of suppuration." (40).

This naturally leads us to the consideration of the amoeba dysenteriae as being the chief etiological factor in hepatic abscess due to dysenteric lesions. There is a tendency to blame the amoebae for everything; we may yet hope that they may be able to prove their innocence.

Amoebae have been found in liver pus, but they never appear in the discharge till the second or third day. When found they are often seen to be actively motile.

Manson believes that the reason for their delayed appearance is that they cannot live in the dead central pus, but only in recent pus or in the membrane lining the abscess wall. Thus all this central pus must first be drained off, before they can make their appearance.

Cantlie thinks that they play no part in the changes leading up to the abscess, and only when the abscess is tapped and has been drained do the amoebae travel towards the diseased region. He says "I have never found the amoeba during the early days of the

discharge, and I have not always found it in the latter." (40).

As pointed out before, some authorities believe that the amoebae are carriers, 'not the actual criminals, but accessories to the fact;' that they do transport duty, carrying bacteria to the scene of action. The objection to this theory is that it implies the presence of the amoebae in liver abscesses from the very beginning, which has not been proved.

Liver pus is curious, not at all like ordinary pus, and 'sterile' when first drawn off. Streptococci and Staphylococci, like the amoebae are not present till the second or third day. This apparent sterility is difficult to explain; with our present pathological and bacteriological knowledge we cannot conceive of suppuration taking place without the presence of bacteria. Cantlie suggests "that owing to the cutting off of the blood supply to a localised area of liver, due to congestion or hyperaemia of the liver itself, this may give rise to a necrotic patch, and that bacterial infection may follow upon, instead of precede, the pathological change." (40).

Lafleur reports that in 19 per cent of amoebic dysentery cases, liver abscess has been found. (6).

II. Continuous Fever. A somewhat rare complication of dysentery. I can only remember seeing one case, and in that the fever appeared first and continued for some weeks before the dysentery started. Johnson records a personal experience, where after being exposed to the heat of the sun in Bengal, while shooting in marshy ground, the same night he had chills alternating with flushes. The next evening there was high fever, delirium and severe dysentery; he ultimately recovered under treatment with scruple doses of calomel (42). This delirium is symptomatic of the fever, not of the dysentery.

III. Malaria. As I have said when discussing the etiology, dysentery and malaria are two distinct diseases. The one may however precede, complicate or follow the other; thus it is not correct to talk of 'malarial dysentery.' Yet repeated attacks of malaria certainly predispose to dysentery, but probably not more so than any other debilitating disease. In many cases of dysentery, where the temperature has gone up, and it has been thought to be complicated with malaria, the increased temperature has really

been due to septic absorption, and not to the malarial parasite.

When the two diseases occur together in the same individual, they may run their course independently of each other, or the dysenteric symptoms may become exaggerated or modified! In a few cases the dysentery is really one of the malarial symptoms. To this category belong cases described by various authors as 'intermittent dysentery' occurring every other day (43); also the so-called 'haemorrhagic dysentery' when large quantities of blood are evacuated daily or every other day(32).

In cases of dysentery complicated with malaria the temperature often runs away up beyond 104° F. If the malaria be severe, the case terminates in an exaggerated form of collapse, resembling choleraic algidity.

The fever-stricken troops in Mauritius in 1866-67 were attacked with dysentery; "the stools were fluid, thin and of a dark smoky colour, consisting of disintegrated blood and water. The patients were very depressed, their bodies became cold, and they died in a condition of exaggerated collapse. Post-mortem examinations shewed in some cases total

sloughing of the large intestine; in others, the only morbid appearance was a prominent state of the glands." (1).

IV. General peritonitis. Ensues after perforation has taken place. Fortunately this accident is rare, but in 108 autopsies recorded by Woodward (3), he found that perforation had taken place in eleven. When an ulcer does perforate through the serous coat, it is generally either in the Rectum or Caecum; the resulting inflammation in the former is called periproctitis, in the latter perityphlitis. The prognosis in these cases is very grave.

V. Scorbutus. The combination of scurvy and dysentery forms one of the most fatal maladies in besieged cities. In scorbutic people dysentery begins very insidiously as ordinary diarrhoea, and the stools are more copious and less frequent than in ordinary uncomplicated dysentery. Colic and straining are slight. The stools consist of faeces, mucus, and a large amount of bloodstained fluid, and may contain sloughs. In this special form of dysentery the prognosis is decidedly grave, as all the cases shew a great tendency to become gangrenous. The ordinary scorbutic symptoms are of course also present,

e.g., spongy gums, leaden colour of skin, haemorrhagic extravasations into skin or connective tissue. (3).

VI. Rheumatism. During the course of some cases of dysentery, the large joints begin to swell one after another until all may be effected; the pain is slight. Generally the swellings gradually subside, and very rarely terminate in suppuration, and, as a rule, there is no rise of temperature. But Trousseau reports a case in which the joint affection was so severe that the synovial effusion caused rupture of the capsule. (44).

VII. Toxaemia. The septic products from the dysenteric lesions are occasionally absorbed into the system, especially in the gangrenous forms of the disease, and gives rise to a state of nervous prostration, accompanied by sleepiness or torpor and a low muttering kind of delirium; the tongue is black, the pulse is feeble and fast, and the patient dies of septic intoxication. Pyaemic symptoms are less common.

VIII. Typhus and Typhoid. Are often complications of the dysenteries of war and famine, and add greatly to the gravity of the prognosis.

IX. Paralysis. Woodward reports eight cases as a sequel to dysentery, a form of paraplegia, due probably to a neuritis. (3).

X. Intestinal stricture. This complication, at least in chronic dysentery, one might expect to find fairly often, but it is very rare; during the late South African war not a single case has been recorded.

XI. Haemorrhages. Large haemorrhages from the bowel may occur in some dysenteric cases, due to the erosion of intestinal bloodvessels. Copious discharges of clotted blood take place 'per rectum.'

It is perhaps unnecessary to give a further detailed list of possible complications. Various diseases of the lungs, heart-failure, dropsy, Bright's disease, may all appear on the scene and hasten the end. It is as well to mention that in the dysenteries of children, 'invagination of the bowel' may occur.

DIFFERENTIAL DIAGNOSIS. The diagnoses of dysentery is not difficult if one remembers the following three points:-

1. Make a careful study of the symptoms.
2. Make a personal examination of the stools, at least once a day.

3. Examine patient's abdomen, (inspection and gentle palpation.

No. 3 will enable you to form a shrewd idea of the main site of the lesions.

By being careless about the second point many a mistake has been made. Dr. Davidson illustrates this by the following case--"a lady who had exhibited some symptoms of hysteria previously, was taken ill. Her motions were scanty and serous; not very frequent, but accompanied by some straining. She did not complain much of the intestinal symptoms, but greatly of dysuria. She was accordingly treated for hysteria by cold baths, and the true nature of the case was not recognised till the protrusion of a tubular piece of mucous membrane from the rectum rendered the diagnosis of dysentery unavoidable." (23).

To distinguish between non-amoebic and amoebic dysentery, the stools must be searched for the amoebae.

The diagnosis between Catarrhal, Acute and Chronic dysenteries may be made by the careful consideration of the symptomatology.

'Colitis.' Pain and straining not marked; calls to stool are less frequent. 'The serious stools like meat-washings' (vide Symptomatology) are absent.

Rectitis. This disease is characterised by

alternation of healthy with dysenteric stools. Thus once or twice daily the patient passes healthy stools, but along with, or independently of these, he passes blood, mucus, and pus constantly, accompanied by more or less straining. The general health of the patient remains good. Examination of the rectum with a speculum will decide the diagnosis. Rectitis yields readily to treatment. (23).

'Bilharzia.' When infecting the lower bowel may give rise to symptoms simulating sub-acute and chronic dysentery. If a portion of the 'stool' be examined under the microscope, the characteristic eggs will be at once detected.

PROGNOSIS.

Mortality. In Persia the mortality amongst dysenteric cases treated in hospital is not more than 2 per cent, but the gangrenous form of the disease is rare and the acute attacks yield readily as a rule to treatment. In all tropical countries the mortality from dysentery has markedly decreased during the last half century. This improvement is not only due to better methods of treatment, but also to improved hygiene.

In the Indian army dysenteric death-rate from 1847-57 was 11 per cent; during the five years 1871-75 only 3.3 per cent. In Martinique, in the two French military hospitals from 1852-72, mortality from dysentery was only 2.8 per cent. This return is specially interesting as it represents the result of treating the disease without giving Ipecacuanha. (23).

As I have said before, the returns from civil hospitals are more trustworthy, because they exclude mild catarrhal dysenteries which are included in all the army returns. The mortality therefore in civil hospitals is, as a rule, heavier. Thus in the civil hospital in Calcutta during the year 1879, the ratio

of deaths from dysentery to admissions was 22.2, in Mauritius during the year 1888, it was 22.7 per cent. (1).

The prognosis becomes graver and more guarded as the disease progresses from one stage to another. Thus in Catarrhal or Climatic dysentery, the prognosis is decidedly good unless the symptoms continue after the first year of residence.

In the acute non-amoebic form, when the lesions are in the lower part of the colon, the prognosis is more favourable than when they are situated in the caecum or ascending colon.

In the first or catarrhal stage, the first sign of improvement is the reappearance of faeces in the stools, the number of motions decrease, the colicky pain and straining disappear, and no more blood is passed. The bowel has suffered but little damage, and there should be no risk of recurrence.

It is therefore of the utmost importance to do our best to arrest the disease while it is in this stage, but unfortunately in civil practice such patients do not consider themselves ill enough to come to you for treatment, and instead try to 'doctor' themselves.

In the second stage the prognosis must be more guarded, though still hopeful. If treatment proves successful, convalescence is apt to be prolonged, and the slightest indiscretion in diet will bring on a relapse. In more advanced cases still, according to Fayrer (36) a hopeful sign is the appearance of 'branny mucus' in the stools, and if this be followed by 'ropy mucus', one is justified in predicting an almost certain cure. In the third stage or gangrenous dysentery, the prognosis is bad; in grave cases, almost hopeless, although one should never give up hope in any case of dysentery. Recovery has been known to take place even after the patient had passed a tubular slough of twelve inches, followed by one three inches long. (36). In connection with these bad gangrenous cases Chevers says, that when a black or coffee coloured slough 'like an old sooty cobweb rolled together' is present in the 'stool', it is the sign of inevitable death.

Hiccough, if present in the early stage of the disease, is not an unfavourable sign, but when it is coincident with collapse, the case is hopeless.

Of course, the presence of complications makes

the prognosis graver, general peritonitis following perforation of the bowel, is generally fatal. Abscess of liver is a dangerous complication; out of five cases operated upon in C.M.S.Hospital, Ispahan three died and two recovered.

The prognosis in amoebic dysentery, as in all caecal forms of the disease is more guarded.

Before giving a prognosis in chronic dysentery many factors must be taken into account, e.g., presence or absence of constrictions, or adhesions interfering with the function of the bowel, severity of the symptoms, resisting power and general health of the patient, etc. Also the circumstances of the patient; is he able to take complete rest, to go to some other country? Then there is a personal element to be considered; is he a man who will carefully follow the directions given him as regards diet, mode of life, or is he careless and indifferent about himself.

Thus in chronic dysentery, one would generally prefer deferring one's prognosis for a year, and watch the progress of the case.

PROPHYLAXIS.

The prophylaxis of dysentery may easily be deduced from its etiology, but one or two points deserve special emphasis.

I would suggest that medical men at home, who are often called upon to pronounce an opinion upon the advisability of people going to tropical climates to reside, should refuse to pass all those, who while living at home in temperate climates, exhibit a tendency to diarrhoea.

Then a very important prophylactic measure--too often neglected--is the wearing of a 'cummerbund' or flannel binder round the abdomen. It would be difficult to under-estimate the importance of this simple measure, more especially in guarding against Climatic dysentery. In this disease, the cummerbund deserves a place under 'treatment,' because it, in itself, has often proved sufficient in Persia to check a slight attack.

Then all chills should be avoided; the drinking water should be always boiled; alcohol should be avoided; no one should ever sleep in wet clothes. The soil round the dwelling-house should be carefully protected from all risk of contamination of faecal

matter. Dysenteric stools should always be carefully disinfected; then, if possible, cremated, or taken some distance away and buried in deep earth.

All these simple precautionary measures are matters of 'common sense,' although the sense of their great importance is unfortunately far from 'common.' I have already referred to prophylactic measures against 'war dysentery,' and I would again draw attention to the importance of enforcing their observance.

TREATMENT of DYSENTERY.

General remarks. How often one hears it said, and sees it too in print, that the treatment of dysentery is purely 'empirical,' that is to say in certain forms of the disease, certain drugs have a curative effect, but in what way the drugs bring about the successful result is unknown. This may be true as far as the giving of Ipecacuanha is concerned, but it is certainly not true as regards the major part of the treatment, and the sound lines upon which it is conducted. One might as well call the ordinary treatment of an inflamed joint, or the putting on of a splint, empirical treatment!

The pathology and symptomatology both combine to indicate the correct method of treatment. Thus, there is known to be internally, an inflamed mucous membrane, which from the presence in the gut of various forms of bacilli, will quickly become the seat of pyogenic organisms, causing ulceration, suppuration, necrosis and gangrene of the part. The patient will exhibit, in a varying degree according to the site and extent of the lesions, special symptoms which have already been fully discussed.

Now for an external inflamed wound two things

are imperatively called for before the wound can heal--rest for the inflamed part, and freedom from irritation; exactly the same are needed for dysenteric ulcerations to heal up, and if only they could be got promptly and completely, dysentery would cease to be such a serious disease. The great difficulty in the treatment of dysentery is to fulfil these two indications. How can rest of the bowel be complete when peristalsis is constantly taking place, and irritating faecal matter constantly befouling the surface of the ulcers! However, rest, complete rest in bed, must be insisted upon in all acute forms of the disease, and the patient must use a bedpan for defaecation, which should be always warmed before giving to be used.

Then 'starvation' alone would completely fulfil the other indication for treatment, but as of course this cannot be done, one must choose a diet in all acute cases, which shall be bland, non-irritating, and which shall ultimately yield very little faecal matter, e.g., milk.

Then external wounds need cleansing, and so do the dysenteric wounds. When the lesions are in the Rectum and Sigmoid flexure, this is fairly easy to

do, by methods that we shall be considering later, but when the lesions are situated in the caecum and ascending colon, they are much harder to reach by these methods, and thus naturally are more troublesome to treat. This question of 'site' which I have emphasised so much in this Thesis, seems to me to be most important as regards prognosis in the treatment of dysentery, much more so than the question whether a case is amoebic or non-amoebic!

I would also like to urge the great importance of prompt and energetic measures during the early stages of the disease. Because some intestinal catarrhs get well without any special treatment, that is no reason for making light of them, for certainly many of them go on to a form of true dysentery if not properly treated.

It is a good rule never to despair of a dysenteric case, unless perforation has occurred; or the patient is lying collapsed, violently hiccoughing-- then one is justified in giving up hope.

The history of the various treatments adopted for dysentery during different centuries is most interesting. The treatment necessarily depended upon what the leading doctors considered the disease to

be from a pathological standpoint. Thus Sydenham (23) believing dysentery to be a fever 'turned inwardly to the bowels' adopted measures to bring the fever outside again, or in his own words, "the indications were plain, and I had nothing else to do but to cause a revulsion of the acrid humours by venesection, by which I tempered the remaining volume of the blood, and then to draw off the aforesaid humours by purging." He tempered his patients' blood to such good purpose, that they often had not enough left for the heart to fulfil its function, and thus died of syncope, while of course, the disease got all the blame not the doctor!!

Later on when the doctrine changed, and dysentery was looked upon as a simple inflammation, the same treatment was retained, but now it was used for the purpose of subduing inflammation!

The Persians regard dysentery as a 'cold disease.' They put their patients on starvation diet in the form of watermelons; then proceed to make them comatose with opium, afterwards bleeding them till they fall into a gentle sleep, from which they too oft awaken--if awake they do--in the Mohammedan's Paradise! Opium is the one drug that the Persian doctor

believes in. It is his panacea for every ill that mortal flesh is heir to! The opium habit which is so prevalent in some of the Persian cities is the natural result of this policy. In Persian dysentery I have very rarely had to use Opium at all; when I was forced to do so, Morphia injected hypodermically seemed to give the best results.

In more modern times (as we saw when discussing the mortality of dysentery), the treatment of the disease has greatly improved, as shewn by the results obtained; but this improvement is undoubtedly to a large extent due to vastly improved hygienic measures. Dysentery nowadays is also of a much milder type, and is seldom complicated with Malaria and Scurvy. Probably the greatly improved facilities for travelling has been of immense use in preventing malignant types of the disease. Our soldiers in India get more frequent furloughs, and thus are not exposed to the disease at the most favourable moment, that is, when they are 'below par' and their tissues devitalised, for there is, unfortunately, no acclimatization for dysentery!

We shall now proceed to discuss the treatment of Non-amoebic and Amoebic dysentery; afterwards

considering more briefly the main points in the treatment of complications and special forms of the disease.

"The special symptomatic indications for treatment in dysentery are--to allay the pain and straining; to soothe the irritation of the inflamed mucus membrane; to support the strength of the patient; to give nourishing diet, which at the same time, must be bland and afford but little faecal residue; and, finally to combat complications." (23).

The special difficulties in the way of treatment are--the large amount of bowel surface involved; the impossibility of getting proper physiological rest for the colon owing to peristalsis; and the presence of irritating particles of food.

I. Non-amoebic dysentery.

I. Catarrhal form. A. Climatic, (as found in Persia).

A. If slight, supervise the diet, forbid fruit, alcohol and tobacco. If not worn before, patient must at once proceed to wear a flannel abdominal binder. A good dose of salts or castor oil should be ordered to be taken first thing on waking in the morning;

then a simple mixture of Bismuth Salicyl. gr x-xx with ginger--a tablespoonful thrice daily--will often complete the cure. The following prescription I have often found useful:-

R Bism. Salicyl. \mathcal{J} ii
Tinct. Zingib. \mathcal{J} ii
Syrupi. \mathcal{J} i
Aq. destill. ad \mathcal{J} viii

Sig. \mathcal{J} ii
t.i.d.

B. If the attack be more acute, the patient must at once be sent to bed and kept there. If, as unfortunately often is the case, he happens to be travelling at the time and has to go forward, there is nothing for it, but to put him under the influence of Opium--either hypodermically with **Morphia**, or per os with Laudanum; Pil Plumbi c Opio, or Pil Opii. This will enable him to reach some city safely, (the big cities in Persia are from 200 to 300 miles apart; ordinary rate of caravan travelling is 3 to 4 miles an hour!). But, as soon as possible, prompt and energetic measures should be taken--rest in bed, flannel binder round the abdomen; the colicky pain can generally be relieved by applying a hot water bottle to the abdomen, or hot fomentations covered

with guttapercha tissue.

There has lately appeared in the market, a patent small tin box containing a slowly burning cartridge of powdered charcoal, known by the name of 'Istra' which has been borrowed from the Japanese. A medical friend has told me how useful he has found it himself when suffering from lumbago, sewn into a flannel bandage and applied over the lumbar region. I see that Dr. Manson recommends it strongly for the relief of colic in cases of acute dysentery--three or four of these small tin boxes with lighted cartridges sewn into a flannel bandage and worn round the abdomen. (24) The patient must not be allowed to get up to pass his 'motions.' He must have milk diet, with arrowroot once or twice in the 24 hours. The milk should be given frequently but in small quantities at a time and should be just tepid, neither hot nor cold, otherwise 'peristalsis' will be excited. If the stools are very frequent and the straining troublesome, a thin starch enema (about a wineglassful) should be injected, once or twice a day. If there is no blood present in the stools then I give opium, either by the mouth, or combine it with the starch enema (mxx of Tr Opii). It is as well to give a dose of salts or

Istra

Castor oil to clear out the bowel before starting the above treatment. Then three times a day I give a tablespoonful of the Bismuth Salicylate mixture. Under the above treatment the dysentery usually disappears, and does not recur.

C. If however the disease becomes chronic with exacerbations and remissions, there is a fear lest it becomes true dysentery. It is then well to advise change of air and work, enjoining the patient to lead a regular life and to be careful of his diet. I have known horse exercise to prove beneficial in some cases, although probably it was the 'desert air' that helped more than the riding. It is no good giving a great number of medicines. A simple Chalk or Bismuth mixture, Opium in some form or other, and in obstinate cases small doses of Quinine gr. 2-4, twice or thrice daily, are well worth trying.

Often the patient may suffer more or less during the whole of his first year in Persia, the disease then disappearing, never to return; in other cases it still continues during the second year, and then the best treatment is to send him back to a temperate climate, if possible by sea; the symptoms will probably have all disappeared before he lands in England.

II. Abortive dysentery. Many a threatening attack may be cut short by taking a good dose of Castor oil with 10 to 20 drops of laudanum. In the special form I refer to--often the sequel in Persia to any great mental strain or anxiety--the treatment is to try and remove the cause, (unfortunately not always possible); give a smart purge of calomel 4 to 6 grs. or Magnesium Sulphate $\mathfrak{J}\mathfrak{i}$ and make the patient wear the flannel binder.

If combined with congestion of liver, patient must rest in bed, drink milk diluted with limewater, and take some simple stomach sedative, as Bismuth Subnit. gr. x \bar{c} Acid Hydrocy. dil mv. t.i.d.

I have discussed the treatment of these minor forms purposely rather fully, because they are worth taking care of, for although some cases would probably get well without any treatment, others, if neglected, become chronic and may terminate in true dysentery.

II. Acute dysentery.

During three years in Persia, I adopted the following routine treatment in the cases of acute dysentery admitted to hospital, with very satisfactory

results. Out of over 100 cases only one died, and he was moribund on admission. All went out of hospital cured, and I can remember only one case relapsing, and he was the one who returned in a moribund condition and died. He went out too soon the first time, refusing to stay in any longer, saying he was cured (after 3 days' treatment), his excuse being that he had to go and sow his corn.

Upon admission the patient is at once put to bed and told to rest. His tongue is next examined, if it is seen to be furred, milk is not given but thin chicken broth is ordered instead; if the tongue is clean, he is allowed milk--boiled, diluted with lime-water and given tepid, in small quantities every two hours. The symptoms are then carefully studied, the 'stools' examined, and the abdomen inspected and gently palpated.

"No opium is given either per os or per rectum, as long as any blood is passed with the stools." I know that this is contrary to the advice of many authorities, but I can only say that in acute dysentery, as seen by me in Persia, the cases do better without opium as long as blood appears in the stools. The presence of blood with the mucus (I am excluding big

haemorrhages from erosion of bloodvessels or other causes) indicates the presence in the inflamed bowel of some irritating particles--faecal or otherwise. If opium be given, the result is that the irritant is retained instead of being expelled, and although the pain be masked by the drug, yet the dysenteric ulcer is still being irritated, and is tending to spread rather than to heal. At least this is my hypothesis, so I omit the use of opium until blood no longer appears in the stools. The great plea for the use of opium is to arrest peristalsis, but as Dr. Davidson points out (1) "attempting to arrest peristalsis by opiates is not without danger," and at least in the milder forms of the disease, sedative enemias, answer nearly as well.

Then orders are given that the dysenteric patient shall have, first thing in the morning upon awaking, half an ounce of the following mixture:-

R Mag. Sulph. ℥ii
Mag. Carb. ℥i
Aq. Menth Pip ad ℥℥

to be repeated during the first day every 4 hours until a good big motion is passed; then half an ounce of the same mixture is given once a day in the early morning (thus on an empty stomach) for the first

week.

The colic is treated by placing a hot water bottle wrapped in flannel upon the patient's abdomen, or by hot fomentations kept hot by covering with guttapercha--to be renewed before they get cold, not after. Turpentine stupes over the abdomen have often proved useful.

The patient is ordered to have three rectal injections in the 24 hours; in the morning an astringent (usually Alum gr. x to the oz.), in the afternoon a thin starch enema (about a wineglassful), in the evening another Alum injection. If the rectum is very irritable, and great straining follows any attempt to introduce the pipe, it is better first to give a Cocaine suppository, or inject a small quantity of a 4% solution.

These injections are given regularly three times a day for the first week, and in nearly all the cases a very marked improvement took place from the first day of the treatment. By the end of the first week, the blood and mucus have disappeared; the stools have been reduced in frequency from 40 to 60 evacuations in the 24 hours to 5 or 6, and they are now faeculent and more like ordinary diarrhoea; colic

and straining have also disappeared. The morning dose of salts is now omitted, also one of the alum injections while twenty minims of laudanum may be injected with the starch. The motions now become formed. After two or three days the injections are omitted, and the patient is placed on a less restricted diet, being discharged cured a fortnight after admission

During the treatment the patient is given some simple chalk or bismuth mixture, a tablespoonful three times a day.

During convalescence if the stools become too frequent, bismuth combined with Dover's powder, will check the tendency to diarrhoea.

As I have said before, the Persian dysentery is probably hardly ever of a malignant type, although I have seen in neglected cases treated by Persian doctors, gangrenous stools, and the patient in a collapsed condition. I fixed upon the above treatment three years ago, before hearing of the saline treatment, after trying other methods, especially Ipecacuanha, because it gave out and away the best results. Ipecacuanha, from my experience of it in Persian dysentery, does not give satisfactory results

although when we come to discuss chronic dysentery, I shall have rather a better word to say on its behalf.

Almost certainly in different tropical countries you find different forms of dysentery; possibly each form has a special specific microbe of its own, though this is unlikely, but certainly each form may require a different kind of treatment, and the one drug that seems specific for the disease of one country, may prove absolutely useless in another.

Ipecacuanha. This drug was till quite recently looked upon as a specific in dysentery, but lately it has been proved to be of little use in some forms of the disease. Thus Clouston in the outbreak of dysentery in Cumberland asylum, tried giving the drug in every form--"by the mouth and rectum, in big and small doses, with and without opium" without success, (28). Davidson found it of no use in the malarious dysentery that attacked the troops in Mauritius (2). The form of the disease that attacked the inmates of the Millbank Penitentiary in 1823-24 resisted every kind of treatment, including Ipecacuanha, mercury alone giving good results. Mayne (46) in the famine dysentery of 1848-49,

occurring in the South Dublin Union workhouse, found Ipecacuanha useless. As I have already said it does not seem to give good results in Persian dysentery, and a Russian doctor practising in South Russia, assured me that he had long ago given up the use of it in the dysenteries he had occasion to treat, because it gave nothing like as good a result as minute doses of Calomel combined with irrigation of the colon with Creolin. The Ipecacuanha treatment was at one time looked upon as a specific for Indian dysentery, but now it is largely being replaced by the 'saline' method. Still it retains a place as one of the most valuable drugs in some forms of the disease, and personally, the one case in which I found it act as a specific, was in a bad case of chronic dysentery imported from Bombay, which resisted all other methods.

Ipecacuanha was described first by Piso in 1649 who pointed out its valuable anti-dysenteric characters. Nearly 40 years later in 1686, Jean Adrian Helvetius, who had studied medicine at Leyden, but who had repaired to Paris to assist his father, got possession of the drug, which had been imported from Brazil by a physician named Legros. Helvetius gained

a great reputation by curing cases of dysentery with large doses of the drug, amongst his successful cases being the son of Louis XIV. He finally revealed the name of the drug to the French government, receiving from them in return a thousand Louis d'or. After this the drug was largely used, but chiefly as an adjuvant, and in small doses; when pressed in large doses, it was generally with the idea of producing emesis. (23).

Balmain (47) at the beginning of the 18th century first re-discovered the method of giving the drug in large doses. He gave two drachms of the drug combined with laudanum, and found "a dose or two enough to remove every dangerous symptom."

Docker (48) in 1858, formulated a scheme of giving the drug as the main part of the treatment in dysentery, which has become the classical method, being still the routine way of giving Ipecacuanha in dysentery.

His method is as follows:- Attack the disease as early as possible; give a preliminary purge if constipation be present. Put the patient in bed and keep him there. Then place a mustard plaster over the pit of his stomach, followed by the administration

of 20 to 60 grs. of Ipecacuanha, as a bolus or in pill form. The patient should have no food for 3 or 4 hours previous to the giving of the drug, and for the same length of time afterwards. He must lie absolutely quiet, and should not be allowed to swallow his saliva, which should be wiped from his lips whenever necessary. If the first dose is not retained, a second dose should be administered first giving *m* 20 to 30 of laudanum, to act as a sedative. In mild cases the dose should be administered morning and evening, in severe cases every eight hours, and the treatment should be persisted in till the motions are faeculent and regular, all colicky pains and straining have disappeared. Manson (24) suggests that each day the dose of Ipecacuanha be reduced by five grains.

I followed this treatment carefully in Kerman, Persia, two years ago. In hospital was a Parsee who had come from Bombay. While there he had contracted dysentery, which had never yielded properly to treatment. Upon admission the dysentery had lasted two years with short intermissions, and he was in a wretched state of health. During the exacerbations he suffered considerably from pain and

straining, and the frequency of the stools would increase to 30 in the 24 hours. He was tried with the routine treatment, already described, with no success. After six weeks he was given Ipecacuanha following Docker's method, and the result was simply marvellous. Although having 60 grs. twice daily, he rarely complained even of nausea, and all the symptoms rapidly disappeared; the stools became faeculent and regular, and he was discharged cured in a fortnight. He was able to resume his merchant's work which he had been obliged to relinquish from the state of his health, and a year afterwards I heard that there had been no recurrence of the dysentery. I would point out that this was a case of chronic not acute dysentery, and was imported from India.

For the dysentery of children if Ipecacuanha be given at all, it is best given combined with Sodium Bicarbonate, or as the Vinum combined with Syrup.

The action of Ipecacuanha in dysentery is certainly 'empirical.' Therapeutically it acts as a nauseant, diaphoretic, laxative, hepatic, pancreatic and intestinal stimulant, but to which of its actions we owe our gratitude in some forms of dysentery

remains unknown. It may act by increasing the exudation into the large gut and so helping to clear it of infective material, thus resembling the action of the salines. Curiously enough, large doses of Ipecacuanha have an irritant effect upon the healthy large intestine causing diarrhoea and melaena. Can its action in dysentery resemble that of Jequirity seeds in Pannus? Against this hypothesis is the fact that even when the drug is not curative, it never seems to cause any exacerbation of the disease.

The Ipecacuanha treatment is contra-indicated in cases of heart disease. I have seen a case where the patient with a mitral lesion has suffered from severe cardiac exhaustion caused by vomiting induced by giving Ipecacuanha.

For the same reason it should be left off in ^a gangrenous cases where the patient has collapsed, and should be at once discarded in any case of dysentery that commences to exhibit symptoms of hepatic congestion.

Finally, treating dysentery with small doses of Ipecacuanha is useless, and often produces a great deal more vomiting than the exhibition of a large dose.

De-emetised ipecacuanha has been largely

tried, but seems of doubtful value. I have had occasion to take it myself, and in my case, it proved a better emetic than the ordinary form of the drug!

Salines. Treatment of acute dysentery by means of saline purges is a comparatively new method, although Dr. Davidson (23) ten years ago drew attention to its great usefulness, and advised its trial in mild cases of dysentery, and in cases where ipecacuanha is not tolerated. Writing now in 1903 one might truthfully say that the saline method in acute dysentery has ousted Ipecacuanha from its first place. Sodium or Magnesium Sulphates are used, the sodium salt being often preferred because it is less irritating, but Magnesium Sulphate gives just as good results. Buchanan (49) has tried this treatment extensively with excellent results. Possibly in considering his statistics, one should remember that some of the cases are probably mere intestinal catarrhs and not true dysenteries, but the brilliant result gained shows that under the saline treatment, promptly given and efficiently carried out, the mortality of the disease can be kept as low as 1 per cent. Buchanan gives two separate reports; the first deals with 555 cases treated, with only six

deaths; the second after having during another year treated 300 more cases with three deaths, giving a total of 855 cases with nine deaths. Average stay in hospital was only eleven days; perhaps, this somewhat brief period accounts for the fact that out of 453 cases, there were 69 relapses (1 in 6.5), and the year after out of 300 cases, 51 relapses, although 37 out of the 51 only relapsed once. Out of the three deaths (in 300 cases) two were very severe gangrenous cases, with frequent 'meat-washing' stools; while the third after the failure of the saline treatment made a wonderful rally after 30 grs. of Ipecacuanha had been given, but died six weeks later with symptoms of chronic diarrhoea. Post-mortem examination shewed ulceration from caecum to rectum. The patient was a feeble, old, toothless man, aged 55.

Buchanan gave the Sodium Sulphate in drachm doses with Aq. foeniculi ad ʒi , 4, 6, 8 times daily continuing till all the blood and mucus had disappeared from the 'stools,' which result was usually gained after two or three days. He apparently would agree with me that Opium is very rarely needed in these acute cases; the salines themselves seem to have a

restraining influence upon the tenesmus. It is interesting to note that he did not meet with a single instance of Tropical abscess amongst the 855 cases. He advocates this treatment in acute dysentery only, and from other sources corroborative evidence is given that 'salines' are useless in chronic dysentery. Buchanan says that this treatment is being largely tried in cases of acute dysentery all over India, and that out of over 60,000 cases treated during the last few years in the Indian prisons, the mortality has been reduced to 7 per cent.

Reports from South Africa during the late war, from hospitals where the 'saline' method has been tried, show that in acute dysentery it gives a much better result than Ipecacuanha.

One of the civil surgeons reports (50):- "cases of true dysentery improve rapidly under saline treatment." But he noticed that the longer the duration of the case before treatment could be given, the less readily it yielded. (Thus corroborating Buchanan's belief that in chronic dysentery 'salines' are of little use). The same writer illustrates this by saying that at Welverdiend, all the acute cases recovered with salines, but out of five chronic cases

treated in the same way, only one recovered, and he had a combination of 'salines' and quinine. Magnesium Sulphate \mathfrak{z} i. with ac. sulph. dil. mxv and Aq. Ment. pip \mathfrak{z} i. was the prescription given, as he found concentrated doses more effective than dilute. This medicine was given every hour till the stools became faeculent, and then in less frequent doses, but continued for at least 48 hours after all the dysenteric symptoms had gone.

Another observer during the war found great benefit in the Magnesium Sulphate treatment in acute dysentery combined with antiseptic irrigation of the bowel (he used warm Boracic lotion), but he makes the interesting observation that the 'saline' treatment was not successful amongst the Indians who were in South Africa, when they contracted dysentery; much better results were obtained by the Ipecacuanha treatment (51)! This seems curious after Buchanan's success with the saline treatment in India.

Dr. Rouget of Mauritius (52) gives an interesting table, contrasting the results obtained from Ipecacuanha compared to 'salines' in that island.

See next page.

Date.	No. of Cases.	Died.	Recov-ered.	Not im-proved.	Treat-ment.
From Jan. 1st. 1898) to May 15th. 1898)	Severe 6 41 Mild 35	3 0	2 28	1 7	Ipecac. followed by Bism. Salicyl. Salol & Opium.
From May 15th. 1898) to Mar. 31st. 1899)	Severe 15 56 Mild 41	0 0	15 39	0 2 (im-proved)	Mag. Sulph. followed by Bism. Salicyl & Benzo-naphthol.

During convalescence he found the following prescription useful:-

dy Bism. Salicylate grs. 15
Benzo-naphthol grs. 10

combined occasionally with grs. 3 or 4 of Dover's powder.

Thus the verdict is unanimous from Mauritius, South Africa, India and Persia, that in cases of acute dysentery, the saline method of treatment is much to be preferred to the giving of Ipecacuanha, but it is only fair to add that two observers during the South African campaign (Washbourne and Richards) report that a combination of the two methods, that is

giving salines and Ipecacuanha together, gave good results (3).

How the salines act in dysentery is unknown, possibly by increasing the exudation into the large gut, and thus helping to clear it of infective material.

Mercury. The treatment of dysentery with this drug dates from the early part of the 18th century. Johnston (53) says of its action in dysentery, "that it does more to resolve irritative fever, to equalise the circulation, disgorge capillary vessels, restore the balance of the nervous power, and open the sluices of the various healthy secretions and excretions, than any other remedy with which I am acquainted", a somewhat long winded way of saying that it acts as an excellent hydragogue cathartic, and hepatic stimulant.

Morehead in 1860 recommended in dysentery that if the patient was too feeble for the orthodox treatment (bleeding followed by the application of a dozen leeches to the anus), local depletion should be got by the administration of calomel gr. x combined with Ipecacuanha and Opium; of each, 2 grs. to be taken at bedtime, and followed by Castor oil next morning. This treatment to be continued for two or three days

then changed for Ipecacuanha, 3 to 6 grs., combined with Blue pill 2 to 5 grs. every 2, 4, 6, 8 hours continued till improvement took place, but not long enough to cause mercurial salivation. If the patient was collapsed, he advised that the blue pill be omitted! (23).

Although, fortunately, for our patients, these somewhat heroic doses of mercury have been discontinued, small doses of the drug are still found valuable in the treatment of acute dysentery. I have myself given minute doses of calomel (gr. $\frac{1}{4}$ to $\frac{1}{2}$) every 2 to 4 hours in place of the salines with good results. Niemeyer speaking of a method where he gave calomel gr.i. combined with opium gr. $\frac{1}{4}$ every two hours, says that he found it "a most trustworthy treatment."

The only drug that seemed any good in the dysentery that attacked the Millbank Penitentiary was calomel, which was used with much fear and trembling after all other methods had failed.

Mayne records that mercury was 'the principal remedy' in the famine dysentery that attacked the inmates of the South Dublin workhouse. (46).
Of course, these outbreaks all occurred long before

the 'saline treatment' became known, but compared with ipecacuanha, mercury gave much better results at that period.

Manson (24) recommends calomel combined with Ipecacuanha and opium, of each a grain, to be taken every 5 or 6 hours in acute dysentery, the patient meantime to be carefully watched for salivation.

For children, in dysentery, Ringer recommends Bichloride of Mercury, one grain in half a pint of water, a teaspoonful to be given every hour until healthy stools are passed.

In the treatment of ordinary acute dysentery calomel cannot eclipse the position now held by 'salines,' but it has a very useful sphere of its own; and Davidson recommends its use in those cases of gangrenous dysentery that have resisted treatment with ipecacuanha. The time of giving must be carefully chosen. It ought not to be tried too hastily before the ordinary remedies have had a fair trial, nor as a 'dernier res^sort' after the gangrene has become extensive, but just as gangrene is starting or threatening to start. He suggests giving the drug in small doses (calomel gr. i, ipecac. gr. 2, opium gr. $\frac{1}{4}$) combined with ipecacuanha and opium, repeated

every hour for five or six hours; then every two hours during the day to be continued for two or three days. Used like this it will not produce salivation. If the discharge from the bowel becomes scanty during this treatment, he gives an enema of ipecacuanha (2 drachms in 10 ounces of water) once or twice a day. (23).

Opium. Has been largely used as an adjuvant to ipecacuanha. While describing the routine treatment of acute dysentery in Persia, I stated my views concerning the use of Opium. With the saline treatment it very rarely needs to be given, and then, in my opinion, it is better to wait until all blood has disappeared from the 'stools.' In Docker's method of giving ipecacuanha, it is often wise to first administer mxx or xxx of laudanum before administering the 'bolus.' When the lesions are chiefly in the rectum, the great pain and straining is best treated, not with opium but by the application of a few leeches round the anus, or hot hip baths, using great precaution against the patient's catching cold. If there is much dysuria or strangury opium is best given in the form of morphia administered hypodermically. As a rule opium is not

needed to quiet peristalsis, and the quieting of peristalsis with opiates is not entirely free from danger.

ANTISEPTICS and ASTRINGENTS.

Remembering the bacteriological factors in the etiology of dysentery, it is not surprising that various attempts have been made to treat the disease with antiseptics either administered 'per os' or injected 'per rectum.' Amongst the former Salol in 15 to 20 grain doses has proved useful. Naphthalin gr. 15 three or four times daily or 'per rectum' dissolved in olive oil has also been recommended.

Benzo-naphthol which passes unchanged through the stomach, breaks up in the intestines, becoming B. naphthol and benzoic acid, is specially useful when the kidney is diseased, 30 to 50 grains in divided doses being given in the 24 hours. It must be given in capsules or dissolved in oil and emulsified.

Resorcin may also be used in the same doses as the benzo-naphthol.

Antiseptic enemata are also largely given in dysentery. Boracic, weak Carbolic, Potassium Permanganate (gr. $\frac{1}{4}$ to the pint) have been especially recommended. I would add Creolin to the above list

a teaspoonful to the pint, for in my hands it has yielded satisfactory results.

There are many astringent injections used; it has been my custom to combine the 'saline treatment' with rectal injections of alum (gr. x to the oz.) and plain thin starch. Tannin, a teaspoonful to the pint, I have also found useful. Besides these Lead Acetate, Zinc and Copper Sulphates have all been used with varying success. Silver Nitrate we shall consider with the treatment of chronic dysentery.

Besides the above medicated enemata, it is well to remember that gentle irrigation of the large intestine with plain warm water often affords great relief to the patient. The water should be allowed gently to enter the bowel by gravitation, the ordinary enema syringe being discarded for a funnel, and the patient lying upon his stomach, and occasionally rolling from side to side to allow the water to, as far as possible, cleanse the whole extent of the large intestine.

The colon of an adult has a capacity for 3 to 4 pints, a large enema would be from $1\frac{1}{2}$ to $2\frac{1}{2}$ pints. In giving enematas, if there is much rectal irritation and pain upon the introduction of the pipe, it

is best to first give a cocaine suppository.

Large enemas are contra-indicated when the bowel is in the stage of bad ulceration, suppuration or gangrene. (2).

The drugs previously mentioned given 'per os' or 'per rectum' in the hope of combating the pathogenic organisms of the large intestine can only be looked upon as auxiliaries or adjuvants to the primary curative agents in dysentery.

Sulphur. During the South African war Dr. Richmond noticed that the veterinary surgeon used sulphur for horses ill with anthrax instead of the ordinary treatment with ipecacuenha. Upon enquiry he found that the reason was because it had been discovered to yield much better results than ipecacuanha. This incident led Dr. Richmond to wonder whether if sulphur was better than ipecacuanha in anthrax, perhaps it might also give superior results in dysentery. He tried it, and the results he records are certainly encouraging, although the sulphur seems to give better results in chronic diarrhoea following dysentery.

He gives 20 grains of sublimed sulphur combined with 5 grains of Dover's powder every four hours, the

patient having been put to rest, and given milk diet. After the first dose, he says "the tenesmus and colic are relieved; the blood and mucus in the stools disappear in two days, and the patient becomes rapidly convalescent." Meat must on no account be allowed till the diarrhoea has been stopped for a full week. During July and August of 1901 in Pretoria, 21 cases of dysentery were treated by this method. Out of the 21 cases, 11 were acute and 10 cases of chronic diarrhoea following acute dysentery. The acute cases were treated with sulphur combined with Dover's powder, the chronic with sulphur only; all the 21 patients were cured!

Dr. Richmond recommends the opium, as it enables the bowel to rest, controls the diarrhoea, and allows the antiseptic qualities of the sulphur to have fair play. He regards sulphur as an ideal intestinal antiseptic, and suggests that the great success of the saline treatment, is not so much that it acts as a purge, washing away cause of the disease, and allowing nature fair play; but that they possibly also inhibit the growth of the micro-organism owing to the presence of sulphur acids.

He also thinks that dysentery in South Africa

is due to a specific bacillus morphologically approaching the type of the B. Typhosus.

I tried this 'sulphur' treatment on hospital dysenteric cases in Persia, and was not satisfied with the result. Upon the whole the treatment did not seem to yield such good results as the routine method I was before using, but I gave some of the powders to a friend who was returning to England, and on the road being attacked with dysentery, he counts that the powders saved his life. He was ill from the time he left Resht (the Caspian Sea port of Persia) until he reached Vienna, and had to stay 24 hours two or three times 'en route.' The only treatment he could have were the powders, and these he took regularly every four hours; sometimes when he was very bad, every three hours. Despite all the adverse circumstances of the case, the inability of resting properly, the perpetual jolting of the train, travelling as he was night and day, yet ere entering Vienna he was cured. He was a doctor himself, and is now a firm believer in the curative power of the sulphur treatment in dysentery. I hope when I am abroad again to have further opportunities of studying this new method of treatment,

if not in Persia, in either Egypt or Palestine.

Monsonia ovata. A South Africa plant. Maberly has used it largely in the treatment of dysentery, and in his hands it seems to have given equally good results in chronic as well as acute forms of the disease. He uses a tincture-- $2\frac{1}{2}$ ounces of the dry plant to a pint of Rectified spirit. (24).

Gangrenous Dysentery. I have already referred to the treatment of this stage with calomel. When the stools have a gangrenous odour from the first, it is well to give ipecacuanha, as 'salines' do not appear to have the same curative effect in this form of acute dysentery. But the ipecacuanha should be at once omitted when the first symptoms of collapse appear. The great indication then is to support the patient's strength. The oil of turpentine often seems to do good in these grave cases, given in drachm doses with a couple of teaspoonfuls of Castor oil daily or every other day, or 20 to 30 m of the oil by itself every two or three hours (23). If there are any symptoms of kidney disease, the turpentine must not be given. When the presence of débris and black sloughs in the stools are noted, there is great risk of septic absorption from the

bowel, and antiseptic enemata should be cautiously administered. When the patient is in a collapsed or algid state, the only treatment is to try to infuse some warmth into the cold extremities by means of hot water bottles; stimulants must be given in small and repeated doses; ether to be injected subcutaneously.

III. Chronic dysentery.

Is a most troublesome disease to treat. 'Salines' seem useless. I have already mentioned a most successful result obtained by using Docker's method of giving ipecacuanha, and it appears of all drugs to give the best result. Manson recommends a preliminary trial of ipecacuanha in all chronic cases, starting with thirty grains and reducing five grains daily, combined with rest and milk diet. During the exacerbations treatment should be given as in acute dysentery--absolute rest in bed combined with ipecacuanha. Astringent injections are very useful especially Silver Nitrate. This is best given by the irrigation method, 20 to 30 grains to the pint, and if possible inject from 3 to 6 pints. It is best to begin with 20 to 30 grains to the half

pint and inject about two pints, the patient's position during the irrigation being as before recommended--lying on his face. These astringent injections should be omitted during very acute exacerbations.

Manson also recommends the systematic daily washing out of the colon with milk, which he says has proved a very valuable remedy. (24). During quiescence

besides the Silver Nitrate injections, Bael fruit has been found very useful, more especially in cases where the bowel is much thickened and ulcerated.

Fayrer suggests its being given combined with Dover's powder or opium alone. (36). The abdomen should

always be very carefully examined for signs of swelling or hardness denoting cicatrices or ulcers. If there is much congestion of the bowel wet compresses will be found useful. If the cicatrices interfere with the evacuations emollient enemas are indicated.

Constipation and diarrhoea should both be guarded against; for the former an occasional dose of Castor oil, glycerine suppositories, a course of Carlsbad waters, or enemas of warm water containing a drachm of salt to the pint; for the latter Bismuth Salicylate, combined with Dover's powder.

Large doses of Bismuth (half a drachm to a drachm

every two hours till 12 to 15 drachms in the twenty-four hours have been taken) have been recommended.

Simaruba (*Ailanthus glandulosa*) is strongly advocated for use in chronic and subacute cases of dysentery. It has been largely used in the East, especially in China, where it is known as 'Rhein's specific remedy for diarrhoea and dysentery' and was bought by the municipality of Shanghai for a large sum of money. The decoction is made thus:-

"Take of Simaruba bark ℥iii., Chinese cinnamon ℥i.; boil in three quarts of water and allow it to evaporate down to a pint. When cool strain into a brandy bottle, add ℥i. of good brandy and fill up by pouring cold water over the bark in the strainer till the bottle is full." (24). The dose is a wineglassful three times a day.

Davidson recommends it to be given during an exacerbation in chronic dysentery combined with *Ipecacuanha* in the following form:-

"infusion of 100 grains each of *Ipecacuanha* root and *Simaruba* bark in ten ounces of water. Two tablespoonfuls to be taken three times a day." (1).

Buchanan (49) points out in his treatment of chronic dysentery in India, the importance of

occasionally giving anthelmintics, as both round and thread worms are often present and help to keep up the irritation.

In cases where the bowel is stenosed, the only possible treatment is systematic gentle massage, warm baths, gentle laxatives to keep the faeces fluid and emollient enemas.

But in chronic dysentery especially, all this drug treatment is subsidiary to more general measures. In tropical climates as soon as a dysentery becomes chronic, the patient should, if possible, be sent to a temperate climate. In India a change to the hills seems to have but little effect. The patient should be made to wear flannel and always be warmly clothed--the abdominal bandage is specially important for him to wear--and he should avoid all draughts. The importance of a sea voyage can hardly be over-estimated; the sea air will often cure some of the apparently most hopeless cases. If the patient wishes to be cured, he must make up his mind to scrupulously obey every direction he receives and regulate his life accordingly. As regards diet, during the exacerbations, he must have only milk as in acute dysentery, but during quiescence he may be allowed a

more liberal diet, although milk should for some time remain his staple food, thus his diet may be supplemented cautiously with eggs, fish and fowl, but he must avoid alcohol.

Some obstinate cases of chronic dysentery seem to respond to a liberal meat diet given during quiescence. Graves strongly recommends this method. He says that "in some cases which had obstinately resisted the most varied remedies assiduously employed, got well after a liberal allowance of meat was given, and the first thing I should do when called upon to treat a case of dysentery of longstanding, would be, to put my patient on a full meat diet." (1).

With regard to 'rest' during exacerbations, the patient must go to bed and stay there, as in acute dysentery, but during quiescence, restricted exercise is beneficial. The patient should go for a carriage drive every day, and if strong enough a short daily walk would do him good.

During convalescence tonics are indicated, especially the perchloride of iron.

Great patience is required in the treatment of these chronic cases of dysentery, both on the part of the patient and the doctor, but they are by no

means hopeless, not even the most unfavourable cases where the bowel is stenosed. So that if only the patient will loyally obey his doctor, he may quite well hope to regain his health and strength again, (Deus volente or as the Persians say ان شاء الله *en shā-Allah*).

I would here again repeat, that all dysenteric 'stools' should be carefully disinfected and then buried deep in the earth, or if possible, cremated.

'Amoebic dysentery.'

The treatment of this form of dysentery seems up to the present to have been unsatisfactory. The general treatment is the same as in the non-amoebic form, the same stress is laid upon rest and milk or egg--albumen for the staple diet. During convalescence a return to ordinary diet must be made very gradually and cautiously. Lafleur (6) recommends turpentine stupes or morphia given hypodermically, for the relief of pain.

As regards curative drugs, Quinine given in five grain doses three times a day in six cases seemed to do no good. Salol, Bismuth and Calomel have all been tried without success.

Ipecacuanha has also been tried by Lafleur seemingly

without success.

Rectal injections or irrigations with antiseptic solutions give better, though not brilliant results, and warrant further trial.

The injections used have mainly been Quinine--1 in 5,000 to 1 in 500 three or four times daily, one or two pints.

Corrosive Sublimate 1 in 5,000, or 1 in 3,000 to be used more cautiously. (There is need for this caution, for I remember while dressing a case of 'fistula in ano' which had been operated upon in Persia, after syringing the wound with 1 in 2000 Corrosive Sublimate, very unpleasant results followed within a few minutes, the patient (one of the chief officers of the Shah's nephew) turning pale, feeling faint, and being seized with severe colic and violent diarrhoea, and this after I had taken every precaution and very little of the solution could have been absorbed. Needless to say more dilute solutions were used afterwards in cleansing the wound).

Silver Nitrate thirty grains in a quart of water has also proved useful.

Dilute Nitric acid has been recommended and ought to be of some use, considering that the amoebae are

known to be killed by an acid media.

The above methods of treatment are for ordinary acute cases; in gangrenous cases small sedative enemas are to be preferred.

Lafleur is not very optimistic over the treatment he recommends for he points out that "the injections may not touch the amoebae embedded in the tissues, but that they may at least prevent the infection of the intact portions of the bowel."

Dr. Symons (36) writing from Sorrento, Italy, in reference to the treatment of amoebic dysentery recommends rectal douches every four hours of a solution of Tannic acid $\frac{3}{4}$ to a pint of warm water. He says that he has found that this treatment reduces quickly the number of the 'stools' and relieves the tenesmus. For the remainder of the treatment he also recommends a little opium by the mouth, rest in bed and milk diet.

No observations seem yet to have been made as to the curative influence of 'salines' or 'sulphur' in amoebic dysentery.

It is curious that Ipecacuanha and Calomel seem to have but little curative effect in amoebic dysentery, and rather strengthens the hypothesis I put

forward that the amoebae are not the primary etiological factors in the disease. For both these drugs are hepatic stimulants, and thus increase the flow of the bile. In amoebic dysentery as a rule the lesions are situated chiefly in the caecum and ascending colon, and here the majority of the amoebae would naturally be found. Now the amoebae have a natural distaste for acid media, so much so that if a stool containing amoebae be left standing till the reaction is acid, the amoebae perish. Thus drugs that increase the flow of bile into the intestine ought to be markedly useful in amoebic dysentery, especially, as the bile acids would at least impede the activity of the amoebae, if they are really the authors of the disease.

Treatment of Complications and special forms of Dysentery.

I. Liver abscess. It would be quite outside the scope of this thesis to discuss fully the treatment of hepatic abscess; here, I can only indicate very briefly the present mode of treatment.

Immediately when symptoms of congestion of the liver make their appearance in any case of dysentery,

intestinal antiseptics should at once be employed, and when the diagnosis of liver abscess is quite clear, ipecacuanha should be dropped (40). Personally, I would suggest leaving off earlier, or not giving the drug at all in cases where congestion of the liver was suspected.

The medicinal treatment of liver abscess is merely to put on poultices and apply counter-irritation over the region of the liver, and to go on treating the dysentery and fever.

The old expectant treatment has now passed away for good, (at least it is to be hoped that it has!). It used to consist in watching and hoping that the pus would become absorbed, or that it would burrow and find a way out for itself by bursting into the right lung and thus being coughed up, or into the intestine to be discharged by the bowel. They feared to operate because the old open method of opening the abscess gave such a high mortality.

The present treatment first proposed and carried out by Manson, is purely surgical. Briefly put, it consists in exploring the liver with a needle for pus, and the diagnosis being confirmed by having found it; the surgeon then proceeds to plunge into

the liver a specially constructed trocar and cannula, the trocar is removed and the abscess is drained. Out of five cases operated upon in Ispahan, Persia, three died and two made a good recovery. We noticed in the three who died, that they commenced going down hill as soon as bile made its appearance. They went on draining away almost pure bile and rapidly became exhausted. The two who recovered lost no bile.

There has been much controversy over this operation--many have not hesitated to call it unsurgical, working in the dark, etc., but the results gained have proved that it is a great advance upon the old method, which although a much more serious operation, yet did not give nearly such good results as this new method.

II. Continuous fever. If the fever is very high give twenty to thirty grains of Antipyrin, otherwise proceed with the treatment of the dysentery.

III. Malaria. In every case of dysentery it is as well to examine the patient's blood and search for the malarial parasite. When malaria complicates dysentery, Quinine must be given in full doses either by the mouth or by intramuscular injection,

alternating with full doses of Ipecacuanha (23) or salines. If the dysenteric symptoms intermit, the chances are that the dysentery is merely symptomatic of the malaria and it is best to treat with full doses of Quinine only.

The Quinine will also probably be sufficient to check the haemorrhages which are often characteristic of Malarial dysentery, if not, Ergotin must be injected.

Davidson speaks of a special form of Caecal dysentery accompanied by malaria and great prostration in which Ipecacuanha is contra-indicated. In its stead he recommends Quinine, small purgative doses of Castor oil combined with 20 to 30 minims of oil of Turpentine, also frequent small doses of wine. (1).

The malarial dysentery which attacked the troops in Mauritius in 1867, resisted all forms of treatment except large doses of Perchloride of Iron.

In all cases of malarial dysentery change of air is urgently needed.

All the iron preparations are useful in dysentery where there is any malarial taint.

IV. Haemorrhage. When this complication occurs in dysentery from the erosion of an intestinal blood-vessel, if the source of the bleeding be situated in

the Rectum or Sigmoid flexure, astringent injections may prove sufficient to stop it; if not, or if the bleeding occurs from a site higher up in the bowel, a full dose of Ergotin (mx) must be injected hypodermically.

V. General Peritonitis following perforation of an ulcer. Case is then hopeless, the only treatment being to keep the patient under the influence of Morphia.

VI. Toxaemia, giving rise to typhoid symptoms. The treatment is the same as that already indicated for gangrenous dysentery, but give large enemas of warm water containing either Carbolic Acid, Permanganate of Potash, or Oil of Turpentine.

If there is much fever present five grains of Quinine may be given three times daily.

If abscesses appear they must be opened and drained antiseptically. (23).

VII. Scurvy. Scurvy in combination with dysentery renders the prognosis much graver. Milk diet is absolutely essential in the treatment, and if possible, the milk should be fresh and unboiled. Plenty of fruit, e.g., grapes, oranges, pomegranates or guavas should be given, the patient being warned to reject the skin and stones, or better still if

able to be procured, fresh lemon and limejuice.

Bael fruit has been recommended in the form of sherbet.

If the motions contain dark liquid blood, give Pernitrate of Iron; if it fail Oil of Turpentine m 15 to 30. If Ipecacuanha is being given, it is wise to combine it with Opium.

Davidson remarks "there is a dysenteric Scorbutus as well as a scorbutic dysentery, the drain on the system causing scorbutic spots to appear." (2). Then the more urgent indication is to stop the dysentery.

VIII. Rheumatism. When this complication appears in cases of dysentery, local applications (e.g., Opium and Belladonna) generally prove sufficient, the affected joints being wrapped up in wool and swathed in flannel.

Day (50) during the South African war observed a case of synovitis complicating dysentery which shewed an apparent exacerbation of the dysentery at the time of the onset of the synovitis. It started at the metatarso-phalangeal joint of the great toe, and shewed all the symptoms of acute gout but would not yield to treatment. The patient suffered a good deal of pain; the temperature varied from 99° to 102.6° F.

Salicylates pushed to salicysm had no effect, but local anodynes gave relief. The ankles and knees also became affected. The attack gradually aborted, and the patient became convalescent.

IX. Paralysis. This complication of, or sequel to dysentery is associated with anaemia and debility and indicates the need for tonics, e.g., iron, quinine, nourishing diet, and if necessary, constant or interrupted currents of electricity. (23).

X. Typhus or Typhoid. If one or other of these diseases complicate a case of dysentery, the treatment will vary according to which disease predominates, and also according to the general condition of the patient.

APPENDIX to THESIS

and

LITERATURE.

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A P P E N D I X.

(A). OTHER CLASSIFICATIONS of DYSENTERY.

I. Davidson (1):-

(A). Non-amoebic form--Varieties,

	(Catarrhal
Acute	(Inflammatory
	(Croupous
	(Diphtheritic
Chronic	

(B). Amoebic form.

(C). Special forms and complications.

II. Osler (3):-

Acute specific dysentery.

Amoebic dysentery. (Acute
(Chronic

Acute catarrhal dysentery,
(Acute ileo-colitis).

Diphtheritic dy- (Primary
senterly. (Secondary.

N.B. Not a good classification; it does not sufficiently distinguish between the amoebic and non-amoebic forms, and omits 'Chronic' dysentery entirely from the latter.

III. Manson (24):-

- i. Catarrhal dysentery.
- ii. Ulcerative "
- iii. Gangrenous "

A. (Continued)

N.B. Unfortunately a too simple classification in the light of our present knowledge of the disease. It is a purely pathological classification according to the gross characters of the intestinal lesions.

IV. Lafleur (6):-

Amoebic dysentery,

- (A). Grave or gangrenous form.
- (B). Dysentery of moderate intensity.
- (C). Chronic form.

V. Davidson (2):-

Non-amoebic dysentery, (i. Epidemic
(classed under (ii. Endemic
'Etiology!'). (iii. Dysentery of war
and famine.

(classed under Morbid Anatomy).

- I. Acute dysentery, (Purulo-gangrenous.
(Fibrinous or
(pseudo-diphtheritic.

N.B. 'Catarrhal' is regarded only as an initiatory stage in both varieties.

- II. Chronic dysentery.
- III. Modified and complicated dysentery,
 - (Malarious
 - (Scorbutic
 - (Arthritic.

A. (Continued)

N.B. I prefer to treat 'Catarrhal dysentery' as a distinct variety of non-amoebic dysentery, as well as an initiatory stage to ^{of} the more serious forms. More especially have I considered its relation to climate under the cognomen 'Climatic dysentery,' where in Persia, at least, it seldom goes on to true dysentery, while it yet has essential characteristics distinguishing it from mere 'summer diarrhoea.'

APPENDIX B. Maps shewing gradual though irregular increase of dysentery upon approaching the Equator, (vide Etiology).

I. Eastern Hemisphere. The figures given represent death-rate per 1,000 and all the statistics are gleaned from Army medical reports.

N₄₀ is the furthest usual limit North for endemic dysentery.

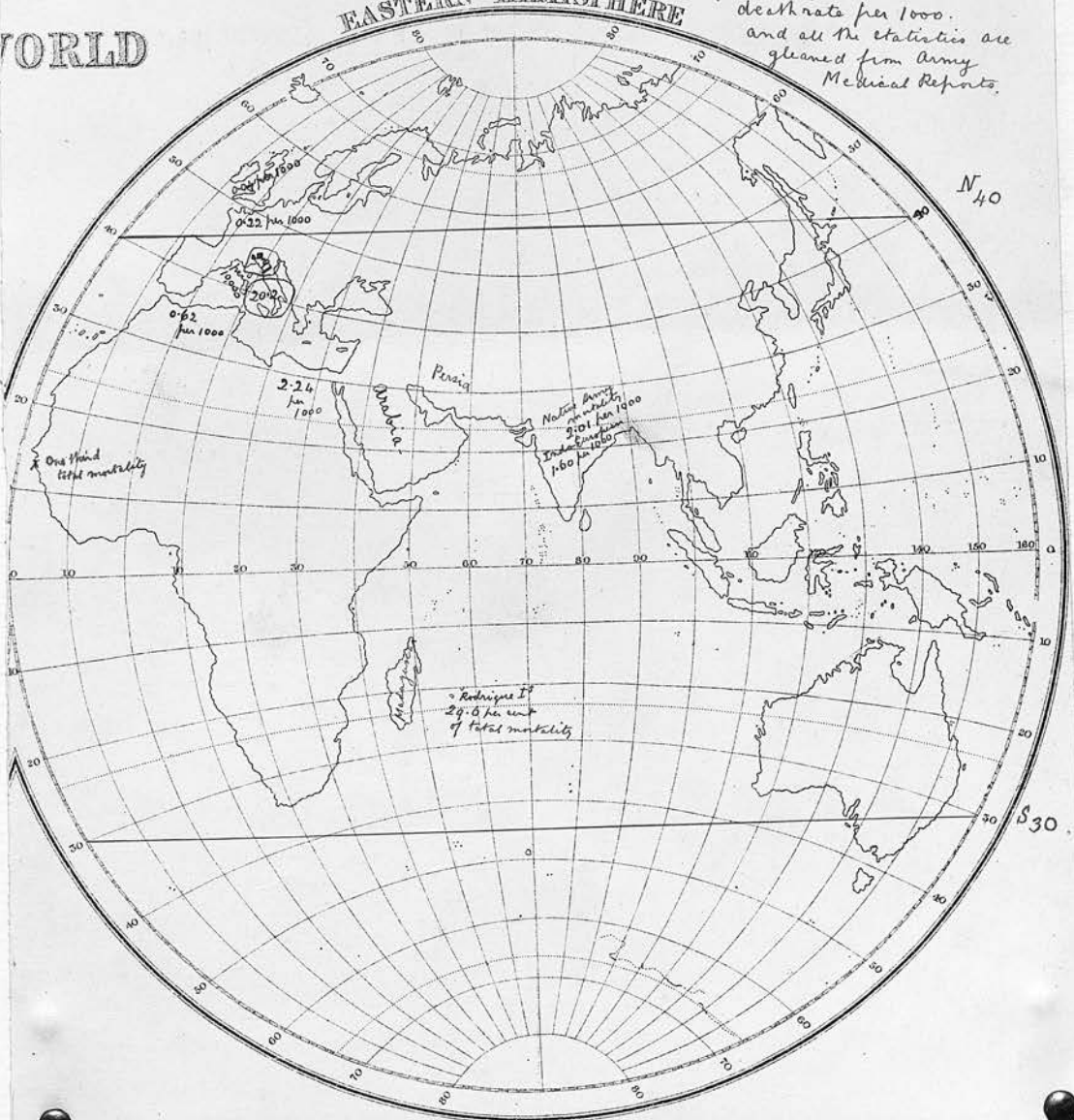
S₃₀ is the furthest usual limit South for endemic dysentery.

Map to show gradual though irregular increase of dysentery upon approaching the Equator.

The figures given represent death-rate per 1000. and all the statistics are gleaned from Army Medical Reports.

WORLD

EASTERN HEMISPHERE



APPENDIX B. (Continued)

II. Western Hemisphere. Map showing increase of frequency of dysentery upon nearing the Equator, the increase in frequency being neither constant or uniform. The statistics are taken from a Table showing ratio of admissions for acute dysentery per 1,000 of the troops.

Numbers on map = ratio of cases per 1,000 of mean strength.

Numbers in brackets = proportion of deaths to cases. N₄₀ is the furthest usual limit North for endemic dysentery.

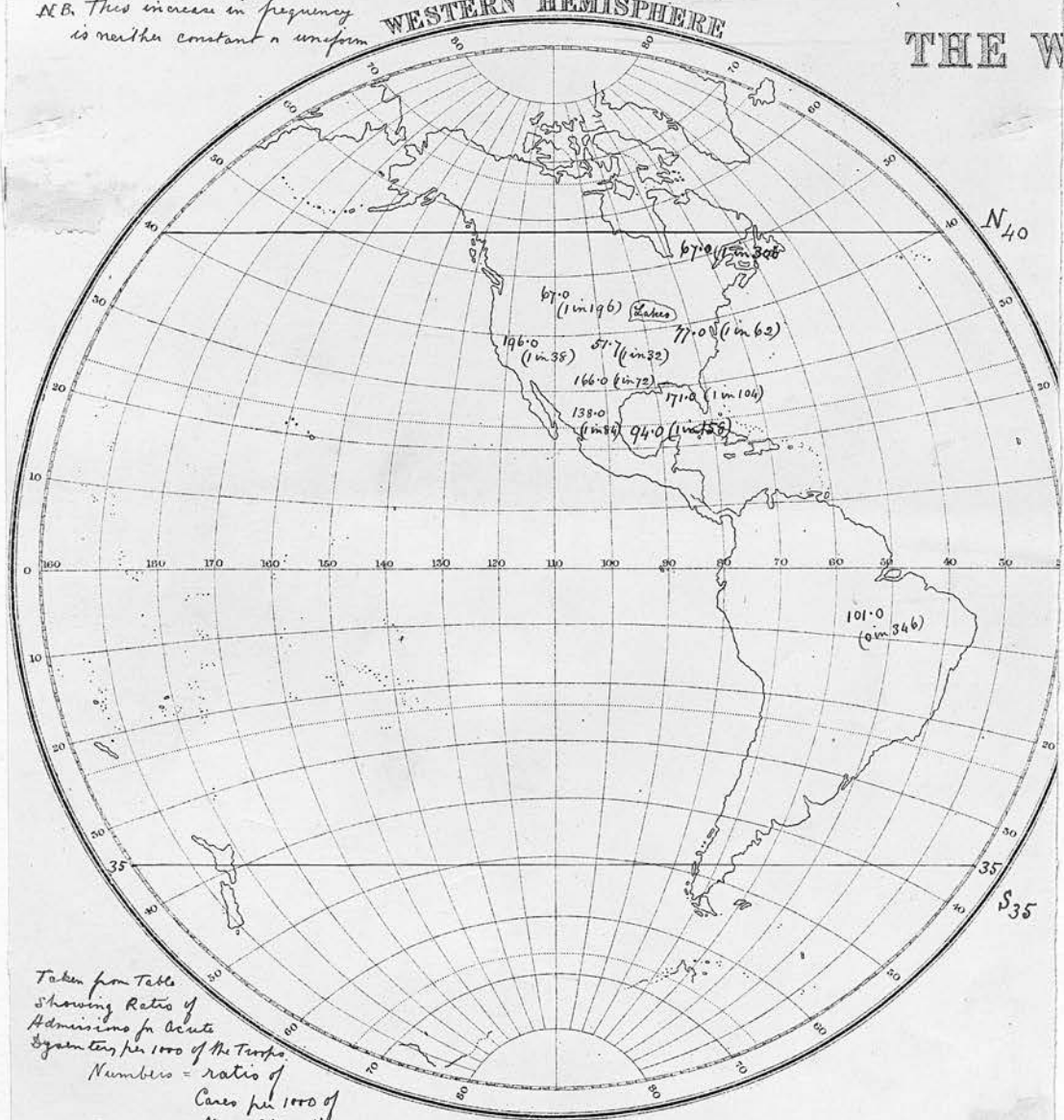
S₃₅ is the furthest usual limit South for endemic dysentery.

Map showing increase of frequency of Dysentery - upon nearing the Equator

N.B. This increase in frequency is neither constant or uniform

WESTERN HEMISPHERE

THE W



Taken from Table showing Ratio of Admissions for Acute Dysentery per 1000 of the Troops. Numbers = ratio of Cases per 1000 of Mean Strength.

Numbers in brackets = Proportion of deaths to Cases. (from Davidson's Hygiene & Diseases of Tropics P 551)

(Statistics obtained from Davidson's Hygiene and Diseases of Tropics, p.551).

APPENDIX C.

Tables compiled by the writer shewing the great similarity in some of the symptoms of:-

- I. Acute Caecal and Acute amoebic dysentery.
- II. Chronic dysentery and Chronic amoebic dysentery.

I. Acute Caecal dysentery.

1. Onset, insidious or sudden.
2. Motions more abundant more faeculent, and less frequent than in Rectal dysentery.
3. Straining not marked, and may be absent.
4. Caecum and ascending colon usual sites of lesions.
5. Characters of stools in different stages of the disease:-
 - A. Faeculent.
 - B. Pale and frothy, one part of same stool may be tinged bright yellow, another green, and all are mixed with blood and mucus.
 - C. Watery, chocolate coloured, extremely foetid.
6. Pain and tenderness to pressure in right iliac fossa.
7. More apt to become chronic than Rectal form.
8. Relapses more common than in Rectal form.
9. Much slower reaction to treatment than in Rectal dysentery.
10. More apt from site of lesions to develop hepatic abscess.
11. Diarrhoea at first painless.
12. Vomiting may be severe.
13. Amoebae morphologically the same as the Amoeba dysenteriae have been found in the stools.

Acute Amoebic dysentery,
(Lafleur 6).

1. Onset may be gradual or abrupt.
2. If onset be gradual motions are at first faeculent, and not very frequent.
3. Straining is not a marked feature in Amoebic dysentery.
4. Caecum and ascending colon are usual sites of lesions.
5. Characters of stools in the different stages:-
 - A. Faeculent.
 - B. Blood and mucus, often greenish (like green scum) or grayish yellow, often containing necrosed masses derived from the ulcers.
 - C. Watery, reddish brown, extremely foetid.
 - D. Same, mixed with shreds & sloughs.
6. Pain and tenderness to pressure may be general or localised in epigastrium or lower abdominal zone.
7. Marked tendency to chronicity.
8. Characterised by exacerbations and remissions.
9. Recovery is slow.
10. Abscess of liver in 19 per cent of cases.
11. Diarrhoea at first painless.
12. Nausea and vomiting are not uncommon.
13. Amoeba dysenteriae always present in the stools.

APPENDIX C. (Continued)

II. Chronic dysentery,
(lesions may or may not be confined to the Caecum).

1. May start insidiously with diarrhoea, which intermits and recurs.
2. Marked by exacerbations alternating with remissions.
3. During quiescence.
Stools may be well formed with thin flakes of mucus attached to their surface, but more frequently they are thin, frothy, offensive, yellow, green or light coloured, mixed occasionally with specks of blood and small masses of mucus.
During exacerbations.
Stools are watery, reddish-brown, mixed with blood, mucus and pus, dysenteric (mawkish) odor.
4. Disease may last from ten to fifteen years.
5. Exacerbations last from 2-10 days.
6. Not much straining except during exacerbations.
7. Pain only during the exacerbation.
8. Death from Asthenia, or intercurrent affections.
9. Fever rare.
10. Patient becomes anaemic, health fails, tongue red and glazed, vomits occasionally, pulse weak and quick, loss of appetite, flatulence, uneasiness after ingestion of food, night sweats.

Chronic Amoebic dysentery.

1. Starts with moderate and painless diarrhoea alternating with short periods of constipation.
2. Characterised by exacerbations alternating with remissions.
3. During quiescence.
Stools become pasty, and even partly formed, abnormal constituents generally disappear except the mucus which is still found mixed with the faeces, or more generally adhering as flakes to the surface.
During exacerbations.
Increase of blood and mucus, becomes more liquid.
4. Chronic cases last months or years.
5. Exacerbations last from 1-10 days.
6. Tenesmus is rare.
7. Ordinary chronic cases are painless.
8. Death from Asthenia, or as result of complications, e.g., Hepatic abscess.
9. Fever rare.
10. Patient becomes anaemic. Pulse quickened in exacerbations. Termination of chronic cases when they prove fatal same as in ordinary Chronic dysentery.

These tables have been compiled from the symptomatology

of Dysentery, and reference has been made to La-
fleur's Art. on Amoebic Dysentery (Allbut's System
of Medicine, vol.2); Davidson's Art. on Non-amoebic
Dysentery (Allbut's System of Medicine, vol.2);
Davidson's Hygiene and Tropical diseases (Art. Dy-
sentry), and other sources.

APPENDIX D.

The sloughs found in dysenteric stools are of all kinds of shape and size. The following classification of these sloughs is associated with the name of Dr. Chuckerbutt, (vide Indian annals of Medical Science, vol.X, p.90):- (45)

"Molecular sloughs or putrilage show disintegration of tissue.

Flaky epithelial sloughs show commencing gangrene in the mucous membrane.

Thin black sloughs, plain or tubular, show primary gangrene of the mucous coat.

Shreddy, ragged and dark olive sloughs indicate gangrene in either mucosa or cellular coat.

Thick pus-infiltrated sloughs show erysipelalous dysentery--a very dangerous form.

Grey or light yellow sloughs, plain or tubular indicate phlegmonous dysentery.

Free, gelatinous or cellular sloughs, when simple, show primary gangrene in the submucous connective tissue; when infiltrated with pus indicates submucous cellulitis.

Ring-shaped sloughs show ring-shaped ulceration in mucous folds.

APPENDIX D. (Continued)

Discoid sloughs indicate circular ulceration in ecchymosed patches.

Ecchymosed sloughs show abraded minute ulcers and intestinal apoplexy.

Tubercular sloughs, if detected, are diagnostic of tubercular dysentery."

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