CHAPTER No. II.

REVIEW OF THE DISEASE
DYSENTERY

1] DYSENTERY & GENERAL CONSIDERATION.

2] AMOEbic DYSEntERY.

3] BACILLARY DYSEnTERY.
We have to see first the two common terms i.e.

1] Amoebiasis

2] Shigellosis. Both terms show an infectious stage.

1] Amoebiasis:- Amoebiasis is an infection of the large intestine produced by Entamoeba histolytica.

2] Shigellosis:- Shigellosis is an acute infection inflammatory colitis due to one of the member of the genus shigella.
Definition:-

A good definition is given by stitt -

The designation dysentery refers to a symptom complex of

1] Small, frequently passed mucous or muco sanguinolent stools and

2] Pains connected with spasm of the sphinctor ani (tenesmus) or intestinal griping (tormina).

The condition may be set up by numerous causes but of these two so outweigh the other that it is usual to have in mind either bacillary or amoebic dysentery when the term is employed

SYNONYMS -

* The Bloody Flux.
* French :- Dysenterie.
* German :- Ruhr.
* Sanskrit :- Pravahika.
DYSENTERY A GENERAL CONSIDERATION.

Since the days of Hippocrates, dysentery and suppurative conditions of the liver are recognised. Before the days of Charak, Susrut, Agasthya and Hippocrates, some of these cases were described under the entity "Atisar" in ancient Indian medicine.

So we can say definitely about the disease which was existed in Egypt and India for centuries before Christ.

According to history in 1898, the Shiga found bacillus of dysentery and that time it was generally accepted. Before that there were so much factors i.e. etiologically epidemiologically described of the subject.

For differentiate conditions of dysentery one is, admixture of mucus and blood in the stool and second is - stool with blood alone.

But many of the old writers failed to differentiate the conditions. But from last hundred years all have considered from the association of mucus and blood with stool as essential in clinical diagnosis.
With the help of better knowledge of etiology we are now recognizing as of dysenteric nature diarrhoeal conditions in which there is an absence of the typical stool of dysentery.

The Term Dysentery.

By the term dysentery we understand a symptom complex of

1] More or less characteristic stool.

2] More or less characteristic pains.

It is described that there is a greenish yellow or dirty brown mucus in little quantity with stool. The blood intimately admixed with the mucus or we can see a whitish grayish muco purulent mass with streaks or fleks blood more or less faculent discharges which are usually small in amount and passed with much frequency.

There are two another terms related with dysentery. One is tormina and second is tenesmus.

The both terms i.e. tormina and tenesmus are used to designate the characteristics of the pains of dysentery.

1] Tormina - It is used for the griping colicky pains, which center about the umbilicas or run in the direction of the large intestine.
2) **Tenesmus** - It is used for the painful spasmodic contraction of the sphincter ani. It is due to sensation of lack of ability to compete the act of defecation leading to straining and justifying Mansooni’s description "glued to the commode".

It is usually stated that

1) The nearer the dysenteric process is to the rectum, the greater tenesmus.

2) The nearer to the caecum the greater the tormina.

**Classification of Dysenteries.**

The classification of Dysenteries in modern ege is based on etiology rather than upon clinical manifestations. There are two main kinds of Dysentery:

I] Amoebic.

II] Bacillary.

I] Amoebic - that caused by *Entamoeba histolytica*.

II] Bacillary - that caused by some strain of *Bacillus dysenteriae*.

We will consider them separately from the other causes of the dysenteric symptom - complex.
Classification of dysentery can be described in three groups.

I] Dysenteries caused by animal parasites.

II] Dysenteries caused by bacteria.

III] Dysenteries resulting from mechanical irritants or poisonous substances.

I] Dysenteries caused by animal parasites.

Dysenteries caused by animal parasites differentiated into two groups.

A] Protozoal Dysenteries.

B] Helminthic Dysenteries.

A] Protozoal dysenteries is classified in four types.

1] Amoebic dysentery (Entamoeba histolytica)

2] Flagellate dysenteries (Lamblia intestinalis, Trichomonas intestinal and chilomastix mesnili)

While in adults these intestinal flagellates usually cause only a diarrhoea with following characteristics.
I] Marked nervousness.

II] They may produce dysenteric symptoms in young children.

III] The onset in children under three years of age may be insidious and attended with fever.

IV] The stool contains much mucus with only a little blood.

In cases of amoebic dysentery, at the same time if there are some diarrhoeal attacks, these diarrhoeal attacks are often associated with the plenty of Flagellates. It may be the cause of complication.

Gallipoli who has reported cases of dysenteric diarrhoea. In these cases he has reported Lamblia (Giardia) were apparently the only parasites involved.

With the involvement of Lamblia stools are often of a yellow ochre color. In Lamblia infection there is a common feature i.e. relapse of infection.

Upper part of intestine is a living sight of Lamblia while Trichomons and Chilomastix have sight of large intestine, especially in the region of the caecum.

Chilomastix mesnili are often found in stools those convalescent from dysentery. But about this there is a general opinion that chilomastix are not
pathogenic. These organisms may be present in diarrhoeal condition in which case it is common to designate such diarrhoeas are called flagellate diarrhoeas.

One hundred and eighty seven cases of pure Lambliasis are reported by Fantham and Porter.

Structure of Lamblia -

It is important in diagnosis to confirm the encysted Lamblia. These are oval shape cysts, about 10.×7 μ and show a curved central line with two latera dots when stained these dots show as chromatin areas. In the faeces these cysts may be found in plenty numbers. The vegetative Lamblia has four pairs of flagella, is about 15 μ long and has a tumbling motion.

3] Ciliate dysenteries (Balantidium coli)

It is a severe type of dysentery may caused by various ciliates, it is very exceptional that others than Balantidium coli do so. These are oval shaped ciliate. These ciliate are long about 60 to 100 microns and broad about 50 to 70 microns. It is a commensal of hogs and the disease in man is usually found in those having care of hogs. Infection of this type of dysentery have been reported form various parts of the
world, temperate as well as tropical region. These ciliates may be found in the faeces of persons apparently well but in such cases symptoms may eventually appear.

The multiplication of parasites take place in the submucosa and the process of pathology is similar to that observed in the large intestine in amoebic dysentery.

**Structure of ciliate parasite:**

It is very simple to detect or impossible to fail to detect in a microscopic examination of the faeces. Because the ciliate parasites are so large and have an active motility. Encysted parasites are round. The onset of disease is rather insidious with diarrhoea which may be followed by dysentery. It is noted that there may be a severe form of anemia.

**4] Dysenteric conditions in the diseases:-**

Two disease are described having condition of dysentery. One is kala-azar and second is malaria.
In the terminal stage of kala-azar (Leishmania donovani) dysentery manifestation is noted and it is also noted in algid pernicious malaria (Plasmodium falciparum). These conditions are taken up under the diseases kala-azar and malaria.

There was a case of dysenteric syndrome of coccidial infection (Isospora hominis) noted by Wenyon. Infections with this parasite about seventy cases have been reported chiefly from soldiers serving in Gallipoli. The usual opinion is that they are non-pathogenic parasite.

The cysts are with one end narrowed, and measure 28*14 microns. There are two sporocysts each of which contains four sporozoites. The cyst when passed is unsegmented.

**B] HELMINTHIC DYSENTERIES**

We have seen the protozoal causes of dysentery above. In addition, we may have some dysenteric symptoms in following infections.

1) Termatodes, especially Schistosoma mansoni and S. japonicum. In these cases we have mucus coated stool with more or less clotted blood in which mucus we may find the diagnostic ova. A rather high eosinophilia is present.
2) There are also dysenteric manifestation caused by infections with gastrodiscus hominis.

3) **Heterophyes** - Heterophyes is a very small cestode. And it has been noted to cause a condition suggestive of dysentery.

4) The Brumpt in 1902 noted the finding of a nematode, *Oesophago-stomum brumpti* in the large intestine of an African native, which caused dysenteric symptoms and more recently, another species, *O stephanostomum*, has been reported as causing a fatal dysentery in Brazilian at Manaus.

5) Cases with dysenteric manifestations which were apparently connected with intestinal myiasis also have been reported.

**II] Dysenteries caused by bacteria.**

1) Dysenteries caused by bacteria are either the more toxic, nonacid mannite strain of shiga, or the less toxic, acid mannite strains of the Flexner group.

2) B.Morgan No.1 :- B.morgan No.1 as the cause of certain bacillary dysenteries has reported by Morgan. Characteristics of B.morgan No.1.
B. morgan No.1 is motile produce indol and in glucose bouillon gives a very slight amount and does not produce a primary acidity in litmus milk. This organism i.e. B morgan no.1. is a frequent cause of summer diarrhoea of children. Flies from houses with such cases often show morgans bacillus.

A clinical picture of colitis may caused by paratyphoid infection, in such cases some time show a large amount of blood in the dysenteric stools. Usually the symptoms are rather those of an entero-colitis or a gastro-enteritis.

3) Dysentery - like epidemic of a very total disease called Ekiri in Japan mostly occur among young children. The organism, the cause of Ekiri is very motile. It produce gas and acid in glucose but not in lactose media it is reported at times show indol production. Organism apparently a member of the Gartner group.

4) Spirillar Dysentery - Another type of desentery reported by LeDantec. The type shows the presence of spiral forms of great number. It is called as spirillar desentery. These organisms are Gram
negative and noncultivable. It is in question whether they belong to bacteria or not. There is no fever in this type of dysentery.

5) Other bacterial causes:– There are cases reported of dysentery caused by organisms namely B. pyocyaneus. Streptococci, a typical B. coli and organisms of the Gartner group.

Dysentery caused by Pyocyaneus infection the colour of the stool would be suggestive. This cause should be born in mind in the dysenteric infection of debilitated children in the tropics. Some of the cases of so-called protomine poisoning due to member of Gartner especially at the commencement of the attack.

III] Dysenteries resulting from mechanical irritants or poisonous substances

A experiment reported from North China that through the use of short lengths of bistles which are given mixed with the food. It was very interesting form of poisoning. The poisoning gives rise to serious illness or death. In this condition there were marked abdominal pain and manifestations of desentery.

Arsenic, antimony, mercury and such type of various
metallic poisons may give rise to dysenteric symptoms. In the disease cancer and syphilis of the rectum there may be a suspicion that the process is an ordinary dysenteric one.

Intussusception show marked tenesmus with bloody rather than muco- sanguineous stools.

In the terminal stages of various chronic diseases especially tuberculosis and cardiac affections, may give the symptom of dysentery. It is also in chronic nephritis, leading to uremia, we may see symptoms of a marked catarrhal or even diphthentic colitis.
AMOEbic DYSENTERY

HISTORICAL BACKGROUND

Lambal - The first man who has noted amoebae in human body 1859. He found amoebae in the stools of a child affected with diarrhoea.

In 1975 Losch who has described first accurately the parasite. He found amoebae in the intestinal ulceration and found it in stool of a patient of chronic dysentery.

Losch also succeed to produce dysentric ulceration in the dog by injecting faeces containing with amoebae in to the rectum of the dog.
Grassi in 1879 noted the encysted forms of amoebae. But the denied their pathogenic importance because he found them in normal people.

Cunningham found amoebae in the stools of cholera patients.

Perrocito found amoebae in typhoid patients.

But both Cunningham and Perrocito put the question of pathogenicity of amoebae as Grassi did.

Hence a firm causal relation between the entamoeba and amoebic dysentery was still not established.

Koch in 1883 was investigating cholera in Egypt. He found entamoeba in the section of dysenteric ulcers and considered that this fact favored the view that amoebae were pathogenic.

Work of Koch was continued by Kartulis. He published his findings in 1886 with 150 cases of dysentery. He found the presence of amoebae in the stool of all these 150 cases. In 1887 Kartulis also found amoebae in the pus of liver abscess.

I Lutz in 1891 noted that amoebae in dysentery contained red cell. In the same year council man described the characteristic lesions in the bowel and named the entity as amoebic dysentery.
Councilman and Lafler came to the conclusion there were two types of amoeba in human body. Out of them one is harmless and the second one pathogenic which was found in the submucosa of intestinal ulcers.

Casagrandi and others put a new view that amoebae only acted as carriers for bacteria. But Kruse and Pasquale in 1893 did an experiment. They injected all the bacterial species isolated from a dysenteric stool in to a cat’s rectum and found negative results.

Hlava and Kartulis first produced dysenteric lesions in cats by injecting per rectum, amoebic stools. Kruse and Pasquale injected bacteria free pus from liver abscess which however contained amoeba in cats per rectum and produced dysenteric lesions.

Many cases of typical dysentery failed to show amoeba so definite relation between amoebae and dysentery was not perfectly established.

Shiga was the first person who has settled this matter. He presented a group of bacilli which were concerned in the production of dysentery.

These findings of Shiga were accepted and confirmed all over world. Deferentiation gradually obtained of cases of dysentery from amoebic infection as well as from bacillary infection.
During the period 1893-1903 Schandinn, Huber, Qunke and Roos demonstrated the life cycle of the parasite and Schandinn clearly established its etiological relation to dysentery (McTiardy - Disease a Month Aug 1957).

Walker (1911) and Sellard (1913) carried out a number of experiment's on human volunteers and described in detail the development and differentiation of Entamoeba coli and Entamoeba histolytica and the pathogenecity of the latter. Walker also described the carrier state in amoebiasis (Faust E.C. Amoebiasis, Springfield III, 1954)

Geographical Distribution

Amoebic dysentery is prevalent in most parts of the world. To be specific the disease is found in the following places.

1. Indo - China.
2. China.
3. Phillipines.
4. In some parts of India.
5. Egypt and North Africa.
6. It has also rooted in South America, especially in Brazil.

7. This disease is also common in West Indies and central America.

8. It is also an important disease in the southern states of USA, in Italy and other parts of southern Europe.

9. It also exists in greater or less degree in most of the tropical and subtropical parts of the world.

**ETIOLOGY WITH HISTORY**

It was very difficult for long time to differentiate between pathogenic and nonpathogenic species.

So the authorities of this subject in Manila took the view that principal factor in the production of dysentery was that of symbiosis between amoebae and suitable bacteria.

Authorities also observed in culture of amoebae evidence of both symbiosis and antagonism on the part of amoebae to certain species of bacteria.
They were convinced that pathogenic amoebae could be cultured on a medium of about 1/10th the strength of ordinary nutrient bouillon or agar and that dysentery could be produced by such cultural amoeba.

This type view taken by authority had an important bearing on epidemiology that where amoeba could be cultured from green vegetables, fruits or water supply. There was a positive evidence of possibility of infection with amoebic dysentery from above sources.

But above conclusion or views are no longer accepted because of work of Walker. Walker was working in Manila with experiments on man. We know that cultural amoebae are without effect in the production of dysentery. There are certainly two well known species of amoebae having man for a host. One is pathogenic and another is non pathogenic or harmless to man. Pathogenic is Entamoeba histolytica and non pathogenic is Entamoeba coli.

More two species of non pathogenic have been described one is Enddimax nana and second is Iodamoeba butschlii.

Some authorities prefer the generic name of Entamoeba i.e. Loschia and Endomoeba.
Description of pathogenic amoeba i.e. E.histolytica given by Schaudinn in 1903 as follows.

1] Distinct, highly refractile and tenacious ectoplasm. Schaudinn concluded this tough external portion of the cytoplasm as the explanation of the ability of the pathogenic amoeba to bore its way into the intestinal submucosa.

2] Eccentric nucleus which was indistinct by reason of little chormatin.

3] Reproduction by peripheral budding in which small aggregation of chromatin reached the periphery of the cytoplasm and, enclosed in a resistant capsule, broke off from the parent amoebae and constituted the infecting stage.

Schaundinn also described non pathagenic amoeba i.e. E.coli as under.

1) No distinction between a granular endoplasm and refractile ectoplasm.

2) Nucleus is centrally placed and sharply outlined, rich in chromatin.

3) Encystment with the formation of eight nuclei which cysts with their nuclei or amoebulae form the infecting stage.
Defferentiation between E histolytica and E coli.

1) The psedopodia of E histolytica are actively projected and as finger like process. They show ectoplasm quite distinctly.

   The psedopodia of E coli are lobed and sluggishly projected and show uniformly opaque greyish color.

2) E. histolytica tends to show contained red cells.

   E.coli never contain red cells but instead of red cell they show bacteria and food particles.

   Viereck and Hartman noted a pathogenic amoeba in 1907. It was with four nuclei in its encysted form. It was given name E.tetragene.

   Observation made by Schaudinn are not accepted by authorities. They consider that there is an error in observation as to existence of peripheral budding for E.histolytica. They recognize two types of encystment one with a larger cyst, and thicker cyst wall, having eight nuclei and an absence of chromidial bodies. The other smaller with a thin cyst wall one to four nuclei and chromidial bodies in the encysted stage. It is pathogenic amoeba i.e. E.histoltica Synonym E.tetragena.

   The human amoebae are best differentiated in the vegetative stage,are best differentiated by the structure of nucleus in stained specimens.
Structure of E.coli:– The nucleus is vesicular with a thick nuclear membrane. The chromatin chiefly deposited on the under surface of the nuclear membrane. Thus chromatin often seems deposited in quadrant aggregation. The karyosome is eccentric.

Differentiation between two types of pathogenic amobae recognised by authority.

1) A histolytica type of nucleus which is formed in dysenteric stools.

2) A Tetragen type which is found in diarrhoeal or more or less normal stools Dobell does not recognize this differentiation.

The histolytica nucleus has a thin nuclear membrane it is poor in chromatin. The tetragena nucleus has more chromatin, showing radial projections from the inner surface the Nuclear membrane and a loose central spherical karyosome which contains a central chromatin dot or centriole, with a clear halo surrounding it. Dobell states he has not been able to note this centriole.

The preencysted E.histolytica has a nucleus resembling that of E.coli. The smaller size and chromidial bodies are differentiating.
Schaudinn, Craing and Wenyon have been unsuccessful to production of dysenteric manifestation by animal experimentation upon kittens with E. coli.

On the other hand material containing pathogenic amoeba injected rectally or fed in kittens authorities succeed to produce typical lesions and dysenteric manifestation.

Darling in his research with kitten has been successful. He compares the colon of a kittege central karyosome. The cysts are spherical or oval in shape. Often these are found in irregular outline and are about 10 microns in diameter. The nucleus is large and eccentrically placed. It has a karyosome which goes to show as a peripherally placed mass. There is almost always present a large glycogen mass in the cyst. The cyst stains intensely with iodine.

**Dientamoeba Fragilis**

It is a rare type of amoeba, established by Jeeps and Dobell in 1918. It is binucleate amoeba. Its average size is 8 to 9 microns. Its nuclei show a fairly large, central granular karyosome and there is no peripheral chromotin. Strands of linin may be seen.
radiating from karyosome to nuclear wall. This organism is said to be frequently mistaken for Blastocystis when the vacuoles coalesce leaving a thin ring of cytoplasm. Cysts are unknown. These are considered non pathogenic organism. Kofoيد and Swezy reported another species of amoeba parasite in man. It is known as councilmania lafleuri. The adult and cystic stages resemble in many respects E.coli. The adult is said to ingest red blood cells. It is said to be pathogenic. But it is not confirmed by all.

**Human Experiments**

Walker and sellards who have been experimenting on human lives. They have published a most important paper on the subject.

The experiments were made at Bilibid prison, in men who were under observation for many years.

The prison food was cooked and the water the prisoners drank was distilled moreover there were complete records of examination for intestinal parasites including entamoeba. They were under complete control and the existence or possibility of natural infection with amoeba was reduced to to minimum. All the
prisoners were fed with pathogenic amoebae after they had volunteered to be experimented upon. In fact they signed an agreement which was in their native dialect.

The first series of experiments were performed with cultural amoebae. This job of experimenting was carried out to refuse statements that amoebae cultivated from water or from other no-parasite sources as well as from dysenteric stools are capable of living in man parasitically or were capable of producing dysenteric symptoms.

Walker and Sellards carried out 20 feeding experiments on 10 men. They were fed with cultures of amoebae but yielded no result in producing dysentery or findings of such amoebae in the stools on being examined under a microscope. They recovered the amoebae in cultures in 13 cases from the faeces from the first to the 6th day but never after that.

Both the research scientists were in a position to definitely state that cultural amoebae are non pathogenic. The next experiments of second series was performed with entamoeba coil. In the twenty cases of person fed with material containing entamoeba coil, there was a total failure to recover them culturally. Also in no instance could dysentery be produced. Seventeen
became parasitized as a result of a single feeding from one to eleven days. The entamoebae were found in the stools and persisting in their appearance in the stools for extended periods. They concluded that Entamoebae coil is an obligate parasite, non-pathogenic and cannot be cultured.

The experiments of third series of 20 feedings was carried on by Walker. This time he did experiments on his own with Entamoeba histolytica. The material was mixed with powdered starch or magnesium oxide and given in gelatin capsules. While performing this experiments he obtained tetragena in the stools of men fed with only motile Entamoeba Histolytica and motile Entamoeba Histolytica in the stools of men were fed only tetragena cysts and finally an alternation of motile E. Histolytica and tetragena cysts in the stools of a man having a recurrent attack of amoebic dysentery.

Results of human experiments.

As a result of experiments carried out seventeen of the men become parasitized after the first feeding, one required three feedings and two who did not become parasitized at the first feeding were held at controls.
The average time taken for parasitization has been found to be nine days. It was also observed that only 4 out of 18 parasitized men developed dysentery. The dysentery effect occurred after twenty, fifty-seven, eighty seven and ninety five days respectively after ingestion of the infecting material.

In 4 cases fed with material from acute dysenteric stools or from amoebae containing pus from liver abscess and containing motile amoebae, there was no resulting dysentery. However, the four cases of experimental dysentery resulted from feeding from normal stools of carriers.

As regards the cases which become parasitized and which did not develop dysentery, it is said that the amoebae live as commensals in the intestine of the host. They penetrate the intestinal mucosa. They become tissue parasites when there occurs depression of the natural resistance of the host or as a result of some lesion of the intestine. That the pathogenic amoebae are more than harmless commensals. This however is shown by the fact that they alone, and not the non pathogenic Entamoeba coli, are capable of penetrating a possibly damaged intestinal mucosa.
**EPIDEMIOLOGY.**

In the spread of amoebic dysentery to be the encysted amoebae in the stools of convalescents or symptomless carriers. It is certainly not the motile amoeba in dysenteric stools. When such carrier has to do with the preparation of food, he becomes a particular source of danger.

Of the spread of amoebic dysentery this fact probably explains the endemic rather than the epidemic characteristics. Because if the innumerable vegetative amoebae in dysentery stools were equally operative with the more sparsely eliminated cysts, there would be epidemics of amoebic dysentery similar to those of bacillary dysentery.

Water, fruit or vegetables from which one can isolate amoebae on culture will have on pathogenic relation to man. The idea, therefore that these are sources of infection must be abandoned.

After the stool is passed vegetative amoebae undergo disintegration in a short time. These therefore, have no concern in amoebic infections, but the resisting cysts may get washed from a dried stool and run into water supply. These could also be transported in the
form of dust and lodgeon unprotected food stuffs. There should be transmitting agents and flies may possibly act as transmitting agents. As bearing on the problem importance of such flies as musca domenstica and Fannia canalicularis in transmitting amoebic infection may be noted the findings of Wenyon. In that he states that the faeces of such flies on cyst-containing human faeces, teem with such cysts. Dissection of 1027 house flies were carried out in Mesopotamia. Buxton found in them ova of parasites of man in 4.00%. The percentage of infection with E histolytica cysts was 0.3%.

**PATHOLOGY**

Weynon is of the opinion that the pathogenic amoebae find their way into the tubular glands of the intestines. They multiply there and there after either by pressure of their pseudopodia or through the disintegrating action of some toxic substance elaborated by them, they force their way into the underlying submucosa. In this location they produce a gelatinous, edematous necrosis which shows a marked absence of polymorphonuclears. But a proliferation of connective
tissue cells. The process is regenerative rather than inflammatory.

Small hemispherical elevation of the overlying mucosa mark the location of the deeper lying necrotic process. The amoebae multiply there and the necrotic process in the submucosa extends. As a consequence tromi is formed in the terminals of the portal vein and possibly in those of the mesenteric arteries, which in the former case may result in emboli being swept up the portal vein to lodge in the liver. There they form a starting point for a similar necrosing process as a result of interference with the blood supply of the overlying mucosa, cause this to undergo necrosis and be cast of as a slough, leaving an oval or irregular ulcer with deeply undermined edges and a floor formed by the muscular coat. The ulcer may be no larger than pin's head or they may be 1 or 2 inches in diameter or by coalescence be still larger. The gelatinous necrosis in the submucosa always extends beyond the limits of the necrosis of the mucosa. This explains the undermining. At times the muscular coats of the intestines are involved. This leads to a slough which involves all coats except the serous one. Bacterial infectino with
coagulation necrosis of the mucosa overlying the amoebic process, is also responsible for some of the tissue destruction.

The ulceration due to amoebae extend above the ileocaecal valve. But it may involve the entire large intestine. Rogers and Lafleur found the lesions most often in the caecum and ascending colon and often limited to this particular area.

In 7% of the Manila autopsies the appendix was involved. The autopsies often show only mild cases of lesions in the caecum. When there is a tendency to perforation the omentum will often be drawn over to the location of the threatened perforation. There is often thickening of the intestine in one place with cicatricial contraction of the lumen and thinning in another. This provides a great irregularity in the appearance.

**SYMPTOMATOLOGY**

In a great majority of cases of amoebic dysentery, the disease runs a chronic course. The period of improvement alternates with recurrences of pains and dysenteric stools. According to walker’s experiments the
period of incubation would appear to be from one to three months. The onset in such cases is very insidious and the complains to the patients more of diarrhoeal than dysenteric showing. When asked such patients often give a history of passing three or four paltaceous stools daily and complain of tenderness in the region of the caecum or along the course of the large intestine. One may determine some thickning of colon in a thin subject.

In the case of amoebic dysentery fever is absent and ther are very few of the toxic manifestations which often accompany bacillary dysentery such as headache, nausea and a mildly delirious state. There is progressive loss of weight and strength with the development of neurasthenic symptoms. The skin becomes dry and earthy. We have picture of a more or less marked secondary anaemia. When such symptoms exist, We should be on the lookout for grayish or grayish brown mucoid masses which can usually be found during an exacerbation sloughs of the gelatinous-like necrosis in the sub-mucosa usually contain amoebae. In amoebic dysentery there may be ulceration. To know the exact location of amoebic ulceration the X-ray can be utilised. Bismuth is used for several day's prior for taking the photograph and fill the sites of ulceration.
Such cases usually show a moderate leucocytosis in which the percentage of large mononuclears is that the area around the caecum becomes tender plus a leucocytosis. Therefore, one may diagnose appendicitis and operate on a normal appendix. Autopsy records however have shown that the appendix is not infrequently invaded by amoebae, but in some of these cases other than findings amoebae in the lumen of the appendix, one is unable to note any change. Cases of amoebiasis confined to caecum and ascending colon may only show symptoms of slight anemia.

Besides the more common insidious chronic type, we may have amoebic dysentery setting in quite actuely with severt griping and frequent scanty grayish green to reddish brown mucoid stools.

Such cases may show anorexia and nausea with some fever but manifestation of toxemia. One associates with a sever case of bacillary dysentery in the tropics is not present.

Very confusing cases are those in which a bacillary dysentery sets in upon an amoebic one and this possibility should always be though of when a severe bacillary dysentery does not respond to serum therapy or an amoebic one to drugs.
In amoebic dysentery gangrenous lesions may occur in amoebic dysentery but not as common as in the case of bacillary infections. Such cases will show extreme prostration and even give the clinical picture of cholera.

**Complications of Amoebic dysentery**

The liver abscess is the most important and serious complication of amoebic dysentery. This condition is treated separately.

Besides liver abscess, quite a number of cases of amoebic abscess of the brain have been reported. 26 such cases have occurred in Egypt alone.

Amoebic abscesses almost always occur in those cases which have developed liver abscess and may appear after the liver abscess has healed.

The pus of such abscesses is viscid and blood tinged, resembling as though the pus is of liver abscess. The amoebae are found in the wall of abscess.

The symptoms are those of brain tumor, meningitis not occurring. Some cases in which amoebae have been found necrotic processes of skin and muscles have also been reported.
Perforation of the large intestine is not rare. Scientist strong has noted 12 perforation in 77 autopsies. These usually occur in the region of sigmoid flexure.

Adhesions are common complications of amoebic dysentery.

**DIAGNOSIS.**

A] Clinical Diagnosis:

It is well to remember that in the clinical diagnosis, many cases of chronic tropical diarrhoeas are really due to amoebic ulceration of the intestine.

We can differentiate bacillary dysentery from amoebic dysentery. Bacillary dysentery is more sudden and acute onset of the former. It accompanies fever and other evidences of toxaemia. The pulse rate is more rapid in bacillary than in amoebic dysentery.

The number of stools in bacillary dysentery is usually greater and the amount of stools each time is less in quantity.

The stool of bacillary dysentery is of a milky whiteness from the large number of pus cells or composed of gelatinous, reddish mucous, while that of amoebic dysentery it is tinged with disintegrated blood giving it a grayish green or brown colour.
In bacillary dysentery the muco purulent mass in bacillary dysentery may be flecked or streaked with blood. The therapeutic results after the administration of antiamoebic infections are of value for diagnosis.

Ganrenous type's of dysentery are similar whether due to bacillary or amoebic infection chronic dysentery of bacillary origin is much like amoebic dysentery clinically.

In the diagnosis of chronic amoebic ulcerations Manson-Bahr and Gregg recommend the use of sigmoidoscope. In the evening the patient takes 1/2 ounce of castor oil and the next morning a soap and water enema is given followed by 15 minims of landanum. The patient is put in the lithotomy position. No anaesthetic is used. The pain in introducing the instrument is greater in chronic bacillary ulceration cases than in amoebic ones. Scrapings can be made for microscopic examinations.

By the use of the sigmoidoscope Nisbet has reported the diagnosis of a case of balantidial ulceration.
Laboratory Diagnosis.

In amoebic dysentery the mucoid mass is often brownish. The pathogenic shows active finger like processes. In acute cases however it shows contents of red cells. In the fresh specimen of the milky mucopurulent mass of bacillary dysentery one observes large number of pus cells and particularly very large phagocytic cells which greatly resemble amoebae. This when stained with Gram's stain one may find Gram negative bacilli in the cytoplasm of this cell.

These large cells which resemble amoebae are often vacuolated. Thus the similarity is intensified. They are non motile and do not show the small ring nucleus which is so characteristic of the vegetative human amoebae. The nucleus of the confusing cells is also larger approximately one fourth of the size of the cell.

Bacillary dysentery stools show an absence of charcot leyden crystals which are often present with amoebic stools.

Walker recommends fixation of thin moist smears in Glemsa's sublimate alcohol for 10 to 15 minutes for bringing out the nuclear characteristics of human amoebae. For the absolute alcohol 1 part sat. Aq. soln.
Bichloride 2 parts should be taken. These smears are then well washed with water and stained with alum haematoxylin for five minutes. The nuclear characteristics are noted under etiology. In such staining the preparations are made on cover slips. This should never be allowed to become dry.

An excellent iron haematoxylin methods that of Rosenbusch.

Rapidly smear out with toothpick a small particle of faeces or other material containing protozoa. While this still moist, fix by Glema's method and after getting rid of the mercury with iodine solution followed by 95% alcohol, treat the smear with a 3.5% solution of iron alum in distilled water for one half hour or over night. Then wash thoroughly in distilled water.

Then stain from five to twenty minutes in the following:

1] 1% solution of haematoxylin in 95% alcohol. It takes at least ten days to ripen.

2] A saturated solution of lithium carbonate, add to 10 cc of the haematoxylin 5 to 6 drops of the lithium carbonate one next wash well and digest with about 1% solution of the iron alum. Again wash in
water, pass through alcohols to xylol and mount in balsam. With vegetative amoebae the result obtained is beautiful with vital staining. This can best be done by tinging the faeces emulsion with a 1% aqueous solution of neutral red. A good result has also been obtained by emulsifying the faeces in a drop of 1 or 2% formalin and then adding a drop of 2% acetic acid. The mixture is then tinged with either neutral red or methyl green.

One can obtain excellent results by emulsifying the faeces in Gram’s iodine solution for distinguishing the encysted form of Entamoeba coli. Owing to the glycogenic reaction given by E.coli, the round amoeba, with its 8 nuclei stand out very distinctly.

For diagnosing the 4 nucleated cyst of the pathogenic amoeba one gets better results with haematoxylin as this brings out not only the 4 nuclei but the chromidial bodies as well. It was formerly customary to recommend the administration of salts prior to examining for amoebae. Walker warns that such a procedure gives us amoebae which are difficult to differentiate the nuclear characteristics of E.coli and the tetragena nucleus of E.histolytica being much alike.
as they both contain much chromatin. The histolytica type of nucleas in a dysenteric stool, containing but little chromatin, does not resemble the nucleus of E.coli.

Walker prefers the examination of formed stools obtained without purgative.

He also notes the advantages of examining a specimen with a 2/3 inch objective as encysted amoebae are easily picked up. In opposition to the usual recommendation of text books to report only on motile amoebae he recommends the making of differential diagnosis on non motile encysted forms. This however is now generally accepted by experienced workers as true.

The preencysted E. histolytica has a nucleus. This resembles much that of E.coli. This presence of the same chromidial bodies one notes in the cysts is an aid in recognising this stage. The four nuclei of the cysts are much smaller than the nucleus of the preencysted or vegetative stage.
# Differentiating Characteristics of Pathogenic Amoebae (After Dobell and O'Connor)

## Motile Stage

<table>
<thead>
<tr>
<th>Entamoeba histolytica</th>
<th>Entamoeba coli</th>
<th>Endolimax nana</th>
<th>Iodamoeba butschlii</th>
</tr>
</thead>
<tbody>
<tr>
<td>Size</td>
<td>20 - 30 u</td>
<td>20 - 30 u</td>
<td>6 - 12 u</td>
</tr>
</tbody>
</table>
| Motility              | Very characteristic.  
    Flows in almost straight line across field. Later becomes less active pushing out a few, large blunt, blade- like pseudopodia which are perfectly hyaline being composed entirely of ectoplasm.  
|                      | Usually sluggish but may show considerable activity when freshly passed. The movement consist chiefly in changes of shape without progression.  
|                      | Slow progressive movement when freshly passed. Later slight changes of shape. Soon rounds and dies.  
|                      | Slight motility with movements similar to E. coli. Quickly degenerates and dies.  

1/1
Cytoplasm: Endoplasm finely granular and uniform in appearance. May contain red blood cells but bacteria probably never seen in normal individuals. Ectoplasm clear and well developed.

Endoplasm: had a bulky granular appearance and contains numerous food vacuoles charged with bacteria and vegetable debris. Never contains red blood cells. No sharp line between endoplasm and ectoplasm.

Endoplasm: finely granular with numerous minute food vacuoles containing bacteria. No sharp line as a rule between the endoplasm and ectoplasm.

Endoplasm: finely granular and homogeneous. Usually contains numerous food vacuoles charged with minute bacteria. Cysts of this amoeba have previously been described as iodine cysts.
<table>
<thead>
<tr>
<th>Nucleus</th>
<th>4-7 u</th>
<th>4-7 u</th>
<th>1-3 u</th>
<th>2 -3.5 u</th>
</tr>
</thead>
<tbody>
<tr>
<td>A delicate vesicle inconspicuous or invisible. Stained shows fine beads of chromatin lining wall. Karyosome small spherical and central.</td>
<td>Distinguishable stained show large beads of chromatin lining wall. Karyosome spherical eccentric and large than that E histolytica.</td>
<td>Vesicular with delicate membrane stained shows wall free from chromatin as a rule. Typically chromatin contained in large irregular eccentric karyosome characteristic.</td>
<td>Small vesicle with distinct membrane stained shows wall free from chromatin as a rule. Typically chromatin contained in large central spherical karyosome zone between nuclear wall and karyosome filled with single layer of small granules.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Entamoeba histolytica</td>
<td>Entamoeba coli</td>
<td>Endolimax nana</td>
<td>Iodamoeba butschlii</td>
</tr>
<tr>
<td>----------------</td>
<td>-----------------------</td>
<td>----------------</td>
<td>----------------</td>
<td>---------------------</td>
</tr>
<tr>
<td><strong>Size</strong></td>
<td>7-15 u</td>
<td>15 - 20 u</td>
<td>7 - 9 u</td>
<td>9 - 12 u</td>
</tr>
<tr>
<td><strong>Shape</strong></td>
<td>Round</td>
<td>Round</td>
<td>Usually oral</td>
<td>More or less rounded.</td>
</tr>
<tr>
<td><strong>Wall</strong></td>
<td>Thin</td>
<td>Thicker than E. histolytica</td>
<td>Thin</td>
<td>Relatively thick</td>
</tr>
<tr>
<td><strong>Nuclei</strong></td>
<td>Typical shows four nuclei. May show one to four. Nucleus at rest structurally similar to that of adult nucleus.</td>
<td>Typically shows eight nuclei May show one to twenty. structurally similar to adult nucleus.</td>
<td>Very small May show from one to four nuclei. Rarely eight structurally similar to adult type nucleus.</td>
<td>One relatively large nucleus Differ structurally from the adult type in that the granules in the clear zone giving an eccentric karyosome</td>
</tr>
<tr>
<td>Chromatoides</td>
<td>Large chromatoids common</td>
<td>Large chromatoids may be present but usually absent.</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>------------</td>
<td>--------------------------</td>
<td>--------------------------------------------------</td>
<td>-------</td>
<td>-------</td>
</tr>
<tr>
<td>Glycogen</td>
<td>Diffuse but not abundant</td>
<td>Relatively abundant in the early stages</td>
<td>Rarely present</td>
<td>Dense glycogen mass in characetic.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
History

The epidemics of dysentery have been noted right from ancient times. The widespread and fulminating nature of such outbreak in times of war and famine have been observed in all ages. The disease have been mentioned in the Ebers papyrus (1600 B.C.).

It was Herodotus who referred to an epidemic of dysentery amongst the Persian Army. The dysenteric syndrome is described by Hippocrates. This disease has been known in India from ancient times.
The etiology of amoebic dysentery was thoroughly investigated. The connection of bacillary dysentery with amoebae was fairly well established during the decade from 1880 to 1890. But it was not until 1898 that Shiga isolated the causative organism of bacillary dysentery. It was true that Chantamesse and Widal drew attention to a bacillus isolated from large intestines mesenteric glands and spleen of cases of tropical dysentery. But the organism was not clearly differentiated from Bacillus coli. Celli isolated an organism which coagulated milk and produced gas in glucose media. This organism which Celli called B. coli dysentericus, differs culturally from B. dysenteriae.

Geographical Distribution

Bacillary dysentery differs from the amoebic form. It tends to appear in extensive epidemics spreading over the following places.

1] Temperate religion.
2] Tropical zones, and.
3] Subtropical parts of the world.

The disease is spread with the movements of the army in any parts of the world like typhoid fever the cause is due entirely to hygienic condition rather than geographical influence.
Dysentery bacilli infection with various strains are important factors in morbidity among infants and young children in whatever parts of the world the question has been investigated. The disease is prone to prevail in lunatic asylum whether these are located in temperate or tropical parts of the world.

**ETIOLOGY**

Shiga the research scientist isolated an organism called *Bacillus dysenteriae* from dysenteric stools of 36 cases during a very total epidemic of dysentery in Japan.

Shiga found this bacillus aggluniated by the serum of the patients. Shiga reported this work in 1898.

Kruse isolated an organism from patients in an epidemic of dysentery in Germany in 1900 which corresponded to that of Shiga. Also in 1900 Flexner, Strong and Musgrave who were working in Manila not only encountered an organism similar to that of Shiga but also an organism of wider fermentative action. Dysentery has resulted from accidental laboratory infections and Strong produced dysentery in a prisoner condemned to death through ingestion of cultures.
The scientists Hiss and Rusel in 1903 isolated an organism from a total case of diarrhoea in a child to which they gave the name "Y".

Dysentery bacilli correspond culturally with the typhoid bacillus except in showing slightly weaker fermentative action on carbohydrates. The main point of difference however is their absolute nonmotility.

The characteristic of nonmotility is of greatest differentiating value and the report of slight motility are probably from misinterpretation of molecular movement as motility. The dysentery bacilli do not form those thread or whip-like filaments so characteristic of typhoid cultures and are somewhat plumper.

The dysentery bacillus is not found in the blood and hence is not eliminated in urine although there have been reported rare cases where dysentery bacilli were isolated from blood. It is found in mesenteric glands. Agglutination phenomena do not show the tenth day from the onset in dysentery patients. So for the purpose of diagnosis. This procedure is of no particular value in diagnosis. However, it is of value to identify an organism isolated from the stools at the commencement of the attack by using serum from an immunized animal or a human convalescent for the agglutination test.
From Shiga strains, it has been revealed that there seems to be only moderate agglutination power in the serum of convalescent. Flexner strains give higher agglutinations but early in convalescence the serum is not apt to have titre of more than 1-150.

A coagulation necrosis of membrane of mucous of the large intestine and occasionally of the lower part of the ileum is produced by bacilli of dysentery. Polymorphnuclears are contained in the fibrin exudate.

It was formerly thought that these lesions were of local origion. The present view is that toxins are produced which being absorbed, are eliminated by the large intestine with resulting necrosis. Flexner injected the rabbits intravenously with a toxic autolystate. This produced characteristic intestinal lesion. The toxin withstand of 7°C without being destroyed. The toxin may cause joint trouble.

There are two main types of dysentery bacilli

1) Flexner - Strong types.
2) Shiga - Kruse types.

1) Flexner - Strong types: - These producing acid in mannite media the acid strains.

2) Shiga - Kruse types: - Those not developing acid in mannite.
The Shiga type is very toxic in cultures, while the flexner type seems to be less so.

An organism resembling the Shiga bacillus but producing indol is the Schmitz bacillus. It does not appear to be pathogenic.

In immunizing horses for the production of antidysenteric serum it is customary to use both Flexner and Shiga strains so as to produce a polyvalent serum.

Four types of dysenteric bacilli recognised by Lentz for the differentiation of which he uses mannite maltose and saccharose bouillon with litmus as an indicator.

<table>
<thead>
<tr>
<th>Mannite</th>
<th>Blue</th>
<th>Red</th>
<th>Red</th>
<th>Red</th>
<th>Red</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maltose</td>
<td>Blue</td>
<td>Red</td>
<td>Blue</td>
<td>Blue</td>
<td></td>
</tr>
<tr>
<td>Succharose</td>
<td>Blue</td>
<td>Red</td>
<td>Blue</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**pidemiology**

Probably there is no disease where those attending the patient are liable to have their hands contaminated with infectious material except in the case of cholera.
The terrible frequency of the stool and the tendency of the mucilaginous mucoid mass to become smeared over the bullocks and clotting of the patient make it onerous for an attendant to carry out methods of personal protection.

The chances of spread of infection in the family in some condition are more where the mother may have to care for a ill child and have to prepare for other children or family members or herself.

Where large numbers make use of the same water-closet accommodation just like in military barracks as well as in other same type of institutions. Therefore there are chances of contamination of the seat by a patient who frequently visit the place by sheer compulsion for evacuation are most probable. The infectious material thus are transferred to others.

The disease Bacillary dysentery is peculiarly an institutional disease and tends to spread in jails, orphan asylums and the like in such institutions a carrier is a particular source of danger.

The danger is not only from a patient who is suffering from Bacillary dysentery but also from those who are convalescing or form chronic carrier, such
carriers are particular sources of danger where they take part in the preparation of food for others. It is how thought that striking prevalence of the disease is in the asylums. In these places it is difficult to make the patients to observe proper care of thier hands as well as their persons.

An outbreak of dysentery, noted by Friendmann, due to Shiga type of bacillus which was instituted by a soldier returning to the barracks form leave.

The disease captured 86 soldiers in the carriers regiment of which 49 belonged to his own squadron. The spread of the disease was traced to the latrines. The epidemic was suppressed by the enforcement of the most rigid rules of cleaniness especially by ordering the soldiers to wash there hands after leaving the latrines.

The Stools of the convalescents were examined and no man was discharged from the hospital until his record of stoools were negative for dysentery bacilli upon three successive tests carried out in fourteen days.

In fourty patients isolation of the bacilli from convalescents was obtained only for periods under fourteen days while with 27 others such carrying of bacilli lasted from two weeks to one months.
Up to certain extent the carrier of dysentery bacillus are less dangerous than the typhoid one because the dysentery bacillus does not invade the blood stream and we do not find in the urine.

There have been reports of isolation of Flexner and "γ" type bacilli from monkeys and rabbits, but there is nothing to indicate that any other host than man is of importance.

Flies are undoubtedly of as much impracticality of infection through the medium of soiled clothes, sent out for washing is to be thought of.

There has also been several instances of transference of the disease by the water supply.

Bacillary dysentery tends to become most important disease among the forces of soldiers in times of war. The military surgeons have a hectic time in encountering this disease. There were 2,85000 case of dysentery in federal army during the civil war.

It is possible that infectious material may be disseminated as dust and thus contaminate food by settling on them.
PATHOLOGY.

Injection of dysentery bacilli into the peritoneal cavity of guinea pigs causes a muco sanguinolent diarrhoea with congestion of and haemorrhage into the caecum. There is also a haemorrhagic peritoneal exudate. In the rabbit lesions similar to those in man are obtained as well as paralysis of the limbs.

So that there are two toxins concerned in the pathology of bacillary dysentery.

1) One is neurotoxin which may cause a peripheral neuritis or joint trouble.

2) Second is which acts on the lower bowel especially the caecum with the production of congestion and coagulation necrosis of the mucosa

Some cases have been reported where the adrenals showed congestion and necrosis, as if subjected to the action of a toxin.

There is an acute inflammation of the mucosa of the large intestine in man. And in the tropics, we frequently find the lower third of the ileum involved as well. The process rarely extends beyonds the ileo caecal valve in amoebic dysentery. A catarrhal process with hyperaemia and sero porulent is first noted, to be
succeeded by fibrin formation in the mucosa. This is a process of coagulation necrosis. There is no involvement of Peyer's patches when the process invades the ileum. Virchow noted the greater intensity of the process noted the greater intensity of the process in the region of the rectum, sigmoid flexure and ileo caecal valve.

As a rule however the entire large intestine is grayish red looking like lustreless red velvet. Later on we may have irregular is lands of grayish membrane formation surrounded by the red swollen congested gut. The solitary glands are usually swollen and may soften and ulcerate having the submucosa as a base.

Ulceration in bacillary dysentery is superficial rather deep as with amoebic dysentery. The ulcers of bacillary dysentery involves the free folds of the intestine and extend transversely while amoebic ulcers run longitudinally. The intervening mucosa is unaffected in amoebic ulceration while in bacillary ones it is inflamed.

Marked congestion of the blood vessels of the mucosa and submucosa with dilated lymph space fully of polymorphonuclear cells we can note it by microscope.
We find an outstanding of pus cells of in the mucosa. These are entangled along with the glandular structures of the mucosa, in a fibrinous exudate which causes necrosis of the mucosa.

According of Rogers in chronic bacillary dysentery the lesions are limited to the lower portion of the large gut and rarely extend above the descending colon.

One finds serpiginous ulceration separated by islands of mucosa in this region well more and savage have noted autopsy findings of what was practically a large granulating surface over the whole large intestine in case which had separately recovered with the exception of a prolonged convalenscence.

**SYMPTOMATOLOGY**

Usually Bacillary dysentery runs an acute course. Rarely there is as relapse but occasionally goes on to a chronic condition. Incubation period of bacillary dysentery is usually from two to seven days. Although accidental infection with bacilli in the laboratory has given as incubation period approximating twenty four hours. Incubation periods longer than a week can probaly
be explained as for cholera such cases being in those who are healthy carriers but by reasons of some gastrointestinal upset the quiescent bacilli take on pathogenic activity.

Particularly when the infecting organism is an a flexner type and in temperate climate, the case may appear as a watery diarrhea associated with colicky pains and anorexia. The stools soon become more scanty in amount frequent in number and associated with straining. This is followed by mucous with staining. This is followed by mucous stools more or less tinged with blood. The temperature is normal or but slightly elevates and the patient does not seem ill.

In the tropics and in temperate climates where the shiga type bacillus is infecting organism the onset is usually rather sudden with malaise abdominal pain and a diarrhea which only temporarily relives such pain.

This initial condition of diarrhea is soon followed by the characteristic dysentery stool and the pain. The pains then tend to centre about the umbilicus and to become continuous. There is mostly loss of appetite and slight nausea and the patient may at times show a very slight tendency of flightiness. The mind
however is usually clear, fever of moderate degree is common and it may be quite marked, say up to $104^0\text{F.}$ Ingestion of food or drink or any movement of the body bring a desire for defecation.

In mild cases the number of stools are about 15 to 30. This may become excessive and even go to about 100 in twenty four hours and the tenesmus torturing. So that excoriations around the anus and at times prolapse of the bowel intensity the distressing clinical picture. In acute cases the stool may be almost pure blood with only an admixture of mucus.

Vesical tenesmus may be present and the urine may be diminished in amount.

The pulse tends become accelerated and weak due to there is a toxic effect on the heart. Bacillary dysentery may show a moderate leucocytosis with increased polymorphonuclear percentage instead of a large mononuclear one as with amoebic dysentery.

At times however the lymphocytes may be the leucocytes showing the greatest relative increase.
**Collapse Types:**

We may have an abrupt onset with rigors, vomiting and high fever, in most severe types of dysentery. This fever gives way to a subnormal temperature and the patients shows signs of collapse and such a case may die without having passed dysenteric stools. On palpation the abdomen is rigid and very tender.

**Entero dysentery:**

In cases where the process extend to the lower the portion of the small intestine the general symptoms are much more severe although the tenesmus is less and the stools less frequent and voluminous. They contain much blood and mucus mixed with feculent material. Shiga calls such type of cases entero dysentery.

In severe cases of the more typical dysentery or colodysentery, as designated by shiga, the stools may change from the mucopurulent mass to a serous discharge. This is very rich in albumin and of an albuminous odor. In such cases emaciations of the patient is very rapid. Such types of cases may show signs of collapse with cold clammy skin and the clinical picture one associate with
cholera. It has been suggested that such case may be due to action of dysentery toxins on the adrenal.

This serous fluid may contain the flesh like particles which the French term as like gut scrapings. During convalescence there may be an arthritis. This however does not impair the function of the joint.

**Complications.**

As above there may be an arthritis, in addition to the arthritis there may be neuritis. In severe cases neuritis may go on to muscular atrophy. In the arthritis the knee joint is that most frequently involved. This complication appear late in the course of the attack. Arthritis may be frequent in one epidemic and absent in another. The joint swelling usually clears up completely. Some of the reported joint involvements are undoubtedly, Serum reactions from antidysenteric serum treatment rarely in shiga infections we may have an iridocyclitis. Subnormal temperature may follow severe attacks.

Dysentery gangrenous manifestations have been common in some epidemics.
This is a very fatal type and is recognized by the passage of dark brown serous discharges containing ashy gray to black sloughs or even, tubules of gangrenous mucosa. The stool having a putrid odour. The general symptoms are pronounced. There is a dry glazed tongue and low muttering delirium with a thready pulse. It is the state of typhoid.

It is used to consider bacillary dysentery as a self limited disease running on to convalescence within ten days to two weeks.

Rogers has called attention to the importance of bearing in mind a chronic condition as well as acute one. In these chronic cases the ulceration are usually located in the descending colon sigmoid flexure or rectum and give rise to frequent stools containing blood and mucus. This causes progressive loss of strength and weight. There is marked digestive disorder and the patient becomes weak anaemic neurasthenic.

**DIAGNOSIS**

Tormina, tenesmus, frequently scanty stools of muco-purulent or muco-sanguinolent character is syndrome of dysentery. In the pressure of this syndrome one must keep in mind the various condition which may gives rise
such manifestations of dysentery and not diagnose a bacillary dysentery until one has excluded tuberculosis, cancerous and syphilitic process as well as those connected with schistosome or other helminthic infections.

**CLINICAL DIAGNOSIS**

Amoebic dysentery is differentiated clinically from bacillary dysentery by the usual absence of manifestation of toxaemia and by its inside onset and chronic course.

It is important however to remember that either bacillary or amoebic dysentery may show gangrenous manifestation and in such cases the clinical picture of the typhoid state is the same whether the process is amoebic or bacillary. Fulminant bacillary dysenteries may greatly resemble cholers in its algid stage.

Tropical liver abscess is a complication exclusively occurring in the amoebic form of dysentery while joint manifestations and evidence of multiple neuritis may be noted in some epidemics of bacillary dysentery again, the toxins of the dysentery bacilli have a tendency to damage the myocardium at present physical consider the good effects of the administration of anti-amoebic drugs as important in the diagnosis of amoebic dysentery.
It is important to remember that chronic dysentery may result from bacillary as well as amoebic infections although a chronic process is more a feature of amoebic dysentery.

The muco-purulent stool of bacillary dysentery is more of a milky whiteness and flecked or streaked with blood or a very viscous bright blood tinged mucus rather than the homogeneous grayish brown gelationus mixture of disintegrated blood and mucus of the amoebic one. The adour is apt to be foetid in amoebic stools but rather albuminous with bacillary dysentery ones.

**Laboratory Diagnosis**

The chief point is to consider whether we are dealing with an amoebic or bacillary infections. While these two kinds of dysentery may coexist it is practical to consider a case in which amoebae with long rapidly extruded finge like pseudopodia and containing red blood cells are found as one of amoebic dysentery.

In bacillary dysentery a fresh specimen of the muco purulent stool shows in addition to the pus cells numerous large phagocytic cells. These may show vacuolation and strikingly resemble amoebae. There is no motility in such cells. But under conditions of lowered
temperature of specimen or from prolonged standing and beginning disintegration the amoebae too fail to show motility. If mounted in gram's Iodine solution, these large cells show a much larger nucleus than that of amoebae and take the yellow staining of iodine more intensely. The best method, however, is to make a smear, fix it by heat and stain by Gram's method or with Loeffler's blue or dilute carbol fuchsin. These confusing cells stain easily and perfectly and in the gram specimen we note the Gram negative bacilli in the cytoplasm. Glemsa's stain, with methyl alcohol fixation or the usual Wright or Leishman technique answer equally well. On the other hand it is rather difficult to obtain satisfactorily stained amoebae in this way. It usually becomes necessary fo fix moist thin smears of the stool with some bichloride fixative, as Zenker's fluid and then carry out the staining with haematoxylin.

The stained smear. The stained smear of material from bacillary dysentery having the presence of pus cells as well as endothelial cells and it has a value to differentiation from an amoebic stool smear stool in which pus cells are rarely seen. A stool smear of the amoebic dysentery shows more the picture of granular debris.
We should must examine stool as soon after it is passed.

If there is a infection of bacillary infection by the microscopical examination we should take a small mass of the stool, wash it in sterile water and then drop it in a tube of sterile bouillon or salt solution. After emulsifying in this tube of bouillon we take up 2 to 3 loopfuls of the emulsion and deposit them on a poured plate; later smearing out with a glass rod, either by successive parallel strokes or by revolving the plate while smearing the surface with the glass rod. It is in the first two or three days of an attack of acute dysentery that we obtain the best cultural results. We note a pure culture of dysentery bacilli from proper material taken at the onset.

Manson-Bahr states that he has never recovered true dysentery bacilli from a purely faecal stool. Even faecal contamination of the mucoid mass makes it difficult to recover the organism.

Dysentery bacilli rapidly die out if the stool is acid so that it has been recommended to make the stool strongly alkaline where it has to be sent to a Laboratory from a distance.
It is believed by the scientist that litmose lactose agar give result more surely than the more restraining faeces plating media still the scientist generally use Endo's fuchsin agar because it is always at hand for typhoid or para-typhoid culturing and gives good results. The colonies of the dysentery bacillus on this medium are like those of typhoid grayish white.

In England they prefer macconkey's neutral red bile salt agar while others use the conradi Drigalski medium. We are now using the tragus medium. On all these media the colonies resemble those of typhoid and the differentiation is more easily made by examining for motility. At the same time one frequently finds lack of motility in bacilli from colonies just isolated on Endo's medium which later on in subculture show motility and are found to belong to the typhoid or paratyphoid group. For the sure determination of dysentery bacilli or for differentiating the flexner and shiga stains one should carry out agglutination tests.

From chronic cases or from convalescents is more difficult as rule and agglutination tests may be more practical. A trouble is that an agglutinating effect may be connected with a apprior infection.
From the onset of the disease, some observers have noted the appearance of agglutinin in the serum of cases of acute bacillary dysentery within three or four days. But it is usual not to obtain agglutination with the patient's serum before the tenth day. With the shiga strains agglutinating power in 1 to 50 is usually accepted as evidence of specificity but for flexner strains we generally have a higher titre so that a dilution to 1 to 150 should be required the test.

Ritchie has tested the sera of 792 normal persons and found that 30% of these individuals agglutinated shiga bacilli in 1 to 32 while with flexner strains 41% agglutinated in 1 to 64 and 30% in 1 to 128. For comparison Ritchie's results with typhoid showed that only 6% agglutinated such bacilli in 1 to 16. There is some evidence that typhoid vaccination increases the agglutinating power of the serum against dysentery organisms. These findings are remarkable as the usual advice is to consider an agglutination of 1 to 30 as fairly specific for shiga infections and 1 to 100 for Flexner ones.

Willmore and savage tried heating serum to 50°C for thirty minutes, but found that such procedure was of
no practical value with dysentery. This has therefore differed from malta fever serum where such a procedure is of value in destroying coagglutinins and thus increasing the specific action. The work of Ohno would indicate that we should trust to the acid producing effect on mannite for differentiating Flexner and shiga strains rather than on agglutination because it was found that agglutinins for an acid strain were not always more specific for such strains than for nonacid ones.

At the same time it is the rule for a Flexner type bacillus to show specificity for its serum and the shiga type for the serum of the more toxic, non-acid-fast shiga strain cases.

The statement of Willmore and Savage that the differentiation of bacillary dysentery infections is a refinement of technique seems a proper view because with a polyvalent serum for treatment one only needs to know that the case is one of bacillary dysentery for proper treatment of course with a monovalent serum. Effective only for shiga bacillus, one would have to determine whether the organism producing the dysentery was of that strain.
In fact it takes considerable time and laboratory skill to carry out reliable cultural and serological test.

Practically we can use the therapeutic polyvalent serum for agglutination and any organism recovered on the plate mode from the faeces which agglutinates in 1 to 50 or 1 to 100 may be considered, as diagnostic of bacillary as against amoebic dysentery.

Often one does not see a case of dysentery until late in the disease and then, provided the condition is serious and the diagnosis points to a bacillary infection. It would be better to inject the curative serum rather than await laboratory confirmation.