Pathogenic Effects. These vary with the nature of parasitic infections:

In protozoal infections, the lesions are greatly influenced by proliferation, multiplication and metastasis to distant organs. In *E. histolytica*, the trophic form secretes a powerful histolytic toxin, causing destruction of the tissues. In plasmodia the parasite, while undergoing erythrocytic schizogony, causes destruction of erythrocytes (R.B.C.).

In the majority of helminthic infections, the adult parasites are found inside the human body and *no multiplication occurs* except in cases of strongyloidiasis and hymenolepiasis. It is the number of invading organisms gaining entrance during primary infection and re-infection that constitutes the most important problem in the development of clinical manifestations in helminthiasis. The effects produced therefore depend upon their habitat, i.e., the sites where the parasites attack the tissues and also on the pattern of laying eggs or larvae. In certain helminthic infections, the normal secretions and excretions of the growing larvae and the products liberated from dead parasites behave like foreign proteins and give rise to various *allergic manifestations*. Allergic state of various helminthic infections may be recognised by skin tests (intradermal) with specific antigens (*vide infra*).

Other pathogenic effects

Some parasitic infections produce an immunosuppressive state, thereby allowing the pathogenic bacteria to invade the tissues which the patient is unable to resist, as in trypanosomiasis, kala-azar and malaria. Immunosuppressive states or agents may help parasitic multiplication, resulting in fulminant parasitaemia, as in falciparum malaria or may favour massive invasion of the tissue, as in strongyloidiasis or may help "opportunist infection", as in toxoplasmosis.

The parasitic infection may contribute to the development of neoplastic growth. Examples are (*i*) adenocarcinoma of the bile duct and primary liver cell carcinoma in fascioliasis and clonorchiasis, (*ii*) colonic, rectal, hepatic and vesical carcinoma in schistosomiasis and (*iii*) Burkitt's lymphoma in malarial infection.

In some helminthic infections, the migrating larvae may carry viruses and Gram negative bacteria from the intestine to the blood and tissues, as in strongyloidiasis, trichinosis and ascariasis.

The pathological changes induced by the parasite may be the result of immunological responses. The following are some examples: (a) Nephrotic syndrome and idiopathic tropical splenomegaly in malaria, (b) Autoimmune haemolytic anaemia observed in malaria and kala-azar, (c) Granuloma formation with consequent fibrosis in schistosomiasis, the result of a cell-mediated immunity, (d) Manifestations of occult filariasis.

Laboratory Diagnosis. Depending on the nature of the parasitic infections, the following materials should be collected for specific diagnosis:

1. *Blood*. In those parasitic infections, where the parasite itself, or in any stage of its development, circulates in the blood stream, examination of blood film forms one of the main procedures for specific diagnosis. Examples are:

In malaria, the parasites are found inside the erythrocytes (R.B.C.).

In kala-azar, *L. donovani* are found inside the monocytes of blood.

In African sleeping sickness and Chagas' disease, trypomastigotes are found in the blood plasma.

In Bancroftian and Malayan filariasis, microfilariae are found in the blood plasma.

In case of leishmaniasis and trypanosomiasis blood culture and animal inoculation are helpful, but not used now as routine procedure.

2. *Stool*. Examination of the stool forms an important part in the diagnosis of intestinal parasitic infections and also for those helminthic parasites, which localise in the biliary tract and discharge their eggs into the intestine.

In protozoal infections, either trophozoites or cystic forms may be detected; the former, during the active phase and the latter, during the chronic phase. Examples are amoebiasis, giardiasis and balantidiasis.

In the case of helminthic infections, either the adult worms or their eggs are found in the stool. Examples are:

(i) Eggs are found in intestinal helminthiasis (ascariasis, hookworm infection, trichuriasis, fasciolopsiasis, intestinal schistosomiasis, taeniasis, diphyllobothriasis, hymenolepiasis and dipylidiasis) and also where the adult worms inhabit the biliary tract (fascioliasis and clonorchiasis).

(ii) In enterobiasis, eggs are *rarely found* in the stool, because they are deposited on the perianal skin and hence *anal swabs* are to be taken for the diagnosis.

(iii) In strongyloidiasis, larvae, not *eggs*, are commonly present in freshly-passed stool.

(iv) Adult worms are found in ascariasis and after a vermifuge in hookworm infection and enterobiasis. Segments of adult worms are found in taeniasis, diphyllobothriasis and other intestinal tapeworm infections.

CHAPTER II

PHYLUM SARCOMASTIGOPHORA

Subphylum: SARCODINA Superclass: RHIZOPODA
Class: LOBOSEA Subclass: GYMNAMOEBIA Order: AMOEBIDA

The protozoal parasites belonging to this group, while in motion, throw out cytoplasms called pseudopodia which represent the organs of locomotion. The genera included in the order Amoebida are:

1. Genus Entamoeba: *E. histolytica, E. coli* and *E. gingivalis*.
2. Genus Endolimax: *E. nana*.
3. Genus Iodamoeba: *I. bütschlii*.

The three genera are distinguished by the structure of the nucleus (Fig. 4) as follows:

In *Entamoeba*—The nuclear membrane is lined by chromatin granules and the compact karyosome is either centrally or eccentrically placed.

In *Endolimax*—The karyosome is a large irregular mass situated peripherally; it may be connected with another small mass.

In *Iodamoeba*—The karyosome is a large circular mass surrounded by refractile globules.

The amoeba infecting man may be classified according to their pathogenicity and habitat as follows:

A. PATHOGENIC

Intestinal Amoeba: *E. histolytica*.

B. NON-PATHOGENIC (HARMLESS COMMENSALS)

1. Mouth Amoeba: *E. gingivalis*.
2. Intestinal Amoebae: *E. coli, E. nana, I. bütschlii* and *D. fragilis*.

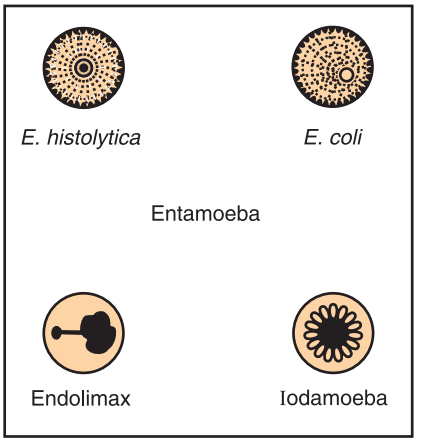

Fig. 4—Nuclear character of various genera under Amoebida.

Genus: Entamoeba

Entamoeba histolytica Schaudinn, 1903

The parasite causing diarrhoea, dysentery and liver abscess in man.

Lambl (1859) first discovered the parasite, Lösch (1875) proved its pathogenic nature, while Schaudinn (1903) differentiated pathogenic and non-pathogenic types of amoebae.

INTESTINAL LESIONS

Genesis of Intestinal Lesions. The metacystic trophozoites* liberated after excystation enter through the crypts of Lieberkühn and penetrate directly through the columnar epithelium of the mucous membrane by their amoeboid activity and by also dissolving the intestinal epithelial cells with a proteolytic ferment they secrete. They then burrow their way deeper and deeper by continuous lysis of tissue cells till they reach the submucous coat (Figs. 10 & 11). Here the amoebae rapidly multiply and increase in number, form colonies, destroy the tissues in their vicinity and utilise the cytolysed material as their food. The amoebae then begin to pass in various directions, dissolving all surrounding tissues, till ultimately, a considerable area of the submucosa is destroyed, undermining the mucous membrane above. The invasion of the tissues by this protozoal parasite brings in its train coagulative necrosis and the formation of abscess which finally breaks down, leading to the development of ulcers.

Intestinal Lesions in Acute Amoebic Dysentery

MACROSCOPIC PATHOLOGY (MORBID ANATOMY)

Distribution of Ulcers. Amoebic ulcerations are strictly confined to the large gut. The lesions may be generalised or localised.

(i) Generalised—The whole length of the large gut as far down as the internal anal sphincter is involved.

(ii) Localised—There are two levels of involvement:

(a) Ileo-caecal region—Here the caecum, ascending colon, ileo-caecal valve and appendix are involved.

(b) Sigmoido-rectal region—Here the sigmoid colon and rectum are involved.

As a rule, the preponderance of lesion in the region (a) is found to be about twice as that in the region (b).

Character of Ulcers. The sites of amoebic ulcers are not easily detected externally (from the peritoneal surface) unless they are deep and extensive. The characteristic appearance of the ulcers is best seen from the mucous surface. The ulcers are discrete and a healthy mucous membrane always intervenes between the ulcers even when they are

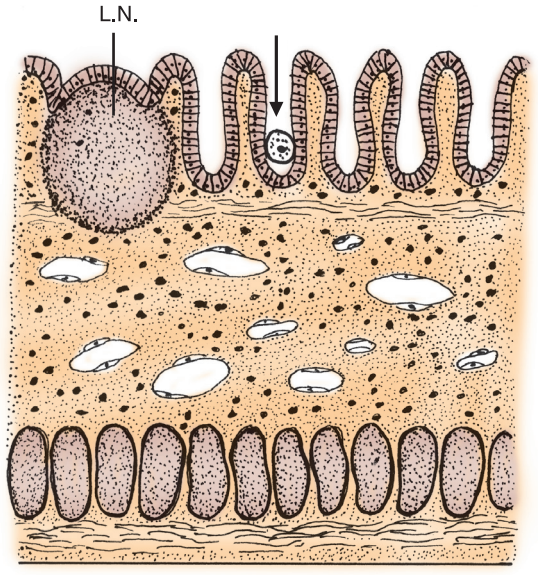

Fig. 10—Microscopic anatomy of the large intestine. 1, crypts of Lieberkühn; 2, muscularis mucosae; 3, submucosa; 4, circular muscles; 5, longitudinal muscles; 6, peritoneum. L.N., solitary lymph node.

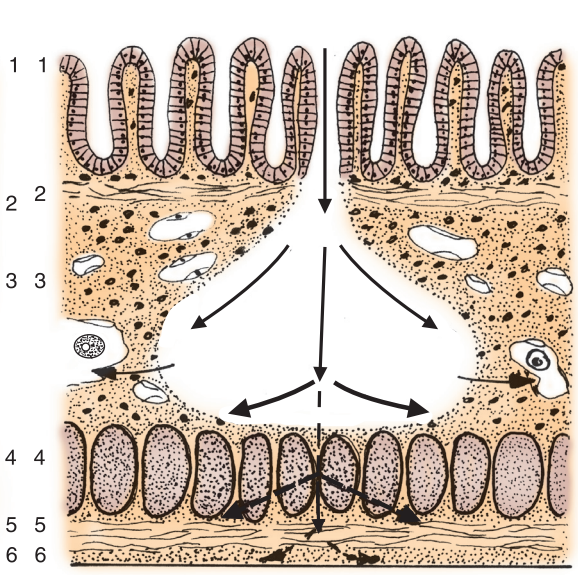

Fig. 11—Invasion of *E. histolytica* through the intestinal wall. Flask-shaped clear area represents the process of tissue necrosis. Continuous lines indicate the usual progress and dotted lines, the occasional approach.

*There are certain strains of *E. histolytica* which may live superficially in the crypts of Lieberkühn's glands of the large intestine, eroding the mucous surface and utilising the mucous secretion as food. At this stage, it metabolises anaerobically and lives in association with certain intestinal bacteria (symbiotic associates) but not feeding upon them. These strains have a low pathogenic index.

(iv) A liver abscess situated on the posterior surface may rupture into: *Inferior Vena Cava*. Such an occurrence, although rare, is invariably fatal (Fig. 24).

Other *sites* of rupture of a liver abscess which have been recorded by different observers include the common bile duct, the pelvis of the kidney and the perinephric tissues of the lumbar region.

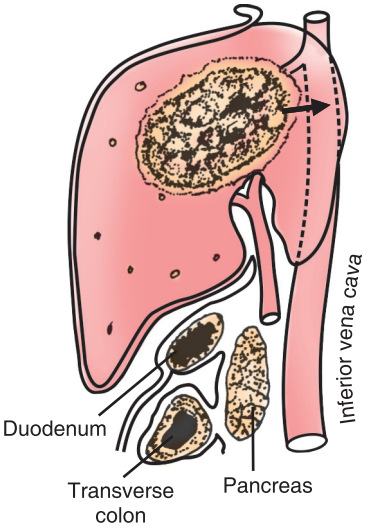

Fig. 24—A liver abscess on the posterior surface may rupture into the inferior vena cava.

METASTATIC LESIONS IN OTHER ORGANS (Fig. 25)

Pulmonary Amoebiasis. Amoebic abscess of lungs may be primary or secondary:

(i) *Primary*. It is a rare condition, occurring independently even without the presence of any hepatic abscess. In these cases, trophozoites of *E. histolytica* gain entrance from the gut-wall *via* the portal circulation into the pulmonary capillaries. Evolution of lung abscess, either single or multiple, occurs in the same way as that of a liver abscess.

(ii) *Secondary*. It arises as a complication of liver abscess by direct extension through the adhesion formed with the diaphragm, the liver and the base of the right lung. In such cases a single abscess, varying in size, may form in the lower lobe of the right lung.

Cerebral Amoebiasis. It is one of the rare varieties of metastatic amoebiasis. In the majority of cases, amoebic brain abscess arises as a complication of either hepatic or pulmonary abscess or both. The abscess is generally single and of small size and is located most commonly in one of the cerebral hemispheres.

Amoebic Pericarditis. Another rare manifestation of amoebiasis is amoebic pericarditis. Recently more cases have been reported in world's medical literature. Pericardial involvement in amoebiasis occurs invariably by direct extension from amoebic abscess of left lobe of the liver. Occasionally, the disease results from an abscess in the right lobe of the liver or lung abscess. High fever, epigastric pain, palpable mass in epigastrium or in left hypochondrium, tenderness over upper abdomen, dyspnoea and pericardial rub are the salient clinical features of the disease. X-ray of chest and upper abdomen show enlarged heart with elevated diaphragm. Ultrasound of upper abdomen and echocardiography are also helpful in making diagnosis. Blood examination reveals anaemia and polymorphonuclear leucocytosis. ECG changes are generalised ST segment elevation, inverted T-waves, or low voltage complexes.

Cutaneous Amoebiasis. Amoebic invasion of the skin is usually found over the region adjoining a visceral lesion, such as, in the areas of drainage of liver abscess or colostomy wound, in the sites of ruptured appendicular and peri-colic abscesses. Extensive necrosis and sloughing of the skin and subcutaneous tissues are caused by the trophozoites of *E. histolytica* in these areas. Besides these granulomatous ulcerations, a granulomatous mass simulating an epithelioma has been seen in the peri-anal region.

Splenic Abscess. It is found in association with hepatic abscess. Transmission of the trophozoites of *E. histolytica* occurs directly through an adherent splenic flexure of the colon.

Amoebiasis of the Penis. Amoebic infections of the penis are extremely rare and clinically mistaken for carcinoma of the penis or ulcerative venereal disease of the penis. A few cases have been reported in world's medical literature. A few cases have been documented from India also.

Enumeration of the Pathological Lesions Caused by *E. histolytica*

I. Intestinal Lesions: Involve the large gut only.
   1. *In Acute Amoebic Dysentery:* Multiple ulcers, deep and extensive. Complications which may arise in the course of the disease are: peri-caecal and peri-colic abscess, amoebic appendicitis, perforation and generalised peritonitis, gangrene of the gut and fistulas.
   2. *In Chronic Intestinal Amoebiasis:*
      (a) Single latent ulcer in the caecum.
      (b) Multiple, small, superficial ulcers scattered throughout the large gut.
      (c) Thickened caecum and colon, with occasional stricture formation.
      (d) Amoeboma in the caecum and other parts of the large gut.
      (e) Pigmented or non-pigmented scars.