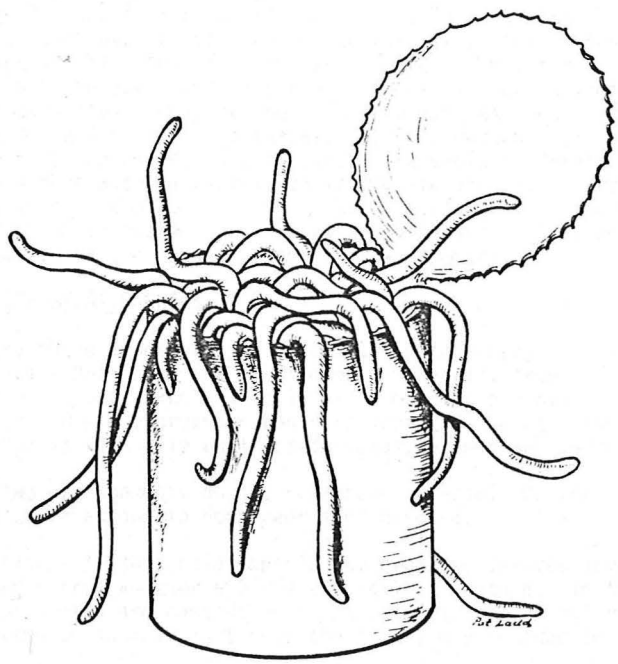
WORMS
AND OTHER PARASITES



MEDICAL GRAND ROUNDS

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Parasitic diseases constitute the most frequent infections in the world. Thirty years ago it was estimated that the "worm" burden of the world was as follows: 644 million cases of ascariasis, 456 million cases of hookworm, and 105 million cases of schistosomiasis. It is estimated that one-third of the world population has a parasitic infestation and many persons harbor more than one organism. On the pediatric service of a large Northeastern hospital approximately 400 cases of helminthic infections are seen each year.

Several factors have contributed to an increase in the variety as well as the incidence of parasitic infections in this country during the past two decades. First, the second World War, the Korean conflict and more specifically the Viet Nam War resulted in military personnel being stationed in areas with a variety of familiar and unfamiliar intestinal parasites. Many of these individuals returned to this country with their parasitic diseases. Second, modern high-speed travel has made the world a small place. Travelers to foreign lands often return home with parasites acquired while abroad. Third, increased immigration, for example, from Puerto Rico, Viet Nam and other areas, has caused us to become aware of parasitic diseases that at one time were considered rare and unimportant in this country.

HOST-PARASITE RELATIONSHIPS

There are three types of symbiotic (living together) relationships.

1) Mutualism - Both host and other organism benefit from the association, (example: termite with its cellulose-producing protozoa).
2) Commensalism - One organism benefits from this relationship, whereas the other is virtually unaffected, (example: man and *Entamoeba coli*).

These two arrangements do not represent disease. On the other hand, the third relationship does represent disease.

3) Parasitism - In this relationship one organism derives advantage or sustenance from another and gives nothing in return. In the medical sense parasites are considered to produce disease or injury to their host although in many instances the injury may be subclinical.

There are many factors that determine the effects that a parasite will have on a particular host. It is obviously advantageous for both the parasite and the host to establish some sort of equilibrium following infection so that they can tolerate each other with as little difficulty as possible. If death of the host occurs, then the parasite obviously also dies. Therefore, in many instances an attempt is made for the host and parasite to get along reasonably well together. For example, a chronic state may follow initial clinical manifestations and may persist throughout the life of the host with only an occasional exacerbation of disease. This chronic state of living together might be considered an "armed truce". Each side is always waiting to take advantage of a weakness in the other.

Whether or not acute disease occurs is dependent upon a number of factors. The size of the infecting dose is important but the most important factor is the relationship between virulence of the parasite and resistance of the host (See Figure 1.).

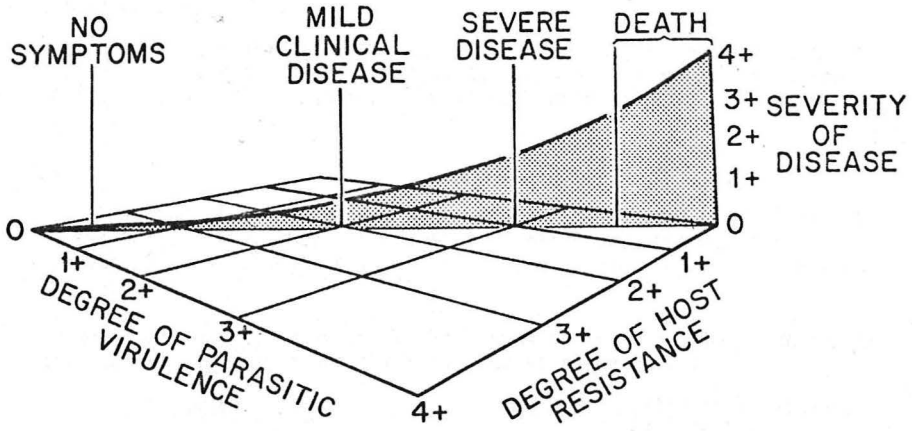


Fig. 1. Degree of disease as a function of virulence of parasite and resistance of host (from Pathology of Protozoal and Helminthic Diseases. Marcial-Rojas, R.A., ed., William & Wilkins Co., Baltimore, Md., 1971).

There are a number of factors that determine resistance in a particular host. Malnutrition, alcoholism, intercurrent disease, debilitating disease, immunosuppressive therapy and immaturity may contribute to a breakdown in resistance and thus lead to acute disease or clinical manifestations of chronic disease.

Hookworm disease is an example of the role nutrition plays in whether or not clinical manifestations occur. Hookworm infection is common in many parts of the world; however, clinically apparent hookworm disease is not. One of the factors determining whether or not a host is symptomatic or asymptomatic is diet. If the diet is sufficient in iron and protein, then hypochromic microcytic anemia and hypoproteinemia (the most common clinical manifestations of hookworm) usually do not occur.

The immune mechanisms of man against parasitic disease in most instances are poorly understood. Sometimes a type of immunity develops

which prevents acute disease although parasites may be present in the host. This "infection immunity" is called premunition (again a type of "armed truce").

Some individuals are refractory to infection by certain parasites presumably as a result of a type of immunity. For example, some people do not develop amoebiasis even though they are fed viable cysts of *Entamoeba histolytica*. Again, the overall state of health and nutrition of the host seem to be important determinants of immune resistance.

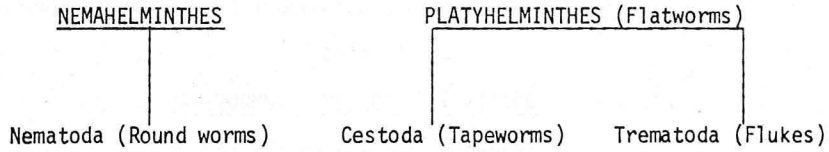
PARASITES INFECTING MAN

The parasites infecting man can be divided into three major categories: 1) the helminths (worms), 2) the protozoa and 3) the arthropods (insects). This discussion will deal only with the helminths and protozoa.

The Helminths

Worms, unlike protozoa, are macroscopic and multicellular. Like the higher vertebrates, they have digestive, excretory, reproductive and nervous systems, though they may be lacking or highly modified in some species.

Worms of medical importance. The worms of medical importance can be divided into two phyla and three classes as shown below:



Nemahelminthes (round worms). All of the round worms of medical importance occur in the class Nematoda. The intestinal round worms include all nematodes with their adult stage in the intestinal tract even though the larval stages may be located elsewhere in the body. Except for Trichinella infections, the intestinal nematode infections may be diagnosed by recovery of the egg or larval stage from the intestinal canal. Occasionally, adult worms may also be identified. With the exception of *Enterobius vermicularis* and *Trichinella spiralis*, the laboratory diagnosis can be made by routine stool analysis.

The nematodes of medical importance are listed in Table I.

Table I

Ascaris lumbricoides
Trichuris trichiura (whipworm)
Enterobius vermicularis (thread or pinworm)
Strongyloides stercoralis
Necator americanus (new world hookworm)
Ancylostoma duodenale (old world hookworm)
Trichinella spiralis

Platyhelminthes (flatworms). Unlike the round worms, the flatworms occur in two classes: the cestodes or tapeworms and the trematodes or flukes. The flatworms in contrast to the round worms have no body cavity. Nearly all are hermaphroditic and some have both asexual and sexual methods of reproduction. Man may be either a definitive host harboring the adult worm or an intermediate host harboring the larval stage of some flatworms. In some instances, as with *Taenia solium*, man can serve as both the definitive and the intermediate host.

Cestodes (tapeworms). Most of the tapeworms that are parasitic in humans reside in the intestinal tract. A few, however, invade body tissues during their larval stages, causing disease. These worms are called somatic tapeworms.

Some people have considered the adult worm as not one individual but a "cast of thousands" linked together (one behind the other) in a chain-like fashion. The chain can be divided into regions. At the proximal end is an attaching organ which is called the scolex. The scolex is round or oval in shape and contains either four suction cups or two elongated sucking grooves. The next link in the chain (following the scolex) is a neck region which is followed by "segments" or proglottides The immature proglottides are adjacent to the neck region. As they mature, the mature proglottides are pushed distally by newly formed immature proglottides. As proglottides mature,they develop male and female sex organs. Once fertilization of eggs occurs, the proglottid (containing fertilized eggs) is called a gravid proglottid. The fully developed adult tapeworm consists of a scolex and neck plus immature, mature and gravid proglottides.

The tapeworms of medical importance are listed in Table II.

Table II

TAPEWORMS OF MEDICAL IMPORTANCE

Taenia saginata (beef tapeworm)
Taenia solium (pork tapeworm)
Hymenolepis nana (dwarf tapeworm)
Hymenolepis diminuta
Diphyllobothrium latum (fish tapeworm)
Echinococcus granulosus
Echinococcus multilocularis

In addition to harboring the adult form of *T. solium* man can also serve as the intermediate host. The larval form of *T. solium* causes a condition known as cysticercosis.

The adult form of *E. granulosus* or *multilocularis* does not reside in man. Man is the host, however, for the larval forms which cause hydatid disease (See Pathologic Effects of Parasites).

Trematodes (flukes). These flatworms are very primitive, and except for the schistosomes, all are hermaphroditic. There are four major groups of flukes: intestinal, lung, liver and blood flukes.

Intestinal flukes. Man is only an accidental host for these flukes as they are usually parasitic for animals. The intestinal flukes of medical importance are listed in Table III.

Table III

Fasciolopsis buski
Metagonimus yokogawai
Heterophyes heterophyes
Gastrodiscoides hominis

Liver flukes. These flukes inhabit the biliary passages of the liver. Those of medical importance are listed in Table IV.

Table IV

Fasciola hepatica
Clonorchis sinensis
Opisthorchis felineus
Opisthorchis viverrini

Lung flukes. Most people believe that the fluke found parasitizing the lungs is of the species, *Paragonimus westermani.*

Blood flukes (schistosomes). There are three species of flukes that reside in the portal system, the mesenteric vessels or the vesicle venules of man. The three of medical importance are listed in Table V. These organisms differ from other flukes in that the sexes are separate, and grossly they resemble round worms more than other flukes.

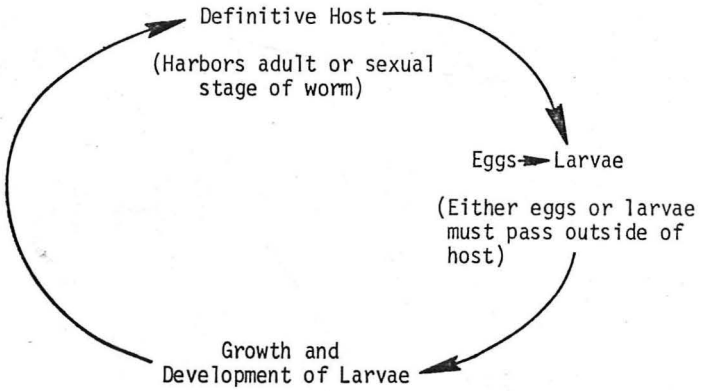
Table V

Schistosoma mansoni
Schistosoma japonicum
Schistosoma haematobium

Geographic distribution. Primarily because of their complicated life histories, many worms may be quite restricted in distribution. Hookworms, for example, require proper soil conditions (temperature, moisture, etc.) plus they must have proper contact with skin (usually soles of the feet) in order to survive and continue their life cycle. Some worms require an intermediate host in order to continue their life cycle (for example, the schistosomes which require certain species of snails).

The worms that seem to occur with greatest frequency in our area are *Ascaris lumbricoides, Necator americanus* (hookworm), *Trichuris trichiura* (whipworm), *Strongyloides stercoralis, Enterobius vermicularis* (pinworm), *Hymenolepis nana* and *Taenia saginata.* Because of the influx of Vietnamese to this country, *Taenia solium* is being seen with increased frequency.

Life cycle. Although variations exist, a basic life cycle is common to all worms as shown on the following page.



The life cycle of *Ascaris*, hookworm and *Strongyloides* is shown in Figure 2.

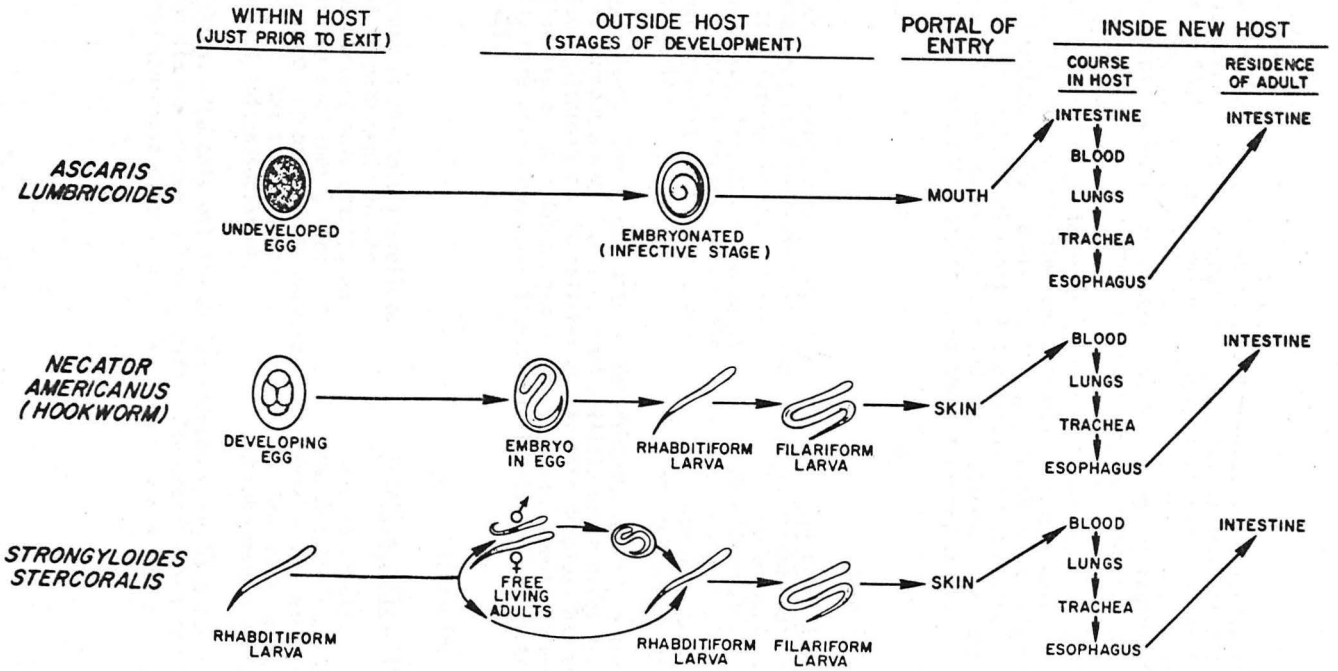
Methods of acquiring infections. The methods of acquiring infection by the more common intestinal tract parasites are listed in Table VI.

Table VI

Method of Acquiring Infection	Organism
Ingestion of ova	Nematodes - *Enterobius vermicularis* *Trichuris trichiura* *Ascaris lumbricoides*
	Cestodes - *Hymenolepis nana* *Taenia solium* *Echinococcus granulosus*
Penetration of skin by larvae	
-soil	Nematodes - *Ancylostoma duodenale* *Necator americanus* *Strongyloides stercoralis*
-water	Trematodes - *Schistosoma haematobium* *Schistosoma mansoni* *Schistosoma japonicum*
Larvae eaten in meat - pork	Cestodes - *Taenia solium*
- beef	Cestodes - *Taenia saginata*
- freshwater fish	Cestodes - *Diphyllobothrium latum*
	Trematodes - *Clonorchis sinensis*

Fig. 2

LIFE CYCLES



The Protozoa

The protozoa, in contrast to the helminths, are unicellular and microscopic. Each unicellular organism is a complete unit capable of performing the physiologic functions which in higher organisms are carried out by specialized cells. The life cycles of most protozoa are relatively simple. Most multiply asexually by binary fission during the trophozoite stage although in the sporozoa (malaria and coccidia) both asexual and sexual reproduction occurs.

Protozoa of medical importance can be divided into the following classes: amebas, flagellates, ciliates and sporozoa. Another method of categorizing these organisms is by the area of the body involved as follows: those involving the intestinal tract, those involving the mouth, vagina and urethra and those involving blood and body tissues. This discussion will involve only the protozoa affecting the intestinal tract.

Amebas. The intestinal amebas are listed in Table VII.

Table VII

	Intestinal Amebas
Entamoeba histolytica is the only ameba that with certainty is pathogenic. Some workers consider *Dientamoeba fragilis* to be pathogenic whereas others do not. The other intestinal amebas are nonpathogenic.	*Entamoeba histolytica* *Entamoeba coli* *Endolimax nana* *Iodamoeba buetschlii* *Dientamoeba fragilis* *Entamoeba polecki*

Flagellates. The flagellates differ from the amebas in that the cytoplasm is surrounded by a well defined pellicle which gives shape to the organism. Although the flagellates appear more complex than amebas and have flagella or an undulating membrane used in locomotion, they are actually more primitive. The intestinal flagellates are listed in Table VIII.

Table VIII

	Intestinal Flagellates
Giardia is the only flagellate that is pathogenic in man. Some workers have attributed diarrhea and other gastrointestinal symptoms to *Trichomonas hominis*, but there is no proof of this association.	*Giardia lamblia* *Chilomastix mesnili* *Enteromonas hominis* *Retortamonas intestinalis* *Trichomonas hominis*

Ciliates. The only ciliate of importance in man is *Balantidium coli* although its occurrence in man is rare. The organism resides in the colon and can cause disease much like *Entamoeba histolytica*.

Sporozoa. This class of protozoa is characterized by the production of spores at some time in their life. There are a number of pathologically important organisms in this class (*Toxoplasma gondii, Pneumocystis carinii* and the malaria organisms); however, the coccidia are the only ones that cause intestinal disease. There are two species of coccidia that infect man and cause coccidiosis. These two species are *Isospora belli* and *Isospora hominis*.

DIAGNOSIS OF HELMINTHS AND PROTOZOA

The diagnosis of intestinal parasites is based primarily on the finding of trophozoites, eggs, larvae, proglottides or scoleces in feces or in material aspirated from the bowel. Adult pinworms and *Ascaris* are sometimes found in feces; however, other adult round worms are not. Adult *Strongyloides* can be obtained by duodenal aspiration. The proper collection and preparation of feces or other body fluid is vitally important in the identification process.

Stool Collections

Feces should be collected in clean, dry containers. The waxed, cylindrical cardboard containers with overlapping lids are adequate. Contamination with water or urine should be avoided. Oily laxatives, antacids and barium (always collect stools prior to barium studies) may interfere with examination of the specimen and therefore should be avoided. Stools should be collected and taken to the laboratory during regular working hours.

The number of stools collected depends on the index of clinical suspicion. Most infections with helminths can be detected on one or two appropriately collected stool specimens. Finding protozoa, however, is more difficult. Therefore, several stools should be collected. At least three fecal specimens should be collected on separate days. If organisms are not found on the initial specimens and index of suspicion is extremely high, purged specimens can be obtained. Purging, however, should not be done routinely.

Stool Handling

It is best to examine the stool specimen immediately. However, if this is impossible, the stool should be divided into three portions. One portion should be refrigerated. Cysts will keep well in a refrigerated specimen if the specimen is kept moist. A second portion should be placed in polyvinyl alcohol (PVA) fixative. This is primarily to preserve trophozoites. A third portion should be placed in 5% formalin. This will preserve cysts, trophozoites and helminth eggs.

Slide Preparation

There are three types of microscopic preparations: direct wet mounts, slides prepared after concentration techniques and permanently stained slides.

Wet mounts. These preparations are made by mixing a small amount of stool with a drop of saline on a glass slide and then applying a coverslip. This type of preparation is useful in detecting trophozoites of ameba, flagellates and ciliates and also eggs and larvae of helminths. The addition of iodine stain to the wet mount preparation will make

the internal structures of ova and cysts easier to identify.

Concentration techniques. There are two types of concentration procedures that are commonly used. These techniques may be performed on fresh or formalin-fixed specimens.

1) Sedimentation technique using formalin-ether: This technique is useful for concentrating all helminth eggs except for *Hymenolepis nana*.

2) Flotation technique using zinc sulfate: This method of concentration appears less helpful. It is not as useful as the sedimentation technique for finding trematode, cestode and infertile *Ascaris* eggs.

Wet mounts of concentrated material are prepared in a similar manner to the direct wet mounts.

Permanently stained preparations. Trichrome, iron hematoxylin, or phosphotungstic-acid-hematoxylin stains may be used to prepare permanent slides of organisms.

Culture Techniques

All of the intestinal ameba and flagellates except for *Giardia* may be cultured on media. Cultures can also be performed to aid in identifying nematode larvae and also to aid in diagnosing the filariform larvae of hookworm and *Strongyloides*. Wet mounts and permanent stained slides of culture material should be made to look for organisms.

Nonfecal Specimen Sources

Specimens other than feces are sometimes obtained in an effort to diagnose parasitic diseases. A list of some of these specimens and the agents sought are listed in Table IX.

Table IX

Nonfecal Specimen Sources and Infectious Agents

Dudoenal aspirate	*Strongyloides stercoralis* *Giardia lamblia*
Anal swab or tape preparation	*Enterobius vermicularis*
Cyst fluid	*Echinococcus granulosus*
Rectal biopsy	*Schistosoma japonicum* *Schistosoma mansoni*
Urinary bladder biopsy	*Schistosoma haematobium*

Microscopic Examination

Identification of helminth eggs. Helminth eggs are round, oval or elongated, depending on the particular organism, and they range in

size from 25-150 mm or longer. Color and thickness of the egg shell is a helpful diagnostic aid. For example, *Ascaris*, *Trichuris* and *Enterobius* tend to have thick shelled eggs whereas hookworm eggs are thin shelled.

Nematode eggs. Nematode eggs are shown in Fig. 3. The eggs of *Enterobius vermicularis* are usually found in rectal tape preparations and only occasionally found in feces. The eggs of *Ascaris* can be found in stool specimens and occur in both fertile and infertile forms.

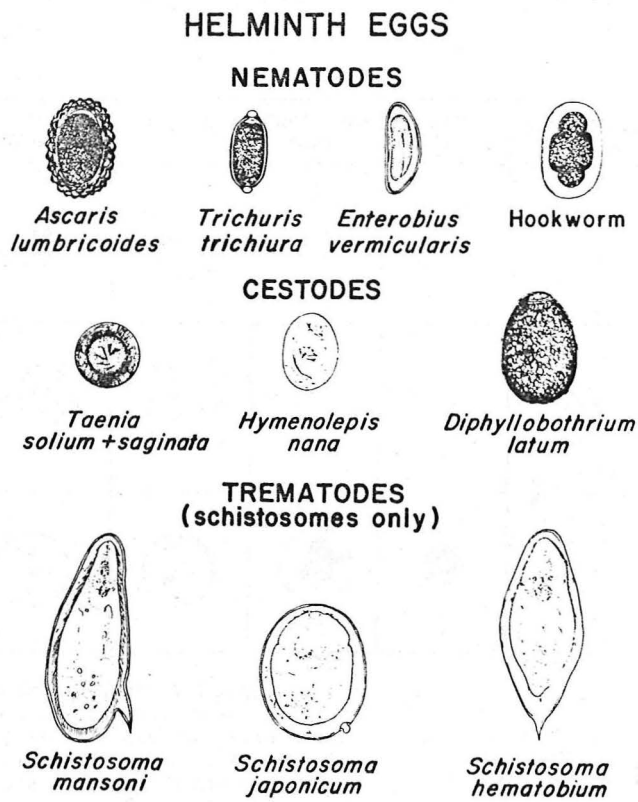


Fig. 3

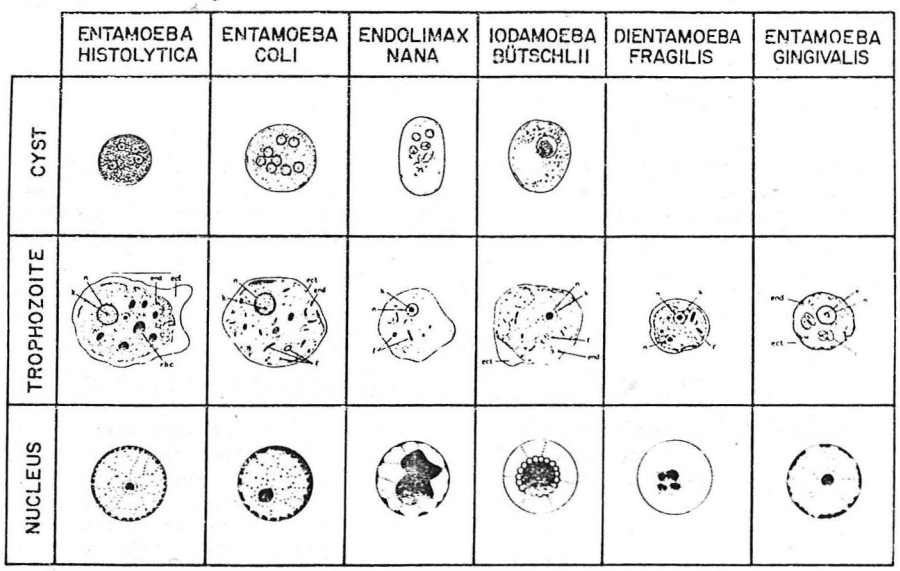
Cestode eggs, proglottides and scoleces. Tapeworm eggs are shown in Fig. 3. Eggs of *Taenia solium* and *Taenia saginata* are uncommonly found in large numbers in stool specimens. The diagnosis of these two organisms is usually made by finding gravid proglottides in stool specimens (see Fig. 7). The diagnosis of *Diphyllobothrium latum* is usually made by finding eggs in feces.

Trematode eggs. Trematode eggs are shown in Fig. 3.

Identification of protozoa. As a general rule, formed stools usually contain cysts of protozoa whereas loose, watery stools usually contain trophozoites.

Amebae. The trophozoites, cysts and nuclei of amebae are shown in Figure 4. The number and appearance of nuclei are very important in the identification of the organisms. The nucleus of *Entamoeba histolytica* has evenly distributed peripheral chromation with a small central karyosome. The nucleus is usually not visible in saline mounts. Mature cysts of *E. histolytica* have four nuclei; immature cysts frequently contain chromatoid bodies with rounded ends.

Fig. 4 AMEBAE



Flagellates. The cyst and trophozoite of *Giardia lamblia* are shown in Fig. 5. The cysts are oval or ellipsoidal and usually have 4 nuclei. The trophozoites are pear-shaped and have 2 nuclei that are not visible in unstained mounts. The organisms have a total of 8 flagella (4 lateral, 2 ventral and 2 caudal).

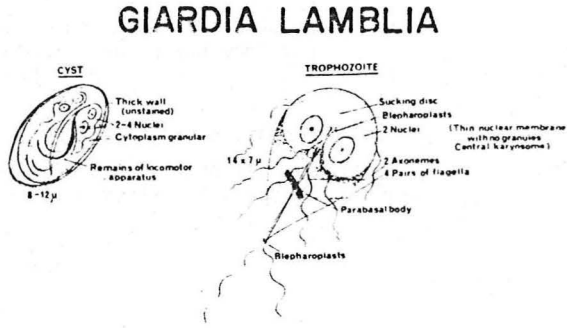


Fig. 5

Ciliates. The cyst and trophozoite of *Balantidium coli* are shown in Fig. 6. The cyst is spherical or oval with one large nucleus that is visible in unstained preparations. The trophozoite is ovoid with a tapering anterior end. The trophozoite has one large and one small nucleus. The large nucleus is occasionally seen in unstained preparations.

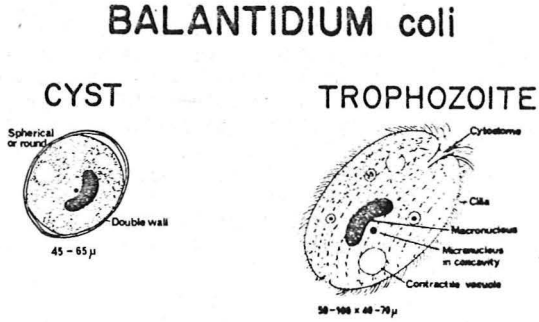


Fig. 6

Artifacts in fecal preparation. One of the major problems in parasitic identification is the differentiation of eggs, cysts and trophozoites from artifacts in the microscopic preparation. Table X lists a number of artifacts that are frequently confused with various parasitic forms.

Table X

Artifacts in Feces

Objects Confused With Helminths	Objects Confused With Protozoa
Plant material	Polymorphonuclear leukocytes
Vegetable cells	Macrophages
Meat fibers	Epithelial cells
Starch cells	Yeasts
Pollen grains	Fungus
Charcot-Leyden crystals	

PATHOLOGIC EFFECTS OF COMMON PARASITIC DISEASES

Nematodes

Strongyloides stercoralis. 51 year old man from Ada, Oklahoma
Two months prior to admission - RUQ pain, nausea and vomiting
UGI and BE x-rays - normal in Ada, Oklahoma
Treatment - cimetidine and Valium

Six weeks prior to admission - fever, chills and skin rash

Two weeks prior to admission - Pain increased in severity; vomiting worsened (occurred 5-10 min. after each meal); 20 pound weight loss

Admitted to Dallas VA Hospital

P.E.: Urticarial skin rash on upper body

WBC: 18,000 (64% eosinophils)

Duodenal aspirate: *Strongyloides stercoralis*

Skin invasion by filariform larvae (see Fig. 2) usually causes pruritus and urticaria. Pulmonary symptoms including fever, cough, dyspnea and occasionally pleural effusions may occur as the larvae migrate through the lungs. Within the lungs the larvae mature into adolescent worms which are usually swallowed and reach the small intestine. Female worms invade the intestinal mucosa (usually upper small bowel) and symptoms are usually caused by mucosal involvement. Microscopic examination of the duodenum may show flattened villi and larvae, eggs and adult worms in the mucosa. Patients may be asymptomatic (with minimal infestations) or may have abdominal pain (usually epigastric), nausea, vomiting and weight loss.

Diagnosis: The best method of diagnosis is examination of duodenal secretions for larvae and adult worms. Examination of stools can also be done, but is less rewarding.

Ascaris lumbricoides. 25 year old man presented to hospital with history of nausea and vomiting which began two days prior to admission. On the day of admission he developed severe abdominal pain.

Physical examination: Generalized abdominal tenderness with guarding

KUB: Air fluid levels in small bowel.

Exploratory laporotomy: A large mass of *Ascaris lumbricoides* was found occluding lumen of ileum.

All patients who harbor *Ascaris lumbricoides* do not present in this dramatic manner. A number of other symptoms may occur. As the larvae migrate from the intestine into the blood stream and through the lungs fever and cough may occur and sometimes a pneumonitis can be seen on x-ray. During this phase mild eosinophilia (usually 5-10%) may appear. Eggs are not present in the stool at this time because the immature worms have not completed their cycle and returned to the small intestine for the maturing process (see Fig. 2).

Symptoms are most often caused by the adult worms. Vague abdominal pain (usually epigastric) and occasionally diarrhea may occur. Most signs and symptoms are due to worms migrating into an orifice. For some reason, worms have an affinity for small orifices. For example, a worm may crawl into the common bile duct causing obstruction which in turn may cause pancreatitis or biliary tract obstruction. Worms may also enter the appendix and cause appendicitis.

Diagnosis: The usual method of diagnosis is by finding characteristic eggs or larvae in fecal specimens. Some times adult worms will be passed from the rectum or will be vomited from the stomach.

Enterobius vermicularis. 30 year old man presented with chief complaint of perianal pruritus.
Further questioning revealed that two children also had similar symptoms.
Cellophane tape preparations from anal region revealed characteristic eggs.

Pinworm is one of the most common infections in humans. In infected households viable eggs can be found on bed linens, towels, furniture, window sills, etc. It is, therefore, easy to understand why many family members are affected and why the entire household must be treated. Eggs can remain viable for up to a week in a cool moist environment but usually survive for only a day at temperatures above 25°C and in dry air. The eggs are usually transmitted by hand-to-mouth contact, inhalation and contaminated food or drink.

Systemic symptoms are rare. The most common symptom is pruritus ani, which is secondary to the nocturnal migration of the gravid females from the anus to the perianal skin where they deposit their eggs. Occasionally, scratching will lead to secondary infections.

Diagnosis: Cellophane tape impressions should be obtained early in the morning before the patient has bathed or gone to the toilet. It usually takes 3-4 examinations to make a diagnosis and seven consecutive early morning preparations should be obtained and examined before concluding that the patient does not have pinworms.

Trichuris trichiura (whipworm). 11 year old child presented with rectal prolapse.
Additional symptoms included right lower quadrant pain, diarrhea and blood-streaked stools.
The patient also had iron deficiency anemia.

Adult worms usually reside in the cecum although they can be found in the entire colon. The ingested eggs (containing infective larval stage) hatch in the jejunum where the adolescent worm develops and then moves to the cecum. There is no visceral migratory phase in whipworm disease.

Many times patients will remain asymptomatic. Vomiting, anorexia, constipation, flatulence and weight loss may occur. Because of localization in the cecum, right lower quadrant pain may occur, and whipworm disease may be confused with appendicitis. Prolapse of rectum may occur when worm infestation is severe. Eosinophilia usually does not occur.

Diagnosis: Identification of typical barrel-shaped eggs (see Fig. 3).

Necator americanus, Ancylostoma duodenale (hookworm). The two hookworms of humans cannot be differentiated in the egg or the larval stages. Therefore, the diagnosis, "hookworm disease" is usually made, and there is no attempt to differentiate between the two.

As shown in Fig. 2, the filariform larvae penetrate the skin and are carried to the lungs. Local tissue reaction with pruritus may occur at the site of larval entry. The patient usually remains asymptomatic with larval passage through the lungs. This is in contrast to the pulmonary symptoms which occur with *Ascaris* and *Strongyloides*. Once the worms reach the small intestine they attach themselves to the mucosa and mature.

Hookworms have been known to remove as much as 0.67 ml of blood per worm per day, but 0.05 ml per worm per day is usual. Heavy hookworm infection, however, can lead to the loss of about 100 ml blood per day.

It is, therefore, easy to understand how iron deficiency anemia develops. Patients may have anorexia, epigastric pain, nausea, vomiting and weight loss. Hypoalbuminemia may occur with heavy infestations, and thus, patients may develop edema. Anemia and hypoalbuminemia are especially common in malnourished patients. Eosinophilia is a common finding.

Diagnosis: The diagnosis is usually made by finding characteristic eggs in feces.

Cestodes (tapeworms)

Taenia solium. The pork tapeworm is acquired by eating raw or poorly cooked pork. The cysticercus (larval form, a single scolex enclosed in a bladder-like cyst) is ingested in the poorly cooked pork (usually muscle). Once it reaches the human small intestine the scolex evaginates and attaches to the mucosa. The larva then develops into an adult worm. The maturation time is about 3 months.

Human cysticercosis may occur. This occurs when man ingests the ovum of *Taenia solium*. In this situation man is serving as the intermediate host. Once eggs reach the intestine an oncosphere, which is an embryo form armed with six hooks, penetrates the intestinal mucosa and is carried throughout the body. Thus, a condition known as human cysticercosis develops. The symptoms produced depend upon the location and number of cysts present.

Symptoms secondary to the adult worm residing in the small intestine are usually minimal. Sometimes vague abdominal pain, anorexia and diarrhea may occur. Mild eosinophilia may be present.

Diagnosis: The ova of *Taenia solium* cannot be distinguished from *Taenia saginata* (see Fig. 3). The definitive diagnosis is made from the appearance of a gravid proglottid or from finding the characteristic scolex (see Fig. 7). Diagnosis of cysticercosis is made by excising the cysticercus and examining it under a microscope.

Taenia saginata. The beef tapeworm occurs in man as a result of eating poorly cooked or raw beef containing cysticercus larvae. Just as with *Taenia solium*, the cysticercus is released in the small intestine of man; the scolex evaginates and attaches to the intestinal wall. In contrast to *Taenia solium*, which measures 9-10 feet, adult *Taenia saginata* can grow to 25 feet in length (usually about 15 feet). Human cysticercosis (with man as the intermediate host) rarely occurs with *Taenia saginata*.

Symptoms are usually secondary to the size of the worm. Mechanical

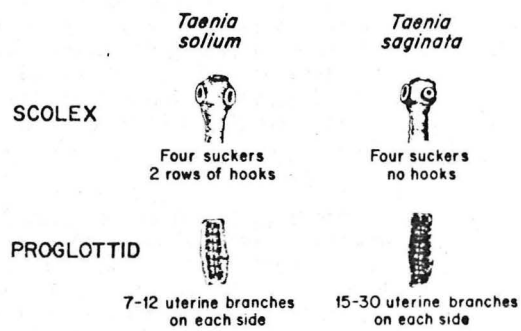


Fig. 7

obstruction of the small bowel can occur; however, most symptoms are due to the fact that the tapeworm diverts nutrients away from the host. Loss of weight may occur along with hyperphagia. Moderate eosinophilia occasionally occurs.

Diagnosis: The definitive diagnosis is made by examining the gravid proglottid (see Fig.7). If the entire worm is passed in response to therapy, examination of the scolex can also be used to make the diagnosis. Biopsy and microscopic examination are necessary to make the diagnosis of cysticercosis.

Echinococcus granulosus. Humans are accidental hosts and do not harbor the adult worms. The ovum of *Echinococcus granulosus* is ingested as a result of eating contaminated food. Once in the intestine, the liberated oncosphere penetrates the intestinal mucosa and passes through the portal system to the liver. Most of the eggs are trapped in the liver where they develop into hydatid cysts. A hollow bladder is formed which is lined by a germinal layer and a true capsule which originate from the parasite. The host reaction (fibrous-connective tissue) forms a false capsule. Hollow secondary cysts develop from the germinal layer of the cyst wall. These secondary cysts are called brood capsules. Invaginated scoleces develop from the inner wall of the brood capsules. Remains of the germinal epithelium, scoleces from the brood capsules and free-floating brood capsules form a substance called "hyatid sand". The cyst fluid contains enzymes and toxins. The original cyst within the liver may grow to as large as a grapefruit and may contain as many as 2 million scoleces. Multiple cysts may develop in the liver as a result of multiple egg infections.

As the cyst within the liver enlarges, normal liver tissue may be destroyed, and liver function tests may become abnormal. The greatest danger, however, to the host is from a ruptured cyst. If a cyst ruptures, hydatid sand leaks into a non-walled-off area, and new cysts may develop. Secondary seeding to any part of the body may occur; the most common sites are lungs, brain, abdominal cavity and bone. Leakage of hydatid sand may lead to allergic reactions and in the most severe cases to anaphylactic shock.

Diagnosis: Most cysts are diagnosed in asymptomatic patients. A calcified cyst wall in the liver is detected on x-ray. In endemic areas a cyst is often diagnosed by finding a slowly growing tumor in the liver. The indirect hemagglutination test and the bentonite flocculation test are the serologic tests of choice for diagnosis.

Protozoa

Giardia lamblia. *Giardia lamblia* occurs in two forms, cyst and trophozoite (see Fig. 5). Most patients with giardiasis are asymptomatic; however, most authorities agree that *Giardia* is a pathogen.

Once the cyst is ingested, it undergoes excystation in the duodenum, and the trophozoite attaches its sucking disk to the intestinal mucosal cells. The small intestinal mucosa may become covered with *Giardia*.

Diarrhea is the most common symptom of giardiasis although steatorrhea and abdominal pain may occur. The pathogenesis of diarrhea and/or steatorrhea is not known although there are several possibilities: 1) the organisms may cause mucosal damage and lead to malabsorption, 2) the massive number of organisms may prevent transport of nutrients across the mucosa, 3) the parasites may cause excessive mucus secretion.

Giardia is a very important cause of traveler's diarrhea. There seems to be a very high incidence of *Giardia* in travelers to the Soviet Union, especially those who travel to Leningrad. There have been several epidemics of *Giardia* reported in the U.S., and it seems that it is especially prevalent in Colorado and other Rocky Mountain areas.

There is an association between gastrointestinal immunodeficiency syndromes and giardiasis. Patients may have deficiencies of IgA, IgM and IgG or may have isolated IgA deficiency. Some of these patients also have small bowel mucosal disease. The mucosal abnormality usually is patchy in distribution and can range from a normal villous architecture to a severely abnormal mucosa which resembles the mucosal disease of celiac disease. Plasma cells may be dramatically reduced in number or may be absent. Some patients also have nodular lymphoid hyperplasia.

Diagnosis: Eosinophilia does not occur in giardiasis. UGI x-ray may show thickening of folds in the upper small bowel, but this finding is non-specific and certainly is not diagnostic. If giardiasis is suspected, stools should be obtained and examined; however, the organism may not be found in stools. The best method for finding giardia trophozoites is by microscopic examination of aspirated duodenal contents. If a duodenal biopsy has been performed, the luminal mucosal surface of the biopsy should be wiped on a glass slide. The slide should be stained with Giemsa's stain for identification of the organisms.

Balantidium coli. As with most amebas and flagellates, *Balantidium coli* has a cyst and trophozoite stage. (See Fig. 6.) Humans acquire the disease by ingesting the cysts. Once in the small intestine, excystation occurs and the trophozoites, for the most part, move to the cecum where they reside. Trophozoites multiply by binary fission. The trophozoites invade the mucosa and cause mucosal disease similar to that of *Entamoeba histolytica*. Many patients, however, with *Balantidium coli* infestation remain asymptomatic.

Diagnosis: Examination of feces should reveal trophozoites or cysts. Several fresh samples should be examined before *Balantidium coli* can be excluded. Proctoscopic appearance of rectal mucosa is very similar to that of amebiasis. Trophozoites can often be found by scraping the base of ulcers.

Ameba

(Please see Grand Rounds by Dr. John M. Dietschy, March 7, 1974 for complete details on amebiasis.)

THERAPY OF PARASITIC DISEASES

Just as with therapy of any disease, the ultimate goal of the physician should be to make the patient well. Unfortunately, many of the drugs used to treat parasitic diseases are also toxic to the host. Thus, the physician must weigh the consequences of parasitic infection against therapy. The presence of a parasite does not necessarily mean that it should be treated.

Protozoa multiply within the host, therefore, it is usually necessary to erradicate them completely. Helminths, on the other hand, usually do not multiply within the host; so that signs and symptoms can often be controlled by simply reducing the number of worms within the host. The drugs of choice used in treating the more common parasitic diseases are listed in Table XI. A complete list of drugs and side effects can be found in The Medical Letter, 20:17-24, 1978.

Table XI

Parasite	Drug	Dose
Ascaris lumbricoides	Pyrantel pamoate	11 mg/kg (max 1 gm) as a single dose
	or	
	Mebendazole	100 mg b.i.d. x 3 days
	Alternate drug: Piperazine citrate	75 mg/kg (max 3.5 gms) 1 dose daily x 2 days
Balantidium coli	Tetracycline[1]	500 mg q.i.d. x 10 days
	Alternate drug: Diiodohydroxyquin	650 mg t.i.d. x 20 days
Dietamoeba fragilis	Diiodohydroxyquin	650 mg t.i.d. x 20 days
	or	
	Tetracycline[1]	500 mg q.i.d. x 10 days
Enterobius vermicularis	Pyrantel pamoate (Antiminth)	11 mg/kg (max 1 gm) as a single dose; may have to repeat after 2 weeks
	Alternate drug: Pyrvinium pamoate (Povan)	5 mg/kg as a single dose (max 350 mg); May have to repeat after 2 weeks

Patient's family also should be treated.

Parasite	Drug	Dose
Giardia lamblia	Quinacrine HCl	100 mg t.i.d. x 5 days
	or	
	Metronidazole[1]	250 mg t.i.d. x 10 days
Hookworm (In the absence of symptoms or anemia, light hookworm infections do not need to be treated.)	Mebendazole	100 mg b.i.d. x 3 days
	or	
	Pyrantel pamoate[1]	11 mg/kg (max 1 gm) as a single dose
Strongyloides stercoralis	Thiabendazole[1]	25 mg/kg b.i.d. x 2 days. Treat for 5 days if disease is disseminated.
Echinococcus granulosus	Surgical resection of cysts is the treatment of choice.	
Hymenolepis nana	Niclosamide[2]	4 tabs (2 gms) daily (single dose) for 5 days
	Alternate drug: Paromomycin[1]	45 mg/kg daily (one dose) x 5-7 days
Taenia saginata	Niclosamide[2]	A single dose of 4 tabs (2 gms)
	Alternate drug: Paromomycin[1]	1 gm q 15 min x 4 doses
Taenia solium[3]	Niclosamide[2]	a single dose of 4 tabs (2 gms)
	Alternate drug: Paromomycin[1]	1 gm q 15 min x 4 doses
Trichuris trichiura	Mebendazole	100 mg b.i.d. x 3 days

1 Considered an investigational drug for this indication by the FDA.

2 In the U.S. this drug is available from the Parasitic Diseases Division Center for Disease Control, Atlanta, Georgia 30333. Telephone 404-633-3311.

3 Niclosamide and paromomycin disintegrate proglottid segments and release viable eggs, thus creating a theoretical risk of causing cysticercosis.

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