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REFERENCES

1. STARR, A. AND EDWARDS, M. L.: *Ann. Surg.*, 154: 726, 1961.
2. CALLAGHAN, J. C.: *Dis. Chest*, 43: 87, 1963.
3. EFFLER, D. B., GROVES, L. K. AND FAVALORO, R.: *Arch. Surg. (Chicago)*, 88: 145, 1964.
4. BLUNDELL, P. E. AND MCGOON, D. C.: The Fate of the Starr-Edwards Valve in the Aortic Area of Calves, paper presented at the 44th annual meeting of the American Association of Thoracic Surgery, Montreal, April 27-29, 1964.

# Pulmonary Manifestations of Parasitic Infestations

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**ABSTRACT**

**Parasitic infestations in man may cause transient or permanent pulmonary lesions. The lesions occur during migration and evolution of the parasites, during parasitemia, or during the final habitat. These manifestations, though infrequent in Canadian medical practice, present difficulties in diagnosis. Life cycles, mode of entry, and migration of parasites in the human body are described and illustrative case histories presented. In this series, transient pulmonary changes were associated with *Ascaris lumbricoides, Ankylostoma duodenale,* filariasis, *Giardia lamblia* and *Trichinella spiralis;* permanent lesions were produced by *Entameba histolytica* and *Tenia echinococcus.* Other parasites which may produce pulmonary changes are *Strongyloides stercoralis* and several types of Filaria (transient); Schistosoma, *Paragonimus westermani* (permanent). A case of amebiasis is presented illustrating the latent stage of infestation which lasted several decades before the organism spread from bowel to para-aortic nodes, to the lumen of the inferior cava, thence to the radicles of the portal vein and lung.**

**SOMMAIRE**

**Chez l'homme, les infestations parasitaires risquent de provoquer des lésions pulmonaires, de caractère transitoire ou permanent, qui se produisent pendant la migration et l'évolution des parasites, pendant une parasitémie ou au cours du stade de la dernière localisation. Ces manifestations, rarement rencontrées en pratique médicale au Canada, présentent des difficultés de diagnostic. L'article décrit les cycles vitaux, le mode de pénétration et la migration des parasites dans l'organisme humain et illustre cet exposé en quelques fiches cliniques. Parmi les malades du groupe étudié, les modifications pulmonaires transitoires accompagnaient des parasitoses par *Ascaris lumbricoïdes, Ankylostoma duodenale,* de la filariose, par *Giardia lamblia* et *Trichinella spiralis.* Les lésions permanentes étaient provoquées par *Entemeba histolytica* et *Tenia echinococcus.* D'autres parasites peuvent affecter les poumons, notamment *Strongyloides stercoralis* et plusieurs types de filaires (changements provisoires); Schistosoma, *Paragonimus westermani* (lésions permanentes). Les auteurs présentent un cas d'amibiase, qui illustre le stade latent de l'infestation qui a mis plusieurs décennies avant que le parasite ne passe de l'intestin aux nodules para-aortiques, jusq'à la lumière de la veine cave inférieure et de là aux racines de la veine porte et au poumon.**

PARASITIC infestations, which may present as transient or permanent pathological changes in the lungs, often pose difficult diagnostic problems. These parasitic infestations have no distinctive clinical traits and in our climate and civilization they are not frequent; therefore the search for the parasite is often delayed until exhaustive diagnostic procedures have failed to identify a more common and more familiar condition.

Parasites may reach the lungs during migration and evolution in the human body, during a parasitemia, or as a final habitat (Fig. 1). Pulmonary invasion may be accompanied by irritating cough especially at night, dyspnea, asthma, blood-stained expectoration and pain in the chest. The symptoms

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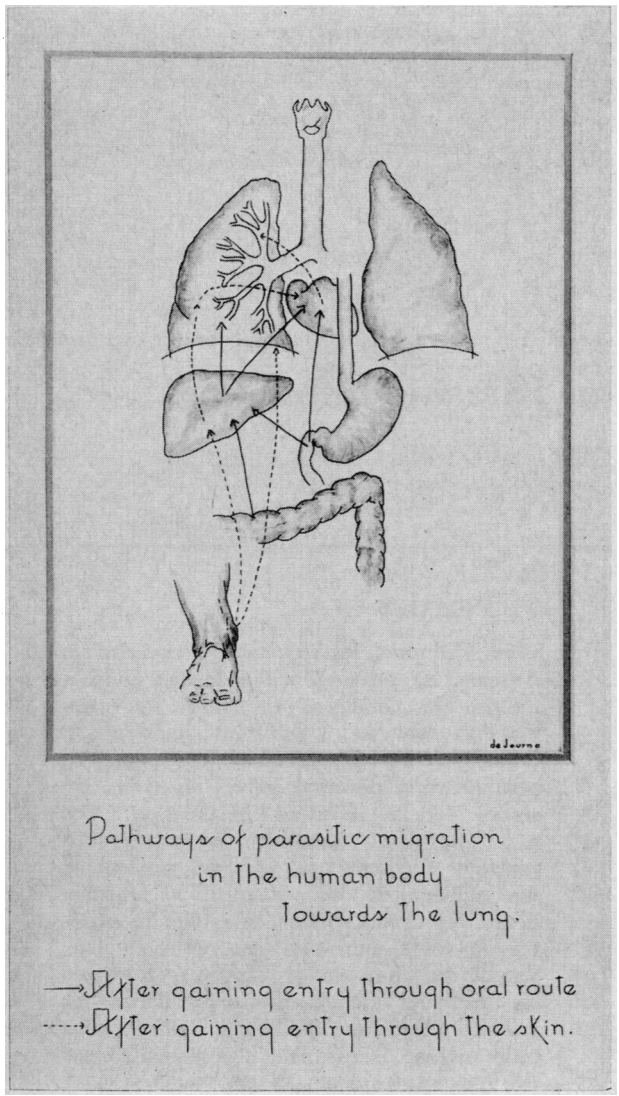


Fig. 1

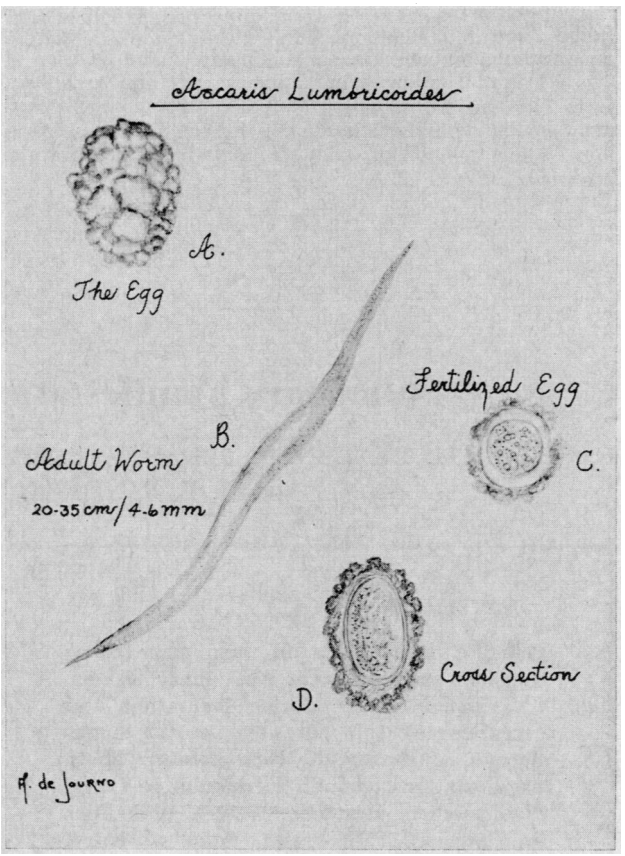


Fig. 2

may be transitory or prolonged, with or without systemic disturbances which may be serious, and sensitivity reactions such as urticaria and eosinophilia.

For a logical interpretation of an eosinophilia caused by parasites, the schema of Lavier is helpful.[1] Particularly in helminthic infestations, the eosinophil count may be considerably elevated, even exceeding 50% of the leukocyte count, in the initial stage of invasion of the parasite and during its evolution to maturity. In a later phase the eosinophil count tends to return to normal or to slightly elevated values, owing either to the host's adapting to the parasite, or the parasite's reaching full maturity and settling in its permanent habitat.

Many cases of transient pulmonary eosinophilia (Loeffler's syndrome), and probably also of tropical eosinophilia of Weingarten,[2] are caused by parasitic invasion of the human body and by passage through the lungs during the larval stage before full maturity and final habitat are reached. It is during this phase that even special diagnostic techniques may not be helpful. The clinical picture may be even more complicated if secondary infection takes place, an event quite frequent in infestations such as strongyloidiasis, amebiasis and hydatid disease.

## PARASITES CAUSING TRANSIENT MANIFESTATIONS

### *Ascariasis*

Ova of *Ascaris lumbricoides* are ingested following fecal contamination of hands and foods. The larvae develop in the duodenum or jejunum, penetrate the mucosa, enter the blood vessels or lymphatics and are carried to the lungs. From the alveolar capillaries the larvae emerge into the bronchial tree and ascend, to be swallowed and thus reach the intestinal tract, their final habitat. During pulmonary migration the larvae reach sexual maturity (Fig. 2).

The larvae may cause hemorrhagic and inflammatory reactions in the form of a pneumonitis or interstitial infiltrate with eosinophilia, that is, Loeffler's syndrome. This pulmonary migration may be accompanied by diffuse peribronchitis, with or without concomitant urticaria. The cause of pulmonary symptoms, such as irritating cough and hemoptysis, common to so many diseases, may remain obscure. Eosinophilia may be prominent. The presence of ova or of mature parasites in

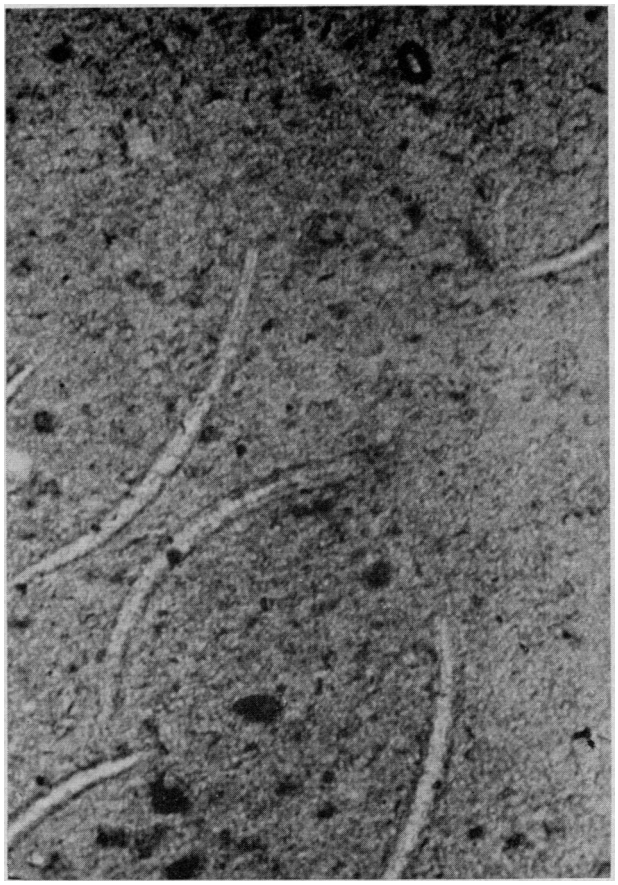

Fig. 3.—Strongyloides stercoralis, size about 15 × 200-300 microns.

the stools directs attention to the true nature of the condition. Rarely a mature worm may be eliminated by vomiting, after a paroxysm of cough, especially in children.

*Strongyloidiasis*

The filariform larvae gain entry through the skin, penetrate blood vessels and are carried to the pulmonary circulation. They escape from the lung capillaries into the alveoli and move up the bronchial tree to be swallowed. During passage through the lungs the larvae reach sexual maturity.

Focal manifestations such as intra-alveolar hemorrhages and inflammatory infiltrations with small areas of pneumonic consolidation are effects of their passing. Some larvae may invade the mucosa of the bronchioles or bronchi.

Bronchial irritation with cough and mucoid, sometimes blood-stained sputum are the clinical manifestations. At times parasites may be found in the sputum, otherwise only a striking eosinophilia is noted and at a later stage the parasites may be identified in stools (Fig 3).

*Ankylostomiasis (Ankylostoma duodenale; Necator americanus)*

This parasite migrates in the same way as *Strongyloides stercoralis* from skin, *via* the blood stream, to the lungs, up the bronchial tree, and finally down to the intestine, its final habitat. Heavy pulmonary infestation may result in a Loeffler's syndrome with irritating cough, eosinophilia with infiltrates of short duration,[3] pleural reaction and blood-stained sputum. Brumpt, observing patients artificially infested for the treatment of polycythemia, did not notice any abnormal physical signs, but he did describe a febrile episode with irritation of the trachea and pharyngolarynx, and a dry, irritating, hacking cough.[4a] The diagnostic investigations are conclusive only when ova or parasites are identified in stools, after the parasite reaches its definite habitat (Fig. 4).

*Filariasis*

*Filaria* (or *Wuchereria*) *bancrofti, malayi* or *Loa loa* gain entry through the infected insect vector, the mosquito (*Culex fatigans* and others), which injects the larvae or microfilariae through the skin. The microfilariae reach the lymphatics and blood vessels, causing a larval parasitemia. The larvae tend to localize in lymph vessels and lymph nodes, particularly those about the external genitalia and the groins. Here the microfilariae reach adulthood and sexual maturity. The fertilized females discharge microfilariae which re-enter the blood vessels, chiefly in a nocturnal cycle around midnight, to be available for infestation of the insect vector.

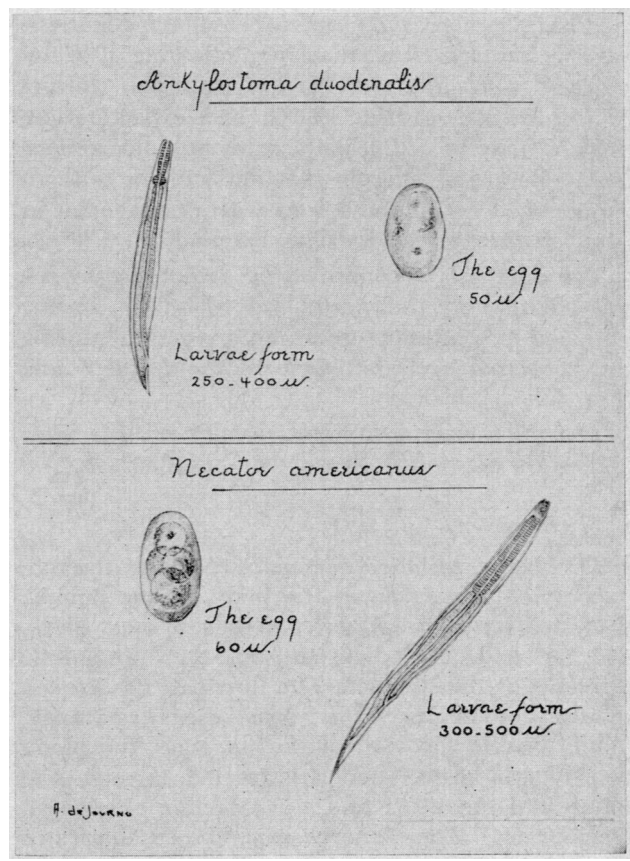


Fig. 4

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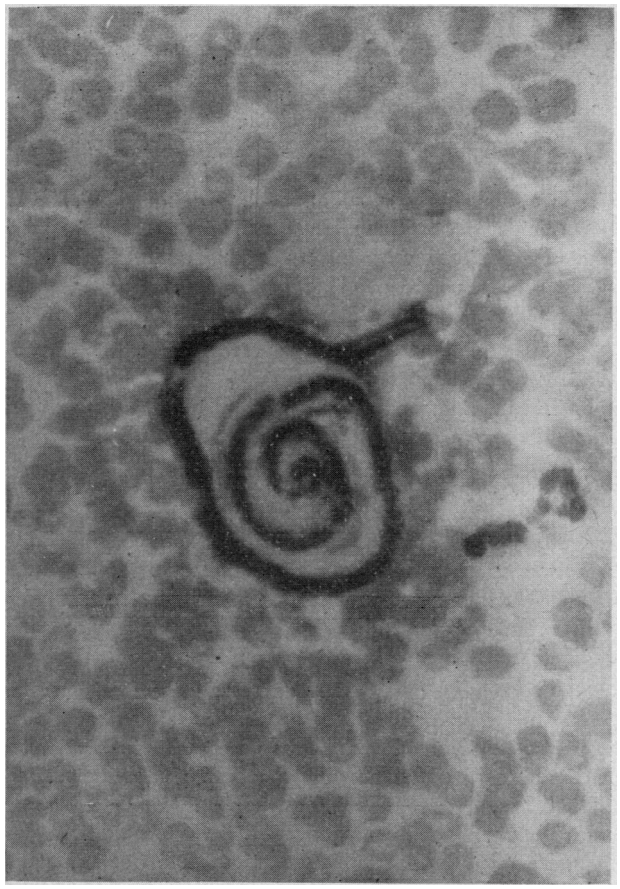

Fig. 5.—Filaria in peripheral blood.

Fig. 6.—Focal lesion of trichinosis with infiltration of lymphocytes in muscle.

The pulmonary symptoms accompanying filariasis may include the tropical eosinophilic lung of Weingarten,[2] with infiltrates of short duration shifting in localization, causing cough and expectoration; or there may be a dry, irritating cough, hoarseness, and generalized allergic reaction with or without pronounced eosinophilia, and with enlargement or even obstruction of the hilar lymphatics.

The diagnosis is confirmed by demonstrating the microfilariae in the peripheral blood, in smears obtained by venipuncture or finger puncture during the nocturnal cycle between midnight and 2 a.m. (Fig. 5).

The pulmonary symptoms usually subside after institution of specific therapy for the filariasis.

### *Trichinosis*

*Trichinella spiralis,* a nematode, passes through a complete life cycle in the body of one animal. Encysted larvae are ingested with raw meat (pork, walrus) and are digested by the proteolytic ferments of the stomach. The liberated larvae pass to the duodenum. Here some reach maturity; others invade the wall of the intestine, the blood and lymph vessels and are carried through the lungs into the heart and the systemic circulation (parasitemia). They emerge from the capillaries to invade many sites, especially striated muscles. The local lesions contain many lymphocytes and eosinophils.

With regard to chest disease, the passage through the pulmonary circulation is temporary, but larvae may invade the diaphragm, the pectoral and intercostal muscles, and serous cavities. Severe chest pain, dyspnea, irritating cough, pleural effusion and pronounced eosinophilia may result from these invasions. Biopsy of the tendinous insertion of the deltoid or gastrocnemius muscles is the best way to establish the diagnosis (Figs. 6 and 7).

### *Giardiasis*

*Giardia lamblia,* a flagellate, is confined to the lumen of the intestine (Fig. 8). But in heavy infestations symptoms of persistent bronchitis with irritating nocturnal cough occur, especially in children. As these flagellates so far have not been shown to leave the intestinal tract, the pulmonary symptoms are considered to be of allergic nature.

## PARASITES CAUSING PERMANENT MANIFESTATIONS

### *Amebiasis*

Amebiasis is caused by *Entameba histolytica,* a unicellular protozoon, pathogenic in man only. *Entameba histolytica* exists in vegetative, cystic or

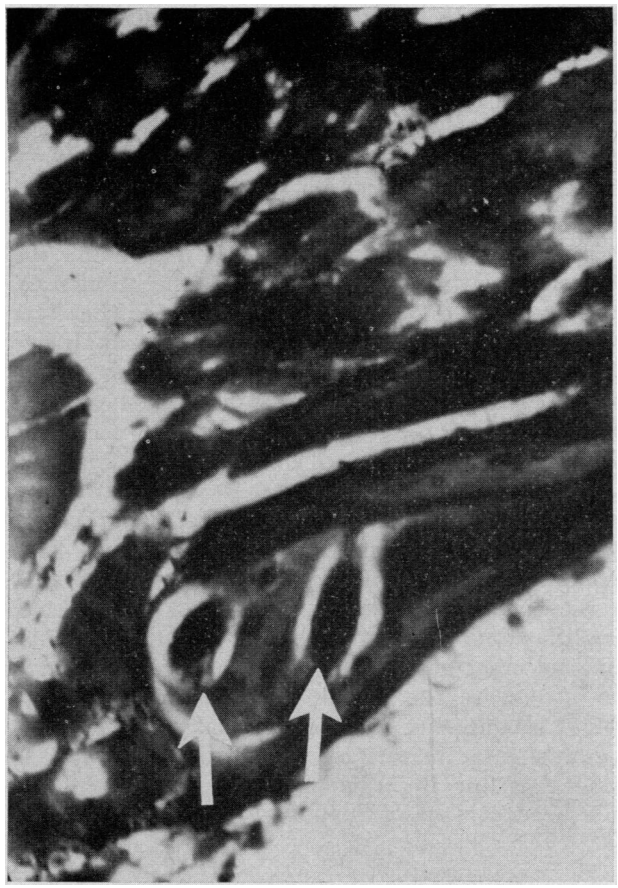

Fig. 7.—Higher power showing *Trichinella spiralis* (arrows).

intermediate forms. In favourable circumstances the cysts may lose their membrane and divide themselves into four new amebic bodies. There are transitory forms of development known as *Entameba nana* and *E. tetragena*. *Entameba histolytica* secretes proleolytic enzymes and hyaluronidases.

Amebiasis is said to be a disease of warm climates, but it is better to consider it as a disease of dirty hands and polluted water, milk, milk products, fruits and vegetables. This statement is borne out by historical and pathological data. Lambl made

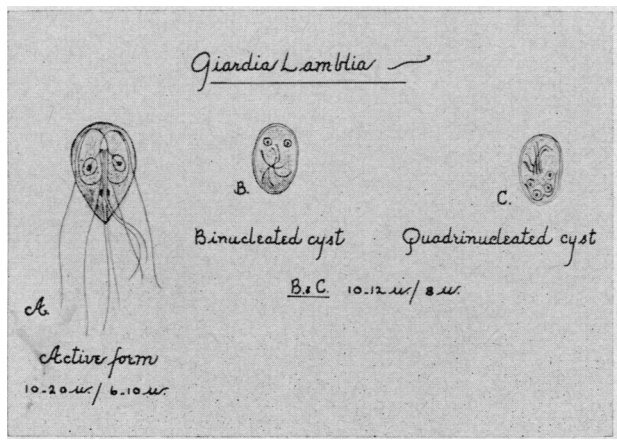


Fig. 8

the first report of amebic dysentery in 1859 from Prague, after he discovered a protozoon in the stool of a child who died from dysentery.[4b] Losch, in 1875 in Petrograd, examining the feces of a patient with dysentery, reported a mobile unicellular parasite, the ameba.[4b]

Approximately 10% of the population of the U.S.A. are said to harbour *Entameba histolytica* cysts in the intestinal tract,[5-7] but only a small percentage of those infested develop clinical manifestations. Although amebiasis is principally a disease of the colon, migration to the liver, lung, heart, bone and brain may occur. Cysts of *Entameba histolytica* may remain latent in the submucosa of the gut for many decades (34 years in our case report), and then resume virulent activity causing either a granulomatous tissue reaction or an exudative, purulent, destructive disease, granuloma or abscess or both.

The spread of the ameba from the intestine to the lungs may take place by the hematogenous route or *via* the liver, by contiguity, lymphatics or blood vessels. The pulmonary manifestations may therefore be a granuloma or an abscess of lung with or without pleural effusion, or an empyema.

Clinically there may be intense chest pain in the right base if the liver is involved; coughing and retching may take place, with evacuation of an abscess. The material eliminated may be chocolate-coloured (anchovy sauce) or greenish-yellow. Irritating cough, fever, sweating, and shortness of breath accompany the acute illness. A cavity containing air and fluid or a pneumohydrothorax, or diaphragmatic deformity are the lesions underlying many of the signs and symptoms. Detection of the amebic cysts in the sputum or in the vomitus, although a rare finding, may establish the diagnosis. The discovery of vegetative or cystic forms in stools or sigmoidoscopic smears is conclusive. Eosinophilia of varying degrees may or may not be present. The sedimentation rate is elevated, as a rule.

### *Bilharziasis*

This disease, very frequent in tropical and subtropical climates, is caused by a nematode of the genus Schistosoma. Three species, *Schistosoma mansoni, japonicum,* and *hematobium,* cause a true parasitemia in man before reaching their final sites in the liver, bowel and urinary bladder. The host is man. Eggs escape in the feces and urine; when deposited in fresh water they evolve to larvae. The larvae infest snails, to emerge as infective cercariae. The cercariae penetrate the skin of man while it is in contact with contaminated water, enter peripheral capillaries, thence to the lungs and through the alveolar capillaries into the systemic circulation. If they infiltrate the pulmonary parenchyma, they may cause grave tissue destruction with bleeding, necrosis and granulomatous

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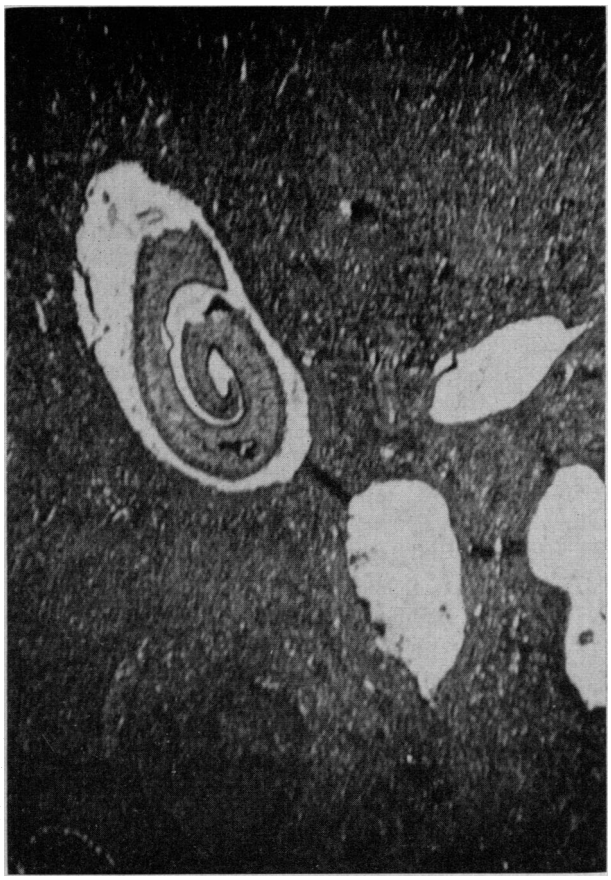

Fig. 9.—Rectal biopsy: Bilharzia in a polyp.

reaction as well as severe systemic response. The presence of ova in the feces clarifies the diagnosis. Rectal biopsy may reveal the worm with ova attached (Fig. 9).

*Pulmonary Distomiasis*

This infestation, limited to the Orient, is caused by *Paragonimus westermani,* the lung fluke. The lungs are the preferred permanent habitat. Ova are deposited here and coughed up in the sputum. If the ova reach fresh water they develop into the ciliated larvae, miracidia, and infest snails. Cercariae emerge from the snails to invade crayfish and crabs, which may be consumed by humans. The cercariae, reaching the intestine, penetrate the wall, enter the abdominal cavity and burrow through the diaphragm to enter the lungs. Presence of the ova in the sputum is diagnostic.

*Hydatid Disease*[8]

Hydatid disease is caused by the echinococcal or hydatid larvae, developed from the ova of the *Tenia echinococcus* or *Echinococcus granulosus.* The parasite, 3-6 mm. long at maturity, inhabits the intestine of the dog. It is a hermaphrodite which at its maturity eliminates eggs measuring 30 microns in the feces of the dog.

Man may become infested by ingesting the ova, as a result of intimate contact with the dog, or by eating contaminated meat of infested animals (intermediate hosts). Herbivorous animals (sheep) may ingest ova, which hatch larvae in the intestinal tract, penetrate the intestinal mucosa and are carried by lymphatics and blood vessels to the tissues of the animal (Fig. 10).

The egg, ingested by man, liberates its embryo in the alkaline medium of the intestine. These larvae penetrate the mucosa and may reach the lymphatics, the thoracic duct, or the capillaries, the vena cava, the subclavian vein or the liver through the tributaries of the portal vein. If the larvae are not retained in the capillaries of the liver lobules, they may reach the right heart and be carried into the lung. In the parenchyma of the lung the hydatid larvae grow, evolve, propagate and over a period of three to five years form hydatid cysts. The commonest site of hydatid cysts is the liver; next commonest, the lungs; and least common, muscles, spleen, kidneys, bone and brain.[9]

The echinococcal larva consists of an external, multilayered, laminated membrane, the cuticula, and an inner germinative membrane, which is rich in glycogen. The germinative membrane has an outer albuminous, white layer and an inner layer producing at maturity germinative buds (broods) which enclose 10-50 scolices (hooklets), resembling the head of *Tenia echinococcus* without segments.

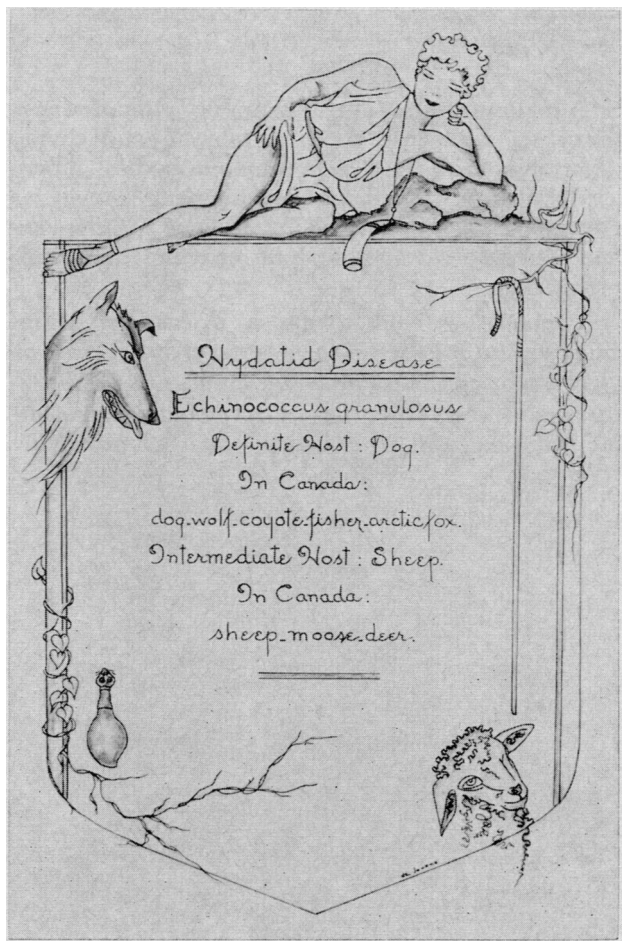


Fig. 10

The capsule includes a limpid fluid, the hydatid fluid. Scolices from the broods may separate from the germinative layer to produce a fine sandlike precipitate in the fluid.

In the pulmonary parenchyma the hydatid cyst may require three to five years to develop fully. It consists of two elements only, the germinative membrane and the hydatid fluid. At first nothing seems to separate the capsule from the pulmonary parenchyma. However, as the cyst increases in size, it compresses the surrounding lung tissue, which forms a collapsed area, deprived of air, and greyish in colour; being rich in blood vessels, the area bleeds easily. This tissue resembles an adventitious barrier. At this stage irritating paroxysms of cough may be associated with hemoptysis. Bleeding around the cyst may further wall it off with a fibrinous layer which is penetrated by new capillaries, with ensuing fibrosis or calcification. The cyst may burst and clear fluid may be coughed up. If the cyst bursts, the following events may take place: the cyst may disappear, or may form a pneumocyst, or may fill with blood forming a hemocyst, or if secondary infection supervenes, form a pyopneumocyst or simple abscess.

The signs and symptoms and the course of the disease vary with the above-mentioned events. Allergic reactions such as urticaria and bronchospasm, cough, pain in the chest, hemoptysis, coughing up of clear, watery liquid and copious purulent sputum with severe systemic reaction, represent variables in the clinical picture. The history, clinical findings, radiographs, bronchoscopic findings, eosinophilia, allergic reactions, presence of scolices in expectorated material, the complement fixation test of Weinberg-Parou and the skin test of Casoni are all important in diagnosis.

Hydatid disease in the lung can be primary or secondary (bronchogenic, hematogenic or metastatic). Thoracic hydatic disease has been noted in the following forms and locations: pulmonary cysts, hepatic cysts with migration to the thorax, pyopneumothorax, cysts of pleural seeding, cardiopericardial cysts, mediastinal cysts and osseous hydatid abscesses. Recently, the findings in 35 cases of hydatid disease from the Toronto area were reported.[10]

## CASE REPORTS CONCERNING PARASITES CAUSING TRANSIENT PULMONARY MANIFESTATIONS

### *Ascaris lumbricoides*

CASE 1.—Mrs. M.P., a 42-year-old Italian woman, had urticaria for two months, involving arms, feet and face. For two weeks she had a dry cough, without fever, the cough being troublesome at night and preventing her from sleeping. Eighteen years before, she had had a similar illness and passed a roundworm per rectum. Examination revealed scattered wheals and excoriations. Rhonchi were heard bilaterally. The chest radiograph was normal. There was an eosinophilia of 8%. Six weeks later she again passed a roundworm per rectum and the symptoms subsided.

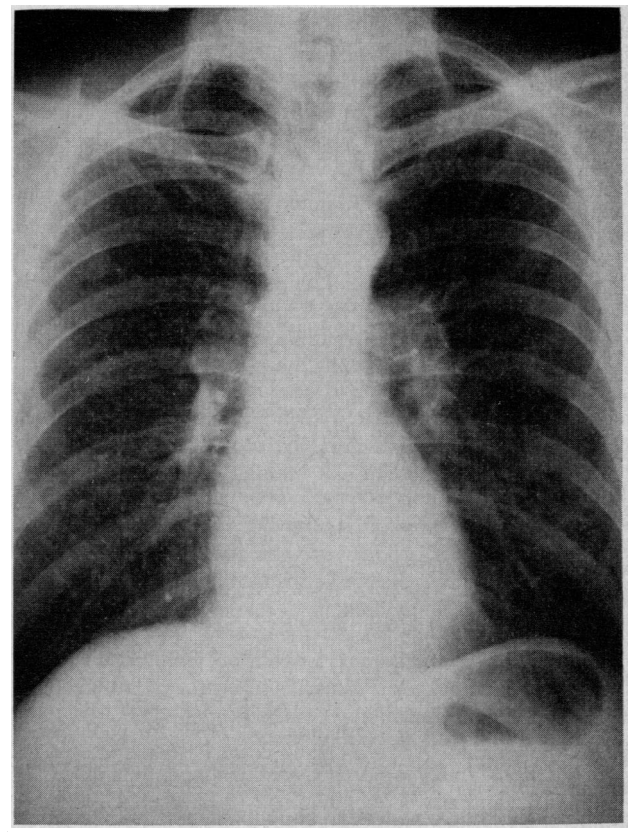

Fig. 11.—Posteroanterior film showing bilateral hilar lymphadenopathy in patient with proved filariasis (Case 3, S.S.). There is a small parenchymal nodule in the left second anterior interspace which may be unrelated.

### *Ankylostoma duodenale*

CASE 2.—G.B., a 43-year-old male immigrant, was anxious and had a variety of symptoms including abdominal pain and loss of weight. The ova of *Ankylostoma duodenale* were identified in the stool. This infestation proved difficult to eradicate. At times, when ova of hookworm were found in the stool, he had attacks of chest pain which limited full inspiration, and he felt breathless. Radiography revealed obliteration of the right costophrenic angle. Repeated tests during the period of infestation revealed an eosinophilia of 5 to 12%.

### *Filariasis*

CASE 3.—S.S., a 52-year-old engineer, was working in Pakistan during 1960. One morning, in February 1960, he awoke with fever and swelling of the scrotum. He was treated with sulfonamides and antibiotic drugs but the fever became more marked. He had pruritus, cough, hoarseness and pain in the chest. During the remainder of the year he was troubled by recurrent fever and attacks of urticaria. On admission in February 1961 there was thickening of the lateral aspect of the scrotum on the right side, and signs of fluid in the scrotum. There were scattered urticarial wheals. Blood films taken between 12 midnight and 2 a.m. revealed *Microfilaria bancrofti* (Fig. 5). A chest radiograph revealed undue prominence and roundness of the lung roots, interpreted as enlargement of lymph nodes

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(Fig. 11). He was treated with diethylcarbamazine citrate (Hetrazan) and the manifestations of filariasis subsided. Chest radiographs were unchanged one month and one year after the commencement of treatment.

*Trichinosis*

CASE 4.—F.R., a 45-year-old man, ate some uncooked, but seasoned, pork and about two weeks later had nausea, vomiting, diarrhea and malaise. Two days later he became febrile. The following day he experienced burning urination and noted a generalized follicular rash. At the same time he had generalized aches and pains most marked in the shoulders and calves. There was periorbital edema and conjunctivitis. On admission his temperature was 104° F. The white blood count was 23,000 per c.mm. and the eosinophils on repeated counts varied between 53 and 71%. On one occasion the total eosinophil count was 13,400 per c.mm. There were an abnormal number of white blood cells in the urine. Biopsies of the deltoid and gastrocnemius muscles showed trichinella organisms. The complement-fixation test for trichinosis was positive with a titre of 1: 16. Evidence of a small pleural effusion and a minimal interstitial infiltration could be seen in the chest films. He improved slowly over the next few weeks and a follow-up chest film was normal.

*Giarda lamblia*

CASE 5.—D.B., a 45-year-old man, had been ill for two months, losing energy and weight and experiencing night sweats and fever. Bowel movements increased to a frequency of two or three per day. For several weeks before admission he had a slight dry cough. On examination the liver was slightly enlarged. The chest radiograph was normal. The following laboratory results were abnormal: eosinophil count 1012 per c.mm., alkaline phosphatase 144 international units (normal range 10-48 IU), serum albumin 3.0 g., serum globulin 3.6 g., bromsulphalein retention at 45 minutes of 10%. Liver biopsy revealed mild cirrhosis. On sigmoidoscopy, shallow rectal ulcerations were noted and scrapings yielded blastocystis and Giardia organisms. A few days after beginning treatment with chloroquine and carbarsone, the fever, night sweats and cough disappeared. He recovered his energy and sense of well-being. One month after treatment the eosinophil count, bromsulphalein retention and serum protein concentrations were normal; the level of alkaline phosphatase in the serum had fallen almost to normal.

The role played by giardiasis in producing the hepatic disease is not certain. The case is included because treatment directed to eradicate the Giardia was associated with cessation of cough.

CASE REPORTS CONCERNING PARASITES CAUSING PERMANENT PULMONARY MANIFESTATIONS

*Amebic Abscess of Lung*

CASE 6.—J.B. Eight weeks before his death in 1952, this 63-year-old man was admitted to hospital because of fever for the previous four months. His temperature would become elevated during the afternoon and evening, when he would feel unwell and often would have a chill. Since 1918 he had had malaria, acquired when he was living in India and Salonika. His attacks of malaria were said to be very similar to the last, febrile illness. Examination on admission revealed flatness on percussion below the inferior angle of the right scapula. There were bilateral basal rales. A Grade II aortic systolic murmur was heard. The liver, which was firm, smooth and not tender, could be felt one fingerbreadth inferior to the costal margin. There was a large para-aortic mass palpable in the abdomen.

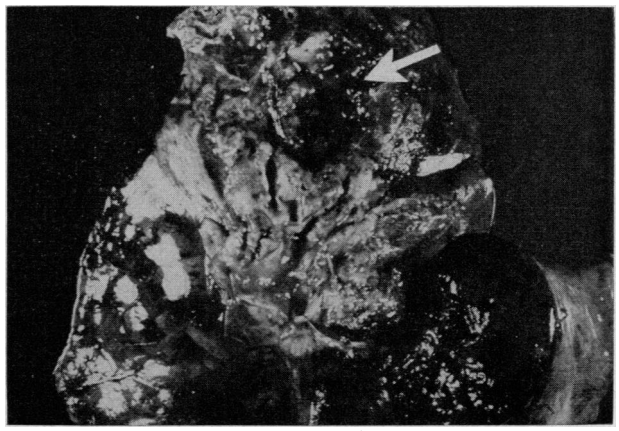

Fig. 12.—Amebic abscess in right upper lobe (arrow).

His fever failed to respond to penicillin, chlortetracycline, or quinine. Blood cultures were negative. No amebae or cysts were found in the stool. The total serum protein was 6.94 g. per 100 ml. with albumin 3.24 and globulin 3.7 g. per 100 ml. The hemoglobin was 71%, red blood cells (RBC) 3.81 million per c.mm., sedimentation rate 97 mm. in one hour. The white blood count was 10,400 per c.mm. with 79% neutrophils. The bone marrow was hyperplastic and contained 70% myeloid series with many myelocytes and bizarre forms, 3% myeloblasts and 6% premyelocytes.

The findings were considered compatible with either septicemia or lymphoma. Because no organism had been isolated and there had been no response to antibiotic treatment, the latter diagnosis was tentatively accepted.

He received a course of nitrogen mustard with temporary subsidence of fever. Urethane and, later, cortisone were administered without benefit. A chest radiograph taken a few days after admission was reported to be normal, but another taken six weeks later revealed a mass in the posterolateral portion of the right upper lobe. The mass extended from the upper pole of the right hilum into the parenchyma of the lung. Hodgkin's disease was considered to be the most likely possibility; therefore, a total of 200 r of radiation was given to the right upper lung anteriorly. He deteriorated rapidly, lapsing into a semiconscious, incontinent state. His respirations became feeble and he died.

The postmortem findings were surprising. The right upper lobe contained an abscess with a shaggy lining (Fig. 12). Thick, green, purulent material oozed from the cut surface of the abscess, which had a diameter of 6 cm. The remainder of the right upper lobe was almost entirely involved by a pyemic bronchopneumonia. There was no evidence of bronchial obstruction. The left lung was also involved in a bronchopneumonic process. The trachea and bronchi appeared acutely inflamed.

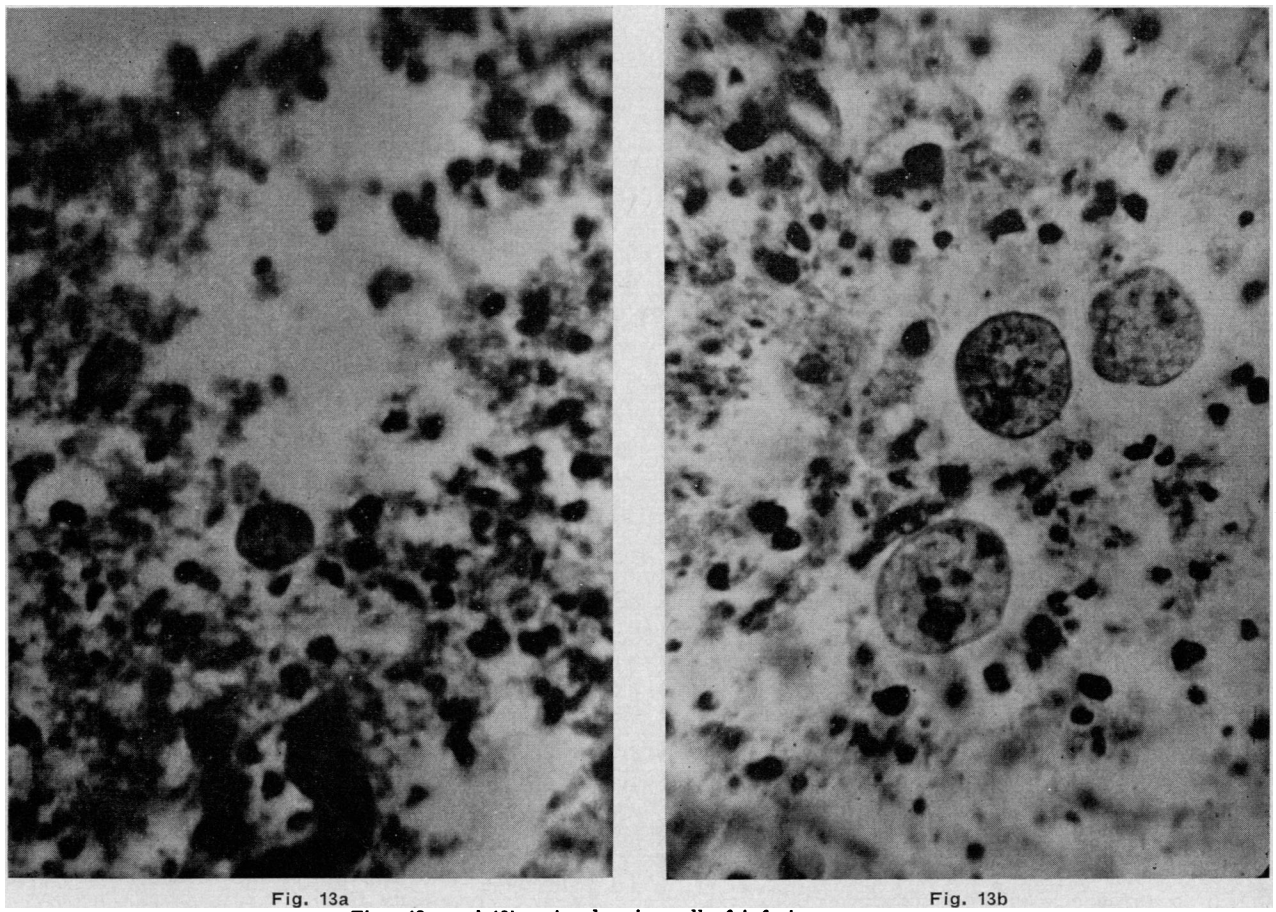

Fig. 13a Fig. 13b

**Figs. 13a and 13b.**—Amebae in wall of inferior vena cava.

The liver weighed 1830 g. and was grossly normal. The spleen was enlarged, weighing 360 g. The kidneys were enlarged and contained small, yellow cortical abscesses. There were yellowish-green streaks suggestive of acute pyelonephritis. Microscopic examination revealed evidence of diffuse subacute and chronic pyelonephritis.

In the abdomen a mass of matted retroperitoneal lymph nodes compressed the aorta and the inferior vena cava. The nodes were smooth and firm, and appeared white when sectioned. The inferior vena cava appeared to have been invaded by the same process which was involving the lymph nodes adjacent to it.

Microscopic study of the lungs, liver, inferior vena cava and adjacent retroperitoneal lymph nodes revealed that these tissues were invaded by *Entameba histolytica*. Sections of the right upper lobe presented the picture of an abscess surrounded by an acute pneumonia. Within the necrotic, fibrinopurulent exudate of the abscess were numerous *E. histolytica*. Sections through the retroperitoneal mass included portions of aorta and inferior vena cava which were encased in dense granulation tissue containing fibroblasts, capillaries and chronic inflammatory cells. No amebae were seen in the lymph nodes or in the granulation tissue surrounding the great vessels. However, the inferior vena cava was invaded by amebae (Fig. 13). Its wall was disorganized and granulation tissue replaced the adventitia and muscularis. The intima was lined by granulation tissue, the most internal portion of which was necrotic. In this necrotic exudate a small number of *E. histolytica* could be seen. The liver contained amebic abscesses (Fig. 14). Amebae were also seen in the mucosa of the colon (Fig. 15). An incidental finding was an astrocytoma in the mid-brain and pons.

*Hydatid Disease*

CASE 7.—Mrs. R.M., a 25-year-old married woman, was admitted because of pain in the right upper quadrant which radiated to the inferior angle of the right scapula and had been present for one day. There had been previous attacks of nausea and vomiting related to eating fatty foods and cabbage. Physical examination revealed splinting and tenderness of the right upper quadrant and a tender mass in the region of the gallbladder. At laparotomy cysts in the liver were seen and excised. Scolices were identified in the cysts.

Eight years later the patient was admitted again because of right pleural pain, and a right pleural effusion. There was a history of transient itchy urticaria and cyanosis. The oral temperature was 104° F. The white blood count was 17,000 per c.mm., of which 32% were eosinophils. Five hundred millilitres of pleural fluid was aspirated. The glucose content was normal. There were 63% eosinophils in the pleural fluid.

The following year she was admitted again because of discomfort in the right upper quadrant which was aggravated by breathing. When the abdomen was explored, echinococcus cysts of the liver were again

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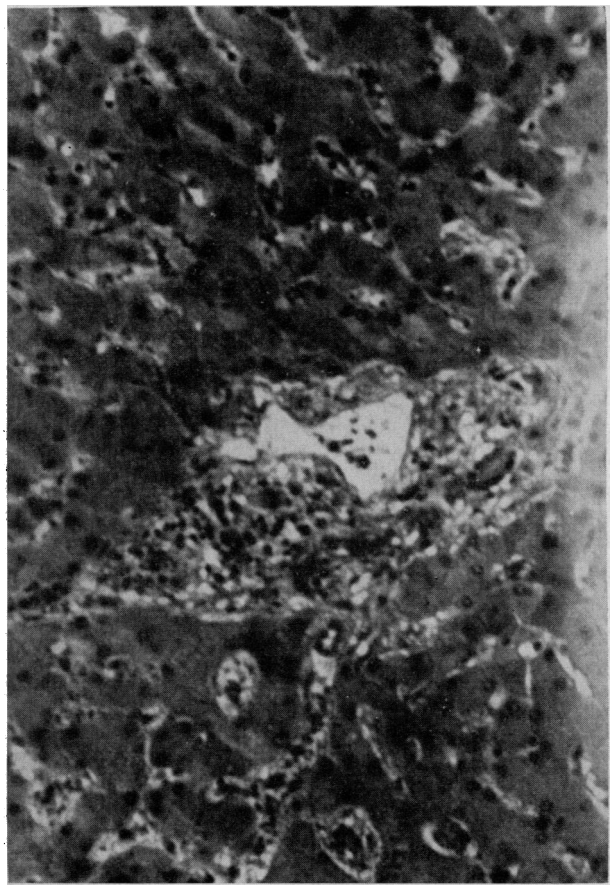

Fig. 14.—Amebic abscess in liver.

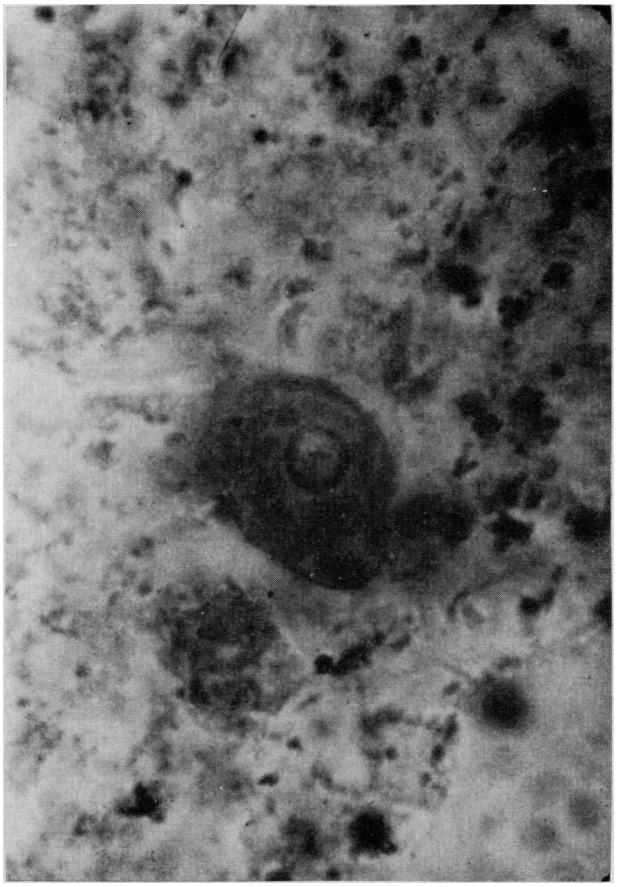

Fig. 15.—Ameba in colonic mucosa.

excised. One of the cystic lesions had invaded the diaphragm.

CASE 8.—When first admitted, A.G. was 45 years old. Born in Roumania, he was interned in a German concentration camp during World War II. After the war he lived in Sweden for three years before emigrating to Canada. While in Sweden he had a pleural effusion, the fluid being slightly hemorrhagic and containing abundant eosinophils. There were from

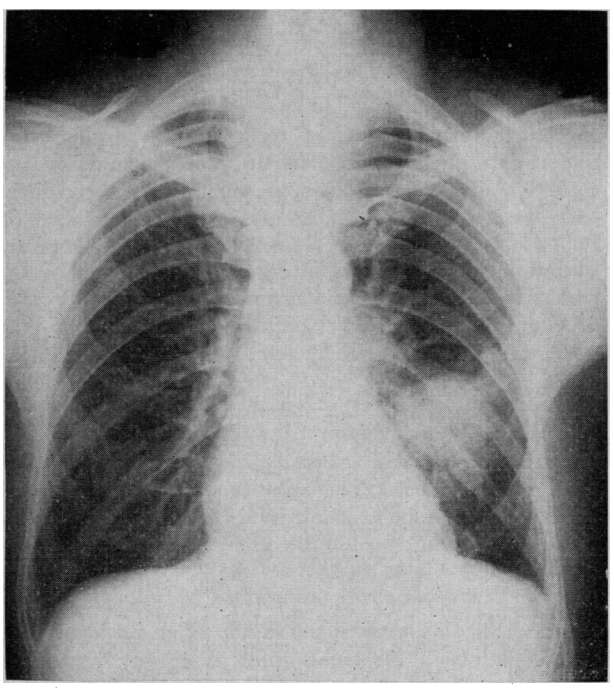

Fig. 16.—Posteroanterior film showing single large hydatid cyst (Case 8, A.G.).

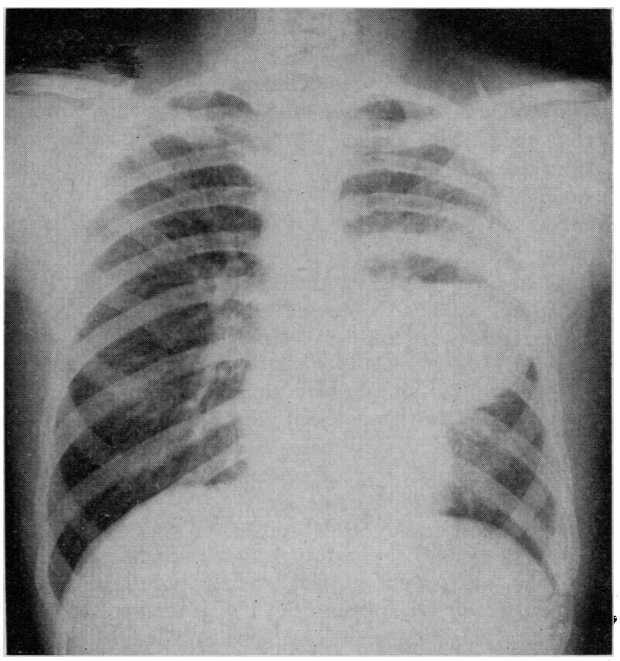

Fig. 17.—Posteroanterior film showing huge hydatid cyst in the left lower lobe with long air-fluid level (Case 9, J.G.), following intrabronchial rupture. Surgical confirmation was obtained.

26 to 40% eosinophils in the blood on repeated tests. The Casoni skin test gave a doubtful reaction. The complement-fixation test was positive, but the result might have been due to the repetition of skin tests. Radiographic examination of the chest revealed a cyst, almost the size of an orange, below the left hilus. This lesion was considered to be a hydatid cyst.

After arriving in Canada the patient again became ill. He had hemoptysis, pain in the left chest and breathlessness. Three weeks after the onset of these symptoms he was admitted to hospital. There was reduced expansion of the left lower chest and a few rales at the left base posteriorly. The tip of the spleen was palpable. The liver was of normal consistency and could be felt at the costal margin. There was an eosinophilia of 26% as well as an excess of eosinophils in the sternal marrow. The erythrocyte sedimentation rate was 100 mm./hr. The Casoni test was positive. Chest radiographs revealed a circumscribed spherical lesion in the apex of the left lower lobe (Fig 16). Lobectomy was performed and the cyst excised. Many hooklets of *Tenia echinococcus* were identified.

Six years later he was admitted again, complaining of choking, difficulty in breathing, sweating and confusion. There was an eosinophilia of 20%. The spleen was enlarged. Several cysts adjacent to the spleen were excised.

CASE 9.—At age 19 J.G. was admitted to hospital because of the sudden onset of cough, white sputum and marked breathlessness two months previously. He became weak, lost his appetite, and complained of a "heavy" pain in the left chest aggravated by deep inspiration. The sputum became yellow and stinking. He had chills during which his teeth would chatter. Three weeks after the onset of the illness a chest radiograph revealed a large abscess, with a fluid level, in the apical segment of the left lower lobe (Fig. 17). Two and one-half weeks later the abscess had resolved partially. However, after a further three weeks he spat up blood which was both bright red and dark. His breath was foul. In spite of these events he had gained 10 lb. in the preceding month.

Examination on transfer to the Toronto General Hospital revealed only a slight reduction in tactile fremitus over the left mid-lung field. The diagnosis was thought to be either bronchiectasis, lung abscess, carcinoma or tuberculosis. When it was learned that the patient, who was born in Italy, had worked on a sheep farm in that country for 10 years until he was 16, the diagnosis of hydatid disease was suggested. The Casoni test was negative and there was no eosinophilia. Bronchoscopy revealed pus coming from the apical segment of the left lower lobe, the mucosa was thick and red, and the lumen was narrow. The lobe was resected. The apical segment contained a centrally placed cyst which had a double wall and was surrounded by an area of infection (Fig. 18). Hooklets were seen on quick section of the wall of the cyst. The outer membrane of the cyst was thin and whitish-blue. The inner membrane was slightly firmer and on its surface were small, pinkish-red nodules, the largest measuring 4 mm. in diameter. In the smear of the fluid from the cyst there were numerous hooklets but no scolices.

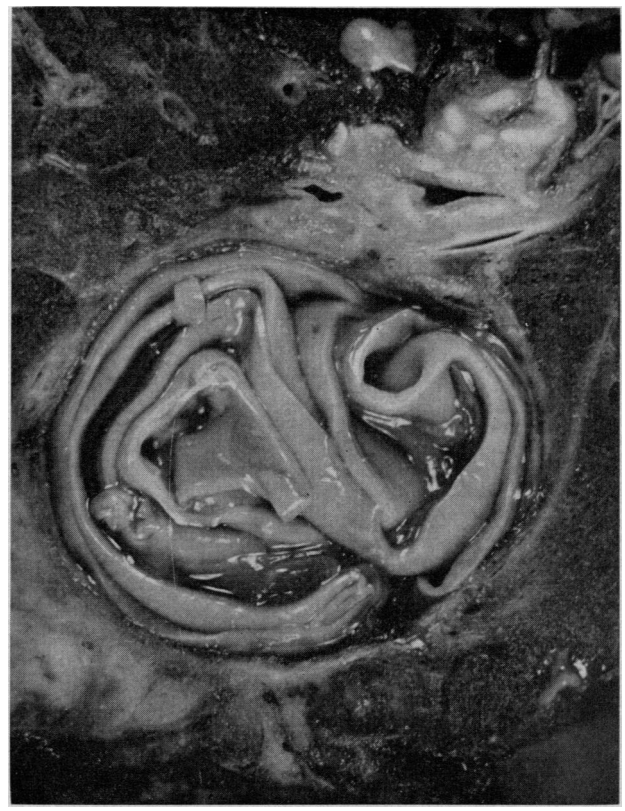

Fig. 18.—Hydatid cyst, apical segment, left lower lobe.

Two years later, on follow-up examination, he was well.

## RADIOLOGICAL CONSIDERATIONS

1. *Pneumonia.*—In Loeffler's pneumonia, regardless of the inciting agent, the radiological findings consist of evanescent patterns of rapidly appearing and rapidly changing infiltrates in any area of one or both lungs which may disappear in 24 hours. These opacities are extremely variable in size, shape, and distribution. Their only characteristic features are: (1) the quickly changing pattern and (2) their association with eosinophilia which is maximal while the transient radiological lesions are most evident. The histopathological change is lobular edema in random distribution. This type of pulmonary disease may occur with many parasitic infestations and is not uncommonly encountered when roundworm larvae migrate through the lungs.

Non-specific pneumonic infiltrates are the rule, but occasionally severe pulmonary edema and interstitial pulmonary hemorrhage may occur (ascariasis, strongyloidiasis). Pleural reactions (ankylostoma, hydatid disease, amebiasis) and effusions may also occur.

2. *Disseminated ("miliary") lesions.*[11]—Infestations with schistosomiasis will on occasion produce a diffuse miliary infiltrate, which is less common in other parasitic diseases. This type of reaction may provoke a chronic interstitial pneumonitis which heals by fibrosis.

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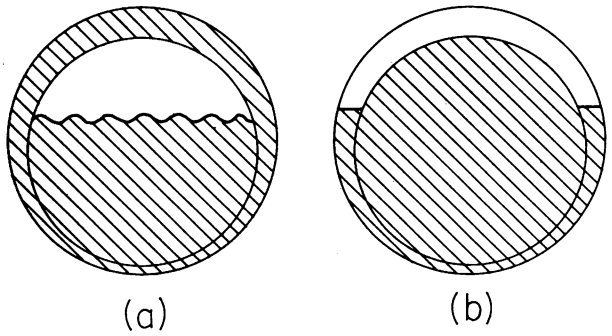


**Fig. 19.**—Pulmonary hydatid cysts: **a.** Diagrammatic sketch showing collapsed parasitic membrane floating on retained fluid following intrabronchial rupture—so-called water lily appearance or "camalote sign". **b.** Pericystic emphysema due to collection of air of crescentic form separating the fibrous host capsule from the intact fluid-filled parasitic membrane. The cyst may contain daughter cysts.

3. *Bronchomediastinal adenopathy.*—Enlargement of hilar lymph nodes often produces a readily recognized roentgen picture but, as with pneumonic manifestations, this too is a relatively non-specific finding. The presence of enlarged lymph nodes, however, is of importance in prompting further

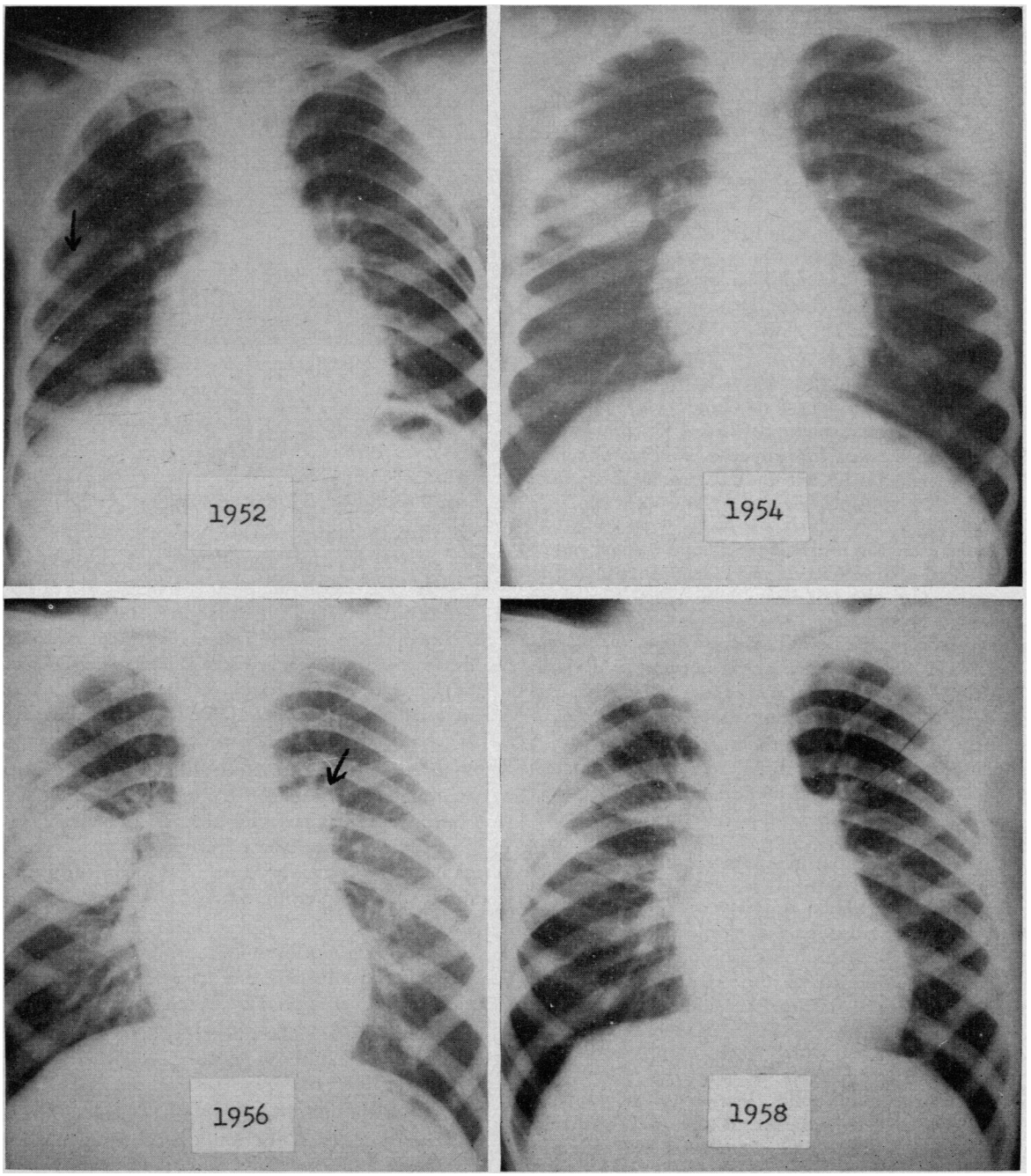


**Fig. 20.**—Serial posteroanterior films showing sequence in development of bilateral pulmonary hydatid cysts. 1952—Single small nodule on the right. By 1954 this lesion has become much larger. By 1956 there is a further slight increase in size and a second lesion is now visible, superimposed on the left hilus. By 1958 both cysts have ruptured and the contents have been evacuated.

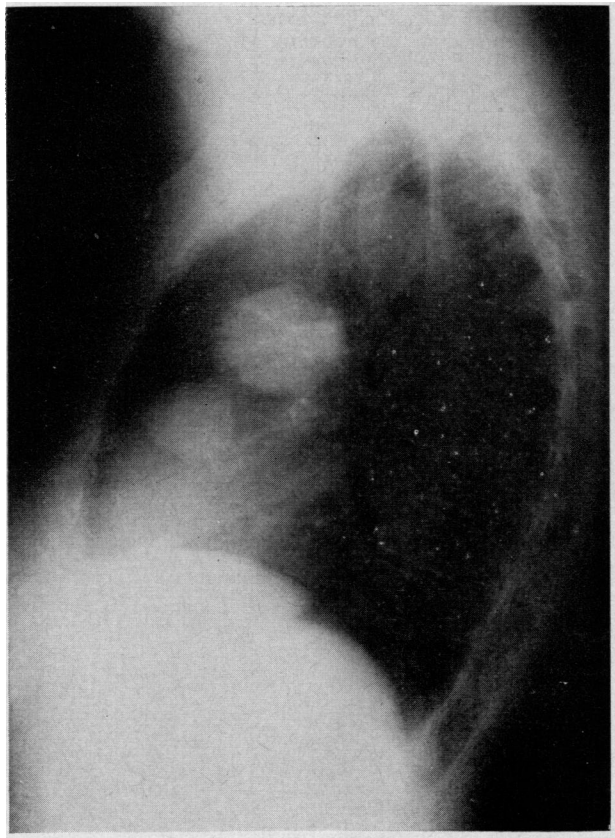

Fig. 21a

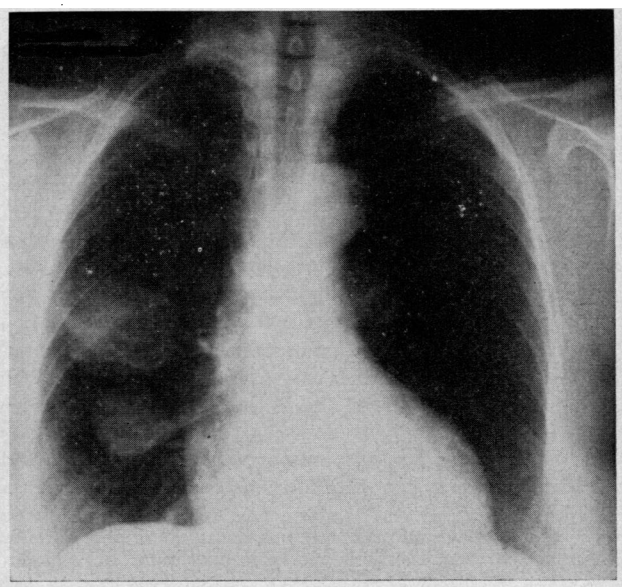

Fig. 21b

Figs. 21a & 21b.—Posteroanterior and right lateral films showing two large nodular hydatid cysts in the right lung without evidence of calcification. Confirmation obtained at operation.

diagnostic studies. Of the parasitic diseases, filariasis may produce radiologically evident enlargement of bronchial and/or mediastinal nodes (Case 3, Fig. 11).

4. *Parenchymal and mediastinal cysts*: (i) *Hydatid disease.*—When hydatid cysts occur in the lungs, mediastinum or heart, radiological diagnosis may be highly accurate. These cysts are round or oval in shape, are sharply defined and may show a distinct alteration in shape with forced inspiration and expiration. They may be single or multiple, may rupture, evacuate or become secondarily infected.[12] The cyst wall, which rarely calcifies in the chest, is made up of two layers. The outside fibrous layer is formed by host tissues, the inner by the parasitic membrane. Collections of air may separate the two layers to produce a halo or crescentic effect, described by Kerley[13] as pericystic emphysema. Should the cyst rupture and collapse but the fibrous capsule remain intact, the inner cyst membrane in its collapsed state will float on the surface of retained fluid content like a floating water-lily.[14] This appearance is described as the sign of the camalote, a South American plant (Fig. 19). Another helpful sign in diagnosis is the tendency of cysts, when multiple, to adhere together and, when touching, to have flattened contacting surfaces. Cysts can cause displacement of adjacent structures or erosion of ribs, sternum, etc. More rarely an alveolar type of hydatid disease may occur which does not have typical signs in the radiograph. Figs 20 and 21 illustrate radiological features in several other cases not described in this paper.

(ii) *Amebiasis.*—In the severe complications of this disease, rupture of amebic liver abscesses into the pleural space and lung may occur; secondary lung abscess, pleural effusion, empyema and bronchohepatic fistulas may result. Rarely, however, true metastatic "amebomas" may occur and produce a nodular or spherical parenchymal lesion.

(iii) *Paragonimiasis (lung fluke).*—This disease is characterized by a peripherally located, small, round or oval cyst measuring 1 to 2 cm. in diameter. The cyst wall is thin above (1-2 mm.) and thicker below (3-4 mm.) and may be surrounded by a narrow ring of pericystic inflammatory reaction.[15] Pleural effusion and calcification of the cyst wall are very uncommon, but pleural thickening is frequent. It is said that 90% of those infested show positive radiological signs.[13]

5. *Soft tissue calcification.*—This may occur in trichinosis in intercostal and diaphragmatic muscles, but the intrathoracic structures are not involved. True parenchymal calcification due to parasitic disease is very rare.[16] In cysticercosis, calcified larvae may occur in the paraspinal muscles and around the shoulder girdle.

## SUMMARY

Pulmonary manifestations of parasitic infestations may be transient (Loeffler's syndrome, pleurisy, bronchospasm and bronchitis) or permanent (cysts and abscesses, miliary lesions, adenopathy about the trachea and bronchi, pleurisy with or without effusion, empyema, and pneumohydrothorax). The transient changes may be caused by a variety of parasites: *Ascaris lumbricoides, Strongyloides stercoralis, Ankylostoma duodenale,* several types of Filaria, *Giardia*

*lamblia* and *Trichinella spiralis*. Permanent lesions result from invasion by *Entameba histolytica, Tenia echinococcus,* Schistosoma of different varieties, and *Paragonimus westermani*. Because these conditions are not well know to most Canadian physicians, diagnosis may be delayed despite the fact that many of the parasites may be detected by microscopic examination of stool or sputum or biopsy specimens, and by skin or serological tests. Eosinophilia and allergic reactions such as asthma and urticaria may be important clues. Illustrative case histories are presented and the radiological features described. A case report of a patient with amebiasis demonstrated that a latent stage lasting several decades may intervene before the organism spreads from the bowel and proliferates in various organs. In this case it appeared that amebae left the bowel, invaded para-aortic nodes, and migrated to the lumen of the inferior vena cava.

We are grateful to members of the staff of the Toronto Western Hospital for their active co-operation, and permission to use their case histories, pathological slides and radiographs. To Mrs. A. du Journo our best thanks for the artistic composition.

REFERENCES

1. SCHNEIDER, J.: Les maladies tropicales dans la pratique médicale courante, Masson & Cie., Paris, 1962, p. 121.
2. WEINGARTEN, R. J.: *Lancet,* **1**: 103, 1943.
3. BONNIN, H. ET MORETTI, G.-F.: *Presse Méd.,* **60**: 221, 1952.
4a. JARNIOU, A-P. ET MOREAU, A.: Les parasitoses respiratoires fréquentes; hydatidose-ambiase, pneumopathies parasitaires avec hyperéosinophilie sanguine, Vigot Frères, Paris, 1958. p. 89.
4b. JARNIOU, A. P ET MOREAU, A.: Les parasitoses respiratoires fréquentes; hydatidose-ambiase, pneumopathies parasitaires avec hyperéosinophilie sanguine, Vigot Frères, Paris, 1958, p. 44.
5. KOPPISCH, E.: Protozoal and helminthic diseases, In: Pathology, edited by W. A. D. Anderson, 4th ed., C. V. Mosby Company, St. Louis, 1961, p. 353.
6. BOYD, W.: A text-book of pathology; an introduction to medicine, 5th ed., Lea & Febiger, Philadelphia, 1947, p. 216.
7. ROBBINS, S. L.: Textbook of pathology, with clinical applications, W. B. Saunders Company, Philadelphia, 1957, p. 364.
8. MELTZER, H. *et al.*: *Canad. Med. Ass. J.,* **75**: 121, 1956.
9. DÉVÉ, F.: L'échinococcose primitive, Masson & Cie., Paris, 1949, p. 79.
10. FINLAYSON, D. M. AND FERGUS, A.: *Canad. Med. Ass. J.,* **88**: 1004, 1963.
11. MIDDLEMISS, H.: Tropical radiology, William Heinemann, Ltd., London, 1961, p. 189.
12. SALMON, M. A.: *Dis. Chest,* **40**: 61, 1961.
13. SHANKS, S. C. AND KERLEY, P., editors: A textbook of x-ray diagnosis, by British authors, Vol. 2, 3rd ed., W. B. Saunders Company, Philadelphia, 1962, p. 663.
14. SCHINZ, H. R. *et al.*: Roentgen-diagnostics, Vol. 3, Grune & Stratton, Inc., New York, 1953, p. 2558.
15. MILLER, F. L. AND WALKER, R.: Radiology, **65**: 231, 1955.
16. BRETLAND, P. M.: *Brit. J. Radiol.,* **35**: 603, 1962.

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