Morphology. In general, the three species of adult worms resemble each other closely; the peculiarities and differentiating features are however shown in the table on p. 138. Schistosomes are long-lived worms, having a *life span* of 20 to 30 years.

MECHANISM OF EGG-LAYING AND EGG-EXPULSION. Oviposition usually occurs in the small venules of vesical plexus. The female, held in the gynaecophoric canal of the male, extends its anterior end far into the smallest venules and deposits the eggs longitudinally, one at a time. Each time an egg is laid, the worm withdraws a short distance and lays another egg immediately behind the first. In this way, the venules are filled with eggs pointing backwards; the worms *in copula* migrate to an adjacent venule. The eggs are held in position by the spines and by the contraction of vessels resulting from the withdrawal of the parent worm. The eggs then work their way through the vessels and the mucosa of the urinary bladder, enter into the cavity and escape with the urine, usually at the end of the micturition.

Differentiating Features of Schistosomes

	S. haematobium	*S. mansoni*	*S. japonicum*
MALE:			
Size:	1 to 1.5 cm by 1 mm.	1 cm by 1 mm.	1.2 to 2 cm by 0.5 mm.
Cuticula:	Finely tuberculated.	Grossly tuberculated.	Non-tubercular.
Testes:	4 to 5; in groups.	8 to 9; in a zigzag row.	6 to 7; in a single file.
FEMALE:			
Size:	2 cm by 0.25 mm.	1.4 cm by 0.25 mm.	2.6 cm by 0.3 mm.
Ovary:	Behind the middle of the body.	Anterior to the middle of the body.	In the middle of the body.
Uterus:	Contains 20 to 30 eggs.	Contains 1 to 3 eggs (usually 1)	Contains 50 or more eggs.
REUNITED INTESTINE:	Long (reuniting about the middle of the body).	Longest (reuniting in the anterior half of the body).	Short (reuniting in the posterior fourth of the body).
EGG:	150 by 50 µm; terminal spine.	150 by 60 µm; lateral spine.	100 by 65 µm; lateral knob.
CERCARIAE: (Cephalic Glands)	2 pairs oxypilic and 3 pairs basophilic.	2 pairs oxyphilic and 4 pairs basophilic.	5 pairs oxyphilic (no basophilic).
INTERMEDIATE SNAIL HOST:	Bulinus (Physopsis) and Planorbarius.	Biomphalaria and Australorbis.	Oncomelania.
DEFINITIVE HOST:	Man.	Man.	Man and domestic animals.
GEOGRAPHICAL DISTRIBUTION:	Africa, Near East and Middle East.	Africa and South America.	Far East (Oriental).
HABITAT:	Vesical and prostatic venous plexus.	Mesenteric plexus of sigmoido-rectal area (inferior mesenteric vein and its radicles).	Mesenteric plexus of ileocaecal area (superior mesenteric vein and its radicles).

Life Cycle. *S. haematobium* passes its life cycle (Fig. 134) in two hosts.

Definitive Host. Man. Adult worm living in vesical and prostatic venous plexus.

Intermediate Host. Fresh-water snails (*Bulinus truncatus* and other species throughout Africa, *Planorbarius metidjensis* in Morocco and Portugal, and *Ferrissia tenuis* in India).

The embryonated eggs are passed with the urine of the definitive host and gain access to water. Ciliated larvae (miracidia), hatched out of the eggs, move freely in water in search of their intermediate host. The miracidium on entering its proper larval host, penetrates into the soft tissues of the snail and ultimately makes its way into the liver. Here it loses its cilia and other organs and in the course of 4 to 8 weeks undergoes developmental changes. The miracidium is transformed into a tubular sporocyst; the latter multiplies and forms a second generation of sporocysts. Several weeks after the infection, when no further multiplication occurs, the daughter-sporocysts give rise to the final larval forms, the fork-tailed cercariae which are infective to man. The cercariae break off from the sporocyst and escape from the snail into water.

Infection results when human beings bathing or wading in this water are infected, the cercariae penetrating the unbroken skin directly. On entry the cercariae cast off their tails (now known as schistosomulae) and gain access to a peripheral venule. From here, they are carried through the right heart into the pulmonary capillaries. It requires some days for the larvae to pass through the capillary bed in the lungs, whence they are carried through the left heart into the systemic circulation. The majority are shunted in the abdominal aorta and gain access to the mesenteric artery, pass

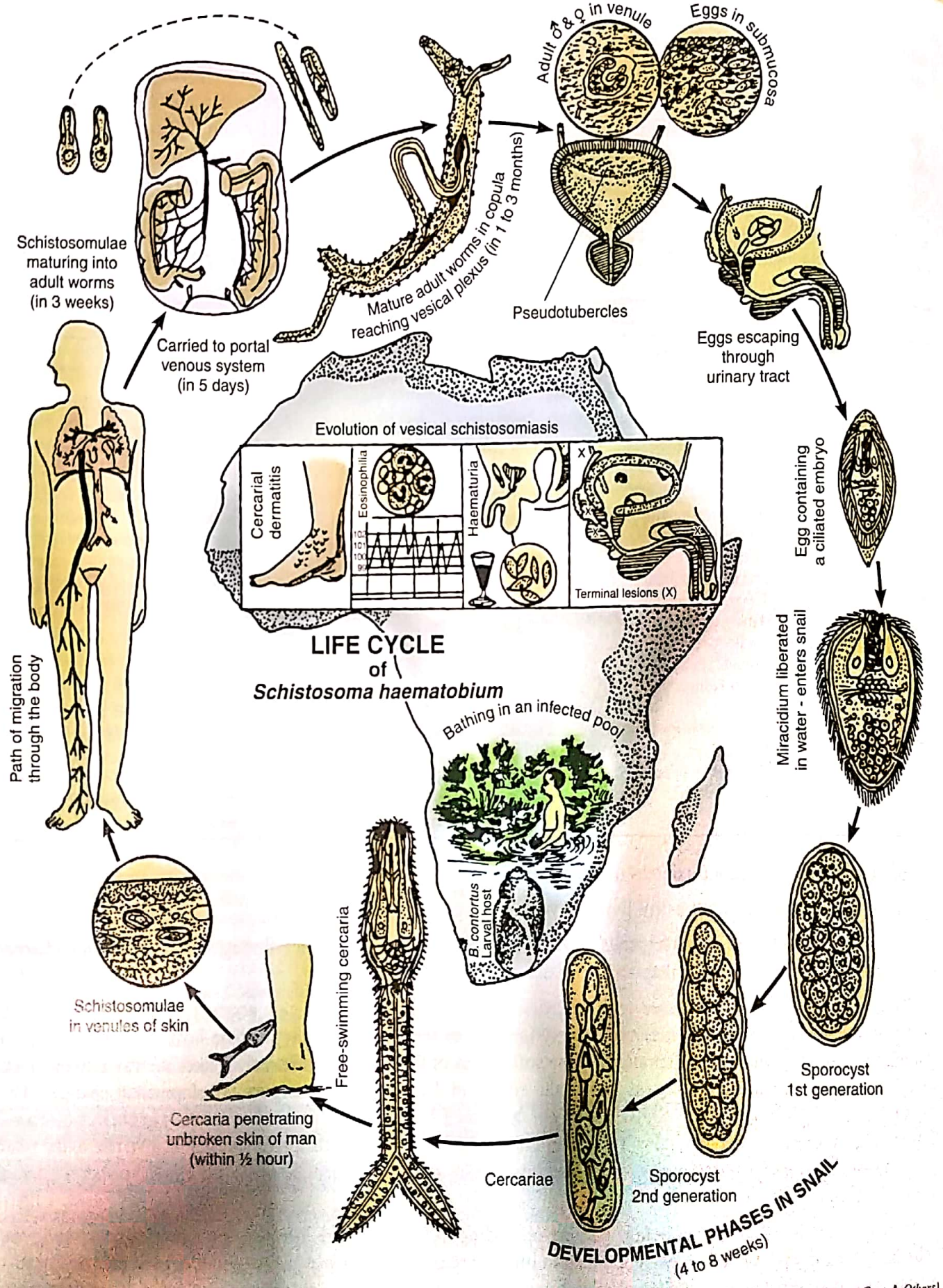

Fig. 134

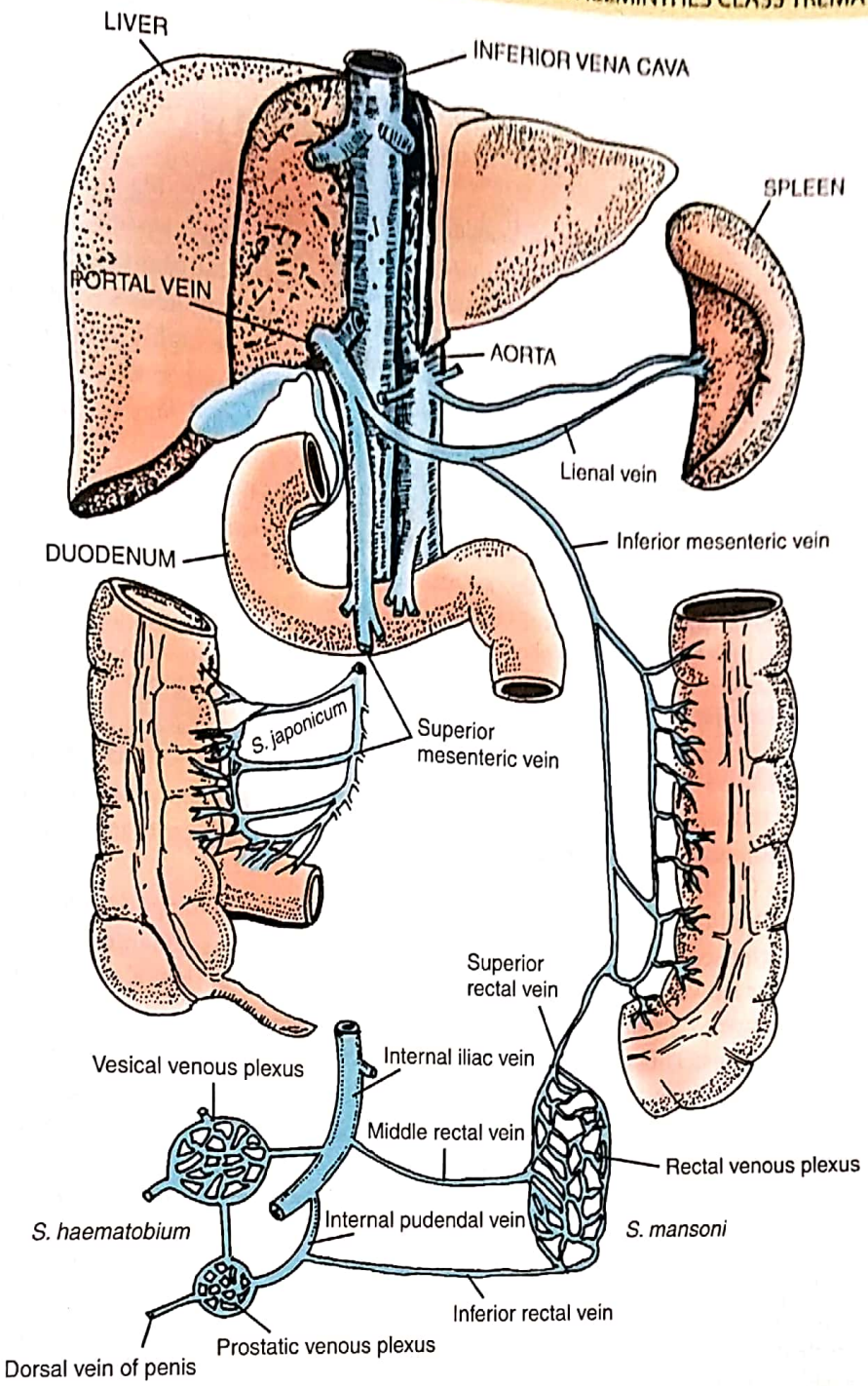

Fig. 135—Portal venous system and its connections.
Route through which adult Schistosomes migrate to their sites of location.

through the capillary bed in the intestine and enter portal circulation (taking 5 days to reach the liver). In the intrahepatic portion of the portal blood stream, the larvae grow into adults (maturing in 3 weeks from the time of entry). After becoming sexually differentiated, they move out of the liver against the blood current, migrating into the inferior mesenteric vein, rectal venous plexus, pelvic veins, and eventually enter the vesical plexus of veins (Fig. 135). It takes about 1 to 3 months for the worms to reach the vesical and pelvic plexuses of veins after the initial exposure of the skin. When the worms are sexually mature, they copulate (the females are enclosed in the males) and the fertilised females lay eggs which are ultimately voided with the urine. The cycle is thus repeated.

Note: Schistosomes are the single exception amongst the digenetic trematodes, where rediae are not produced, asexual multiplication taking place only in the sporocyst stage.

Pathogenicity. An individual bathing in an infected pool or coming in contact with contaminated water is liable to be infected. The cercariae stick to the surface of the skin of the swimmer or bather, by means of their ventral suckers (acetabula) and as the water begins to evaporate, penetrate the skin.

Infecting Agent—Cercariae. These have a free-swimming existence and can live in this state for a maximum period of 3 days.

Portal of Entry—Skin. *Site of Localisation*—Vesical plexus of veins (urinary bladder).

PATHOGENESIS. The terminal-spined eggs of *S. haematobium* may erode blood vessels and cause *haemorrhages*. Schistosome eggs, deposited in the tissues, act like foreign protein and have an irritative effect leading to round cell infiltration and connective tissue hyperplasia. The tissue reaction in these cases produces what is known as formation of a "*pseudotubercle*" around each egg (egg-granuloma). The early nodules are highly cellular and are composed of eosinophils, giant cells, monocytes and lymphocytes; later on, the cellular reaction tends to disappear and is replaced by a whorl of fibrous tissue, in the centre of which degenerated and calcified eggs may be found. Large and progressive granulomas are found only around the eggs and may cause a diffuse fibrosis.

Schistosome granuloma. The Schistosome eggs secrete soluble substances which pass through the pores of the egg-shell and provoke a granulomatous reaction, a manifestation of cell-mediated delayed hypersensitivity. In sensitised individuals, granulomatous reaction becomes accelerated and enhanced on second exposure to eggs. The host-reaction is thus an immunological one and the sensitisation can be transferred by lymphoid cells and inhibited by immuno-suppressive agents (Warren, 1972)*.

The immunological reaction to Schistosome eggs plays a defensive as well as a destructive role, because (i) it can sequesterate antigen *in situ* and potentiate antigen catabolism and (ii) it can synthesise antibody locally in the cells that surround the mature granuloma.

* Warren, K. S. (1972). *Trans. Roy. Soc. Trap. Med. & Hyg.*, **66**, 417-32.

Clinical Features. The disease caused by infection with *S. haematobium* is referred to as schistosomiasis haematobia (urinary schistosomiasis or bilharziasis; endemic haematuria). Evolution of the disease passes through 3 phases:

(i) By the cercariae at the site of entrance: Local reaction (dermatitis). This is particularly seen with the cercariae of non-human schistosomes (adult worms in birds or small mammals).

(ii) By the toxic metabolites liberated during the growth of schistosomulae in the portal blood of the liver: General anaphylactic reaction characterised by fever, urticaria, eosinophilic leucocytosis, enlarged tender liver and palpable spleen. The symptoms appear between the 4th and the 5th week of the infection (k.a. Katayama fever in Japan). It is commonly seen in infection with *S. japonicum* and rarely with *S. haematobium*.

(iii) At the time of laying eggs: This may be regarded as a localising symptom, generally occurring within 3 to 9 months of the infection. The characteristic manifestation is a painless terminal haematuria. In course of time, the adjacent structures of uro-genital apparatus are involved, at first by the reversible granulomatous inflammatory reaction to eggs and later by the irreversible fibrosis and calcification.

It has been observed that a close relationship exists between vesical schistosomiasis and vesical carcinoma, particularly in areas where the infection is highly endemic.

ECTOPIC LESIONS. These are the result of an overflow phenomenon due to heavy infection. The eggs and worms escape into the pelvic veins and are carried to the lungs where the eggs excite a granulomatous reaction leading to fibrosis, pulmonary endarteritis, obstruction to pulmonary blood flow, pulmonary hypertension and finally chronic cor pulmonale. Eggs and adult worms have also been carried from the portacaval system into the distant parts of the body causing lesions in the brain (a space-occupying lesion, common in *S. japonicum*) and the spinal cord (transverse myelitis-like syndrome, common in *S. haematobium* and *S. mansoni*).

Diagnosis. This is based on the demonstration of eggs of *S. haematobium* in:

(a) A microscopical examination of urine (centrifuged deposit). Now-a-days sophisticated filtration techniques give quantitative estimation of egg excretion.

(b) Examination of stool: Direct smear of the stool is not sensitive. Concentration methods (Keto thick smear or its modified method) may detect the eggs of schistosoma species.

(c) A piece of vesical mucosa is removed by cystoscopic biopsy. The excised tissue is divided into two pieces. One piece is compressed between two slides and examined for eggs under the low power of the microscope. The other piece is placed in a fixative for histological examination.

OTHER TESTS: (i) *Blood Examination*. This should include:

(a) Eosinophilic Count—Increased in early cases.

(b) Aldehyde Test—Often positive (due to high globulin value).

(c) Complement Fixation Test—Sera of patients react positively with cercarial antigen obtained from infected snail's liver.