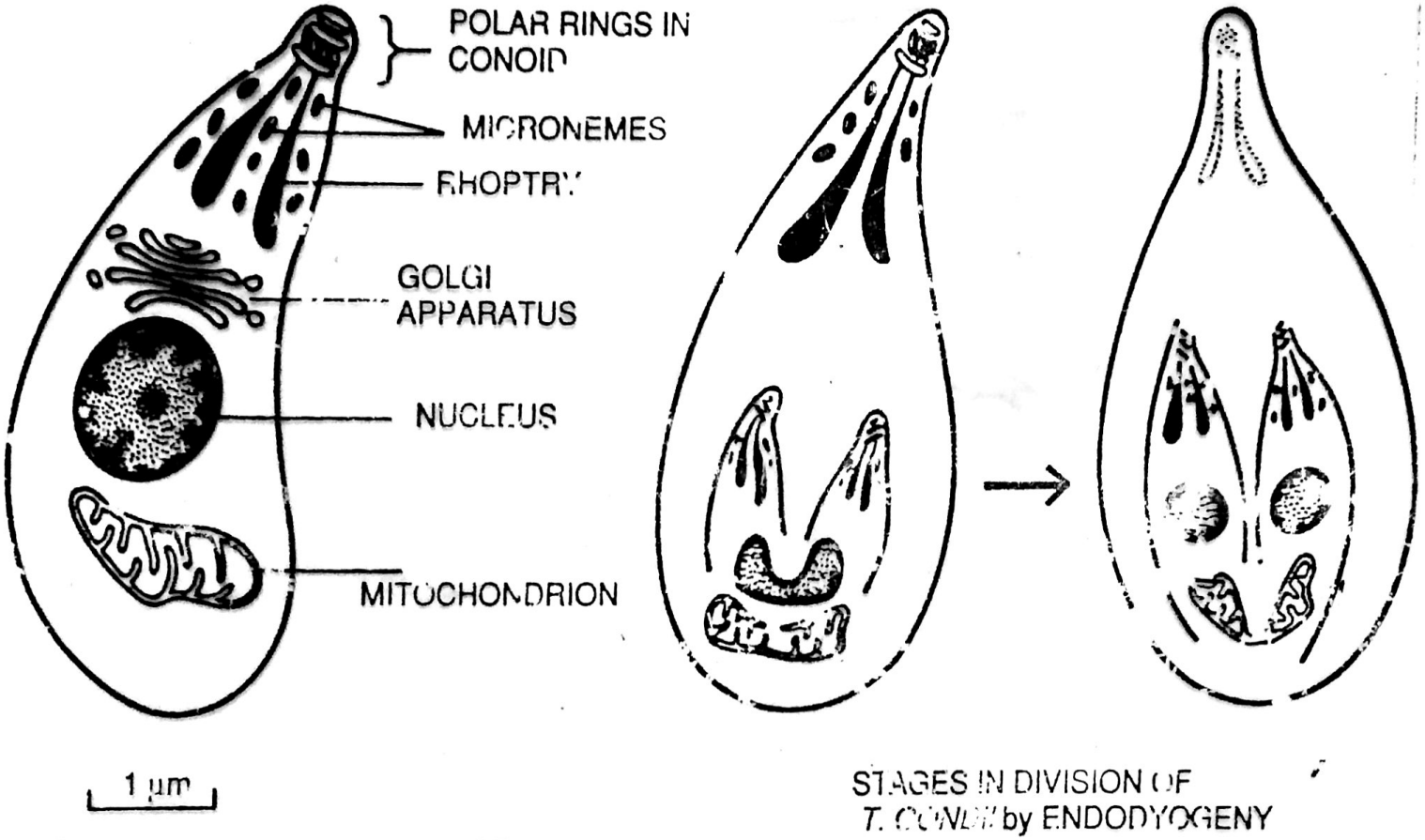

Figure 3–12. Diagrammatic ultrastructure of *T. gondii* tachyzoite and stages in division of tachyzoite by endodyogeny. (Redrawn from Pfefferkorn, ER, Cell Biology of *Toxoplasma gondii* in Modern Parasite Biology, edited by DJ Wyler, 1990, W.H. Freeman and Co., New York.)

TOXOPLASMA GONDII

APICOMPLEXA

ENDODYOGENY

TACHYZOITE

ZOITE

OOCYST — CYST

CONGENITAL TOXOPLASMOSIS

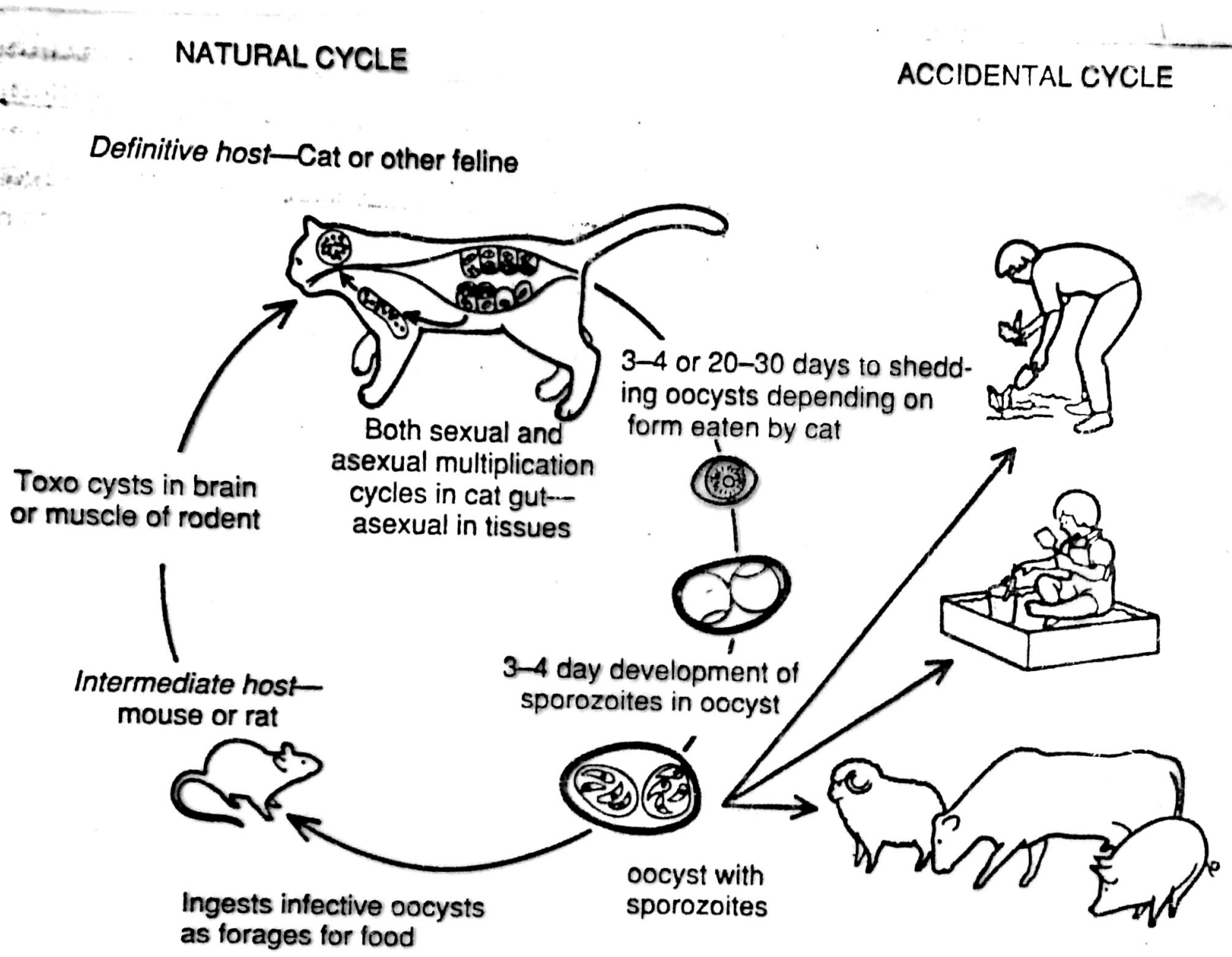

Figure 3–14. Transmission cycle of *Toxoplasma gondii*.

TRYPANOSOMA

BRUCEI RHODESIENSE

BRUCEI GAMBIENSE

TSTSE FLY

SLEEPING SICKNESS

COSIS

FREE LIVING AMOEBA

NAEGLERIA FOWLERI

ACANTHAMOEBA

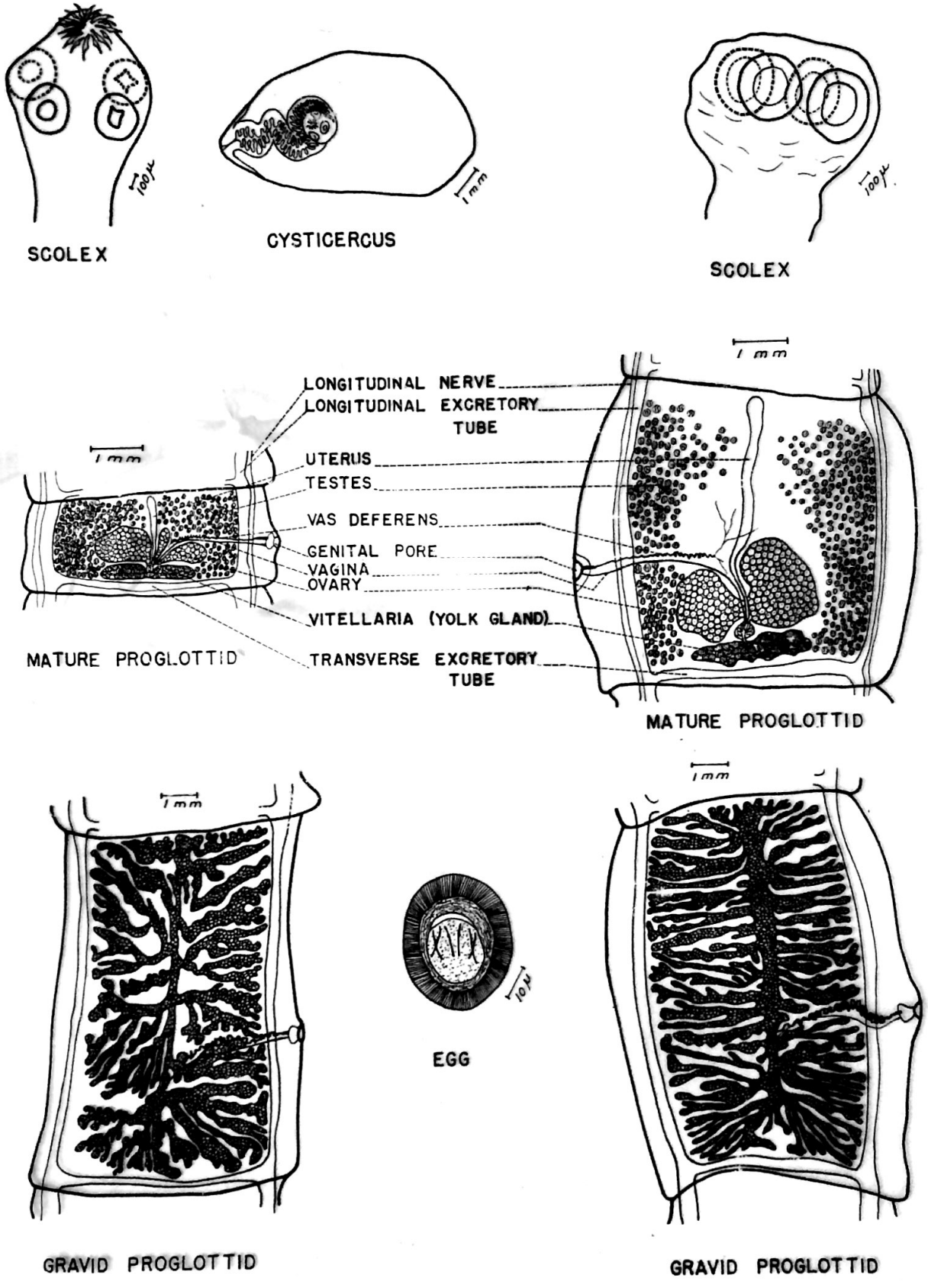

Figure 9–5. *Taenia solium* and *T. saginata*—a diagrammatic comparison.

ingestion of food or water contaminated with human feces, and (2) autoinfection by the unclean hands of unsuspecting carriers of the adult worm. Infection with the larval stage can also occur if proglottides are regurgitated into the stomach of an individual infected with the adult tapeworm, but this mechanism may be more theoretical than real. Only about 25% of patients found to have clinical cysticercosis give a history of known infection with a tapeworm. Since cysticercosis comes to medical attention only when vital functions are affected, eg, the central nervous system, the infection is more prevalent than reported. The disease is most common in China, India, many countries of Africa, Central and South America, and Mexico. In many Latin American countries neurocysticercosis is the most common cause of epileptic seizures in adults, and suspected cases occupy 10% to 15% of beds in neurologic wards of hospitals.

Patients with cerebral cysticercosis are being encountered with increasing frequency in medical centers of cities with large Hispanic immigrant populations. A recent report estimated that 10 of 138 cases reported over a 4-year period in Los Angeles were acquired within the United States.

Some of these cases, as illustrated by the report of neurocysticercosis in Jewish families who had never left Brooklyn, probably represent transmission from infected domestic employees within the household. The possibility also exists that infected migrant farm laborers could initiate foci of natural transmission within the United States.

PATHOLOGY AND SYMPTOMATOLOGY. The cysticerci, often multiple and even numbering into the hundreds, may develop in any tissue or organ of the human body. The most common sites are striated muscles and the orbit, but they also occur in the subcutaneous tissues, eye, heart, lung, and peritoneum. The growing cyst may produce some inflammatory reaction, but on the death of the larva, which may survive up to 5 years, there is an increase of the cystic fluid and a pronounced tissue response to the parasitic material. The degenerating parasite usually undergoes calcification. The pathology depends upon the tissue invaded

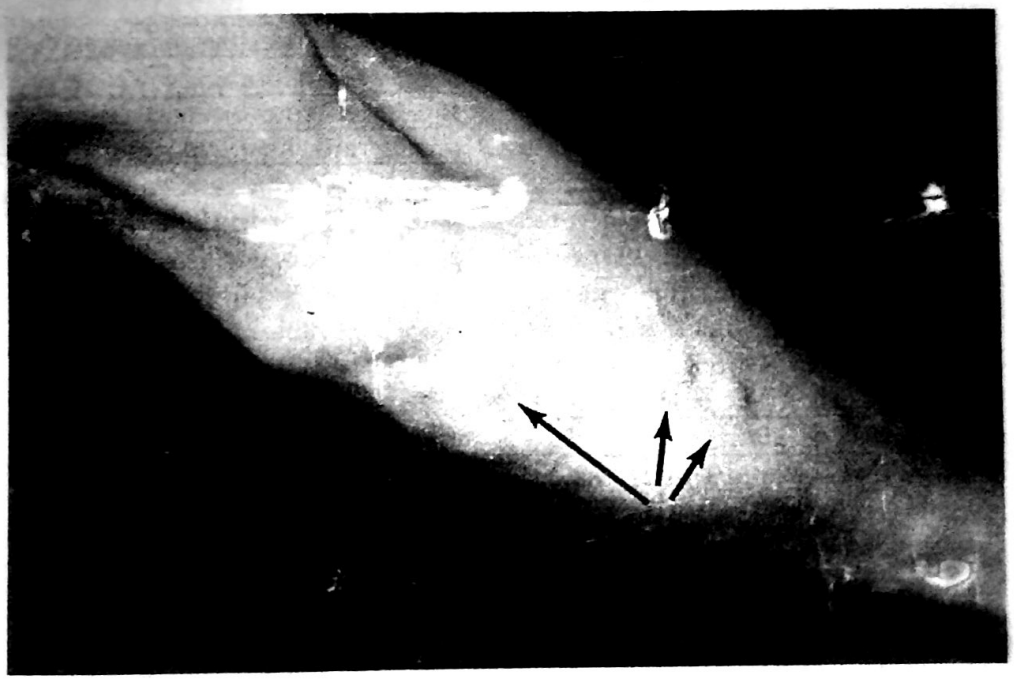

Figure 10–5. Multiple subcutaneous cysticerci on the inner surface of the arm. This was a patient in mainland China.

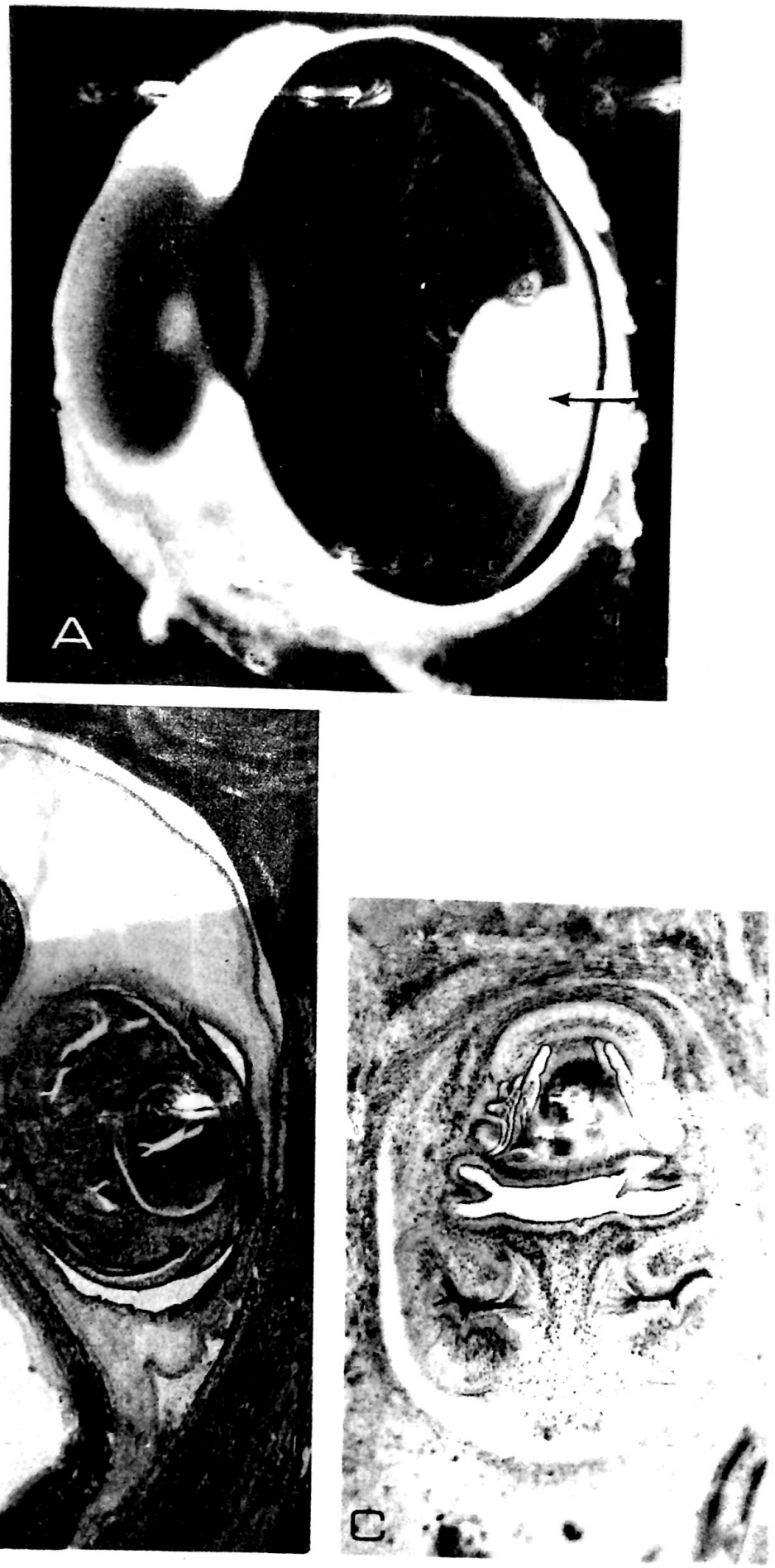

Figure 10–7. *Taenia solium* cyst in eye. **A.** Gross. **B.** Cyst embedded in retina. **C.** Section of scolex showing suckers and hooks. *(Courtesy of JAC Wadsworth, MD, Institute of Ophthalmology, NY.)*

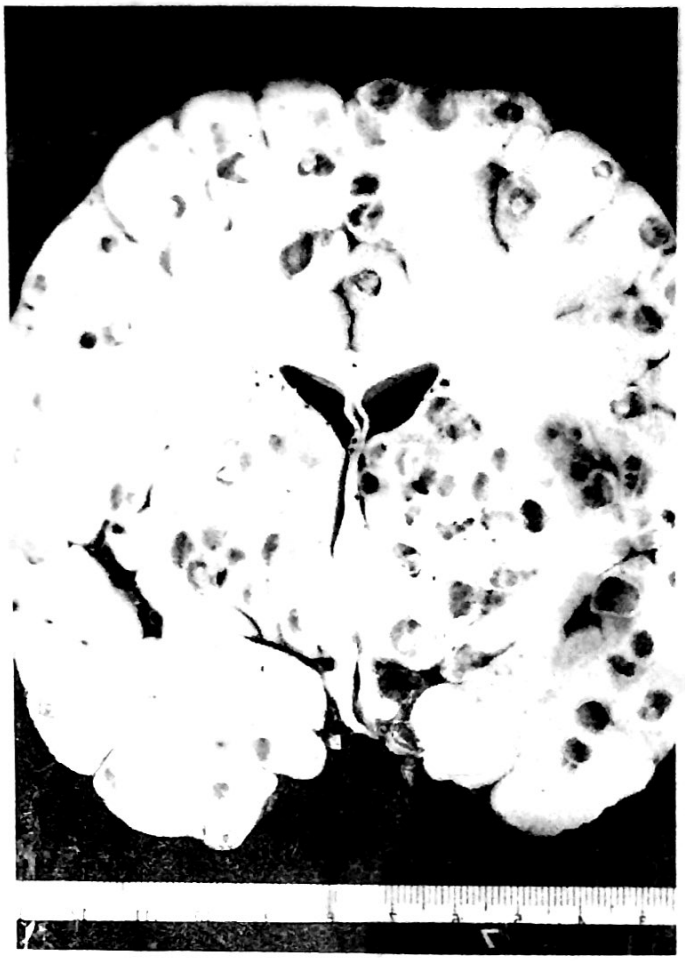

Figure 10–6. Human brain showing multiple cysticerci. The patient had a clinical picture of acute encephalitis that was fatal.

and the number of cysticerci. Invasion of the brain and eye causes serious damage (Figs. 10–5, 10–6, and 10–7).

A syndrome referred to as pseudohypertrophic myopathy, presumably the result of large numbers of cysticerci in skeletal muscles, has been reported. We are unaware of any other clinical syndromes in which systemic manifestations are attributed to a single heavy exposure to *T. solium* eggs and invasion of nonneural tissues by developing larvae. Some authors claim that muscular cysticercosis is more commonly seen in Asia than in other areas of the world.

The serious manifestations of the disease occur in cerebral cysticercosis, usually associated with the unrecognized general cysticercosis. Cysticerci may be present in the cerebral cortex, meninges, ventricles, and, less often, in the cerebral substance. They are usually found near the surface of the brain over the frontal and parietal lobes and along the middle cerebral arteries; they are found occasionally in the occipital region and the cerebellum. Severity of the cellular reaction to the cysticerci varies considerably, but they are generally tolerated well while the larva is alive. With death of the larva, the resulting inflammatory reaction and local edema are likely to trigger symptoms and signs of its presence. The cellular reaction eventually destroys the parasite and leaves a calcified nodule.

Neurologic symptoms often do not occur for [illegible] or more, but problems may arise earlier depending upon the location

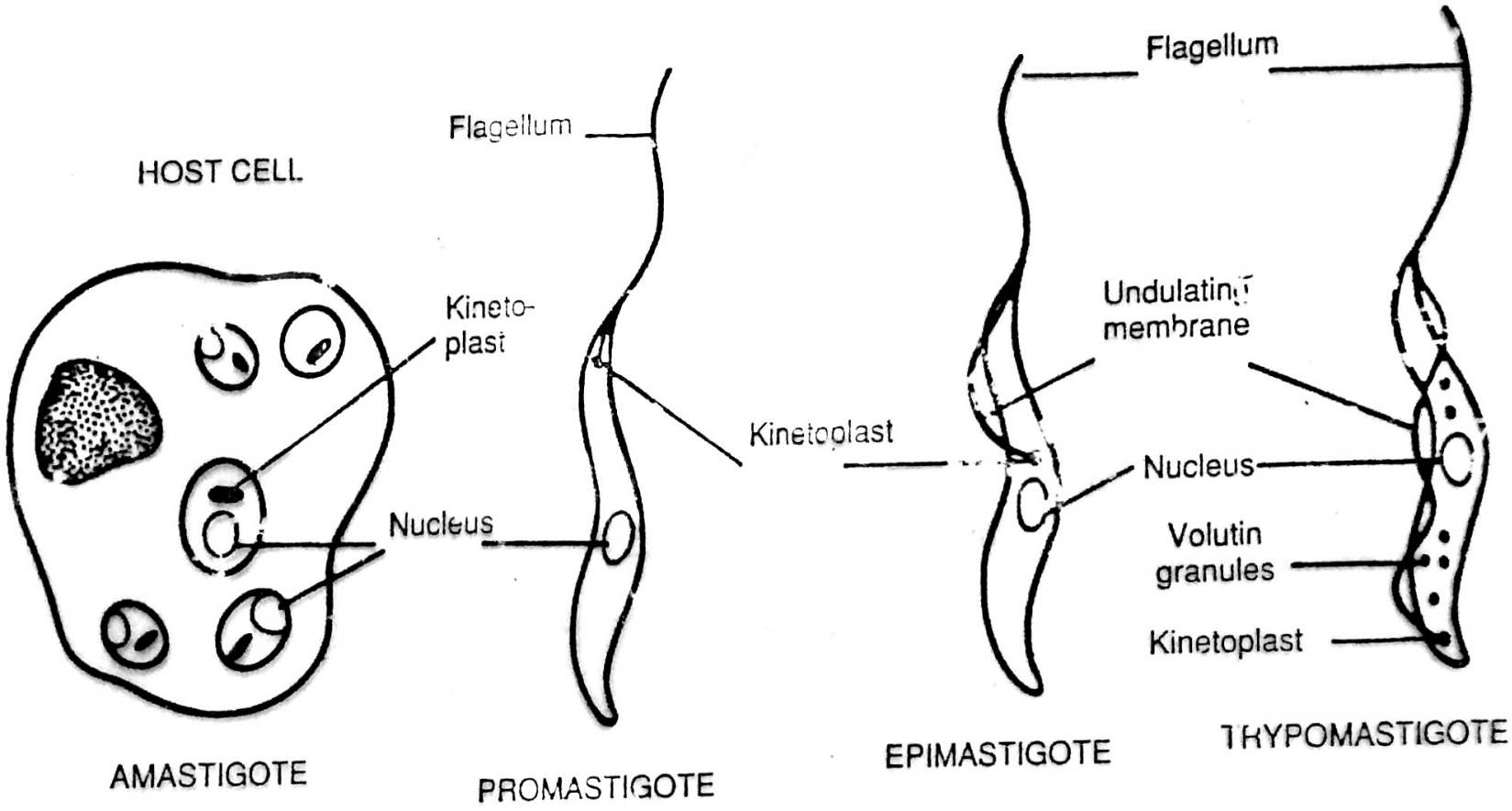

Figure 4–1. Developmental forms of Trypanosomatidae.

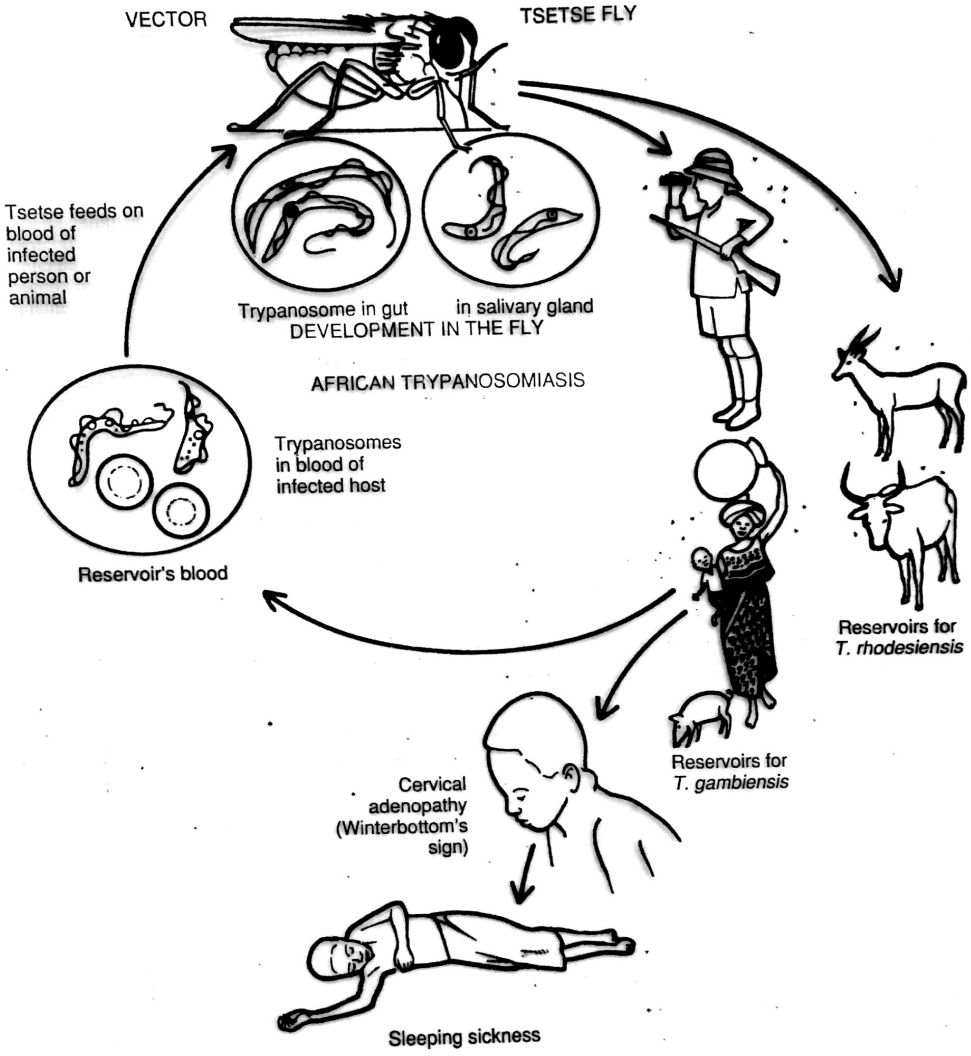

Figure 4–3. Life cycle of *Trypanosoma b. gambiense* and *Trypanosoma b. rhodesiense.*