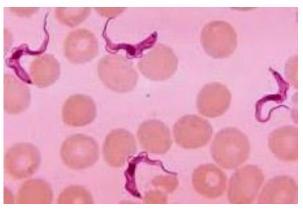
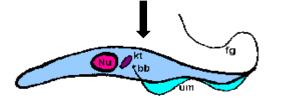
- 1- Clinical picture
- 2- Demonstration of trypanosomes:
- Microscopic examination of thin and thick films unstained or stained blood films
- -Culture on suitable medium (N.N.N OR Weinmann's media to detect **Epimastigote**)





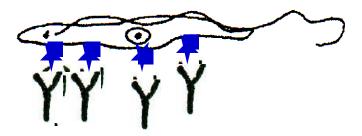


- Animal inoculation



3- Serological test:

<u>Increased total IgM</u> level in serum due to <u>antigenic variation</u> of the surface coat of the parasite.



Trypanosome posses genes that code for about 1000 variant forms of their surface glycoproteins (SVG). Switch to a different variant produces a new generation not susceptible to attack by immune factors specific to the previous generation.





<u>C.S.F</u>



Treatment

For the acute stages of the disease the drug of choice is suramin with pentamidine as an alternative.

Inchronic disease with CNS involvement, the drug of choice is melarsoprol.

Alternatives include trypars amide combined with suramin.

Trypanosoma cruzi causing Chagas' disease

Morphology

Trypomastigote (Monomorphic)

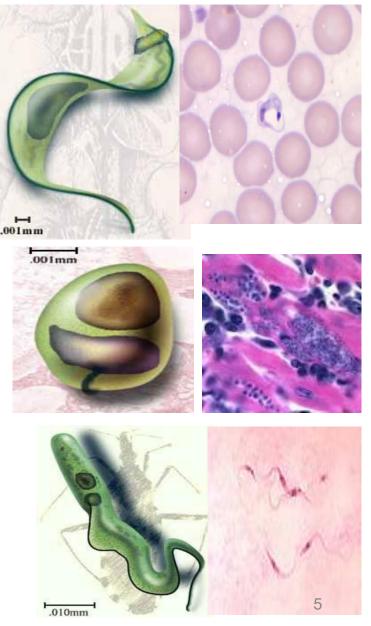
Slender shaped (20μ) – Central nucleus – C or U-shaped –Free flagellum 1/3 body-Large bulging peripheral kinetoplast

Amastigote

Obligatory intracellular – mainly in cardiac & Skeletal muscles – Brain meninges – Nerve ganglia – cells of GIT etc

Epimastigote (Vector only)

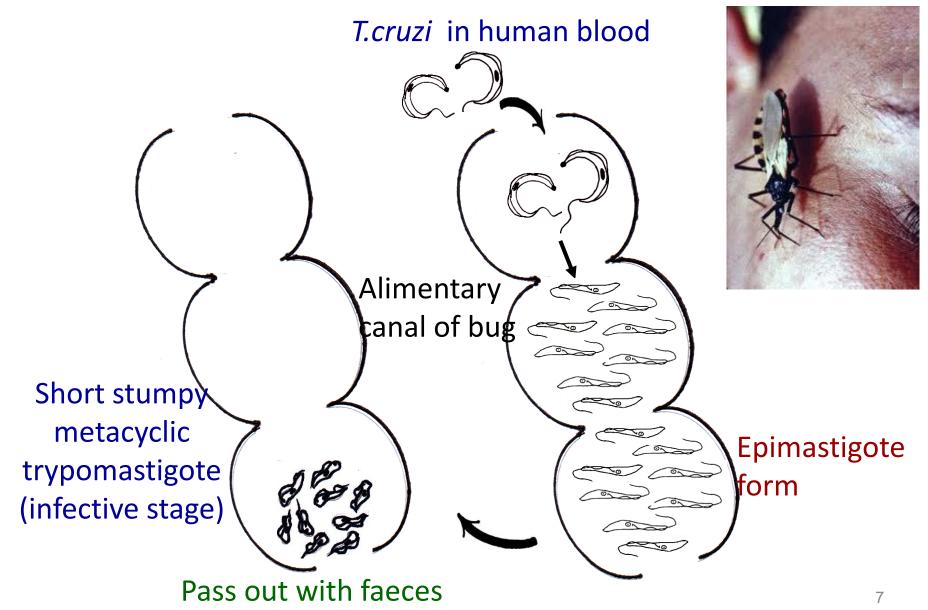
Spindle shape– Kinetoplast anterior to central nucleus– Undulating membrane is short – terminal free flagellum



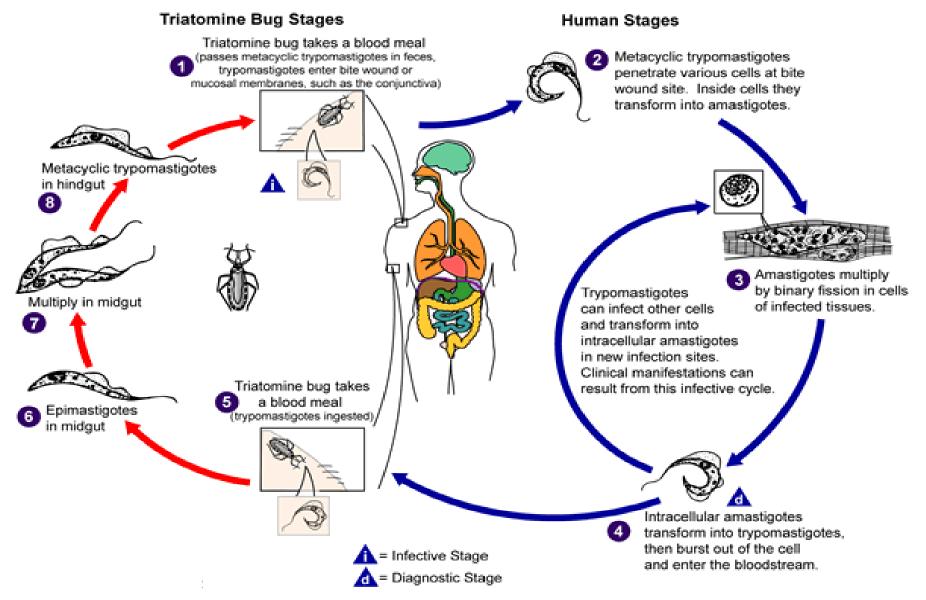
Geographical Distribution of Americam Trypanosomiasis



Mechanism of disease transmission by winged bug



LIFE CYCLE OF Trypanosoma cruzi



Mode of infection

Mainly by

Contamination of skin abrasion by winged bug faeces

Rarely by

Through infected blood transfusion Through infected mother's milk Through the placenta





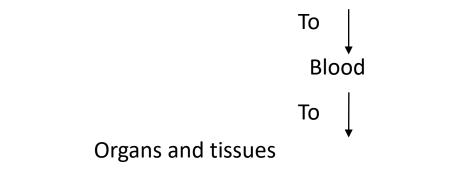


Pathogenesis and Clinical Picture



Chagoma occurs at the site of bite.

Parasite reaches regional lymph nodes

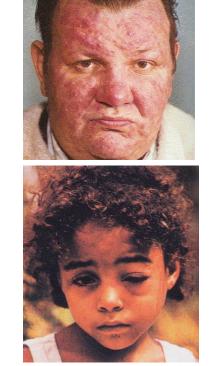


Fever, enlarged lymph nodes, skin rash, enlarged liver & spleen.

<u>**Romana's sign**</u> (Unilateral conjunctivitis appear suddenly togetherwith oedema of upper & lower eye lids & cheek)

Meningoencephalitis, heart failure

Death or pass to <u>C</u>hronic form



Pathogenesis and Clinical Picture

II- Chronic form

- Parasite produces antigens similar to patient's self antigens:
- The body produces auto-antibodies that
- cause damage to:
- Heart muscle fibres: congestive heart failure.
- Oesophageal muscle fibres: megaoesophagus and dysphagia. Destruction of Auerbach's plexus
 Colon muscle fibres: megacolon and constipation.
- CNS or thyroid gland Exacerbation of infection in immunosuppressed patients.

Amastigote form of *T.cruzi*





Finding the parasite in:

Blood film (C-shaped T.cruzi)

Biopsy from lymph node, liver or spleen (amastigotes)

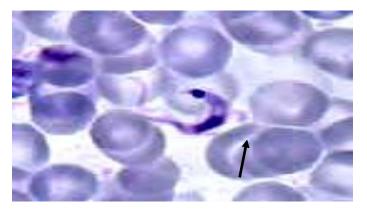
<u>Culture</u> (Epimastigotes)

Xenodiagnosis

Serological tests

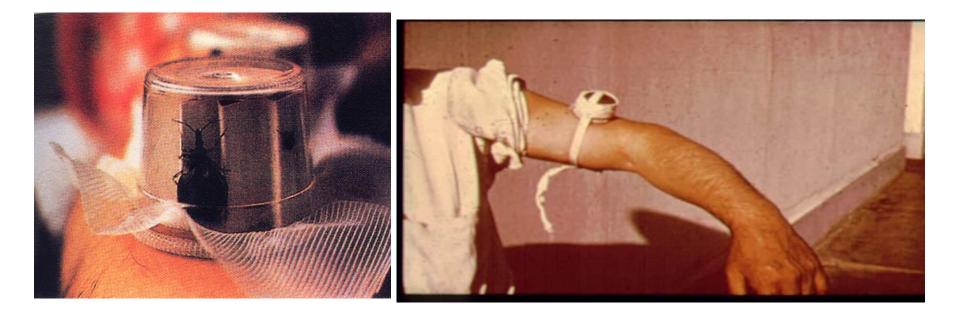
Cruzin test (I.D.)

Molecular techniques





Diagnosis (Xenodiagnosis)

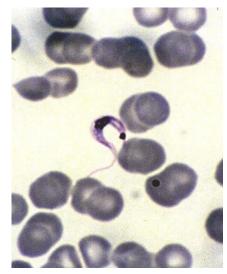


Highly efficient – demonstrate low level of parasite in blood <u>Method:</u>

A Laboratory bred winged bug is starved for 2 weeks then fed on suspected patient's blood – 30 days later, it faeces & gut examined for trypanosomes.

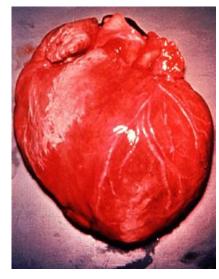


Trypomastigote



Winged Bug







Chagoma

Romana's sign



Treatment

Sleeping Sickness

In early stage of the disease:

Pentamidine OR Suramin

In late stages of the disease: Tryparsamide

For both early and late stages of the disease:

Eflornithine (DFMO) Ornidyl

Chagas Disease

Nifurtimox

- inhibits **intracellular** development .
- Drug of choice in acute and early chronic

OR

Primaquine destroys **Trypanosoma** in blood

Control

Sleeping Sickness

Treatment of patients

Control of vectors (Glossina)

Pentamidine as prophylactic drug

Chagas' disease

Treatment of patients

Control of vectors (*Triatoma*)

Elimination of reservoir hosts