TRYPANOSOMA SPP.

Introduction

1- **West African Trypanosomiasis:** " West African Sleeping Sickness" caused by *T. brucei gambiense*.

2- **East African Trypanosomiasis:** " East African Sleeping Sickness" caused by *T. brucei rhodesiense*.

3-**Chronic form:** caused by *T. brucei gambiense.* While **Acute Form** is caused by *T. brucei rhodesiense.*

4-**African Sleeping Sickness** *is* the 3rd important parasitic disease globally after **Malaria & Schistosomiasis.**

5-**West African Sleeping Sickness** is in regions along riverside while **East African Sleeping Sickness** is in Forest regions (Savannas).

Causes

Trypanosomiasis

- West African Trypanosomiasis
- East African Trypanosomiasis
- American Trypanosomiasis

T.brucei gambiense *T.brucei rhodesiense* → Sleeping sickness

Transmitted by
Glossina (tsetse fly)

T.cruzi → Chagas' disease

Transmitted by
Triatoma (winged bug)

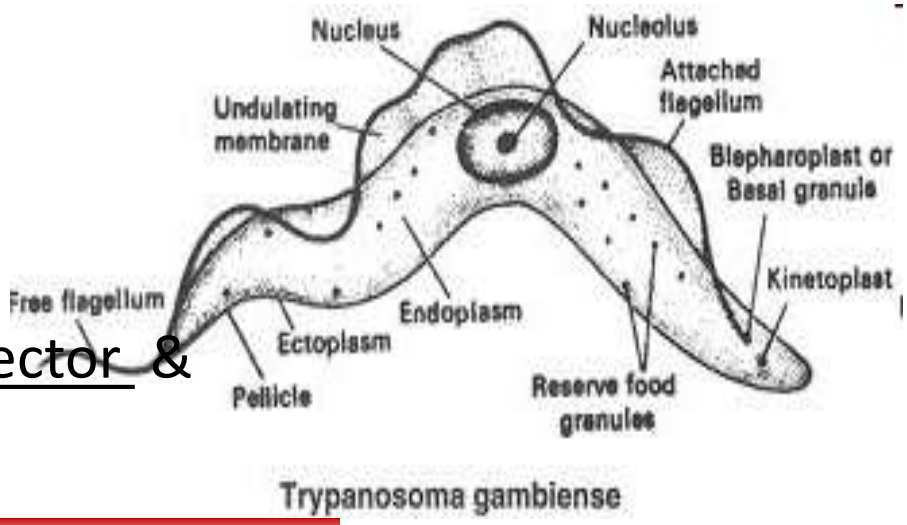
T. brucei complex

Morphology

Exist into 2 inter forms:

Trypomastigote in Blood/ Lymph / tissue space of various organs & C.N.S is terminal & fatal

Epimastigote in salivary gland of vector & Culture media.



Trypanosoma gambiense

Trypomastigote (Polymorphic Trypanosomes

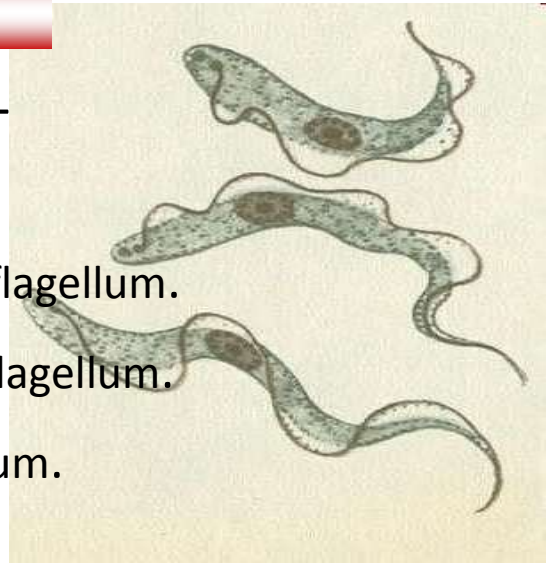
Spindle shaped – Central nucleus – free flagellum – undulating membrane.

3 forms

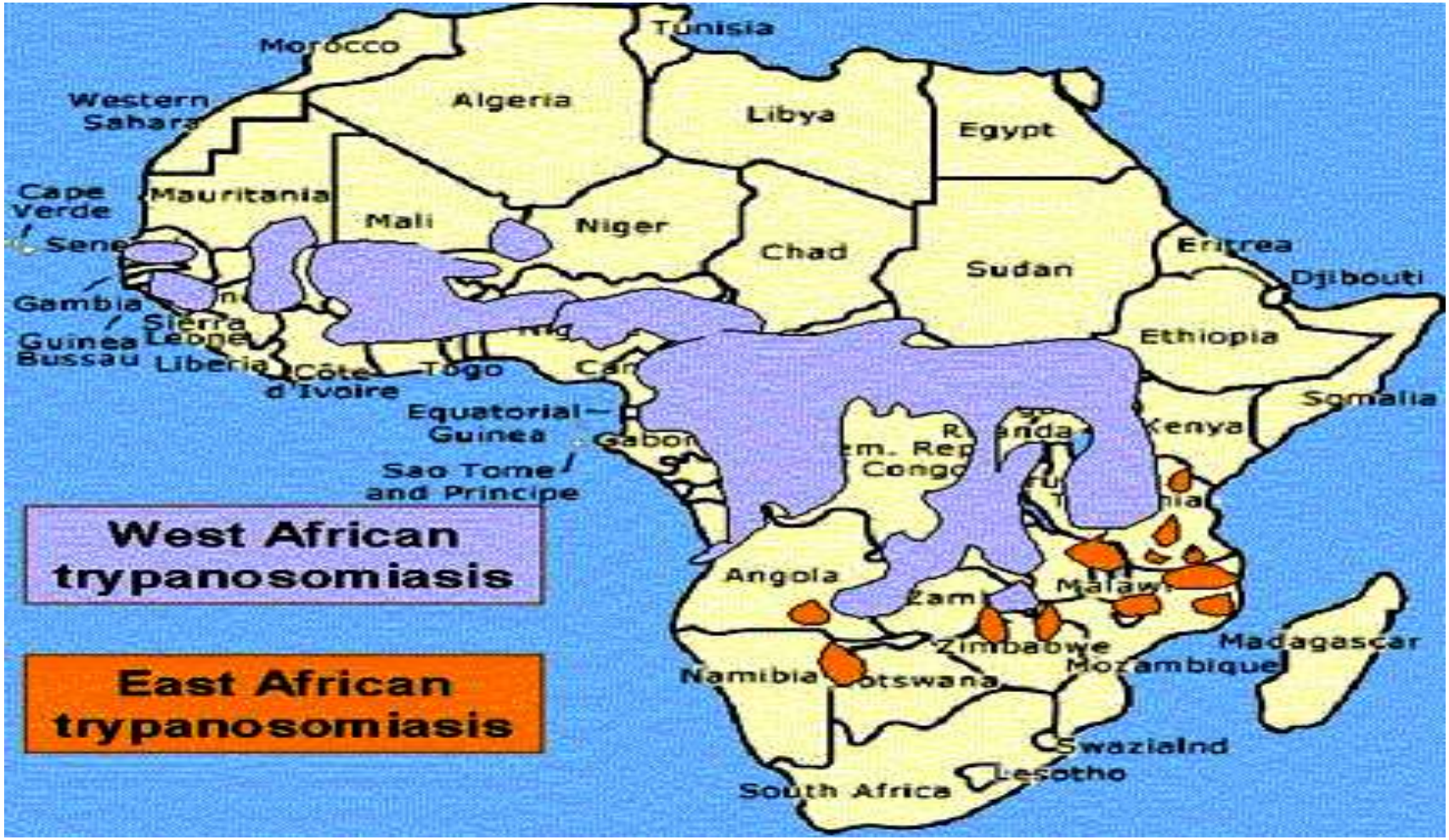
1- long Slender Form (30µ): active motile with free flagellum.

2- Short stumpy Form (15µ): sluggish without free flagellum.

3- Intermediate Form (20µ): with a short free flagellum.



Geographical Distribution of African Trypanosomiasis



Mechanism of disease transmission by *Glossina*

Bite of ♂ & ♀ *Glossina*

Trypomastigotes

Diagnostic stage

3 weeks

Epimastigote

Salivary gland

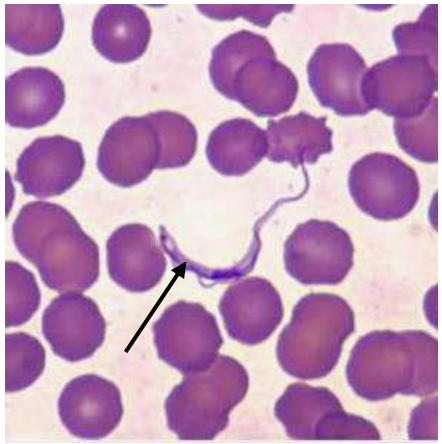
Full of short stumpy metacyclic Trypomastigote

Infective stage

Trypanosoma brucei causing Sleeping Sickness

West Africa

T.brucei gambiense



Less plentiful

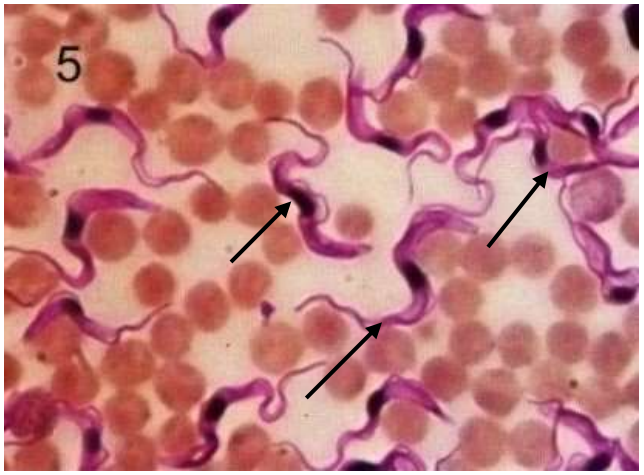
Cannot live in lab animals

Reservoir host: goats, cattle & pigs

Transmitted by: *G.palpalis*

East Africa

T.brucei rhodesiense



More plentiful

Can live in lab animals

Nucleus is shifted posteriorly

Reservoir host: wild game animals

Transmitted by: *G.morsitans*

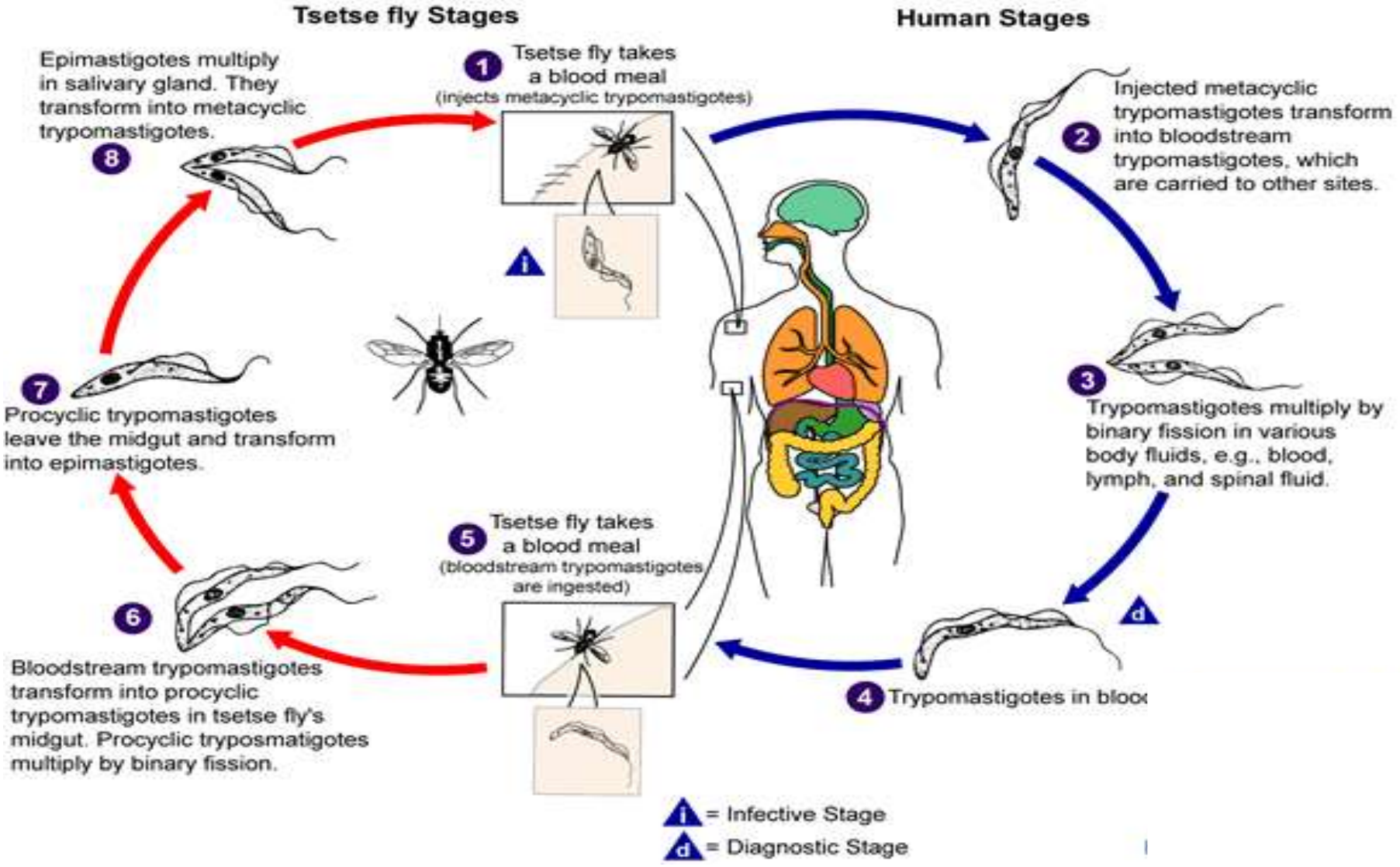
G.palpalis

In West Africa

G.morsitans

In East Africa

African Trypanosomiasis life cycle



Life cycle of *Trypanosoma brucei gambiense* & *T. b. rhodesiense*

Pathogenesis and Clinical Picture

Incubation period (2 weeks)

Trypanosoma chancre (at the site of bite)

Via lymphatics: enlarged lymph nodes

especially posterior cervical region. (Winterbottom's sign)

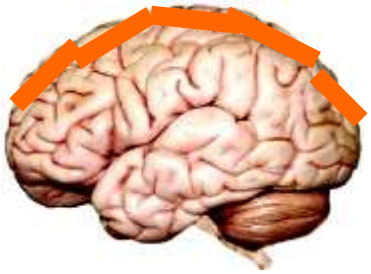
Via blood stream: headache, fever(fluctuating),

muscle & joint pain, irregular erythematous rash.

Invasion of bone marrow (hypoplastic anaemia)

Enlarged liver & spleen, generalized weakness.

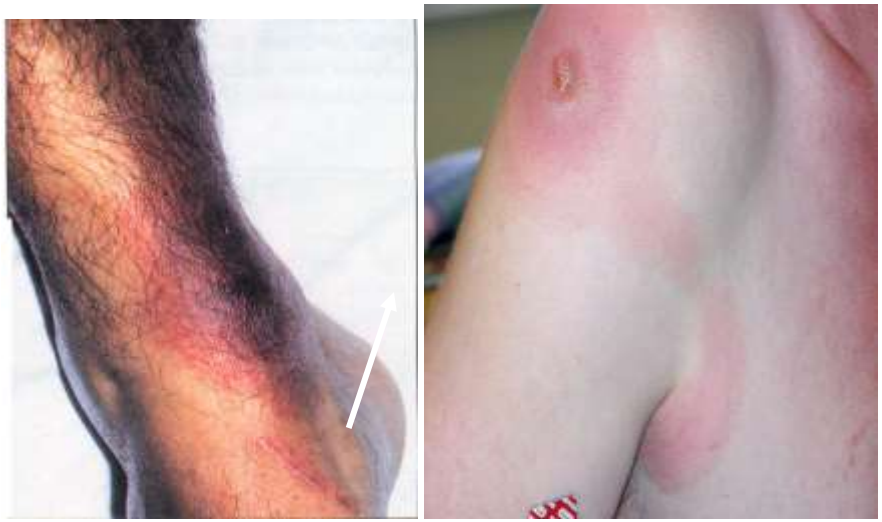
Invasion of CNS: Chronic cases severe headache, mental apathy, slow speech , deep sleep, coma & death



In East African Trypanosomiasis:

Disease runs more rapid & fatal course

Pathogenesis and Clinical Picture



Trypanosoma chancre (ulcer)

Winterbottom sign

Emaciation

Coma before death

Progressive disease may lead to the following C.N.S manifestations:-

1- Insomnia wakefulness 2- Mood changes (dullness / apathy)

3- Motor & Sensory Disorders: (Hyperesthesia / slurred speech / abnormal gait

4- Convulsions 5- Epilepsy

Terminal stage: →

1- Permanent Sleep.
2- 2ry Bacterial infection.
3- Coma & Death.

Diagnosis

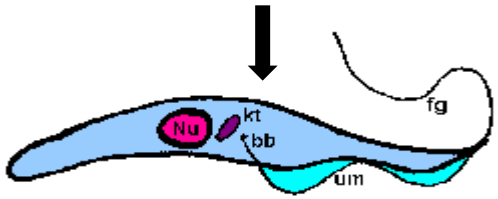
1- Clinical picture

2- Demonstration of trypanosomes:

Polymorphic 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**)

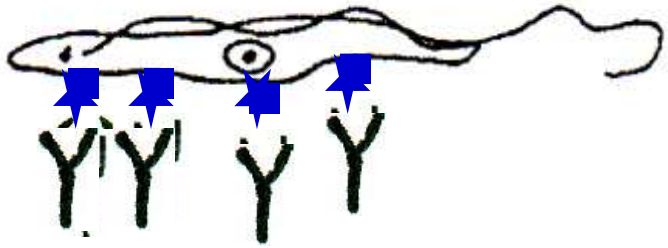


- Animal inoculation

Diagnosis

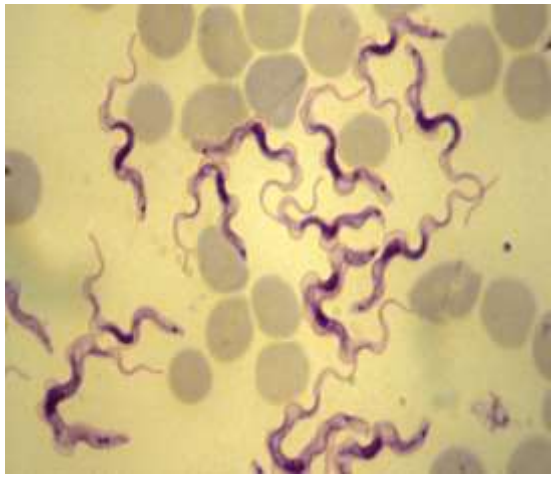
3- Serological test:

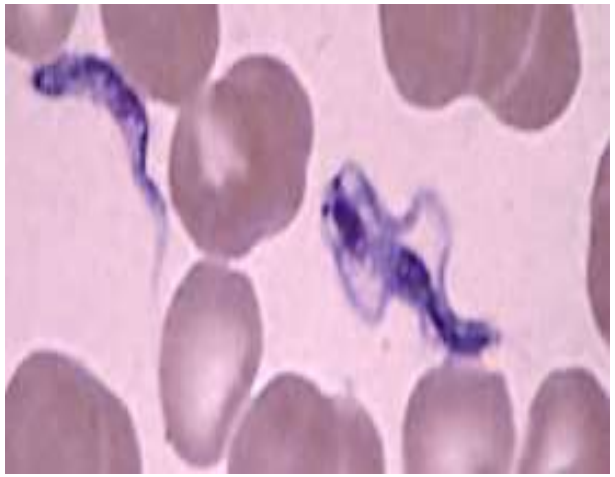
Increased total IgM level in serum due to **antigenic variation** of the surface coat of the parasite.

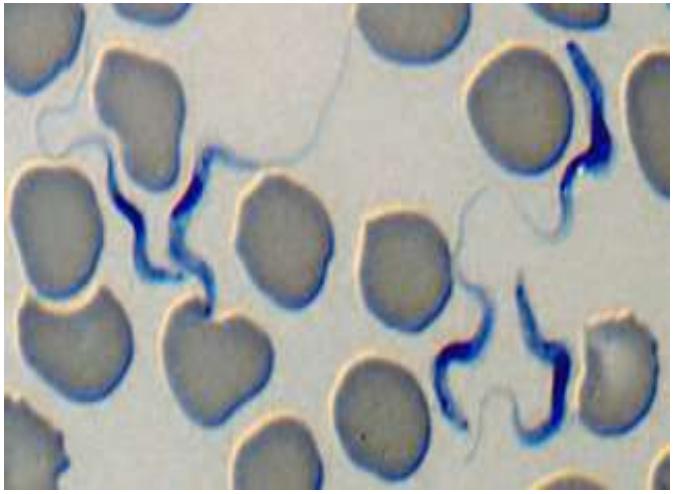


Trypanosome posses genes that code for about 1000 variant forms of their surface glycoproteins (SVG). Switch to a different variant produces a new generation not susceptible to attack by immune factors specific to the previous generation.

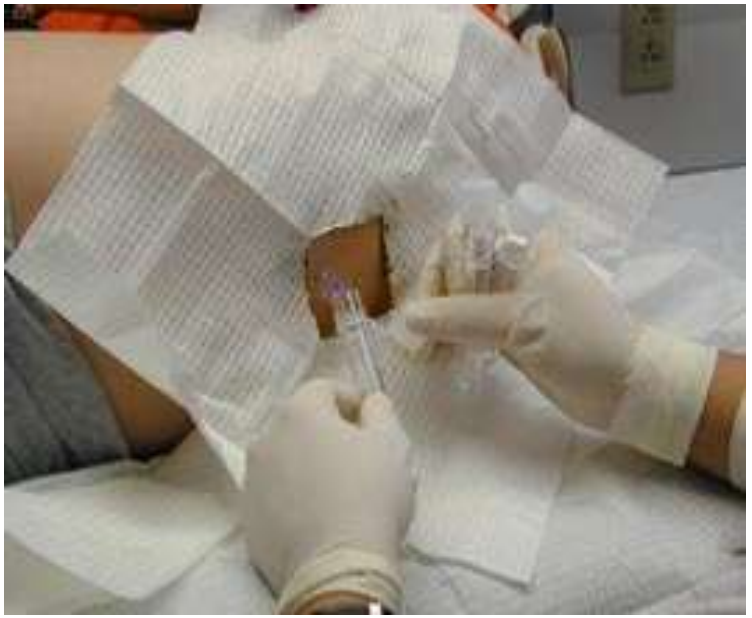
Diagnosis







C.S.F



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.

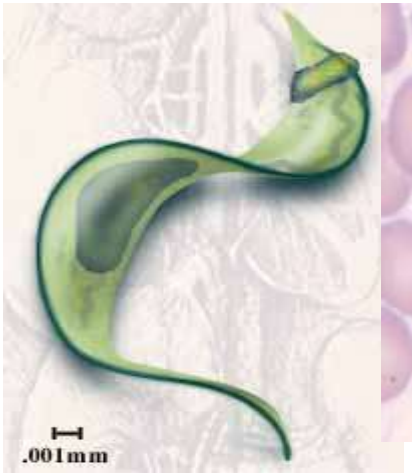
Alternatives include trypars amide combined with suramin.

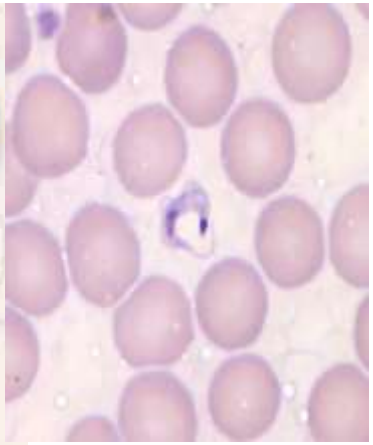
Trypanosoma cruzi causing Chagas' disease

Morphology

Trypomastigote (Monomorphic)

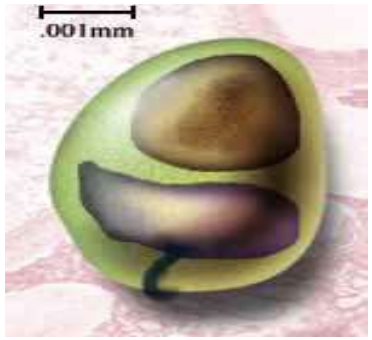
Slender shaped (20µ) – Central nucleus – C or U-shaped –Free flagellum 1/3 body- Large bulging peripheral kinetoplast

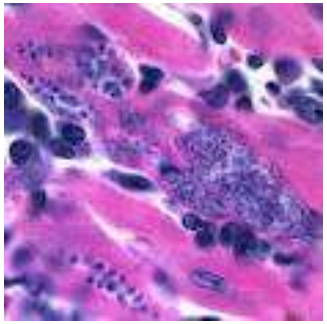




Amastigote

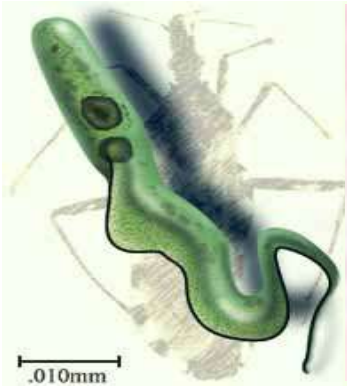
Obligatory intracellular – mainly in cardiac & Skeletal muscles – Brain meninges – Nerve ganglia – cells of GIT …. etc





Epimastigote (Vector only)

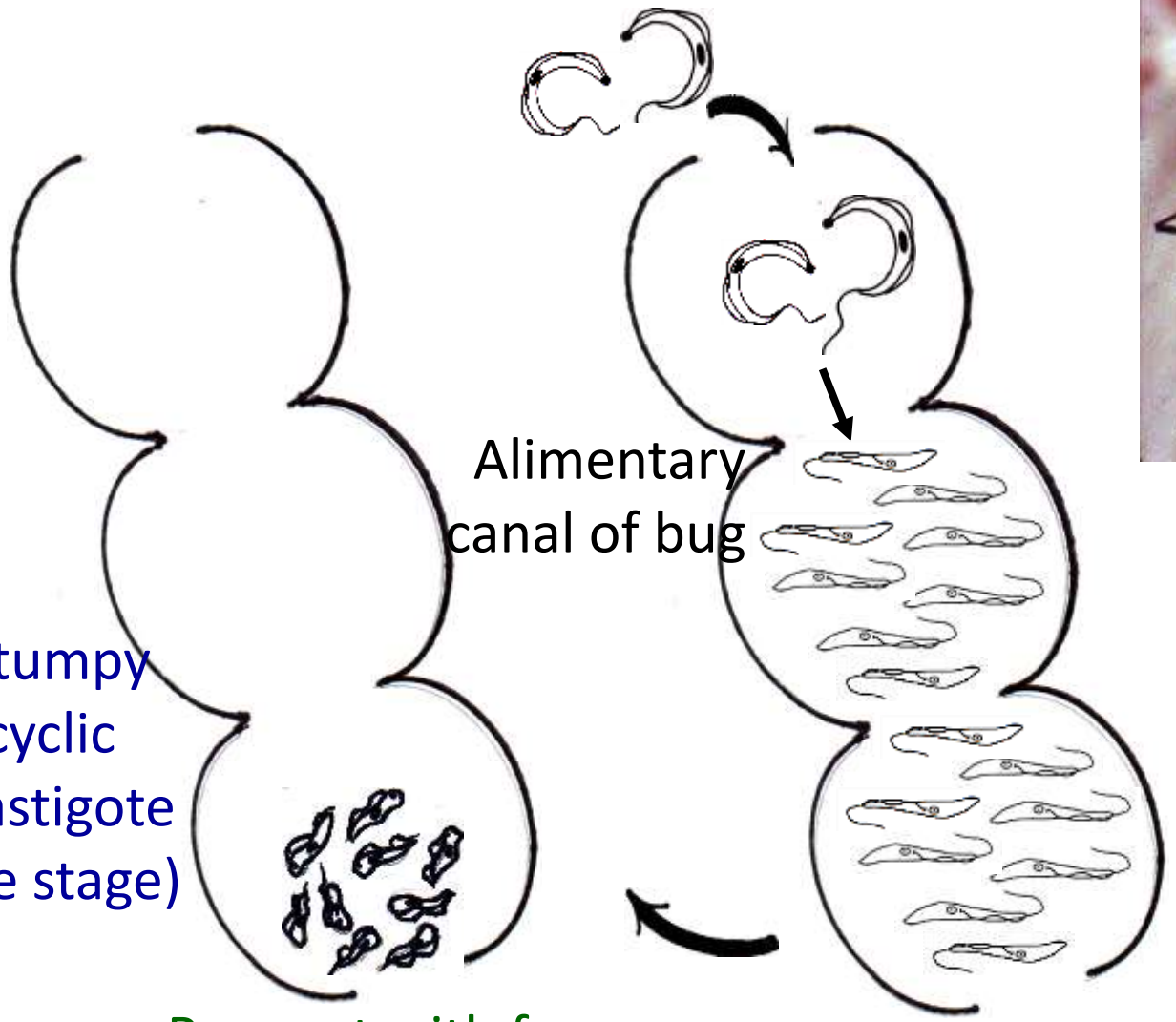
Spindle shape– Kinetoplast anterior to central nucleus– Undulating membrane is short – terminal free flagellum



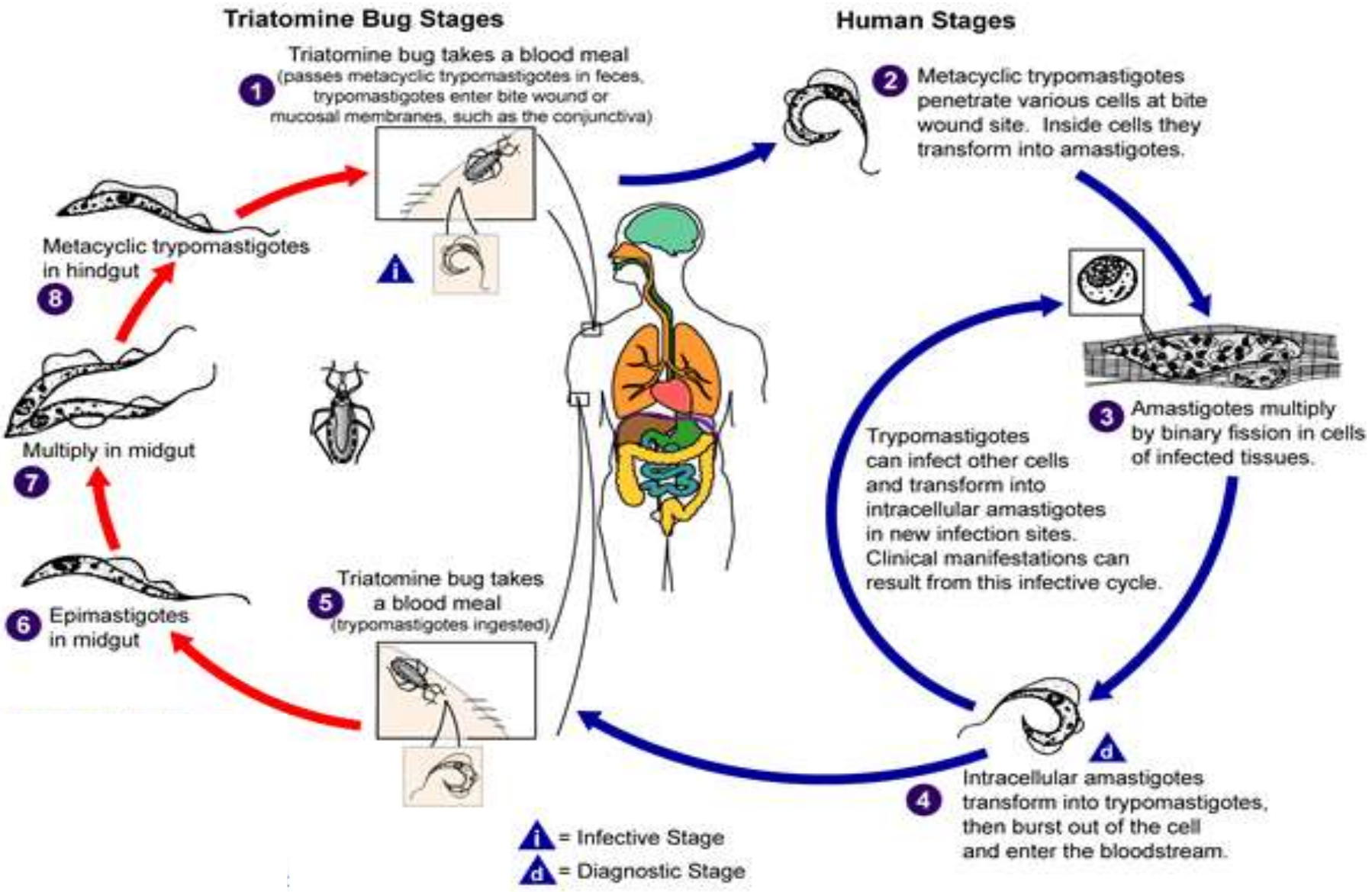
Geographical Distribution of Americam Trypanosomiasis

Mechanism of disease transmission by winged bug

T.cruzi in human blood



LIFE CYCLE OF *Trypanosoma cruzi*



Mode of infection

Mainly by

Contamination of skin abrasion by winged bug faeces

Rarely by

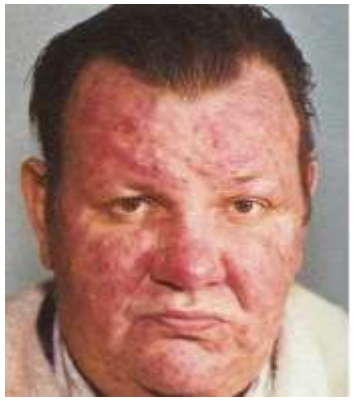
Through infected blood transfusion

Through infected mother’s milk

Through the placenta

Pathogenesis and Clinical Picture

I- Acute Form



Chagoma occurs at the site of bite.

Parasite reaches regional lymph nodes

To ↓

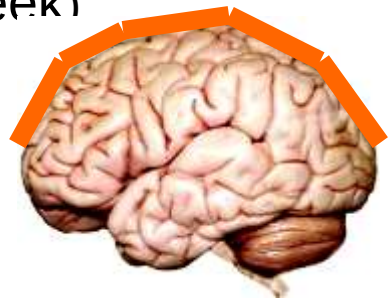
Blood

To ↓

Organs and tissues

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

Romana's sign (Unilateral conjunctivitis appear suddenly togetherwith oedema of upper & lower eye lids & cheek)



Meningoencephalitis, heart failure

Death or pass to **C**hronic form

Pathogenesis and Clinical Picture

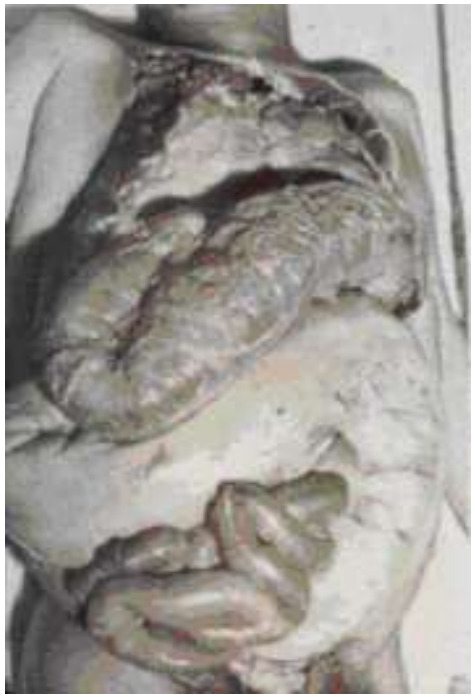
II- Chronic form

Parasite produces antigens similar to patient's self antigens:

The body produces auto-antibodies that cause damage to:

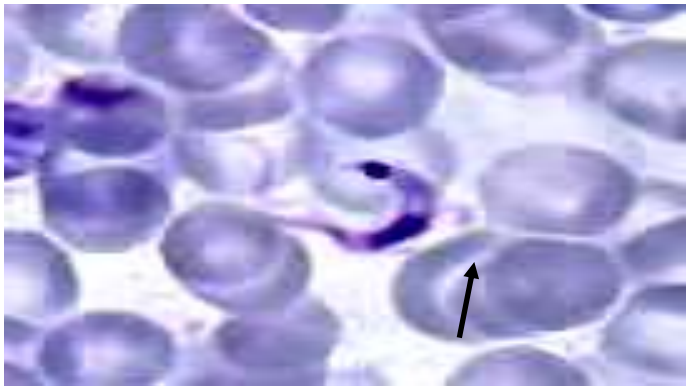
- ❑ Heart muscle fibres:
  congestive heart failure.
- ❑ Oesophageal muscle fibres:
  megaoesophagus and dysphagia.
  Destruction of Auerbach's plexus
- ❑ Colon muscle fibres:
  megacolon and constipation.
- ❑ CNS or thyroid gland
  Exacerbation of infection in immunosuppressed patients.

Amastigote form of *T.cruzi*



Diagnosis

Finding the parasite in:



Blood film (C-shaped *T.cruzi*)

Biopsy from lymph node, liver or spleen (amastigotes)

Culture (Epimastigotes)

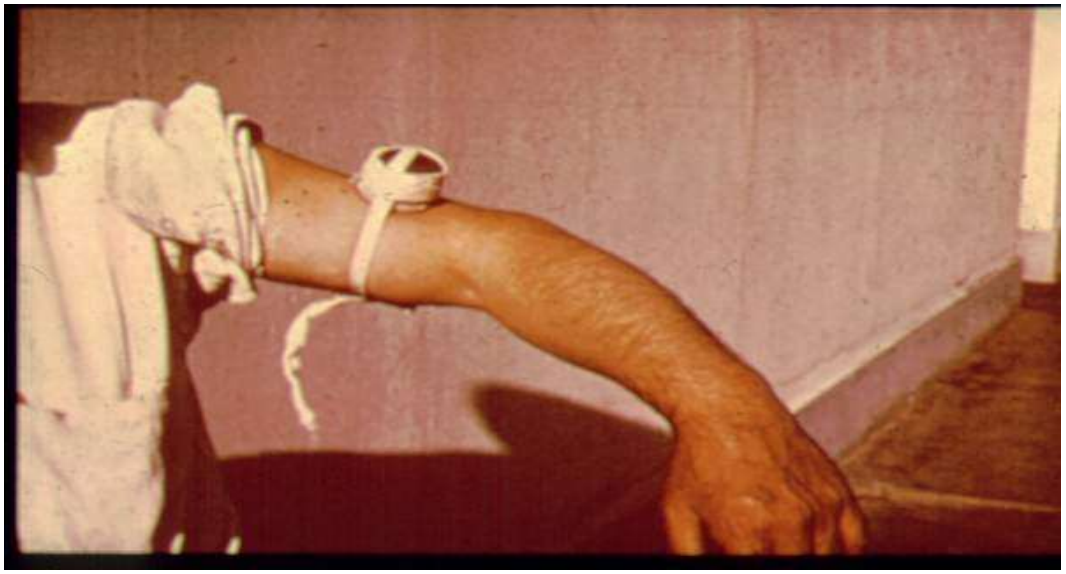
Xenodiagnosis

Serological tests

Cruzin test (I.D.)

Molecular techniques

Diagnosis (Xenodiagnosis)



Highly efficient – demonstrate low level of parasite in blood

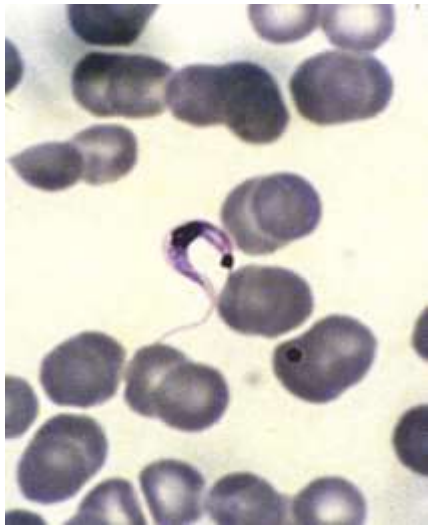
Method:

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.

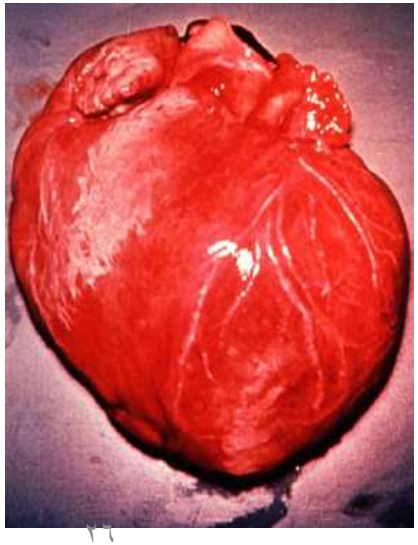
Diagnosis

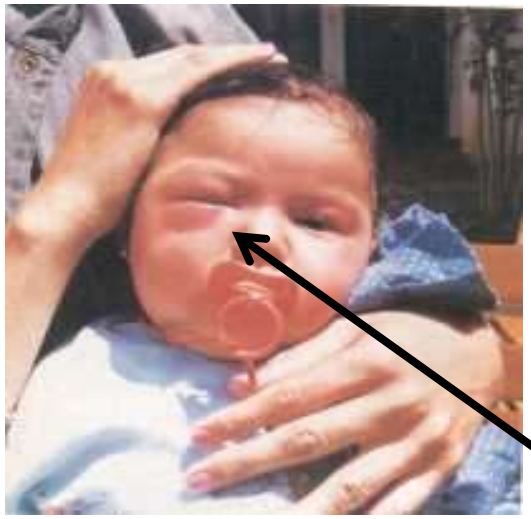
Amastigote

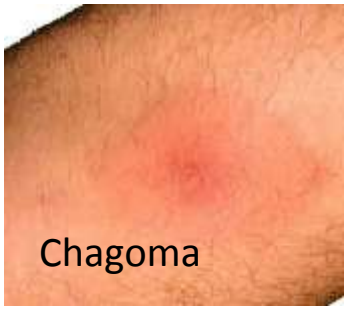
Trypomastigote



Winged Bug

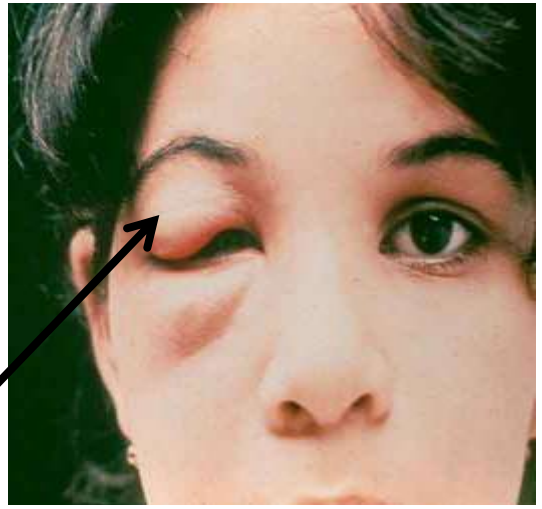






Chagoma

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