

# Syphilis

## Urogenital Tract Module

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# Outlines

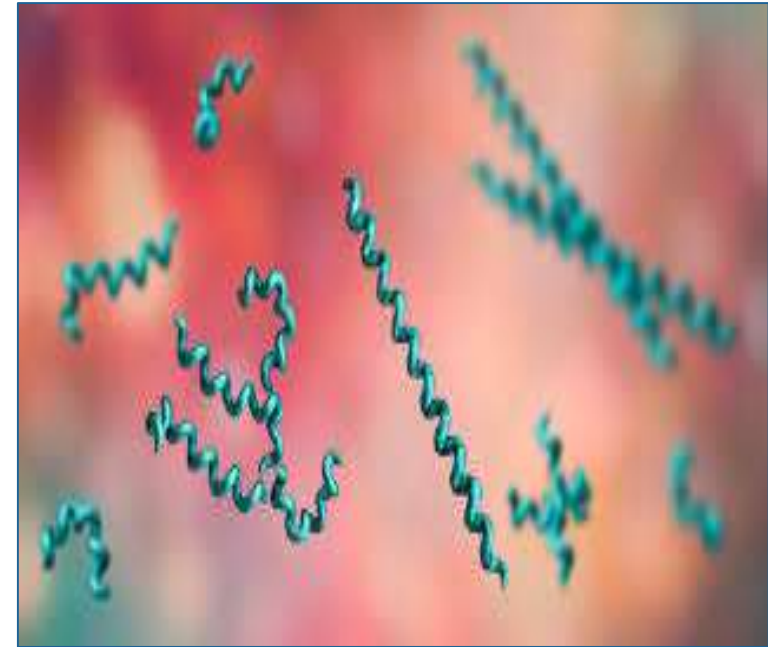
- Introduction
- Etiology: pathogen, pathophysiology, and transmission
- Clinical features:
  - Primary syphilis
  - Secondary syphilis
  - Latent, and tertiary syphilis
  - Congenital syphilis
- Diagnosis
- Treatment

# Syphilis: Introduction

- Syphilis is a predominantly bacterial sexually transmitted infection caused by the spirochete *Treponema pallidum*.
- **Epidemiology:**
  - Worldwide annual new cases: 11 million
  - Incidence in the United States is rising.
  - Men > women (8:1) (Homosexual)
  - Most common age group: 20–29 years old

# Syphilis: Etiology- Pathogen

- **The causative organism:** *Treponema pallidum*
- **Basic features** of Treponema species:
  - Gram negative Spirochete (spiral shaped) bacteria
  - Microaerophilic
  - Cannot be grown in culture

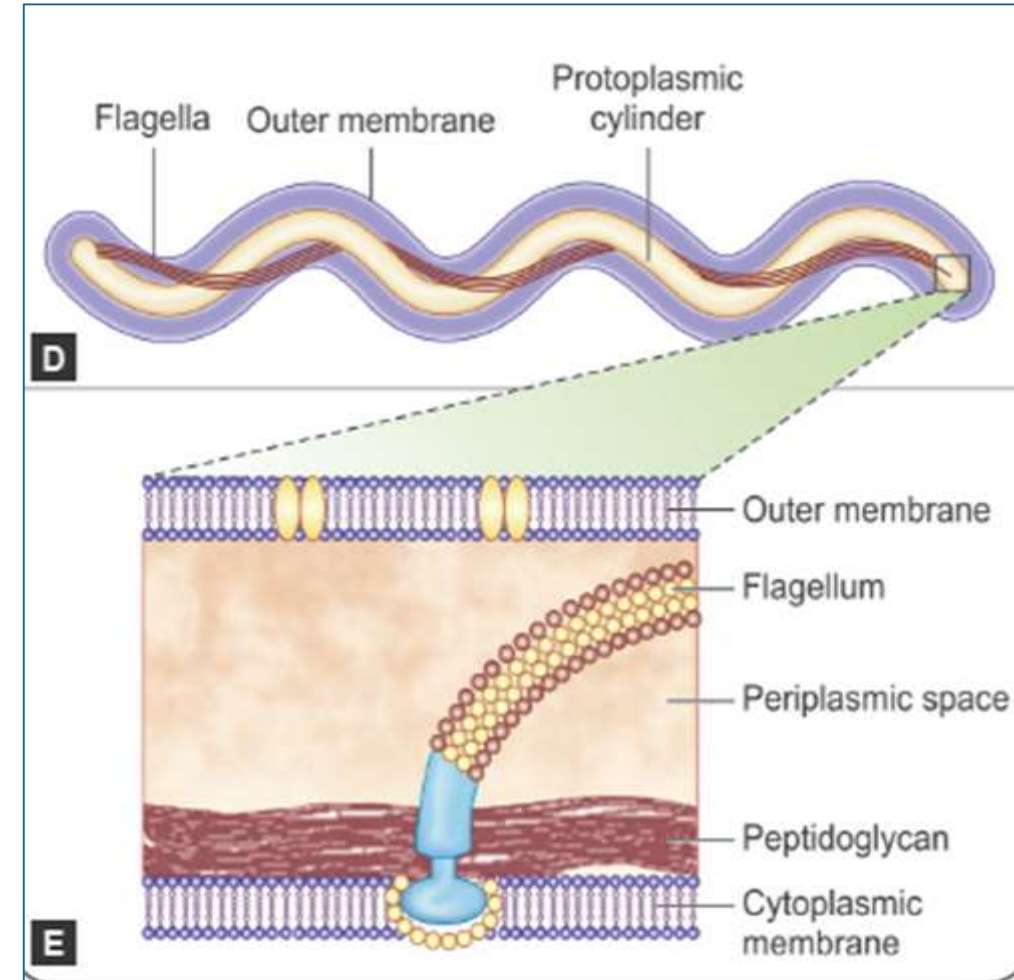


# Syphilis: Etiology- Pathogen

- **Basic features** of Treponema species:
  - Endo-flagellated:
    - 3 flagella originate from each end.
    - Located in the periplasmic space
  - Classic motility: Rotates rapidly about its longitudinal axis

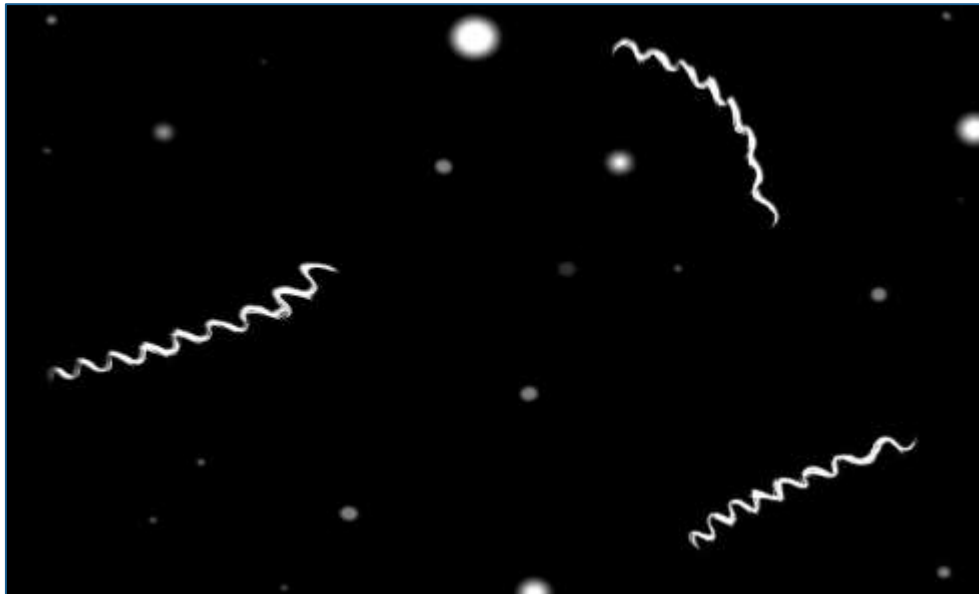


**Corkscrew motility**



# Syphilis: Etiology- Pathogen

- **Basic features** of Treponema species:
  - Visualization: Dark-field microscopy because its too slender to be visualized using Gram or Giemsa stain



# Syphilis: Etiology- Pathophysiology

- **Transmission:**

- Humans are the only reservoir, and transmission is through human-to-human contact.
- Sexual contact
- Direct contact with infectious lesions
- Vertical (congenital syphilis)

# Syphilis: Etiology- Pathophysiology

- *T. pallidum* adheres to skin or mucosal membranes → hyaluronidase production → tissue invasion
- The organism **coats itself in the host's fibronectin** → prevents recognition and phagocytosis by the immune system → development of the chancre (initial ulcerative lesion)
- Eventual local immune control → resolution of chancre
- During the primary period, some spirochetes move into local lymph nodes.
- Later stages:
  - Spirochetes multiply and disseminate through the bloodstream → invade other organs and tissues
  - Host immune-inflammatory response → systemic clinical manifestations



# Syphilis: Etiology- Pathophysiology



- **Obliterative endarteritis**—is a hallmark pathological finding in syphilitic lesions.

*T. pallidum* shows a **marked preference** for invading the **endothelium of arterioles and capillaries**.



The host mounts an intense perivascular immune response



Thickening of the endothelial lining, Proliferation of the intima, Narrowing or occlusion of the vessel lumen, and **Ischemia of surrounding tissue**

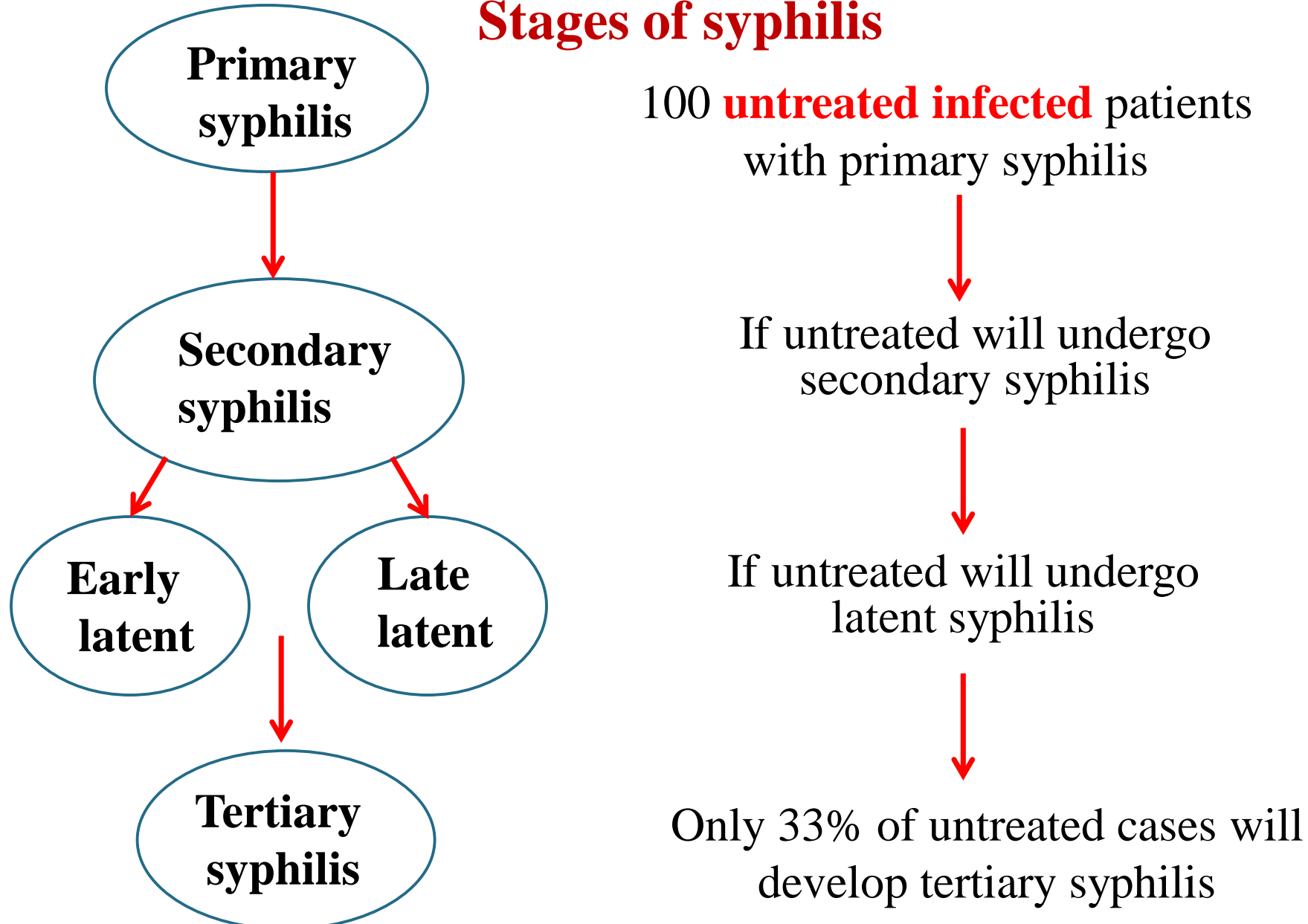
# Syphilis: Etiology- Pathophysiology

- Syphilis is a multistage disease. It has 4 clinical stages: primary, secondary, latent, and tertiary, each with different pathological mechanisms and tissue involvement.

# Pathogenesis and Clinical Manifestations

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## Stages of syphilis

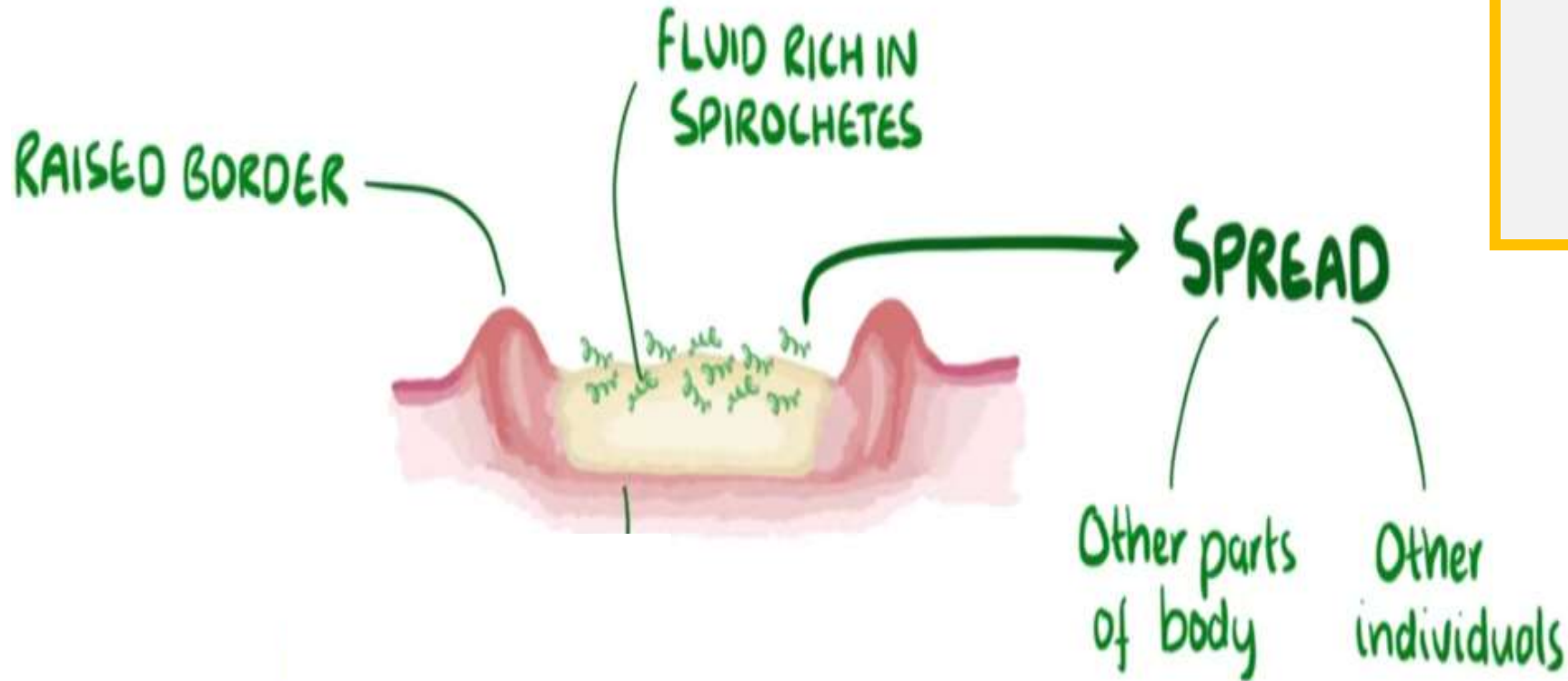


# Syphilis: Clinical features- Primary syphilis

- Localized disease
  - 90–95%: genital primary syphilis
  - 5–10%: extragenital primary syphilis (most commonly, the oral cavity, finger, and anus or perianal region)
- Primary lesion (**chancre**):
  - Typically starts out as a solitary, raised papule (usually on the genitals)
  - Evolves into a painless, firm ulcer with indurated borders and smooth base
  - The primary lesion appears at the site of inoculation, usually persists for 4–6 weeks, and then heals spontaneously.
- Nontender regional lymphadenopathy (e.g., involvement of the inguinal lymph nodes in genital primary syphilis)

# Syphilis: Clinical features- Primary syphilis

## Chancre



**P**rimary

=

**P**ainless

# Syphilis: Clinical features- Primary syphilis

## Chancre



# Syphilis: Clinical features- Secondary syphilis

- Disseminated disease due to the **systemic spread** of the spirochetes, inducing an immunologic reaction
  - Begins approx. 2–12 weeks after primary infection and typically lasts 2–6 weeks
1. Constitutional symptoms: fever, malaise, myalgias, headaches, anorexia, weight loss, nausea
  2. Generalized nontender lymphadenopathy

**Secondary**  
=  
**Systemic**

# Syphilis: **Clinical features-** **Secondary syphilis**

## 3. Polymorphic rash (all are highly contagious)

### **A. Generalized rash:**

- Nonpruritic, Macular, papular, or nodular. Scaly or smooth
- Red, reddish-brown, or copper colour
- Involves the trunk, extremities, palms, and soles

### **B. Superficial mucosal erosions (mucous patches)**

- Commonly involve the oral or genital mucosa.
- The typical mucous patch is a **painless silver-gray erosion** surrounded by a red periphery.



# Syphilis: Clinical features- Secondary syphilis



# Syphilis: Clinical features- Secondary syphilis

3. Polymorphic rash (all are highly contagious)

## C. Condylomata lata

- **In warm, moist**, intertriginous folds (commonly the perianal region, vulva, and scrotum), and on oral mucosa papules can enlarge to produce **painless broad, moist, pink or gray-white, highly infectious lesions** (condylomata lata).
- Cauliflower-like mass
- In 10% of patients with secondary syphilis



# Syphilis: Clinical features- Latent syphilis

- **Pathophysiology:** *T. pallidum* is **not eradicated**; instead, it remains **dormant** in certain tissues. The immune system keeps the bacteria in check but cannot clear them.
- **No clinical symptoms**, despite **seropositivity**
- The disease can resolve, relapse with skin/mucosal lesions, or progress to tertiary syphilis.
- May last months, years, or even for the entire life of the patient
- Classified based on the duration since initial infection:
  - Early: < 1 year after initial infection
  - Late: > 1 year after initial infection

# Syphilis: Clinical features- Tertiary syphilis

## “Immune-Mediated Destruction”

- Seen in 33% of untreated cases
- Occurs 1–30 years after the initial infection
- Caused by **delayed-type hypersensitivity reactions** to *T. pallidum* antigens, not by direct spirochete activity.

### A. Gummas:

- Soft, solitary, granulomatous lesions with central necrosis
- Variable in size
- Destructive (leaves scars)
- Occurs on skin, bones, or organs
- Bone involvement may cause deep, boring pain



# Syphilis: Clinical features- Tertiary syphilis

## B. Cardiovascular syphilis:

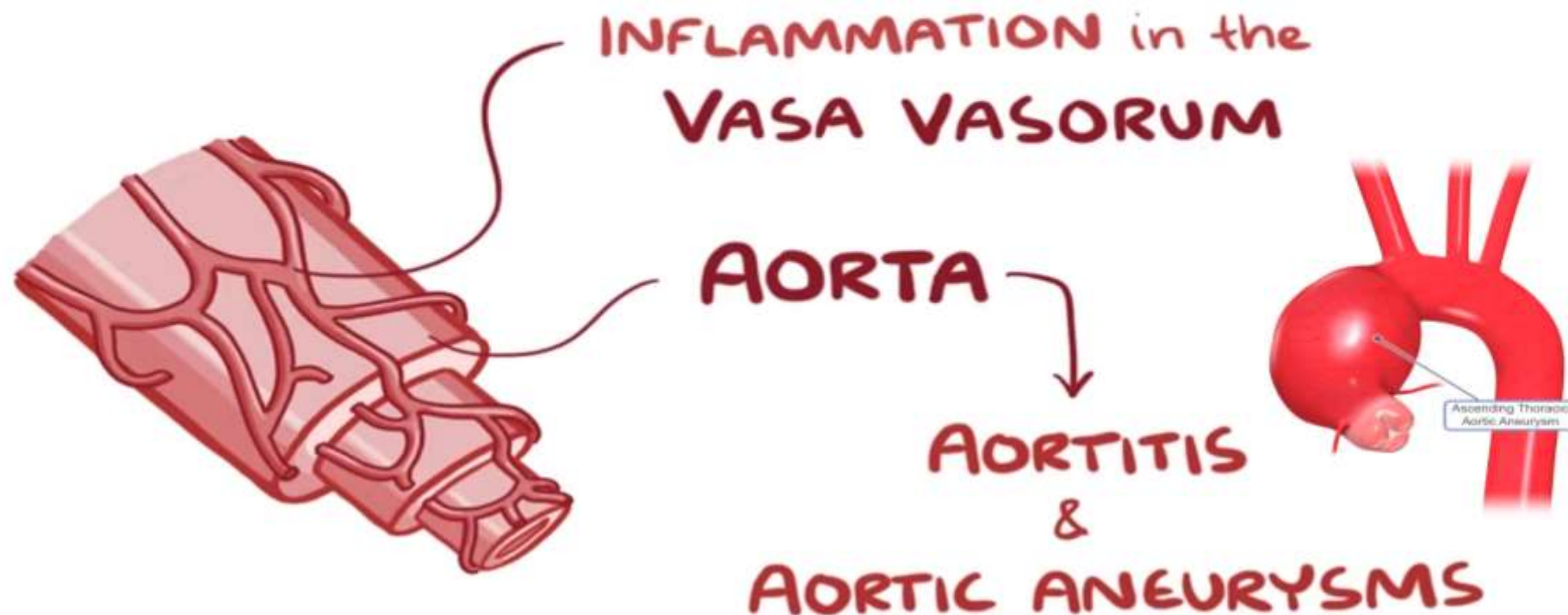
- A consequence of Treponema-induced vasculitis of the vasa vasorum of the large vessels (especially the aorta), resulting in vessel wall atrophy, and thereby, aneurysm formation
- Findings: Aortitis, ascending aortic aneurysm (thoracic aortic aneurysm), and aortic root dilation → aortic regurgitation



# Syphilis: Clinical features- Tertiary syphilis

## CARDIOVASCULAR SYPHILIS

### – ENDARTERITIS



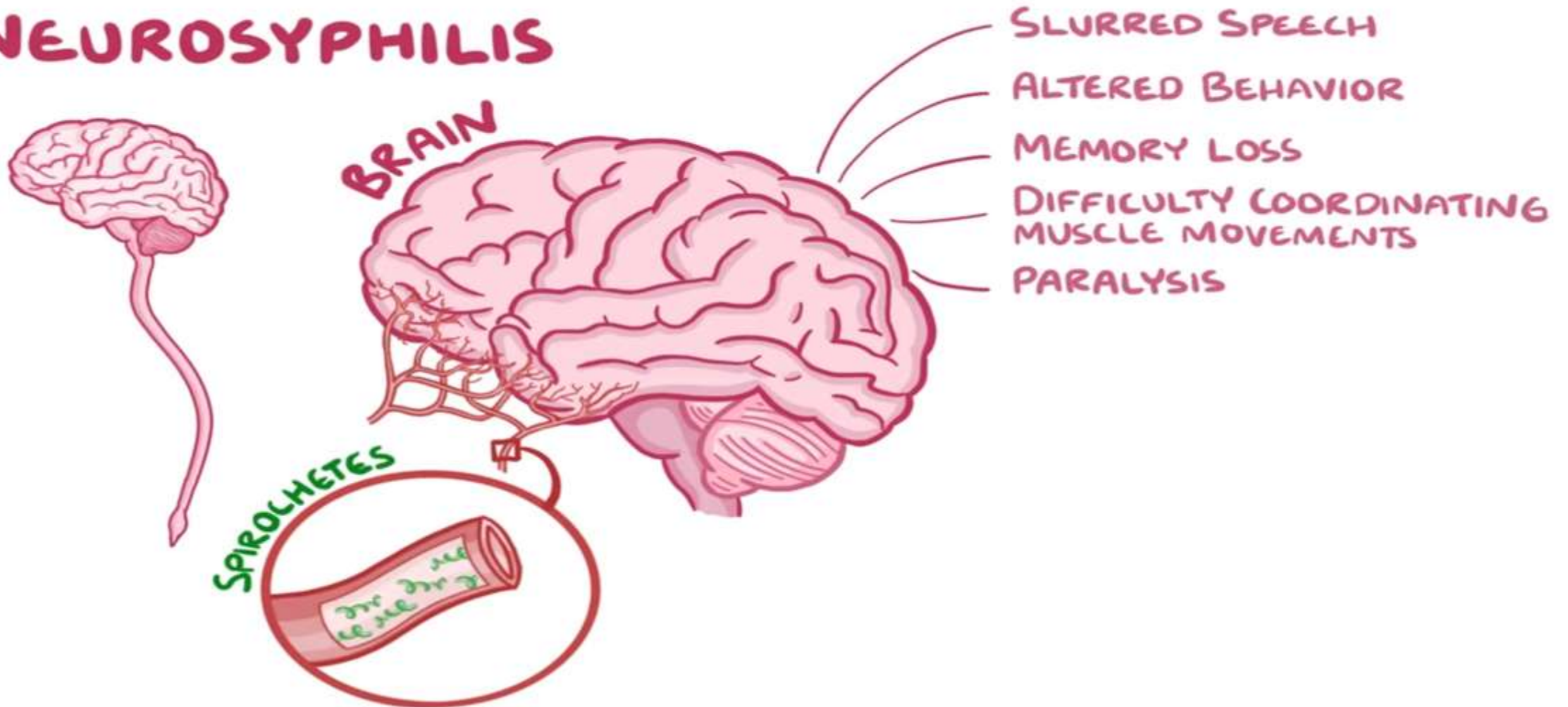
# Syphilis: Clinical features- Tertiary syphilis

## B. Neurosyphilis:

- Neurosyphilis is an infection with treponemal invasion of the CNS (e.g., meninges, cerebral vasculature and/or parenchyma).
- Clinical features: highly variable
  - Acute syphilitic meningitis
  - Subacute stroke, meningitis, and/or cranial nerve disorders
  - **Paretic Neurosyphilis** (also called **general paresis of the insane**): *T. pallidum* invades the cerebral cortex, particularly the frontal and temporal lobes.

# Syphilis: Clinical features- Tertiary syphilis

## NEUROSYPHILIS



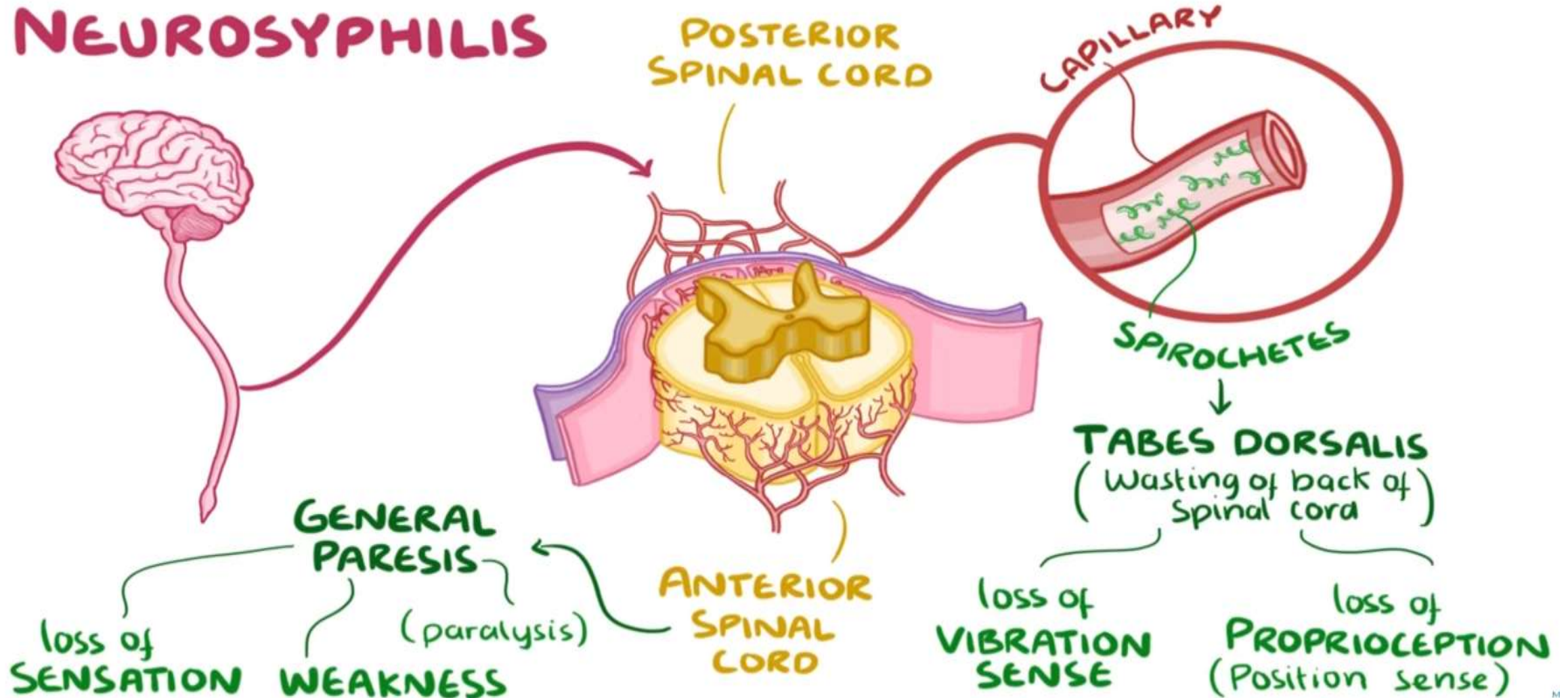


# Syphilis: Clinical features- Tertiary syphilis

## **B. Neurosyphilis:**

- Tabes dorsalis: Demyelination of the dorsal columns and the dorsal roots
  - Loss of vibratory sense and proprioception, Loss of sensation, predominantly in the lower extremities.

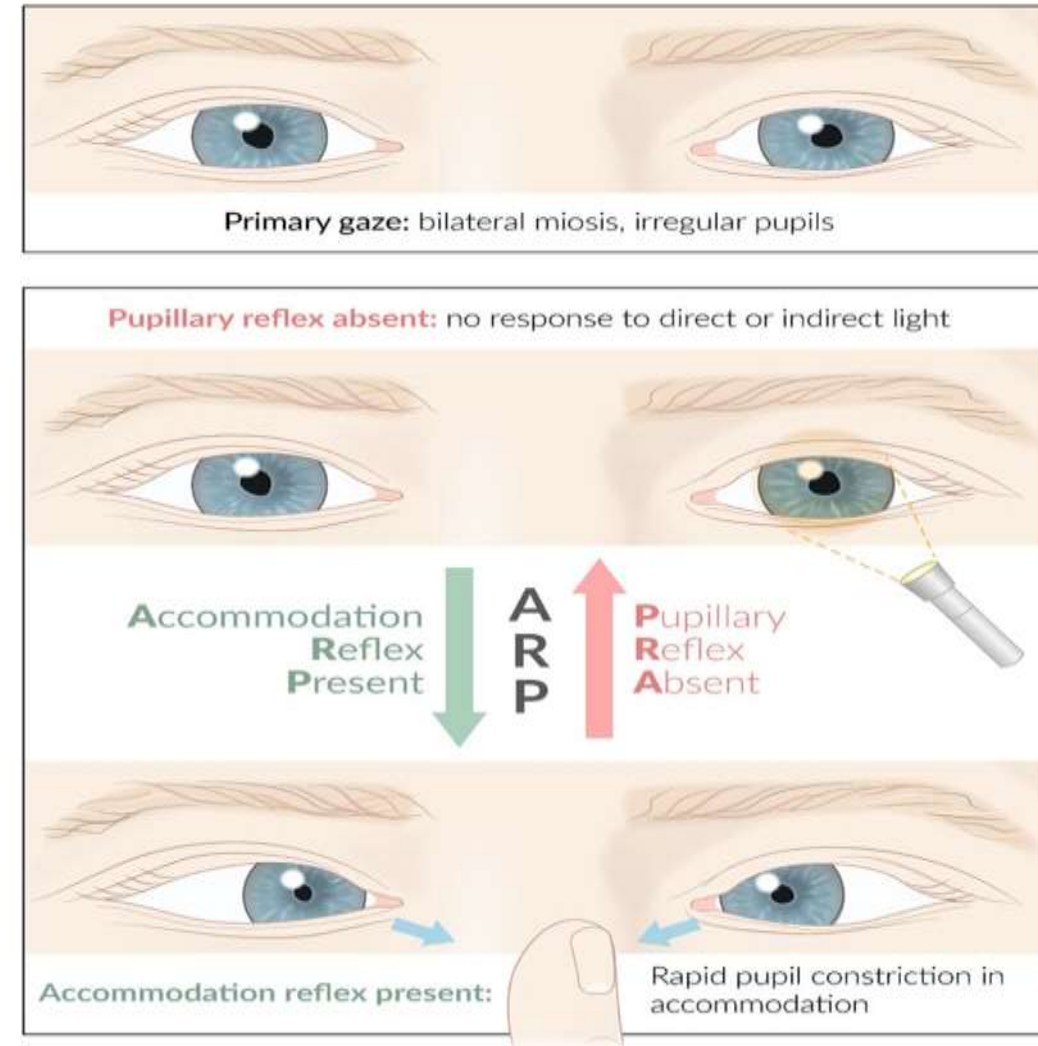
# Syphilis: Clinical features- Tertiary syphilis

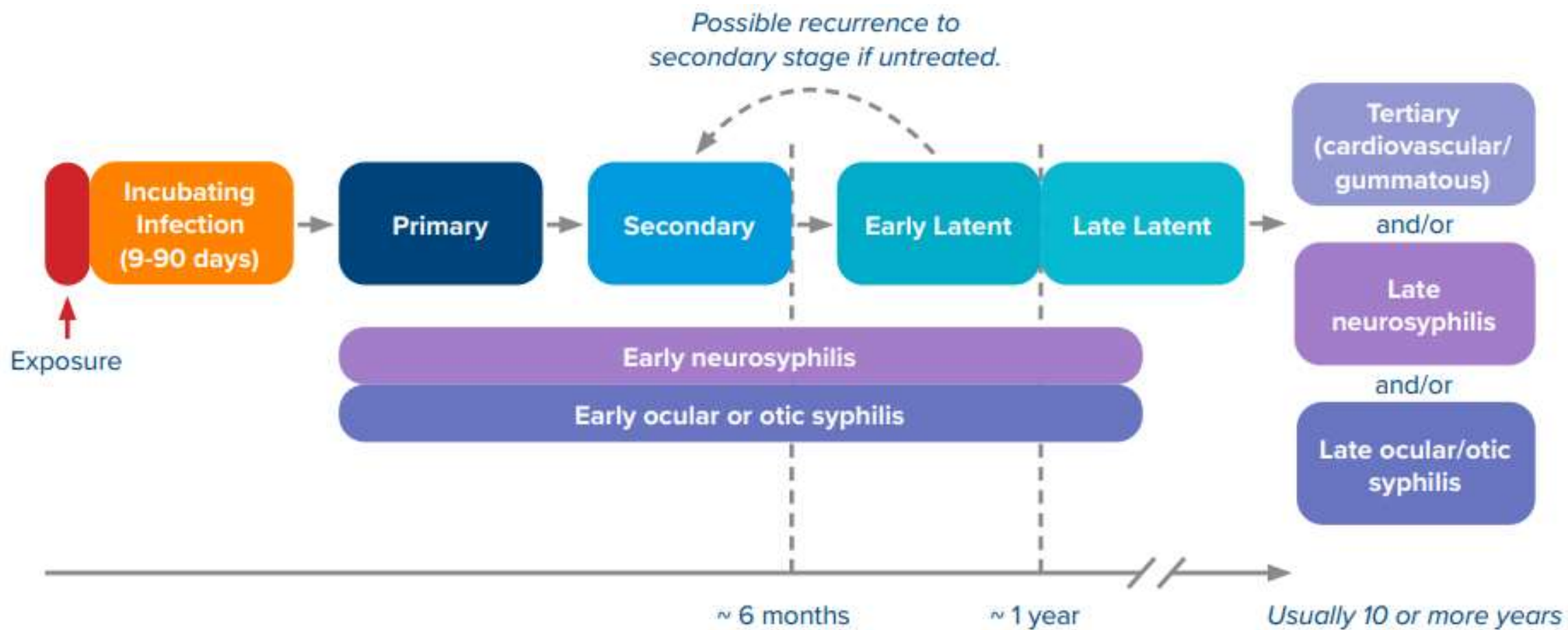


# Syphilis: Clinical features- Tertiary syphilis

## B. Neurosyphilis:

- **Argyll Robertson pupil:** bilateral small pupils that **fail to constrict in response to bright light** but exhibit constriction during near vision tasks





Adapted from New York City Department of Health and Mental Hygiene and New York City STD Prevention

# Syphilis: **Clinical features-** **Congenital syphilis**

- **Transplacental transmission** from infected mother
- Clinical features of congenital syphilis:
  - In utero syphilis: Miscarriage, Stillbirth
  - Early congenital syphilis (onset < 2 years of age)
    - Hepatomegaly and jaundice
    - Rhinorrhoea with white or bloody nasal discharge (also called “snuffles”)
    - Maculopapular rash on palms and soles

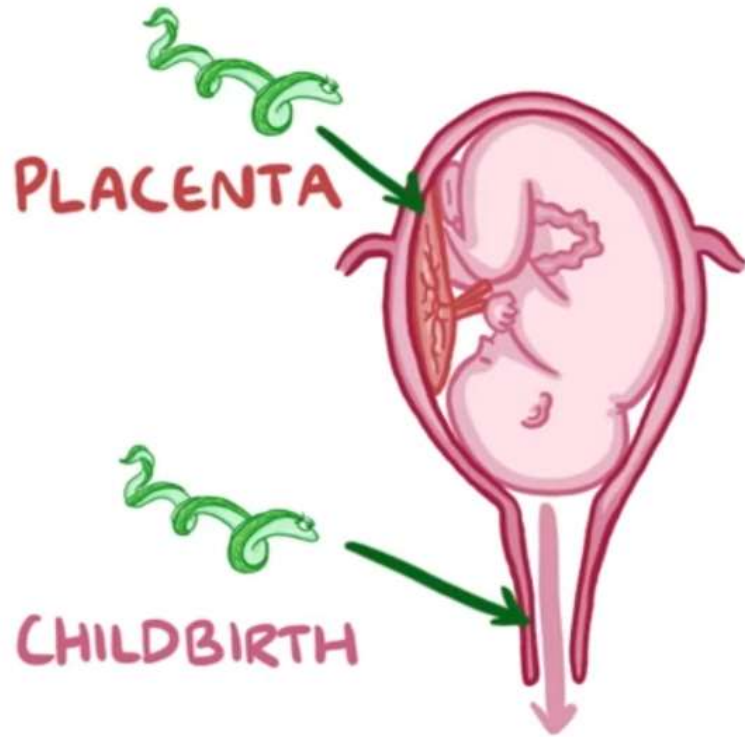


# Syphilis: Clinical features- Congenital syphilis

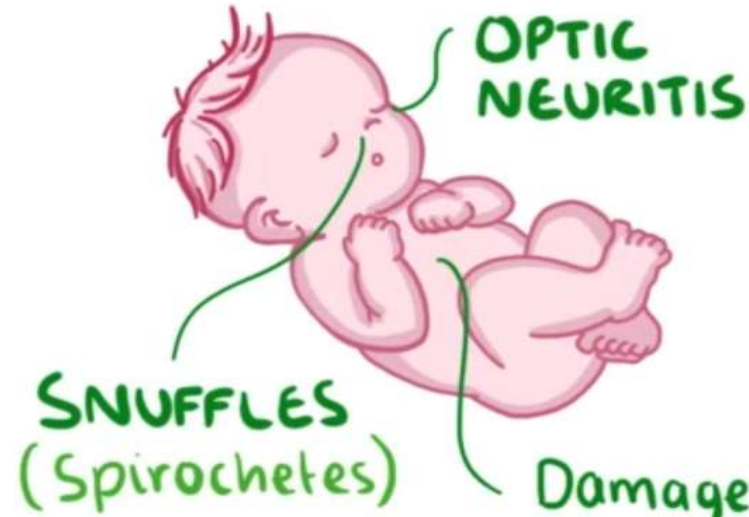
- Late congenital syphilis (onset > 2 years of age)
  - Typical facial features: saddle nose, frontal bossing
  - Dental findings: Hutchinson's teeth (notched, widely spaced teeth)
  - Saber shins: An anterior bowing of the tibia
  - Hearing loss



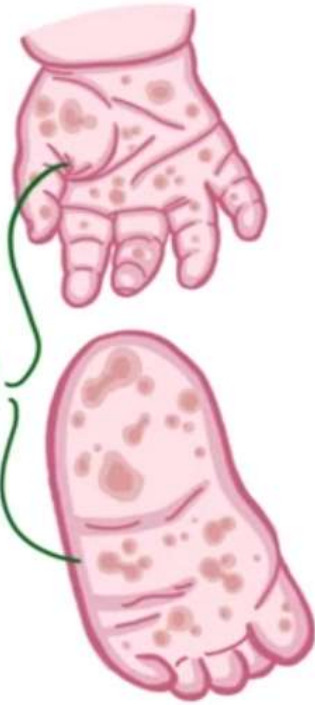
# CONGENITAL SYPHILIS



## EARLY DISEASE (First 2 years)



STILLBORN  
DYING IN WOMB  
MACULOPAPULAR  
RASH



## LATE DISEASE (> 2 years)



# Syphilis: **Diagnosis**

## **Direct detection:**

- Darkfield microscopy: visualization of motile spirochetes on wet mount under a dark-field microscope
- Direct fluorescent antibody for *T. pallidum*: visualization of immunofluorescent antibodies on the specimen
- Nucleic acid amplification (e.g., PCR)



# Syphilis: **Diagnosis**

## Serological studies

### A. Nontreponemal testing

- Use for screening purposes since the tests are sensitive, but not specific.
- Detects anticardiolipin antibodies
- Options: Rapid plasma reagin test, VDRL

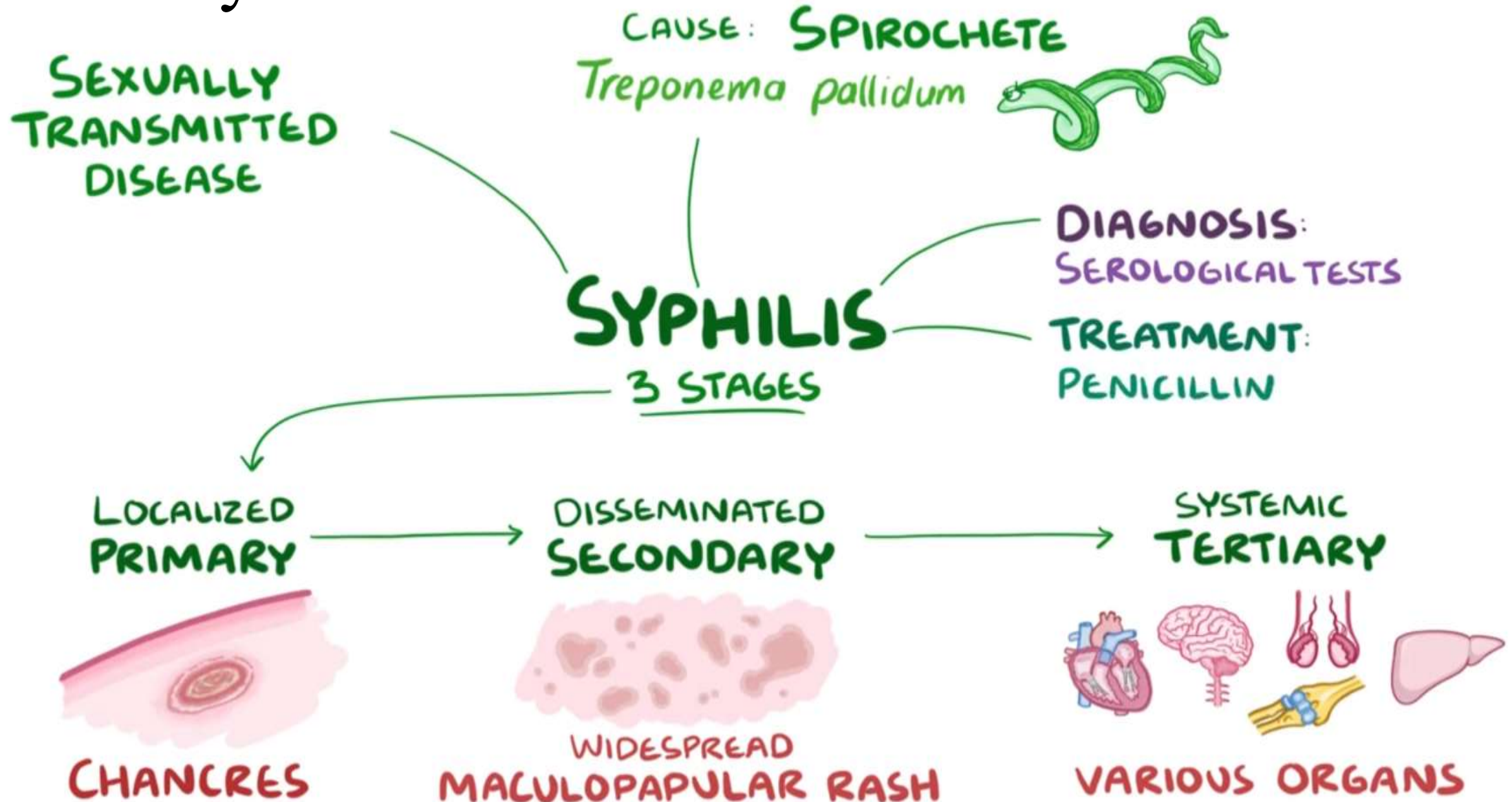
### B. Treponemal testing

- Confirmatory tests that detect antibodies to Treponema antigens
- *T. pallidum* particle agglutination test (TPPA)
- *T. pallidum* enzyme immunoassay (TP-EIA)

# Syphilis: Treatment

Penicillin G is the first-line therapy for all patients.

# Summary...



Thank you

