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ENCEPHALITIS

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ENCEPHALITIS

ENCEFPHALITIS -

damage of the brain tissue with :

- inflammatory
- Inflammatory - allergic
- allergic and
- toxic

signification

CLASSIFICATION

I. Histological criterion :

- polioencephalitis (grey tissue alteration)
- leucoencephalitis (white tissue alteration)
- panencephalitis (white +grey)

II. Morphopathological criterion :

- infiltrative - proliferative
- hemorrhagic

CLASSIFICATION

III. Pathogenic criterion :

- primary
- secondary
- para- and postinfectious

IV. Evolutional criterion:

- acute
- subacute
- chronic

CLASSIFICATION

V. Etiological criterion :

- viral
- bacterial
- fungal
- parasitic

CLASIFICAREA ENCEFALITELOR

VI. Epidemiological and seasonal criterion :

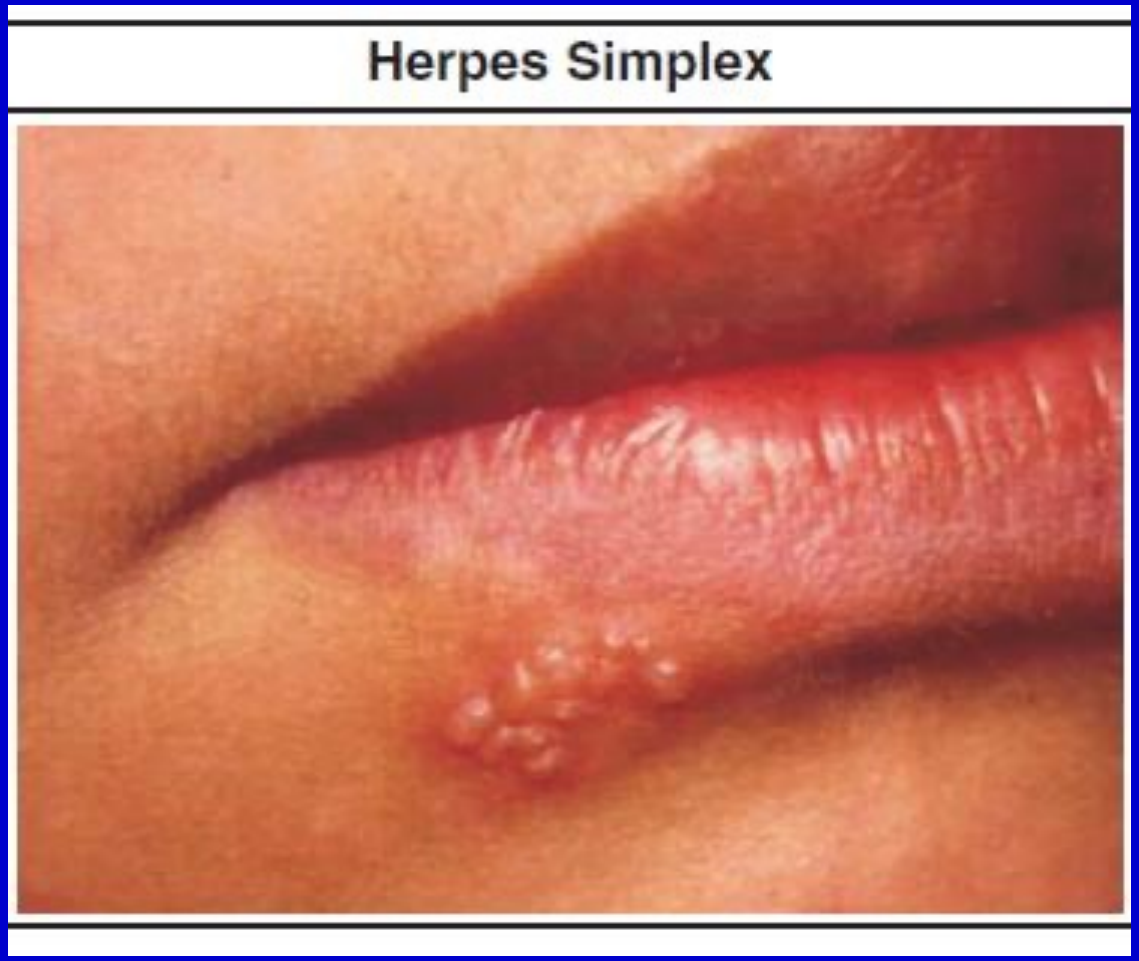
- **seasonal**
- **non-seasonal**
- **endemic**
- **epidemic**
- **sporadic**

Herpes simplex encephalitis

Etiology. **HSV-1 and HSV-2.**

Biological basis. Primary HSV infections are usually oral (gingivostomatitis), corneal (keratitis), or genital (penile, vaginal, cervical). Once transmitted, the virus then enters peripheral sensory nerves, travels by retrograde axonal transport to the neuronal cell body, and establishes a latent infection that can persist for the life of the host.

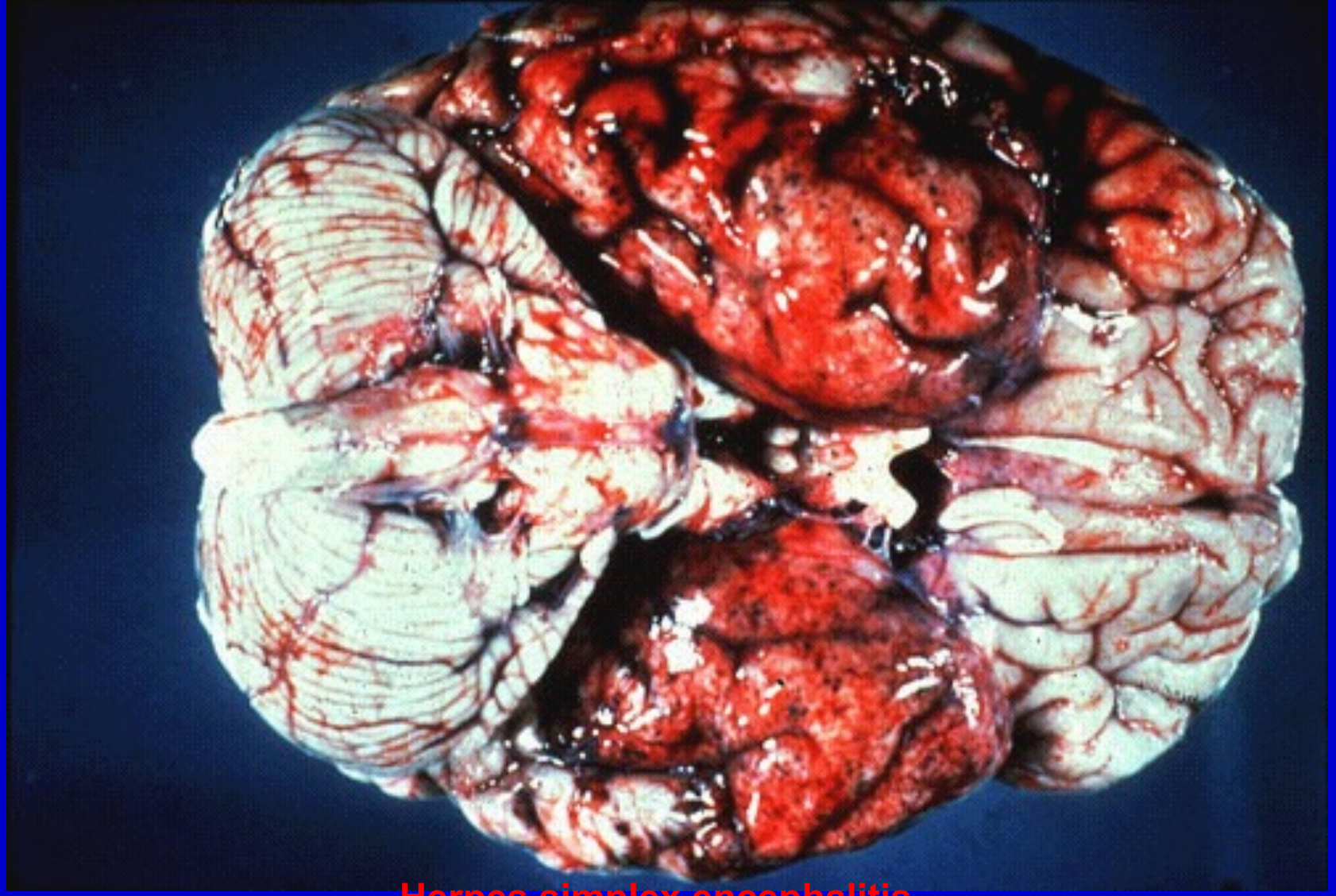
Herpes simplex encephalitis



Herpes simplex encephalitis

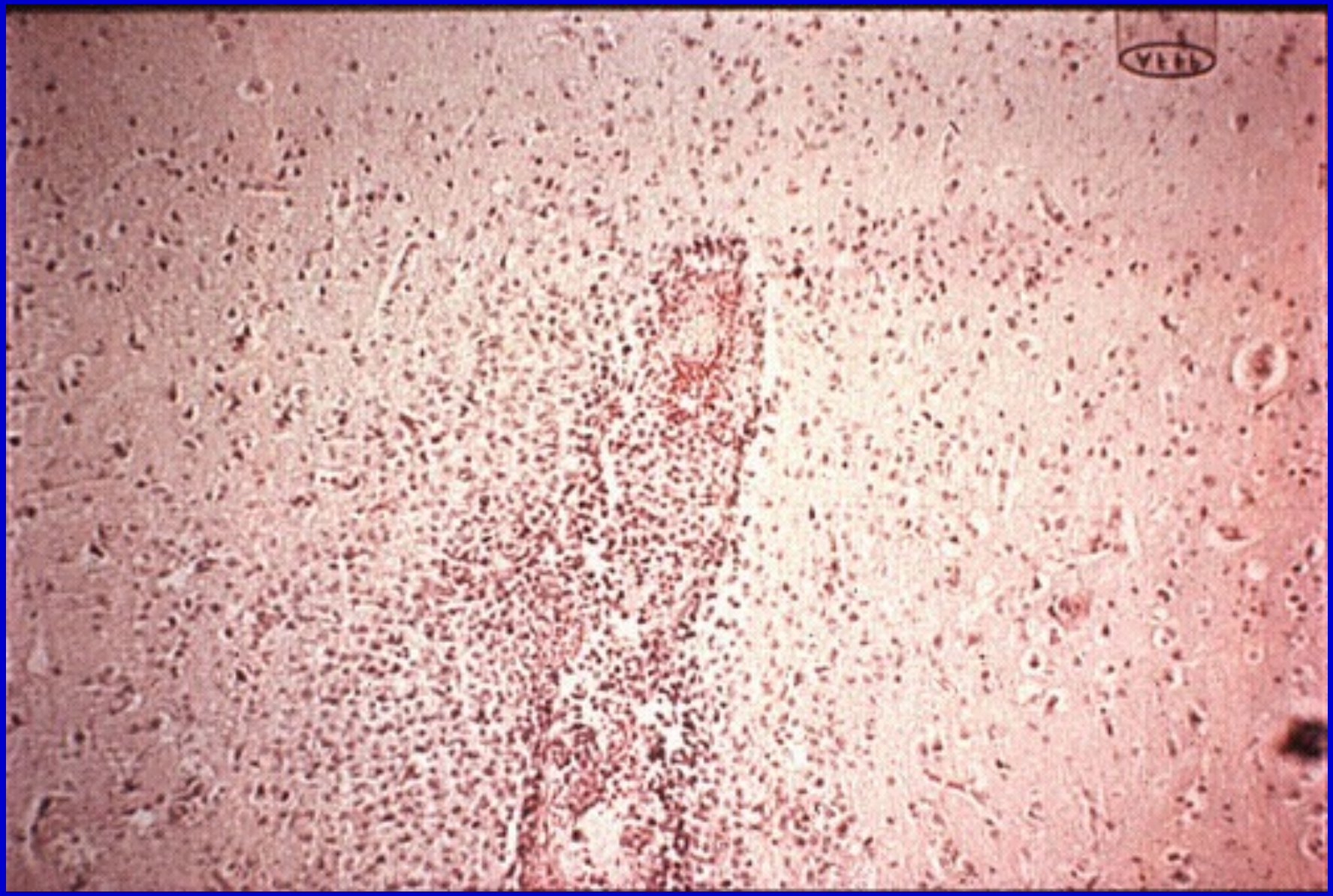
Pathology .

The lesions take the form of an intense hemorrhagic necrosis of the inferior and medial parts of the temporal lobes and the orbital parts of the frontal lobes. The temporal lobe lesions are usually bilateral but need not be symmetrical. Cases described in past years as *acute necrotizing encephalitis* and inclusion body encephalitis were probably instances of HSV encephalitis. In the acute stages of the disease, intranuclear eosinophilic inclusions are found in neurons and glial cells, in addition to the usual microscopic abnormalities of acute encephalitis.

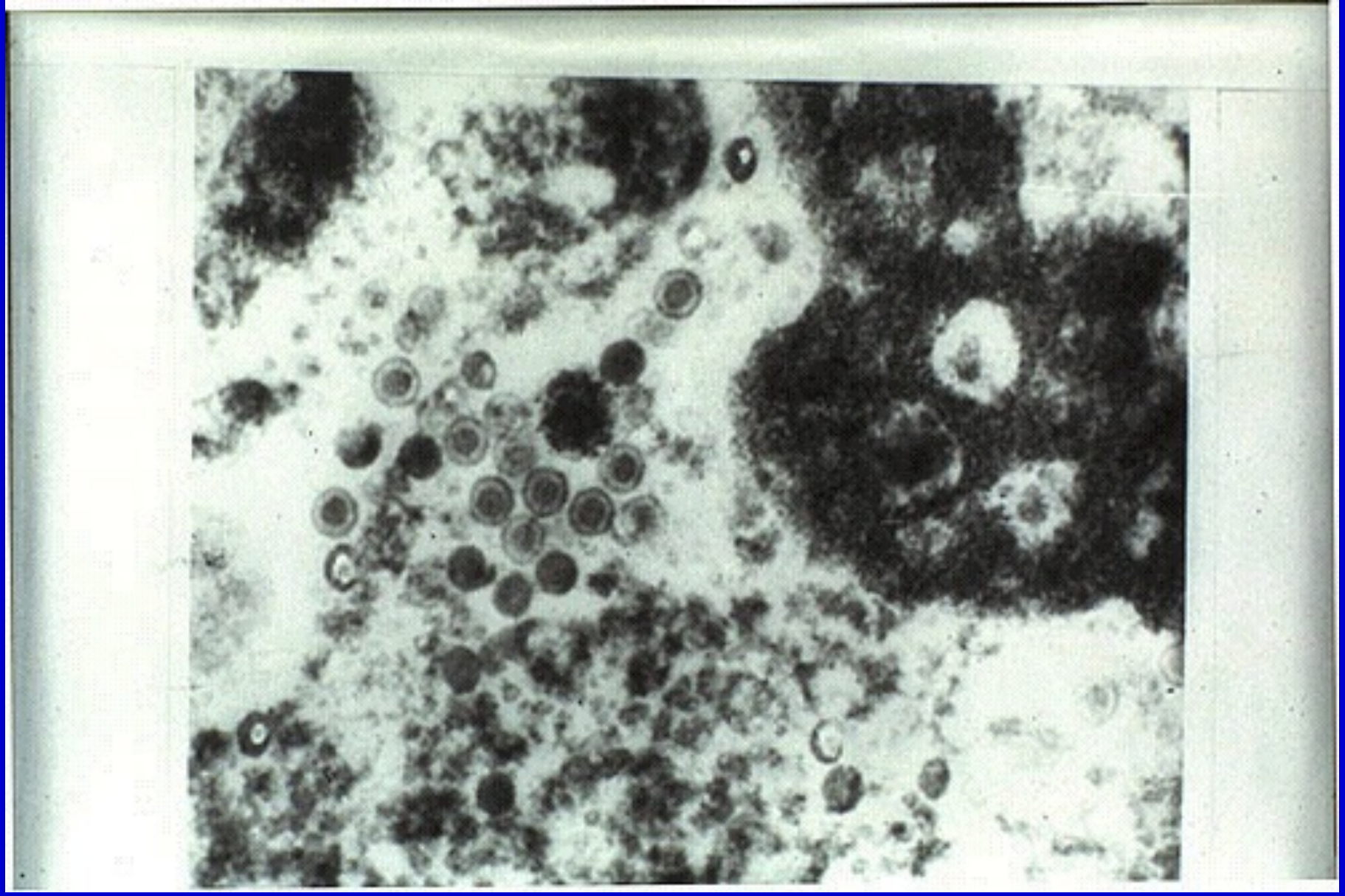


Herpes simplex encephalitis

intense hemorrhagic necrosis of the inferior and medial parts of the temporal lobes and the orbital parts of the frontal lobes.



usual microscopic abnormalities of acute encephalitis



intranuclear eosinophilic inclusions are found in neurons and glial cells

Herpes simplex encephalitis

Clinical Features.

The symptoms, which evolve over several days, are in most cases like those of any other acute encephalitis - namely, fever, headache, seizures, confusion, stupor, and coma. In some patients these manifestations are preceded by symptoms and findings that betray the propensity of this disease to involve the inferomedial portions of the frontal and temporal lobes.

Herpes simplex encephalitis

Clinical Features.

The latter manifestations include olfactory or gustatory hallucinations, anosmia, temporal lobe seizures, a brief period of personality change, bizarre or psychotic behavior or a delirium, aphasia, and hemiparesis. Very rarely an affection of memory can be recognized. Swelling and herniation of one or both temporal lobes through the tentorium may occur, leading to deep coma and respiratory arrest during the first 24 to 72 h.

Herpes simplex encephalitis

Diagnosis.

The CSF is often under increased pressure and almost invariably shows a pleocytosis (range, 10 to 500 cells per cubic millimeter, usually less than 200). Mainly the cells are lymphocytes, but occasionally there is a significant number of neutrophils. In a few cases, 3 to 5 percent in some series, the spinal fluid has been normal in the first days of the illness, only to become abnormal when reexamined.

Herpes simplex encephalitis

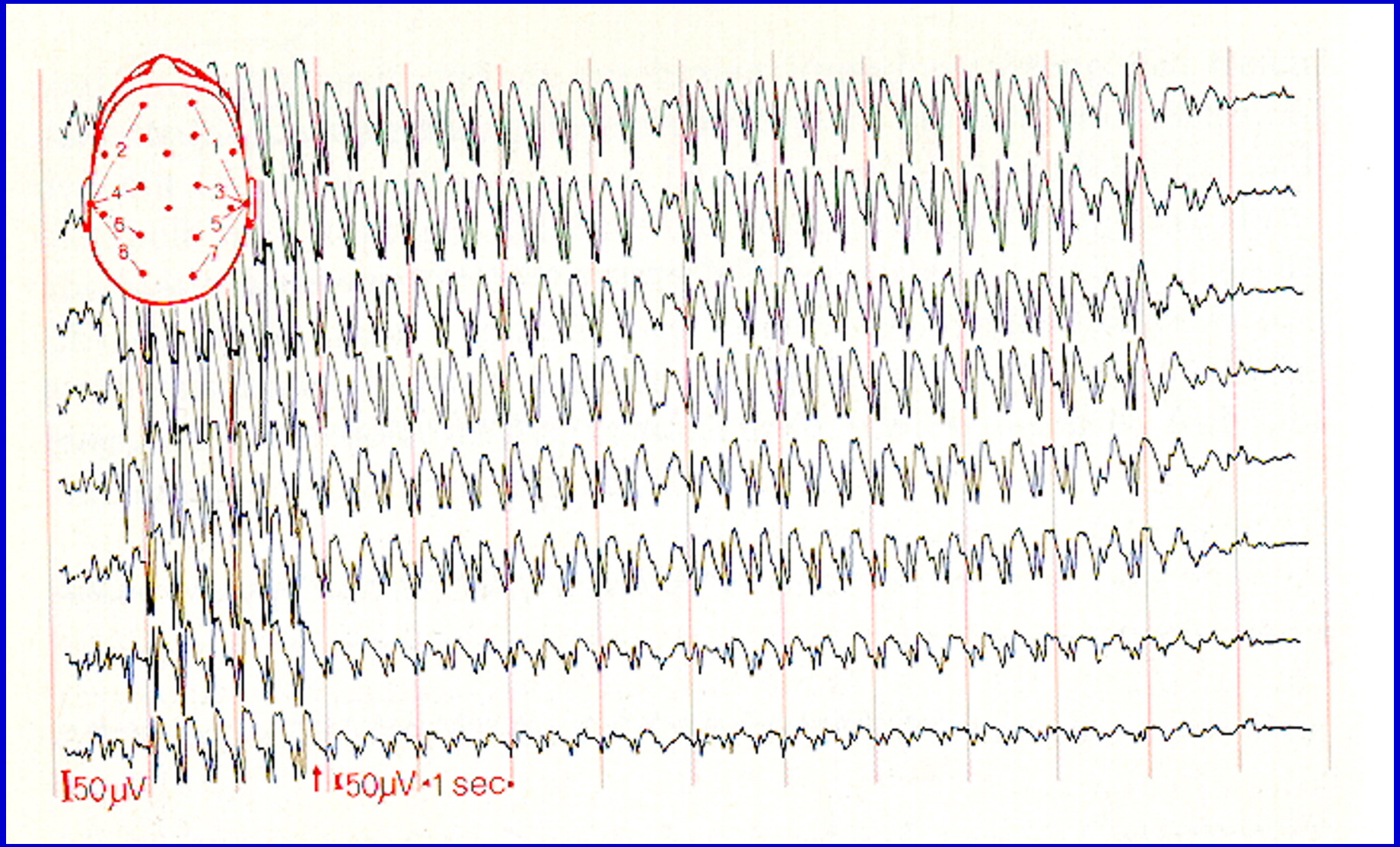
Diagnosis.

Recently developed tests for the detection of HSV antigen in the CSF and the application of the polymerase chain reaction to amplify DNA from the CSF are proving to be useful in diagnosis in the first few days of the illness, while the virus is replicating, and enable one to avoid brain biopsy. The only other absolute way to establish the diagnosis of acute HSV encephalitis is by fluorescent antibody study and by viral culture of cerebral tissue obtained by brain biopsy.

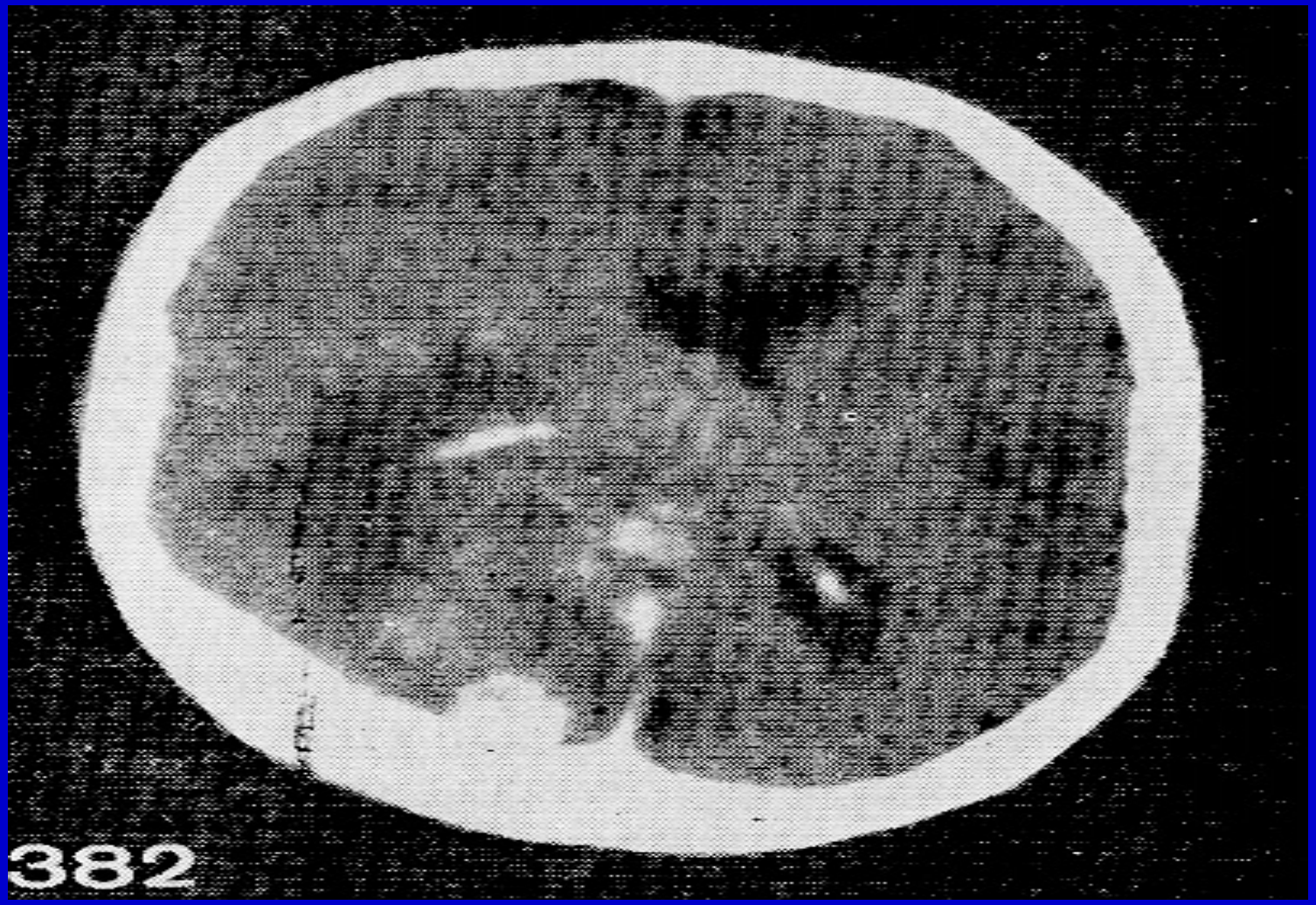
Herpes simplex encephalitis

Diagnosis.

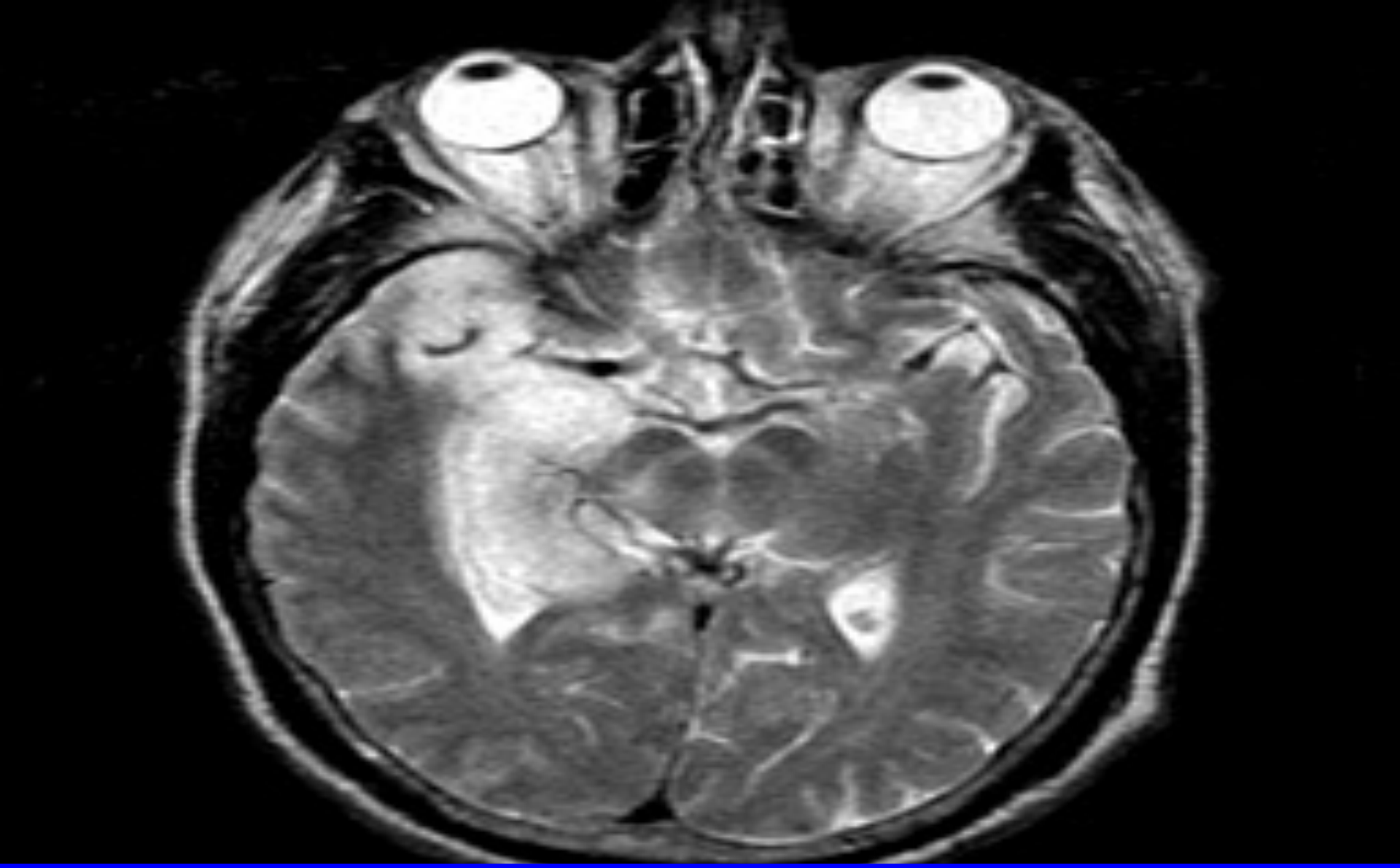
In a minority of cases, red cells, sometimes numbering in the thousands, and xanthochromia are found, reflecting the hemorrhagic nature of the lesions, but more often there are few red cells. The protein content is increased in most cases. Rarely, the CSF glucose levels may be reduced to slightly less than 40 mg/dL, creating confusion with tuberculous and fungal meningitides.



The EEG changes, consisting of lateralized periodic high-voltage sharp waves in the temporal regions and slow-wave complexes at regular 2- to 3-per-second intervals, are suggestive though not specific for the disease.



Computed tomography shows hypoattenuation of the affected areas in 50 to 60 percent of cases



MRI T1-weighted images demonstrate areas of low signal intensity

Herpes simplex encephalitis

Treatment.

Acyclovir is given intravenously in a dosage of 30 mg/kg per day and continued for 15 to 30 days in order to prevent relapse. As indicated above, newer laboratory procedures have obviated the need for brain biopsy in most cases. Acyclovir carries little risk and can be discontinued if further clinical or laboratory features point to another diagnosis.

Herpes simplex encephalitis

Treatment.

All measures used in the management of brain edema due to mass lesions should be applied.

Corticosteriods.

Seizures are usually brought under control by high doses of conventional anticonvulsants.

Herpes simplex encephalitis

The outcome.

If the patient is unconscious, the outcome is uniformly poor.

However, if treatment is begun within 4 days of onset of the illness in awake patients, survival is increased to 92 percent (Whitley).

The neurologic sequelae are of the most serious type, consisting of a Korsakoff amnesic defect or a global dementia, seizures, and dysphasia (see Drachman and Adams).

Influenza encephalitis

Features:

It has a seasonal character.

It manifests after an apparent satisfactory state.

Morphopathological hemorrhagic component is present.

Clinical forms: focal *(mezodiencephalic, cochleovestibular, bulbar)*, with diffuse neurological symptoms.

To the treatment are added gamma-globulin, remantadine, desensitizing agents.

Rheumatic encephalitis

Etiology: ß-hemolytic streptococcus group A.

Clinical forms:

Chorea Sydenham ("chorea Sancti Viti")

Hyperkinesis like tics

Distal stereotypic hyperkinesis

Myoclonic hyperkinesis

With diffuse neurological symptoms

Paralytic ("chorea mollis")

Atypical (whiplash, pseudo-hysterical, psychotic, with pronounced intracranial hypertension syndrome)

Rheumatic encephalitis

Epidemiological data: Age frequently affected 6-15 years; girls predominate (2: 1); most cases are observed in March April and November-December.

The onset *may be sudden, but it is often insidious within 2-3 weeks.*

Cardinal sydromes:

1. *Choreic movements*
2. *Muscular hypotonia*
3. *Emotional lability*

Rheumatic encephalitis

Choreic movements are:

involuntary, spontaneous, illogical, short, sudden,

disordered, large amplitude, contradictory,

never symmetrical, in different muscle groups,

never synchronous, without purpose, arrhythmic,

unforeseen, first on the face and hands,
then generalizes,

exaggerated by mental efforts.

Rheumatic encephalitis

Complementary examinations:

- Somatic exam

Heart damage in 1/3 cases (myocarditis, endocarditis, pericarditis)

Articular changes

Mild hyperthermia

Pulse accelerated

- Laboratory tests

Mild leukocytosis

Relative lymphocytosis

Relative eosinophilia

Increased sedimentation rate of red blood cells

Increased antistreptolysins

SCF: Normal

EEG: dysrhythmias

EMG: irregular spontaneous activity

Rheumatic encephalitis

Differential diagnosis with:

Infantile encephalopathies

Huntington Chorea (infantile and juvenile forms)

Hemiplegic Chorea

Hysteric Chorea

Neurosis of Obsesive Movements

Pregnancy Chorea

Encephalitis with Chorea in syphilis

Thyrotoxicosis

Senile Choreea

Chorea syndromes of toxic origin: carbon monoxide, L-Dopa, neuroleptics

Chorea syndromes in neoplasms

Rheumatic encephalitis

Treatment

I. Bed rest.

II. Varied and rich in proteins and vitamins nutrition, reduced amount of carbohydrates.

III. Etiological anti-rheumatismal treatment.

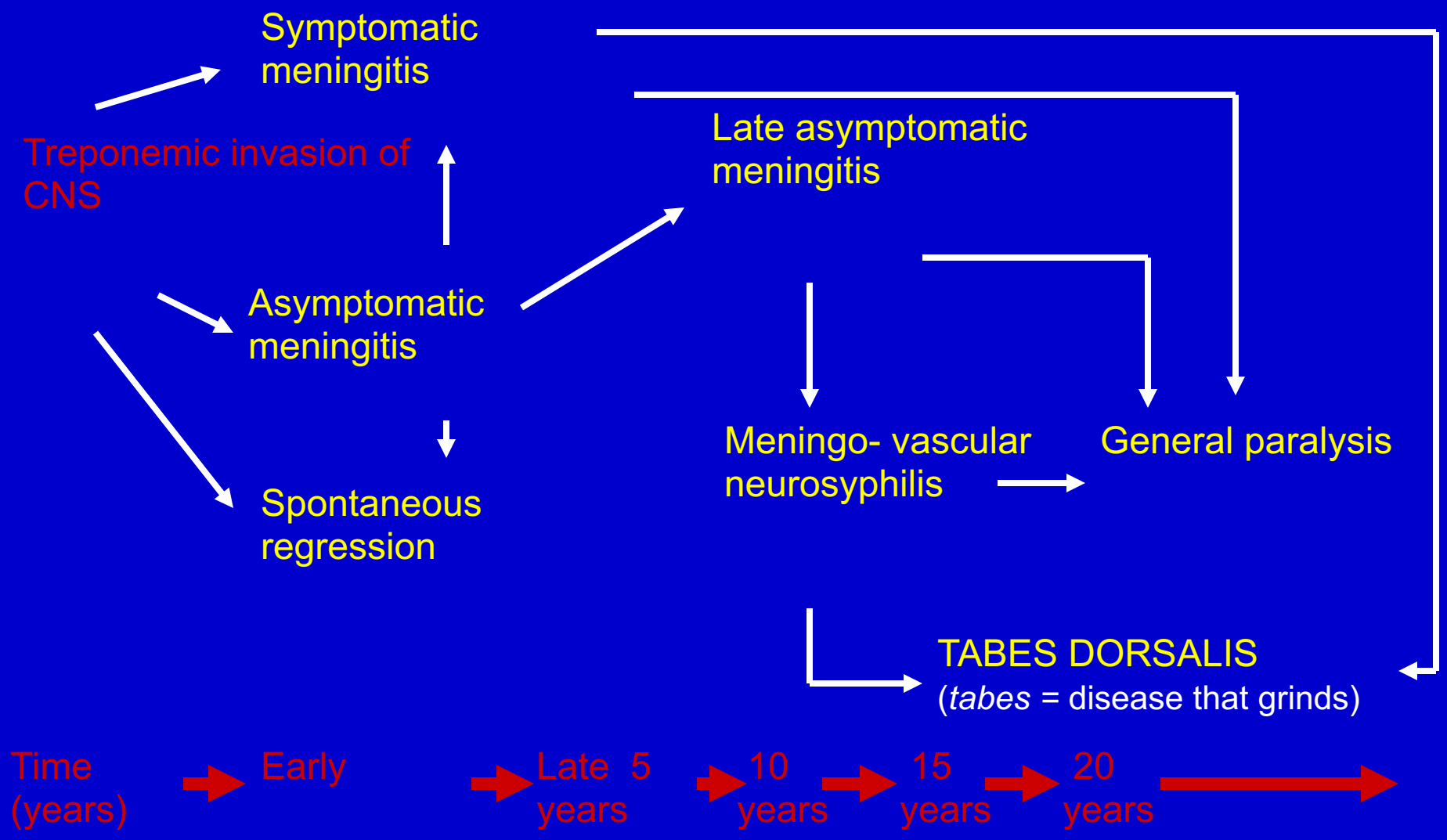
IV. Symptomatic treatment:

- tranquilizers

- other substances with sedative and neuroleptic action (chlorpromazine, thioridazine, phenobarbital, thioproperazine, / aminazine /, haloperidol).

V. Prophylactic anti-rheumatic treatment.

NEUROSYPHILIS



Simplified diagram of neurosyphilis evolution

NEUROSIPHILIS
GENERAL PARALYSIS (PARALYTIC DEMENTIA)

Pathology. Specific macroscopic and microscopic changes.

Symptoms. Sudden onset is accompanied by transient neurological signs: seizures, motor deficits, aphasia.

The insidious onset has three stages:

1. incipient; 2. development of psychosis; 3. terminal.

Gradually the patients deteriorate mentally. Argyll Robertson sign is present in 90% of cases. Optical atrophy occurs in 5% of cases.

Argyll Robertson sign: bilateral small pupils that reduce in size on a near object (i.e., they accommodate), but do not constrict when exposed to bright light (i.e., they do not react to light). They are a highly specific sign of neurosyphilis;.

Complementary investigations: CSF - moderate pleocytosis + hyperproteinorachia, positive reactions for syphilis.

NEUROSIPHILIS: TABES DORSALIS

(Progressive locomotor ataxia)

Morphology. Are predominantly affected posterior columns and posterior spinal cord roots.

Symptoms.

Abolition of myotactic reflexes of the lower limbs. +

Argyll-Robertson sign + *Romberg sign*

Painful crises of the type "throwing", "figurative", "in the belt".

Muscular hypotonia, more evident at the inferior limbs. Sphincter disorders. Disorders of cranial nerve function. Trophic disorders. Visceral seizures. Psychiatric disorders.

NEUROSIPHILIS: TABES DORSALIS

Fleeting and repetitive lancinating pains, primarily in the legs or less often in the back, thorax, abdomen, arms, and face.

Ataxia of the legs and gait due to loss of position sense occurs in half of patients.

Paresthesias, bladder disturbances, and acute abdominal pain with vomiting (visceral crisis) occur in 15–30% of patients.

The cardinal signs of tabes are loss of reflexes in the legs; impaired position and vibratory sense; Romberg's sign; and, in almost all cases, bilateral Argyll Robertson pupils, which fail to con- strict to light but accommodate.

NEUROSIPHILIS: TREATMENT

BENZYLPENICILLIN. 12 – 24 mln u. i/v/day for 14 days

In case of penicillin allergy it is recommended:

TETRACYCLINE 500 mg x 4 times/day for 30 days

or

ERYTHROMYCIN 500 mg x 4 times/day for 30 days

or

CHLORAMPHENICOL 1 g i/v every 6 hours for 6 weeks under the control of erythropoiesis

or

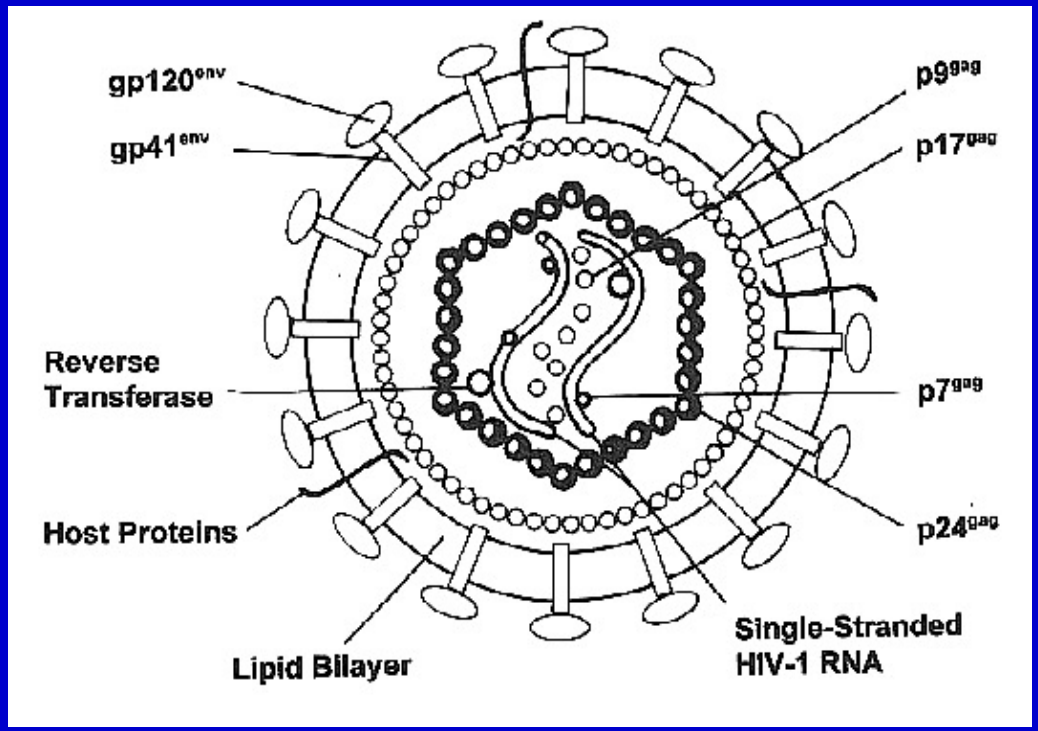
CEFTRIAXONE 2 g 1 time/day i/v or i/m for 14 days

HIV / SIDA

HIV (human immunodeficiency virus)

HIV **- Lentivirus from the subgroup of retroviruses with tropism to:**

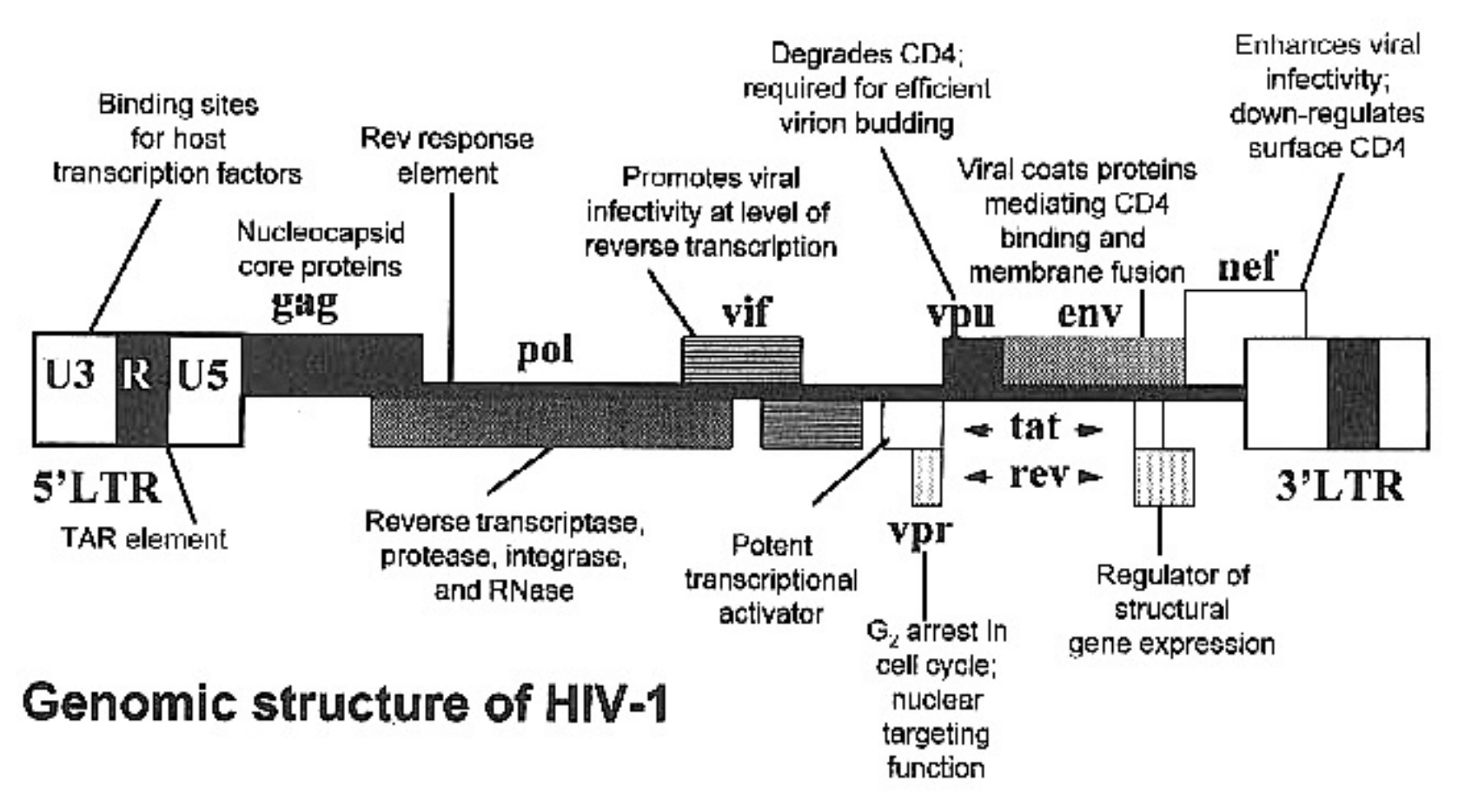
1. Macrophage-monocyte line (in S.N.C. their counterpart is microglia).



2. Lymphocytes T-helper (CD4 +).

Neurological manifestations result from the combined infection of the two cell types.

HIV / SIDA



The HIV-1 virus was isolated from brain tissue, spinal cord, cerebrospinal fluid, and peripheral nerves.

HIV / AIDS

HIV (human immunodeficiency virus)

HIV causes AIDS (Acquired Immunodeficiency Syndrome)

Human immunodeficiency virus and AIDS are the two stages of one and the same nozological form - HIV / AIDS

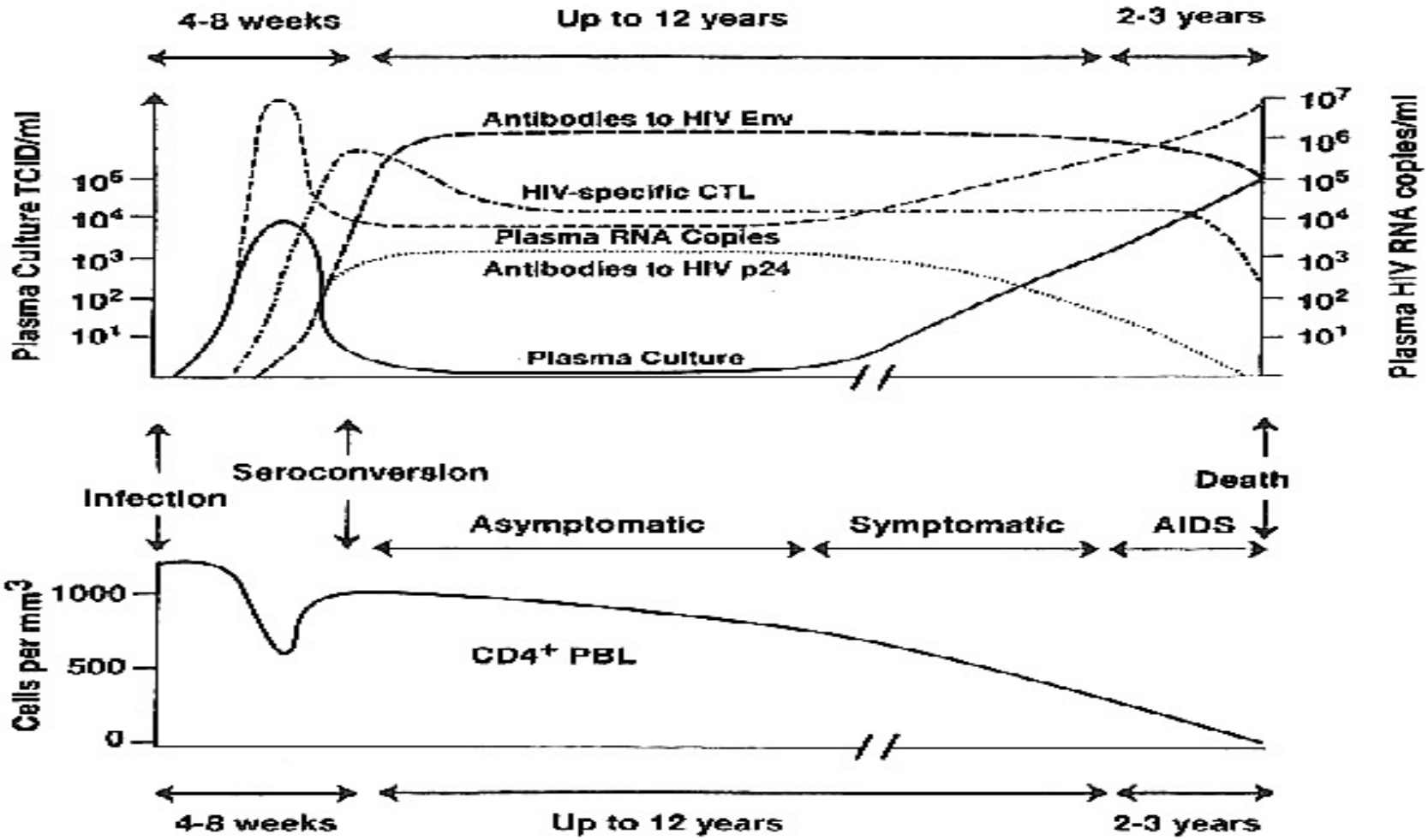
CLASSIFICATION

The clinical-immunological classification of HIV / AIDS infection is based on the clinical criteria and the amount of CD4 + T lymphocytes in the blood. This classification was developed by WHO in 1990 and modified by the U.S. Centers for Disease Control, published in 1993.

Clinical-immunological CLASSIFICATION of HIV / AIDS infection

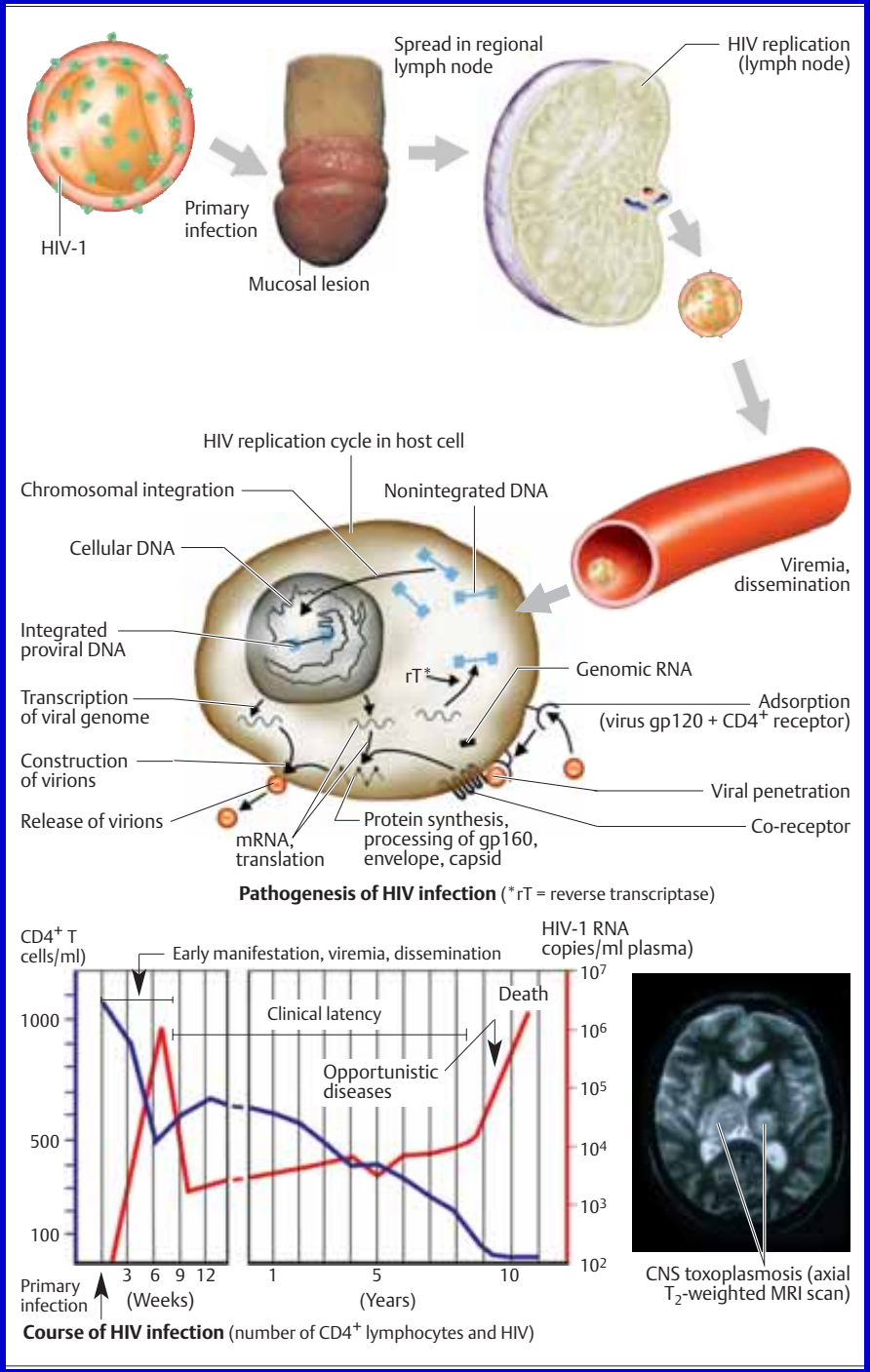
Number of CD4 + T-lymphocytes in 1 mcl	Clinical categories		
	A Asymptomatic, acute HIV or Persistent Generalized Lymphadenopathy	B Symptomatic, not A, not C	C AIDS-indicator diseases
>500 (>29%)	A1	B1	C1
200 - 499 (14 - 28%)	A2	B2	C2
>200 (<14 %) = AIDS-indicator	A3	B3	C3

HIV / AIDS



EVOLUTION OF THE INFECTIVE PROCESS

HIV


Spread in regional lymph node
HIV replication (lymph node)
HIV-1
Primary infection
Mucosal lesion
HIV replication cycle in host cell
Chromosomal integration
Nonintegrated DNA
Cellular DNA
Viremia, dissemination
Integrated proviral DNA
rT*
Genomic RNA
Transcription of viral genome
Adsorption (virus gp120 + CD4+ receptor)
Construction of virions
Viral penetration
Release of virions
Co-receptor
mRNA, translation
Protein synthesis, processing of gp160, envelope, capsid
Pathogenesis of HIV infection (*rT = reverse transcriptase)
CD4+ T cells/ml)
HIV-1 RNA copies/ml plasma)
Early manifestation, viremia, dissemination
Clinical latency
Death
Opportunistic diseases
1000
500
100
10^7
10^6
10^5
10^4
10^3
10^2
3 6 9 12
1
5
10
Primary infection
(Weeks)
(Years)
CNS toxoplasmosis (axial T2-weighted MRI scan)
Course of HIV infection (number of CD4+ lymphocytes and HIV)

1. Diseases of the nervous system directly determined by the HIV virus.

The cognitive-motor complex (HIV encephalopathy).

Aseptic meningitis.

Headache.

Polymyositis.

Guillain - Barre syndrome.

Encephalitis.

Chronic inflammatory demyelinating polyneuropathy.

Myelopathy.

Neuropathy.

Radiculopathy.

Myopathy.

2. Diseases of the nervous system AIDS-indicating.

NEOPLASMS OF THE NERVOUS SYSTEM:

Primitive lymphoma of the nervous system ≈ 5% of patients with AIDS.

Non-Hodgkin's lymphoma.

Kaposi's sarcoma.

HIV / AIDS

HIV encephalopathy (subcortical dementia)

Motor dysfunction of the lower limbs

Balance disorders, ataxia

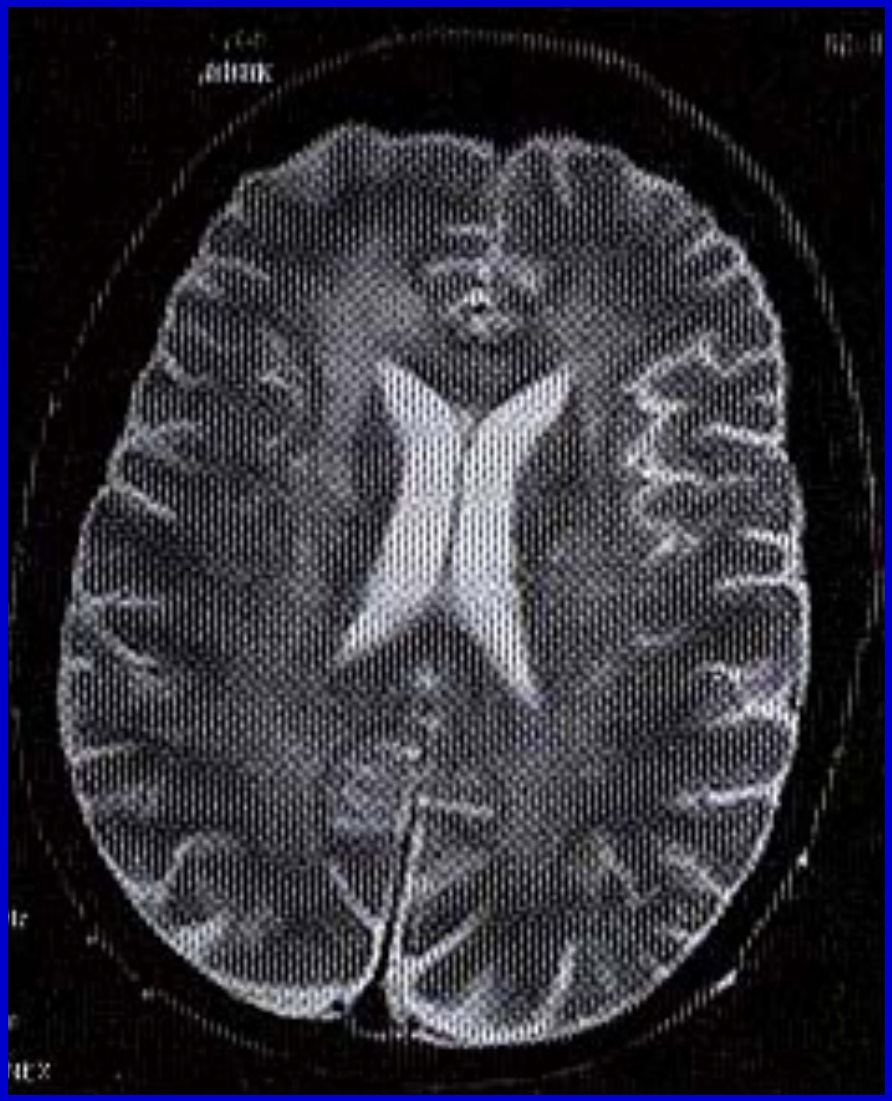
Tremors

Pyramidal signs (hyperreflective, Babinski +)

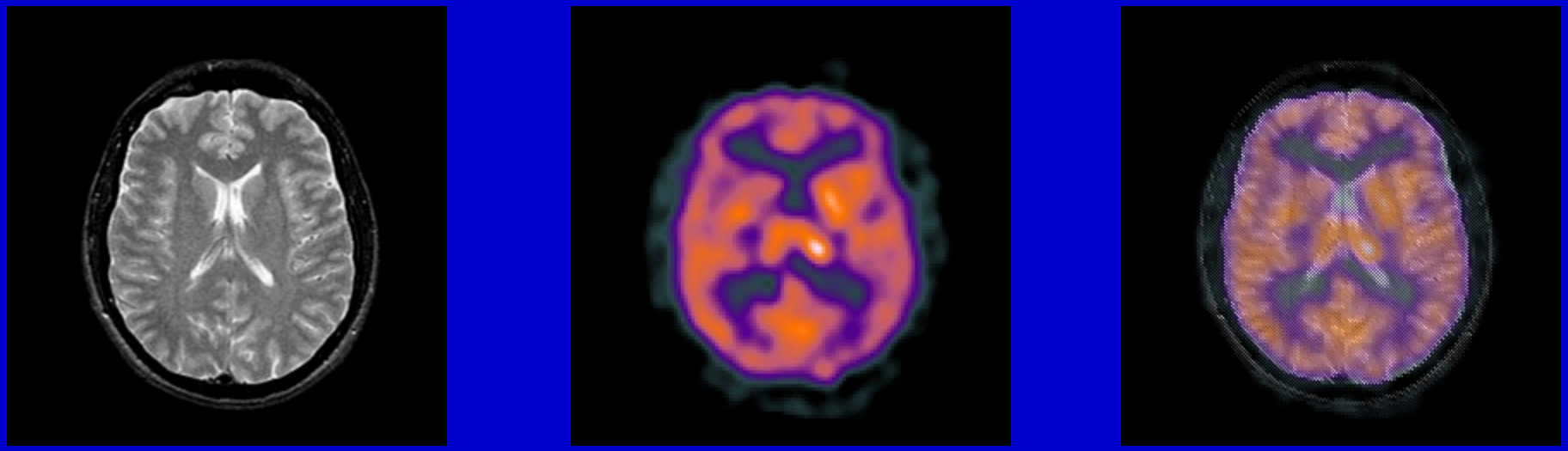
C.S.F. : 20% of patients show lymphocytic pleocytosis (<50 mm3 cells) 60% of patients have hyperproteinrachia B2-microglobulin concentrations correlate with the severity of encephalopathy

CT and MRI shows typical diffuse cerebral atrophy with enlargement of the grooves and ventricular system

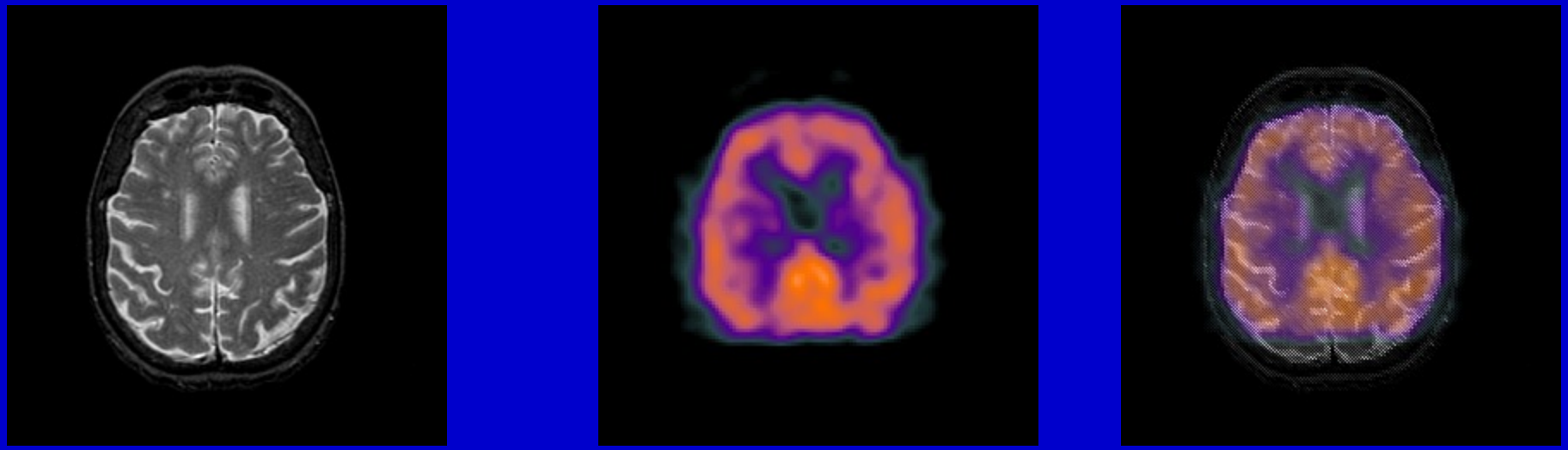
HIV / AIDS



A 30-year-old man with AIDS. T2-weighted MRI image: cortical atrophy in association with hyperintense signal of white matter.



MRI, PET and SPET image of a healthy person



HIV encephalopathy: MRI, PET and SPET imaging

2. Diseases of the nervous system AIDS-indicating

OPPORTUNISTIC INFECTIONS OF THE NERVOUS SYSTEM:

Bacteria (*Mycobacterium tuberculosis, Mycobacterium avium-intracellulare, Treponema pallidum, Nocardia, Salmonella, Listeria monocytogenes*).

Viruses (Cytomegalovirus, Herpes simplex viruses 1 and 2, Varicella zoster virus, JC virus, Epstein-Barr virus).

Fungi (Cryptococcus neoformans, Candida Coccicioides immitis, Aspergillus, Histoplasma capsulatum).

Protozoa (Toxoplasma gondii, Trypanosoma cruzi, Acanthamoeba).

HIV / AIDS

There is no etiological treatment!

MANAGEMENT.

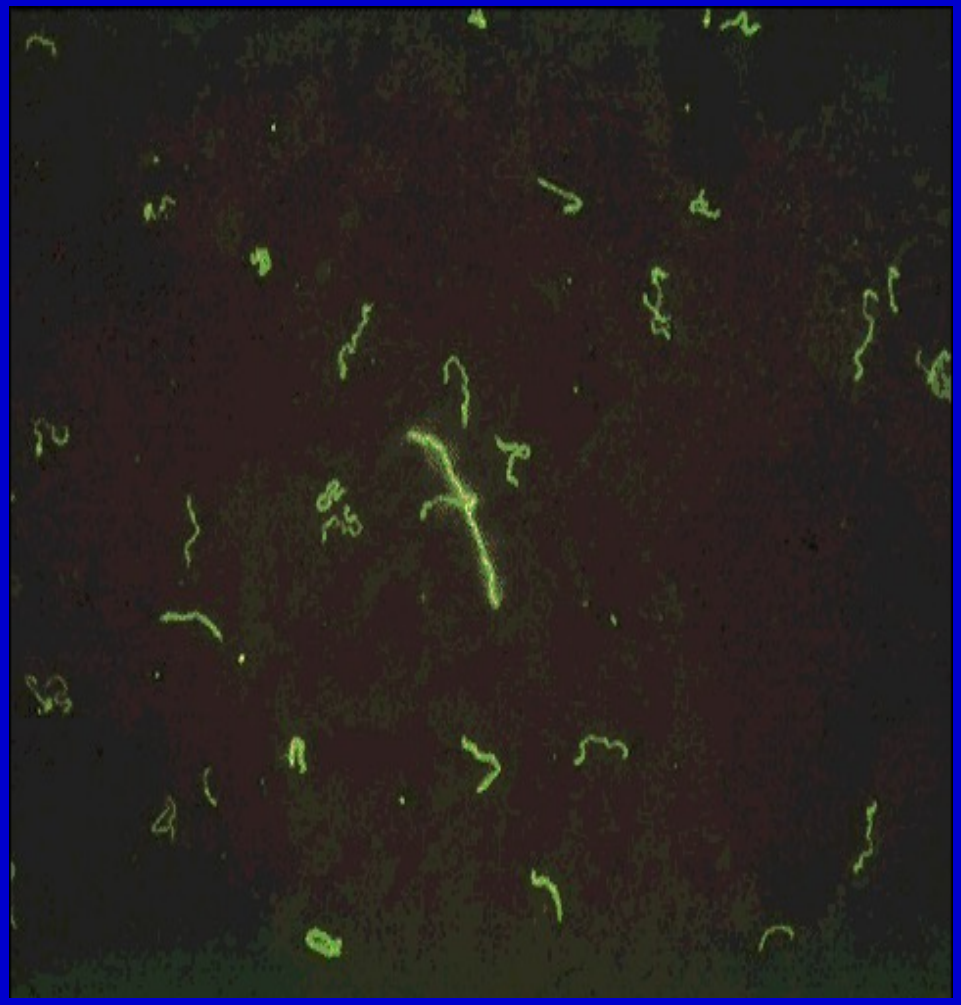
1. Early diagnosis

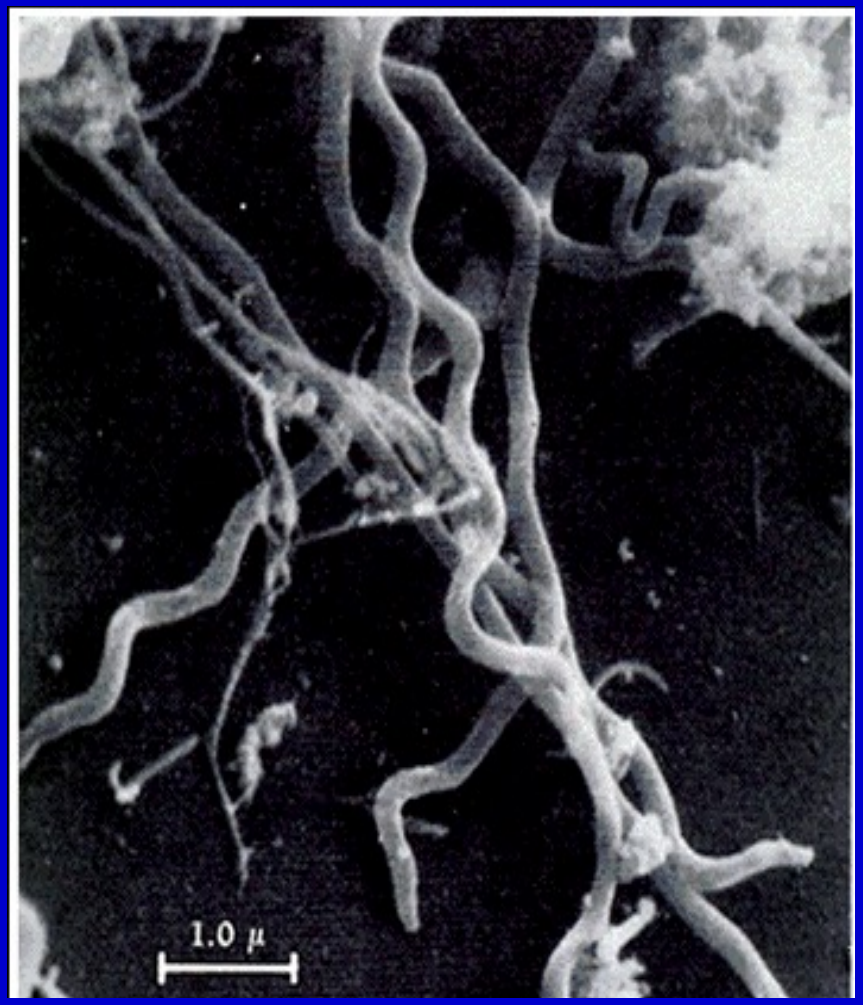
2. Primary antiretroviral therapy.
 -Zidovudine
 -Didanozine
-Dideoxycitidine
-Stavudine
-Lamivudine
-Nelfinavir

3. Prophylaxis and treatment of opportunistic infections.

LYME DISEASE (BORRELIOSIS)

The pathogen agent Spirochaete *Borrelia burgdorferi*





Borrelia burgdorferi

LYME DISEASE (BORRELIOSIS)

The pathogen agent — Spirochaete *Borrelia burgdorferi*

Vector — Ixodes ticks

IXODES TICKS

LYME DISEASE (BORRELIOSIS)

The pathogen agent — Spirochaete *Borrelia burgdorferi*

Vector — Ixodes ticks

ROE DEER CAPTURED BY TICKS

LYME DISEASE (BORRELIOSIS)

The pathogen agent — Spirochaete *Borrelia burgdorferi*

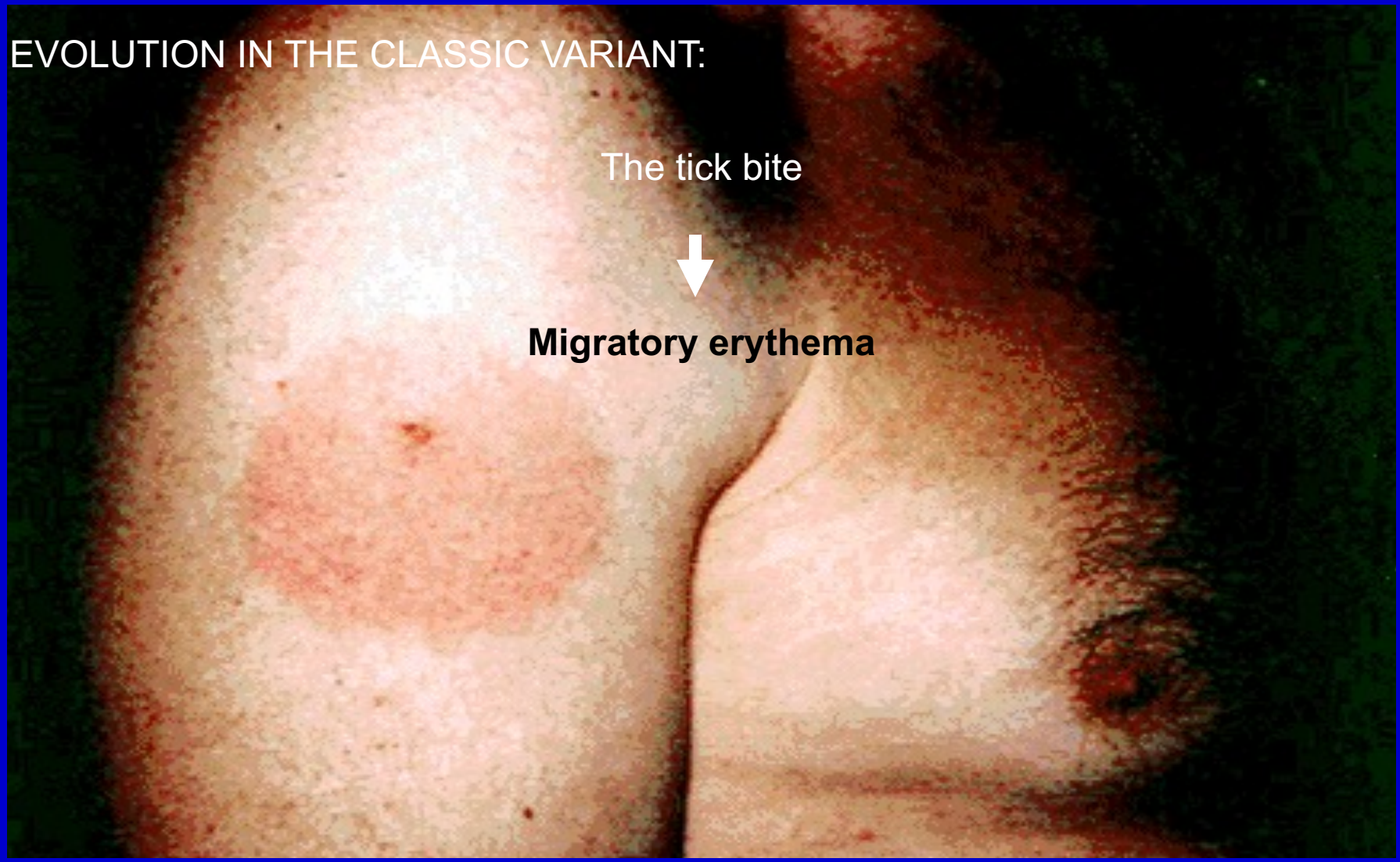
Vector — Ixodes ticks

EVOLUTION IN THE CLASSIC VARIANT:

LYME DISEASE (BORRELIOSIS)

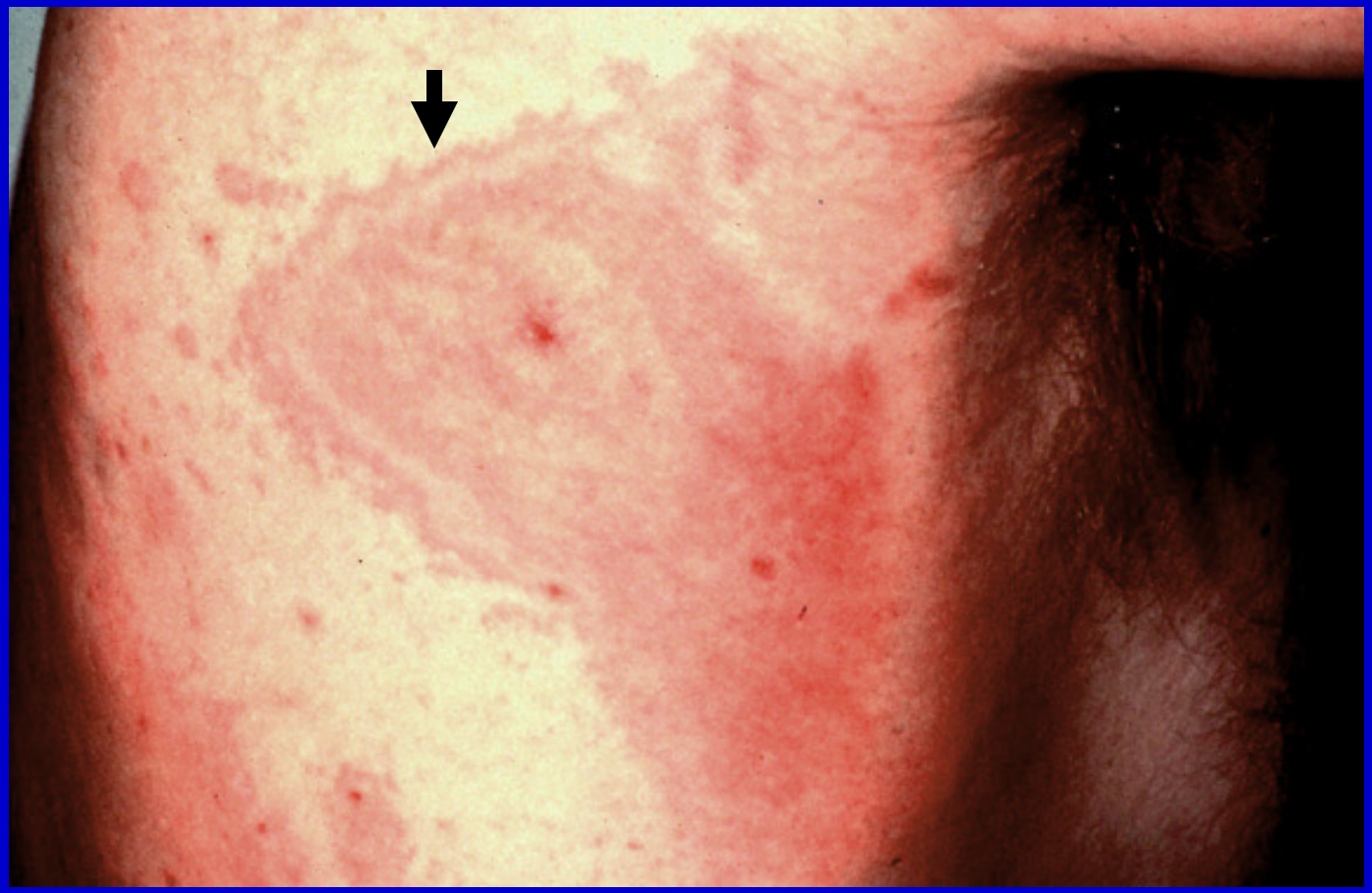
The pathogen agent — Spirochaete *Borrelia burgdorferi*

Vector — Ixodes ticks



Clinical Manifestations of Lyme Disease

Up to 90% of infected patients will develop the characteristic *Erythema Migrans*



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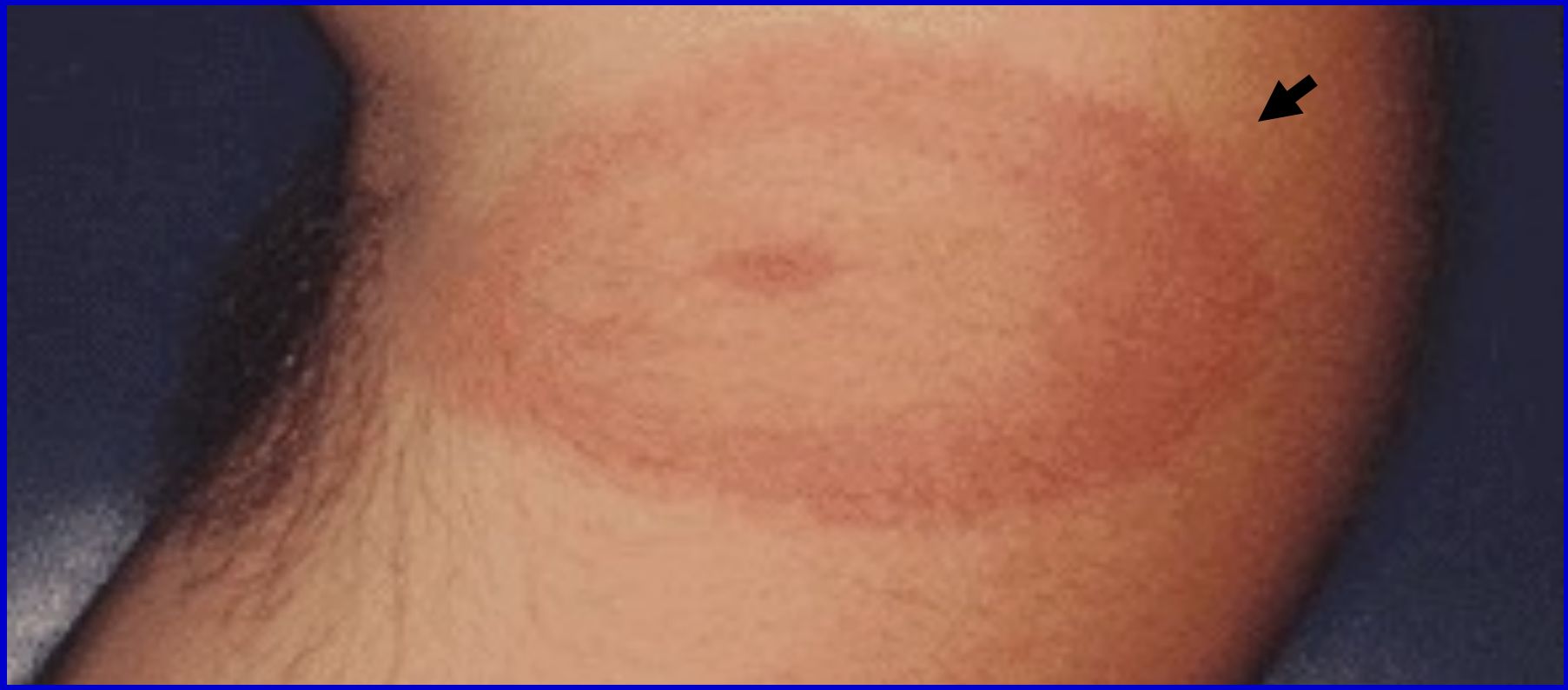

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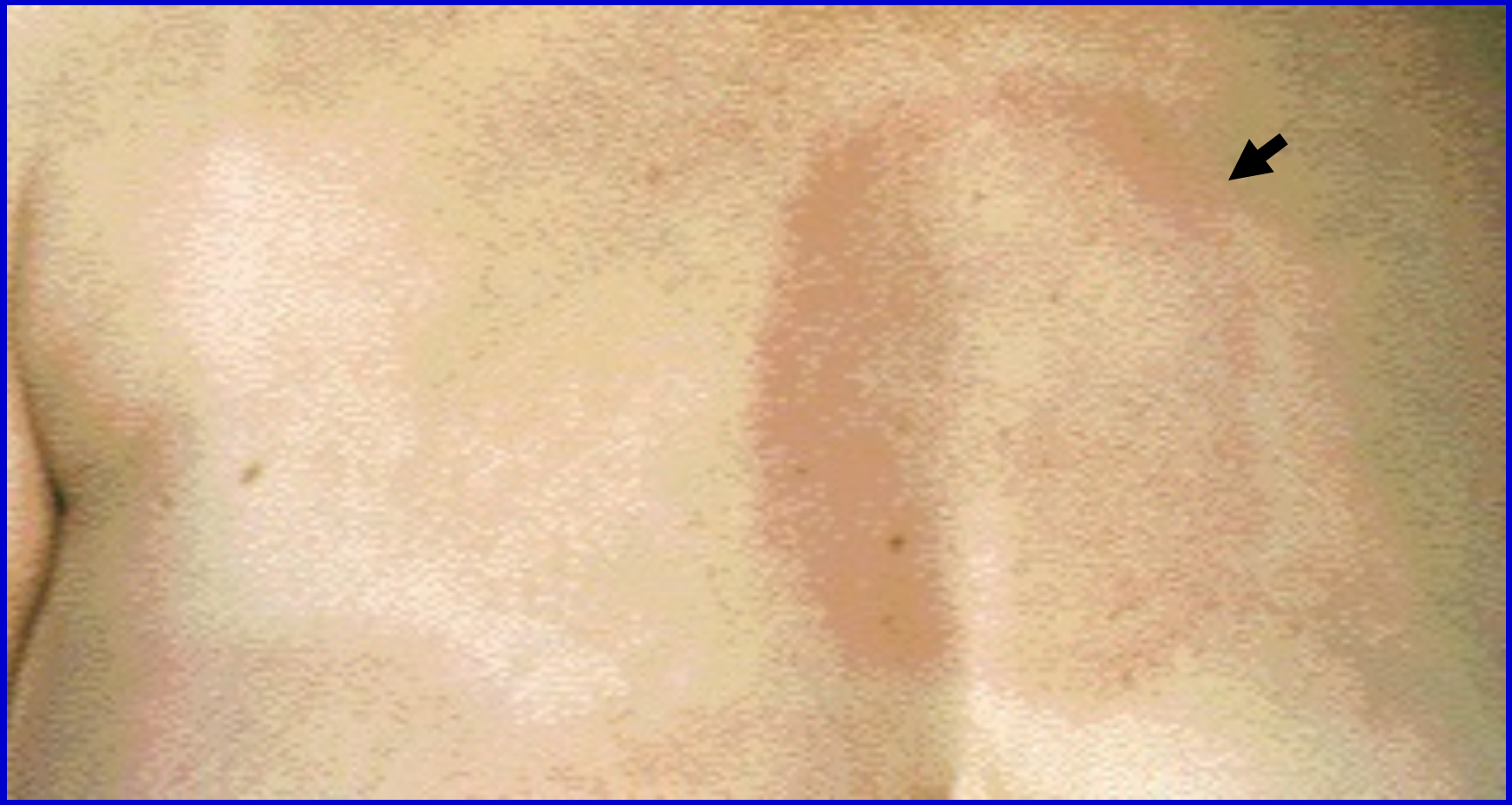


Neurological Manifestations of Lyme Disease (approximately 15% of patients)

Tick Bite

↓

Erythema Migrans (Stage 1)

↓

Flu-like syndrome

fever, chills, extreme malaise, stiff neck and diffuse aches and pains

↓

NEUROLOGICAL MANIFESTATIONS (15% of patients)
(in Stage 2 and/or in Stage 3)

Clinical Manifestations of Lyme Disease

Stage 1. Localized.

Stage 2. Disseminated.

Early infection

Stage 3. Persistent.

Late infection (may occur within 1 year and many years later)

LYME DISEASE (BORRELIOSIS)

NEUROLOGICAL MANIFESTATIONS IN SECONDARY PHASE:

- Mono / multi / polyradiculitis
- Mono / multi / cranial neuritis
- Chronic subacute lymphocytic meningitis

NEUROLOGICAL MANIFESTATIONS IN TERTIARY PHASE:

- Borrelic meylitis
- Borrelic enchephalitis / enchephalopathy

Clinical Manifestations of Lyme Disease

Approximately 1/3 of all patients with erythema migrans will have no further manifestations of Lyme disease, while 2/3 of patients will develop further symptoms.

Clinical Manifestations of acute Neuroborreliosis (stage 2 of LD)

Bannwarth syndrome (triad):

1) lymphocytic meningitis,

2) cranial neuritis,

3) and painful radiculitis.

Clinical Manifestations of acute Neuroborreliosis (stage 2 of LD)

Rarely:

Plexitis,

Guillain-Barré syndrome ,

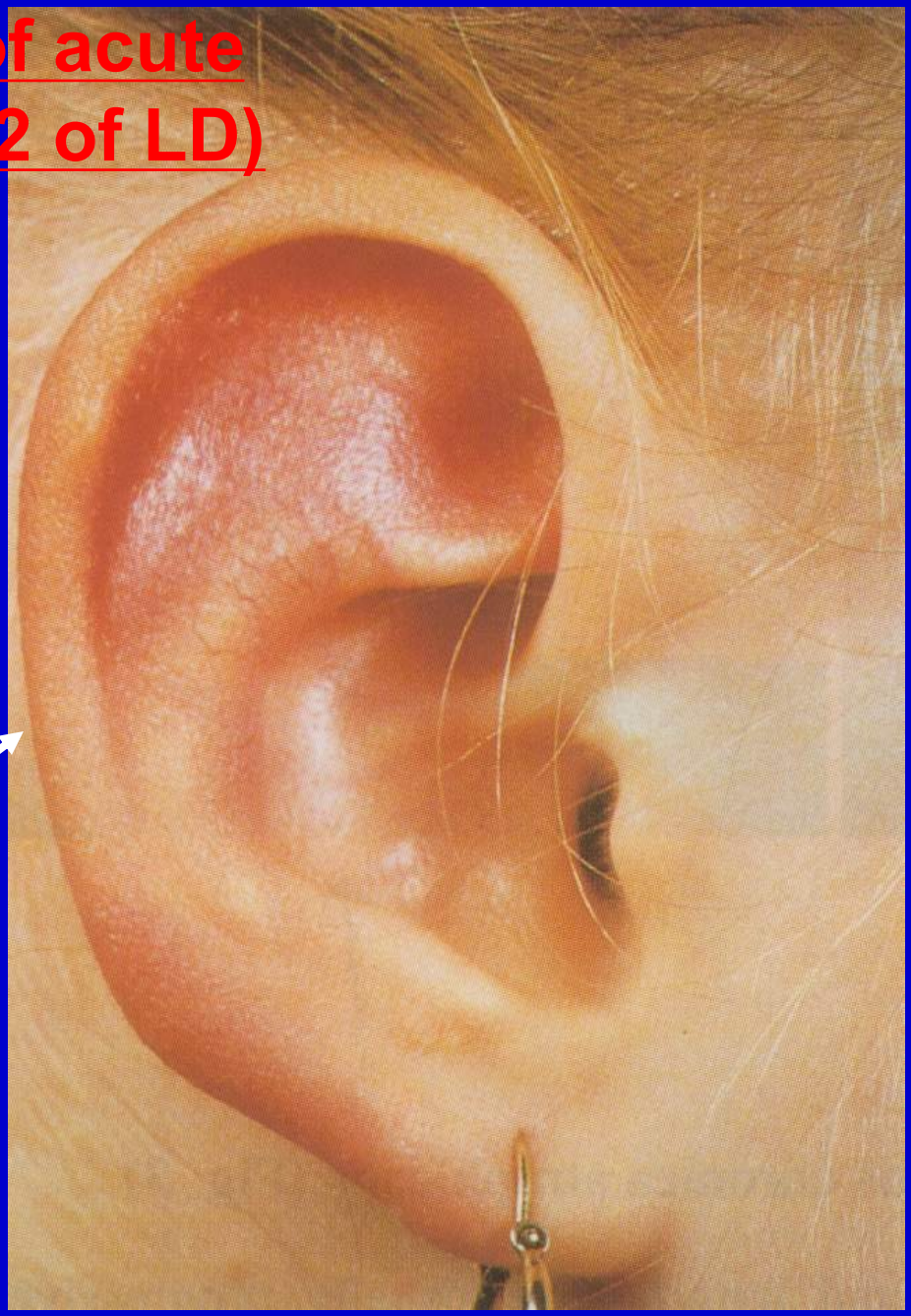
Transversal Myelitis,

Encephalitis.

Clinical Manifestations of acute Neuroborreliosis (stage 2 of LD)

Another lesion of the skin in this stage can be associated to neurological manifestations of LD:

LYMPHADENOSIS BENIGNA CUTIS



Clinical Manifestations of acute Neuroborreliosis (stage 2 of LD)

CRANIAL NEURITIS:

- Virtually any cranial nerve may be involved:
n. VII > n.II > nn. VI; III; IV > n.V > nn. IX-XII

- The seventh is the most common
- most often occurs within 4 weeks of the appearance of erythema migrans
- headache and fatigue
- it may be bilateral

N.B.: bilateral facial palsies commonly may be in:

- Lyme Neuroborreliosis

- Sarcoidosis

- Guillain-Barré syndrome

Clinical Manifestations of acute Neuroborreliosis (stage 2 of LD)

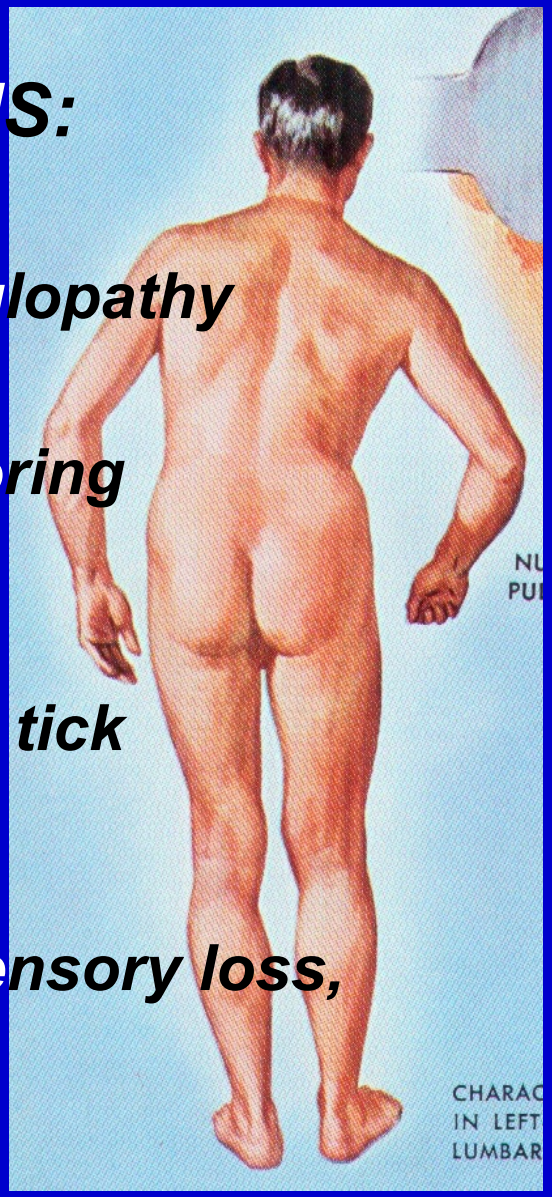
PAINFUL RADICULITIS:

- *may mimic a mechanical monoradiculopathy*

- *severe sharp, jabbing, or deep and boring pain in a radicular nerve distribution.*

- *often, the limb that was the site of the tick bite is the site of the pain.*

- *within days to weeks there may be sensory loss, weakness, or hyporeflexia in the limb.*



Clinical Manifestations of acute Neuroborreliosis (stage 2 of LD)

TRANSVERSAL MYELITIS:

- often occurring at the same level as nerve root inflammation in the syndrome Bannwarth and vary usually between Th 4 and Th 10;

-can develop in hours, days or months from the onset of the infection;

- some patients develop a complete Brown-Sequard syndrome.

Clinical Manifestations of late Neuroborreliosis (stage 3 of LD)

FOCAL ENCHEPHALOMYELITIS:

- Prominent white matter involvement

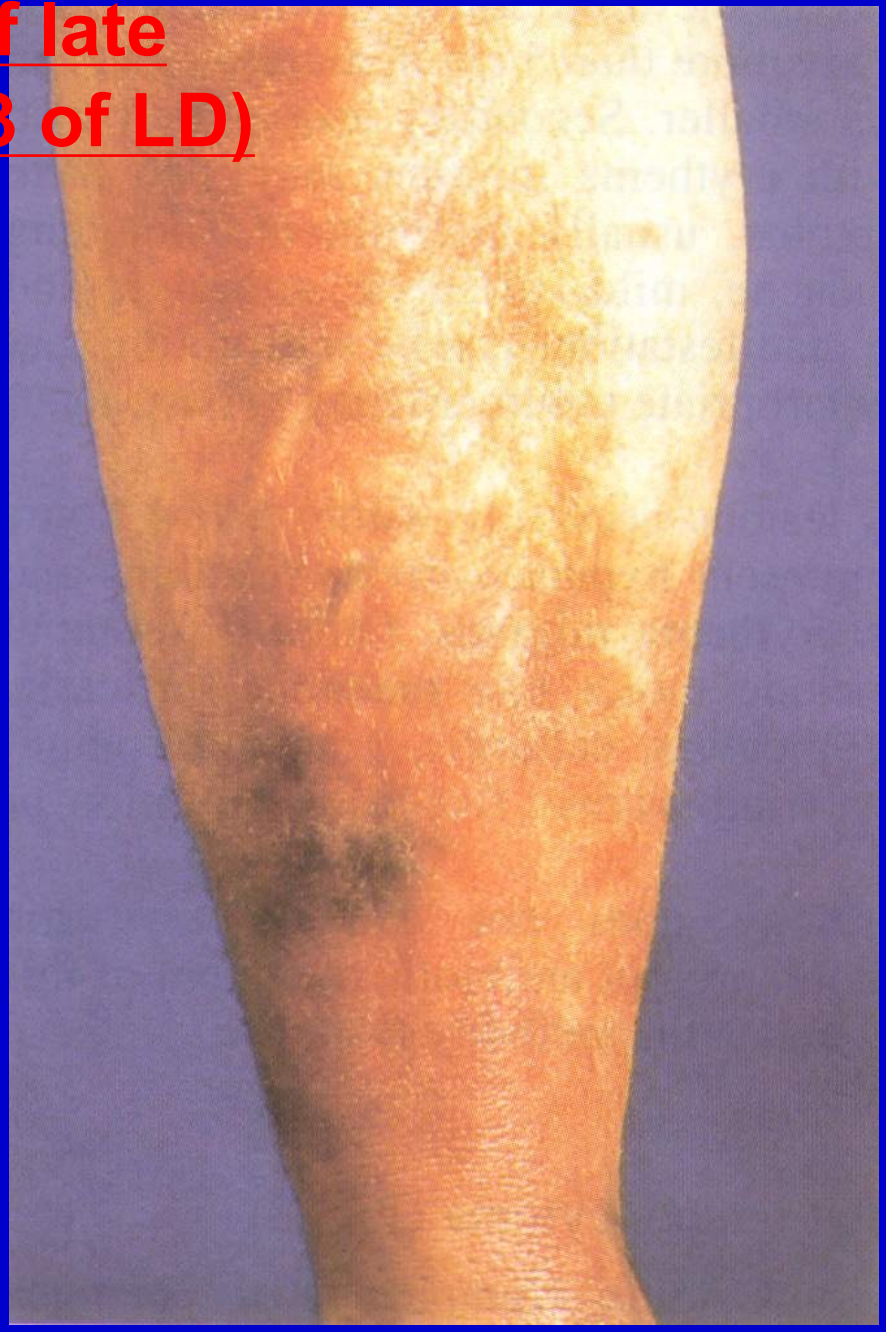
- Examination of the CSF demonstrates a lymphocytic pleocytosis, a mildly elevated protein concentration and the intrathecal production of anti-Borrelia burgdorferi antibodies.

Clinical Manifestations of late Neuroborreliosis (stage 3 of LD)

PERIPHERAL NEUROPATHY:

sensory symptoms, particularly distal paresthesias in a stocking and glove or a stocking distribution.

Often occur with *Acrodermatitis Chronica Atrophicans*, *a chronic cutaneous manifestation of Borrelia infection seen in Europe, but rarely in North America.*



Clinical Manifestations of late Neuroborreliosis (stage 3 of LD)

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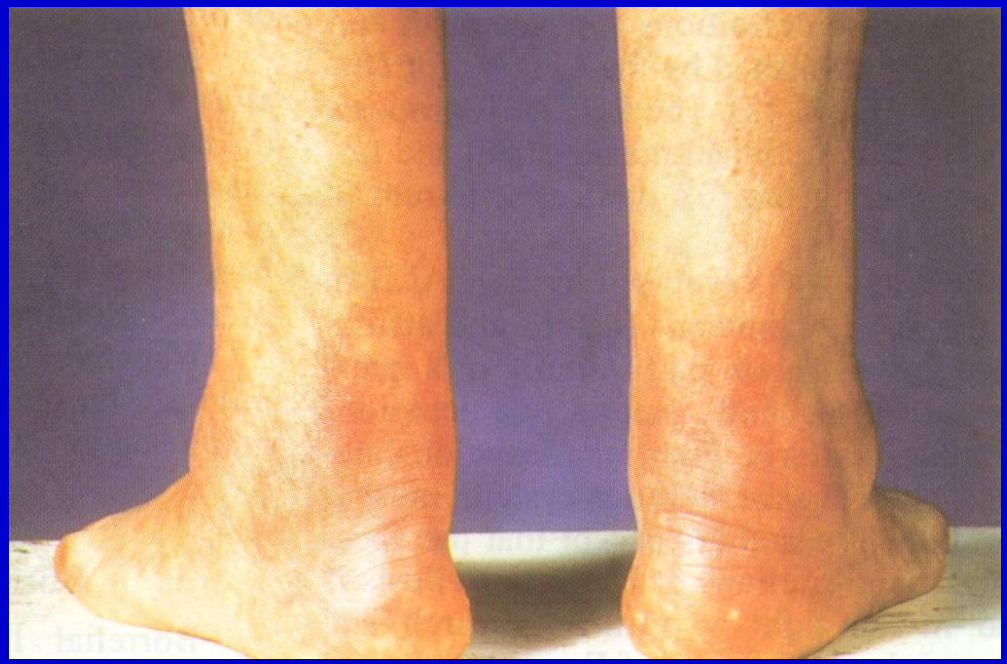
sensory symptoms, particularly distal paresthesias in a stocking and glove or a stocking distribution.



Often occur with *Acrodermatitis Chronica Atrophicans*, *a chronic cutaneous manifestation of Borrelia infection seen in Europe, but rarely in North America.*

Clinical Manifestations of late Neuroborreliosis (stage 3 of LD)

PERIPHERAL NEUROPATHY:

Electrophysiological studies demonstrate a mild sensorimotor radiculoneuropathy with axonal loss, minor decreases in the distal compound action potential amplitudes (sensory greater than motor), little or no slowing of nerve conduction velocities, and mild to moderate distal and paraspinal muscle denervation.

The examination of the CSF is usually normal.

Clinical Manifestations of late Neuroborreliosis (stage 3 of LD)

LYME ENCEPHALOPATHY:

Difficulty with memory and cognitive slowing often accompanied by fatigue and malaise.

Neuropsychological testing is abnormal.

CSF abnormalities may or may not be present.

Evidence from quantitative analysis of SPECT scans suggests that Lyme encephalopathy patients with measurable memory deficits have reduced perfusion affecting primarily subcortical frontotemporal white matter and basal ganglia.

Differential Diagnosis of Lyme Neuroborreliosis

- Multiple Sclerosis

-Other chronic infections of Nervous System

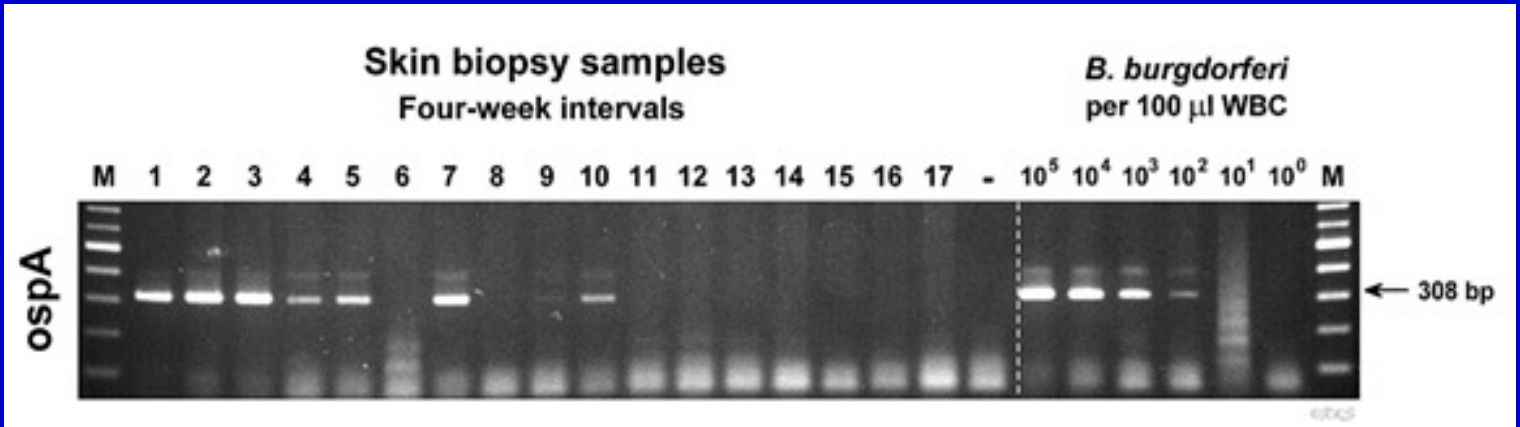
- syphilis
- leptospirosis
- mononucleosis
- parvovirus infection
- rheumatoid arthritis

Diagnostic Workup of Lyme Neuroborreliosis

- Borrelia burgdorferi can be grown in vitro and can often be cultured from the typical cutaneous lesion (erythema migrans, acrodermatitis chronica atrophica)

- The polymerase chain reaction (PCR) to detect bacterial DNA

Diagnostic Workup of Lyme Neuroborreliosis

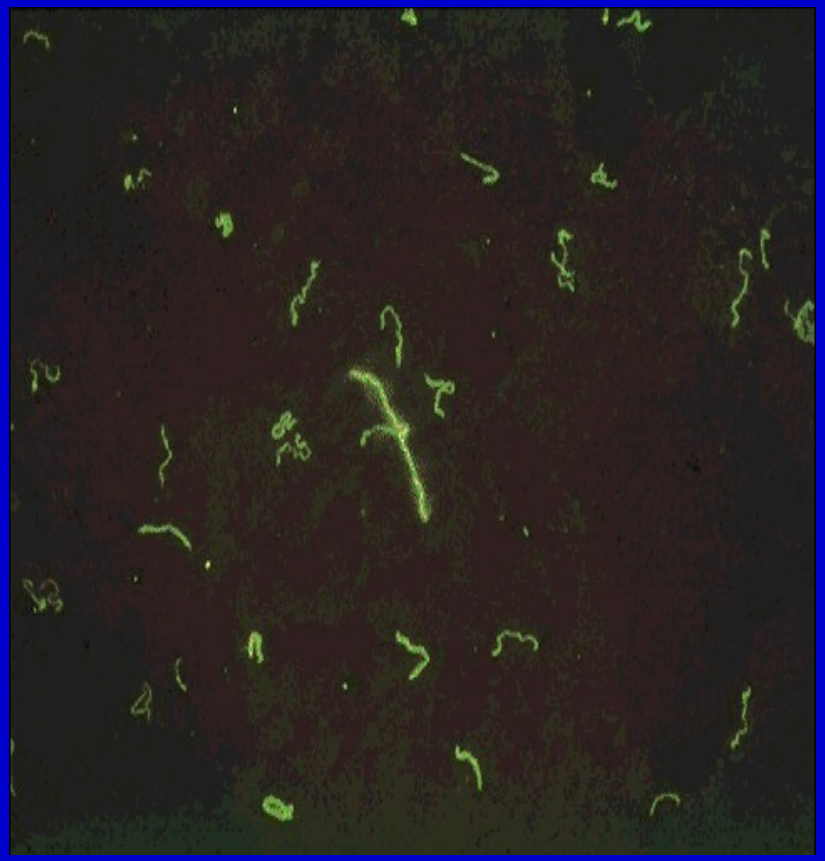


Detection of B. burgdorferi-specific DNA by conventional qualitative PCR and agarose gel electrophoresis.

Diagnostic Workup of Lyme Neuroborreliosis

Techniques have been used to detect antibodies to borrelial antigens:

- immunofluorescence (IFA)



Diagnostic Workup of Lyme Neuroborreliosis

Techniques have been used to detect antibodies to borrelial antigens:

- immunofluorescence (IFA),

- enzyme-linked immunosorbent assays (ELISAs)

- Western blots

comparing specific CSF and serum antibody!

Management of Lyme Neuroborreliosis

ANTIBIOTICS

1. Tetracyclines doxycycline and monocycline.

2. Penicillins amoxicillin and oral penicillin V.

3. Cephalosporins of third (advanced) generation: cefuroxime axetil, ceftriaxone, cefotaxime.

4. Macrolides and azalides (advanced): azithromycin, clarithromycin, erythromycin.

MYELITIS

GENERAL ASPECTS

Myelitis is a general term for inflammatory processes affecting the spinal cord, either in isolation or in the context of a systemic infectious illness, an allergic reaction, or an (allergic) demyelinating disease.

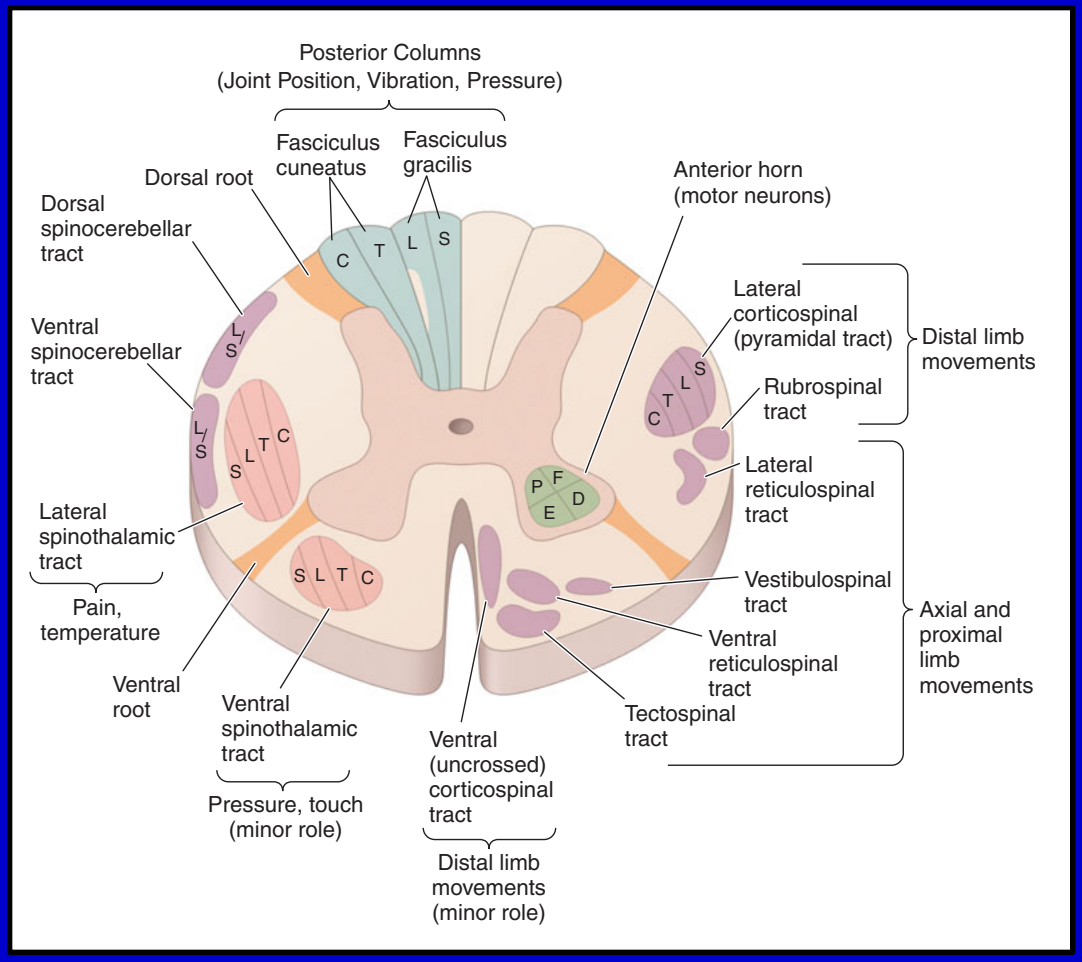
MYELITIS

GENERAL ASPECTS

Most *myelitides* involve multiple spinal cord tracts simultaneously, with or without concomitant encephalomyelitis, and can produce more ore less complete spinal cord transection syndromes, sometimes in more than one level at the same time.

Transverse section through the spinal cord

the principal ascending pathways (*left*)



the principal descending pathways (*right*)

The lateral and ventral spinothalamic tracts (*blue*) ascend contralateral to the side of the body that is innervated. C, cervical; T, thoracic; L, lumbar; S, sacral; P, proximal; D, distal; F, flexors, E extensors.

SPINAL CORD LEVELS RELATIVE TO THE VERTEBRAL BODIES

SPINAL CORD LEVEL	CORRESPONDING VERTEBRAL BODY
Upper cervical	Same as cord level
Lower cervical	1 level higher
Upper thoracic	2 levels higher
Lower thoracic	2 to 3 levels higher
Lumbar	T10-T12
Sacral	T12-L1

MYELITIDIS

- Are frequently devastating.
- They produce quadriplegia, paraplegia, and sensory deficits.
- Many are reversible if recognized and treated at an early stage.

MYELITIS

CAUSES:

- Leptospirosis
- Rickettsial diseases
- Measles
- Mumps
- Herpes simplex
- Other viruses (HIV-1, HTLV-I)
- Postvaccinal (smallpox, rabies)
- Paraneoplastic
- Undetermined

- The pathogen can be determined only by virologic testing.

CHRONIC MYELOPATHIES

RETROVIRUS-ASSOCIATED MYELOPATHIES

The myelopathy associated with the human T cell lymphotropic virus type I (HTLV-I), formerly called tropical spastic paraparesis, is a slowly progressive spastic syndrome with variable sensory and bladder disturbance.

Diagnosis is made by demonstration of HTLV-I–specific antibody in serum by enzyme-linked immunosorbent assay (ELISA), confirmed by radioimmunoprecipitation or western blot analysis.

There is no effective treatment, but symptomatic therapy for spasticity and bladder symptoms may be helpful.

A progressive myelopathy may also result from HIV infection.

Inflammatory and Immune Myelopathies (Autoimmune Myelitis)

This broad category includes MS and postinfectious myelitis, both of which are demyelinating in nature, as well as connective tissue disease.

Recurrent episodes of myelitis are usually due to an immune-mediated disease such as a demyelinating disease, SLE, or sarcoid; or to infection with herpes simplex virus (HSV) type 2.

POLIOMYELITIS

EPIDEMIOLOGY

This endemic and epidemic *enteroviral infection* is spread by oral ingestion of virus particles derived from the stool or respiratory secretions of an infected person.

The virus produces neurologic manifestations in only 1-2% of infected persons. The incidence of poliomyelitis is now practically zero in countries in which active vaccination is practiced.

POLIOMYELITIS

HISTOPATHOLOGY

The disease affects the central gray matter, particularly the anterior horn of the spinal cord, which are acutely lost and replaced with gliotic scar tissue.

POLIOMYELITIS

Clinical Features

- *Incubation period* (3-20 days)

- Febrile *prodromal phase* (several days)

- *Main phase* (fever, a general feeling of illness, headache, and meningeal signs). *Weakness* develops after 1-4 days and progresses over a few hours or days to marked paresis or paralysis. Paresthesiae or other purely sensory abnormalities are not part of the clinical picture, though there may be pain and tenderness in the involved muscles.

POLIOMYELITIS

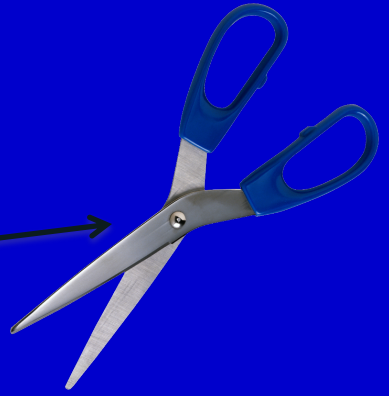
Clinical Features

- *Spinal form (most common)*

- *Predominant brainstem involvement (occasional)*

- *Encephalitic form (extremely rare)*

POLIOMYELITIS



CSF Findings : scissors

Main phase: pleocytosis (100 or > cells/1 mm^3) – predominantly polymorphonuclear neutrophils at first, with a rapid transition to lymphocytic predominance.

The cell count falls over the ensuing 1-2 weeks, while the SCF protein concentration rises: "*albumino-cytologic dissociation*".

POLIOMYELITIS

Prognosis & Evolution

Cases with brainstem involvement and respiratory paralysis: mortality 50%

Paralysis usually resolves incompletely, leaving a variable degree of residual weakness, muscular atrophy, and areflexia, as well as stunted growth of the affected limb(s) if the illness strikes in early childhood.

POLIOMYELITIS

Vaccination

Oral vaccine of Sabin.

Subcutaneous immunization with inactivated polioviruses (e. g., Salk vaccine), followed by a first booster in 6–8 weeks and a second booster in 8–12 months.

Paralysis as a complication of vaccination is exceedingly rare.

Following recovery, as many as 20% to 30% of individuals who develop paralytic poliomyelitis may suffer from *post-polio syndrome*, which produces muscle weakness, pain, atrophy, and fatigue many years after acute illness.

POLIOMYELITIS summary

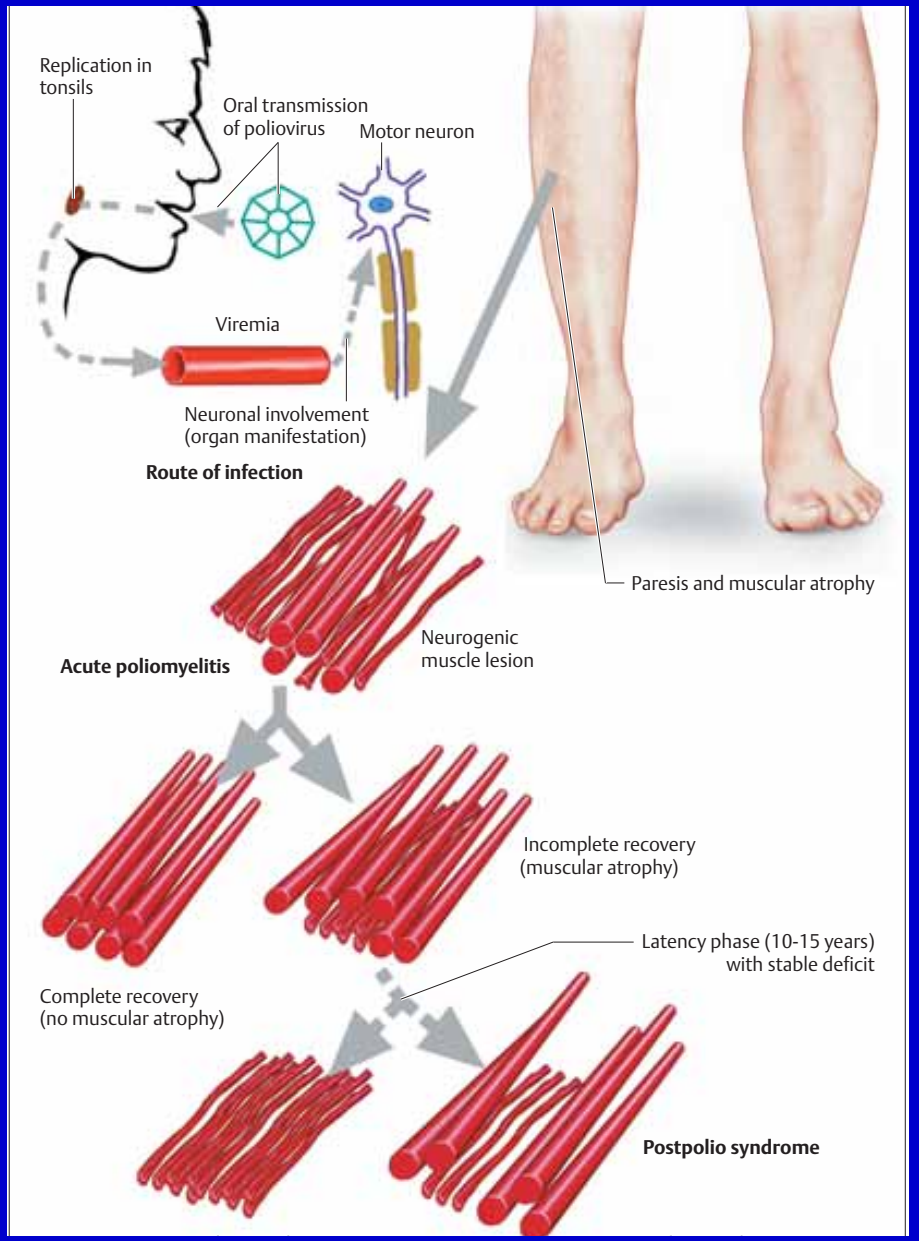
Key points:

Enterovirus

Aseptic meningitis, polioencephalitis, bulbar poliomyelitis, and paralytic poliomyelitis

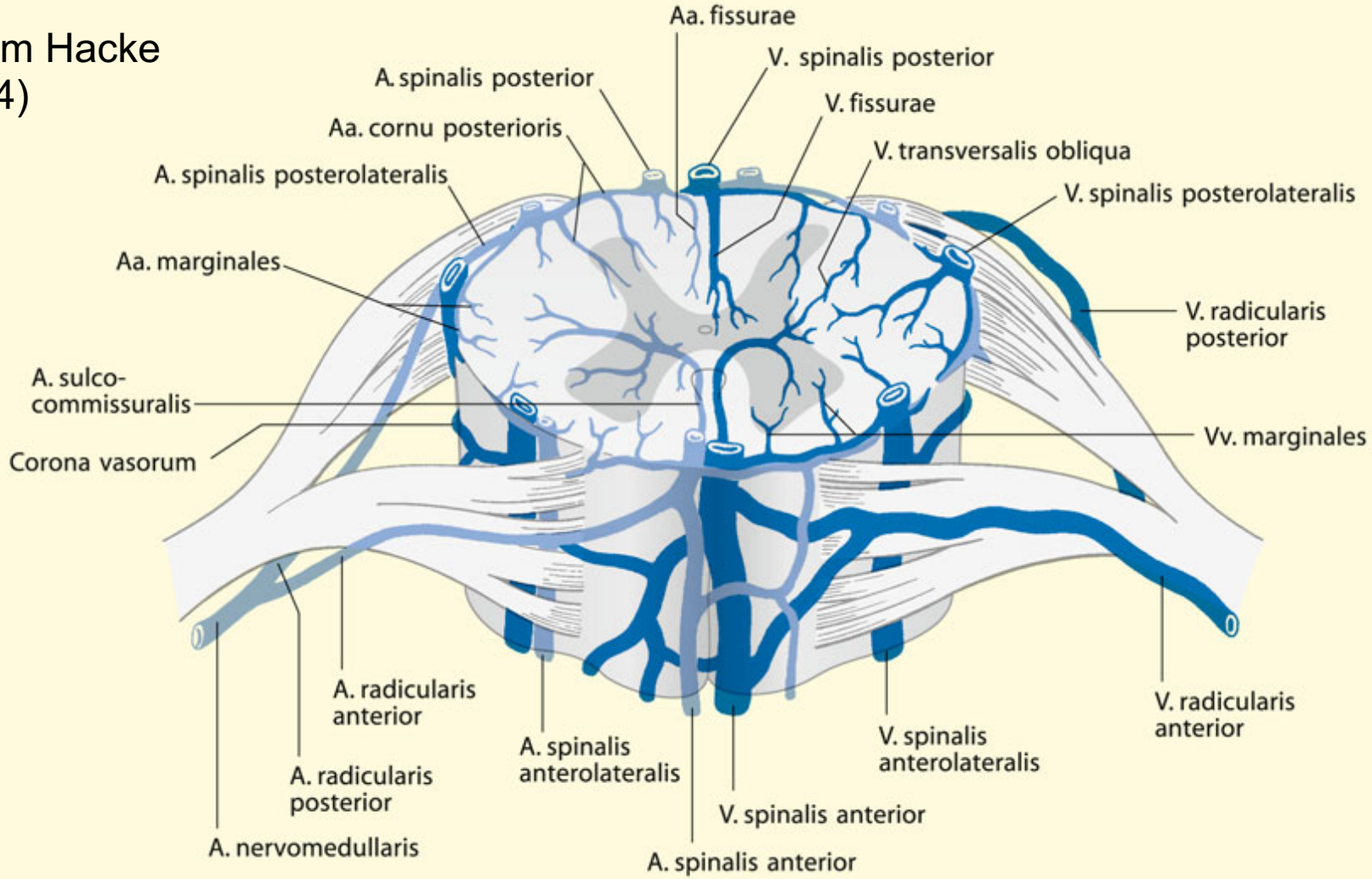
post-polio syndrome,

There has been a large worldwide effort for poliomyelitis eradication, with a decrease of polio cases by over 99% since 1988



spinal cord blood supply

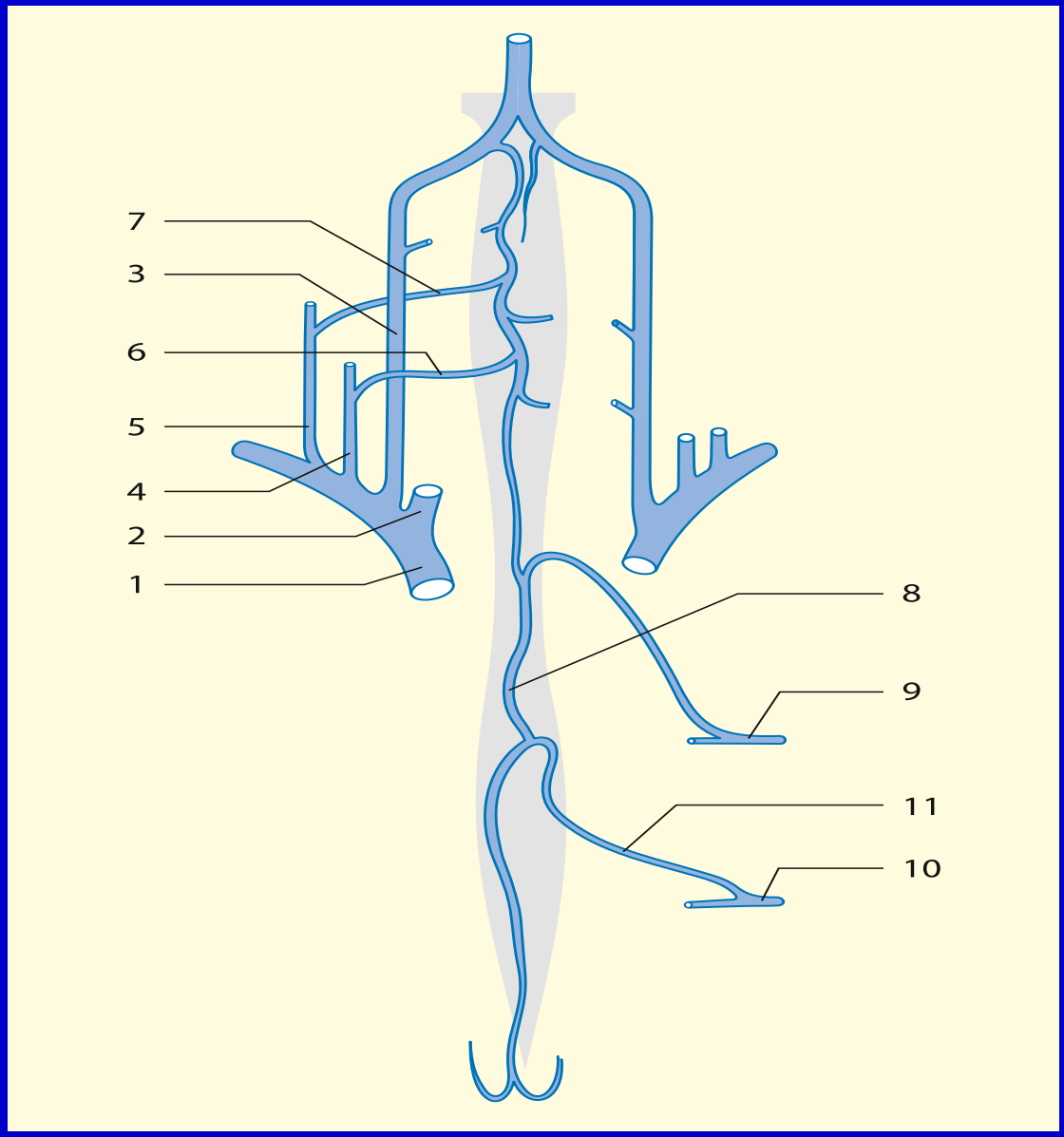
(From Hacke 1994)



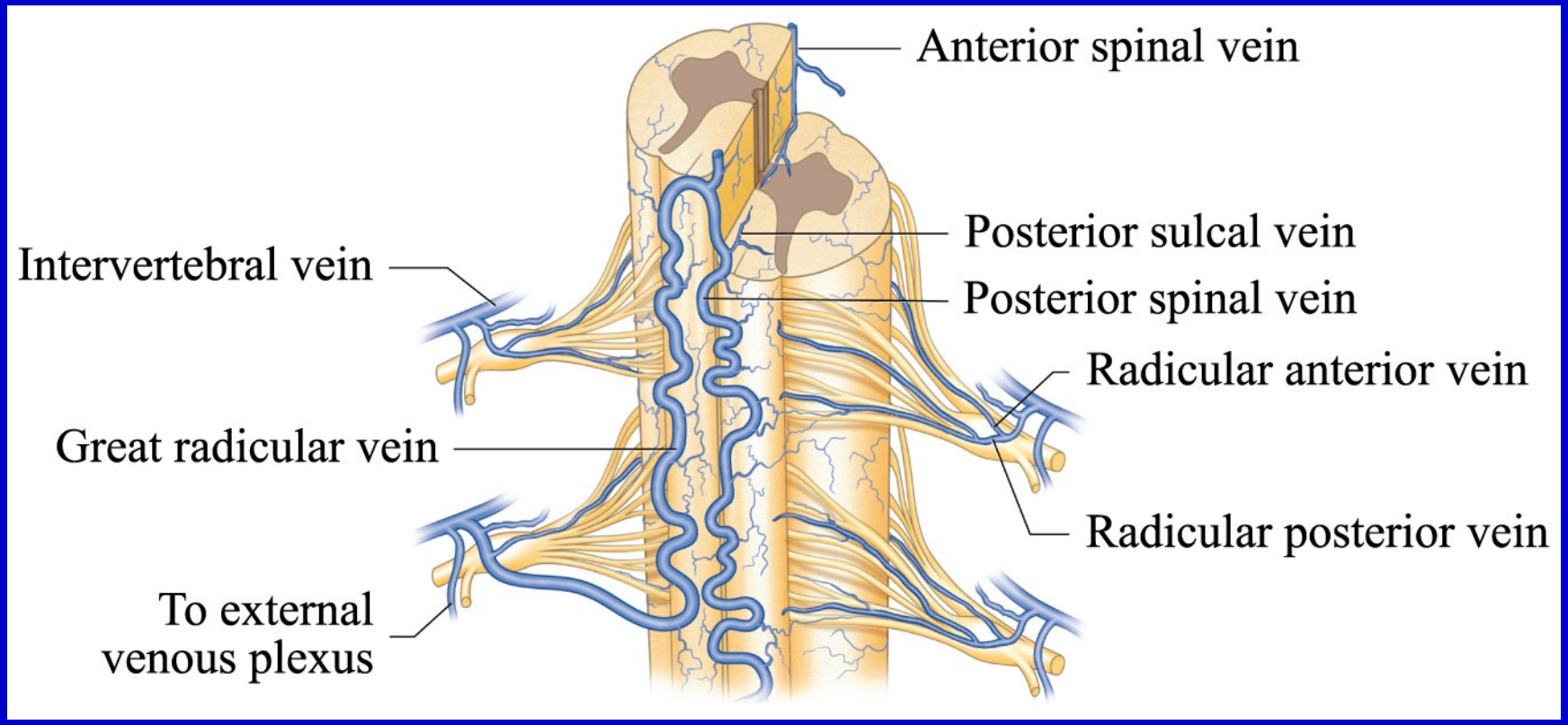
spinal cord blood supply

1 – Truncus brachio-cephalicus
2 – A. carotis
3 – A. vertebralis
4 – Truncus thyreocervicalis
5 – Truncus costocervicalis
6 – A. radicularis anterior C6-C8
7 – A. radicularis anterior C4-C5
8 – A. spinalis anterior
9 – A. intercostalis posterior Th4-Th6
10 – A. intercostalis posterior Th9-L1
11 – A. radicularis magna Adankiewicz

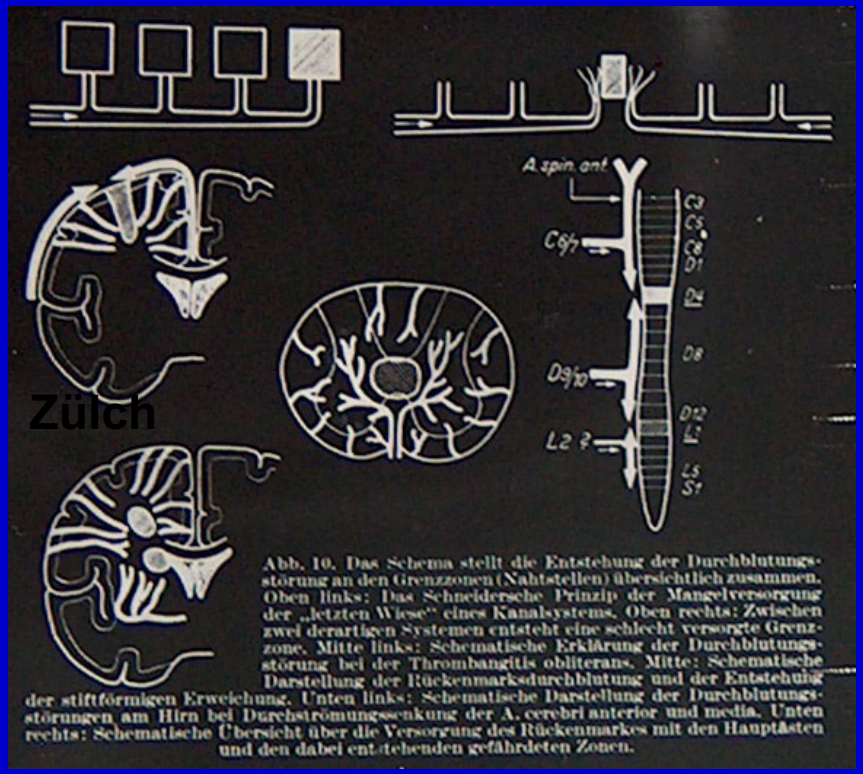
(Thron, Aachen)



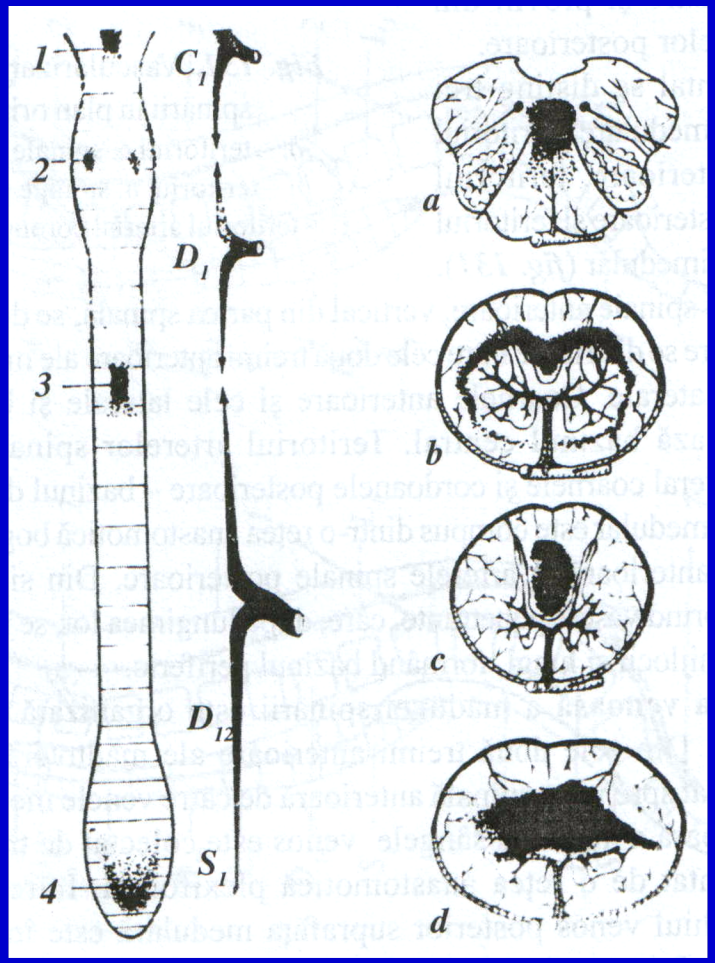
spinal vascular anatomy



spinal cord's ischemia: watershed zones



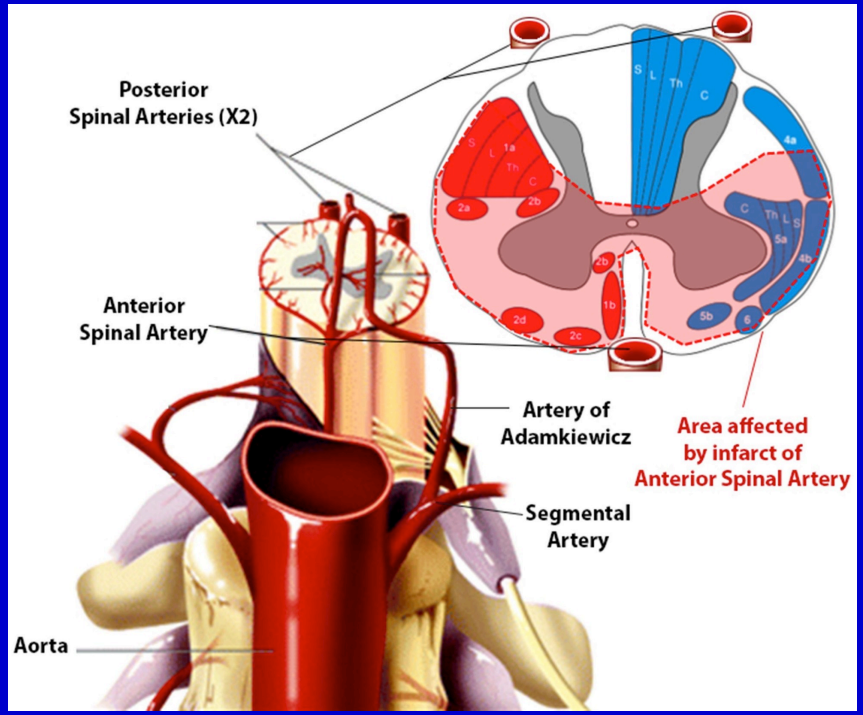
Zülch, 1951



Gherman, 1964

spinal cord infarction/transient ischemia : territorial syndromes

- Anterior spinal artery syndrome
- Posterior spinal arteries syndrome
- Transverse infarction of the spinal cord
- Central cord infarction
- Venous infarction
- Lacunar cord infarction



causes of spinal cord infarction

Aorta disease	**Aortic dissection, traumatic rupture of the aorta, aortic thrombosis, aortic aneurysm, coarctation of the aorta**
Aortic procedures	**Aortic surgery, aortography, left subclavian coverage**
Systemic hypoperfusion	**Cardiac arrest, systemic bleeding**
Cardiogenic embolism	**Atrial myxoma, mitral valve disease, patent foramen ovale, bacterial endocarditis, cardiac catheterization**
Vasculitis	**Systemic lupus erythematosus, polyarteritis nodosa, Behçet's disease, giant cell arteritis, varicella-zoster virus, primary angiitis of the CNS**
Infection	**Bacteria, borreliosis, syphilis, viruses, mucomycosis, TB**
Hematologic disease	**Hypercoagulable condition, sickle cell anemia**
Spine disease	**Cervical spondylosis, fibrocartilagenous embolism**
Nonaortic surgeries	**Spine surgery, epidural steroid injections**
Miscellaneous: nonsurgical	**Cocaine abuse, vertebral artery dissection, spinal vascular malformation, decompression sickness**
Miscellaneous: surgical	**Sympathectomy, subclavian artery catheterization, celiac plexus neurolysis, lumbar epidural anesthesia, renal artery embolization, single radicular artery ligation, thoracoplasty, postcaval shunt placement, intrathecal injection of lidocaine or phenol**

Nardone, R *et al.* (2016) Current and emerging treatment options for spinal cord ischemia. *Drug Discovery Today*, 10, 1632-1641.

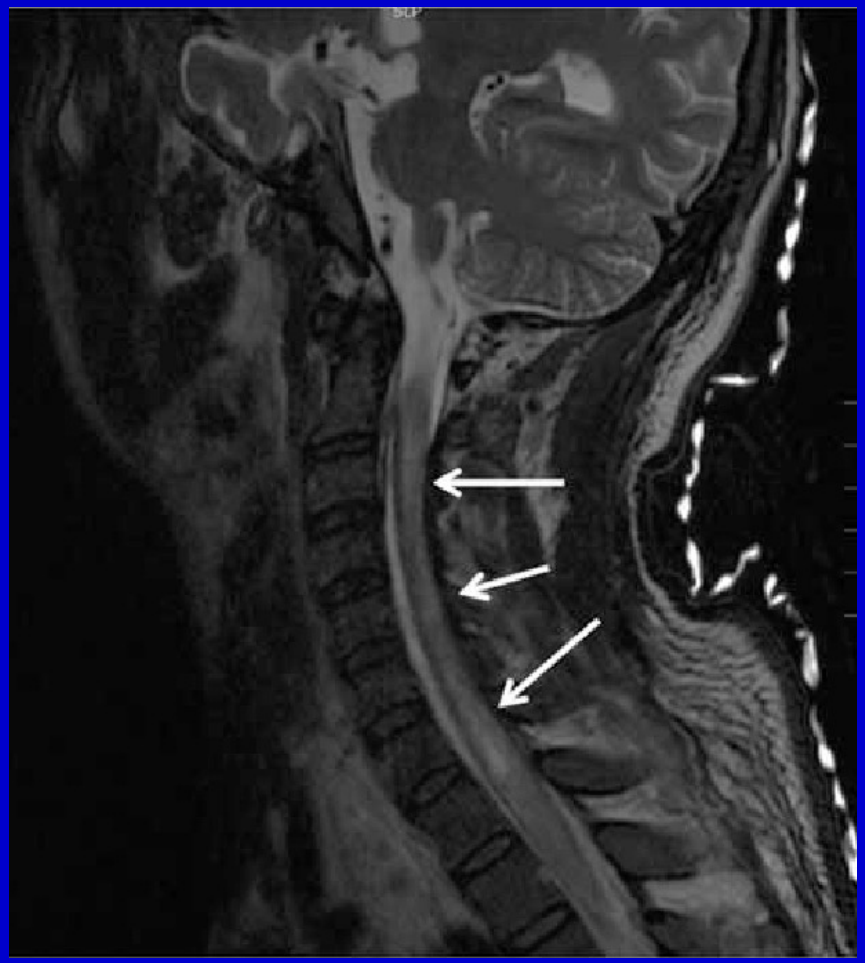
Spinal Cord Infarction

With systemic hypotension, cord infarction occurs at the level of greatest ischemic risk, usually T3 - T4, and also at boundary zones between the anterior and posterior spinal artery territories.

Onset may be sudden and dramatic but more typically is progressive over minutes or a few hours, quite unlike stroke in the cerebral hemispheres.

Spinal Cord Infarction

Acute infarction in the territory of the *anterior spinal artery* produces paraplegia or quadriplegia, dissociated sensory loss affecting pain and temperature sense but sparing vibration and position sense, and loss of sphincter control (“anterior cord syndrome”).



Sagittal T2-wighted MRI

Anterior Spinal Artery Syndrome

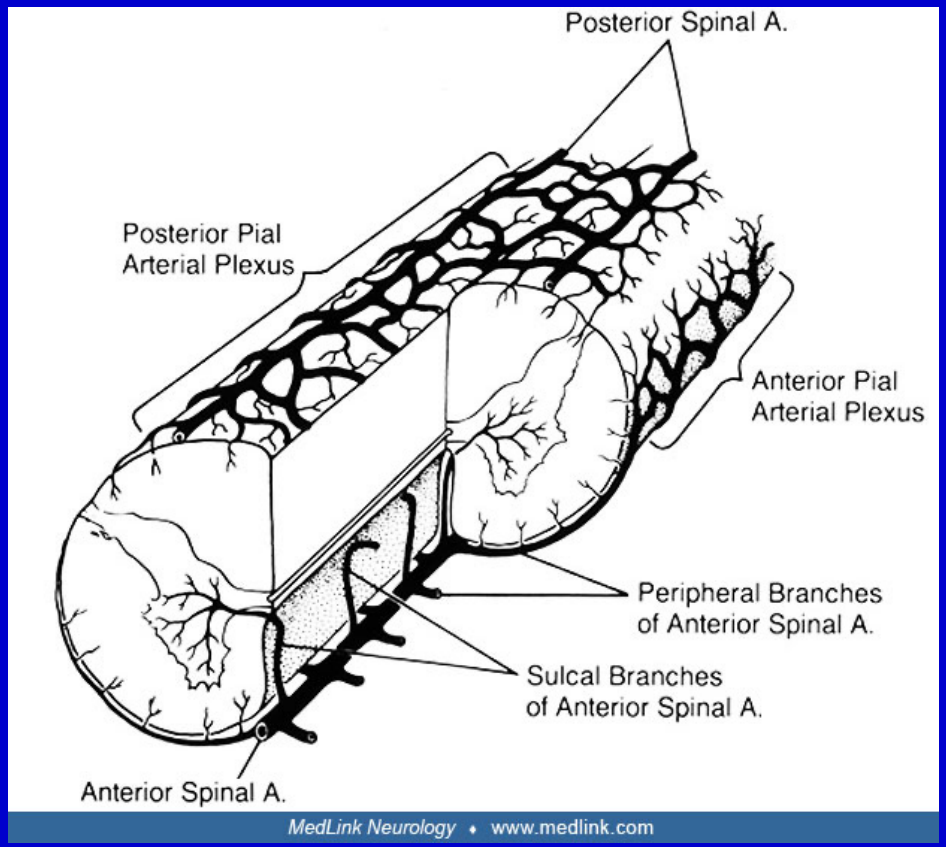
Infarction of the cord is generally the result of occlusion or diminished flow in this artery.

The result is extensive bilateral tissue destruction that spares the posterior columns.

All spinal cord functions—motor, sensory, and autonomic—are lost below the level of the lesion, with the striking exception of retained vibration and position sensation.

Posterior Spinal Arteries Syndrome

Less common is infarction in the territory of the *posterior spinal arteries,* resulting in loss of posterior column function.



Spinal Cord Infarction treatment

In cord infarction due to presumed thromboembolism, acute anticoagulation is probably not indicated, with the exception of the unusual transient ischemic attack or incomplete infarction with a stuttering or progressive course.

The antiphospholipid antibody syndrome is treated with anticoagulation.

Drainage of spinal fluid has reportedly been successful in some cases of cord infarction but has not been studied systematically.

You are tired. I know.

PROTECT YOUR BRAIN
and
SPINAL CORD!


merci
obrigado
grazie
gracias
kiitos
спасибі
danke
tack
köszönöm
хвала
teşekkür ederim
ačiū
hvala vam
Dank u
tak
þakka þér
ddiolch 'ch
σας ευχαριστώ
thank you
dziękuję
děkuji
Takk
Ďakujem
hvala
ありがとう
תּוֹדָה
Tapadh leibh
falemnderit
благодаря
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спасибо
mulţumesc
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trugarez
Ви благодарам
谢谢
धन्यवाद
Go raibh maith agaibh
Paldies