VIRAL MENINGITIS

DEPARTMENT OF GENERAL MEDICINE

MADURAI MEDICAL COLLEGE

- Viral meningitis is inflammation of the leptomeninges as a manifestation of CNS infection. Viral meningitis syn. aseptic meningitis
  .
- In uncomplicated viral meningitis, the clinical course is usually self-limited, with complete recovery in 7-10 days.

  complicated- meningoencephalitis or meningomyelitis, the course can be significantly more protracted.

- Enteroviruses account for more than 85% of all cases of viral meningitis.
- Herpes family viruses: HSV-1, HSV-2, VZV, EBV, CMV, and human herpesvirus 6 collectively cause approximately 4% of cases of viral meningitis, with HSV-2 being the most common offender.

Meningitis

Inflammation of meninges

ENCEPHALITIS

Inflammation of brain parenchyma

Inflammation of Meninges.

Leptomeningitis – Subarachnoid & Pia.
Pachymeningitis – Dura (Local trauma)
Meningoencephalitis – + Brain.

Aetiologic Types:

Infective

- Bacterial
- Viral
- Fungal
- Ricketsial
- Parasitic/Protozoal

Chemical – Drugs.

Carcinomatous – metastasis.

- Viral
  - HSV especially in infants
  - Enterovirus (coxsackie, echovirus)
  - HIV
  - Lymphocytic choriomeningitis virus
  - Arbovirus
  - Mumps
  - CMV
  - EBV
  - VZV
  - Adenovirus
  - Measles
  - Rubella
  - Rotavirus
  - Influenza and parainfluenza

AT RISK

- The incidence of viral meningitis drops with age.
- Neonates are at greatest risk and have the most significant risk of morbidity and mortality.

- The incidence during the first year of life is 20 times higher than in older children and adults

AT RISK

- Asplenia
- Drugs that suppress immune system
- Immunocompromised
- HIV, malignancy
- Malignancy
- Alcoholism

AT RISK

- Patients with cardiac and pulmonary anomalies may spread septic foci,bronchiectasis, occasionally pneumonia). Common with pulmonary AV fistulae and R to L cardiac defect
- T-cell defects (HIV)
- Neural tube defects- Staph aureus, enteric organisms
- Terminal compliment deficiency- Neisseria
- septic foci from thrombophlebitis
- iv drug abusers

CONDUCIVE ENVIRONMENT

- **Airborne**- when the person coughs, laughs, talks, or sneezes
- **close contact** -spread between people who are in (crowded day-care situation or a military recruit camp, schools, colleges)
- **Sharing-** food, drinking glasses, eating utensils, tissues, or towels
- **poor hygiene-** most commonly enteroviruses that normally live harmlessly in people's bowels. someone who comes in contact with the stool — such as a child in day care

ROUTE OF ENTRY

- **Hematogenous**
- infection of the skin, urinary system, gastrointestinal or respiratory tract can spread by the bloodstream
- **Colonization** of nasopharynx by bacteria or viruses

- **Direct infection**
- skull fractures possess abnormal openings to the sinuses, nasal passages, and middle ears.
- surgical procedures or who have had foreign bodies surgically placed within their skulls.

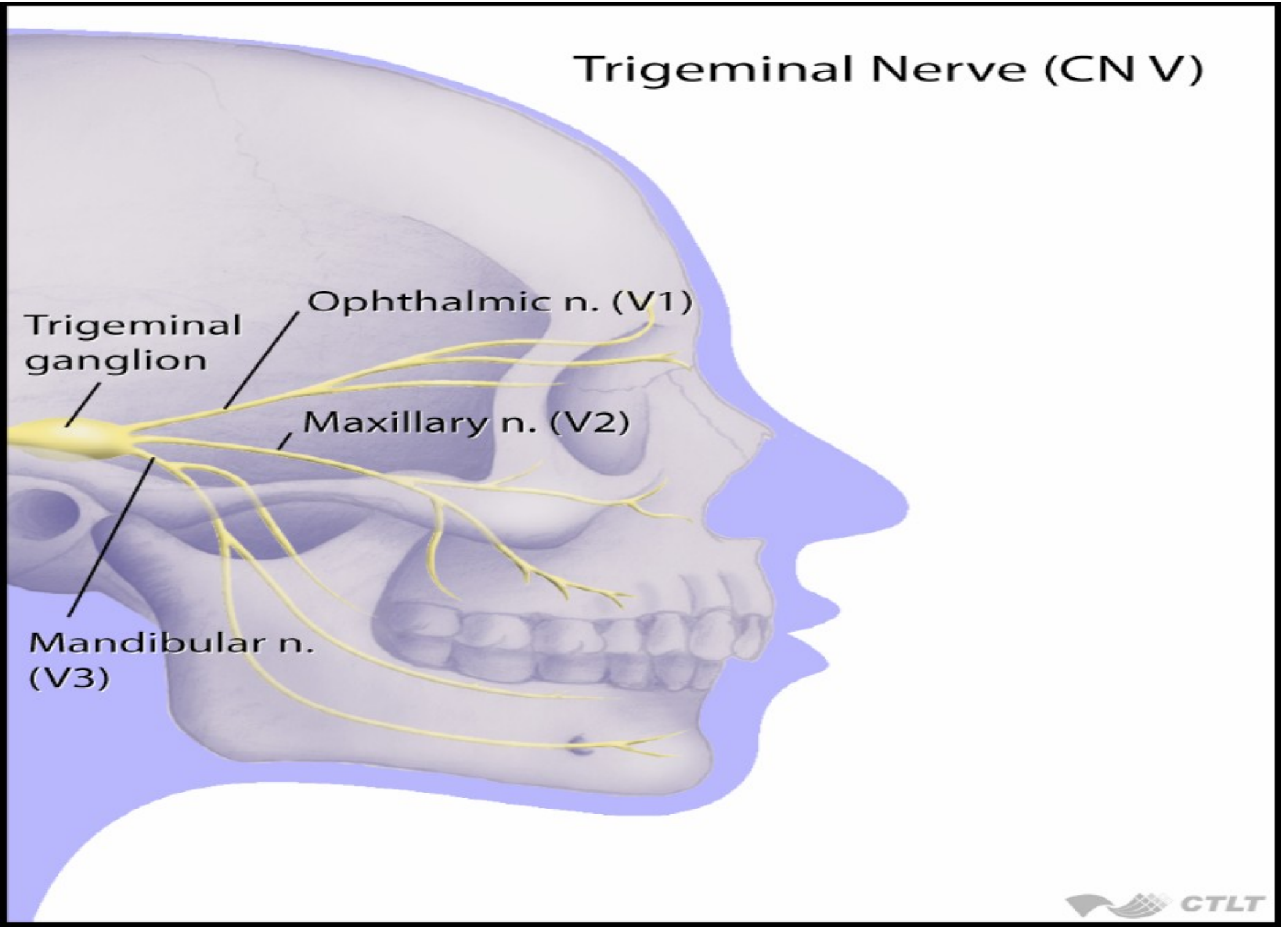
- otitis media, mastoiditis, Osteomyelitic foci in the skull, sinusitis, Penetrating cranial injuries, Brain or spine surgery, Ventriculo-peritoneal shunt, lumbar puncture.

- Extradural abscess, Subdural empyaema, Spinal epidural abscess

ROUTE OF ENTRY

- Neurotropic
- intraneural spread-- spreading along a nerve, and using that nerve as a kind of ladder into the skull, where the organism can multiply and cause meningitis.

  (eg)
- Herpes simplex virus
- Rabies
- HSV


Trigeminal Nerve (CN V)
Trigeminal ganglion
Ophthalmic n. (V1)
Maxillary n. (V2)
Mandibular n. (V3)

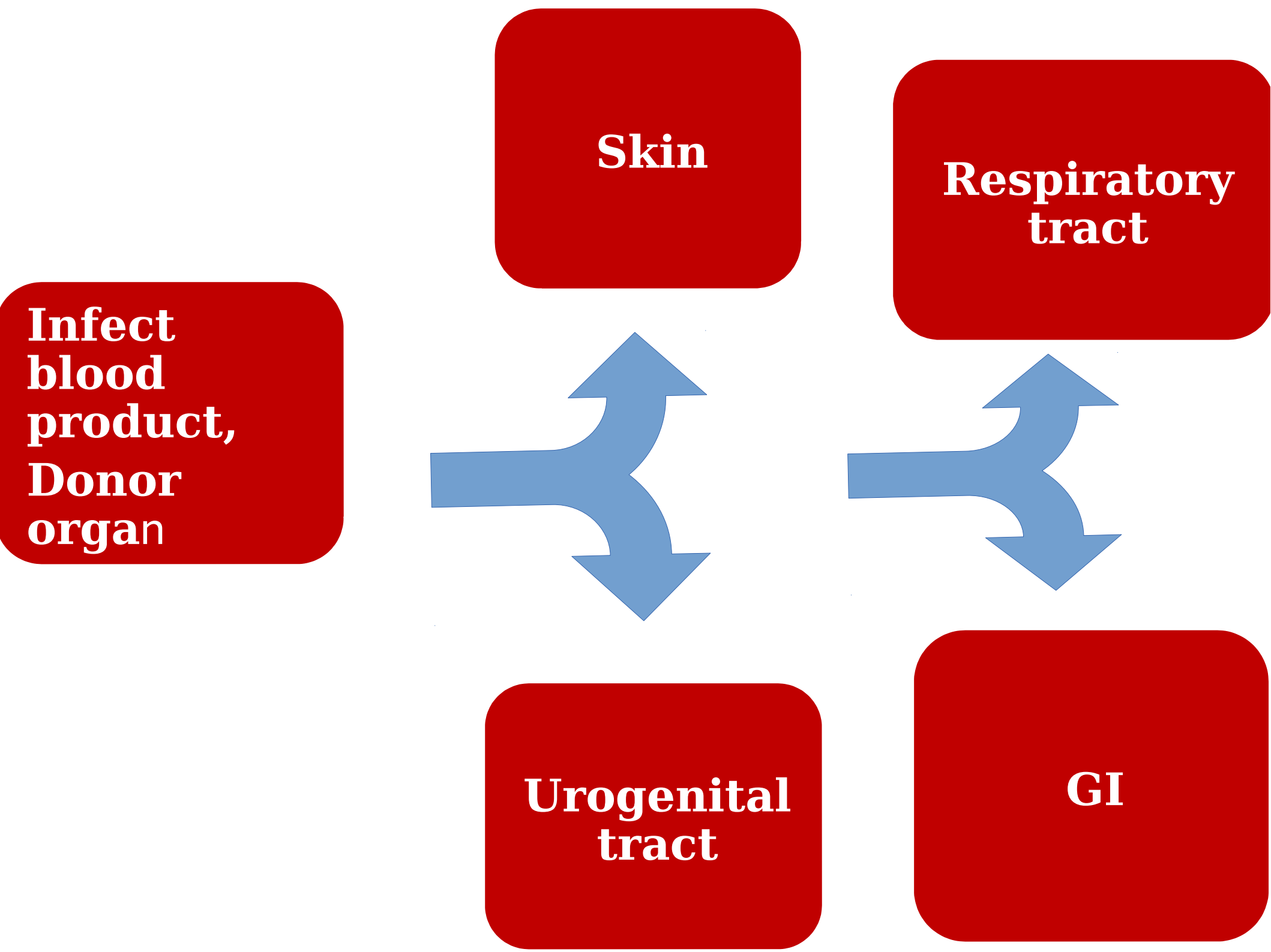
pathophysiology

Pathophysiology

- Multiple host defenses (local and systemic immune responses, skin and mucosal barriers, and the blood-brain barrier) prevent viral inoculum from causing clinically significant infection.

Mortality/Morbidity

- Excluding the neonatal period, the mortality rate associated with viral meningitis is less than 1%; the morbidity rate is also low.


Skin
Respiratory tract
Infect blood product, Donor organ
Urogenital tract
GI

Pathogenesis of Meningitis

- The virus or bacteria replicates in the initial organ system (ie, respiratory or gastrointestinal mucosa) and gains access to the bloodstream. Primary viremia or bacteremia introduces the virus or bacteria to the reticuloendothelial organs (liver, spleen, and lymph nodes.)

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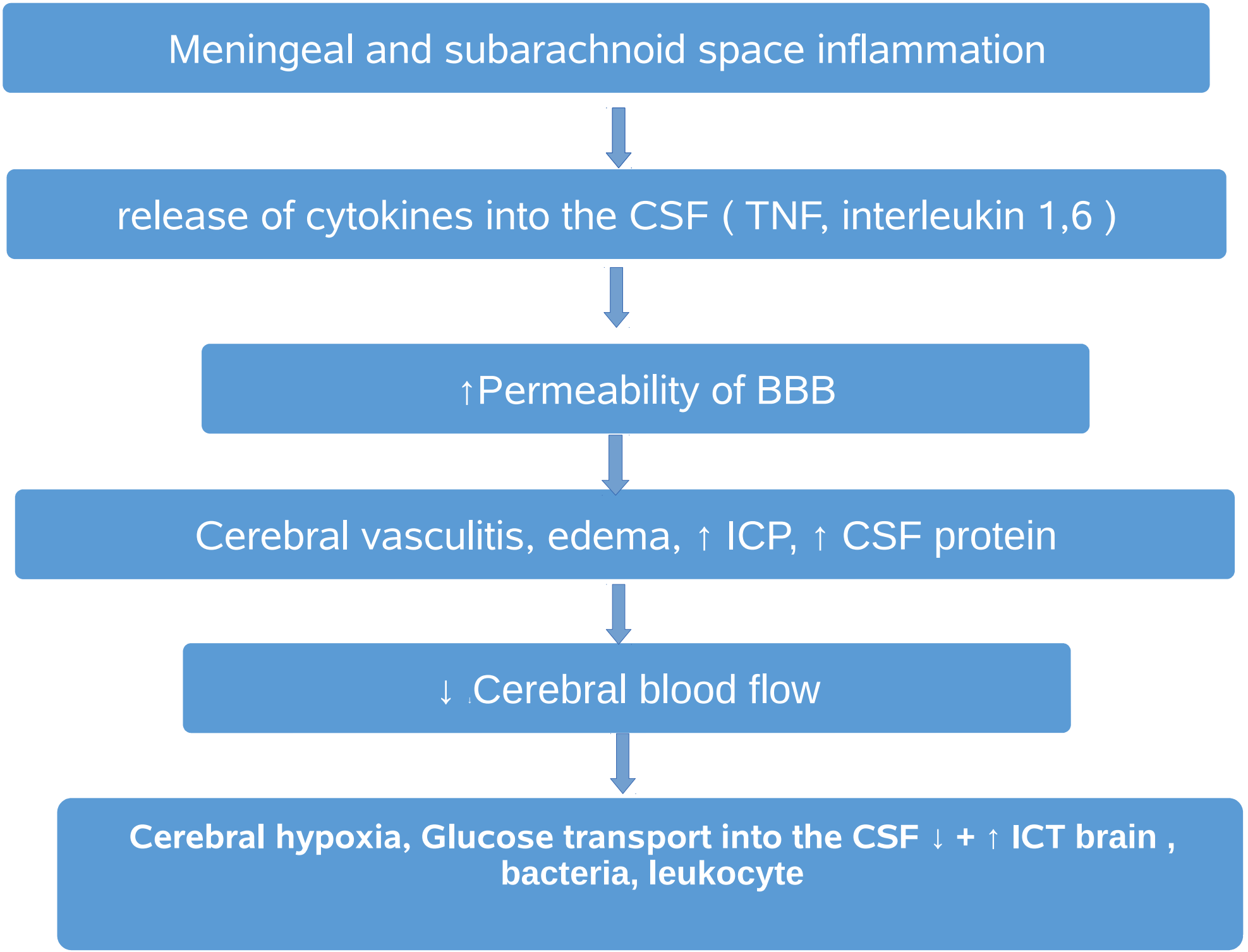
Meningeal and subarachnoid space inflammation
release of cytokines into the CSF (TNF, interleukin 1,6)
↑Permeability of BBB
Cerebral vasculitis, edema, ↑ ICP, ↑ CSF protein
↓ Cerebral blood flow
Cerebral hypoxia, Glucose transport into the CSF ↓ + ↑ ICT brain , bacteria, leukocyte

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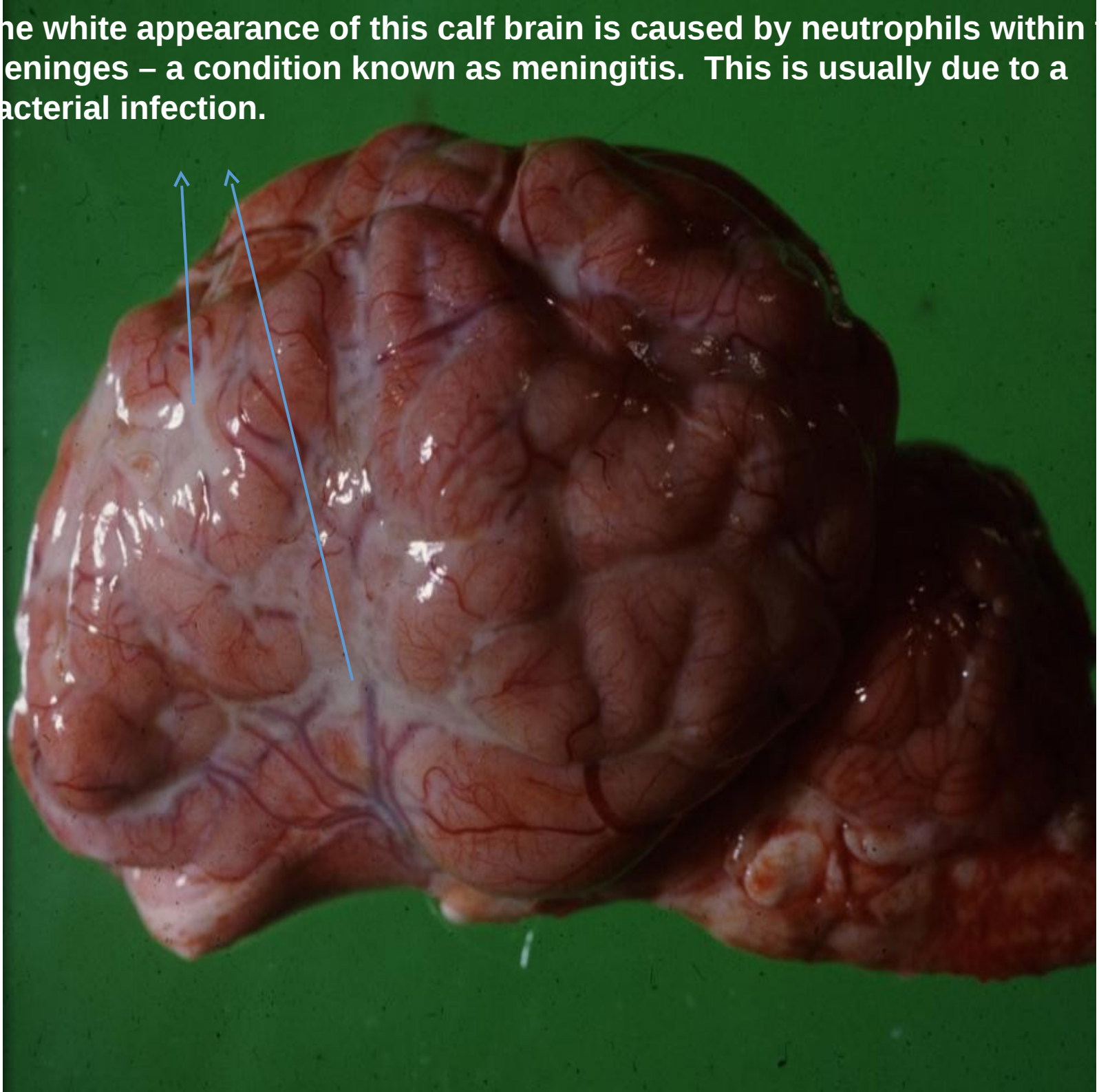
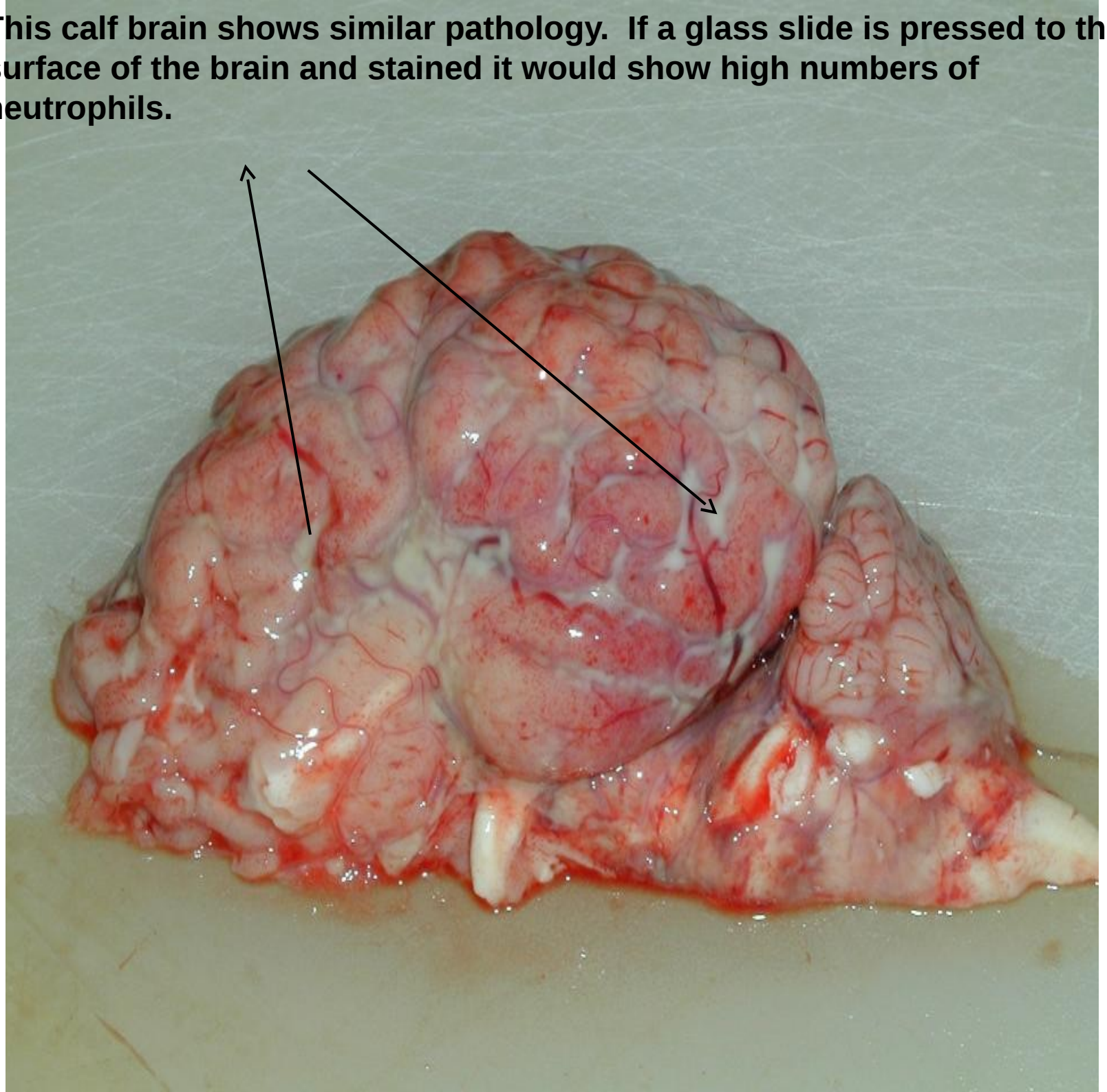
PATHOLOGY

he white appearance of this calf brain is caused by neutrophils within
eninges – a condition known as meningitis. This is usually due to a
acterial infection.

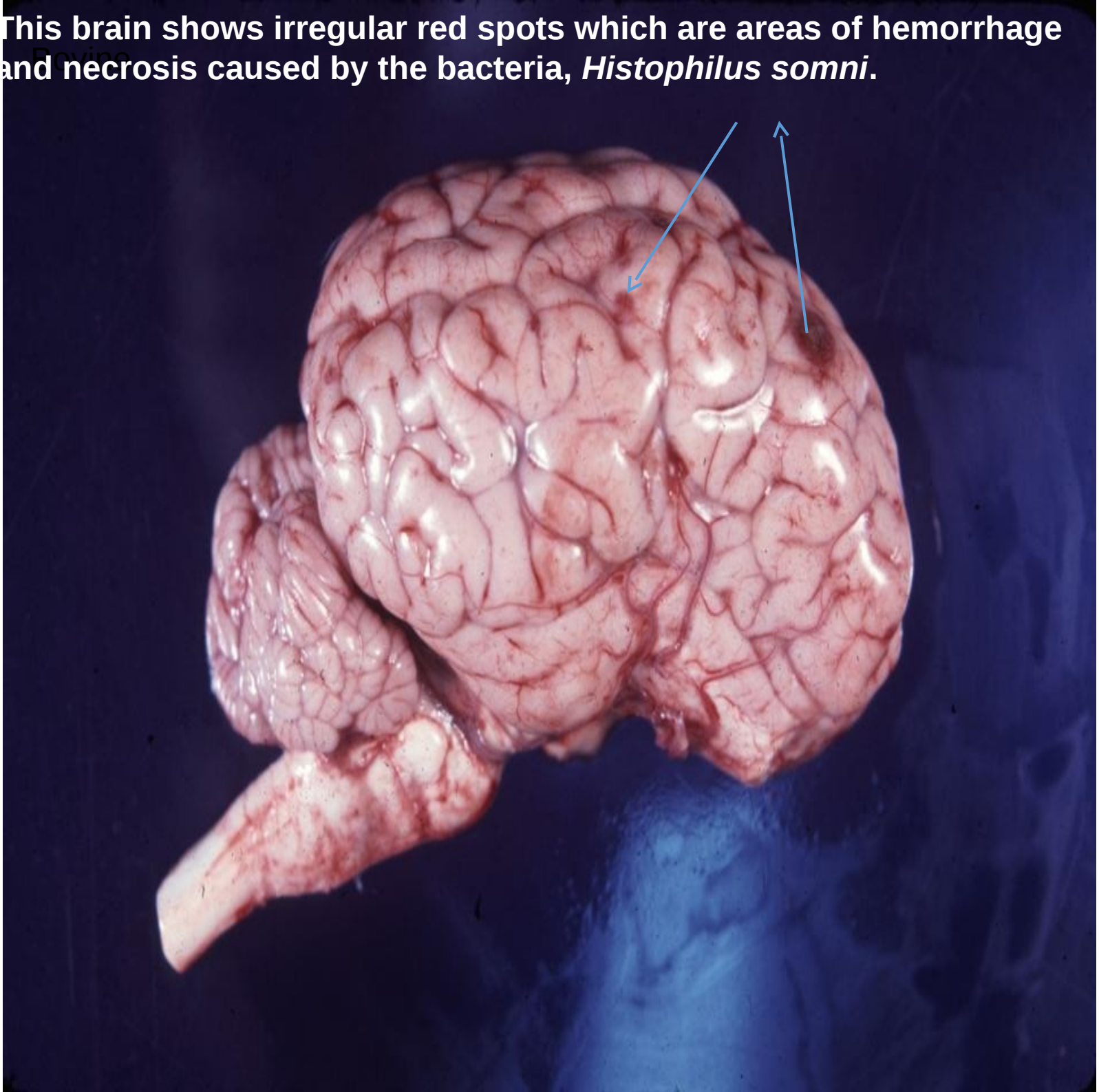


This calf brain shows similar pathology. If a glass slide is pressed to the surface of the brain and stained it would show high numbers of neutrophils.



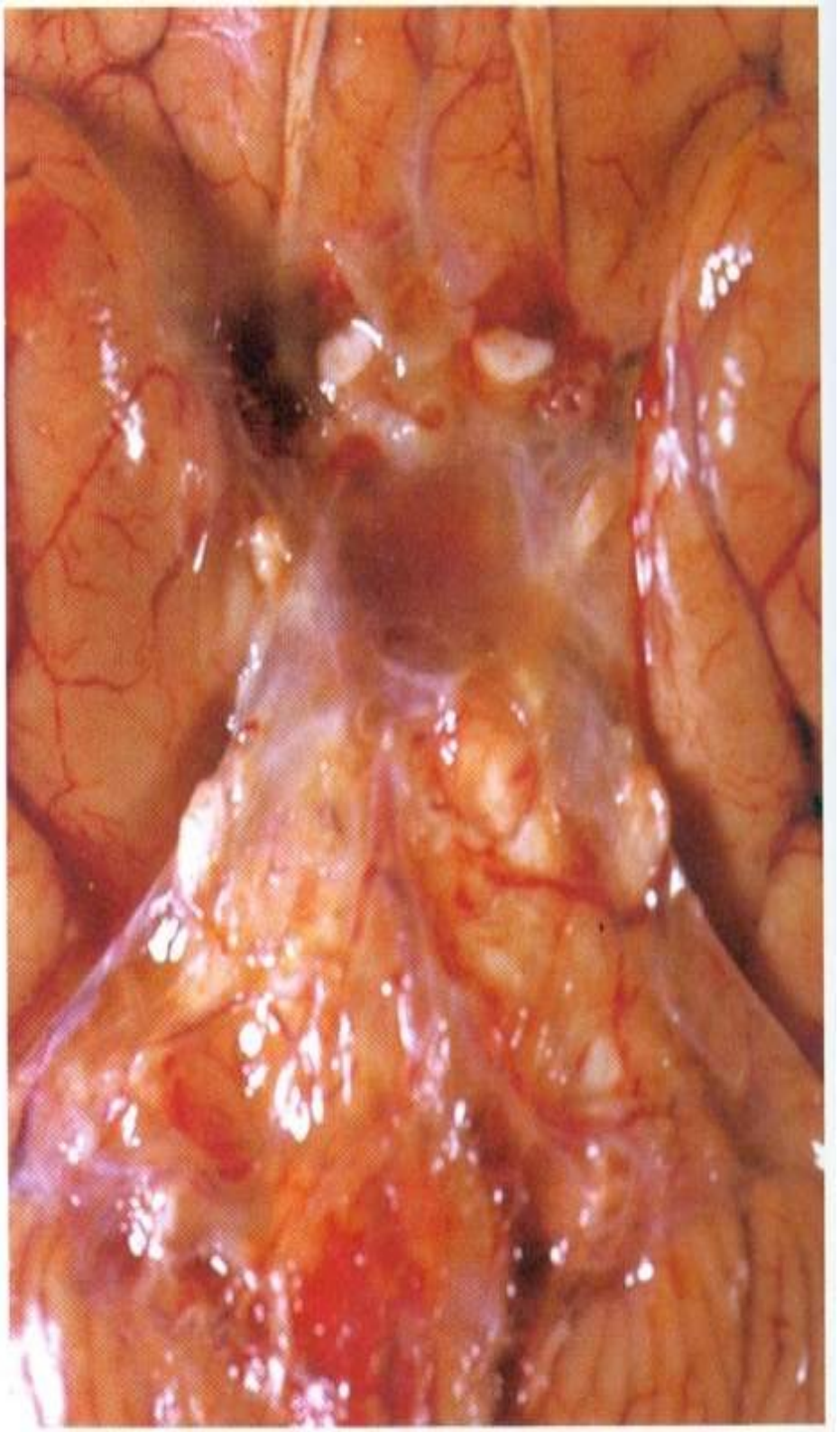
Bovine

This brain shows irregular red spots which are areas of hemorrhage and necrosis caused by the bacteria, ***Histophilus somni***.



Brain:

meningitis.



Symptoms and signs

Signs & symptoms of Meningitis

- Non-specific complaints
  - Fever
  - Headache
  - Nausea and vomiting

  --Nuchal rigidity
  - Lethargy
  - Irritability
  - Restlessness
  - Poor feeding
  - Back pain
  - Altered mental status (seizure, coma)

Other signs of specific viral infection can aid in diagnosis:

Pharyngitis and pleurodynia in enteroviral infections

Skin manifestations, such as zoster eruption in VZV,

maculopapular rash from measles and enteroviruses,

vesicular eruption by herpes simplex,

herpangina in coxsackievirus A infections.

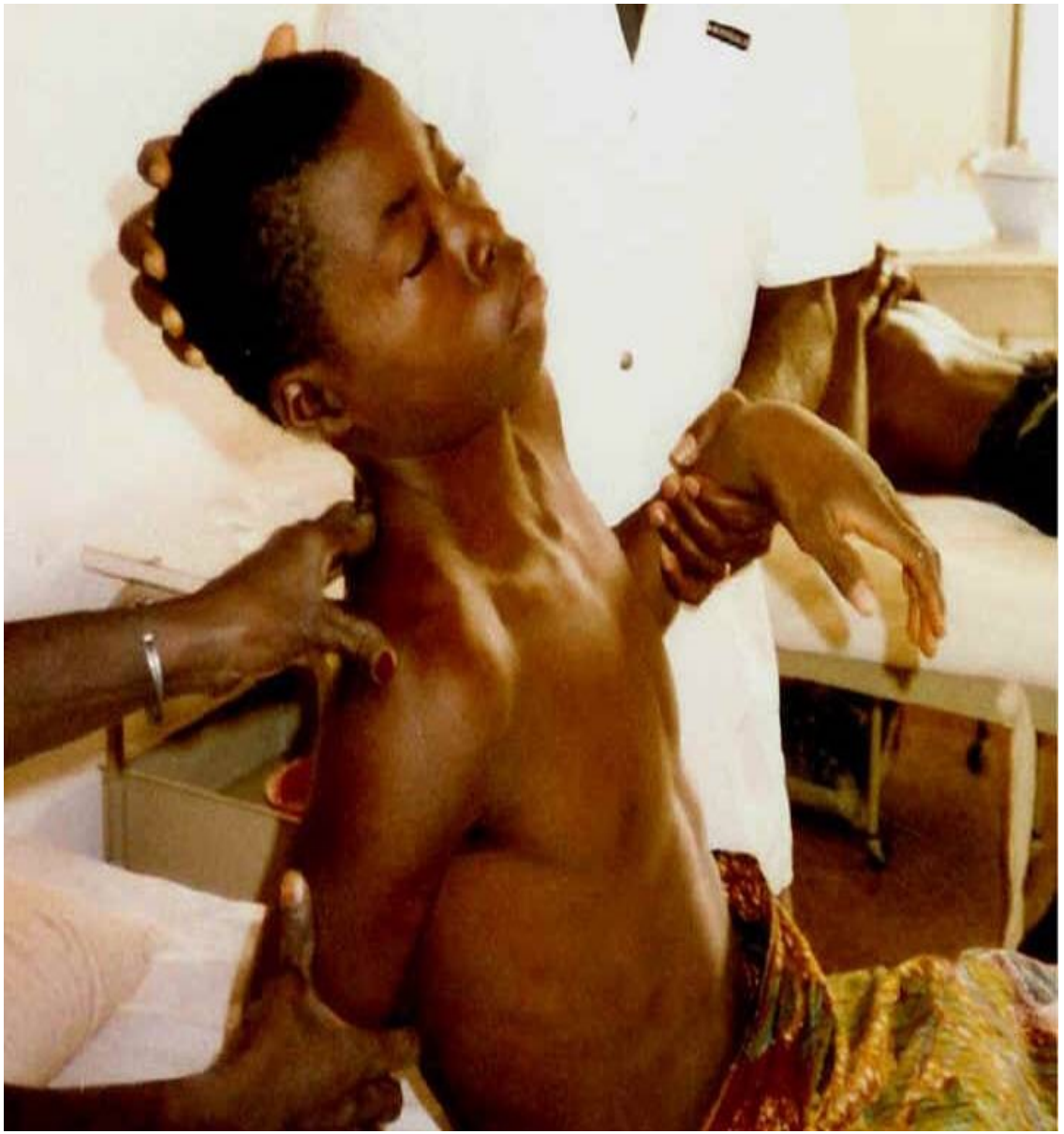
Pharyngitis, lymphadenopathy, and splenomegaly suggest Epstein-Barr virus infection.

Immunodeficiency and pneumonia should suggest adenovirus, cytomegalovirus , or HIV as the causative agent.

Parotitis and orchitis can occur with mumps

Gastroenteritis and rash occur with most enteroviral infections.

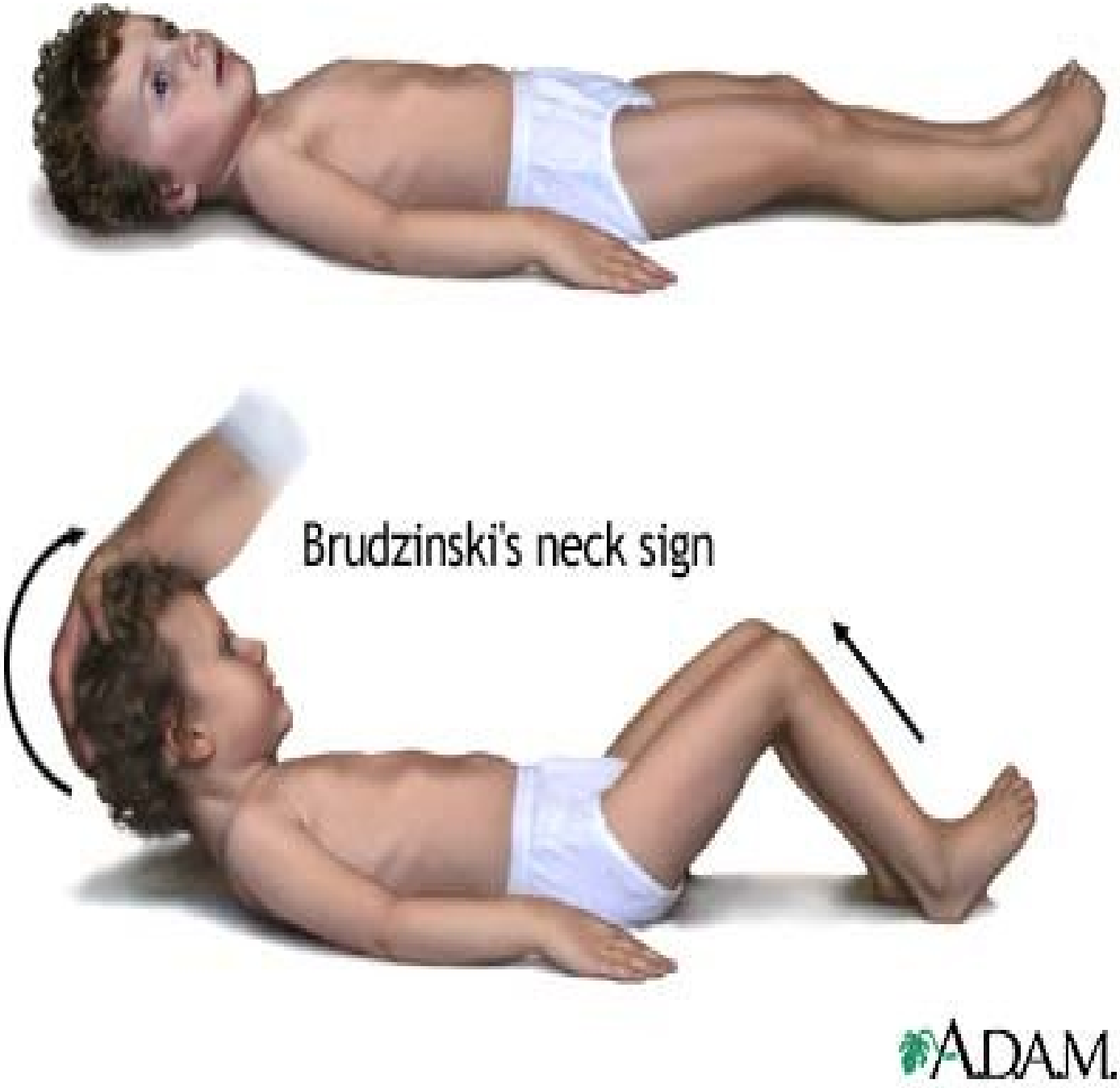
Neck Stiffness:



* Pathogenesis: Meningeal irritation

Kernig's Sign

- Patient placed supine with hips flexed 90 degrees. Examiner attempts to extend the leg at the knee
- Positive test elicited when there is resistance to knee extension, or pain in the lower back or thigh with knee extension

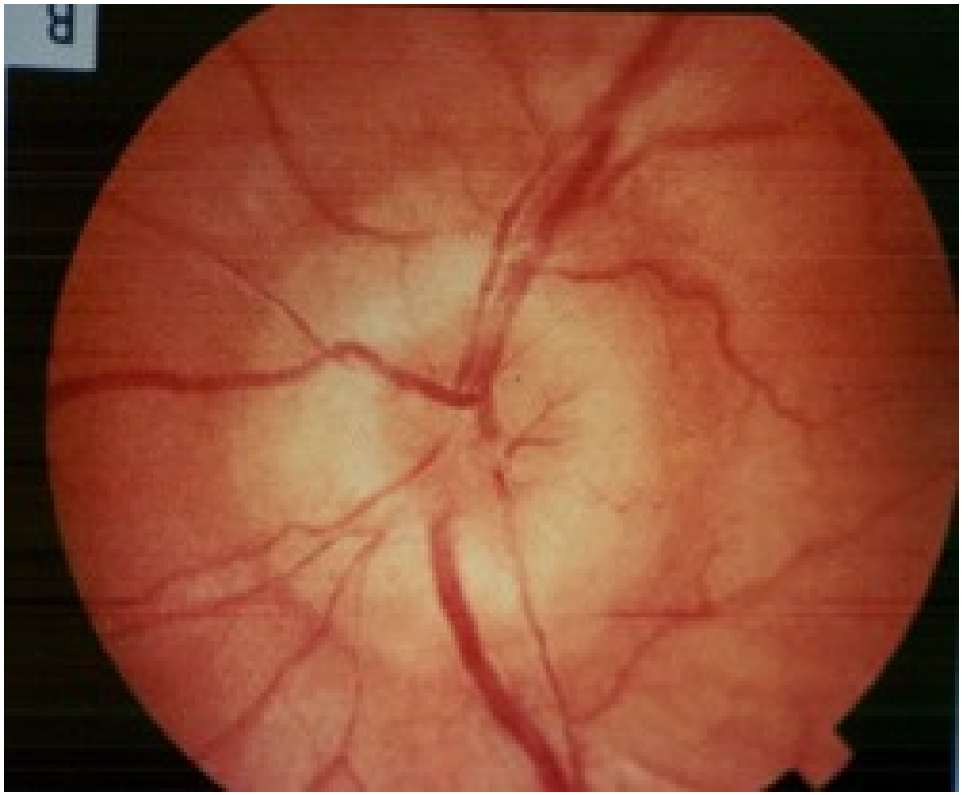

Brudzinski's neck sign
A.D.A.M.

Brudzinski's Sign

- Patient placed in supine position and neck is passively flexed towards the chest
- Positive test is elicited when flexion of neck causes flexion at knees and/or hips of the patient

Normal fundus

papiloedema



Increased intracranial pressure (ICP)

- Papilledema
- Cushing's triad
  - Bradycardia
  - Hypertension
  - Irregular respiration
  - Severe headache
  - herniation
  - vomitting
- ICP monitor (not routine)
- Changes in pupils


MD Challenger Sample Photo

Petechiae

- Is due to small skin bleed
- All parts of the body are affected
- The rashes do not fade under pressure

- Pathogenesis:

a. Septicemia
b. wide spread endothelial damage
c. activation of coagulation
d. thrombosis and platelets aggregation
e. reduction of platelets (cosumption)
f. BLEEDING 1.skin rashes
2.adrenal hemorrhage

Adrenal hemorrhage is called Waterhouse-Friderichsen Syndrome.It cause acute adrenal insufficiency and is uaually fatal

Complications and Outcome of Meningitis

- Infection can spreading to
- Dura – pachymeningitis
- Leptomeninges - leptomeningitis
- Brain – encephalitis
- Spinal cord – myelitis
- Ventricles - ventriculitis

Complications and Outcome of Meningitis

Seizures/motor and neurological deficits / Behaviour problems,Learning difficulties

Psychosocial and emotional problems - low self esteem and difficulties coping

Subtle complications - poor concentration, clumsiness and mood swings

SIADH

Subdural effusions & empyema

CNs Palsies (esp deafness)

Cerebral edema

Complications and Outcome of Meningitis

- Serum amylase level may be elevated in cases caused by mumps even in the absence of parotitis.

Septic sinus or cortical vein thrombosis

Arterial ischemia / infarction (inflammatory vasculitis)

Septic shock / multi-organ failure

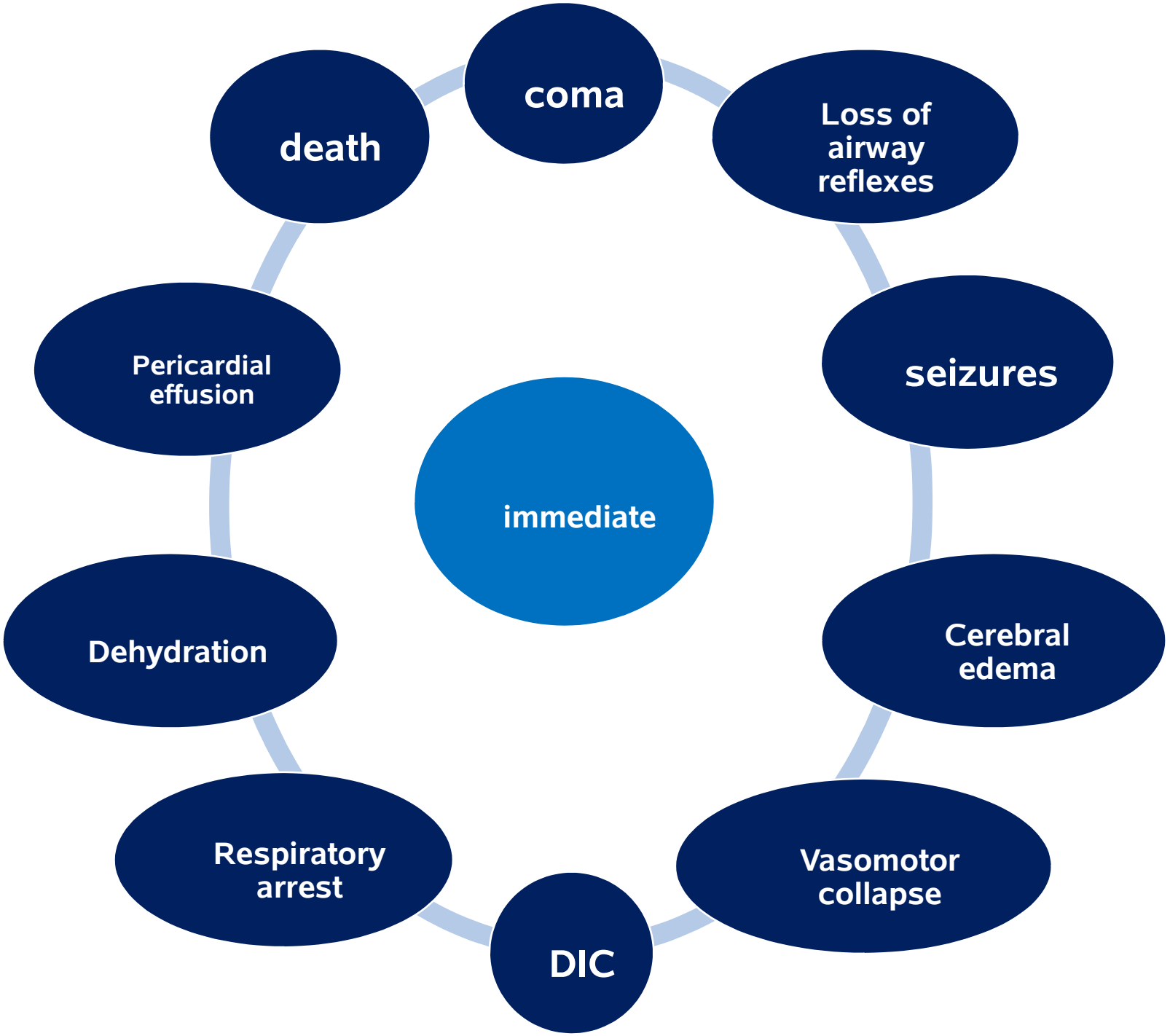
Disseminated intravascular coagulation (DIC)

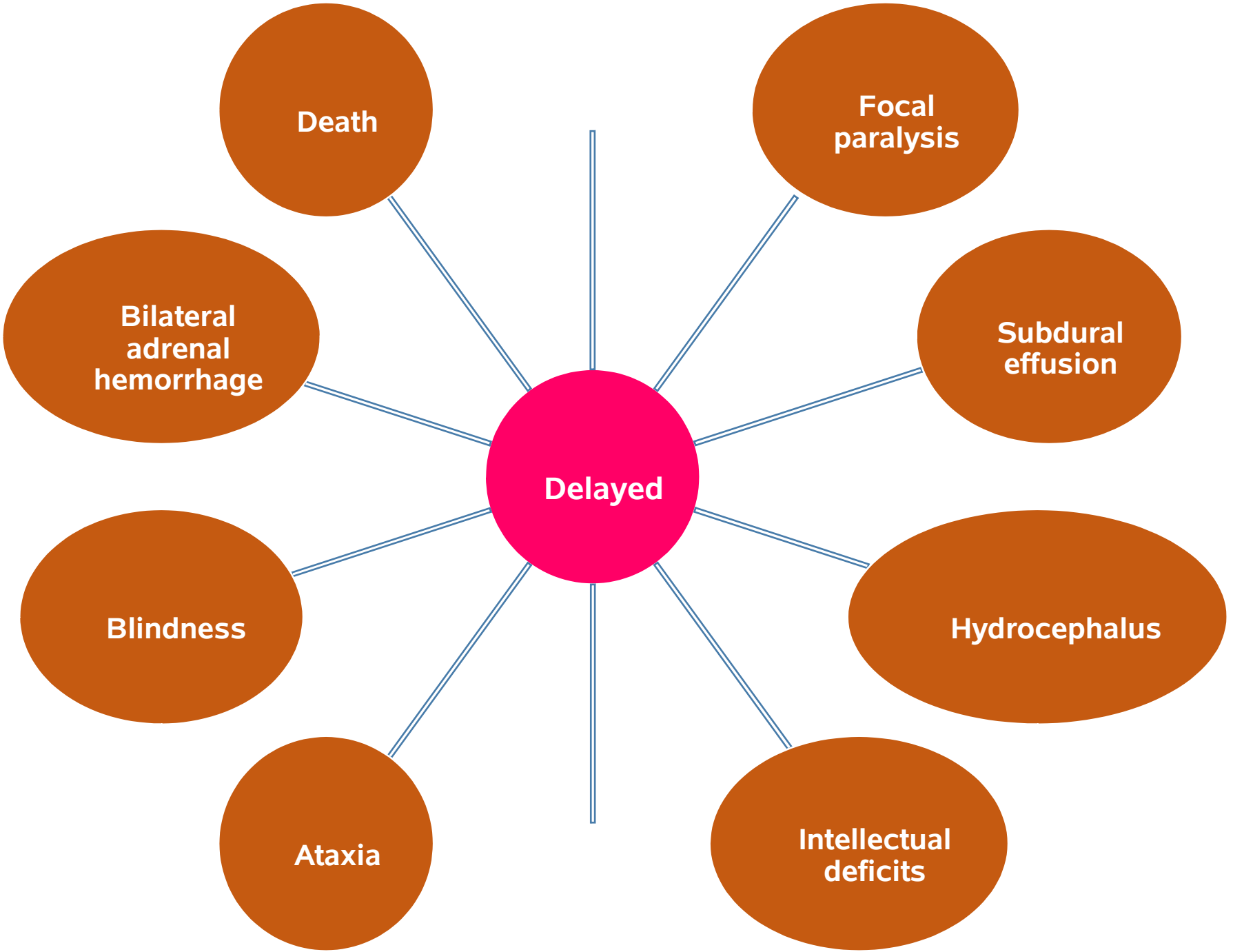
HYDROCEPHALUS

DILATATION OF THE VENTRICULAR SYSTEM

NONCOMMUNICATING: Due to obstruction within the ventricular system, e.g., tumor, aqueductal stenosis

COMMUNICATING: Due to obstruction of CSF flow in the subarachnoid space with decreased reabsorption


coma
Loss of airway reflexes
seizures
Cerebral edema
Vasomotor collapse
DIC
Respiratory arrest
Dehydration
Pericardial effusion
death
immediate


Death
Focal paralysis
Bilateral adrenal hemorrhage
Subdural effusion
Delayed
Blindness
Hydrocephalus
Ataxia
Intellectual deficits

Differential Diagnosis of Meningitis

- Brain abscess

- Encephalitis

Subdural and epidural empyema

- Tumor like astrocytomas,oligodendrogliomas,ependymomas
- Mixed gliomas
- Metastatic tumor

Differential Diagnosis of Meningitis

- Chemical meningitis: Rupture of tumor

- intracranial haemorrage like
- Epidural
- subdural
- Subarachnoid
- intraparenchymal

Differential Diagnosis of Meningitis

- metabolic encephalopathy
- hyperglycaemic coma
- uremia
- hepatic encephalopathy
- vit B deficiencies
- vascular diseases (amyloid angiopathy, vasculitis, berry aneurysms, A-V malformations

CSF & LUMBER PUNCTURE

- Contraindications
- - INR > 1.4 or other coagulopathy
- - platelets < 50
- - infection at desired puncture site
- - obstructive / non-communicating hydrocephalus
- - intracranial mass
- - high intracranial pressure (ICP) / papilloedema

CSF & LUMBER PUNCTURE

- An LP may safely be performed without first doing a CT head in a young previously healthy patient with no history of seizures, a normal level of consciousness and a normal neurological exam.
-

CT scan usually is performed prior to LP to rule out intracranial hematoma, mass effect, or obstructive hydrocephalus.

CSF & LUMBER PUNCTURE

- Cell count and differential
- Chemistry (protein, glucose)

  Culture and Gram stain
-

CSF & LUMBER PUNCTURE

Other tests to consider

India ink and / or Cryptococcal Ag (for Cryptococcus neoformans)

AFB and / or PCR for TB

Viral PCR (includes HSV, CMV, EBV)

arbovirus / WNV, echovirus

CSF & LUMBER PUNCTURE

- VDRL
- fungal culture
- PCR and /or antibody titers for Lyme ds.

Summary of Typical CSF Findings

	Normal	Bacterial	Viral	TB
Cells	0-5	>1000	<1000	<500
Polymorphs	0	Predominate	Early	+/- increased
Lymphocytes	5	Late	Predominate	Increased
Glucose	60-80	Decreased	Normal	Decreased
CSF plasma: Glucose ratio	66%	<40%	Normal	< 30%
Protein	5-40	Increased	+/- Increased	Increased
Culture	Negative	Positive	Negative	+TB

Viral Meningitis-Treatment

- Mostly supportive-
- Rest, hydration, antipyretics, and pain or anti-inflammatory medications. The most important decision is whether to initiate antimicrobial therapy empirically for bacterial meningitis while waiting for the cause to be identified.
- Seizures should be treated immediately with IV anticonvulsants such as lorazepam, phenytoin, midazolam, or a barbiturate.

Patients with signs and symptoms of meningoencephalitis should receive acyclovir early to possibly curtail HSV encephalitis

Enteroviruses and HSV are both capable of causing viral septic shock in newborns and infants. In these young patients, broad-spectrum antibacterial coverage and acyclovir should be instituted as soon as the diagnosis is suspected. .

Assessment and stabilization

- Aggresssive management in septic shock, hypoxemia, seizure, cerebral edema, hypotension from dehydration
- Preexisting condition may complicated the pt. dz. - Recent neurosurgery, trauma, leukopenia, immunocompromised, DM.
- Acute cerebral edema or increase ICP → immediate intubation and adequate ventilation +- osmotic agent (mannitol, diuretic)

TREATMENT of MENINGITIS

- Therapy can be modified as the results of Gram stain, cultures, and PCR testing become available. Patients in unstable condition need critical care unit admission for airway protection, neurologic checks, and prevention of secondary complications.

TREATMENT of MENINGITIS

- since SIADH has been reported. Fluid restriction, diuretics, and rarely hypertonic saline infusion may be used to correct the hyponatremia

TREATMENT of MENINGITIS

- Cerebral edema does occur in cases of severe encephalitis and may require intracranial pressure control by infusion of mannitol (1 g/kg initial dose followed by 0.25-0.5 g/kg 6 hrly), IV dexamethasone, or intubation and mild hyperventilation, with arterial PCO2 around 28-30 mm Hg

TREATMENT of MENINGITIS

- Use of adjunctive corticosteroids:
- - prior to or along with initial antibiotics, administer Dexamethasone 10 mg IV for suspected bacterial meningitis (based on cloudy CSF, CSF WBC counts > 1000 or + Gram stain)
- - continue 10 mg IV 8 hrly x 4 days

TREATMENT of MENINGITIS

- Isolation & Contact Prophylaxis:
- - Generally isolate cases of bacterial meningitis for up to 48 hours of appropriate antibiotics
- - Concern is to reduce transmission of meningococcal infections

TREATMENT of MENINGITIS

- can be taken out of isolation after this time or if alternative pathogen identified
- - department of health should be notified of pathogens in pyogenic meningitis

TREATMENT of MENINGITIS

- Close contacts (family members, partners, co-workers or school children) should receive prophylaxis if meningococcal or haemophilus influenzae type B (if not vaccinated):
- - rifampin 600 mg PO bid x 2d
- - ciprofloxacin 500 mg PO single dose

TREATMENT of MENINGITIS

- dexamethasone 0.6 mg/kg/day IV 6-8 hrly for 4 days
- reduces incidence of neurologic sequelae, i.e., hearing loss

- electrolyte abnormalities - SIADH

Prognostic Factors

1. Age
- 2. Level of Consciousness (50% mortality if unresponsive or minimally responsive on admission)
- 3. Seizures early in course
- 4. Strep. pneumoniae meningitis
- 5. CSF results (lower glucose & WBC counts, higher protein)

THANK YOU