

## **PATHOLOGY OF THE NERVOUS SYSTEM**

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### **REFERENCES**

Veterinary Neuropathology (Summers, Cummings and de Lahunta)  
Veterinary Neuroanatomy and Clinical Neurology (de Lahunta)  
Thomson's Special Veterinary Pathology (Carlton and McGavin)

## NEUROPATHOLOGY

The pathology of the nervous system can be intimidating - the terminology is different from other organ systems; the anatomy is complex; and the nervous tissue itself is prone to artifact. Familiarising yourself with basic neuroanatomy will maximise your understanding of disease processes so please do review your anatomy notes if you feel rusty. *It is primarily your responsibility to ensure you have a grasp of basic neuroanatomy.*



**When you see this symbol, answer the question**

### **I. RESPONSE OF CNS CELLS TO INJURY**

**All but the neurons are  
classed as glial cells, glia or  
“neural glue”**

#### **A. CNS cells types**

There are six major cell types: (a) Neurons

(d) Microglial cells

(b) Astrocytes

(e) Ependymal cells

(c) Oligodendrocytes

(f) Choroid plexus epithelial cells

#### **B. Response of neurons to injury**

The neuron is especially vulnerable to injury since it has:

- A high metabolic rate (i.e. high requirement for O<sub>2</sub> and glucose)
- Little stored energy (i.e. a high requirement for O<sub>2</sub> and glucose !!)
- No regenerative capacity
- A very dependent axon

What is  
this?

The axon has no **Nissl** substance = it cannot make its own protein

The cell body must work for the entire axon - it produces the protein for flow of axoplasm  
along the axon and is responsible for disposing of waste

Death or damage to the neuronal cell body → axonal compromise or degeneration

The main ways neurons **respond to injury** are: Acute necrosis (including laminar cortical necrosis)  
Chromatolysis  
Wallerian degeneration  
Vacuolation

## 1. Acute necrosis

This is a common response of neurons to a variety of injuries:

e.g.	Ischaemia (↓ blood flow)	Nutritional deficiency
	Hypoxia	Trauma
	Hypoglycaemia	Toxins (e.g. heavy metals)

**Laminar cortical necrosis** is a form of acute necrosis that occurs **in a distinct pattern**. The **distinct pattern** is due to **selective destruction of neurons** in the deeper layers of the cerebral cortex (they are most sensitive to hypoxia). This occurs in several instances:

- i) Ischaemia (e.g. seizure-related in dogs)
- ii) Thiamine deficiency in ruminants (polioencephalomalacia or cerebrocortical necrosis [CCN]) for which the exact pathogenesis is still unclear
- iii) Salt poisoning/water deprivation in swine and ruminants
- iv) Lead poisoning in cattle
- v) High sulphur intake

**GROSS:** Most likely see nothing at all but can see oedema → brain swelling; herniation  
Thin, white, glistening line along middle of the cortex; fluoresces under UV light in ruminants; cortex ultimately becomes necrotic and collapses

## 2. Chromatolysis

THIS IS NOT NECROSIS

It is an *adaptive response* to deal with injury but it *can* lead to necrosis

Cell body swells and the Nissl substance *disperses* → allows cell body to produce proteins for re-building

Q ? In which equine disease is chromatolysis a PATHOGNOMONIC LESION?

Answer:.....

## 3. Wallerian degeneration

This is breakdown of an axon and its myelin sheath *distal to the point of injury*

CLASSIC CAUSE = Axonal transection

Other causes include vascular, inflammatory, compression, crushing, stretching and toxic

Fig 1. Wallerian degeneration

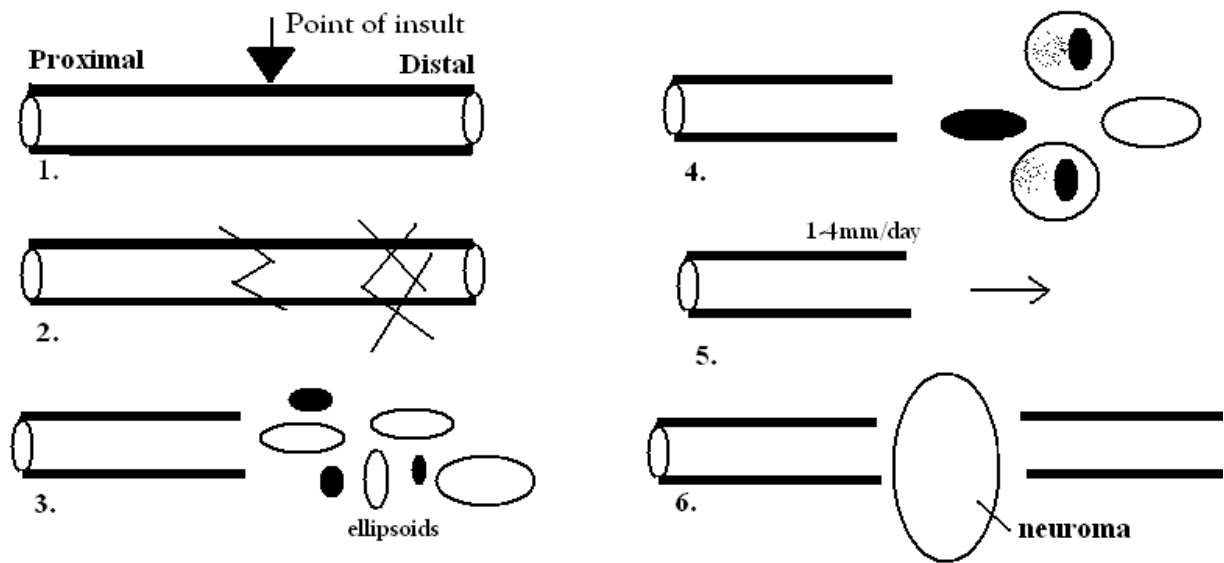


Table 1. Wallerian degeneration occurs in the PNS and the CNS but subsequent regeneration is unlikely in the CNS:

	CNS	PNS
<b>Phagocytosis</b>	Inefficient	Efficient
<b>Remyelination</b>	Oligodendrocytes very lazy	Schwann cells very good at this
<b>Basal lamina to guide new growth</b>	No	Yes
<b>Axonal sprouting</b>	Inhibited by central myelin	Peripheral myelin doesn't inhibit

#### 4. Neuronal vacuolation (vacuolar degeneration)

Hallmark of transmissible spongiform encephalopathies (BSE, Scrapie)

However, it can occur under other circumstances (artifact of fixation, toxicoses, sometimes normal)

#### C. Response of glial cells to injury

CNS cells vary in their susceptibility to injury:

MOST SUSCEPTIBLE: Neurons > oligos > astrocytes > microglia > blood vessels: LEAST SUSCEPTIBLE

Response of oligodendrocytes: Prone to hypoxia → **degeneration**

Death of oligodendrocytes → **demyelination**

<i>Response of astrocytes:</i>	Hyperplasia = astrocytosis Hypertrophy = astrogliosis (usually in preparation for formation of glial fibres, a form of scar tissue in the CNS)
<i>Response of microglia:</i>	Functions include immunosurveillance and repair (phagocytic) Probably of monocyte origin Hypertrophy, hyperplasia, phagocytosis, act as antigen-presenting cells and produce nitric oxide, cytokines and chemokines

## **II. OTHER GENERAL RESPONSES TO INJURY**

1. Oedema
2. Myelination disorders
3. Vascular disturbances

### **1. Oedema**

There are three main types of cerebral oedema (Table 2).

*Table 2. Types of cerebral oedema*

<b><i>Type of oedema</i></b>	<b><i>Cause</i></b>	<b><i>Outcome</i></b>
<b><i>Vasogenic</i></b>	Vascular injury & breakdown of BBB This is most common form in animals	Extracellular accumulation of fluid (white matter typically affected)
<b><i>Cytotoxic</i></b>	Altered cellular metabolism (ischaemia leading to energy deficit so can't pump out Na <sup>+</sup> and water)	Intracellular accumulation of fluid
<b><i>Hydrostatic (interstitial)</i></b>	↑ ventricular hydrostatic pressure (i.e. hydrocephalus)	Extracellular accumulation of fluid (periventricular)

NB: BBB = blood brain barrier

One of the most important consequences of oedema is that ***the brain increases in size*** & attempts to escape the confines of the skull → ***herniation***

If herniation occurs at the foramen magnum → compression of the medulla → DEATH

## 2. Demyelination

This is loss of myelin that has already formed and is **initially normal** - may be 1° or 2°

1° demyelination: The myelin sheath is selectively affected; the axon remains intact

RARE but a feature of canine distemper, visna and CAEV

2° demyelination: Loss of myelin following damage to the axon, i.e. in Wallerian degeneration

Q ? How do you think **demyelination** differs from **dysmyelination**?

Answer.....

## 3. Vascular diseases

Can lead to complete or partial blockage of blood flow → ischaemia

Consequences of ischaemia depend on

- i) Duration and degree
- ii) Size and type of vessel involved
- iii) Susceptibility of the tissue to hypoxia

These consequences include:

1. Acute neuronal necrosis (see above)
2. Vasogenic oedema: The ischaemia is not a direct cause of the oedema but the two could occur together if the ischaemia was the result of vascular damage (see above)
3. Infarct
  - a. Necrosis of tissue following obstruction of its blood supply. Causes include
    - i. Thrombosis: Uncommon in the CNS of animals (common elsewhere) but can see with DIC or sepsis
    - ii. Embolism: Bone marrow emboli following trauma or fractures in dogs
    - iii. FCEM = Fibrocartilaginous embolic myelopathy
    - iv. Vasculitis: e.g. hog cholera (pestivirus), malignant catarrhal fever (herpesvirus) and oedema disease (vasculitis caused by *E. coli* toxin)
4. Malacia
  - a. Definition: Grossly appreciable softening of brain/spinal cord, usually resulting from necrosis
  - b. It follows that malacia occurs in infarcted tissue

- c. Malacia is **not specific to infarcts**, but can occur in other disease processes e.g. hypoxia, toxicosis, nutritional, infectious or metabolic disease
- d. The **pattern** and **location** of malacia are often more diagnostically helpful than the lesion itself

Q ? Fill in the gaps below by ticking the appropriate column for each cause

	Symmetrical malacia	Asymmetrical malacia
Infectious		
Vascular (e.g. infarcts)		
Nutritional		
Trauma		
Toxic		
Metabolic		
Genetic		

### III. INFLAMMATION OF THE NERVOUS SYSTEM

Inflammation is usually the result of infection: Bacteria, fungi, protozoa and viruses can invade the CNS and induce an inflammatory response. They enter by one of four main routes:

- Routes of entry:**
1. Haematogenous (most common route)
  2. Direct implantation (trauma; iatrogenic)
  3. Peripheral nerves (within the axoplasm of axons e.g. rabies, *Listeria*)
  4. Local extension (from nasal cavity, middle ear or paranasal sinuses)

#### **Localisation of infectious organisms:**

After entry, organisms tend to establish in one (or more) of four main areas (Fig 2). The inflammation can take the form of localised abscesses or it may be more diffuse or generalised. The form it takes is generally dictated by the route of infection.

Fig 2. Asterisks mark where infection can become established

	<b>Skull bone</b>
*	<b>Epidural space</b>
	<b>Dura</b>
*	<b>Subdural space</b>
*	<b>Leptomeninges</b>
*	<b>Brain</b>

1. **Epidural or subdural inflammation** tends to result in abscesses, which are uncommon
2. **Leptomeningitis** (inflammation of the leptomeninges; tends to be diffuse)
3. **Encephalitis** (inflammation of the cerebral parenchyma)

Leptomeningitis and encephalitis can be classified based on cause but are usually classified based on the nature of the exudate. Different types of exudate are usually associated with particular groups of pathogen. The main exudates are outlined in Table 3 below.

Table 3. Inflammatory exudates used to classify leptomeningitis and encephalitis

<b>Type of exudate</b>	<b>Type of infection</b>
Fibrinous	Bacteria (including Mycoplasma)
Suppurative	Bacteria (including Mycoplasma) and fungi
Lymphoplasmacytic (non-suppurative)	Viruses
Granulomatous	Bacteria or fungi
Haemorrhagic (rare)	Usually associated with septicemia or infarcts

#### **A few examples:**

##### *i) Suppurative leptomeningitis*

Most common form of leptomeningitis and typically caused by bacteria (*E. coli*, *Streptococcus*, *Haemophilus*, *T. pyogenes*) that spread to the meninges in the bloodstream. The brain swells and the meninges are opaque but MAY BE NO GROSS LESIONS. Microscopically, neutrophils predominate initially; usually fatal.



ii) *Suppurative encephalitis*:

Suppurative inflammation in the brain is usually the result of bacterial infection and manifests as abscesses, some of which may be very small (microabscesses). When they arise in the brain, the abscesses may be single or multiple depending on the route by which they gain entry. They vary in size with a central, liquefied cavity (necrosis). The bacteria are often the same species as those that cause leptomeningitis and both the brain and meninges may be inflamed concurrently (meningoencephalitis). Bacterial infection with *Listeria monocytogenes* is different and outlined below in (iv).

iii) *Lymphocytic / lymphoplasmacytic meningoencephalitis*:

Usually due to viral infection but also some protozoal infections. Often, the meninges and brain are affected concurrently, hence meningoencephalitis. In the brain, the inflammation is perivascular. Main routes of viral invasion of CNS are neural and haematogenous. There are usually no gross lesions but common **hallmark lesions of CNS viral infections** are neuronal necrosis, gliosis and perivascular lymphoplasmacytic cuffing. A few important viral examples affecting the CNS are outlined in Table 4.

Table 4. Types of viruses affecting the nervous system

Neurotropic	Endotheliotropic	Pantropic
Rabies (rhabdovirus)	Infectious canine hepatitis (canine adenovirus)	Canine distemper (morbillivirus)
Aujeszky's disease (herpesvirus)	Classical swine fever (pestivirus)	Infectious bovine rhinotracheitis (bovine herpesvirus type 1)
Visna (ovine lentivirus)	Equine herpesvirus type 1 (herpes)	

iv) *Listeriosis*:

Listeriosis is caused by *Listeria monocytogenes*, an important bacterial pathogen causing suppurative encephalitis, notably in sheep, but its pathogenesis is different to most other bacterial infections in the CNS.

Bacteria gains entry via oral mucosa → trigeminal nerve → trigeminal ganglion in brain

CNS form mainly occurs in ruminants eating silage, especially where pH is too high

Usually NO GROSS LESIONS

Microscopic lesions: Microabscesses **AND** lymphoplasmacytic cuffing of vessels

Clinically: Circling, unilateral facial nerve paralysis, drooling, recumbency, paddling

**DEATH**

iv) *Granulomatous*: Fungal diseases and mycobacteriosis; idiopathic forms recognised (mainly dogs).

iv) *Eosinophilic meningoencephalitis*:

**NOT USUALLY DUE TO INFECTION.** This is eosinophilic inflammation in the leptomeninges *AND* brain. In the meninges, it's rather like suppurative leptomeningitis except eosinophils predominate instead of neutrophils. In the brain parenchyma the inflammation is perivascular. Most likely causes are (i) salt poisoning and/or (ii) water deprivation, mainly occurring in pigs.

vi) *Prion diseases (transmissible spongiform encephalopathies)*

- A group of fatal neurodegenerative diseases which occur in a number of species, including man
- BSE (cattle); scrapie (sheep and goats); chronic wasting disease (elk)

AETIOLOGY: Still highly controversial

Prions are protein based infectious agents which lack nucleic acid and are composed of PrP<sup>Sc</sup>; the host-encoded prion protein (PrP) changes its structure into an abnormal isoform in the brains of affected animals and humans

Long incubation period; difficult to diagnose; agent is highly resistant

#### **IV. TRAUMA**

CONCUSSION: Temporary loss of consciousness following head trauma; full recovery is usual but if repeated or severe → neuronal loss and haemorrhage, even death

CONTUSION: "Contusion" = focal haemorrhage

LACERATION: Tearing of CNS by bone within the skull (e.g. fractured skull) or by penetrating objects (i.e. bullets). This is the most severe and most serious form of traumatic injury as it carries additional risk of contamination/infection

HAEMORRHAGE: As well as following contusion injury, haemorrhage may also result from endothelial damage. Locations of haemorrhage may be epidural, subdural, leptomeningeal or cerebral.

## **V. RESPONSE OF THE SPINAL CORD TO INJURY**

Spinal cord, like brain, can be affected by infectious, inflammatory, toxic, nutritional and neoplastic disease and similar basic lesions can occur in its parenchyma as occur in the brain parenchyma. Traumatic injuries to the spinal cord include concussion, contusion, haemorrhage and compression. The first three are similar to those described above in the brain. Compression requires more attention.

### **COMPRESSION**

May arise from within or outside the spinal cord

Causes:

- *Abscess* (Extradural, vertebral, intervertebral)
- *Fracture* (Traumatic or pathological fracture of vertebral bodies e.g. due to abscess, metabolic disease, neoplasia)
- *Neoplasia*
- *Intervertebral disk disease* Prolapsed disk can cause acute or chronic compression
- *Malformations (esp. vertebral)*
  - Wobbler horses (stenotic myelopathy): Vertebral canal narrows due to malformation and malarticulation of the cervical vertebrae (usually C3-C4)
  - Cervical vertebral malformation-malarticulation in dogs: Similar pathogenesis to wobblers
  - Atlantoaxial subluxation of toy dogs - hypoplastic dens leads to subluxation

**Lesions associated with focal compressive spinal cord injury are similar regardless of cause**

Gross: Spinal cord may be indented or flattened

Microscopic: Essentially Wallerian degeneration but may progress to neuronal loss and malacia (probably due to associated vascular damage)

## **VI. CONGENITAL MALFORMATIONS**

Congenital malformations may involve the spinal cord, brain, meninges, calvaria or vertebral column and typically result from defects in neural tube development. They are present at birth (or before) and manifest as either (a) a morphological or (b) a functional problem. This section largely deals with

morphological problems, since the functional malformations tend to arise as biochemical abnormalities, such as the lysosomal diseases or leukodystrophies. A selection is outlined here.

## CAUSES

1. Environmental (e.g. toxic, infectious, nutritional, radiation): By far the most common cause
2. Inherited

## TYPES OF CRANIAL/BRAIN MALFORMATION

- A. Hydrocephalus
- B. Cerebellar defects
- C. Weird and wonderful

### A. Hydrocephalus

This is an increased accumulation of fluid in the cranial cavity (see Table 5)

Table 5. Types of hydrocephalus

Type of hydrocephalus	Location of fluid
Internal	Within ventricles
External	Within arachnoid space
Communicating	Within ventricles <i>and</i> arachnoid space
Hydrocephalus <i>ex vacuo</i>	Dilation of ventricles 2° to loss of cerebral tissue; also known as <i>compensatory hydrocephalus</i>

Internal hydrocephalus is most common type

- a) Acquired: Supposedly the result of obstruction, usually due to inflammation or compression  
Space-occupying lesions include neoplasms, abscesses and cholesteatomas  
Inflammation of meninges and/or ependymal cells can lead to hydrocephalus (e.g. feline infectious peritonitis)
- b) Congenital: An obstructive lesion is often not found  
Cranium may be abnormal (e.g. doming)  
Common in brachycephalic dogs and in small breeds; sporadic in cattle  
Malformed mesencephalic aqueduct may be involved

## B. Cerebellar defects

Most important defects are: *Cerebellar hypoplasia* and *cerebellar abiotrophy*

### a) Cerebellar hypoplasia:

Occurs in all domestic species; one of most common congenital CNS defects

Aetiology: Inherited: Suspected in Arab foals, Jersey cattle, Chows, Corriedale sheep

Environmental: Teratogens selectively attack mitotic germinal cells of the cerebellum that are the source of neurons which migrate late in gestation, leaving this stage vulnerable to teratogens

Teratogens: **\*bovine virus diarrhea (BVD)**, **\*feline parvovirus**, classical swine fever (USA: hog cholera), canine herpesvirus

### b) Cerebellar abiotrophy

Premature or accelerated degeneration of nervous tissue elements **after they have formed (i.e. really a form of atrophy)**; probably inherited in most cases

## VII. NEOPLASIA

Less important and less frequent than in man

PRIMARY: Meningioma, glial tumours and primitive neuroectodermal tumours (PNETs; very rare)

**Meningioma** Most frequent in cats and dogs

Originates in the meninges, as the name suggests

Acts as a compressive, space-occupying lesion which **seldom invades**

### Glial tumours

*Astrocytoma*: The most common of the glial tumours

Predilection for brachycephalic breeds (boxer, bulldog)

Solid, firm, grey-white; sometimes mottled red with areas of necrosis/haemorrhage

*Oligodendroglioma*: Most common in dogs; predilection for brachycephalic breeds

Soft, grey to pink/red and often gelatinous

*Ependymoma*: Occur mainly in ventricles (primarily lateral); may spread in the ventricular system via CSF; expansile but can be invasive and destructive

*Choroid plexus tumours:* Rare; mainly dogs and largely in the fourth ventricle  
Metastasis can occur via the CSF and ventricular system

## SECONDARY (METASTATIC)

A number of malignancies can metastasise to the brain, e.g. *haemangiosarcoma, lymphoma, mammary or pulmonary carcinomas*. Some tumours invade from adjacent tissue (e.g. *pituitary or nasal carcinomas*)

## VIII. IDIOPATHIC DISEASES

### Idiopathic epilepsy

Definition of seizure: A brain disorder manifested as a paroxysmal cerebral dysrhythmia

Sudden onset, ceases spontaneously but tends to recur

Pathogenesis: Small group of neurons periodically and spontaneously depolarise. This can occur due to structural, biochemical or unknown causes.

Structural: Neoplasms, inflammation or trauma

Biochemical: Hypocalcaemia, hypoglycaemia, hepatic encephalopathy

Idiopathic: No cause found (i.e. epilepsy); individuals believed to have a low seizure threshold which predisposes their neurons to depolarise of their own volition

**\*\*\* Be aware that there are idiopathic diseases which are often breed-related (e.g. pug dog encephalitis); granulomatous meningoencephalitis (adults of all breeds of dog) \*\*\***

## IX. PATHOLOGY OF THE PERIPHERAL NERVOUS SYSTEM

The peripheral nervous system (PNS) consists of the dorsal and ventral roots, the spinal ganglia, the spinal and peripheral nerves, cranial nerves and peripheral components of the autonomic nervous system.

### A. Trauma

Some nerves are predisposed to injury because of

- (a) their superficial location
- (b) their proximity to bone
- (c) dystocia (Calving paralysis – obturator nerve damage leading to downer cow)
- (d) proximity to injection sites

## B. Infectious

Some infectious organisms can involve peripheral nerves

Examples:

- a) *Neospora caninum* has a predilection for dorsal roots in the dog
- b) Equine guttural pouch mycosis → inflammation in the recurrent laryngeal nerve → vocal cord hemiplegia
- c) Macaw wasting disease (aka proventricular dilatation syndrome).

Non suppurative inflammation of the central, peripheral and autonomic nervous systems

Leads to gastrointestinal dysfunction (wasting, anorexia, depression) and neurological signs (ataxia and seizures). This was considered idiopathic but now *believed* to be caused by borna disease virus; most common in macaws and parrots

## C. Idiopathic/immune-mediated

e.g. Coonhound paralysis (does not occur in the UK). This is a polyradiculoneuritis linked in some cases to raccoon bites and leading to quadriplegia. Cause not known. Lesions are in ventral roots of spinal nerves and in peripheral nerves.

## D. Neuropathies

Several neuropathies and polyneuropathies are often breed-related (suggesting an inherited pathogenesis)<sup>7</sup>

## E. Degenerative

e.g. Equine laryngeal hemiplegia (“roarers”)

This disease is **COMMON**

Pathogenesis: Idiopathic degeneration of left recurrent laryngeal nerve → atrophy of the left dorsal cricoarytenoid muscle → inability to abduct arytenoid cartilage and vocal fold → airways partially obstructed on inspiration

## F. Toxic

Uncommon

Heavy metals: Lead, thallium, mercury

e.g. lead damages Schwann cells → peripheral demyelination

## G. Neoplastic Uncommon; occasionally see in the dog

## H. Nutritional/metabolic

- a) Vitamin A, Vit B, pantothenic acid and copper deficiency can lead to neuropathy
- b) Copper deficiency: Swayback in newborn lambs and enzootic ataxia in older lambs; pathogenesis unclear
- c) Some metabolic states are associated with neuropathies, such as diabetes mellitus and hypothyroidism

## X. LYSOSOMAL STORAGE DISEASES:

*Mostly* inherited group of diseases which cause lesions in the CNS and PNS

Lysosomes are responsible for “waste disposal” in the cell

Substrate → enzyme destruction → product → disposal from cell
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Basic problem is lack of enzyme, leading to build of substrate in cell

Krabbe's (globoid cell leukodystrophy) is most relevant to the CNS because it is essentially a failure to break down myelin during normal turnover of this substance.



### Some general rules of CNS injury

1. Neurons **do not** regenerate.  
have small energy stores and a high metabolic rate. They depend on an intact blood supply for O<sub>2</sub> and glucose.
2. Following transection of axons **in the CNS**, there is little or no regeneration.
3. Axons in the Peripheral Nervous System (PNS) **can** regenerate, however.
4. CNS lesions heal by proliferation of astrocytes. Capsules are not as strong as those produced by fibroblasts elsewhere.
5. Blood brain barrier stops drugs, infectious agents and antibodies entering brain.
6. Once infected, the CNS is the least resistant tissue.
7. Consequences of focal or even small lesions often more severe than in other organs. In the skull, there is little room for maneuver and no regenerative capacity (compare to other organs, such as the liver or kidney).
8. In clinical terms, the LOCATION of the lesion is the critical factor and its nature of secondary importance.

## **Neuropathology Glossary**

Encephalo	Brain
Meningo	Meninges
Myelo	Spinal cord
Leuko	of the white matter
Polio	of the gray matter
Encephalitis	Inflammation of the brain
Meningitis	Inflammation of the meninges
Meningoencephalitis	Inflammation of the brain and meninges
Myelitis	Inflammation of the spinal cord
Malacia	Necrosis
Astrocytosis	Increased numbers of astrocytes (hyperplasia)
Astrogliosis (or gliosis)	Increased numbers of glial fibers
Gemistocytes	Highly reactive astrocytes with abundant pink cytoplasm and distinct cell borders
Gemistocytic astrocytosis	Astrocytic hyperplasia in which gemistocytic forms predominate
Leptomeninges	Pia and arachnoid mater together
Pachymeninges	Dura mater
Leukoencephalomalacia	Necrosis of the white matter in the brain
Leukomyelomalacia	Necrosis of the white matter in the spinal cord
Polioencephalomalacia	Necrosis of the gray matter in the brain
Poliomyelomalacia	Necrosis of the gray matter in the spinal cord
Ganglioradiculitis	Inflammation of cranial and spinal ganglia and roots
Polyradiculoneuritis	Inflammation of multiple spinal or cranial nerve roots

Telencephalon = cerebral hemispheres

Diencephalon = thalamus and hypothalamus

Mesencephalon = midbrain

Metencephalon = cerebellum and pons

Myelencephalon = medulla