TRAUMATIC INJURIES

- **Concussion (Commotio cerebri)**
- **Contusion (Contusio cerebri)**
- Coup
- Contre coup
- **Laceration**
- **Skull Fracture**
- **Spinal Cord Injuries**

TRAUMATIC INJURIES

Concussion

Concussion is a transient loss of consciousness and reflex activity following a sudden injury to the head.

Full recovery is expected, and it is assumed that in mild cases.
There is no morphologic injury.

The mildest degrees of concussion produce **no visible microscopical structural damage**, but there is some experimental evidence that plasma substances, including proteins, may be transported in vesicles through the endothelial cells, and the brain and cord may lose their capacity for autoregulation of blood flow in local areas of static injury.

Contusion

Contusion means **bruising;** the architecture of the **nervous tissue is retained**, but there is **hemorrhage into the meninges and about the blood vessels in the parenchyma.**

Contusions may be diffuse or focal injuries, although often those coexist.

Same pathogenetic factors as those causing concussion, but of greater magnitude.

- A **coup** contusion is located at the impact site, and a **contrecoup** contusion at a location on the opposite side of the brain.

- The development of contre-coup depends on the sudden movement of the brain to the point of impact (coup) if the head was immobilized and the brain does not move, contre-coup injury won't happen.

- **Laceration**

Laceration is a traumatic injury in which there is disruption of the morphology of the tissue.

The mechanics of lacerations are, in general, the same as those of contusion.

- **Penetrating injuries** are analogous to, but more severe than, those that result in focal contusions.
- Lacerations may also occur with **blunt injuries** of the type that cause displacement of the brain.

Lacerations caused by penetrating injuries are always liable to secondary infections.

- **Fracture of the skull**

Fractures of the skull can provide **a pathway of infection to the sinuses, meninges, and brain.**

Fractures of the base of the skull **may involve the middle ear and allow the escape of cerebrospinal fluid (CSF) and the entrance of infection**.

Injuries to the spinal cord

It is possible for direct injuries to the spinal cord to occur without obvious injury to the vertebrae.

Much more common are indirect injuries to the cord acquired in the course of vertebral luxations or fractures with dislocation.

The most common of indirect injuries to the cord are produced by extruded nucleus pulposus in dogs, and **by compression of the cervical cord** in the syndrome known as **"wobbler"**

DEGENERATION IN THE NERVOUS SYSTEM

- Meninges
  - Collagenous and osseous metaplasia
  - Spherical mineralized nodules (psammoma bodies)
  - Hyalinization of dural collagen
  - Ossifying pachymeningitis (osseous metaplasia of the dura observed frequently in large breeds of dogs)

DEGENERATION IN THE NERVOUS SYSTEM

- Choroid plexuses
  - Hyaline degeneration
  - Cholesteatosis, cholesteatomas (*cholesterol Granulomas)*

    15-20% of old horses

    Lateral ventricules cause hydrocephalus by obstructing the interventricular foramen

    More frequently in the plexuses of the fourth ventricle than in the lateral ventricles

- Atrophy in the brain and spinal cord

  Senility,

  Congenital lipofuscinosis

  Progressive internal hydrocephalus

- **Anoxia and anoxic poisons**

The pathologic effects of anoxia on nervous tissue are of 2 types:

- **Ischemic neuronal necrosis** followed by glial repair
- Greater degrees of anoxia, sufficient to necrose astroglia as well as neurons, result in **softening**.

Histotoxic: respiratory enzyme blockage
Hypoxic: lack of oxygen in inhaled air
Anoxic: hemoglobin is not free
Ischemic: decreased blood volume (common)

Anoxia and anoxic poisons

- ✓Cyanide poisoning
- ✓Nitrate/nitrite poisoning
- ✓Fluoroacetate poisoning
- ✓Carbon monoxide poisoning
- ✓Hypoglycemia

MALACIA AND MALACIC DISEASES

Malacia means grossly observable **softening**, and is used to signify necrosis of tissue in the Central nervous system.

Encephalomalacia: necrosis in the brain
Myelomalacia: necrosis in the cord
Encephalomyelomalacia: cerebral and spinal necrosis

Poliomalacia: softening of gray matter
Leukomalacia: softening of white matter

MALACIA AND MALACIC DISEASES

- Focal symmetrical poliomyelomalacia syndromes
- **Polioencephalomalacia of ruminants**
- **Thiamine deficiency**
- Nigropallidal encephalomalacia of horses
- Salt (NaCl) poisoning
- Mycotoxic leukoencephalomalacia of horses
- **Lead poisoning**

▪ Polioencephalomalacia of ruminants (PEM)

- "**Cerebrocortical necrosis**" or "**laminar cortical necrosis**
- Cattle, sheep, goats and pigs
- Salt poisoning in swine, occasionally in lead poisoning of cattle, and described among the residual neurologic lesions of cyanide poisoning.
- Deficiency of thiamine or a disturbance in its metabolism is implicated in PEM.
- Course in sheep and goats is shorter and sheep in the age group from weaning to **18 months** are the most affected.
- Disease of young cattle.

▪ Polioencephalomalacia of ruminants (PEM)

- Blindness and dullness, a tendency to head press against obstacles, and cessation of feeding.
- Remain on their feet and show, in addition to the noted signs, muscular tremors, opisthotonos.
- The animals are recumbent. There is twitching of the face, ears, and eyelids, intermittent grinding of teeth, drooling, and bulbar paralysis in some.

Thiamine (Vitamine B1) deficiency in ruminants

- Thiamine deficiency in cattle and sheep has been termed **polioencephalomalacia.**

- Rumen microbes are able to synthesize thiamine.

- Only very young ruminants, whose rumens have not yet been populated by thiamine-producing microbes, are susceptible to thiamine deficiency.

- The disease is seen most commonly in cattle 6 to 18 months of age fed concentrated rations. In sheep, most cases occur in younger age groups (2 to 7 months).

- Clinical signs in cattle and small ruminants may include depression, stupor, ataxia, head pressing, apparent cortical blindness, opisthotonos, convulsions, and recumbency with paddling and then death.

- **Gross lesions**, if present, are limited primarily to the cerebral cortex.

- Initially, 2 days after onset : cerebral edema

- In rare cases with more severe brain swelling: brain displacement with herniation of the parahippocampal gyri beneath the tentorium cerebelli and the vermis of the cerebellum into the foramen magnum can occur.

Microscopically;

- Neuronal necrosis and edema
- Macrophages and gitter cells to phagocytose necrotic debris
- Laminar separation (at the gray matter–white matter interface) in which there are prominent accumulations of macrophages

LEAD POISONING

The most consistently important poison in **farm animals**.

- ✓Common and fatal in **cattle**;
- ✓Less common but fatal in sheep;
- ✓Occasionally observed in horses, dogs, and cats;
- ✓Rare in swine.

The disease in cattle is probably always **acute**,
in horses is virtually always **chronic**.

Usual sources of lead for cattle: ***paint* and *metallic lead in storage batteries.***

Lead is usually obtained by **ingestion**, but only a small proportion **(1-2%)**

Absorbed lead is slowly excreted in bile, milk, and urine;

and is deposited in tissues, → in liver and kidneys in acute poisoning

↘ in bones in chronic poisoning.

Clinical syndromes are chiefly neurologic :

Acute poisoning in cattle usually leads to death in 12-24 hours

Staggering, muscle tremors, recumbency, convulsions, opisthotonos, champing of the jaws, hyperesthesia

The diagnosis of lead poisoning is necessarily **chemical** because lesions are either absent or nonspecific;

Possible **lesions**;

- ✓ The lower gut may contain a small volume of dark fetid feces, attributed to lead sulfide.
- ✓ moderate brain swelling
- ✓ The capillaries and venules are congested
- ✓ Endothelial swelling and proliferation
- ✓ Laminar cortical necrosis
- ✓ Irregular, acid-fast, intranuclear inclusion bodies can be found in the renal tubules

NEURODEGENERATIVE DISEASES

- CENTRAL NEURONOPATHIES AND AXONOPATHIES

Compressive optic neuropathy

Organomercurial poisoning (Minamata disease)

- CENTRAL AND PERIPHERAL NEURONOPATHIES AND AXONOPA

Organophosphate poisoning

Arsenic poisoning

Neonatal copper deficiency (swayback, enzootic ataxia)

- PERIPHERAL AXONOPATHIES

Mononeuropathy: lesion involving single peripheral nerve

Mononeuropathy multiplex: several nerves are randomly involved

Polyneuropathy: bilaterally symmetrical involvement of several nerves. It carries the implication of a systemic disturbance.

Equine laryngeal hemiplegia

Neonatal copper deficiency (swayback, enzootic ataxia)

- ✓ Characteristic neurologic disease of lambs, goat kids, and piglets.
- ✓ Caused by **maternal/fetal copper deficiency**.
- ✓ Copper **is required for the catalytic activity of enzymes that are essential for neural function.**
- ✓ Copper deficiency can be primarily form the result of copper-deficient soils and inadequate intake from diet or secondary from defective absorption because of interactions between copper, molybdenum, zinc, cadmium, or inorganic sulfates.

- ✓ The provision of adequate copper supplementation of pregnant animals at risk can effectively eliminate the disease in their offspring, and treatment of affected animals with copper may produce some remission of signs.

- ✓ Other manifestations of copper deficiency, such as **"steely wool," osteoporosis, and hypopigmentation of black wool**, could be expected in affected sheep flocks

Neonatal copper deficiency (swayback, enzootic ataxia)

- ✓ Clinical swayback in lambs occurs in a congenital form and a delayed form, also called "**enzootic ataxia**" in which, after being normal at birth, lambs suddenly develop signs at any time between 1 week and several months of age.
- ✓ Lesions occur in the **cerebrum, brainstem, and spinal cord in the congenital form**, but only in the brainstem and spinal cord in cases with a postnatal onset.
- ✓ Affected lambs can be born dead, weak, or unable to stand. If mobile, they are ataxic. Enzootic ataxia is characterized by ataxia.

Congenital swayback

- ✓ Bilateral and symmetrical gelatinous **softening** or **cavitation** in the cerebral white matter (may be restricted to the occipital pole or may involve the entire corpus medullare).
- ✓ Histologically, **edema** with mild fibrillary **astrogliosis** and a loss of myelin can be expected.
- ✓ Some myelin degradation products are usually present, but never in great quantity, and gitter cells are sparse.

Delayed swayback

- ✓ Do not have lytic lesions in the cerebral white matter, they consistently have changes in both gray and white matter in other parts of the neuraxis.
- ✓ Extensive Wallerian degeneration is concentrated in dorsolateral and ventromedial tracts throughout the spinal cord.
- ✓ The pattern of tract degeneration is suggestive of a distal axonopathy.
- ✓ Many neurons undergo central chromatolysis, and some have nuclear rhexis and lysis. Few may be undergoing neuronophagia.

Equine laryngeal hemiplegia

- ✓ Denervation atrophy of the intrinsic muscles of the left side of the larynx.
- ✓ The resultant inability to adduct the arytenoid cartilage and the vocal fold leads to partial obstruction of the airway on inspiration, and inspiratory stridor on exertion, referred to as roaring.
- ✓ Idiopathic degeneration of the left recurrent laryngeal nerve.
- ✓ Decrease in myelinated fibrils and an increase in connective tissue.
- ✓ Demyelination and Waller degeneration.

MYELINOPATHIES

- **Hypomyelination/dysmyelination**

Ovine and caprine hypomyelinogenesis

- **Leukodystrophic and myelinolytic diseases**

Globoid cell leukodystrophy (Krabbe disease)

- **Spongy myelinopathies**

Idiopathic spongiform myelinopathies

Toxic/metabolic spongiform myelinopathies

- **Nonmyelinic Spongy Encephalomyelopathies**

Idiopathic - Toxic/metabolic spongiform myelinopathies

- Branched-chain α-ketoacid decarboxylase deficiency

(Maple syrup urine disease)

- Hepatic and renal encephalopathy
- Hexachlorophene toxicosis
- Halogenated salicylanilide toxicosis
- *Stypandra* toxicosis
- *Helichrysum* toxicosis

Nonmyelinic Spongiform Encephalomyelopathies

- The term "status spongiosus" applies to a variety of lesions in which the microscopic appearance of the neural tissue is transformed by numerous vacuoles or microcavities.
- The changes results from either the swelling of astrocytes or the vacuolation of neurons and neurites.
- Usually considered in the context of the diagnosis of scrapie and related conditions.
- It is distinctly different from the foamy vacuolation seen in the lysosomal storage diseases

Nonmyelinic Spongiform Encephalomyelopathies

- Citrullinemia
- Neuronal vacuolar degeneration of Angora goats,
- Multifocal spongy encephalomyelopathy in dogs
- **Scrapie of sheep and goats,**
- **Bovine spongiform encephalopathy (BSE),**
- Chronic wasting disease (CWD) of deer and elk,
- Transmissible mink encephalopathy (TME),
- Feline spongiform encephalopathy (FSE),
- Exotic ungulate encephalopathy of captive wild ruminants,

- Human prion diseases include

✓ *Kuru,*
✓ Creutzfeldt-Jakob disease CJD,
✓ *Gerstmann-Straussler-Scheinker disease* (GSS),
✓ *Fatal familial insomnia* (FFI).

SCRAPIE

- The first **prion disease** to be recognized and described.
- Clinically affected animals are usually in the 2- to 5-year age group

- **Pathogenesis**;

➢Infection occurs probably via ingestion

➢The agent proliferates in the lymphoid tissues and lower intestine

➢May take up to 2 years to reach the nervous system.

Clinical signs;

✓ Affected sheep are initially alert but excitable,

tremble when excited, and may have seizures.

✓ Agitated rubbing against posts and trees,

behavior that gave rise to the colloquial name “scrapie.”

✓ Self-trauma can cause extensive loss of wool and

abrasions of the skin.

✓ Progressive dysmetria and emaciation,

and finally paralysis and death.

- No significant **gross lesions**, and no inflammatory changes.

The most characteristic finding is the presence of **large intraneuronal vacuoles** in the medullary reticular, medial vestibular, lateral cuneate, and papilliform nuclei.

Spongy vacuolation of the neuropil in gray matter is the result of vacuolation of neuronal processes

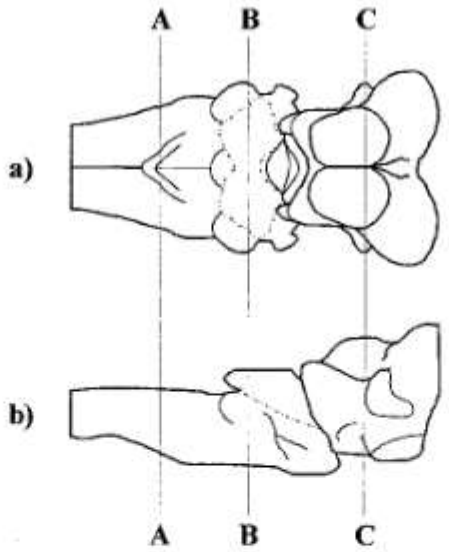
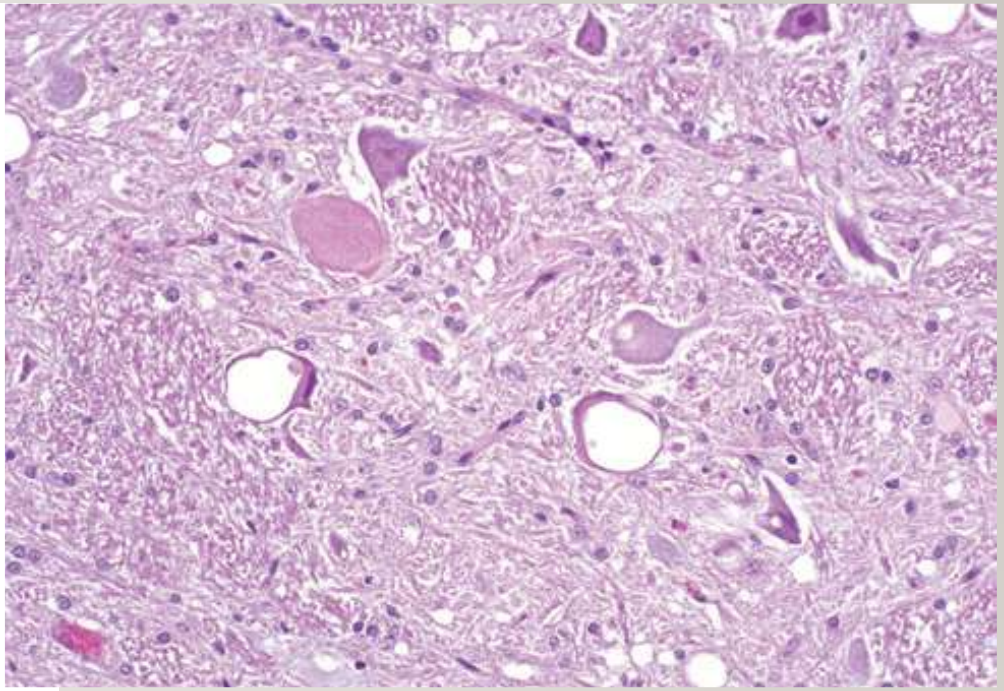


Fig 1. Brainstem after the removal of the cerebellum, from a) dorsal, and b) lateral aspects. Recommended levels at which sections should be taken: A–A = medulla, at the obex; B–B = medulla through caudal cerebellar peduncles; C–C = midbrain through rostral colliculi.

https://science.vla.gov.uk/tseglobalnet/confirmatory-diagnosis.html



Spongiform Encephalopathy (Scrapie), Brain, Motor Neurons, Sheep,

Neuronal cell bodies contain one or more discrete and/or coalescing clear vacuoles *(V)*. There are no inflammatory cells in this disease.

BOVINE SPONGIFORM ENCEPHALOPATHY (BSE)

- Ingestion of feed contaminated with **infectious prion** is the most likely origin for BSE.

- Cattle 3-6 years of age become **apprehensive, hyperesthetic, and dysmetric**.

Display **fear** and **aggressive behavior**, with progressive gait disturbances leading to frequent falling.

- The pathologic features of BSE resemble those of scrapie

- Vacuolation of neuronal cell bodies and processes is prominent in the dorsal vagal, medullary reticular, vestibular, solitary, spinal trigeminal, and red nuclei;
- accompanied by moderate degrees of spongiform change in neuropil.