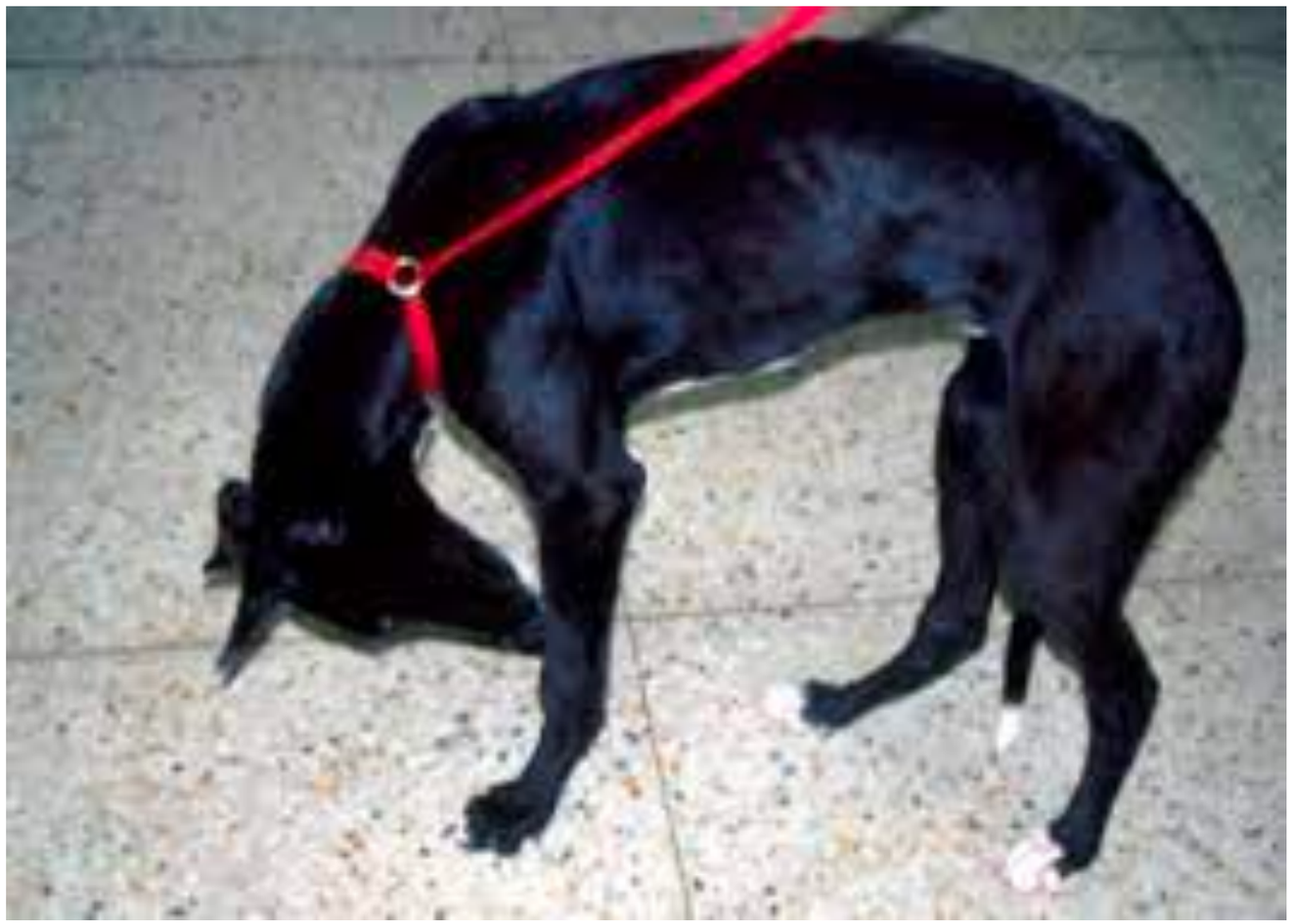
Disease of Nervous system
Part 2



Dr. Karima Al Salihi

Technical terms:

- Encephalitis: inflammation of the brain.
- Myelitis: inflammation o f the spinal cord.
- Neuritis: inflammation of the nerve tissue.
- Meningitis: inflammation of the brain covering
- Pachymeningitis: inflammation of the dura matter.
- Leptomeningitis: inflammation of the pia matter.
- Neuralgia: pain along the coures of a nerve.
- Ataxia: Failure of muscular coordination or irregularity between several members of a group of muscles with out paresis, paralysis or involuntary movement.
- Tremors (trembling): faint, rapid, intermittent contraction relatively of short periods.
- Spasms: Abnormal muscular contractions.

- **Somnolence (Sleepiness):** The patient stands with the head drooping and the eyes closed.
- **Stuper:** Disturbance o f equilibrium when standing.
- **Lassitude:** Tiredness- weakness.
- **Syncope (Fainting):** Loss of consciousness caused by
- inadequate glucose or oxygen to the brain causing neurogenic o r cardiovascular disturbances.
- **Coma:** Loss o f consciousness with absence o f reflexes and loss of muscular tone, but the cardiac and respiratory functions are still maintained.
- **Nystagmus:** Rapid, rhythmic side to side movement o f eye

Technique adopted to investigate neurological disorders:

(1) Clinical neurological examination:

Previous clinical approach is highly effective fordiagnosis; prognosis and treatment.

(2) Radiological examination:

It helps to identify traumatic and congenital lesions of the vertebral column and skull.

(3) Radio isotope scanning:

It is used to screen lesions like tumor, infective changes in the brain. Gamma emitters are injected IV and their sites o f localization are scanned through a recorded.

(4) Electro encephalography:

(EEG) To record the electrical activity of cerebral cortex and interpret function state o f cortex.

(5) Neuro- opthalmology:

The optic nerve is a part of brain and lesions of brain and brain stem are reflected in the form of opthalmological symptoms; e.g. Brain edema is characterized by papilloedema and protrusion of optic disc.

1) Nystagmus (involuntary oscillation of eye ball):

Is concerned with the lesions of cerebellovestibular apparatus.

2) Non-functional droopy eyelids:

Are associated with seventh cranial nerve paralysis.

(6) Nerve conduction velocity:

Here the rate of passage of nerve impulse following direct stimulus is recorded. This is applied to diagnose paresis and •paralysis.

(7) CSF pressure:

1) It increases in cases of:
1- Avitaminosis A
2- Increased intracranial contents (tumor, abscess, cyst).
3- Hydrocephalus.
4- Inflammation (meningitis, encephalitis).

2) It decreases in cases of:
1- Shock
2- hypotension
3- Long standing degenerative disease.

Diseases of the brain

•These diseases include:

- encephalitis
- encephalomalacia,
- hemorrhage of the brain
- hydrocephalus
- cerebral anemia
- sunor heat stroke
- chorea
- neurosis
- coenurosis
- epilepsy,
- toxoplasmosis
- leptospirosis
- tick paralysis
- listeriosis and
- cerebrospinal nematodosis in equines.

Encephalitis

It is the inflammation of the encephalon (brain), characterized by initial hyperexcitability followed by paralysis and unconsciousness. It may be primary or secondary or consequence to other diseases.

Etiology:

(1) Viral agents:

Cattle: Sporadic bovine encephalomyelitis; Bovine malignant catarrh; Rabies; Infectious bovine rhinotracheitis•**Horse:** Infectious encephalomyelitis.

Sheep and Goats: Scrapie; Louping ill.

Dog: Canine distemper.

(2) Bacterial agents
Listeria, Necrobacillus, Enterotoxaemia, Salmonella, Erysipelus.

(3) Parasitic agents:
Migrating larvae, Multiceps multiceps, Toxoplasmosis, Nervous coccidiosis.

(4) Toxic agents:
Lead, Arsenic, Salt poisoning, Ipomia plant.

(5) Fungal agents:
Cryptococcosis.

Pathogenesis:

1. Infecting agents cause irritation and degenerative changes in the brain tissues.
2. formation of multiple necrotic foci of micro- abscess.
3. The lesions depend on the sites and nature o f causative agents.
4. There may be acute edematous swelling.This may obstruct blood flow and interfere with cerebral function.
5. There is gradual increase in intracranial pressure.
6. Irreversible brain injury is associated with cervical rigidity, ataxia, tremors, convulsions followed by coma and paralysis.

Clinical findings:

(1) High rise of temperature.
(2) Mania, aggressiveness, depression and stupor.
(3) Circling movement, head pressing.

(4) Clonic convulsion, muscular tremor and pawing on the ground.
(5) Frothy salivation from commissar o f mouth.
(6) Champing of jaws and hyperaesthesia.
(7) Depression o f consciousness.
(8) Spastic type o f paralysis may be in either sides.
(9) Ataxia or incoordination of gait.
(10) Nystagmus o f eye ball.
(11) Unilateral facial paralysis due to listeriosis.
(12) Blindness of eye reflex.
(13) Prostration, unable to stand, head is drawn under the body.

Clinical pathology:

Examination of CSF for biochemical, cellular and microbiological aspects.

Diagnosis & differential diagnosis:

Differentiated from:
(1) Acute cerebral edema: Less excitement; No fever; History of salt poisoning.

(2) Poisoning: Salivation; History of poisoning; No fever; Blindness; Acute onset.

(3) Avitaminosis-A: Occur in young animal (calf); No fever; Ataxia, Hyperaesthesia; Respond to Vitamin-A therapy.

(4) Encephalomalacia: History of grain engorgement; Thiamine Deficiency, which respond to vitamin B 1 therapy.

(5) Meningitis: Temperature reaction; Hyperaesthesia; Rigidity of muscles; Examination of C.S.F. will assist in diagnosis.

Treatment:

(1) Complete rest, try to remove & treat the real causes.
(2) Use o f sedative and tranquilizers during the excitement stage.
(3) Nervous stimulants during the period o f depression.
(4) High dose o f broad spectrum antibiotics for 5-7 days.
(5) Use o f corticosteroid.
(6) Specific antidote against poisoning.
(7) Specific treatment for specific agent.
(8) Use of parenteral fluids for nourishment and dehydration.
(9) Use of mannitol to reduce intracranial pressure.

•Encephalomalacia

It is the degenerative changes of brain.

Causes:

(1) Nutritional deficiency: Copper, vitamin E, thiamine.
(2) Infectious: Clostridium perffingens type D, pulpy Kidney.
(3) Ingestion of toxic chemical: Lead, mercury, arsenic. Salt causes edema of brain.
(4) Hepatic encephalopathy: Damage of liver lead to indigestion of protein & ammonia intoxication causing damage of CNS.

Pathogenesis:

•It may occur as a result of endothelial injury.

•Clinical symptoms:

It may be acute or subacute.

(1) Acute form:

- Muscle tremor (more pronounced in head)
- Frothy
- Salivation & Champing of jaws
- clonic convulsions
- Opisthotonos condition
- Nystagmus o f eye ball
- Death within 24 hours in young age group (6-9 months).

(2) Subacute form:

- Anorexia & depression
- Ataxia
- Circling movement
- Head pressing
- Atony of rumen
- Bradycardia
- Severe weakness.

Diagnosis:

The disease occurs in young age & differentiated from:

(1) Lead poisoning: seen in all ages, history of poisoning, salivation, blindness, mortality rate is very high.
(2) Avitaminosis A: occurs in very young animal as two forms Cerebral form (tremors, inco-ordination & convulsion) & Ocular form (blindness & keratitis). It responds to vitamin A therapy.

Treatment:

The disease is irreversible but apply supportive treatment including B- complex, fresh rumen juice, corticosteroid to reduce intracranial pressure.

Cerebral apoplexy(Hemorrhage in the brain)

This means rupture o f a blood vessel in the brain. This condition may occur in cattle, horses and dogs.

Etiology:

(1) High increase in blood pressure during violent exertion or parturition.
(2) Traumatic injury o f the skull.

Symptoms:

(1) Nervous shock:, Animal falls unconscious with convulsions.
(2) The intracranial pressure leads to lethargy, coma and death.
(3) The formed haernatoma will compress on part o f the brain causing loss of functions controlled by centers located in this part; and consequently hemiplegia, paraplegia or monoplegia.
(4) Breathing is slow and sonorous.

Treatment:

(1) Keep the animal quiet in a calm place.
(2) Apply cold compresses to the head.
(3) Elevate the head above the body level.
(4) Give laxatives and diuretics.
(5) Stimulants are contraindicated.
(6) Do not attempt to push the animal to stand.
(7) Slowly IV injections of 50% sucrose (one ml/lb BW).

Hydrocephalus

It is an accumulation of cerebrospinal fluid in the cerebral ventricles due to defect in the normal drainage of the cerebrospinal fluid.

Etiology:

(1) Congenital hydrocephalus: It is due to embryological defect in the change canals and foramina between the individual ventricles or between the ventricles and subarachnoid space. The cerebral hemisphere becomes distended with fluid and the growing skull becomes greatly enlarged.

(2) Acquired hydrocephalus: It is caused by obstruction of drainage by some local space occupying lesion or inflammation. It may be acute or transient as in cholesterol granulomas, compression of the brain occurs in hypovitaminosis A in calves due to failure of growth of the cranial bone to accommodate the growing brain.

Symptoms:

(1) Gradual onset of general paralysis.
(2) Depression, disinclination to move.
(3) Chewing in slow intermittent and incomplete.
(4) Reaction to cutanous stimulation is reduced.
(5) Frequent stumbling in coordination and abnormal posture.
(6) Brady cardia and cardiac arrhythmia.

Diagnosis:

The diseases must be differentiated from encephalitis.

Treatment:

No treatment.

Cerebral anaemia anoxia (Cerebral ischaemia)

Cerebral anoxia occurs when the supply of oxygen to the brain is reduced. This may be acute or chronic depending on the severity of the deprivation.

Etiology:

(1) Sudden and severe loss o f blood on general anaemia due to chronic loss o f blood.
(2) Acute hydrocyanic acid and nitrite poisoning.
(3) Acute cardiac failure due to severe copper deficiency in cattle.
(4) Terminal stages o f pneumonia and congestive heart failure.
(5) Sudden rush o f blood from the brain to internal organs as in too rapid removal of transudate or exudate from a body cavity.
(6) Sudden evacuation of gas from tympanic stomach.
(7) Allergic shocks.
(8) Increased intracranial pressure with compression of the cerebral vessels.

Symptoms:

Acute cerebral anoxia is manifested by:

(1) Loss o f consciousness.

(2) Muscular tremor, beginning about the head and spreading to the trunks and limbs followed by recumbency.

(3) Chronic convulsions and the animal fall to the ground.

(4) Vomiting may occur.

(5) The mucous membrane is pale and the pupils dilated.

Chronic cerebral anoxia are manifested by:

(1) Pale mucous membrane.

(2) Lethargy, dullness, atexia.

(3) Muscle tremor or convulsions in some cases.

Differential diagnosis:

(1) Hypoglycemia in which similar signs occur.
(2) Lead and arsenic poisoning and encephalitis.

Prognosis:

(1) Favorable as long as the pupil can react to light.
(2) If there is no reaction especially when there is convulsions, the prognosis is bad.

Treatment:

(1) Remove the real cause.
(2) Respiratory stimulant such as inhalation o f ammonia.
(3) Artificial respiration may keep the animal alive for few minutes.
(4) Injection o f stimulants as campher.
(5) Put the head in a lower position than the body level to supply the brain with blood.

Sun stroke (Heat stroke)

It is affection not necessarily due to exposure to sun rays, but also to action of great heat combined with increased humidity. This frequently result from direct exposure of the sun rays during the hot season, also inability to prespire readily is a predisposing factor.

Etiology:

(1) Direct exposure o f the animal to sun rays during a hot and humid weather.

(Heavy hair coat or fatty animals in crowds in a hot humid ill-ventilated place.
(3) Damage to the hypothalamus due to spontaneous hemorrhage.
(4) Inadequate water intake and insufficiency o f the tissue fluids to permit heat loss by evaporation.

Pathogenesis:

(1) Heat stroke will cause vasodilatation of the cranial vessels, the result is drop in blood pressure.
(2) Respiration increases in rate and depth, the temperature is elevated, **the heart** rate becomes fast and irregular, the urine secretions is reduced.

Symptoms:

(1) Onset is sudden, the animal stops work and refuse to continue.
(2) Staggering gait and the animal fall to the ground unconsciousness
(3) Mucous membranes are congested.
(4) Pulse is fast and irregular.
(5) The temperature is elevated up to 42°C, shivering.
(6) Convulsions are evident and the animal dies in a state of coma within two hours.

Diagnosis:

The condition should be excluded from acute infective diseases.

Treatment:

(1) Put the animal in a well ventilated shaded place (cool place).
(2) Spray the patient with cold water and apply ice bag to the head.

(3) Circulatory stimulants and vasoconstrictors are indicated, camphor in oil 20-30 gm for large animals + 1.5-0.5 gm for small animals (hypodermically).
(4) IV o f 5% dextrose saline solution slowely.

NB: When the body temperature begins to fall, regulate or stop cold water because rapid drop o f temperature below the normal is dangerous.

Neuroses of pregnancy, parturition and lactation

It occurs in cases of:

(1) Milk fever.

(2) Grass tetany.

(3) Acetonaemia.

(4) Neurosis and or neuritis.

Coenurosis (sturdy)

The disease is caused by the presence o f multiceps in the brain, which inhabits in the small intestine o f the dog and fox.

It occurs most commonly in sheep, much more rarely in otherherbivores in the following order o f frequency (cattle, goats, horses, camels and rabbits).

Causes:

•The tape worm Tania multiceps inhabit in the small intestine o f the dog. Infestation occurs as the result of ingestion o f proglottids o f tape worm from grass or water contaminated with the feces o f infested dog.

•Symptoms:

In sheep:

The first stage **of acute meningoencephalitis begin 10-14 days after invasion:**
(1) The animal lags behind the rest o f the flock. Often interrupts its grazing to stand with lowered head.
(2) In severe case there is dullness, scalp hot and painful to touch, lateral bending o f the head, salivation girding of the teeth, sometimes the animal rushes along a straight line or in a circle, stumbles then fall, finally developing convulsions.

The second stage, **after 3-6 months:**
(1) Disturbance of consciousness from increased intracranial pressure. The animal lifts the feet too high in walking, head is lowered or raised in the air.
(2) Epileptic attacks.
(3) Circular convulsive movement. In cattle, the most common type of movement is circular.

Treatment:

(1) Cold compression on the head.
(2) Trephinning and removal of the cyst

Toxoplasmosis

(1) The clinical signs in calves are dyspnea, cough, fever tremors and shaking of the head, grinding of the teeth, depression, recumbency with bicycling motions of the legs, weakness and prostration.

(2) Death after a period of 2-6 days.

(3) In adults there is hyper excitability more than depression in the early stages.

(4) Diagnosis depends on demonstrations of the organisms by animal inoculation or histologically there is no knowledge yet of the life cycle nor is there a treatment. Autotoxin has been demonstrated in the serum of adult dogs.

(5) Post mortem, mild hyperemia or no lesions in the CNS the toxin acts on the control and not on the peripheral nervous system.

Tick paralysis

(1) A Rapidly progressive ascending flaccid paralysis. It first affects the hind legs and eventually may reach the medulla oblongata and death results from respiratory failure.

Calves, sheep, goats, pigs, dogs and cats are affected as well as man.

(2) Recovery without treatment may occur in sheep in 48 hours. Dipping or spraying will stop the diseases quickly in a flock.

(3) The cause is a toxin injected into the host by rapidly engorging female ticks (toxin in the tick's body).

(4) Young animals are more susceptible than adults. The toxin causes paralysis. Adults can with stand heavy infestations because of previous exposure.

Listeriosis

(1) A highly fetal infectious disease in ruminants caused by ***listeria monocytogenes*** affecting cattle, sheep and goats most commonly.
(2) The symptoms are those o f encephalitis and brain lesions. Listeria isolated from abscesses o f liver and from lymph glands, spleen, heart and spinal cord.
(3) Cattle o f all ages are affected. The affected cow shows dullness and isolates herself from the herd and wanders around from place to place may stand with the head pushing against a wall or byre.
(4) The cow may circle at pasture, the circling becoming evident when she is confirmed to a yard. The animal particularly young cows, circles using the hind legs as a pivot. Circling may be to the right or left but is usually in the same direction. The head may be carried low on one side, salivation conjunctivitis and nasal discharge may be seen. Paralysis o f the pharynx, paresis and coma precede death. The disease is usually sporadic. It has a tendency to reappear periodically on the same farm. (5) Enzootics may appear in beef cattle. Morbidity is low and mortality is high and most affected cattle die.
(6) Diagnosis by symptoms serologic tests are not successful. It should be differentiated from rabies and lead poisoning.
(7) Treatment by penicillin and IV sulphamezathin

•Diseases of the meninges

Meningitis

It is the inflammation of the meninges; it affects brain and spinal cord.

Etiology:

(1) Bacterial: due to streptococci, corynebacteria, haemophilus, pasteurella, listeria & leptospira, and tuberculosis.
(2) Viral: Malignant head catarrh and bovine encephalomyelitis.
(3) Extension of inflammation in cases of encephalitis.

Mode o f infection:
(1) In bacterial infection: infection is usually haematogenous.
(2) Sporadic cases occurs as a result of penetrating wounds of the skull, otitis media & after dehorning.
(3) As a result o f umbilical infections in newborn animals.

Symptoms:

(1) The onset o f acute cases is sudden.
(2) Fever and toxemia & cutaneous hyperaesthesia.
(3) Tonic spasms o f neck muscles causing retraction of head & muscle tremors.
(4) Respiration is usually slow and deep.
(5) Excitement, mania followed by convulsions & finally death.

Diagnosis:

(1) Clinical signs
(2) CSF examination, which shows, high protein, turbidity, high cell count and bacteria.

Treatment:

(1) Antibacterial drugs over a long period (7-10 days).
(2) Analgesics in severe pain.
(3) Oral salicylates in chronic cases.
(4) Intrathecal administration of drugs is advised in severe cases.

Diseases of the spinal cord

- They include traumatic injury and myelitis.
- Traumatic Injury

This is a sudden severe trauma of the spinal cord causes complete flaccid paralysis caudal to the injury.

Etiology :

(1) Dislocation or fracture o f the vertebrae.
(2) Concussion or contusion without structural damage to the bones o f the vertebral column.
(3) Migration o f parasitic larvae as in cerebrospinal nematodiases.

Pathogenesis

The lesions cause compression on the nervous tissue due to the displaced bone or haematoma. The initial response is that of spinal shock which is manifested by complete flaccid paralysis. The lesion must effect at least the ventral third o f the cord before spinal shock occurs.

Symptoms:

(1) Immediate spinal shock manifested by flaccid paralysis.
(2) Fall in blood pressure due to vasodilation.
(3) Local sweating.
1) The extremities are affected and the animal is unable to rise.
2) Anaethesia occurs at and caudal to the lesion and hyperaesthesia may be observed at the anterior edge of the lesion due to irritation by local inflammation and edema.
3) There is no systemic disturbance but pain may causeanorexia and an increase in heart rate.
4) Recoveiy may occur in 1-3 weeks if nervous tissue is not destroyed; when there is extensive damage to a large section o f the cord, there is no recovery and disposal is advisable.

Diagnosis:

(1) Depends upon the history and symptoms.

(2) X-ray examination may reveal the site and extent of injury.

Treatment:

(1) Careful nursing on deep bedding with turning.

(2) Massage of bony prominence and periodic plunging.

Myelitis

It is the inflammation of the spinal cord which is usually associated with viral encephalitis.

The initial signs of initiation are followed by signs of loss of function. This is common in rabies, may be ended by paralysis.

Viral diseases of CNS in animals

Disease	Cause	Incidence	Clinical Signs and pathology	Course and prognosis	Diagnostic tests	Treatm
Encephalo myelitis	Togavirus	Variable; horses, dogs	Depress, fever, anorexia, circling, primarily cerebral signs	Acute, prognosis; recovery or brain damage	History, CSF, serology, virus isolation	Support
Scrapie	"Slow" virus	Sporadic; sheep more than 2 year old	Pruritus, cerebellar ataxia, death; neuronal and spongi- form degeneration of brain	Chronic, pro-gressive; always fatal	History and signs, histopathology	Non
Malignant catarrhal fever	Herpes virus	Sporadic; adult cattle	Depression, blindness, seizures, death; nasal and ocular discharge	Acute, progres-sive; usually fatal	History and signs, histop-athology	Non
Bovine spongi-from encephalopathy	Possibly scrapie virus	Cattle	Abnormal behavior, gait and posture, aggressive, hyperreactive to stimuli.	Chronic, progressive; usually fatal	Signs, histopatholog, mouse inoculation	Non
Equine infection anemia	Retrovirus	Rare CNS; horses	Behavioral changes, blindness, ataxia, weakness	Chronic, progressive	Serology	Support
Caprine artheritis- ence-phalomyelitis	Retrovirus	Sporadic; young goats	Artheritis, ataxia, paresis, affect pelvic limb then limbs; signs of cerebellum or cranium.	Acute to chronic progressive or fatal	History, signs, CSF, serology	Support
Louping ill	Flavivirus	Sheep	Ataxia of head and trunk leaping gait	Acute, Progressive; about50% fatal	Presence of ticks, serology, virus isolation	Support

Bacterial (1-5), mycotic (6-8), protozoal (9-12) and parasitic diseases (13,14) of

Disease	Cause	Incidence	Clinical signs and Pathology	Course and Prognosis	Diagnostic Tests
1.Meningitis	Staphylococcus, Pasteurella, others	Variable, but generally uncommon	Generalized or localized (especially cervical) hyperaesthesia; degree of illness variable	Acute or chronic Prognosis good with early treatment	CSF (protein >20 mg/dl, neutropjils Culture and sensitiv test
2.Meningo-encephalomyelitis	As meningitis	Uncommon	As meningitis, blindness, seizures, ataxia, cranial nerve deficits	Usually acute: Prognosis good with early treatment, but neurologic deficits are common	Same as meningiti EEG may indicat encephalitis
3.Abscess	As in meningitis	Rare	Focal signs and may be signs of meningitis or meningoencephalitis	May be chronic: Progression may be rapid once signs are obvious	As meningo-encephalitis
4.Tetanus	Clostridium tetani	Rare except in horses	Extensor rigidity of all limbs, often opisthosonos; erected ear, contraction of facial muscles, prolapsed nictitating membrane; infected wound	Acute onset. Often lasts 1-2 wk, animals may die; Prognosis fair if treated	Signs, history, isolation of organis from wound
5.Listeriosis	Listeria monocytogenes	Sporadic in ruminants	Depression, asymmetric ataxia and paresis, cranial nerve signs, central vestibular signs	Acute progressive in sheep and goats, more chronic in cattle; poor prognosis if CNS signs are present	History, sign, CSF protein, mononucle cells, Histopathology, fluorescent antibod isolation of Listeri

6.Nocardiosis	Nocardia species	Low	Respiratory or cutaneous forms; CNS abscesses and osteomyelitis	Chronic; poor prognosis	Smears, cultures, (protein, neutroph
7.Actinomycosis	Actinomyces species	Low	Similar to nocardiosis	Chronic; poor prognosis	As Nocardiosi
8.Aspergillosis	Aspergillus sp.	Primarily in large animals	Encephalitis, guttural pouch infection	Chronic; poor prognosis	Culture, CSF
9.Toxoplasmosis	Toxoplasma gondii	Common infection but infrequent clinical problem	Immunosuppression; CNS, eyes, lungs, gastrointestinal tract and skeletal muscle often affected	Chronic; fair to poor prognosis	Serum titer, oocys stool, biopsy, C (Protein, mononu & neutrophile
10.Babesiosis	Babesia sp.	Rare	Parasite of RBC ; rarely CNS, But infarction and hemorrhage more common	Acute to chronic; Poor prognosis	Peripheral bloo smears
11.Trypanosmiasis	Trypanosoma cruzi	Rare	Parasite of RBC; rarely causes CNS disease	Chronic, fair prognosis	Peripheral bloo smears
12.Coccidiosis	Several species	Common enteric, rare CNS,	Enteric may cause CNS signs Sarcocystis spp. may cause myopathy	Variable	Fecal exam. Orga in muscle biop
13.Larva migrans	Toxocara canis and other species	Rare	Granulomas in brain or spinal cord from migrating larvae	Acute or chronic; Prognosis depends on severity of signs	None, necrops
14.Coenursis	Coenurus sp.	Rare; in sheep	CNS signs depend on location of lesion	Acute to chronic; Prognosis poor	Palpated sheep sk radiographs

Sciatic paralysis

Etiology:
(1) In prolonged unilateral recumbency.
(2) Secondary to femoral neck or pelvic fracture (pubis, ischium).
(3) Septic infection arising from intramuscular injections.

Clinical sings:

(1) The limb is entirely non- weight bearing.
(2) The limb hangs loosely and there is forward jerk while the animal attempts to walk.
(3) Sensation is lost from the limb distal to the stifle except, the medial aspect of the mid-metatarsal region.

Prognosis:

•Is hopeless so that early slaughter is advisable.
•Tibial nerve paralysis
•It is rare in animal. It is caused by paralysis of the extensor of the hock and flexor of the digits resulted in slightly flexed fetlock while the sole is in apposition with the ground.

Other paralysis

(1) Spastic paralysis denotes paralysis in contractile stage.

(2) Flaccid paralysis indicates paralysis in relaxation stage.

Lesions of Nerves of the Thoracic Limb and Neurologic Examination Fina

Roots	Nerve	Muscle Atrophy	Gait and Posture Deficit	Spinal Reflex Alterations	
C6-C7	Suprascapular	1.Supra. 2. Infra;spinatus	1.None	1.None	
C7-C8	Axillary	1.Deltoid	1.None	1.None	1.
C6-C8	Musculocutaneous	1. Biceps brachii	1.No flexion of elbow	1.Decreased or absent flexion of elbow during flexor reflex	1
C7- T2	Radial	1.Triceps brachii 2.Extensor carpiradialis 3.Ulnaris lateralis 4.Common and lateral digital extensors	1.No extension of elbow, carpus and digits 2.Unable to support weight on limb	1.Decreased or absent of triceps tendon reflex & extensor carpi radialis muscle response	
C8- T1	Median and Ulnar	1.Flexor 2.Superficial & deep capri radialis digital flexor 3.Flexor carpi ulnaris 4.Deep digital flexor	1.No flexion of carpus. 2.No flexion of digits.	1.Decreased or absent flextion of carpus and digits during flexor reflex	
L5-L6	Obturator	1.Pectineus 2. Gracilus	1.Slight abduction of hip	1.None	
L4-L5	Femoral	1.Quadriceps femoris	1.Unable to extend stifle 2.Limb collapses with weight	1.Decreased or absent patellar tondon reflex	

L6-S1	Sciatic	1. Semimembranosus 2. Semitendinosus 3.All muscles of peroneal and tibial nervea	1.Unable to actively flex stifle 2.Hock flexes and extends passively 3. Hock dropped 4.Knuckled onto digits 5.Caudal gluteal muscle involvement will produce adduction of hip	1.Decreased or absent flexor reflex 2.Decreased or absent cranial tibial muscle response 3.Decreased or absent gastrocnemius tendon reflex	
L6-S1	Tibial	1.Gastrocnemius 2.Superficial and deep digital flexors	1.Dropped hock and tarsus 2.Over flexion of hock and over extension of digits	1.Decreased or absent gastrocnemius tendon reflex	
L6-S1	**Peroneal (fibular)**	1. Peroneus longus 2. Cranial tibial 3.Lateral and long digital extensor	1.Stand knuckled onto digits 2.Overextension of hock and overflexion of digits	1.Decreased or absent cranial tibial muscle response	

Flaccid paralysis of hind quarter in dog.

Spastic paralysis of hind quarter in dog.

Plate 6 (c) Diseases of nervous system

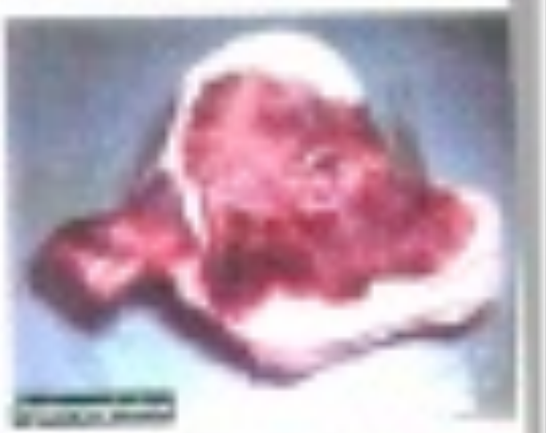


Plate 6 (c) Diseases of nervous system