Digestive system
Diseases of the alimentary tract

Introduction:

The primary functions of the alimentary tract are:

Prehension, Digestion and Absorption of food and water. By the modification of the absorbed material the internal environment is maintained. The above functions fall into 4 major modes which are Motility, Secretion, Digestion, and Absorption.

Principals of alimentary tract dysfunction

Abnormality in any or all of the four major modes of function therefore gives diagnosis of digestive tract disease. This should be directed to find which mode or modes of function are disturbed before proceeding to the determination of the site & nature of the lesion and the specific disease. The principals of alimentary tract dysfunction are:

I- Motor function:

A) Hyper and Hypo-motility:

The most important types of the alimentary tract motility are:
a. Peristaltic movements, which move the ingesta and feces through the alimentary tract and this, depend on.
1- Segmentation movements. 2- Tone of the sphincter.
b. Prehension - mastication - swallowing are other types of alimentary tract motility. There may be increased or decreased motility, the first cause diarrhea, and the second cause constipation.

B) Distension:

Rapid accumulation of gases, engorgement of the lumen of the stomach or gut by solids or liquids, in pyloric or ilecoaecal valve, causing obstruction with consequent distension of the tract. When the gases and fluids fail to pass through, this causes pain reflexly to spasm and motility of the adjacent gut segments. Further secretion of fluid occurs which
aggravates the distension. Finally pain disappears and a stage of paralytic ileus develops in which all muscular tone is lost.

C) Abdominal pain:
Alimentary tract disease is the major cause of abdominal pain.
Causes of abdominal pain:
1- Stretching of the wall of the organs stimulates the free pain endings of the autonomic nerves in the wall. Contraction itself does not cause pain but the direct reflex; distension of neighboring segment together with the action of peristaltic wave is the causes.
2- Odema, failure of local blood supply to alimentary tract (embolism) causes pain.
3- Stretching and inflammation of serous membranes (peritoneum).

D) Dehydration & shock:
1- The effect of distension will result in stimulation of further secretion of fluids and electrolytes in diseased segments. This will result in loss of fluids & electrolytes, thus causing dehydration, which may cause fatal ending by shock before death. Dehydration is accompanied by acidosis or alkalosis. The production of shock when there is a distension of segments is mainly due to a marked reflex depression of vasomotor, cardio-vascular and respiratory functions.
N.B.: In addition to the above disorders there may be vomiting and diarrhea as a result of motor function disorders.

II - Secretory functions:
Abnormalities of the secretions, either gastric or intestinal or pancreatic are rare in animals. Such examinations depend on laboratory work and is essential in other individuals

III- Digestive functions:
They depend on the motor and secretory functions. In herbivores the microflora play the major role in conversion of cellulose and its products to volatile fatty acids, in addition to conversion of nitrogenous substances to ammonia and protein. The activity of the flora is ceased in many cases when the pH is altered due to:
I- Incorrect diet.                2- Prolonged starvation.
3- Antibiotics & sulphonamides administration.
4- Protozoal diseases.

**IV - Absorptive function:**
It is affected by increased motility or the presence of a disease affecting the intestinal mucosa.

**V - Auto-intoxication:**
The theory of auto-intoxication is that the toxic amines and phenols produced by putrefaction of protein in the large intestine are normally detoxified in the bowel wall. When they are regurgitated into the small intestine they will be absorbed and cause depression & weakness.

**Manifestations of alimentary tract dysfunction:**
1) Inanition is the major physiological effect of alimentary tract dysfunction when the disease is chronic.
2) Dehydration in acute cases.
3) Shock in bad cases.
4) Abdominal pain.
5) Other manifestations include abnormalities of prehension, mastication and swallowing as follow:
   A - Prehension:
   **Including grazing and drinking. This may be affected by:**
   1) Paralysis of the muscle of the jaw or tongue.
   2) Deficiencies of the incisor teeth.
   3) Malposition of the jaws (as in rickets).
   4) Pain from stomatitis.
   in all cases, unless there is anorexia, due to systemic disease the animal is hungry, attempts to feed but can not do so.
   B - Mastication:
The disorders are manifested by slow jaw movement, interrupted by pauses, expression of pain if the cause is bad tooth. In painful stomatitis there is usually complete, refuse to chew. Incomplete mastication is evidenced by the dropping of food from the mouth while eating and the passage of large quantities of undigested materials in the faeces.

C - Swallowing:
Disorders in swallowing occur due to:
- Is usually due to a physical obstruction by a foreign body or tumors in the pharynx or the esophagus.
- Inflammatory swelling, in esophageal dilatation due to partial paralysis.
- Dysphagia is manifested clinically by forceful attempts for swallowing accompanied by extension of the head and neck, by forcible flexion & violent contraction of the muscles of the neck and abdomen.
- Lesions in the pharynx cause regurgitation of food or water through the nostrils and coughing them out.

**Diarrhea**

Inflammation or autonomic imbalance, in the alimentary tract leads to diarrhea. Lack of fluid absorption makes the faeces soft and bulky.

The common causes of diarrhea include:

1- Enteritis.
2- Incomplete digestion with the passage of excess fiber or other feed constituents.
3- Functional diarrhea occurs in excitement.
4- Hepatic fibrosis.
5- Increased venous pressure in the portal circulation caused by congestive heart failure.

**Constipation**

When the alimentary tract motility is reduced, constipation occurs. There is increased time afforded for fluid absorption in case of constipation.

**Common cases of constipation include:**

1- Severe debility. 2 - Deficient dietary bulk.
3- Dehydration.
4- Partial obstruction of alimentary tract.
5- Painful conditions of the anus, Paralytic ileus.
6- Grass sickness of horses is a specific disease in which severe constipation is accompanied by degenerative lesions in sympathetic ganglia.
7- It is a common sign in chronic zinc poisoning in cattle.

**Vomition (Emesis)**

**Definition:**
It is a forcible expulsion of the contents of the stomach through the mouth and nose which is accompanied by nausia.

**Occurrence:**
Vomition is not a diseases, but a symptom of a disease, it occurs frequently in carnivores (as dogs and cats) but rare in herbivores. Horses do not vomit, but if vomit it indicates rupture of the stomach or very serious gastric dilatation. Vomition in adult cattle and sheep is rare, but it may occur in young calves.

**Etiology:**
Vomition may be induced by causes which irritate the afferent nerves of the stomach (reflex vomition) or causes that act upon the vomiting centre (central vomition).

**A) Reflex Vomition: Induced by the following causes:**
1- Overloading of the stomach with palatable food.
2- Irritation of the stomach by fermented food, mouldy silage or drugs such as apomorphine or chloroform.
3- Gastric catarrh (gastritis) or gastro-intestinal catarrh specially in dogs.
4- Foreign body in the stomach or feeding on poisonous plants.
5- Torsion of the small intestine which is usually accompanied by colic.
6- Acute peritonitis.
7- Obstruction of the pylorus by tumors or a large number of gastrophilus larvae.
B) Central vomition

It is rare in animals but when present it will be due to the followings:

1- Injury or diseases of the brain specially the medulla oblongata where the vomiting centre is located.
2- In cases of uremia and hepatitis.
3- Action of some drugs as arecolin or apomorphine

Clinical findings:
It occurs in two forms.

Projectile vomiting:
It is based on reverse peristalsis and is not accompanied by retching movements. Large amounts of fluid material are vomited with little effort. This is the common form of vomiting in horses & cattle as a result of overloading of the stomach with food or water,

True vomiting:
It is accompanied by retching movements including contraction of both the abdominal wall and of the neck muscles with extension of the head.

Effect:
Loss of fluids & electrolytes or it may be followed by aspiration pneumonia.

Diagnosis:
The significance of vomiting depends upon its causes and the disease producing it. if it is due to over loading of the stomach it is usually not repeated, but repeated vomition indicates gastritis or peritonitis or causes of central vomition. Examination of the vomitus is essential to determine its site of origin.

Treatment:
1- You have mainly to treat the original causes.
2- Temporary treatment is to give the animal crushed ice per mouth, also you can give sedatives and narcotics such as chloral hydrate or you can inject novalgin intravenously or morphine intramuscularly (1/4 - 1 cc. for small animals).
3- Do not give food or water at least 24 hours, followed by easily digested food till complete recovery.

**Hematemesis (Blood vomition)**

It means bloody vomition from the stomach or from the neighboring hollows.

**Aetiology:**

The condition is common in dogs and mainly met with in cases of severe gastritis, tumors of the mucosa of the stomach or injuries of the mucous membrane of the stomach by foreign bodies. In horses, it is very rare but it may occur due to the presence of large numbers of gastrophilus larvae in the stomach which may cause obstruction of the pylorus.

**Clinical findings:**

Bloody vomiting, which is dark brown in color and acidic in reaction. If the blood passes downwards to the intestine, it will be seen mixed with the faces and causes passage of a black colored faces which will have a foetid odor. If the condition goes on, the animal suffers from anemia.

**Treatment:**

1- Absolute rest of the animal.
2- Give crushed ice continuously orally.
3- Injection of vitamin “K” to increase coagulability of the blood.
4- Calcium injections to improve coagulability.
5- Inject ergotamine subcutaneously in a dose of 5-10 cc. for large animals and 1/2-1cc. for small animals as nerve blocker.
6- Give only nutrient fluids and easily digested food.
7- If hemorrhage is severe, then give blood transfusion.

**Alimentary tract hemorrhage**

Hemorrhage into the stomach or intestine may occur as a result of
1- Ulceration and erosion of blood vessels.
2- Acute vascular engorgement such as that occurring in cases of:
a) intestinal obstruction.
b) Thrombosis of mesenteric arteries.
c) Acute gastritis or enteritis especially when caused by helminthes or protozoa which penetrate more deeply than bacteria or virus.

**Clinical findings:**
1. Hemorrhage in the stomach causes faeces in this case to be black or very black brown or has tarry appearance (melaena).
2. If the hemorrhage is in the intestine the changes in the faeces varies with the level at which blood originates as follow:
   a) If the blood originates from the small intestine the faces is brown black.
   b) If from colon-or caecum the color will be red.
   c) If from the lower colon and rectum the stools will contain clot of whole blood.

N.B.: Severe hemorrhage causes anemia or acute peripheral circulatory failure.

**Stomatitis**

**Definition:**
Is an inflammation of the oral mucosa and includes glossitis (inflammation of the lingual mucosa) and gingivitis (inflammation of the gums). Clinically it is characterized by:
- Partial or complete loss of appetite.
- Profuse salivation.

It is frequent in occurrence in cattle either as a primary (purely local) or secondary conditions (e.g. indigestion, Avitaminosis C and along the course of some infectious diseases.

**Types of stomatitis**

I - **Simple stomatitis (Catarrhal stomatitis):**
This type of stomatitis is an acute mild inflammation of the mm. of the mouth characterized by:
Salivation, redness and swelling of the oral mucosa.

**Aetiology:**
1- Traumatic injuries: While dosing, sharp foreign bodies awns plants and drinking hot liquids. Injuries to the mucosa may result from a metal speculum or mouth gag and during oral passage of a stomach tube, rough use of balling gun.

2- Chemical injuries:
   a) Is more common to be due to caustic drugs such as aromatic spritus, chloral hydrate or formaline.
   b) Prolonged use of mercury-arsenic iodide,
   c) Pastures may contain some irritant plants.
   d) Simple stomatitis may accompany indigestion in cattle, impaction of rumen and gastro.-intestinal catarrh.
   e) Infection may extend from abscess of cheeks or pharynx.

Clinical findings:
1- Initial sings are partial or complete refusal of food with painful mastication and salivation.
2- Drooling of saliva which may contain pus or shreds of epithelial cells.
3- Foam at the commissures of the lips.
4- The tissues of the mouth are red and swollen.
5- Fetid odour is present on the breath only if bacterial invasion of the lesion has occurred.
6- Enlargement of the local lymph nodes if there is bacterial invasion.

Pathogenesis

Physical and chemical agents produce lesions directly to the mucosa or gaining entrance to the mucosa by minor abrasions, or localization in the mucosa in the case of septicaemic diseases.

II - Other types of stomatitis may be classified according to the cause:
1- Bacterial stomatitis:
Is usually necrotic and is manifested by ulceration and suppuration. The only common cause is oral necrobacillosis caused by sphaerophorus necrophorus.

2- **Mycotic stomatitis:**
It is in most cases caused by infection with Monilia spp. fungi. It usually takes the form of a heavy white deposit with little inflammation or damage to the mucosa.

3- **Viral stomatitis:**
Caused by specific viruses and this type assume a number of forms:

a) **Vesicular stomatitis:** The vesicular lesions are usually thin-walled vesicles 1-2 cm in diameter, filled with clear serous fluid. When the vesicle rupture it leaves sharp edged shallow ulcers as in cases of foot and mouth disease, vesicular stomatitis and vesicular exanthema.

b) **Erosive stomatitis:** Erosive lesions in viral stomatitis are shallow, usually discrete areas of necrosis. This type of lesions tends to occur most commonly on the lingual mucosa and at the commissures of the mouth the necrotic tissues may remain but usually shed leaving a very shallow discontinuity of the mucosa with a dark-red base.

c) **Suppurative stomatitis:** The lesions penetrate more deeply to the lamina propria. The erosive and secondary ulcerative stomatitis occurs in Rinderpest, mucosal disease, blue tongue, infectious ulcerative stomatitis.

**N. B.:** Ulcerative dermatitis, sheep-pox and contagious exanthema are primarily skin diseases but may involve the alimentary tract including the oral cavity.

d) **Proliferative form** occur in proliferative stomatitis, papular stomatitis and in rare cases of papillomatosis.

Many other causes of stomatitis are met with in the field but cannot be defined as belonging to any of the above conditions. An example is ulceromembranous gingivitis seen in sheep where ulceration begins at the gum-tooth margin and penetrates down into the alveoli causing expulsion of the teeth. Another example is allergic stomatitis.

4- **Gangrenous stomatitis:**
May be a complication of any of the above-mentioned types. It is characterized by a rapid extending necrosis and destruction of the m.m. Ulcers are covered by a slimy yellowish material. Mouth has a very bad fetid smell.

General clinical findings:
1- Partial or complete anorexia.
2- Salivation may be frothy or profuse, and drools if the animal does not swallow normally.
4- Saliva contains pus or shreds of epithelial tissues.
5- A foeted odor if bacterial invasion of the lesion has occurred.
6- Enlargement of local lymph nodes with swelling of the face in some cases where a cellulitis or Phlegmon extends to involve soft tissues.
7- Increased desire for water.
8- Manipulation and examination of the mouth is painful to the patient.
9- Toxemia may be present when the stomatitis is secondary to a systemic disease.

Local lesions are dealt with in the different types above.

Lab. examination:
1- Materials from lesions could be examined for the pathogenic causative agent.
2- Transmission experiments with filtrate or swabs in cases of suspicion of viral agents.

Diagnosis:
1- Particularly in cattle diagnosis is important and the signs with the history can be used to differentiate between the different causes.
2- Examination of the other organs.
3- PM. examination in suspension of viral diseases.
4- Laboratory examinations is necessary to define the causative agent.

Treatment:
1- Isolation of diseased animals which should be fed and watered from separate utensils.
2- Specific treatment are dealt with under the specific diseases.
3- non specific treatment includes:
  a) Frequent application of a mild antiseptic solutions such as:
     - 2% sol. of copper sulphate.
     - 2% suspension of borax.
     - 1% suspension of sulphonamides in glycerin.
     - 1 % suspension of acriflavine in glycerin.
     - 2% sol. of pot. chlorate or alum.
  b) In ulcers diphtheroid lesions require curettage or cauterisation with a silver nitrate stock or tincture of iodine (2.5%) in 10% glycerin.
  c) Care should be taken to teeth especially in cases of trauma.
  d) There may be need in some cases to antibiotic application.

**NB.:** You have to repeat this treatment daily till complete recovery.

**B - Diseases of the salivary glands**

**1 - Salivation (Ptyalism)**

It means secretion of saliva in abnormally excessive amounts. It is not a disease but a symptom of various affections.

**Aetiology: It is due to the following causes:**

1- Diseases of the mouth and throat: inflammation of the salivary glands, defective tooth or presence of foreign bodies in the mouth.

2- Reflex stimulation may cause profuse salivation in cases of choke (oesophageal obstructions) or in acute indigestion. It is also found in impaction of the abomasum.

3- Salivation may be excessive due to administration of certain drugs as injections of arecoline or acetylcholine or when giving nux vomica.

4- It may occur in the course of some specific infectious diseases as foot and mouth disease, rabies, thieleria affection in cattle and certain brain diseases.

**Clinical findings:**
Increased salivation and the saliva flows from the commissures of the mouth in the form of long strings. If salivation is continued for sometime, the animal becomes emaciated due to weakened mastication and the empty movement of swallowing.

**Treatment:**
Temporary relief is obtained by subcutaneous injections of atropine sulphate in a dose of 30-50 mg in large animals and from 2-3 mg in small animals (given as atropne sulphate sol. 2%).

**2 - Parotitis**
It means inflammation of the parotid gland or any other salivary gland.

**Aetiology:** may be due to:
1- Mechanical injuries as entrance of foreign bodies (calculi) in the stensons duct (parotid duct) which joins the parotid gland with the mouth or due to trauma from outside.
2- May be due to extension of the inflammation from the adjoining parts as pharyngitis or stomatitis.
- Occur in the course of some specific infectious diseases as strangles in horses or distemper inflammation, in the presence of calculi in the salivary duct as well as in the presence of Actinobacilosis and tuberculosis in cattle.

**Clinical findings:**
1- In the acute form, there is hot painful swelling of the parotid salivary gland accompanied by Dysphagia. There is increased salivation. The animal uses its jaws slowly and carefully. Abscess formation may take place in the parotid gland.
2- In the chronic form, the parotid gland will be hard intense and swollen, but it is painless and it may lead to induration of the part.

**Prognosis:**
The acute type is always favorable and if the condition is not complicated with pyogenic micro-organisms it will subside gradually. The chronic type when accompanied with the presence of calculi will be difficult to be treated.

**Diagnosis:** It is based on:

Symptoms and you have to distinguish this diseases from pharyngitis and the other infectious diseases mentioned above.

**Treatment:**

1- In the acute form, without abscess formation, the local treatment consists of applying cold fomentation socked in a weak antiseptic solution as (1-2% carbolic acid solution, or Lysol solution or 0.5% potassium permanganate solution on the affected gland. Then apply resolvent ointment as iodine 10% or ecthyol ointment 20- 30% or 10% compher ointment,

2- If there is abscess formation in the gland, you have to ripen it by using local hot fomentations and then ecthyol ointment and later on open surgically but you have to avoid the formation of fistula. It is better before opening surgically to use the intramuscular injections of antibiotics for 3 days and sometimes it may respond to the antibiotic treatment without need to opening.

3- In the chronic form of parotitis where there is induration, you have to fasten the resorption by the local application of tincture or injecting the affected gland with lugol’s iodine solution into various parts (injecting 5 cc. at intervals of 7 to ten days) You can give also potassium iodide internally orally in a dose of 8 grams daily for cattle for few days, then stop for 5 days and carry again and so on (to prevent the symptoms of excess iodine in the body).

4- If the cause of parotitis is the presence of a calculus in the stenson’s duct, then you must get rid of it surgically but avoid the formation of fistula.

**C - Diseases of the pharynx**

1) **Pharyngitis:**

**Definition.** It is an inflammation of the pharynx and it is
characterized clinically by:
1- Coughing. 2- Painful swallowing.
3- Lack of appetite. 4- Regurgitation through the nostrils.
5- Drooling of saliva may occur.

Aetiology:

It occurs commonly as a part of some other primary disease.

1- Ingestion of foreign bodies (cereals-awns-or gelatin capsules which may lodge in the pharynx) all these cause local ulceration and irritation.
2- Ingestion of irritant chemicals or hot or cold substances that cause stomatitis & pharyngitis.
3- May occur as a secondary condition in some specific diseases as strangles in horses, oral necrobacilisis, Actinobacillosis, Granulomatous lesions, pharyngeal anthrax in dogs and horses.
4- Upper respiratory tract diseases may involve the pharynx and cause pharyngitis.

Clinical findings:

1- The animals may refuse to eat or drink.
2- If it eats, swallowing will be with evident pain.
3- Manual compression of the throat causes paroxysmal cough.
4- Opening of the jaw for examination is restricted.
5- Mucopurulent nasal discharge, sometimes blood is evident.
6- Regurgitation of food through nostrils & oral medication in such cases is impossible.
7- Affected animals often stand with head extended, drool saliva and make frequent jaw movements.
8- If local swelling is severe there may be obstruction of respiration.
9- Regional lymph nodes are enlarged.
10- In pharyngeal Phlegmon in cattle there is an acute onset with high fever.
11- Severe toxemia may accompany the local lesions especially in oral necrobacillosis.

Diagnosis
1-The syndrome is manifested by acute onset and local pain. In pharyngeal paralysis the onset is slow. In obstruction with a foreign body there is a severe distress and continuous expulsive coughing.
2-Palpation of the pharyngeal region may be made with the aid of a gag if a foreign body is suspected.
3-Endoscopes examination through the nostrils is often of diagnostic value.

**Clinical Pathology:**

Identification (by culture) of the causative agent from nasal discharge or oral lesions.

**Prognosis:**

- Acute cases subside in 3-4 days.
- Chronic case may persist for weeks especially if there is ulceration or a persistent foreign body.

**Treatment:**

I-Primary disease must be treated first, usually by the use of antibiotics or sulphonamides.
2-In horses, drugs may be mixed with syrup, as an electuary or as a topical spray. Potassium chlorate could be added to drinking water (40-50 gram).
3- Inhalations in the recovery stage (creoline, pine oil or turpentine 150 gram/gallon of water.
4- Electuaries containing sedative expectorants may be administered.
5- Pharyngeal Phlegmon is fatal so early treatment with broad spectrum antibiotics may save the animal.

6- Apply expectorants as follow:

a) Ammonium chloride 16 gram
Ammonium carbonate 16 gram
Camphor 4 gram
Fluid ext. belladonna 30 ml
Syrup 500 ml
For large animals give 15-30 ml orally every 4 hrs.
b) Put 15-30 cresol in a pail of steaming hot water. Allow the animal to inhale vapour for 10-12 minutes, several times daily.

2- Pharyngeal obstruction

**Obstruction of the pharynx is accompanied by:**
1- Stertorous respiration. 2- Coughing.
3- Painful swallowing. 4- Lack of appetite.

**Aetiology:**
1- Enlargement of the retro pharyngeal L.N. in cases of tuberculosis, Actinobacillosis & lymphomatosis especially in cattle & in strangles in horses.
2- Presence of obstructive foreign body-as solid sharp pieces or wire…etc.
3- Diffuse enlargement of lymphoid tissue in the pharyngeal wall (cattle & pigs).

**Pathogenesis:**
Reduction in caliber of pharyngeal lumen will interfere with swallowing.

**Clinical findings:**

**Difficulty in swallowing.**
- The animals attempt to swallow without success and the food is coughed up through the nostrils.
- The presence of snoring respiration which is often loud and is heard some meters away. The inspiration is prolonged & accompanied by marked abdominal effort.
- Auscultation over the pharynx reveals loud inspiratory Stertorous sound.
- Palpation on the pharynx may reveal the nature of the lesion.
- Emaciation when the disease is prolonged.
- Aspiration pneumonia may occur when a nasal tube is passed and a L.N. abscess ruptures.

**Clinical pathology:**
1-T.B.test.
2- Blood picture must be carried out to evaluate the animal condition.
3- Nasal swabs for bacteriological examination especially for the presence of streptococcus equi.

**Diagnosis:**
1- Signs of the primary disease may aid in diagnosis of the Actinobacillosis, Strangles .... etc.
2- The presence of stertor (snoring sounds) during respiration.
3- Rejection of ingested food.
4- It is important to differentiate between obstruction & paralysis when rabies is present in the area where absence of pain & respiratory obstruction is clear in paralysis.
5- Laryngeal stenosis causes snoring but swallowing is not impeded. This is very important to differentiate between pharyngeal obstruction and laryngeal stenosis.
6- Nasal obstruction is manifested by noisy breathing more wheezing than snoring. This is also can be used for differentiation between pharyngeal and nasal obstruction.

**Treatment:**
1- Removal of the foreign body through the mouth.
2- Actinobacillosis-lymphadenitis are treated with iodides.
3- Parenteral treatment of abscesses with penicillin.

**3-Pharyngeal paralysis**

**The paralysis is manifested by:**
- Inability to swallow.
- Absence of pain and respiratory obstruction.

**Aetiology:**
1- Accompanies rabies, or encephalitis or botulism.
2- Trauma leading to peripheral nerve damage.
3- Spread of suppurative process or pressure by tumor or abscess.
4- In horses, nerve damage may result form the formation of diphtheritic membranes in the guttural pouch.

**Clinical findings:**
1- Animal is hungry but food drops from the mouth during attempts to swallow. The animal may regurgitate the food from nostrils during coughing.
2- Salivation occurs constantly.
3- Failure to stimulate swallowing by external compression.
4- Rapid loss of condition.
5- Clinical signs of the primary disease may be evident.
6- The condition known as cud-dropping occur in cattle is a of partial paralysis. There is difficulty in controlling the regurgitated bolus and it drops from the mouth.

**Diagnosis:**
Physical examination and the use of endoscope to exclude pharyngeal obstruction.
2- Paralysis is a typical sign in rabies, botulism and other encephalitic diseases.
3- Absence of pain.

**Treatment:**
No specific treatment but local application of heat may be attempted. Feeding by nasal tube may be tried. Disappearance of paralysis seems to be probably without specific treatment.

**Diseases of the esophagus**

**4- Oesophagitis**

**Inflammation of the esophagus which is accompanied by signs of:**
1- Spasms and obstruction.
2- Pain on swallowing and palpation.
3- Regurgitation of blood-stained slimy materials.

**Causes:**
1- Ingestion of chemicals or irritant materials.
2- Laceration of the mucosa by foreign bodies introduced into the rumen, stomach tube etc.

N.B: Death of hypoderma lineata in the submucosa of the esophagus causes acute local inflammation.

3- Secondary to some infectious diseases causing also stomatitis either viral or bacterial.

**Pathogenesis:**
Inflammation leads to an increase in muscle tones and involuntary movement with local edema and swelling leading to functional obstruction.

**Clinical findings:**
1- Salivation and attempts to swallow which cause severe pain particularly in horses.
2- In case of swallowing soon the food is regurgitated mixed with fresh blood and mucus, coughing, retching movements, with vigorous contraction of cervical & abdominal muscles.
3- Palpation on the cervical part of the esophagus (in the jugular furrow) causes pain when inflammation is in this region.
4- If perforation has occurred (in the cervical part there is local pain swelling & crepitus. Local cellulitis may occur causing fistula to the exterior. Regional hifiltration may occur leading to obstruction and toxemia.
5- Perforation of the thoracic esophagus leads to fatal pleurisy.
6- Signs of viral diseases (mucosal disease, malignant head catarrh) where the lesions being mainly erosive.

**Diagnosis**
1- Oesophagitis resemble pharyngitis but in pharyngitis the attempts to swallow are not so severer however both may occur together.
2- Local palpation may help to localize the lesion.
3- Surgical interference could be adopted in case of foreign bodies to ensure the cause.
4- Complete oesophageal obstruction is accompanied by bloat.

**Treatment:**
1- Food should be stopped for 2-3 days and intravenous injection of glucose should be supplied.
2- Parenteral antibiotic administration especially if laceration or perforations occurs.
3- If the animal can swallow astringent & antibacterial electuaries should be given at frequent intervals.

5- Oesophageal obstruction

Oesophageal obstruction may be acute (shock) or chronic. The major clinical signs are:
1- Inability to swallow.
2- Regurgitation of food and water.
3- Bloat in ruminants.

Causes:

**Dogs:** Feeding of bones or other objects during playing.

**Cattle:** Obstruction caused by solid objects as turnips, potatoes, comb of maize or upper parts of sugar cane.

**Horses:**
1- Incompletely masticated and unsalivated dry fed.
2- While dosing with gelatin capsules.
3- Cordial obstruction caused by carcinoma of the stomach.

**All animals:**

**Chronic obstruction may result from:**

a) Stenosis after Oesophagitis.

b) Pressure from enlarged lymph nodes due to any cause as TB, tumor, etc.

c) Persistent right aortic arch in new-born animals.

**Pathogenesis:**

Physical inability to swallow and prehension.

Eructation which leads to bloat.

**Clinical findings:**
Acute obstruction or choke:

Cattle:
1- The site is usually in the cervical esophagus just above the larynx or at the thoracic inlet.
2- Animal stops eating and shows anxiety and restlessness.
3- Forcible attempts to swallow.
4- Salivation-coughing-continuous chewing movements,
5 - Incomplete obstruction with bloat.
6- Ruminal movements are continuous and forcible.
7- Systolic murmurs are heard on heart auscultabon.
8- Passage of stomach tube is impossible.
9- Persistent obstruction causes pressure necrosis of the mucosa and perforation may occur, or stenosis may result due to fibrous tissue constriction.
10- Acute signs may subsides within few hours due to relaxation of the oesophageal spasm, but tympany persists. Many obstructions may pass spontaneously, other may persist for several days.

Horses:

Site:
Obstruction is often in the terminal part of the thoracic esophagus and cannot be seen or palpated.

Clinical signs:
Like that of cattle in general, but in horses it takes a violent picture. Gelatin capsules may remain in situ 3-4 days.
Death may occur in either species due to aspiration pneumonia and dehydration.

Chronic obstruction:
1- Absence of acute signs.
2- Chronic bloat which persists for very long period without the appearance of other signs.
3- Rumen hypermotility at the first weeks occurs then the tone is usually depressed (cattle).
4- In this form swallowing movements are affected where they are normal at first till the bolus reaches the obstruction then replaced by forcible movements.
5- Dilation of the esophagus may cause a pronounced swelling at the base of the neck.
6- Notice the swallowed material if either passes slowly through the stenotic area or accumulates and is regurgitated.

**Clinical pathology:**
1- Roentgenological examination may be helpful to outline the site of stenosis, diverticulum, or dilatation.

**Diagnosis:**
1- The clinical picture is quite sufficient to diagnose the case, but may be mistaken with Oesophagitis, in Oesophagitis local pain is more apparent and there may be stomatitis and pharyngitis.
2- Differential diagnosis must be carried from pharyngeal paralysis by absence of pain etc.
3- Chronic obstruction may be difficult to diagnose but the following articles are helpful.
   a) History of acute obstruction suggests cicatrical stenosis.
   b) Enlargement of lymph nodes are accompanied by the specific signs of TB, or lymphomatosis.
   c) Chronic Ruminal tympany, due to atoriy of Ruminal wall, is characterized by abnormal movements.
   d) Diaphragmatic hernia and vagus indigestion are another causes of chronic tympany but all these are accompanied by systolic murmur, and the passage of a stomach tube is impaired.

**Treatment:**
1- In acute obstruction sedate the animal at first by any ataractic drug or chloral hydrate may help to relax the oesophageal spasm or the use of other relaxants such as:
- Atropine sulphate (16-32 mg).
- Fluid extract of belladonna (1-2 ml).

2- Passage of stomach tube to locate the obstruction.

3- Gentle attempts may be made to push the obstruction onward avoid damage of the oesophageal mucosa.

4- In cattle where solid obstruction is in the upper esophagus, you can reach it by passing the hand or large forcipes into the pharynx through a speculum and by the help of an assistant to push the foreign body upward. A wire bent as a loop may help also to pull the object upward. In cattle you have to watch tympani. In emergency cases trocar the rumen and leave the cannula until obstruction is relieved. If all these fail to pull it out, leave the animal for a while and apply relaxants for the esophagus.

Horses:

1- Some difficulty is met with in removing those materials which accumulate in the lower esophagus in horses. In this case, small quantities of warm saline are introduced through a stomach tube that passed to the site of obstruction-then the saline is pumped or siphoned out. Repeat this several times until fluid comes clear. If obstruction is still present fluid extract of belladonna should be administered before removal of the tube. Further attempts could be made at shorten the intervals but avoid aspiration pneumonia.

2- In case of a palpable obstruction in the neck vigorous squeezing from exterior may brake it up and aid in its removal.

3- Surgical removal by oesophagotomy may be necessary if other measures fail. Treatment of chronic obstruction is usually unsuccessful.

Complications

Suffocation, asphyxia and death.

2- Subcutaneous emphysema if the obstructive material causes perforation of the esophagus.

3- unrelieved choke may lead to fatal paralysis and necrosis of the esophagus.
4- Death may take place from drenching pneumonia which arises from the passage of the saliva to the respiratory tract.
5- Tympani appears and will be severe one if there is complete choke.

6- **Stenosis of the esophagus**

**Definition:**
It means partial obstruction of the esophagus.

**Etiology:**
1- Due to thick mucous membrane of the esophagus in cases of chronic Oesophagitis.
2- Presence of abscess or tumor in esophagus itself or in other organs in the neighborhood which press on the esophagus form outside and causes narrowing of the lumen.
3- It is caused by the enlargement of the mediastinal lymph glands in case of tuberculosis.
4- May be due to nervous troubles.

**Clinical findings:**
Difficulty in swallowing and only small quantity of food could be taken. Pain is noticed during and after feeding vomiting is a symptom in the dog and lastly the animals refuse to take its food. Emaciation is noticed. Sometimes swallowing may be normal but rumination is obstructed. There is a recurrent tympany or a chronic one due to the stenosis of the esophagus or of the pylorus.

**Treatment:**
Stenosis due to pathological changes in veterinary medicine is incurable.

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**D - Diseases of the stomach**

1 - **Gastric dilation**

**Dilation of the stomach is accompanied by:**
- Severe abdominal pain.
- Projectile vomiting.

**Aetiology:**
1-Sudden & complete obstruction of the pylorus by foreign body (Hair balls) or achalasia.
2- Gross overeating or drinking.
3- External compression by lipoma in horses.
4-The condition may occur in old debilitated animals fed for long periods on indigestible roughages
5-Wind-sucking in horses.

**Pathogenesis**

Dilation of the stomach stimulates vomition with consequent increase in gastric motility leading to powerful peristalsis towards the pylorus causing pain.

- Reflex depression of the cardia and peripheral vascular system results in the occurrence of shock and their may be a reflex depression of respiration.
- Excessive secretions & loss of fluids causes fatal dehydration and alkalosis,
- Local damage to gastric mucosa may cause additional shock.

Increase permeability of mucosa to byproducts produced by putrefaction causes toxemia.

- Rupture of the stomach may also occur.
- In case of wheat eating in cattle in large quantities, there will be production of large quantities of lactic acid in all parts of the rumen and consequently this will affect the bowel causing an increases in the osmotic pressure of it, which leads eventually to the passage of much fluid into the lumen, and this will eventually leads to sever dehydration with mild acidosis and the condition finally will cause laminitis.

**Clinical findings**

1-Vomiting is a cardinal signs in acute gastritis. Vomiting is usually projectile in nature. in the horse vomiting materials may come form the nostrils (terminal event) followed by rupture.
2- In case of grain engorgement in horses vomiting does not occur as grain absorb much of fluids.
3- Abdominal pain is severe in horse. This is manifested by: sweating rolling, kicking at the belly, sitting on the hunches. The dog sitting in horses helps the animals to relief pressure on the diaphragm to facilitate breathing.
4- Dehydration, looseness of the skin elasticity sunking of the eyes.
5-When the alkalosis develops the signs are tetany, tremors and rapid shallow respiration.
6- Pulse rate is increased (rapid and progressive).
7- Passage of stomach tube results in evacuation of a large quantities of foul smelling fluid.
8- In chronic dilation there is anorexia, mild continues or current pain scanty faeces, loss of weight, occasional vomiting after feeding and moderate degree of dehydration.
9- If rupture has occurred, vomiting cases, the animal stands quite, cold, sweating tremors occurs, subnormal temperature and severe congested m.m., pulse reaches 120/rn and is very weak. Peristalsis are absent.

**Lab. diagnosis:**
1- Examine the vomitus to determine the origin and nature of the contents.
2- Roentgenological exam. may be of value in young animals.

**Diagnosis:**
1- Profuse projectile vomiting more than in enteritis & intestinal obstruction. Both contain bile and are alkaline in nature.
2- History of overeating.
3- Relaxant drugs for functional suspect of pyloric obstruction in case of no history of over feeding and to differentiate between the functional from physical causes.
4- Rectal examination in horses is of diagnostic value:
Displacement of the spleen occurs medially and caudally within the abdominal cavity. In severe cases a sharp, firm edge may be found in the middle of the pelvic inlet.
5- In case of stomach rupture the examination reveals the very flaccid intestine due to complete absence of peristalsis.
Treatment:
1- Sedate the animal by giving 30-40 gram chloral hydrate i/v or orally by a stomach tube.
2- Evacuation of the stomach by the aid of a large stomach tube could he tried and in most cases is unsuccessful. The procedure is done by pumping 1-2 gallons of normal saline then siphoned out. The difficulty is met with when there are grains.
3- Another trial is the administration of mineral oil (one gallon) or 2 liters linseed oil followed by a parasympathetic stimulant. There is a danger as this latter drug may cause rapture of the stomach.
4- It is preferable to keep the animal in the standing position to prevent rolling otherwise torsion is likely to occur.
5- Periodical removal of gastric fluid may relief discomfort, but in this case i/v administration of electrolyte solutions is necessary.
6- Repeated administration of belladonna preparations may help in relaxation of the pylorus. Horses: 30-60 mg S/C two to four times daily.
7- Gastrotomy is advisable in young animals when physical action due to tumors or foreign bodies are in suspect
8- Chronic atone may be treated by administration of: R/14 ml tincture of nux vomica given twice daily.
9- You can give orally salicylic acid 15-25 gr. (large animals) to control fermentation.

2 - Gastritis

Definition:
Inflammation of the stomach causes abnormal motility and it is manifested clinically by vomiting. This might be acute or chronic & is commonly associated with enteritis in the syndrome of gastro-enteritis.

Aetiology:
The inflammation may be caused by:
1- Physical agents:
a) Grass over-feeding causing gastric dilation and is usually accompanied by secondary gastritis. Ingestion of coarse fibrous feeds as straw bedding or bad teeth leading to faulty mastication has the same effect.

Foreign bodies may lacerate the mucosa as in reticuloperitonitis in cattle.

Damaged feeds, mould & fermented hay cause moderate gastritis.

2-Chemical agents:
Caustics, irritant poisons (arsenic, lead, copper, mercury and phosphorus nitrate) cause severe gastro-enteritis.

Excess production of lactic acid in the rumen after grain engorgement causes ruminates & gastroenteritis.

3-Bacterial agents:
Examples: oral necrobacillosis, hemorrhagic enterotoxaemia, colibacillosis in calves, salmonellosis etc.

4-Viral agents:
Rinderpest and mucosal diseases in ruminants causes abomasal and Ruminal lesions.

5- Fungal agents:
Fungi cause ulcerative gastritis in newly born animals.

Exam: Moniliasis.

6- Metazoan agents:
b) Horses: Larvae of Habronema megastomum cause ulcerative lesions of the stomach which lead to perforation and

Pathogenesis:
Inflammation of the stomach may be either:

1-Acute reaction where increased motility causes pain increase peristalsis) with rapid emptying of the stomach by vomiting or via the pylorus in animals which do not vomit.
2-Chronic reaction causes increased secretion with delayed food indigestion which permits putrefaction and leads to further inflammation to the intestine.

**Clinical findings:**

**Acute gastritis:**
1- Vomition: The vomitus contains much mucous, may be tinged with blood. Repeated vomiting is accompanied by retching movements.
2- The appetite is always reduced or absent.
3- Excessive thirst.
4- Breath has a rank smell.
5- Diarrhoea is present then there is gastro-enteritis.
6- Faeces are pasty and soft.
7- Signs of the primary disease.
8- Dehydration & alkalosis with tetany & rapid breathing may develop if vomiting is excessive.

**Chronic gastritis:**
1- Signs are less severe with depraved appetite.
2- Vomiting occurs sporadically (after feeding) the vomitus contains viscid mucous.

**Diagnosis:**
1- Gastritis & gastric dilation have some similarities. In the latter the vomitus is more profuse and more projectile.
2- Oesophageal obstruction: vomitus is not acid or rancid.
3- Intestinal obstruction: always contain bile and alkaline in reaction.

**Lab. diagnosis:**
1- Exam, of the vomitus.
2- Estimation of gastric acidity. Faecal examination.

**Treatment:**
Treat the primary cause.
Many preparations could be prescribed. In the market there are lot of patent preparations.

1. Gastric sedatives: Preparation of:
   - Magnesium hydroxide.
   - Magnesium carbonate
   - Kaolin-Pectin-charcoal.
   - Bismuth subnitrate or carbonate.

2. Gastric asthngents’ Preparation of:
   Tannic acid and catechu.

3. Fluid therapy:
   To replace the electrolytes lost by vomition. (These electrolytes are Na, K, Ca, Mg.). These are given by I/v. and orally when vomiting stops.

4. Some chemotherapeutic drugs and antibiotics are also prescribed as guanirnycin and sulphaguanidine.

5. If purgatives are used to empty the alimentary tract use mineral oil to avoid further irritation by other purgatives.

6. During convalescence, give the animal soft, highly nutritional feed.

7. Alimentary tract stimulants as strychnine preparations ammonium carbonate to hasten the return of gastric motility.

8. Vitamin LB6 in a dose of 50-100 mg every other day.

9. Glutamic acid (capsules) one capsule with each meal.

3 - Gastritis in dogs

Aetiology: A) Primary causes:
1. Over eating.
2. Allergy to certain feed materials.
3. Eating of decomposed feeds.
4. Ingestion of foreign bodies as small pieces of hard rubbers (in acute forms) or prolonged presence of foreign body (in chronic form).
5- Chemicals (Phenols or any other harmful chemical).
6- Poisonous materials (as P, ... etc.).
7- Thermal causes.
8- Parasitic causes (migration of ascaris larvae or presence of tap worm in the intestine).
9- Poisonous plants

**B) Secondary causes:**

Along the course of some infectious diseases as leptospira, infections canine hepatitis and chromic nephritis.

**Clinical findings:** The gastritis in dogs may be acute or chronic:

**a. Acute gastritis:**

1- Repeated vomition which varies in its nature according the cause, thus in over-eating the animal has to repeat the vomited material and if the cause decomposed food the animal vomits explosively, allergic causes the vomition is periodical followed transitory enteritis and sometimes by Urticaria, cause is chemical or poisonous material or plants the vomition is severe and in some hemorrhagic. In other cases the vomition is repeated varies in its frequency and severity according severity and nature of the affection.

2- Abdominal pain (usually severe in nature).

**b. Chronic gastritis:**

1- Vomition is occasional.

2- Intermittent weak appetite.

3- Gradual emaciation.

4- Anemia.

5- Disturbed gastric digestion and function, with increased formation of mucus.

**Treatment:**

1- Administration of fluid electrolytes i/v to treat dehydration if present.

2- Oral administration of bismuth nitrate in solution containing belladonna to protect gastric mucosa and lessen gastric motility in acute cases.
3- Give antiemetic drug (Diphenyldramine hydrochloride I/v 12 mg/kg body weight. or vit B6.
4- Give liquid easily digested food for 24-48 hours.
5- In chronic gastritis give nerve tonics (strychnine).
6- R/ Sod. bicarbonate 0.3 gm
   Cal. carbonate 0.6 gm
   Mag. trisilicate 1.0 gm
   Glucose 1.0 gm
Sig. mix and give at once as one dose.
7- Give nimarol table spoonful 3 times daily.
8- Sulpha drugs and antibiotic by oral rout.
9- Sufficient water must be given especially in ruminants.
10- During convalescence give soft palatable food.
11- Antiemetic.
12- Gastric nerve tonics e.g. strychnine preparations.

3- Gastric ulcer

Definition:
Ulceration of the gastric mucosa is characterized by the presence of a collection of symptoms including anorexia, abdominal discomfort; intestinal motility giving rise to either constipation or diarrhoea and in some cases gastric hemorrhage.

Aetiology:
Gastric ulcers in farm animals are usually traumatic or it may result from the following causes:
1- Long standing chronic vagus indigestion (specially in the abomasum).
2- Light feeding on corrosives or chemicals for a long time which will cause shallow erosion giving rise to ulcers.
3- Neglected treatment for cases of gastritis.
4- Tumors of the stomach may lead to ulcers.
5- It may occur in the course of some specific infectious diseases as Rinderpest.
6- Due to long standing parasitic infection in the stomach such as gastrophilus species or habronema megastoma.
7- In pigs, it may arise from high level feeding with antibiotics and arsenical compounds which lead to ulcers and ending with sudden death from hemorrhage.
8- It may occur due to vitamin C deficiency in pigs.

N.B.: The effects of gastric ulcers are largely reflex causing spasms of the pylorus and increased gastric motility.

**Symptoms:**
1- In uncomplicated cases, there are mild and intermediate signs of abdominal pain, anorexia and either constipation or diarrhoea.
2- In vomiting animals, vomition occurs and the vomitus contains mucous and is not highly acidic.
3- Haemorrhage may occur giving rise either to severe hemorrhagic anemia or sudden death from coetaneous Hematemesis.
4- The faeces are black in color, usually pasty in consistency and of small volume due to the accompanied spasms.
5- In cows, milk yield is suppressed with diminished appetite and pain can be detected by percussion on the abdomen. In the complicated cases, there is perforation in the stomach with rupture or severe hemorrhage of the stomach.

**Diagnosis:**
1- Depends upon the symptoms mentioned above.
2- Any animal comes to your clinic with a case history of emesis or Hematemesis, you have to suspect the probability of the presence of gastric ulcer.
3- Blood picture of such animals reveals sharp rise in total leukocytes and neutrophil percentage.
4- In dogs, X-rays help in diagnosis.
5- Faecal examination is done to denote the presence of eggs of stomach parasites.
Treatment:
1- Protective and astringent drugs are used (as in gastritis) and recommended for longer periods.
2- If hemorrhage takes place and is severe, blood transfusion or haematinie drugs are useful. In addition give anticoagulants as injections of calcium and vitamin K.
3- Food containing irritant materials should be avoided.
4- Surgical repair is useful.

N.B.: This disease is seen frequently more in carnivorous animals.

5- Rupture of the stomach
The rupture of the stomach occurs usually in its great curvature and always seen in the horse (as it cannot vomit).

Causes:
1- Acute indigestion and severe impaction.
2- Severe and serious cases of tympanitis.
3- Strongly spoiled and fennented food materials.
4- Severe attack with spasmodic colic.
5- Gastric ulcers.
6- Degeneration or weakness of the stomach muscles.
7- It may occur from carelessness by giving big doses of drugs as arecolin, pilocarpine or eserin.

Clinical findings:
The disease is most frequently seen in horses, In such conditions, there are severe symptoms of abdominal pain followed by sings of relief and vomition, then profuse sweating and the animal will sit on its hunches as in dog’s sittings in order to relief pressure on the diaphragm and lungs. The animal inclines its head fowards and there is a sudden fall in temperature followed by death in few hours.

E) Diseases of the intestine

I - Acute intestinal obstruction
**Definition:**
Intestinal obstruction includes volvulus, intussusceptions and strangulation. The clinical signs include: acute abdominal pain, severe shock, absence of defecation and the Dosage of blood and mucus.

a) Volvulus: Is an acute obstruction of the bowels caused by twisting of a loop of bowel around itself.

**Aetiology:**
1- indigestion associated with severe colic is the most frequent cause.
2- Violent movement and rolling or jumping and following struggling during casting in horses.
3- Sudden or irregular increase in penstaltic movements of the intestine after dosing with parasympathetic stimulant as carbacol or colityl.
4- Severe spasmodic colic.
5- Heavy infestation of nodular worms (Oesophagostomum columbianum) in sheep or ascaris in all animals may cause intestinal obstruction.

**Clinical findings:**
1- Severe continuous pain, proceeded by signs of colic (History of violent exercise) pain is mild at first followed by anxiety an finally distress and delirium.
2- Profuse sweating.
3- Violent movements, pawing, kicking at the belly and ear dropping. From time to time the animal falls on the ground then slowly rises up again.
4- The animal may assume dog sitting.
5- Congestion of the mucosa.
6- lab breath-Pulse 50-70 at first then increases to 100/m and becomes poor in quality.
7- Slight rise in temperature then suddenly falls (rising of temperature indicates bad prognosis).
8- Anorexia for food and water is complete.
9- Trembling is frequent.
10- In torsion of the colon persistent bloating is the rule.
11- Vomiting of retching type may occur (Especially in volvulus of small intestine).
12- Bowel evacuation is suppressed.
13- Peristalsis is absent.
14 The course is from 8-24 hrs. terminating in death.

**Rectal findings:**
In torsion of the small intestine examination in the upper left part of the abdomen reveals:
1- Irregular tympany.
2- One may locate painful spot that when touched causes the animal to kicks the belly.
3- Loops of small intestine may be found displaced to the right above the colon.
4- In cases of volvulus of ileum, a tense cord like twist of mesentery in the region of the left kidney is felt
5- Torsion of the colon leads to tympany.
6- Anterior to the pelvic rim you feel a cord like mass of colic vessels.

**Treatment:**
No useful treatment. Even surgical attempts are useless.
1- In the horse, manual reduction per rectum may be attempted when the large colon is affected and the twist is only partial.
2- Try to give large doses of liquid paraffin or oil of turpentine or linseed oil in cases of partial torsion and in addition apply back racking and enema of soft soap and warm water.
3- In complete torsion it is definitely useless.

**6 - Intestinal strangulation**
**Intestinal incarceration**
It is the occlusion of the intestinal lumen by pressure from the outside. It occurs:
I-When a loop of the intestine passes through a natural or artificial opening in the peritoneum and held there.

2- The bowl may be strangulated by the long stem of a pedunculated tumor or by other fibrous cords or bands.

3- In stallions, incarceration in the inguinal canal may take place due to inguinal hernia.

4- In bovines, the most frequent locations are the peritoneal adhesions. A segment of the intestine may also sometimes passes through a slit in the diaphragm (diaphragmatic hernia).

5- Rolling from colic seems to be a pre-disposing cause to such conditions.

**Symptoms:** The same as in torsion.

**Treatment:** as in torsion, but if there is hernia, early operation may be necessary.

C) **Invagination (Intussusceptions)**

This is a form of acute intestinal obstruction caused by the telescoping Invagination of a section of the bowl into a portion immediately behind it. The affected part forms a sausage shaped, firm and painful swelling composed of 3 segments an outer, a middle and an innermost layer. It occurs mostly in dogs and cattle. The more common seat is the ileo-caecal junction (as the ileum is always invaginated into the caecum or the colon).

**Aetiology:**

1- Irregular and violent intestinal peristaltic movement.

2- Tumors in the lumen of the bowel.

3- Severe enteritis particularly in young animals.

**Symptoms:**

1- Commences as a rule with subacute abdominal pain which rapidly becomes acute and resembles those of torsion.

2- The faeces may be passed tinged with blood.

3- Complete anorexia.
4- Pulse 80-100/m.
5- Breathing is shallow & irregular.
6- Bowel evacuation is suppressed.

**Diagnosis:** as in torsion.
1- Rectal palpation reveals a finny painilil circumscribed swelling just anterior to the tim of the pelvis in the right flank.
2- X-ray is a good diagnostic means specially in dogs.
3- In dogs, external palpation of the abdomen reveals the sausage-shaped material which could be clearly felt with fingers.

**Treatment:** -
1- As in torsion begin the treatment by giving large doses of liquid paraffin orally and give sedatives.
2- Surgical interference may be tried as quick as possible.

**Verminus aneurysm:**
Migration of the larvae of strongylus spp. worms into the wall of the cranial mesenteric artery and its branches occurs in horses which may cause restriction of the blood supply to the intestine, so it should be differentiated from intestinal obstruction which is accompanied by tympany:

<table>
<thead>
<tr>
<th>Thrombosis</th>
<th>Tympany</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pain</strong></td>
<td><strong>Tympany</strong></td>
</tr>
<tr>
<td>- Is less sever</td>
<td>- Is less severe.</td>
</tr>
<tr>
<td>- There may be passage of blood with faeces.</td>
<td>- Flatus is passed through the rectum.</td>
</tr>
<tr>
<td><strong>Rectal exam.</strong></td>
<td></td>
</tr>
<tr>
<td>Reveal the knobbly thickened obstructed mesenteric vessels in the midline level of the caudal pole of left kidney.</td>
<td>- May reveal no obstructed intestinal loops.</td>
</tr>
<tr>
<td>It pulsate with each pulse wave</td>
<td></td>
</tr>
</tbody>
</table>

2- **Enteritis**
Definition:
It is inflammation of the intestinal mucosa characterized by:

• Increased motility of the gut.
• Decreased absorption with increased secretion.
• Clinically it is manifested by: abdominal pain, diarrhoea and sometimes dysentery. In many instances it occurs with gastritis.

Aetiology:
There are many causes and the disease varies with type and severity of the causative agent. These causes are subjected to many factors from the infected host i.e. immunity, stresses, nutritional conditions, age .... etc.

1- Bacterial enteritis:
Colibacillosis, salmonellosis, pasteurellosis and enterotoxaemia (caused by clostridium perfringes types B and C) are all manifested by enteritis. In addition shigellosis in foals and anthrax.

N.B.: Antibiotics in some species, may alter the intestinal flora and permit the growth of Staph, proteus and fungi and cause diarrhoea.

2- Viral enteritis:
Cattle are the only species in which viral enteritis are common. Rinderpest-mucosal disease- bovine malignant head catarrh cause enteritis.

3- Protozoal enteritis: Coccidiosis.
Intestinal trichomoniasis may cause enteritis in horses.

4- Chemical agents:
Poisoning by lead, arsenic, phosphorus, copper and other chemicals causes enteritis when taken in large doses. Many poisonous plants cause severe enteritis. Overdosing with oral iron preparation may cause chemical enteritis in young pigs.

5- Parasitic enteritis:
Most common in farm animals. Examples are stomach flukes (Paramphistomum spp.). Trichostronylus spp., Copperia spp., Chabertia spp., and Nematodirus spp. One of the
important causes in cattle is:
Hookworms (Bunostomum spp. which cause enteritis in calves).
Heavy tapeworm (Monezia spp.), infestation can cause enteritis in sheep.
In horses Strongylus spp. and Trichonema spp. and Ascaris.
6- Physical agents:
Ingestion of large quantities of sand or dust causes enteritis especially in horses (Sand colic).
7- Overfeeding with grains which result in large quantities of lactic acid with the occurrence of enteritis.

**Pathogenesis:**
1- It depends upon the causative agent. There is either catarrhal inflammation with severe hemorrhagic enteritis or erosive and necrotic destruction of intestinal mucosa. The clinical signs vary accordingly.
2- Reaction of Epith. to inflammation.
Is desquamation of epithelium lining with increased motility which will cause absorption of fluids and impaired digestion which will lead to passage of the contents in a fluid state (diarrhoea). Consequently there will be incomplete digestion and putrefaction of protein with carbohydrate fermentation. Consequently there will be sour odour of faeces.
There will be loss of fluids and proteins leading to dehydration. In addition denotation of epithelium will lead to permission of absorption of toxic products with shock and consequent shedding of large areas of mucosa. This will be seen in the form of shreds or casts of mucosa in the faeces.
In chronic enteritis the intestinal wall becomes thickened, mucus secretion is stimulated with resultant decreased absorption and faeces will be thin watery and in addition it contains much mucus.

**Clinical findings:**
1- Gastritis commonly accompanies enteritis and signs of gastritis may be present.
2- In acute enteritis there is abdominal pain with its manifestation either in horse or other species. In chronic enteritis pain is seldom presents or it may be indistinct.

3- Diarrhoea is the charlatanistic sign of acute enteritis. Faeces are soft or fluid in consistency and have unpleasant odour.

4 Faeces may contain blood, shreds of mucosa and mucus especially in chronic type.

5- Anorexia is complete in acute enteritis but the appetite is normal in chronic type and thirst is usually increased.

6- Straining may occur especially in calves and may be followed by intussusceptions or rectal prolapse.

7- The distribution of the faeces on the hind parts may suggest the disease e.g. in case of smudge pattern appearance the infection with coccidiosis and straining as in these cases the faeces comes in a horizontal way. In helminthes infestation there is little sweating on the pine bones but the tail and inside of the hocks are largely coated.

8- Auscultation of the abdomen reveals increased motility and fluidity of the intestinal contents. Pain may be evident on firm palpation.

9- There may be shock, in heart rate in creased severe cases.

10- Typical signs of the specific diseases may be present, e. g. fever, lesions etc.

11- Acute cases may terminate within 24 hrs, but chronic enteritis persists for several months.

12- Signs of dehydration if diarrhoea is severe.

**Clinical pathology:**

1- Faecal examination to determine the causative bacteria, helminthes, protozoa .... etc.

2- It is very important to determine the fluid state, bicarbonate level and electrolytes as a base sound therapy.

**Diagnosis:**

1 It is not difficult because of the nature of the faeces. Diarrhoea occurs in other forms of hypermotility-but there is absence of abnormal odour or excessive mucous and
systemic reaction.

2-Chronic enteritis due to John’s disease and parasitic infestation must be differentiated by laboratory examination.

3- In cases of diarrhoea due to specific diseases, there are the signs of these diseases.

4- In sand colic of horses, the faeces usually contain sand. This could be also estimated quantitatively.

**Treatment:**

1- Primary treatment will depend on the nature of the causative agent and the application of the specific treatment.

**Basically treatment includes:**

a) Removal of the causative agent from the intestine.

b) Administration of astringent preparations.

c) Replacement of lost fluids and electrolytes.

2- Evacuation of the bowel by purgatives may sometimes be necessary to remove the toxic materials from the intestine but this may result in superpurgation (esp. horses) sand could be removed by repeated treatment with mineral oil.

3- Astringent and protective preparations as described under gastritis may reduce fluid loss.

4- Spasmolytic preparations are also useful in relieving pain especially in exited animals to prevent self injury.

5- Replacement of lost fluids and electrolytes is effective by parenteral administration.

6- Blood transfusion may be of some value in young animals.

7- Saline solution.

8- If antibiotics are used-therapeutic levels should be used because of the danger of creating resistant strains of bacteria.

9- Heart stimulants may be advised in some cases.

10- Nutrients in the form of i/v injection of dextrose. The following tables show the
doses of antibiotics commonly used in veterinary practice and in cases of enteritis.

**Antibiotics**

The following groups of preparations are available for parenteral and/or oral administration:

- Slowly absorbed sulphonamides.
- Short-acting sulphonamides.
- Long-acting, sulphonamides.
- Sulphonamide-trimethoprim combination.

- The initial dose in the case of slowly absorbed sulphonamides and short-acting sulphonamides should be 60-100 mg/kg body weight.

For the newer long-acting sulphonamides and the sulphonamide-trimethoprim combinations, a dosage of 20-30 mg/kg body weight is considered sufficient.

- Follow-up doses of 1/2 to 2/3 of the initial quantity are required at 12 to 36 hours intervals. Therapy should be continued for 3-5 days.

11- Give the specific vermifuge for the treatment of the various helminthes.

12- For toxic gastro-enteritis:

a) Remove the cause of the disease.

b) Wash the stomach with normal saline or magnesium sulphate solution by means of a stomach tube. This procedure is contra indicated in poisoning with couosive.

c) Give the specific antidotes for each sort of poison.

**Examples: for lead poisoning:**

**a) Spasmodic colic:**

A severe attach of abdominal pain caused by functional disturbance in the intestine, characterized by intermittent fits of colic, rapid course and favorable termination.

**Aetiology:**
Excitement in shows, races, drinks of cold water when the hours is hot and sweating after work are all predisposing causes.

**Pathogenesis:**

The above factors leads to:

increase in parasympathetic tone causing, Hypermotility with consequent spasmodic colic, resulting in pain With consequent evacuation of intestinal contents. Then spasmodic contractions case and the peristaltic movements remain active for sometimes.

**Clinical findings:**

1- The disease is characterized by short attacks of abdominal pain. The pain is intermittent. The horse manifest rolling, pawing & kicking these signs last for a few minutes, then stands normally for few minutes until the bout of pain occurs.
2- Loud intestinal sounds are often audible some distance from the horse.
3- Auscultation reveals a loud borborgymi sounds.
4- Pulse is elevated to above 60/min.
5- Sweating in patches.
6- The signs usually disappear spontaneously within few hours. A similar undrome occurs in calves and cows but the intestinal sounds are not usually increased.

**Diagnosis:**

1- Spasmodic colic may resemble enteritis because in both there are increased intestinal sounds and pain.
Diarrhoea is usually present in enteritis: But care could be taken that parasitic enteritis (Horse) may be an exception.
2- May be confused with intestinal obstruction but the presence of blood; mucus in the rectum and failure to pass faeces suggest obstruction.
3- Obstructive urolithiasis in horses has a similar posture but there are absence of sounds.
4- Abdominal pain due to uterine contractions may also occur but again there is no hypermotility.
Treatment:
1- The best treatment for hypermotility is by Spasmolytic agents as atropine. In horses as standard treatment is 1/4 -1/2 grain followed by the administration of mineral oil (2 liters).
2- Injection of pethidine as an analgesic and Spasmolytic.
3- Novocain 0.1 gram/100 kg bwt injected very slowly i/v may produce an immediate relief.
4- Promazine derivatives (tranquillizers) have also a Spasmolytic effect.
5- Novalgin 50% as i/v injection (20-25 ml) for a horse produced a rapid relief.
6- Rch1oral hydrate 30.0 oil of turpentine 36.00 sp. aeth: nitrosi 75.00 linseed oil ad 800.00
Give at once by a stomach tube. Dissolve the choral hydrate in a little quantity of water.
7- Enema with soft soap & warm water may stimulate and regulates the intestinal peristalsis in some cases.

b) Dietetic scour:

Aetiology:
1- Dietary abnormalities which is followed by secondary colibacillosis.
2- Drinking too rapidly.
3- Feeding of excessive quantities of milk at too long intervals and at temperature below body heat.
4- Feeding of a high milk fat. Poor clotting of milk leads to scour. Milk with a very low level of casein or calcium or with high level of sodium or high pH clots poorly.
5- Sudden change from whole milk to milk substitute.
6- Calves fed on milk from cows grazing on very lush pastures.
7- Occasional cases occur in foals and lambs when the dams have profuse milk supply.

Pathogenesis:
Ingestion of milk of a high casein content leads to the formation of a much denser abomasal curd which is indigestible. With the entrance of more milk, more curd will be
formed with more enlargement, this will cause abomasal dilatation consequently milk will pass in an undigested state leading to formation of scour.

**Clinical findings:**

1- Dietetic scour is characterized by the passage of soft fluids faeces varying from white to yellow to green color, depending upon the diet.
2- The appetite is good, but the calves loss their weight rapidly.
3- If the condition is prolonged, dehydration may become severe.

**Diagnosis:**

Dietetic scour resembles enteritis but the faeces are bulky and pasty rather than watery. Animals respond to palliative treatment (ordinary direct and correct treatment: -

**Treatment:**

1- Milk feeding should be stopped and the animal must be fed orally electrolyte solutions for 24 hrs then gradually re-introduced.
2- In foals they should be muzzled and allowed only limited access to the mare. Do not forget to make hand stripping to relieve tension in the udder of the dam.
3- Astringent and protective preparations are used (Refer to enteritis).
4- In severe cases antibacterial drugs are used.
5- Feeding system should be corrected after giving colostrums (after birth till 48 hours) followed by milk low fat content and the calves should be fed at last 3 time daily.
6- The addition of lime water (1: 2 parts of milk) aids digestion.
7- Oral transfers or rumen transplants is recommended and aids in digestion in young calves.

c) **Impaction of the ilio-caecal valve:**

The disease occurs chiefly in horses, causing a syndrome of subacute abdominal pain followed by acute one. It is commonly fatal.

**Etiology:**

The common cause is feeding on low-grade finely chopped roughage.

**Pathogenesis:**
The finely chopped straw collects in the terminal part of the ileum at the ileoaelcal valve which causes complete intestinal obstruction.

**Clinical findings:**
1- Initially there is subacute pain for a period of 8-12 hrs. In this period there is no increase in the pulse rate or respirations.
2- Intestinal sounds are increased in frequency & intensity.
3- Rectal palpation may reveal the enlargement of the impacted ileum in the upper right flank at the base of the caecum. Care should be taken to differentiate this from impaction of small colon.
4- At the end of this phase pain increases in severity, there is depression, patchy sweating and coldness of the extremities. Animal sits on haunches and rolls and straggles violently. The abdominal pain becomes severe and continuous. Pulse rate rises (80-120/m) and is weak. Respirations are increased (over 401m). No abdominal sounds. Aspiration of fluids by a nasal tube may amount several gallons of sanguineous fluid.
   • - Rectal exam. in this phase reveals that the large intestine is small and contracted and the small intestine is so tightly distended with gas and fluids.
   • - Death usually occurs 36-48 hrs. after the onset of he disease.
   • - Rupture of the intestine may occur.

**Diagnosis:**
1- In early stages the disease may be confused with spasmodic colic or enteritis but the palpation of the impacted ileum may differentiate the two. The continuation of the illness suggests the ileoaelcal valve impaction.
2- In the second phase it resembles acute tympany except that the small intestine is obstructed.
3- In acute intestinal obstruction shock is sever.
4- The characteristic feature of ileoaelcal valve impaction are the gross accumulation of fluid and the relatively long course.