**Treatment:**
1- Removal of the fluids from the stomach and replacement of the lost fluids.
2- Sedatives.
3- Removal of the obstruction surgically if the animal survives but this is without hope.
4- A large doses of mineral oil (1/2 - 1 gallon).

**D) Intestinal tympany:**
Intestinal tympany causes distension of the abdomen and severe abdominal pain and is sometimes accompanied by the passage of much flatus.

**Etiology:**
1- Occur secondary to obstruction of the intestinal lumen.
2- Tympany of small intestine: are caused by:
   a) Acute intestinal obstruction.
   b) Constricting adhesions from perforated gastric ulcer or ilio-caecal valve impaction.
3- Tympany of large intestine:
   Primary causes: ingestion of large quantities of highly fermentable green pasture, spoiled or mouldy food or atony of the bowl.
   Secondary causes: are caused by acute intestinal obstruction or stenosis (Veminous aneurysm-fibrous tissue formation).

**Clinical findings:**
1- Abdominal distension and the distended loops may be visible through the abdominal wall in thin animals.
2- Pain is acute & continuous, pawing violently and the horse lies down very carefully.
3- Peristaltic sounds are reduced but fluids may be heard moving in gas-filled loops producing tinkling and metallic sound.
4- rectal exam reveals gas-filled loops of intestine.
5- In primary tympanitis much flatus is passed and the anus may be in a state of continuous dilatation.
**Diagnosis:**
1- Primary tympany is always difficult to differentiate from secondary tympany. But the presence of faeces, flatus and the history of engorgement on lush pasture may differentiate the two.
2- Primary tympany involves nearly the whole of the tract.
3- Typany due to obstruction terminate fatally in a short time.

**Treatment:**
1- Differentiate between primary and secondary tympanitis.
2- Sedatives: i/v injection of Novalgin 50%.
3- In primary tympany the administration of carminatives and intestinal stimulants are useful. It is given in the form of an oral mixture of ammonium carbonate & charcoal in equal quantities dissolved in water.
4- External abdominal massage, also through the rectum may stimulate the peristalsis. Soft soap enema has the same action.
5- Intestinal Antiferment suppresses the formation of gas as follow:
   Carbolic acid (Sol 2%) 200.00
   or formaline solution (10-20 ml) dissolved in 2 liters water to be given orally.
   OR
   Mineral oil (1/2 - 1 gallon) containing oil of turpentine (30- 60 ml), formalin (30 ml) or chloroform (30 ml).
6- In severe cases trocarization with a long small-caliber, intestinal trocar and cannula may be necessary. This can be performed per rectum or through the upper right or left flank depending on the site of maximum distension.
7- In secondary tympany, permanent relief can be obtained only by correction of the obstruction.

e) Impaction of the large intestine:
Impaction of the large intestine causes commonly in horses moderate abdominal pain, constipation, general depression and anorexia.
Etiology:
1- Debility is a predisposing cause and the diminished intestinal muscle tone is incapable to move the ingesta.
2- Feeding on low-grade indigestible roughage, particularly old hay or sorghum.
3- Bad teeth (improper mastication).
4- Over-fed, fat horses are more susceptible.
5- Interference with the blood supply to the intestine as in Verminus mesenteric arthritis may interfere with the muscle tone.
6- Presence of fiber-walls & enterolith.

Clinical findings:
1- Moderate abdominal pain is the typical sign in affected horses, and is continuous for 3-4 days and sometimes for 2 weeks, and in the latter case it is accompanied with caecal impaction. Fits of pain occur at intervals of up to 1/2 hours.
2- Anorexia & constipation. Feces are passed in small amounts, hard and covered with thick sticky mucous.
3- Intestinal sounds are reduced or absent and much decreased in intensity.
4- Rectal palpation reveals the following:
   a) Impaction of the pelvic flexure of the large colon is the commonest site. Solid-loops of the intestine could be palpated at the pelvic rim or to the right of the midline.
   b) Impaction of the caecum can be palpated in the right flank extending from high up and passing downwards and anteriorly.
   c) Impaction of the small colon may be felt dorsally to the right of the midline.
5- Pulse rate is moderately increased (50/min).
6- Animal does not eat but may drink small quantities of water.
7- When death occurs this may be due to rupture of the intestine or from exhaustion after a large course in debilitated horses.
8- In foals retention of the meconium causes continuous straining with elevation of the
tail and walking backwards. Hard fecal balls can be palpated with the finger in the rectum.

**Diagnosis:**

1- Other causes of constipation as in peritonitis and dehydration should be considered. In peritonitis there may be toxemia as a complication.
2- In other forms of pain as gastric dilatation, acute intestinal obstruction and spasmodic colic the pains is more severe, and have much shorter course.
3- Palpation of the cranial mesenteric artery must be done to differentiate the disease from Verminus mesenteric arteritis.

**Treatment:**

1- Administration of 1/2 - 1 gallon of mineral oil with 15-30 gram chloral hydrate in 1 - 1.5 liters of water by nasal tube. Repeat the treatment if the impaction is not relieved in 12 hrs. In this case you can inject s/c, in addition parasympathetic stimulant. Do not use parasympathetic stimulant without prior administration of mineral oil otherwise rupture of intestine occurs. Linseed oil can be also used.
2- Detergents combined with mineral oil could also be used.
3- Violent purgatives as anthracene purgatives are also used but be careful about the dose to avoid super purgation.
4- Enema with soft soap and worm water may be used but have doubtful effect.
5- Retention of the meconium in foals could be treated with the injection of mineral oil (90 ml) or glycerin (30 ml) into the rectum by the use of a rubber tube. The enemas are repeated until soft feces appear and the animal is comfortable. Oral doses of 120-250 ml of mineral oil are also indicated; small doses of parasympathetic stimulants could also be injected (1/8 or 1/16 of the adult dose).
6- Pain is relieved with parentral administration of an ataractic drugs.
4 - Equine Colic

Those diseases of the horse, which cause abdominal pain, are generally called equine Colic. A summary is presented in the following table.

The most prominent feature is distention.

This may be (1) static (physical colic) when there is an accumulation of ingesta-gas or fluid.

(2) Or transient (Functional) when local, periodic distention occurs as the result of spasm with increased peristalsis of the intestinal segments.

(3) Stretching of the peritoneum.

Clinical signs:

1- Pain is manifested by looking at the flank, rolling, lying down on the back (careful in lying down and getting up). The horse often sitting, like a dog. Geldings protrude the penis without urination.

2- Restlessness manifested by pawing & kicking at the belly or by sitting, getting up and lying down frequently.

3- Pain is generally subacute or acute. It is intermittent in early stages and the severity is correlated with the severity of the illness. In most severe cases the pain is almost continuous.

4- Obvious signs of shock, profuse sweating with respiratory disturbances.

**Diagnosis:** Diseases which may be conflicted with colic are:

**Laminitis:** There is more immobility than restlessness. The feat are held together. No abdominal pain and the pain are obviously in the feet.

**Hepatitis:** Pain is dull and almost continuous the horse does not roll or keep abnormal posture and jaundice is common.

**Tetany:** Extremes tetany-third eyelid prolapsed and hypersensitivity the titanic convulsions and sweating may lead to incorrect diagnosis of colic. A horse with colic can always rise but a horse with tetanus that can rise has unmistakable clinical signs.

**Urolithiasis:** Pain is with attempts to urinate and the passage of few drops of blood
urine with the appearance of a distended urinary bladder on rectal palpation.

**Peritonitis:** Pain is evident on percussion or deep palpation and fever is present.

**Notice:**

(1) Pulse rate less than 80/mm is favorable - a rate over 100/indicates danger.

(2) Too low temp. Indicates the development of shock, while fever suggests other cause of the signs observed.

(3) Absence of peristalsis suggests paralytic ileus.

(4) Gas-filled loops of intestine is an indication of flatulent colic and if accompanied by much fluid indicates intestinal obstruction.

(5) Long, unbroken columns of feces is an indication of impaction.

(6) A passage of stomach tube may be necessary to detect if there is suspect of inflammatory exudate in peritoneal cavity:

The more common colic are:

**Acute:** Gastric dilation, ileoacaecal valve impaction, intestinal emergency colic (Intussuception-volvulus-strangulation) diaphragmatic hernia, enterolith, enteritis flatulent colic and hemorrhage.

**Subacute:** Impaction of colon, caecum, spasmodic colic and retained meconium.

**Chronic or recurrent:** Mesenteric Verminus arteritis constriction of gut by adhesion and peritonitis.

The most common recurrent colic is simple impaction of caecum and colon due to poor condition of molar teeth, too frequent heavy feed and debilitated old age animals.

**Treatment:** Generally it depends on the nature of every case and the situation of the lesion. The following principals are recommended:

1) Analgesia: To prevent self injury and without masking the use of pethidine (Meperidine) or chloral hydrate are satisfactory drugs. Synthetic analgesic is an excellent pain relief in horses (2 mg/kg b.w.).

2) Evacuation of the bowel in case of impaction with fecal material is necessary by:

a- Mineral oil
b- Anthroquinone purgatives (much smaller in size-but variable in efficiency).
c- parasympathomimetic drugs as arecolin,
  • phystostigmine and other could be used alone or with lubricant. In very impacted colon
  the use of these stimulants may cause unnecessary pain.
d- Antispasmodics as atropine are to be the avoided unless there is a functional motility.,

3) Treat the fluid loss.
5) Surgical m some cases of physical colic

**Etiological classification of equine colic**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Primary etiological agent</th>
<th>Pathogenesis</th>
<th>Causes of distension</th>
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<td>b) Physical</td>
<td>Low grade roughage</td>
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<td>Parasitism (strongylosis)</td>
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Colic | Bacteria (Salmonella) | Peristalsis.
---|---|---
% | Viral (Equine viral arteritis) | 
% | Physical (sand colic) Chemical poison Excitement Thunderstorm Cold drinks or chilling Reflex from other viscera Grass sickness Venomous mesenteric arteritis | Imbalance of autonomic nervous system (Spasmodic colic)

**Diseases of the stomachs of the ruminants**

**Introduction**: Ruminants utilize mainly fibrous substances. These fiber are delayed and subjected to extensive microbial fermentation in the rumen before their passage to the true stomach and intestinal tract.

- There is always constant supply of food and water in the rumen temperature is held relatively at 39°C and pH at 6.8.
- Importance of the rumen lies in that 70-85% of the digestive dry matter is utilized in the rumen. 70% of the total cellulose digestion occurs in the rumen and the rest (30%) occurs in the large intestine.
- As far as mammals do not secret cellulose enzymes in their digestive juices, therefore the degradation of cellulose is achieved by the cellulolytic activities of the microbial population.
- Rumen microbes play an important role in protein synthesis, vitamin B complex formation, fat alteration and soluble carbohydrate digestion.
- The retention of the ingests in the rumen (1-3 days) allows sufficient time for bacterial disintegration of food stuffs before passage to abomasums and intestine.
- Reticuloruminal contractions promote intermixing of he bacteria, ingests and ruminal fluids, thus promoting bacterial fermentation.
- The principal products of ruminal digestion are the volatile fatty acids.
- The end products of ruminal carbohydrate fermentation are acetic acid, Propionic and
butyric acids. These usually perform 95% of the contents of the ruminal ingests. These fatty acids are absorbed by the reticulorumininal epithelium. The fate of these fatty acids after absorption are as follow:

1) Propionic acid is converted into glucose and then to lactose.
2) Acetic acid may be assimilated into body fat and oxidized to provide energy or converted to glucose and stored in the liver as glycogen.

- Proteins and non-protein nitrogenous compounds (urea and amino acids) are hydrolyzed by the ruminal bacteria into free ammonia in the rumen which is synthesized into bacterial cell protein by multiplication of bacterial cells. These bacterial cells pass into the posterior digestive tract where the cellular protein is digested and absorbed by the host.
- Young suckling ruminants need; most of vitamin B complex to be added as far as rufen is largely nonfunctioning.

**Indigestion**

**Definition:** It is impairment of the process of digestion as a result of the changes in the two main function of the stomach which are:

1) Microbial digestion and fermentation.
2) Physical maceration by contraction of the stomach.

N.B.: Contractile power and movements of the walls of the fore stomach depends on afferent and efferent nerves ascending out by: the vagus nerve. For this there is no intrinsic contractile power in the muscles of the fore stomach and any injury in these afferent or efferent nerves will lead also to indigestion (Vagus indigestion). The term indigestion is applied to all types of digestive disorders, but usually is reserved for rumen disturbances. It could be classified according to its types into. . S

**Primary indigestion:** These originated in the pre-stomachs themselves and the defect may be:

A) In the wall of the rumen or
B) in the contents

**Secondary indigestion:**

1) Is a sequelle to diseases in other organs as bacterial or parasitic disease such as anthrax, malignant head catarrh, fascioliasis, coccidiosis ... etc.
2) Indigestion may be associated with acute mastitisendometritis - abomasal displacement. Post-parturient paresis.
3) Accompanies poisoning with nitrate or hydrocyanic acid peritonitis, pericarditis ...etc.

**Pathogenesis:**

1) If the ruminal contents are frothy it will be impossible for the cardia to be cleared and eructation to occur and consequently tympany occurs.
2) The atony of the stomach by dietary causes produced by putrefaction of proteins that forms toxic amides and amines, which include histamine. These causes atone and in advanced cases cause laminitis.
3) Changes in pH depress the ruminal motility. This occurs in cases of impaction when fermentation of carbohydrates by Gram + ve cocci leads to this condition (acidity) due to lactic acid formation.
4) The fall in milk yield may be attributed to the sharp falls in volatile fatty acids production in the atonic stomach.
5) Death may occur in acidosis due to histamine production & dehydration. Injuries to the vagus nerve lead to disturbance in motility (Vagus indigestion).

**Simple indigestion**

**Aetiology:**

1) **Dietary abnormalities:**
   - Indigestible roughage particularly which have low protein content.
   - Mouldy food.
   - Over feeding with grains (change of pH).
   - Eating the placenta.
   - Rumen atony.
2- Water limitation (deficiency of drinking water).
3- Administration of sulfa drugs or antibiotics because of their effect on the rumenal flora.
4- Toxic amides and amines produced during protein putrefaction may include histamine-which cause Ruminal atony.
5- High protein diet including feeding large amounts of diet that causes alkalinity which depress ruminal motility.

**Predisposing causes:**
- Lesions of the peritoneum (traumatic peritonitis).
- Advanced pregnancy.
- Fatigue, change of food and lack of water.

**Pathogenesis:**
- Changes of the pH (acid or alkali) affect the motility of the rumen and cause death of the microflora.
- Putrefaction of protein leads to liberation of histamine which causes atony of the rumen.

**Clinical findings:**
1) Reduction of the appetite is the first sign.
2) Severe drop in milk yield.
3) Mild depression and dullness.
4) Suppression of rumination.
5) Ruminal movements are depressed in quality & rate or may be absent.
6) There is constipation with scanty feces, although diarrhea may be present.
7) There may be moderate tympany.
8) There is no systemic reaction (pulse respiration & temp. are not altered).
9) No pain on percussion.
Clinical pathology:
1) Differentiate between indigestion & Acetonaemia.
2) Examination of ruminal fluids.

Diagnosis: Differentiate between the followings:

Acetonaemia: Always 2 months after calving with the presence of ketone bodies in the urine.

Traumatic reticuloperitonitis:
- Characterized by sharp fall in milk yield.
- Mild elevation of temperature.
- Pain on percussion.

Abomasal displacement:
- Usually after parturition: accompanied by Ketonuria.
- Pain on percussion.

Vagus indigestion: - Which is not accompanied by fever and there are hypo-or hypermotility and there may be abdominal distention.

Acute ruminal impaction:
- Signs of dehydration.
- Nervous derangement.
- History of engorgement with grain ingestion.

Treatment: Because of the variety of the causes, the chief object of treatment is:
- To establish contraction of the rumen.
- To evacuate the digestive tract.

I) Milk of magnesium: (antacid + antiferment + milk laxative).

R Milk of Mg. prepared as follows:
Mg. Hydroxide. 250 g dissolved in 5.00 c.c. water

II) Parasympathetic stimulants:
Used as ruminal tonic e.g. Eserin or neostigmine
Dose 5 mg/kg.
or Carbacol or coletyl or pilocarpine (2-4 ml) s/c

III) This is to increase the motility of the rumen. Alkaline therapy in cases of acidosis:
Mg Oxide 250-400 g

or Mg. carbonate 200-300 g

or calcium carbonate 200-300 g

V) Regulate the pH in cases of alkalinity:
- Lactic acid 50-70 ml in 8-10 liters water
Vinegar 200 ml/K B.W. diluted in 300 ml of water
V) Ruminal juice transplantation.
One litre of ruminal juice (fresh) is mixed with water or saline.
VI) Antihistaminic.
- Benadyl 0.2 -1 mg/K b.w.
VII) Antiferment as oil of turpentine (30 gm orally or creoline).

**Acute impaction of the rumen**

**Rumen overload-rumen acidosis**

**Definition:** Ingestion of large amounts of highly fermentable carbohydrate feeds causes:
1) Acute illness due to the excess production of lactic acid.
2) Clinically the disease is manifested by severe toxemia dehydration, recumbency, complete ruminal stasis and high mortality rate.

**Causes:**
1) Ingestion of large quantities of highly fermentable carbohydrate whole or ground grains.
2) Sudden change of the ration.
3) Excessive feeding especially during dry season where high percent of fermentable carbohydrates will be eaten.

**Pathogenesis:** Rapid fermentation of the carbohydrate by the Gram-positive cocci usually Strept. Bovis causes the formation of large quantities of lactic acid, which increase the osmotic pressure into the rumen leading to dehydration and haemoconcentration accompanied by anuria. Rumen motility decreases as pH falls, Rumen stasis occurs Rumen microflora are destroyed and replaced by lactobacilli and streptococci bacteria.

- There is severe depression of volatile fatty acids production due to the destruction of the ruminal bacteria and protozoa.
- Histamine production occurs which leads to laminitis.
- Bacterial infection leads to the development of metastatic abscesses in the liver with subsequent formation of hepatitis which may be symptomless.
- Necrosis and gangrene may affect the forestomachs and may lead to acute diffuse peritonitis and toxemia ending by death.

**Clinical findings:**
1- The sings varies with the nature and the amount of the feed, being faster with ground food than the whole grains.
2- The first sign may be abdominal pain with kicking at the belly.
3- There is profound depression and desclination to move.
4- Respiration is increased in rate and may be accompanied by grunting.
5- Anorexia is complete and affected animals usually do not drink much water.
6- Distention of the abdomen and slight Ruminal tympany (may not be marked).
7- Palpation on the left paralumbar fossa reveals a firm and doughy material.
8- Nose is dry and mucopurulent exudate accumulates in the nostrils.
9- Grinding on the teeth.
10- Diarrhea with the passage of soft or watery faeces is seen.
11- In severe cases there may be profuse diarrhoea accompanied by the passage of much mucus and some blood.
12- Increase in pulse rate up to 120-140/min and the pulse is weak.
13- Temp is below normal.
14- Ruminal movements are completely absent but gurgling sounds of gas may be heard (accumulation of fluids).
15- Animals gragger and appear to be blind; and the eyes have not eye preservation reflex.
16- Laminitis and recumbency may follow after 48 hrs but in may be an early sign when animals lie, they are often with the head turned into the flank (resembles parturient paresis).
17- Death occurs in 24-72 hrs in most fatal cases. Return of normal ruminal movements, pulse rate, Passage of large amounts of soft feces may indicate a phase toward recovery.
18- Some animals may appear to have a temporary improvement but become severely ill again on the 3rd and 4th day.
Death may follow because of diffuse peritonitis.

N.B.: The above signs are seen in severe cases-but as mentioned above it varies according to the type of feed and its amounts.
Clinical pathology:
1- Measurement of Haematocrite (PCV) may rise up to 50 or 60 (Normal 30-31%).
2- Blood pressure falls.
3- Urine pH falls, Oliguria may follows with anuria.
4- Blood glucose and phosphates levels rise.
5- Blood glucose and phosphate levels are elevated.
6- pH of the ruminal fluid falls: 4.5 - 5 (Moderate) less than 4.5 (severe).
7- Motility of inifisoria (microflora) disappear.
8- Gram positive microflora appears.
9- A degree of proteinuria is noticed.

Diagnosis:
- History of engorgement with grains when this is not available the disease may resemble parturient paresis and can be differentiated as follow:
  - Parturient paresis: are seen in recently calved or in late pregnancy or in early lactation. There is no peripheral circulatory collapse + heart rate is not greatly increased and feces are hard and dry.
Differential diagnosis from the following condition:
- Acute hepatic insufficiency: accompanied by blindness and staggering gait but there is usually jaundice and heart rate is approximately normal.
- Arsenic poisoning: Enteritis is more severe and death is quicker.
- Lead poisoning: Is manifested by severe nervous signs. In both lead and arsenic poisoning, faecal analysis should be adopted.
- Enterotoxaemia: Caused by Cl. perfringens type D that may occur in well fed calves and lambs and could be diagnosed on the basis of the toxicity of bowel filtrates.
- Early stages of traumatic reticuloperitonitis: Here there is severe pain on abdominal percussion and changes in the white cell count.

Treatment:
1) Mild cases could be treated as in simple indigestion.
2) Prevent access to further eating of grains.
3) Exercise vigorously for half an hour 3 times daily.
4) Allow water but in limited quantities at time.
5) Inject antihistaminics.
6) Give oral antibiotics.
7) Give alkaline purgatives.
There are the broad lines of treatment.

**Prescriptions:**

- Magnesium hydroxide or Mag. carbonate to be given 250-500 grams as an initial dose followed by 120 gm at 12 hrs intervals.
- Oral penicillin: from .0.5 - 1.0 million for sheep orally. These restrict the growth of bacteria, which produce lactic acid. In cattle higher doses are given up to 10 million I.U.
- Oral tetracyclines: 5 - 10 grams and repeat it at 12 hrs intervals.
- Repair the dehydration by injection of large quantities of isotonic solutions 4 - 10 litre.
- Repair the acidosis by the (i/v) injection of 500 ml 2.5% solution of sod bicarbonate. -- Inject slowly and watch the animal if you repeat the dose otherwise alkalosis may develop, oral doses may be safer (120 gm twice a day).
- Injection of thiamine 2 - 4 grams is excellent in such cases, or bakers yeast 500 gm daily given per os.
- Ca. borogluconate is said also to help recumbent animals.
- Emergency rumenotomy should be performed in serious cases and the rumen should be washed out. The contents should be replaced by mixture of hay + some water + rumen transplants or 1-2 litre of fresh ruminal fluid gained from healthy individuals.

**Prevention:**

1- Gradual changes of the ration when start feeding on grains until adaptation of the new diet over a period of 6 days.
2- The addition of alkalinizing agents to the ration of beef cattle fed heavily on grain to the level of 7% with a mixture of ground limestone and sod. Bicarbonate.

**3- Ruminal tympany**

**(Bloat)**

Def: Over- distension of the rumen and reticulum with gases, either free or mixed with the ingesta. The incidence of the disease has increased with the heavy application of fertilizers.

**Aetiology:**

**Primary ruminal tympany:**

a) Dietary factors:
1- Grassing on very succulent pasture, particularly young rapidly growing legumes in the pre-bloom stage (the important cause).

2- May also occurs when cattle grassing on cereal crops; cabbages, leguminous vegetable crops (peas-beans) young grass pasture with a high protein content.

b) Animal factors:

1- Cattle vary in their susceptibility to tympany perhaps due to hereditary factors.

2- Foaming of the ruminal contents:

Foaming of the Ruminal contents produced by the high viscosity or surface tension. This is caused by:

- The pH of the rumen is an important factor to make the foam stable. Stability occurs at a pH of about 6.0 the composition of the rumen diet and the activity of the microflora influence this factor (pH).

- The rate of flow and composition of the saliva has an effect on the tendency for a tympany to occur as saliva act as a buffer on the pH of the rumen. Salivary mucin prevents the formation of foam but the mucinolytic bacteria in the rumen may destroy the mucin and permit bloating. The amount of saliva may dilute the ingesta. The low amount of saliva may increase the susceptibility of cows to develop tympany.

- An increase in slime producing bacteria may be the cause of foaming (frothiness) as these microorganisms produce polysaccharides, which encourage froth formation. Moreover, this bacteria digest plant lipids which have an anti-foaming effect.

Secondary ruminal tympany:

1) Physical obstruction: This prevents eructation and is caused by foreign body stenosis-pressure from enlargements outside the esophagus or obstruction inside the esophagus or at the cardia.

2) Vagus indigestion and diaphragmatic hernia: These interfere with oesophageal function and cause chronic tympany. The condition may occur in tetanus in young lambs due to spasms of the oesophageal musculature.
3) Interference with the nerve pathways:

These are responsible for maintenance of eructation reflex. The receptor organs in this reflex are capable to differentiate between gas and foam liquid lesions of the vagus nerve may interrupt the reflex action, which is important to remove gas from the rumen.

4) Atony in normal tone of the musculature of the rumen and reticulum. Sudden change in pH either to acidity or alkalinity may cause such atony.

5) Persistent or enlarged thymus may cause chronic tympany in calves up to 6 months.

**Pathogenesis:**

1) Free gas ruminal tympany is due to oesophageal obstruction or atony.

2) Frothiness of the ruminal contents causes the obstruction of the cardia and this inhibits the eructation reflex consequently cranial sphincter of the esophagus fails to open. Rumen movements are stimulated by distension causing hypermotility which increase frothiness and terminally there is loss of muscle tone and motility.

**Clinical findings:**

**Primary pasture bloat:** Which appears clearly in the upper left flank in the form of:

1) Obvious distension of the rumen, which occurs suddenly 15 minutes.

2) Discomfort and the animal may get up and lie down, frequently kicks at the belly and even roll.

3) Dyspnoea is marked which characterized by mouth breathing, protrusion of the tongue, salivation and extension of the head.

4) Respiratory rate is increased up to 60 min.

5) Projectile vomiting may occur and the faeces may be expelled in a stream.
6) Ruminal movements are at first increased but later on the sounds are reduced in volume. When the distension is severe, the movements are decreased or completely absent.

7) Percussion produces tympanic sound allover the rumen.

8) In acute stages rumination and eructation disappear (may increase before clinical signs).

9) Pulse rate is increased up to 100-120.

10) Death occurs due to:

a- The absorption of the toxic gases (H$_2$S, toxic amines, histamine).

b- Depression of the cardiovascular, respiratory systems. Death can occur within 3-6 hours from the onset of the clinical signs.

Secondary bloat:

1) There is excess of free gases on the top of the ruminal contents.

2) As in pasture bloat there is usually an increase in rate and force of ruminal movements in the early stages followed by atony.

3) Passage of the stomach tube or trocarization induce the expulsion of large quantities of gas and the retention subsides. (Also by the stomach tube you can detect if there is oesophageal obstruction).

4) Pulse is increased; systolic murmur is often audible which may be due to displacement of the diaphragm and distortion of the base of the heart.

**Diagnosis:** Simple in primary bloat but the difficulty in secondary bloat especially when the bloat is chronic. Passage of the stomach tube will detect the oesophageal obstruction or stenosis. Vagus indigestion and diaphragmatic hernia may have a previous history of traumatic reticuloperitonitis and partial anorexia.
- Tetanus is manifested by limb rigidity, prolepses of the third eyelid and hyperethesis.
- Carcinoma and papilloma of the oesophageal groove and reticulum are difficult to be diagnosed without rumenotomy.

**Treatment:**

1) Treatment of the secondary tympany depends upon the removal of the cause.
2) In any form adopt the first aid to save the animal.
   a- Tying a stick in the mouth.
   b- Let the animal to stand with the front feet raised.
   c- Drench with non-toxic vegetable oil or mineral oil
   d- Smear the back of the tongues with wood tar.
3) Passage of the stomach tube to relief gases, and in emergency cases trocarization must be performed.

**Primary bloat:** The aim in this type is to reduce the stability of the foam by:

1) Oral administration of any vegetable or mineral oil (300-800 ml) for large animals and 60-100 ml for sheep, oils could be mixed with a detergent. A stomach tube to avoid aspiration and to remove excess free gas could administer this.
2) Oral administration of poloxalene (25-50 gm) to reduce stability of the foam.
3) In severe cases trocarization should be adapted and oils can be introduced through the cannula by a syringe with a long nozzle.
4) Turpentine oil (30-60 ml) reduces the viscosity of the foam.
5) Purgatives may be administered with the oil to facilitate evacuation of the tympany-produced ingesta.
6) Anti-histaminics may be recommended but their use in frothy bloat may have a dangerous effect.
7) Severe cases may need rumenotomy.

8) Secadine (Anti bloat drug).

4- Vagus Indigestion

It is usually seen in cattle and buffaloes and is characterized by:

- Delayed passage of food in the intestine due to paralysis of stomachs.
- Distension.
- Anorexia.
- Passage of soft pasty faeces in small quantities. Caused by lesions, which involve the vagus nerve supply to the forestomachs and abomasums.

Causes:

1) The commonest cause is traumatic reticulo-peritonitis.

2) Scar tissue lesions affecting the ventral branch of the vagus nerve, which ramifies over the anterior wall of the reticulum.

3) Lesions, which prevent oesophageal, groove reflexes or which cause adhesions between rumen and abomasums.

4) Actinobacillosis of the rumen and reticulum may act also as a cause.

5) Involvement of the vagus nerve as a result of enlargement of L.N. due to tuberculosis or lymphomatosis. Similar disturbances may occur as a result of diaphragmatic hernia.

6) In sheep’s peritonitis caused by sarcosporidia and cysticercus tennicollis.

Pathogenesis:
1) Varying syndromes depending upon the affected branches of the nerve and to the varying degrees of immobilization caused by adhesions of reticulum to diaphragm this leads to: Development of achalasia of the reticulo- omasal and pyloric sphincters which result in accumulation of food material in the rumen.

2) Failure of the oesophageal groove to permit the passage of food into the rumen may accompany pyloric achalasia.

Clinical findings: 3 major syndromes.

1) Distension of the rumen with hypermotility:

- This is not related to pregnancy or parturition.
- Moderate to severe ruminal tympany although the animal did not eat with strong continuous movement of the rumen.
- The faeces are normal or pasty but scanty.
- No fever.
- Heart rate is usually slower than normal and sounds are accompanied by systolic murmurs, which wane with respiration being loudest at the peak of inspiration. This disappears when tympany is relieved.

II - Ruminal distension the atony:

- This occurs in late pregnancy and may persist after calving. The cow is clinically normal but does not eat well.
- Cow passes only small amount of faeces - soft and pasty.
- Tympany does not respond to treatment.
- Ruminal movements are seriously reduced or absent.
- No fever, no pain on percussion of the abdomen.
- Rectal palpation may reveal a distended rumen to the •extent that it blocks the pelvic inlet.
- Animal losses weight rapidly and become very weak and eventually recumbent. At this stage heart rate is increased and the animal dies from inanition.

**III Pyloric obstruction:**

- Most cases of this type occur in late stage of pregnancy.
- Anorexia and reduced volume of pasty faeces.
- No obvious abdominal distension and no systemic reaction until the late stages when the pulse rate rises rapidly.
- There is a distension of the abomasums and you can palpate it on the floor of the abdomen on rectal examination (but this is impossible when the cow is pregnant). The abomasums is firm and not distended with gas or fluid.
- Rumen movements are completely absent and death occurs slowly due to inanition.

**Remarks:** Combination of these types may occur especially distension of the rumen combined with abomasal obstruction.

**P.M. Findings:**

1) Firm distended abomasums half filled with digested fibers and the rest is filled as that of the rumen contents.
2) Ulcers in mucosa.
3) Intestine may be empty.
4) Lesions (adhesions) in the reticulum may suggest a history of traumatic reticulo-peritonitis.

**Clinical pathology:** Moderate neutrophilia + neutrophilic shift to the left relative monocytosis which reflects chronicity.

Diagnosis: (Differential diagnosis)

Indigestion due to:

1) Left abomasal displacement:
   a- There is audible abomasal sounds.
   b- Left lower flank of the abdomen is sunken rather than distension.
   c- Ruminal movements are not absent or exaggerated but the sounds are modified.
2) Chronic reticulo-peritonitis - impossible to differentiate this from vagus indigestion unless without rumen atony.

3) Subacute abomasal torsion (Right displacement): here the distended abomasums can be palpated filling the right half of the abdominal cavity.

4) Lipoma or fat necrosis, causing partial obstruction of the intestine could be palpated by rectal palpation.

**Treatment:**
- Rumenotomy and empty of rumen may be followed by slow recovery over a period of 7-10 days when there is hypermotility.
- Abomasotomy is not recommended as the motility of the abomasums does not return to normal.
- Conservative treatment has no good effect.
- Slaughtering is recommended.

**Traumatic reticulo-peritonitis and allied syndromes**

Perforation of the wall of the reticulum by a sharp pointed foreign body produces:
- Initially and acute local peritonitis; this may spread to cause acute diffuse peritonitis.
- This may also causes:
  - Vagus indigestion.
  - Diaphragmatic hernia.
  - Other organs may be involved.

Resulting in pericarditis, cardiac tamponad, pneumonia, pleurisy, hepatic or diaphragmatic abscesses.

**Traumatic reticulo-peritonitis**

**Definition:** Perforation of the wall of the reticulum by sharp pointed foreign body produces initially acute local peritonitis, which is characterized by: Sudden anorexia, drop in milk yield, fever, ruminal stasis and local pain in the abdomen.
Causes:
1) Sharp pointed objects: nails, wires ... etc; are the most common causes of injury.
N.B.: Sheep and camel could be also affected.

Pathogenesis:
- Lack of oral discrimination in cattle lead to ingestion of foreign bodies.
- These may be lodged in the upper esophagus and cause obstruction - or it may be in the oesophageal groove and causes vomiting.
- The sharp pointed foreign body may pass to the reticulum where the vigorous contraction and with the fixation of the nail into the wall it will penetrate it. Usually most perforations occur in the lower part of the anterior wall, other sites may be toward the spleen or liver.
- Some foreign bodies may remain fixed without penetration and without serious results and will gradually be voided away.
- The illustrated diagram explains the sequelle of perforation.

Clinical findings:
1) Onset is sudden (with complete anorexia) and a sharp fall in milk yeild be about a third of the previous yield.
2) Subacute abdominal pain in all cases.
3) Animal is reluctant to move and if it moves it moves slowly.
4) Walking downhill is often accompanied by grunting. Most animals prefer standing for long period and lie down with great care (habitual recumbency is characteristic in some animals).
5) Arching of the back “tucked up abdomen”.
6) Defecation and urination cause pain.
7) In rare cases an attack of acute abdominal pain with kicking at the belly, stretching and rolling is the earliest sign. In others there is recumbency and inability to stand.
8) Rise of temp (39.5 - 40°C), pulse rate rises to about 80/mm, resp. rate 30/mm and respiration is shallow. If pleura is affected audible expiratory grunt is heared.
9) Rumination is stopped and ruminal movements are absent or severely depressed (1 - 2/5 min).

10) Mild tympany with a moderate distension of the left flank.

**Sequelle of traumatic perforation of the Reticular wall by foreign body**

**(Common sequelle & Uncommon sequelle)**

<table>
<thead>
<tr>
<th>Common sequelae</th>
<th>Uncommon sequelae</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perforation</td>
<td>1- Rupture of left gastroepiploic artery</td>
</tr>
<tr>
<td>local Acute peritonitis</td>
<td>(Death due to Internal Haemorrhage)</td>
</tr>
<tr>
<td></td>
<td>2- Splenic abscess.</td>
</tr>
<tr>
<td></td>
<td>3- hepatic abscess.</td>
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<tr>
<td></td>
<td>4- Diaphragmatic abscess.</td>
</tr>
<tr>
<td></td>
<td>5- Pleurisy and pneumonia.</td>
</tr>
<tr>
<td></td>
<td>6- Mediastinal abscess.</td>
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<td></td>
<td>7- Endocarditis.</td>
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<td></td>
<td>8- Nephritis.</td>
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<td></td>
<td>9- Arthritis</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Recovery</th>
<th>Chronic Acute local peritonitis</th>
<th>Acute diffuse Pericarditis</th>
<th>Acute Peritonitis</th>
<th>Rupture of coronary artery or ventricular wall causing cardiac tamponade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vagus indigestion</td>
<td>Diaphragmatic hernia</td>
<td>Death due to congestive heart failure</td>
<td>Chronic pericarditi</td>
<td></td>
</tr>
</tbody>
</table>

11) Constipation is always present.

12) Pain tests: pain could be induced by:

a- Vigorous palpation of the abdominal wall just behind the xiphoid process of the sternum. The closed fist or the knee can exert pressure also.

b- Pinching the withers to cause depression of the back.

c- Sharp elevation of animal under the abdomen. A positive response to any of these tests is a grunt of pain audible by the ear, but you detect it more clearly by auscultation of the trachea. Pain accompanies reticular or ruminal contractions and could be watched during examination as an aid of diagnosis.
13) Stage of acute local peritonitis subsides quickly (24 hrs). In cases of recovery or by the application of conservative treatment signs disappear by the 4th day. The chronic peritonitis persists. The appetite, milk yield do not return completely to normal, pain is evident, but the gain may be slow, grunting may occur during rumination, defecation, and urination. In this stage rumination is depressed and chronic moderate bloat may be present.

**The development of acute diffuse peritonitis:**

In this form the signs are:
- Profound toxemia evident (1-2) days from the onset of local peritonitis.
- The alimentary tract movements cease completely.
- Severe depression and temperature is high or subnormal in fatal cases.
- Pulse rate rises (100-120/min).
- This stage is followed by acute collapse and peripheral circulatory failure with all pain responses having disappeared.
- A terminal stage of recumbency and coma may develop.

N.B: Mine detectors are of low values because about 80% of the dairy cattle give positive results because of the presence of many metallic objects in the rumen or reticulum.

**Clinical pathology:**

1- Total leucocyte counts rises up to 8000-12000/mm3 in the first day and remain 24 hrs, then subsides after the 3rd day.

2- Neutrophils and unsegmented neutrophils count show a similar behavior. Neutrophils (30-32% normal rises to 50-70%).

3- In chronic cases levels do not return to normal for long periods with persistent elevation of monocyte (5-9%).

**Diagnosis:**

- Acute local peritonitis: can be differentiated from indigestion, impaction, Acetonaemia by the presence of:
1) Local pain.
2) Sharp fall in milk yield and appetite.
   - Polynephritis: presence of pus and blood in urine.
   - Displacement of the abomasums: presence of abomasal sounds in the left flank.
   - A hepatic lesion is not necessarily accompanied by ruminal stasis and there is pain over the posterior ribs on the right side.
   - Impaction of rumen: is accompanied by an increase in heart rate, staggering, recumbency, blindness; hypothermia no history of engorgement no signs of Haemoconcentration.

**Acetonaemia:**

- There may be some degree of secondary Acetonaemia in T.R.P. Differentiation may be difficult if peritonitis is of 3-4 days duration. Response to treatment may aid in the diagnosis. The history also is essential in both cases.
- In complication which include involvement of the pericardial sac, liver, or spleen, there is high fever, high pulse and toxemia, all had high total leucocytic count and increased neutrophils percentage.
- Extension from a case of metritis: In metritis there are signs of the primary disease.
- Peritonitis due to perforation of an abomasal ulcer is characterized by evidence of pain on palpation over a much larger area of the abdominal wall and in early stages this is most marked on the right hand side. There is difficulty of course if this disease is associated with diffuse peritonitis.

**Treatment:**

**A) Conservative treatment:**

1- Immobilize the animal by tying it to a place and front feet should be elevated about 35 cm above the floor. This facilitates the formation of adhesions. Removal of the foreign body may occur feeding and watering are carried out on the spot.
2- Feed especially roughage should be reduced.
3- Antibiotic drugs: combination of penicillin and streptomycin for 3 days.

N.B.: The sulphonamides may decrease the activity of rumen bacteria but it has a good local effect on the lesion.

4- Bar magnets 7.5 cm long & 2.5 cm diameter with rounded ends may be used as for prophylaxis against T.R.P. and for treatment. Firm embedded nails may not be returned but loose ones may fall, for prophylaxis it is advised before introducing the magnet to reduce the roughage 24 hrs. Otherwise the magnet goes to the rumen.

5- The use of magnetic retrievers has been newly introduced which could be introduced to pass through the rumen to the reticulum and by the aid of metal detector one can locate the retriever.

**Surgical removal of the foreign body:**

For diagnosis and also as a treatment. The recovery rate varies depending upon the time at which surgery is undertaken.

Failure to improve by surgery is usually due to treat the animal conservatively for 3 days and if marked improvement had not occurred in the last 3 months of pregnancy it needs rumenotomy. Movement of the cow during early stages of the disease is not desirable because of the risk of breaking down the adhesions.

**Control:**

- All chopped feed should be passed over magnets to remove metallic material before being fed to cattle.

- Chronic cases of traumatic reticuloperitonitis are best treated by rumenotomy.

- Acute diffuse peritonitis should be treated by broad spectrum antibiotics + intraperitoneal injection of normal saline (2 or 3 grams of oxytetracyline or tetracycline in 4 litre of saline or electrolyte solution.
Diaphragmatic hernia

- Herniation of a portion of the reticulum through a diaphragmatic rupture caused by weakening of the diaphragm, by lesions of T.R.P. or congenital defects causes chronic Ruminal tympany. There is loss of condition, moderate tympany, grinding of the teeth; feces are pasty and reduced in volume.

- Rumination does not occur and the animal may vomit when a stomach tube is passed. No fever and pulse is slower. A systolic murmur is usually audible on auscultation and the intensity of the heart sound; may suggest displacement of the heart usually anteriorly or to the left.

- More severe syndrome is recorded in cases where viscera other than a portion of the reticulum are herniated.

- Peristaltic sounds may be audible in the thorax and there may be interference with respiration and signs of pain with each reticular contraction. Death from inanition in 3-4 weeks after the onset of bloat.

**Abomasal displacement**

*(Dislocation of the abomasums)*

- In this disease, the abomasum is displaced from its normal position (on the mid abdominal floor slightly to the right side) either to the left or to the right or into an anterior position in the right side.

- In the left displacement the sac of the abomasum comes to lie in a position behind the omasum and passes under the rumen. The great curvature of the abomasum passes under the rumen and is imbedded between the rumen and the left abdominal wall and lies in the left lower flank. This is the most common type.
- In the anterior displacement; the clinical picture is very similar to that of the left dislocation but the abomasum or the major part of it, displaced anteriorly and comes to lie between the reticulum and the diaphragm.

- In the right displacement, the abomasum is displaced to the right and is found lying between the liver and the right abdominal wall. The displacement to the right must be always associated with some degree of torsion of the pylorus.

**Predisposing causes:**

1. Abomasal displacement occurs: near parturition, either a short time before or a few weeks after.

2. Dystockia or parturient paresis: This also predisposes to the condition due to the fact that the pregnant expanding uterus may be distended in these cases under the rumen instead of being deflected to the right as is considered in normal. In such cases the abomasum may be pushed forward and to the left. As parturition takes place, the rumen subsides and returns to its normal position, trapping the abomasum on the left side especially if it is atonic or distended with food.

**Causes may be due to the followings**

1. Long standing cases of chronic indigestion where gases accumulate in the upper part of the abomasum; and the condition progresses worsely without proper treatment, more gases accumulate in the upper part of the abomasum which lastly takes the form and shape of a balloon pulling all the organ upwards and displacing finally the position of the abomasum (specially to the left side).

2. Atony of the abomasum. Heavy feeding with grains in late pregnancy.

3. Laboratory incisions, surgical interference as a method for treatment of chronic indigestion in cattle play also a role.
4- unusual activity including jumping on other cows during estrus may be the cause of such cases.

5- A few cases have been found to be associated with ulceration of the abomasum, producing adhesion either between the left thoracic wall and the abomasum or between the left lateral surface of the rumen and the abomasum. It is thought that in such cases the adhesions may hold the abomasum in the abnormal position after parturition.

**Symptoms:**
1- The disease is characterized by sudden decrease in appetite general symptoms of indigestion, signs of abdominal pain, scanty feces which is black in color and soft in consistency.
2- Dullness and marked loss in weight of the animal.
3- Acetonuria.
4- Auscultation at each intercostals space on an imaginary line from the ventral position of the left paralumbar fossa in a slightly downward and curved direction to the left elbow will reveal the characteristic tending sounds. These tending or splashing sounds which resemble the sounds of church belling are of more fluid nature than ruminal sounds, may be heard frequently or as long as 15 minutes a part, these become more obvious when palpating strongly the same position.

In few cases, the abomasum may be so distended that its gaseous fundic portion can be observed and palpated behind the last rib in the left Para-lumbar fossa. In such conditions, an obvious bulge caused by this distended abomasum may develop in the anterior; part of the lower left Para-lumbar fossa and this may extend up behind the costal arch almost to the top of the fossa.

The swelling is tympanic gives resonant sound on percussion (gasses + fluid).
5- Milk production decreases rapidly and the animal becomes emaciated with great reduction in the abdominal size.
6- Upon rectal palpation, the rumen is left small while the abomasum is palpable to its left or may be felt so high in the right side.

**N.B.:**

All the above mentioned symptoms are defined to the left displacement of the abomasum; while in the anterior and right displacement, the clinical symptoms are similar to those described above except that the normal Ruminal sounds could be heard in the distended abomasum just behind and above the heart an on both sides of the chest.

**Diagnosis and differential diagnosis:**

1- From the symptoms described above and the consideration of the nearness of parturition.

1) History of parturition and auscultation findings.

2) You - have to differentiate between this disease and chronic Acetonaemia, in the latter-chronic uncomplicated Acetonaemia, the field test for the detection of ketone bodies in urine or milk is strongly positive while in displacement of the abomasum it is only mildly positive. The feces in chronic Acetonaemia are scanty and firm, while in displacement scanty also but soft in consistency. In primary Acetonaemia, the rumen may be always palpated in the left Para-lumbar fossa and it will be less active and firmer than normal, while in the displacement. The rumen cannot be palpated in the left paralumbar fossa (if it is left displaced because it has been pushed medially by the displaced and distended abomasum). In addition, displacement of the abomasum does not respond to treatment of Acetonaemia except for 2-5 days after which clinical signs re-occur.

3- Differentiate between this disease and traumatic pericarditis and pyelo-nephritis.
Treatment:

1) In subacute cases laboratory through the right flank with drainage of the abomasum and correction of the torsion has been carried out successfully but in many cases abomasal atony persists.

2) Rolling of the animal on back, vigorously and the rolling is stopped abruptly in the hope that the abomsum will free itself.

3) Supportive treatment with large quantities of isotonic fluids is important to maintain the animal’s fluid and electrolyte balance.

Diseases of the liver

Usually diseases of the liver occur as secondary to diseases arising as part of a generalized disease process or by spread from another organ.

A primary disease of the liver seldom occurs in farm animals except as a result of food poisonings.

Principal of occurrence of hepatic dysfunction

This usually occurs through either diffuse or local hepatic disease. Diffuse diseases of the liver are more commonly accompanied by signs of insufficiency than that of focal diseases, which produce their effect either by toxins found in the lesions or by pressure on their organs, including the biliary system. Diffuse diseases of the liver can be classified as hepatitis and heptoses according to the pathological change, which occurs. Clinically the differences between these two diseases are not marked, although some assistance can be obtained from clinico-pathological examination. Variations in hepatic dysfunction occur in the acuteness and severity of the damage, but the effects are the same and the clinical manifestations vary only in degree major hepatic functions are:
1- The maintenance of normal blood sugar levels by providing the source as glycogen.

2- The formation of some of the plasma proteins.

3- The formation and excretion of bile salts and excretion of bile pigments.

4- The formation of prothrombin.

5- Detoxification and excretion of many toxic substances. Abnormalities in this function will produce clinical signs.

**Manifestations of liver and biliary disease**

**(Jaundice)**

It is a clinical sign which often arises in diseases of the liver and biliary system but also arises in diseases in which there are no lesions of these organs. It does not always occur and may be absent in acute hepatitis. The jaundice is more intense in cases of obstructive and hepatocellular jaundice than in hemolytic jaundice. The staining of jaundice is due to staining of tissues, especially elastic tissue and not due to accumulation in tissue fluids so that it is best detected clinically in the sclera.

**Classification of jaundice:**

Types of jaundice with impaired bile flow and jaundice without impaired bile flow is the examination of the urine for the presence of bilirubin and urobilinogen and the determination of the relative amounts of direct and indirect bilirubin present in the serum. The kidney does not excrete indirect bilirubin, which has not passed through hepatic cells,, so that in hemolytic jaundice, the indirect bilirubin content of the serum is markedly increased and although the urine contains an increased amount of urobilinogen, no bilirubin is present. In those cases in which jaundice is caused by
impairment of bile flow there is a marked increase in the serum level of direct bilirubin, and the bilirubin content of urine is greatly increased.

Etiology:

A) Pre-hepatic causes (Affection of R.B.Cs):

1- Bacterial toxins (Bacillary Hb urea and leptospirosis).

2- Protozoa (Babesia and Anaplasma).

3- Viruses (Equine anemia virus).

4- Immunological reactions.

B) Hepatic causes: (Toxic and infections):

Any one of diffuse liver diseases causing degeneration of liver eels e.g. virus hepatitis.

C) Post hepatic causes: (Obstructive):

In causes of obstruction of bile duct by calculi, tumors or abscess pressing on the bile duct and causing its obstructing.

Clinical findings of hepatic dysfunction:

1) Yellow coloration of the mucus membrane and unpigmented portions of the skin (the color varies from lemon to orange yellow or greenish yellow). The sweat, milk and exudates coming from the body also contain bile pigments, while lacrimal and salivary secretion did not contain bile pigments.

2) Reduced appetite.

3) Albuminuria.
4) Presence of excitability and dyspnoea.

5) Presence of nervous signs including hyper excitability convulsions, coma, muscular tremors psychic disturbances including dullness, head pressing and failure to respond to signal.

6) Odema manifested commonly in the intermandibular space, and it may be sever and extended but it will be limited to the abdominal cavity as in cases of obstruction of portal circulation.

7) Emaciation.

8) The presence of alimentary tract syndrome comprising anorexia, vomition (in some species as pigs) and constipation with attacks of diarrhoea form time to time and the faecal color is pale.

9) Photosensitization may be also present.

10) Alteration in the liver size where it is grossly enlarged.

11) Hemorrhagic diathesis (prolongation of coagulation time).

12) The presence of abdominal pain.

**Pathogenesis:**

1- The appearance of yellow coloration in the mucous membranes and unpigmented skin is due to increased amount of bile pigment.

2- There are many factors cooperating to produce the nervous signs, of these hypoglycemia, failure of normal hepatic detoxication mechanism leads to accumulation of excess amino acids and anemia.
If the damage of the liver occurs more slowly the hypoglycemia will be less marked and will be accompanied by inability to perform work. In case of the presence of persistent hypoglycemia, structural changes occur in the brain which will lead to drowsiness.

Tissue wasting and a fall in the plasma proteins manifest 3- Failure of the liver to anabolize acids and plasma proteins during hepatic insufficiency. This may be sufficiently severe to cause oedema.

4- In hepatitis and hepatic fibrosis there is incomplete formation or absence of bile salts, which cause the alimentary tract syndrome.

5- As most of photosensitizing substances are excreted in the bile after their absorptions, thus in hepatic or retarded liver function photosensitization will results.

6- Hemorrhagic diathesis results in severe diffuse diseases of the liver this could be attributed to deficiency in the prothrombin formation resulting in prolongation of coagulation time.

7- Abdominal pains result from both distention of the liver (causing increased tension on its capsule) and lesions in the capsule and liver parenchyma.

8- Alteration in the size of the liver is often a result of advance congestion of the liver and multiple neoplastic lesions and to a less extend in acute hepatitis.

Treatment:

1) Treat the original cause if it is known.

2) In cases due to biliary congestion try to give the following purgatives:

R (for horses)
Aloes 18.00

Calomel 4.00

Sig.: Make it as one bolus and give it directly per os at the root of the tongue.

R (for cattle)

Magnesium sulphate  60.00 gm

Sodium bicarbonate  30.00 gm

Sodium citrate   10.00 gm

Sig.: to be given orally as a drench after being mixed and dissolved in sufficient quantity of water for 5 successive days.

3) Give atropine sulphate solution by intramuscular injection (1/4 - 1/2 grain for the horse and 0.001 - 0.002 grain for dogs) so as to dilate the bile duct and allow free outflow of the biles.

4) Flush the liver with large quantities of glucose that are given by 1/V injection.

5) Give calcium by both oral and parenteral injection.

6) Give also polyvitamines daily.

7.) Give easily digested laxative food (for horses and cattle give boiled linseed and little fodder while for dogs give barely water) at the same time you must avoid all the digestive troubles.

8) Lipotrophic factors should be given as choline.
Hepatitis:

This includes a diffuse degenerative and inflammatory which affect the liver and in addition the common types of cirrhosis as far as the causes are the same except that liver cirrhosis runs less actus coarse and slower than in hepatitis.

There are 5 types of hepatitis as follow:

a- Toxic hepatitis.

b- Infectious hepatitis.

c- Parasitic hepatitis.

d- Nutritional hepatitis.

e- Congestive hepatitis.

Etiology:

A) Toxic hepatitis:

1- Inorganic poisons including phosphorus, arsenic and possibly selenium.

2- Organic chemicals particularly carbon tetrachloride, hexachloroethane, gossypol from cottonseeds, cresols from coal tar pitch and chloroform.

3- Poisonous plants.

4- A number of fungi including pithomyces charlarum, aspergillus flavus, penicillin rubrum and periconia spp. and some algae.

5- Bacterial toxins.
B) Infectious hepatitis:

As in cases of salmonellosis, leptospirosis, septicaemic listeriosis and infectious equine anemia.

C) Parasitic hepatitis:

1- Massive liver fluke infestation.

2- Migration of larvae of ascaris.

1)) Nutritional hepatitis:

1- Deficiency of cystine and thiamine at one time.

2- Methionin deficiency.

3- Multiple dietary deficiency may be also a cause of nutritional hepatitis.

E) Congestive hepatitis:

Due to congestive heart failure, which causes increased pressure in the sinusoids of the liver, leading to anoxia and compression of surrounding hepatic parenchyma, which causes centritobular degeneration.

Pathogenesis:

Hepatitis are usually the same as follow:

1- The usual lesions in toxic hepatitis is centrilobular varying form cloudy swelling up to necrosis with a terminal venoculsive lesions as in some plant poisoning.

2- In infectious hepatitis the lesions varies form local cell to diffuse cell necrosis affecting all or most of hepatic parenchyma.
3- In parasitic type the changes depend upon the number and type of migrating or infesting parasites. This in massive fluke infestations, sufficient damage may occur or cause acute hepatic insufficient damage may occur or cause acute hepatic insufficiency, manifested particularly by submandibular oedema. In more chronic cases extension from a cholangitis may also cause chronic insufficiency.

4- hepatic fibrosis develops particularly if there is massive hepatic necrosis, which destroys entire lobules. Degeneration is not possible when the necrosis zonal, and fibrous tissue replacement occurs. Fibrosis is a terminal stage of hepatitis that may have developed acutely or chronically and is manifested by the same clinical syndrome as that to hepatitis except that the signs develop more slowly.

**Clinical findings:**

1- Anorexia is manifested on the animal accompanied by constipation, which is intermittent, by fits of diarrhoea with faeces, which are lighter in color than normal.

2- Nervous signs (Pawning or coma or hyper excitability with muscle tremors and convulsions) there may be dummy syndrome in which the animal pushes with the head, do not responds to stimuli and may be blind.

3- Vomition may occur in pigs.

4- Subacute abdominal pain may be present manifested by arched back and pain upon liver palpation.

5- Jaundice and oedema may or may not be present.

6- Photosensitization may also occur when the animal is given green food and exposed to sunlight.
7- In chrome hepatic fibrosis the signs are similar to those of hepatitis but develop more slowly, and persist for longer periods (often months).

8- Ascites and oedema of the dewlap may also occur.

**Clinical pathology:**

Urine and blood samples and liver biopsy specimens may be submitted for laboratory examination as outlined in jaundice and tests of hepatic dysfunction.

**Diagnosis:**

1- Clinical findings.

2- Differential diagnosis must be carried from

A) Encephalitis, which may be differentiated by the presence of jaundice and photosensitization.

B). Cardiac diseases by examination of the heart.

C) Acute diseases of the alimentary tract particularly those manifesting signs of nervous derangement by the case history and clinical examination which usually suggests a primary involvement of the alimentary tract.

**Treatment:**

1- J/V injection of glucose and electrolyte solutions especially, if the animal is off food.

2- Avoid giving proteins as far as possible because of the danger of ammonia intoxication and the given diet should be high in carbohydrate and calcium contents and consequently low in proteins and fats.
3- Give enema with soft soap and warm water in common with giving the animal purgatives.

4- Oral administration of antibiotics (broad spectrum).

5- In case of hepatic fibrosis, treatment is useful.

**Focal diseases of the liver**

**A) Hepatic abscess:**

It is a local suppurative infection of the liver but it may be also massive or extensively metastatic. When the condition is local suppurative type no clinical signs of hepatic dysfunction can be detected. The clinical findings of hepatic dysfunction can be detected in case of massive extensively metastatic suppurative hepatic case. They may however in case of suppuration of the liver give signs of toxemia because of the destruction tissue, or liberation of potent toxins.

**Etiology:**

1- Corynebacterium pyogenes.

2- Sphaerophorus necrophorus invaded from the lesions may also lead to hepatic infections by sphaerophorus necrophorus or other organisms.

3- Abscessed livers are common in cattle fed heavily on concentrates.

4- Toxins liberated from clostridium novyii in case of black disease, and from clostridium haemolyticum in bacillary haemoglobinuria and in focal hepatic necrosis.
Clinical findings:

The clinical findings of these specific diseases are included under the discussion of each disease and the only finding to all is local pain on palpation or percussion over the liver.

Treatment:

1- Give massive doses of broad spectrum antibiotics.

2- Give the animal diet as in case of hepatitis.

3- Finally when no response to antibiotics, surgical treatment procedure must be done to remove the abscess as far as you can.

B) Tumors of the liver

Metastatic lesions of lymphomatosis in calves are the commonest neoplasms encountered in the liver of animals although primary adenorila, Adencarcinoma and metastases of other neoplasms in the area drained by the portal tract are not uncommon especially in ruminants. They produce no signs of hepatic dysfunction but they may cause sufficient swelling to be palpable and some abdominal pain by stretching of the liver capsule. Primary tumors of the gallbladder also occur rarely and do not as rule cause clinical signs.

C) Diseases of the peritoneum

Peritonitis:

Definition:

It is an inflammation of the peritoneum. The disease is accompanied by abdominal pain, which varies in degree with the severity of peritonitis. Typical manifestations are:
- Rigidity of the abdominal wall.

- Constipation.

- Systemic reaction.

**Etiology:**

1- Perforation of alimentary tract as in traumatic reticuloperitonitis in cattle or goats-abomasal ulcer in cattle.

2- Perforation of the genital tract as occurs in rupture of the vagina during violent coitus or rupture of the uterus at parturition of during manual correction in Dystockia (Shock + Hemorrhage).

3- Failure of the uterus to heal may be followed by peritonitis.

4- perforations of gastric ulcers caused by larvae of gastrophilus spp. & Habronema megastomum in horses.

5- Rupture of the stomach or intestine in acute dilation or obstruction (Shock + internal hemorrhage).

6- passage of infection through the gut wall (the wall is devitalized and permeable)