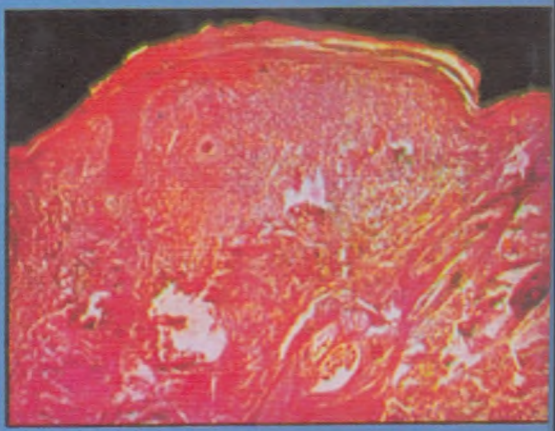
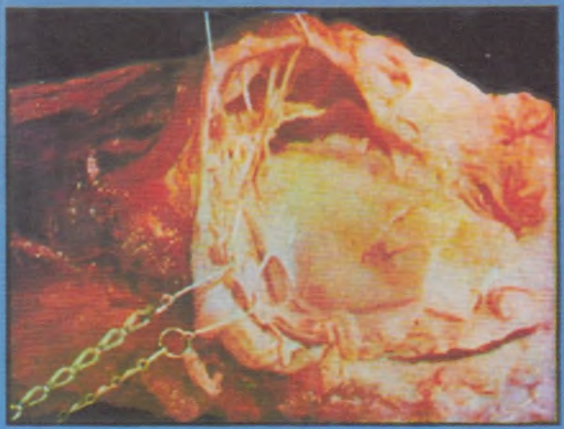
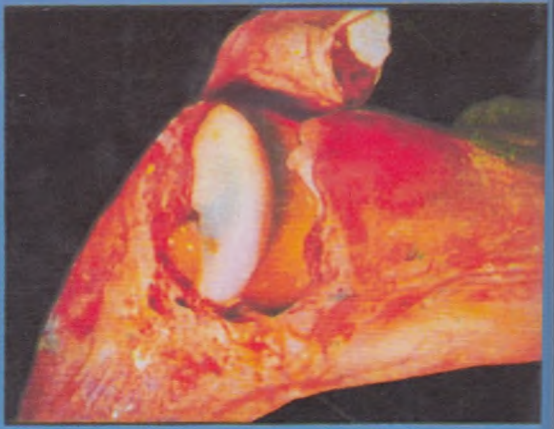
Veterinary Pathology

SEVENTH EDITION

Ganti A. Sastry
Revised & Enlarged by : P. Rama Rao







Dedicated to my late father

G.Y. Somayajulu Garu

Veterinary Pathology

SEVENTH EDITION

Part I : GENERAL PATHOLOGY
Part II : SPECIAL PATHOLOGY
Part III : INFECTIOUS DISEASE

Ganti A. Sastry
G.M.V.C., B.V.Sc., M.Sc., M.S., Ph.D. (Kansas)
Retired Principal & Professor of Pathology,
College of Veterinary Science, Tirupati - 517 502

Revised & Enlarged by

P. Rama Rao
M.V.Sc., F.R.V.C.S. (Sweden), Ph.D.
Principal, Andhra Pradesh Agricultural University,
College of Veterinary Science, Tirupati - 517 502

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Ph: 49344934 • Fax: 011-49344935
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Branches:

- ***Bengaluru:*** 2975, 17th Cross, K.R. Road, Bansankari 2nd Stage, Bengaluru - 70 • Ph: +91-80-26771678/79 • Fax: +91-80-26771680 E-mail: cbsbng@gmail.com, bangalore@cbspd.com
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- ***Kochi:*** Ashana House, 39/1904, A.M. Thomas Road, Valanjambalam, Ernakulum, Kochi • Ph: +91-484-4059061-65 Fax: +91-484-4059065 • E-mail: cochin@cbspd.com
- ***Kolkata:*** 6-B, Ground Floor, Rameshwar Shaw Road, Kolkata - 700014 Ph: +91-33-22891126/7/8 • E-mail: kolkata@cbspd.com
- ***Mumbai:*** 83-C, Dr. E. Moses Road, Worli, Mumbai - 400018 Ph: +91-9833017933, 022-24902340/41 • E-mail: mumbai@cbspd.com

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PREFACE FOR THE SEVENTH EDITION

In an effort to update the informations on poultry diseases according to VCI syllabus and needs of poultry industry. Dr. P. Rama Rao, Dr. Gopal Yadgirkar and Dr. S. Rafeeque Ahmed former staff members of the Department of Pathology in the faculty of Veterinary Science, Acharya NG Ranga Agricultural University have revised the book.

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   Bailliere Tindali, 3rd edition, 1990.

DR. P. RAMA RAO

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Continued demand for my book by the student body had necessitated the production of this Sixth edition. It is no little satisfaction to the author to find that his book is felt needed and is found useful by most of the Veterinary students of the country.

In this edition some parts are revised and many facts and conditions have been added to bring the contents of the book uptodate. As such the number of pages had to be increased. To make the information more authentic. I have made a departure in this edition, by requesting Professors of other colleges as well as some of our own college, to subscribe chapters or conditions in which they have specialised. By this means the students get benefitted by the experience and expertise of these Professors. I am grateful for the following persons for contributing on the subjects noted against their names to this part :

Dr. D. Anjaneya Prasad	— Bovine Ketosis and revision of chapter on Disturbances in Mineral Metabolism.
Dr. P. Babu Rao	— Ionizing radiation.
Dr. B. Sambamurthy	— Viruses and Tumorigenesis.
Dr K Sathyanarayana Rao	— Electron Microscopic structure and function of normal cell.
Dr. A. Sreerama Murthy	— Hereditary defects and disease resistance.
Dr. D. Sunderasiva Rao	— Revision of chapter on Neoplasms.
Dr. J.L. Vegad	— Chapters on Inflammation and Healing.

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It is the fond hope of the author that this book will continue to be useful to the Veterinary Students and that it will help lay a sound foundation in the better and rational understanding of disease processes.

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GANTI A. SASTRY

CONTRIBUTORS

Part I

Dr. D. Anjaneya Prasad, BVSC, MVSC, PhD (Kansas) Professor of Animal Sciences, College of Veterinary Science, Tirupati (A.P.)

Dr. P. Babu Rao, MSC, PhD, Lecturer in Physics, S.V. College of Arts and Science, Tirupati (AP.)

Dr. B Sunbamurthy, GMVC, BVSC, MS, (Kansas) Retired Associate Professor of Microbiology, College of Veterinary Science, Tirupati (A.P.)

Dr. K. Sathyanarayana Rao, GMVC, BVSC, MS, (Kansas) Principal & Professor of Physiology, College of Veterinary Science, Tirupati (A.P.)

Dr. A. Sreeramamurthy, BVSC, MSC (Vet), PhD, Head, Department of Animal Genetics and Breeding, College of Veterinary Science, Tirupati.

Dr. D. Sunderasiva Reo, M D. Retired Principal and Professor of Pathology, Rangaraya Medical College. Kakinada, A P. *Now* Pathologist, Amala Cancer Hospital and Research Institute, Puzhakkal, Kerala.

Dr. J.L. Vegad. BVSC, ASSOC, IVRI ; PhD. (New Zealand). University Professor and Head, Department of Pathology, College of Veterinary Science & AH. Jabalpur, M.P.

Part II

Dr. M.S. Kwatra, MVSC, PhD. Senior Veterinary Investigation Officer, College of Veterinary Science, Ludhiana. (Mulberry Heart Disease and Manchester wasting Disease of cattle).

Dr. B B. Mallick, D S.C. (Sarborne) ; FCAI., Assoc IVRI, BSC, G VSC (Hons) Head, Division of Virology, IVRI, Mukteswar. (Immunological Diseases).

Dr. A. Rajan, BVSC, MVSC, PhD. Professor of Pathology, College of Veterinary Science, (PO) Mannuthy. Trichur, Kerala. (Revision of "Endocrine Glands").

Dr. P. Rama Rao, BVSC, MVSC, I RVCS (Sweden) PhD. Head, Department of Pathology, College of Veterinary Science, Tirupathi. (Revision of "The Reproductive System").

Dr. J.L. Vegad. BVSC, Assoc IVRI, PhD (New Zealand). University Professor and Head, Department of Pathology, College of Veterinary Science, & AH, Jabalpur (The Nervous System).

Part III

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Dr. M. S. Kwatra, MVSC, PhD, Senior Veterinary Investigation Officer, College of Veterinary Science, Ludhiana, for contributing on 'Degnalla Disease',

Dr S J. Seshadri, MVSC, Professor & Head. Department of Pathology Bangalore Veterinary College for his contribution on Bacterial and Viral Diseases of Poultry,

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Dr. A. Venkataratnam, GMVC, BVSC, DAP, MSC (L'pool) Associate Professor of Parasitology, College of Veterinary Science, Tirupathi, for reviewing chapter on "Diseases caused by Protozoa ",

THE CARDIOVASCULR SYSTEM

THE HEART

The function of the heart is to maintain sufficient supply of blood to meet the needs of the body tissues.

"The cardiac reserve represents the ability of the heart to meet increased demands" that may arise during fever, exercise, anemia, pregnancy etc The heart is able to adapt itself to varying physiological needs and to pathological abnormalities. This ability is known as "compensation". For example, in a developing pulmonary fibrosis or chronic nephritis, when there is resistance to free flow of blood, the heart becomes hypertrophied so that the force of the heart is increased to overcome the peripheral resistance, thereby maintaining the volume flow and blood pressure in the vessels.

But in the face of an ever increasing peripheral resistance together with the increased work load put on the heart, the heart is not able to cope up with the demands and so becomes fatigued and fails. This state in which the heart is no longer able to compensate is called "decompensation". Decompensation is gradual and results in dilatation of the ventricles.

The factors that lead to cardiac failure are three fold:

1. **Alteration in the return of venous blood.** To a limit the heart is capable of adapting itself to increased inflow of blood. But if this condition persists, then the heart becomes fatigued and fails

If the venous return is inadequate, the heart is not capable of compensation to meet the situation and so finally fails.

2. **Increased resistance to out-flow:** We have already mentioned that heart compensates for increased resistance to out-flow. But in time the reserve power of the heart is exhausted and so fails. The causes of increased resistance to outflow are: hypertension, narrowing or dilatation of valvular orifices, thrombosis and arteriosclerosis.

3. **Impaired cardiac contraction:** Any disease that injures the myocardium reduces the contractile power of the heart and so the compensatory mechanism cannot work. In man the most important cause for myocardial injury is coronary vascular insufficiency.

Developmental anomalies

1. **Patency of foramen ovale:** Very soon after birth, the foramen ovale through which the right atrium communicates with the left, should become closed and blood diverted to the lungs. But sometimes this does not happen and so communication exists between the two areas. In human pathology, this condition gives rise to the "Blue babies". A small opening may not be of significance. But a large patent foramen leads to hypertrophy of the right ventricle. Patent foramen ovale has been met with in calves. Paradoxical embolism occurs in this condition.

2. **Interventricular foramina** Small openings connecting the ventricles are of no consequence. But if the openings are ½ to 1 cm. in diameter then the following functional changes are noticed:—elevation of pressure in the ventricle and pulmonary artery and hypertrophy of the right ventricle. A systolic murmur is heard.

3. **Patent ductus arteriosus:** Since in the fetus there is no necessity for blood to go to the lungs for oxidation, a shunt connects the pulmonary artery and the aorta. This is the ductus arteriosus, which should be obliterated within a few weeks after birth, Sometimes this closure does not happen, causing increased pressure in the pulmonary artery, leading eventually to hypertrophy of the right ventricle. Animals are cyanotic. Thrombosis of the ductus arteriosus, if it is patent, is a hazard.

4. **Persistance of the right aortic arch:** This is seen in dogs mostly and in bovines rarely. The esophagus is encircled and constricted by the ductus arteriosus of this aorta leading to obstruction of the esophagus resulting in dysphagia and then to dilatation of the esophagus proximal to the obstruction. Dilatation can be seen by x-ray examination. Puppies vomit immediately after taking food.

5. **Coarctation of the aorta:** This is narrowing of the lumen of the aorta, most commonly occurring close to the heart or between the origin of the common brachiocephalic artery and the ductus arteriosus. This leads to hypertrophy of the left ventricle as there is hindrance to flow of blood.

6. **Transposition of the aorta:** This is a serious condition in which the aorta arises from the right ventricle or sometimes from both the ventricles This is incompatible with life.

7. **Congenital aneurysm** of the aorta or pulmonary artery denotes dilatation of the respective vessels. These may cause pressure atrophy of the neighbouring structures. Fatal hemorrhage may result due to thinning of the vessel wall.

8 **Pulmonic stenosis :** This condition in which the pulmonary valves become fused together resulting in stenosis, is the most common anomaly seen in dogs. The right ventricle hypertrophies, the wall becoming twice as thick as normal. A precordial thrill and a harsh systolic murmur are present.

9. **Sub-aortic stenosis :** This condition is seen mostly in Alsatians and Boxers. A ring of fibrous tissue immediately below the cusps of aortic valves causes stenosis, resulting in hypertrophy of the left ventricle. Clinically, the following symptoms are observed; poor condition; inability to exercise: frequent fainting fits; tachycardia with poor pulse volume; systolic murmur and cardiac dilatation in X-ray pictures.

10. **Tetrology of Fallot :** In some animals the following pathological changes may be present. (i) right ventricular hypertrophy, (ii) stenosis of pulmonary valves. (iii) defects in the interventricular septa and (iv) dextraposed aorta. In man this condition also gives rise to "blue babies". Affected animals are stunted and the mucous membranes are cyanotic. Clinically, the pulse and respiratory rates are increased and a murmur is heard.

11. **Endocardial fibro-elastosis :** This condition may be seen in dogs, cattle and cats. The endocardium of the ventricles is thickened due to increase in elastic fibres of the subendothelial connective tissue. Death occurs soon after birth. Cause is not known.

12. **Other defects are : (a) ectopia cardis** in which the heart is found outside the thorax, usually in the neck region or abdominal cavity. (b) **acardia :** in which there is complete absence of the heart. (c) **diplocardia :** in which two hearts are present. These defects are incompatible with life.

THE PERICARDIUM

Abnormal contents :

Hydropericardium : In this condition, there is excess of serous fluid. The causes are :

(a) Cachectic diseases.

(b) Congestive heart failure (in this condition there is increased pressure in the coronary veins and capillaries).

(c) Renal disease.

(d) Chronic stomach worm infection leading to hypopoteinemia

(e) Damage to capillary endothelium as occurs in (i) many infections due to toxins or (ii) anoxic conditions.

(f) Anemia, especially hemolytic.

(g) Liver insufficiency—stasis of portal circulation, hypoproteinemia and general edema.

(h) Mulberry Heart Disease in pigs.

(i) Tumors : (i) implanted metastases on pericardium, (ii) lymphogenous metastases to the myocardium. (iii) primary tumors at the base of the heart and in the anterior mediastinum

The fluid is straw-colored and clear. In some cases as mulberry heart disease and infectious diseases, floculi may be present. In these conditions, due to damage of the capillary endothelium much protein flows out into the exudate·

If the fluid in the pericardial sac persists for a long time, it may become turbid and organised, giving a shaggy (bun-butter) appearance to the pericardium and epicardium.

Hemopericardium : In this condition there is accumulation of blood in the pericardial sac. If the clot completely encloses the heart, the condition is known as **cardiac tamponade.**

Causes : Trauma and rupture of heart, arota or coronary artery.

Pneumopericardium : Gas accumulates in the pericardium The sources may be :

(a) Activity of gas producing organisms, which may enter the pericardium with a penetrating body

(b) Gas may escape into the pericardium in traumatic reticulitis.

(c) From the lungs gas may enter the pericardium when a lesion involving the lungs and pericardium breaks down.

(d) In compound fractures of ribs, gas may enter from the outside.

Pyopericardium : Pus in pericardium is mostly seen in traumatic pericarditis. It may also be found in (i) tuberculosis.

(ii) purulent pleuritis
(iii) purulent pneumonia } as secondary infection.

(iv) a rupturing myocardial abscess.

Serous atrophy of subepicardial fat : In animals suffering from cachectic diseases, the fat present in the grooves of the heart becomes transformed into a gelatinous mass. The fat is lost and in its place edematous fluid accumulates. The interstitial tissue also is edematous. White foci of necrosis may be seen in these lesions.

Hemorrhages on pericardium : Petechial hemorrhages are common in shock, toxemia and hypoxemia. Most common causes are the toxins of bacteria and viruses causing various diseases. Purpura hemorrhagica of horses is a condition in which petechiae of the pericardium are common. Ecchymoses and diffuse hemorrhages are common in sweet clover poisoning.

Pericarditis : Inflammation of pericardium is commonly seen in animals. It is usually due to bacterial infections and secondary to other diseases. The routes of infection may be :—

1. **Hematogenous:** occurs in septicemic conditions and other specific diseases.

2. **Lymphogenous :** from the inflammatory processes of neighbouring tissues—myocardium, pleura, bronchial or mediastinal lymphnodes.

3. **Trauma :** Directly from outside as in bullet wounds. Or by a foreign body entering through the rumen and reticulum (traumatic pericarditis).

Pericarditis in animals is found in the following conditions :—

Horses : Pericarditis is a complication of pneumonia, influenza or strangles. Streptococci are the organisms usually present.

Cattle : In bovine encephalomyelitis, pasteurellosis, contagious bovine pleuropneumonia, black quarter and coliform infection of the new born through the umbilicus.

Swine: In erysipelas, pneumonia (Mycoplasma Spp) pasteurellosis streptococcal infections, salmonellosis and hog cholera and infection by *Hemophilus suis* (Glasser's Disease)

Sheep: In pneumonia (Pasteurellosis), salmonellosis.

Dogs Rare, may be a complication of distemper and produced by secondary bacterial invaders and may also be found in leptospirosis.

Fowls: Fowl cholera and pullorum disease.

Pericarditis is classified according to the nature of the exudate.

Fibrinous Pericarditis: Pathogenesis: In the beginning, the surface of the serous membrane is dry following congestion. The glistening luster is lost. This phase is soon followed by marked exudation of serous fluid both within the subserosal tissue (thereby thickening it) and into the sac. Proliferation and desquamation of the mesothelial cells occur. Fibrin is deposited on the membranes as well as into the fluid and gives rise to the condition. "Shaggy heart" and "Bread and butter appearance"

Inflammatory cells consisting of neutrophils, lymphocytes, plasma cells and macrophages infiltrate into the subserous connective tissue. In infections by pyogenic organisms, the leucocytic infiltration into the fluid is so great that it becomes turbid and pyopericardium results.

If the cause is removed early exudate may be removed (the fluid part is absorbed through the blood and lymphatics and the solid matter liquefied by the proteolytic enzymes of the leucocytes which absorb it) mesothelium proliferates and fills in the places where it is lost and complete resolution is possible. But in severe infections with large amount o serofibrinous exudate such complete resolution is not possible and so adhesions occur. This is brought about by organisation of the exudate. This process involves proliferation and growth of granulation tissue from the subpericardial tissue. The organisation and resultant scar tissue may be focal or diffuse. In the latter case the cavity is obliterated and the animal finally dies of decompensation. Occasionally caseation necrosis and calcification of the exudate may occur.

Suppurative pericarditis: This occurs more frequently in cattle due to bacterial infestation with a metallic foreign body passing through rumen, reticulum and diaphragm (Traumatic pericarditis). In poultry salmonellosis is accompanied by suppurative pericarditis. This condition may be a complication of suppurative pleuritis and bronchopneumonia of animals. Nutritional anemia in pigs, due to deficiency of iron and copper, is accompanied by suppurative pericarditis.

In this condition, especially in cattle of traumatic pericarditis, the pericardium is filled with pus. A thick membrane of fibrin forms on both the surfaces. Resolution in such cases is impossible and organisation supervenes, resulting in adhesion between the perietal surface and the epicardium—*cor rugosum* or shaggy heart. Sometimes the whole of the exudate is organised and so the pericardium becomes very thick.

The exudate and adhesions compress the heart, **constrictive pericarditis** though initially hypertrophy of the myocardium may occur. Due to mechanical interference and degeneration by toxins, the heart is not able to work properly,

chronic venous congestion develops and the heart stops. Death due to toxemia may supervene long before chronic venous congestion and edema develop.

In chronic constrictive pericarditis, the thickened pericardium that is tightly attached to the heart compresses it and so its normal diastolic filling is interfered with. So also contraction of the heart during systole is hampered. Since the roots of the great veins are compressed blood flow into the heart is obstructed. These result in chronic venous congestion and reduced output of the heart producing nutmeg liver, cardiac cirrhosis, splenomegaly, ascites, effusion into the pleural cavity and deposit of fibrin on the liver and spleen (sugar icing).

In traumatic pericarditis severe leucocytosis and extreme shift to the left are noticed.

Uric Acid pericarditis: In fowls suffering from visceral gout, the needle shaped salts of the uric acid and urates are deposited on the parietal and visceral layers of the pericardium. This gives the appearance of fine frost.

The urates being irritants cause inflammation of the epicardium, with the resultant formation of granulation tissue and hence organisation of the deposits occurs.

Specific inflammations: Tubercular pericarditis is common in cattle and rare in dogs.

Myocardium

Hypertrophy: In hypertrophy of the heart muscle, there is increase in the size of the individual muscle fibres so that the walls become thicker. Usually the left side is more often affected and the ventricles suffer more frequently than the atria. Physiologically hypertrophy may occur when greater strain is put on it as in hearts of the race horses and grey hounds. The causes for hypertrophy may be intracardial or extracardial hindrance to the flow of blood.

Intracardial hindrances: Stenosis or insufficiency of cardiac valves give rise to this condition.

(a) **Aortic valves;** Hypertrophy of the left ventricle occurs due to stenosis or insufficiency of aortic valves.

(b) **Mitral valves :** Lesions of these valves lead to hypertorophy of left auricle.

Ultimately, the lesions mentioned above lead to brown induration of the lungs, resulting in hypertrophy of the right ventricle

(c) Lesions of the valves of pulmonary artery produce hypertrophy of the right ventricle.

Extracardial hindrances: Right ventricle hypertrophies in pulmonary emphysema (heaves), chronic interstitial pneumonia, brown induration of lungs and pneumoconiosis.

Usually, hypertrophy and dilatation of the heart occur together.

The heart is enlarged with thickened walls. If the lumen of the chambers is narrowed, it is called **concentric hypertrophy.** Mere hypertrophy is called **simple** but if accompanied by dilatation it is called **eccentric hypertrophy.** Hypertrophy of the right side increases the width of the base, while the left sided

hypertophy increases the length of the heart. But bilateral hypertrophy causes the heart to be rounder.

The increased size of the cardiac muscle needs more nutrition, which may not be adequately supplied to every muscle fibre by the coronary vessels. Because of inadequate blood supply, metabolites formed in and arround the fibres are not removed and so accumulate. Due to these two factors, the muscle fibres get degenerated and in face of continued work of the heart under the circumstances that were originally responsible for the hypertrophy, atrophy of the muscle fibres results. Consequently, the compensated heart falters and is no longer able to meet the demands and so decompensation sets in, ultimately ending in heart failure. Dilatation of the heart that is a sequel to stasis in pulmonary circulation is called **cor pulmonale.**

Dilatation. In this condition, one or more chambers may undergo pathological enlargement, usually due to deficient emptying during systole. Frequently the right ventricle is affected.

Causes: Dilatation occurs in various infectious and intoxicating diseases in which myocardial degeneration or myocarditis is present and so the heart is not able to expell all the blood received into it and hence blood accumulates in its chambers dilating them.

Sudden acute dilatation is attributed to the cumulative action of toxic products on cardiac muscle in severe diseases.

Chronic dilatation usually occurs with hypertrophy in which it is a terminal lesion.

In dilatation, the heart is rounder and more globular. The walls are thinner and the papillary muscles are attenuated. Very great dilatation of the heart of man is called **cor bovinum.** Dilatation leads to congestive heart failure.

Cardiac failure: We have already noticed that the heart has great reserve powers and that it can adjust itself to increased demands placed upon it due to increased venous return and to increased resistance to outflow. In long continued states where this demand still exists decompensation sets in, especially when there is impaired cardiac contraction, resulting in cardiac failure.

Though failure of one side of the heart ultimately produces excessive strain on the other side, it is convenient to study the pathological physiology of each side separately.

Left-sided heart failure: The most common causes are :

(1) Hypertension
(2) Arotic valvular disease } Stenosis and incompetence due to
(3) Mitral valvular disease } endocarditis
(4) Congenital heart disease
(5) Myocarditis
(6) Myocardial degeneration
(7) Adhesive pericarditis
(8) Nephritis in dogs.

Majority of the clinical symptoms in left-sided heart failure stem from a) diminished blood flow through the various organs and tissues of the body

and (b) from pulmonary congestion caused by venous stasis. The following changes in the organs with related symptoms occur :

Lungs : Due to damming back of blood in the lungs and diminished cardiac output, there is venous congestion in lungs which is transmitted to the alveolar capillaries resulting in accumulation of the edema fluid in the alveoli. Sometimes, small capillaries may rupture resulting in small hemorrhages into the alveolar spaces (heart-failure cells are seen in such an event).

Impaired exchange of gases and reduced vital capacity of the lungs that result due to edema, cause hypoxic stimulation of the carotid sinus and the respiratory centre and so reflex dyspnoea occurs. Cough occurs due to the irritation of the respiratory mucosa by the edema fluid.

Kidneys : In the congestive left-sided heart failure, there is significant alteration in renal function. Renal excretion is impaired due to renal anoxia that results from impairment of circulation (Vasoconstriction). Salt and water are retained, raising thereby the blood volume which further adds to the work-load of the heart. Further impairment in renal blood flow results in diminished excretion of nitrogenous substances—prerenal uremia. The condition is called prerenal because, the fault lies not in the kidneys, but in some prerenal or cardiac lesions. Retention of sodium and water accentuates edema, noticed first in the dependent parts of the body.

Brain : Anoxia of the brain results in increased, irritability restlessness and in far advanced cases, stupor and coma.

Right-sided heart failure: Right-sided heart failure usually occurs with the left-sided heart failure and it is only rarely that right-sided heart failure in a pure form occurs. Because, congestion of pulmonary vessels that occurs in left-sided failure must ultimately embarrass the right side of the heart. Hence the causes for right-sided failure include those enumerated for the left side, especially mitral stenosis and incompetence, which greatly increase the pressure in pulmonary vessels.

Causes for the pure right-sided heart failure are:—

1. Myocarditis. As the right ventricle is weaker than left, it fails more rapidly when myocardium is damaged.
2. Myocardial infarction and degeneration.
3. Causes producing increased resistance for the flow of blood in the lungs—emphysema (heaves in horses) and chronic interstitial pneumonia.
4. Constrictive pericarditis: in this condition the flow of blood into the heart is blocked.
5. Hydropericardium. Here also blood is blocked from entering the heart.
6. Endocarditis, especially of tricuspid valves, producing incompetence and stenosis.

In right-sided heart failure, the primary disturbance is damming back of blood in the systemic and portal venous circulations. with consequent decreased flow of blood into the left auricle from the lungs. Anoxia that is caused thereby produces renal pathology in which there is retention of salt and water increasing the blood volume. So edema results. In the horse and ox, edema is

seen subcutaneously, while in the dog, ascites is manifested and in the cat pleurisy is seen.

Liver; Because of congestion the liver is enlarged. In the acute and severe phase, pulsation may be present. There may sometimes be actual hemorrhage round about the central vein. Atrophy and necrosis of the hepatic cells around the central vein are present. If the animal lives long enough (usually animals do not survive for this length) fibrosis of the central part will occur—*cardiac cirrhosis* Slight hepatic dysfunction and icterus may be evident. But clinically impaired hepatic function is not a feature.

Kidneys: In the right-sided failure, congestion and anoxia of the kidneys is more marked than in the left-sided failure. Hence renal function is much affected and there is, therefore, greater retention of salt and so the volume of blood is much increased. Due to the decreased renal blood flow there is decreased formation of glomerular filtrate and hence reduced sodium filtration occurs. The tubules reabsorb the little salt that is excreted. So along with the salt water is also reabsorbed and hence the increase in the blood volume; which together with venous stagnation produces interstitial edema, manifested in the dependent parts.

Spleen: Splenomegaly is seen due to congestion. Hemorrhages may be present liberating hemosiderin followed by organisation and fibrosis. Such areas may be calcified. Metaplasia of the reticulum to fibrous tissue causes hardening of the organ.

Other Symptoms: Due to congestion in the portal system, the stomach and intestines may also manifest venous stasis with resultant digestive troubles—diarrhoea. Cyanosis is also seen. In the horses epistaxis may occur.

In summary, therefore the pathology and manifestation in the left-sided heart failure are essentially results of stasis in pulmonary circulation, while stasis in the systemic circulation is the cause of the pathology seen in right-sided failure.

Cardiac failure may be unilateral but usually it is bilateral. Heart failure, therefore, is a syndrome of failing circulation in various organs. Hence to detect heart failure clinically or at autopsy by just examining the heart is very difficult We have to examine other organs to arrive at a correct diagnosis.

Fatal Syncope of pigs, Herztod

This is a very serious disease in pigs, which suddenly fall down and die while being driven about or while feeding or during mating. If animals could be observed while alive, symptoms of dyspnoea, paralysis and spasms can be seen.

Cause: The exact cause of the disease is not yet clear. It is probably a nutritional condition. There may be hormonal imbalance, the hormones concerned being the Thyrotropic horn one, the ATCH and the Growth hormone. A diet rich in carbohydrates and poor in proteins (feeding on potatoes) producing vitamin and protein deficiency has been cited a cause. An allergic etiology is also put forward.

Lesions: Serous cavities contain large quantity of serous fluid. All organs show passive congestion Heart shows grayish streaks and patches of

hyaline degeneration with round-cell infiltration. Lungs are hyperemic and edematous. Skeletal muscles show hyaline degeneration or cloudy swelling with loss of cross striations or even atrophy. Passive congestion of the liver and severe passive congestion of the intestinal mucosa are also observed.

Thyroid is congested due to extreme dilatation of the blood vessels. The follicles show collapse due to separation of the epithelium from the basement membrane by subepithelial edema. Infiltration by lymphocytes is observed The colloid is watery and thin. The adrenals are atrophied and the immediate cause of death may be adrenocortical insufficiency. (See page 149 for fatal syncope in cattle caused by copper deficiency)

Round Heart Disease

Sudden deaths in hens under eight months of age may occur, especially in summer months.

Causes: A diet rich in carbohydrates and poor in protein is suspected. Poisoning by zinc is suggested by some workers. No evidence of infection has been seen.

Macroscopically, the heart is greatly enlarged and appears rounder, the apex losing its normal conical shape The left ventricle especially, is hyperrophied and dilated. Gray streaks or spots may be seen on the myocardium. Hyperemia of liver, kidneys, intestines and lungs may be observed. Pericardium may contain fibrinous exudate. Edema of the subcutis and lungs may be noticed. Comb is cyanotic.

Microscopically, the myocardial fibres are hypertrophied. In some areas fatty degeneration of the heart may be seen. In others small nodules consisting of accumulation of histiocytes and lymphocytes may be observed.

DISTURBANCES IN METABOLISM

Cloudy swelling: This condition is seen in different toxemias and septicemias, and in febrile conditions.

Macroscopically, the heart is slightly enlarged, has a cooked, pale appearance and is friable.

Microscopically, the muscle fibres are sightly swollen and their cytoplasm is granular, cross striations are indistinct.

Since autolytic changes occur soon after death simulating cloudy swelling care should be taken to distinguish one from the other.

Fatty degeneration: In this condition, the irritants are more severe than those causing cloudy swelling. Fatty degeneration is met with in prolonged infections; piglet anemia, toxemia, phosphorus, arsenic and chloroform poisoning, purpura hemorrhagica in horses, pyometra; avitaminosis. E.

Macroscopically, the heart is enlarged and yellowish in color. In severe fatty degeneration yellowish striping of the endocardial surface-"tigering" is seen. "Thrush-breast" heart is observed when there is mottling of subendocardial fibres, especially on the papillary muscle

Microscopically, minute droplets of fat, detected with special stains are seen in the muscle fibres, which normally do not contain any. The nuclei are degenerated.

Obesity: It is normal to find deposition of fat subepicardially. But fat is not found in the myocardium. In some cases of obesity, fat of coronary adipose tissue infiltrates between the myocardial fibres—fatty infiltration. This fat crowds out the fibres and may interfere with the action of the heart resulting in its failure.

Hyaline degeneration: Hyaline degeneration is best seen in the conditions, called white-muscle disease in calves and stiff lamb disease in lambs due to vitamin E deficiency. This may also be seen in myocarditis, in gossypol poisoning, 'Herztod' in swine and copper deficiency (falling disease) in cattle. The muscle fibres are homogeneous and glassy. This is a prenecrotic lesion, terminating usually, in necrosis and calcification.

Necrosis of the myocardium is usually focal and is seen as grayish spots scattered in the muscle. This is a common lesion seen in white muscle disease of calves and lambs. Thiamine deficiency in pigs has been found to produce coagulative necrosis of the myocardium.

Hyaline degeneration and necrosis of the myocardium are seen in Foot and Mouth Disease in cattle; in Equine Viral Arteritis and in Swine Fever.

Calcification: Dystrophic calcification of necrotic myocardial fibres may occur. So this is common in white-muscle disease. In cattle poisoning by organic compounds of mercury causes hyaline degeneration of Purkinje fibres, followed by calcification.

In puppies excessive vitamin D or calcium therapy results in calecification of the myocardium.

Disturbances in circulation

Infarction of heart is relatively uncommon. This condition is due to occlusion of the coronary artery by a thrombus, atheroma or arteriosclerosis. Rarely emboli arising from cardiac vegetations (in swine) may give rise to infarction.

The sudden occlusion of a large artery may cause necrosis and deaths. But gradual obstruction of smaller branches will cause atrophy and replacement fibrosis, which being weak, is a place for dilatation to occur. This is called *cardiac aneurysm*.

Hemorrhages: petechiae under the epicardium and larger ecchymoses under the endocardium are common in cattle and sheep and are encountered in toxemias, septicemias and in death due to asphyxia.

MULBERRY HEART DISEASE

(By Dr. M. S. Kwatra)

Mulberry heart disease in pigs is a dietetic microangiopathy which is characterised by the sudden death due to heart failure, hydropericardium and typical linear hemorrhages on the heart giving it an appearance of a bunch of mulberries. The piglets belonging to Landrace breed are relatively more susceptible to this disease and the cases mostly appear on the farm during the spring season. The disease has recently been noticed in Assam. Though the morbidity

rate is generally low, mortality rate is quite high. The disease commonly occurs among 3-4 month old pigs. especially in active healthy looking ones. The etiology or the cause is unknown. The disease is considered to be a dietary deficiency and addition of vit E and selenium to the diet has a beneficial effect Generalised edema is suggestive of damage to endothelium which may be due to toxins of bacterial origin. In majority of cases no symptoms are observed as the course of the disease is extremely short. Some cases may show incoordinated gait, dyspnoea, muscular tremors and weakness. The temperature is usually within the normal range. The chronic cases show nervous symptoms and poor weight gains

The characteristic gross lesions are :

1. Edema and emphysema of the lungs
2. Hydropericardium
3. Hydrothorax

} The fluid is so rich in fibrin that it clots

4. Diffuse hemorrhages on the epicardium and endocardium giving the heart a mulberry appearance.
5. Ascites .
6. Hyperemia of gastrointestinal tract.
7. In animals with delayed death softening of the cerebral gyri (due to anoxia consequent to pulmonary lesions)

Microscopic Changes

A. **Heart**

(i) The blood vessels of the myocardium reveals generalised, hyperemia and typical fibrinoid degeneration in several blood vessels.

(ii) The myocardial fibres show necrobiotic alterations.

(iii) Hemorrhages and edema in between the bundle fibres are seen.

B. Liver sections reveal acute passive hyperemia and degenerative changes of hepatic cells in the cetrilobular areas.

C. Lungs also show passive hyperemia, enlargement of the lining cells of the alveoli, marked alveolar emphysema and edema.

D. Leucoencephalomalacia in white matter of the cerebrum is generally observed.

The mulberry heart disease appears to have no etiological relationship with 'Gut Edema Disease'.

Myocarditis

Though myocarditis is a common lesion found in many systemic diseases primary condition is rare.

Non-suppurative myocarditis: This is found as a hematogenous infection in generalised septicemias, toxemias and bacteremias.

Cattle: pasteurellosis, Foot and Mouth Disease.

Dogs: extension from valvulitis: from infective focus somewhere else—streptococcus or coliform septicemia:hapatitis contagiosa canis; leptospiral infecions.

Horses : infectious anemia

Extension of infection may occur from pericarditis also.

The lesions of myocarditis are usually focal and consist of pale or yellowish or grayish areas. In acute Foot and Mouth Diesase in young calves with supervening death, there is myocardial degeneration and diffuse myocarditis. With necrophorus infection, gray areas of coagulative necrosis are found.

Microscopically, there is degeneration and necrosis of the muscle fibres in the acute stage with infiltration of lymphocytes, plasma cells, macrophages and eosinophils.

In the chronic stage, there is much fibrosis since the heart muscle does not regenerate and healing occurs by granulation tissue.

Acute suppurative myocarditis: This is found in pyemia that occurs in mastitis, metritis and joint ill. The spread is by way of the coronary arteries. Direct extension from purulent pericarditis, endocarditis, pleura, lungs and bronchial lymph glands is also possible. Infection may also occur through a foreign body penetrating the myocardium through the reticulum

Macroscopically, the heart reveals abscesses with hyperemic borders. Some abscesses may be encapsulated.

Microscopically, the typical appearances of an abscess are seen with neutrophils abounding. Healing may occur by organisation and scar formation. Sometimes abscesses may be calcified.

Specific inflammations: Tubercular myocarditis is common, especially hematogenous. Extension from neighbouring tissues is also possible. Actinomycosis of the myocardium is also met with.

Parasites of myocardium:

1. Heart worms *(Dirofilaria immitis)* are found in the right ventricle of dogs, sometimes completely filling the lumen. Depending on their number, these round worms may cause thrombosing endocarditis leading to cardiac dilatation and hypertophy or thrombosis of pulmonary arteries.

2. In the heart muscle are found the following:
   (a) *Sarcocystis tenella*. These are found universally in the hearts of cattle, sheep and pigs. The muscle fibres contain the Miescher's tubes in which are found the spores—Rainey's corpuscles.
   (b) *Toxoplasma gondii* may be present in the heart muscle fibres as pseudocysts. If these cysts rupture, a focal myocarditis develops. The lesion consists of a necrosed centre surrounded by inflammatory cells—neutrophils, histiocytes and lymphocytes. Sometimes the necrosed tissue may be calcified.
   (c) *Cysticercus bovis* in ox heart is the bladder worm of *Tenia saginata* of man.
   (d) *Cysticercus cellulosae* in pig heart is the bladder worm of *Tenia solium* of man.
   (e) *Cysticercus ovis* in the sheep is the bladder worm of *Tenia ovis* of dog and fox.
   (f) Hydatid cysts of *Echinococcus granulosus* of dog contain scolices and clear fluid.

Endocarditis

Inflammation of the endocardium is rather common in animals and is almost always caused by bacteria. Though the endocardium of all chambers and valves can be affected, it is the valvular endocardium that is mostly affected primarily. Form here, inflammation may extend to the mural endocardium.

Among animals, swine are more frequently affected.

Endocarditis is a constant lesion seen in chronic septicemic diseases, in which the circulating bacteria infect the endocardium The following organisms have been found to be causative.

Horse. *Streptococcus equi* (strangles), *Shigella equirulis* (umbilical infection) Migrating larvae of *Strongylus spp*, *Actinobacillus equili* and *Meningococcus spp*, (while hyperimmunizing with meningococci) are other causative agents.

Cattle : *Corynebacterium pyogenes* (secondary to a liver abscess or peritoneal abscess); Streptococci of intestinal origin (white scours); embolic infection from traumatic reticulitis, suppurative metritis and mastitis can occur.

Dog : Not usually observed but may exist in association with streptococcal infections of mouth, teeth and pharynx. Leptospirosis may also be associated with endocarditis.

Pig : *Erysipelothrix rhusiopathiae*, *Corynebacterium pyogenes* and *Streptococci*. Of these the first is most important.

Sites In the order of frequency of occurrence, these are :

In horse : Aortic valves: right auriculo-ventricular valve, left auriculo-ventricular valve and pulmonary valve.

In Cattle ; right auriculo–ventricular valve; left auriculo-ventricular valve; pulmonary valve and aortic valve.

In pig and dog : mitral valve, aortic valve; tricuspid valve and pulmonary valve.

The valves are more often affected because these are exposed to the circulating bacteria and to the force of blood during systole. Bacteria that are present in the blood get implanted on the valvular endocardium. The surface of the valve that is exposed to the force of blood is more often affected, viz, the auricular surface because when the blood is forced into the ventricle, it is this side that is continually brushed. Besides, it is the edges of the valve (lines of closure) which are most exposed to stress and trauma and so are more often affected. The implanted organisms grow on the injured endocardium. The toxic metabolic materials released by these bacteria damage the local cells, with the liberation of thromboplastin which converts fibrinogen into fibrin. Fibrin is a good medium for the bacteria to grow and so more and more of fibrin is formed due to the liberation of greater quantities of thromboplastin by the cells that are increasingly destroyed by the growing organisms. Thus a thrombus is formed. The enlarged valve (with the thrombus) injures the adjacent surface during its movement and during this motion the thrombi may break off and form emboli.

The lesion being chronic, the thrombus is formed slowly but progressively, This is friable and resembles the head of a cauliflower and so is called vegetation. Endocarditis in which these vegetations are present is called

PART II

Special Pathology

tative endocarditis. From the basal area of the valve granulation tissue invades the thrombus which is thus organised in the deeper layers. But so long as the organisms are alive, complete organisation and healing are not possible

In swine excessive vegetations are common. But in cattle due to C *pyogenes* excessive fibrin is not common but fibrosis is frequently seen Extension of infection from the tricuspid and b cuspid valves to the chordae tendinae makes them weak and degenerated, resulting in their rupture.

Mural endocarditis is only an extension of the inflammatory process from the valves. In cattle affected with black quarter, the wall of the left auricle is usually affected showing roughened endocardium.

In the dog. with uremia and leptospirosis, ulcerative endocarditis is common. Greenish ulcers are present in the left auricle and ventricle, pulmonary artery and aorta.

Microscopically, the lesion is a thrombus in the centre of which bacteria are seen, Leucocytes are present in the intima, Fibrosis in the chronic cases is a feature noticed.

Effects of Endocarditis : The vegetations which are formed at the points of contact of the cusps of the valves prevent the closure of the valves and obstruct the lumen, thereby hindering free passage of blood through the lumen. These conditions thus give rise to valvular insufficiency or stenosis. The effect of these conditions is to cause accumulation of blood in the chamber just preceding the lesion. The following sequelae are met with in lesions of different valves

Tricuspid valve : Dilatation of the right auricle, general venous congestion and ultimately hypertrophy of the right auricle.

Pulmonary valve: Dilatation of the right ventricle, general venous congestion and hypertrophy of the right ventricle.

Mitral valve : Dilatation of left auricle, pulmonary congestion, edema and pneumonia (brown induration), hypertrophy of the left auricle, hypertrophy of the right ventricle.

Aortic semilunar valves: Dilatation of the left ventricle; hypertrophy of the left ventricle and later general venous congestion The final outcome of these lesions is congestive heart failure. Fragments of the thrombus (vegetation) may be detached to form emboli.

The results of embolism are:

1. If the emboli are contaminated with pyogenic organisms, abscesses will be found in the kidneys (acute embolic nephritis). spleen and liver from emboli arising from left side; pulmonary abscesses from emboli arising from the right side and myocardial abscesses from emboli of coronary vessels.

2. If the emboli are bland and sterile, infarction will be caused in the kidneys and spleen from emboli arising from the left side and pulmonary thrombosis from emboli arising from the right side. Myocardial infarction may occur if emboli enter coronary circulation.

Endocardiosis in dogs:- Otherwise known as "Chronic valvular disease", in this condition the mitral valve is mostly affected. The affected valve is shrunken and distorted and so is incompetent producing gradually congestive heart failure.

The atrio-ventricular cusps are thickened, become shorter, the chordae tendinae are thickened and may sometimes be ruptured. The thickening is due to proliferation of fibroclastic tissue with abundant mucous ground substance.

MANCHESTER WASTING DISEASE OF CATTLE
(By Dr. M. S. Kwatra)

(Synonyms: Enteque seco, pasteur disease, Naalehu, Calcinosis, Calcific arteriosclerosis.)

The disease is characterized by progressive wasting, stiffness of forelegs and back and deposition of calcium in the arteries, heart muscle, lungs and kidneys. The condition has recently affected large number of Corriedale sheep and a few cattle in Ludhiana district of Punjab The disease is also found in Jamaica, Brazil and Hawaii and causes severe losses in livestock industry. The disease generally affects cattle over 15 months of age.

Symptoms: The affected animals waste away progressively; the joints of the limbs become stiff, and fore limbs are often severely affected. The animals cannot walk properly and sometimes put weight on the knees. As the disease advances, the animal is disinclined to move and stands with arched back for long periods. On auscultation heart murmurs may be heard. On exercise animals appear to be distressed Blood calcium level is increased in affected animals.

Cause: In different localities grazing on different plants containing excessive amounts of vitamin-D or toxic substances causing hypercalcemia results in the calcification of arteries, heart, pulmonary tissues and kidneys. *Solanum malaecoxylon Cestrum diurnum. Trisetum flavescens* and other plants have been found to contain toxic concentration of vit. D or some other toxic substances with similar action.

Gross Changes: The general condition of the animals is usually poor. The aorta and major arteries show plaque formation in the intimal layer. The walls of the affected vessels are hard, thick and brittle, particularly during the advanced stages of the disease In worst cases mitral valves, myocardium, lungs and kidneys may show calcium deposition. In mild cases the plaque formation is seen in the terminal portion of the aorta and in moderate cases it may extend upto the aortic arch. In severe cases whole of aorta, mesenteric,and carotid arteries are affected while in advanced cases the myocardium, kidneys and lungs also show calcific deposits. Hydropericardium, hydrothorax, hydroperitoneum may be present in cases in which the cardiac valves and lungs are involved.

Microscopic Changes; the medial layer of the aorta and arteries is intensely involved and muscular fibres show degenerative changes and deposits of calcium salts. The tunica intima shows slight proliferation of intimal connective tissue while the tunica adventitia remains, unaffected. The cardiac muscle fibres undergo calcification and degenerative changes. The coronary arteries may also reveal deposition of calcium in the tunica media and intima. In the kidneys the arterioles of the cortex and medulla are calcified. The calcification of the lungs is confined to the thickened interalveolar septa. The alveolar epithelium undergoes foetalization,

Neoplasms of the heart: primary: Not common, Rhabdomyosarcoma, fibroma and myx fibroma have been described.

Secondary: These are the commonest. The most common secondary neoplasm of the heart is the lymphosarcoma among cattle. Tumor masses may project into the pericardium or into the chambers. Diffuse infiltration of the myocardium is also seen and the neoplastic cells may be found between the muscle fibres, which undergo pressure atrophy.

DISEASES OF THE ARTERIES

Hypertrophy, Compensatory hypertrophy of the walls of the arteries is common and may affect one or all of its components. Mostly the muscle and elastic lamina are hypertrophied to withstand increased blood pressure, This is seen in renal vessels of dogs suffering from chronic interstitial nephritis and in lungs of cats

Arteriosclerosis: The exact meaning of arteriosclerosis is "hardening of the arteries" This condition is common in man, characterised by intimal thickening following proliferation of connective tissue, hyaline degeneration, infiltration of lipoids and finally calcification.

In animals there is no comparable lesion as occurring in man, The dog (with hypothyroidism) is most frequently affected among animals. Usually, the older animals are affected. One difference in the dog is that infiltration of lipoid is unusual.

In man, the lesion is described under the heads: atherosclerosis, Monckeberg's medial sclerosis and diffuse arteriolosclerosis.

Atherosclerosis: This condition affects the larger elastic arteries. Athere means in Greek a soft, mushy, gruel - like substance. In this condition such a substance is formed in the intimal layer.

The aorta and its primary branches are mostly affected though carebral and coronary vessels may sometimes be involved.

Pathogenesis: 1. The lesion commences as a focal degenerative change in the subendothelial tissue. The mucinous ground substance of intima is increased and in these foci fine fat droplets appear. These fatty deposits consist of cholesterol, neutral fats, fatty acids and cholesterol esters. Elastic fibres disintegrate.

2. Foam cells (macrophages laden with lipoids) are found in these foci It is conjectured that macrophages transport the fats to the lesions directly from the blood

3. These macrophages cluster in the subendothelial area.

4 Subsequently the macrophages die, disintegrate and liberate the fats into the tissue spaces.

5. The fats (cholesterol crystals especially) stimulate the proliferation of the connective tissue around these foci, especially towards the luminal side. This newly formed tissue becomes hyalinised.

6. This is the plaque (atheromatous plaque), with a central debris consisting of granular, lipid-rich material and acicular crystals of cholesterol. Hemorrhages and hemosiderin granules may also be seen,

7. A well-formed plaque is supplied with numerous capillaries, which are the source of hemorrhage.

8. A few lymphocytes may be found around the lesion.

9. Fibrous tissue increases in quantity and with further deposition of lipoids, the atheroma enlarges in size and reaches the endothelial layer, which may be pushed into the lumen.

10. Along with the changes noticed in the intima, degenerative changes are noticed in the media. Edema first occurs separating the muscle and the elastic fibres. In these foci collagen is formed and scarring occurs. The elastic fibres degenerate in the focal areas but proliferate in the adjacent areas.

11. The fate of plaque : (i) it may be converted into a dense inflammatory scar, containing cholesterol clefts; or (ii) it may be calcified or (iii) islands of heterotopic bone may be formed in it; or (iv) the thin endothelium may be necrosed due to the subendothelial accumulation of macrophages and fatty debris, resulting in ulceration into the lumen of the blood vessel—the atheromatous ulcer. A thrombus may subsequently form on this ulcer.

Causes of atherosclerosis: No definite cause is attributed. The following are some of the etiological factors suggested in man.

1. Senility or ageing process, in which there is progressive degeneration of the walls of arteries leading to fatty degeneration of the tissue in which lipids accumulate.
2. High blood cholesterol and lipid content.
3. Hypertension.
4. Intramural hemorrhages, which may be the starting points of the lesions.
5. Probably some endocrine deficiency.

Heredity, obesity, stress, physical activity and smoking habits may be contributory factors.

Atherosclerosis as occurring in man is found only in the pig among animals. Atherosclerosis encountered in the aorta of pigs was reported from India. Abdominal aorta was found to be more often affected.

Maroscopically, the surface of the aorta revealed fatty streaks. Frank fibrous plaques were not seen.

Microscopically, there was edematous swelling of the ground substance in the intima, deeper parts of which showed deposition of variable amounts of lipid. The number of smooth muscle cells increased and marked collagenization was observed in a few cases. It was thought that the initiation of fatty streaks in the pig was not mediated through the thrombotic mechanism, though in man microthrombi composed chiefly of platelets were supposed to be the primary initiating factor in the development of atherosclerotic lesions.

It has been mentioned earlier that among animals, atherosclerosis is more often seen in dogs, suffering from hypothyroidism with hypercholesterolemia. The pathogenesis is slightly different. The lesion commences in media (as against the intima in man and fowl) in the middle and outer layers of which lipoids are deposited. Foam cells appear here and the lipoid may be found within the muscle fibres which show hydropic degeneration. Due to the pressure of lipoids the fibrous connective tissue proliferates and replaces other structures. This becomes hyalinised and later impregnated with iron salts. In the adventitia lymphocytic infiltration is seen, sometimes to such an extent as to form nodules.

Along with the changes in the media the intimal elastic fibres are disrupted, destroyed and reduplicated. The endothelium shows hydropic degeneration and appears thickened. Later, there is fibroblastic proliferation that occurs around these thickened areas (plaques) of intima.

Macroscopically: the affected vessel is enlarged and less pliable and the walls are thickened. On opening the aorta, raised oval or round areas (plaques) may be seen. These are white or yellow and may be 0.1 m.m to 2 cms, in diameter.

Monckeberg's medial sclerosis : In this condition the medium sized muscular arteries are affected. The muscular tissue undergoes hyaline and fatty degeneration followed by necrosis and calcification. Sometimes heterotopic ossification occurs. This condition is found in older people but is not necessarily associated with hypertension. The disorder is considered to be related to prolonged vasotonic influences—prolonged action of epinephrine and nicotine. Hypervitaminosis D may also produce similar lesions. Medial calcification is seen in some dogs suffering from chronic interstitial nephritis.

The lesions are found only in the media and do not encroach on the intima and vessel lumen. The endothelium is intact.

Arteriolosclerosis : In this condition, there is thickening of the walls and narrowing of the lumina of small arteries and arterioles. Two types are recognised; (1) hyaline arteriolosclerosis and (2) hyperplastic arteriolosclerosis. Both forms result from hypertension. Hyaline arteriolosclerosis occurs in a slowly developing hypertension while the hyperplastic variety is a more acute condition developing due to a sudden elevation of blood pressure.

In tha hyaline arteriolosclerosis, there is homogeneous pink collagenous fibrosis and thickening of the walls of the arterioles. The cellular details of the tissue are completely lost. In the hyperplastic arteriolosclerosis an "onion skin" appearance is found due to the proliferation and concentric arrangement of the endothelial cells, subintimal fibroblasts and even the muscle cells of the media. Sometimes, there may be an admixture of the two forms. Being a reaction to increased blood pressure, arteriolosclerosis is met with in all the small arteries and arterioles, particularly those of kidney, spleen, pancreas, adrenal and small intestines.

In general, arteriolosclerosis in animals is not common and is not of clinical significance. Atheromatous plaques with calcification are frequent in the aorta of adult cattle, caused by *Onchocerca armillata* and found only at post-mortem. In dogs atheromatous ulcers may be found in infection by *Spirocerca lupi* in the migratory life cycle of which the larvae at one stage pass through the aorta.

Arteritis

The inflammation of the wall of arteries is arteritis, which may be acute or chronic.

Acute arteritis may be caused by parasites, bacteria, viruses or fungi. The routes of infection are (1) from the outside of the vessel, extending through the wall: (2) through the vasa vasorum and (3) from the lumen of the vessel.

Extension of inflammation from the adjacent tissues is common as in pneumonia, metritis and mastitis. Infection may also occur due to pyogenic bacteria, the primary lesion being elsewhere—umbilical abscess. In such cases the pulmonary vessels are the favoured places where emboli lodge and produce inflammation.

Inflammation of the intima results in the formation of a thrombus at the site—thromboendarteritis.

Equine viral arteritis : A primary acute arteritis in the horse is caused by a virus which cannot be grown on egg embryos or propagated in experimental animals. Serial passage through tissue culture attenuates it and such an attenuated virus confers strong immunity when vaccinated into horses. The clinical picture is:—fever, leucopenia, conjunctivitis, rhinitis with mucopurulent nasal discharges, palpebral edema, respiratory distress, depression, incoordination and edema of the limbs and abdominal wall, colic and diarrhoea, abortion in pregnant animals.

Macroscopically, petechial hemorrhages are seen in all serous membranes, in the lungs and gastric mucosa. Edema of the eyelids is prominent. All serous cavities contain excess of fluid with high protein content. The intestinal wall is thickened by edema, which is present in the mesentery and lungs also. Enteritis is seen.

Microscopically, the lesions are found in the media of smaller arteries. The muscle fibres are necrosed and replaced by a hyaline fibrinoid material. There is edema of the adventitia with lymphocytic infiltration. The intima and endothelium are usually intact showing no thrombosis. But the arteries of the intestines and lungs are severely affected resulting in thrombosis. Infarcts may occur in the mucosa of cecum and colon.

Chronic arteritis : This is exemplified by the arteritis of the anterior mesenteric artery in the horse due to *Strongylus vulgaris*, which by its presence causes chronic irritation. The artery is dilated, fibrosed and the wall loses its resiliency. The intimal surface becomes roughened where a thrombus forms. Due to loss of elastic tissue and continuous pressure of the blood, the wall dilates and an aneurysm forms. Sometimes, the aneurysm may rupture with fatal results.

Polyarteritis nodosa: (Also known as periarteritis nodosa) This is one of the group of collagen diseases.

There is inflammation involving all the layers of the wall. Bacteria, viruses and allergy to drugs and streptococci have been thought of to be of etiological significance. Among others, sulfonamides, arsenic, iodum and desoxycorticosterone acetate have been incriminated. In some animals sarcosporidia have been suspected. Probably this parasite serves as a long acting antigen. Lesions very similar to the naturally occurring condition in man have been produced in horses by sensitising the animals to foreign protein. Among animals periarteritis nodosa is met with in viral diseases—malignant catarrhal fever in cattle, in equine infectious anemia, and in sporadic encephalomyelitis in bovines.

Small and medium sized arteries located in general musculature, myocardium, subepicardial fat and mammary gland are involved. The inflammatory changes begin in the adventitia or media with edema in these places. Fibrinoid

necrosis of the media occurs converting it into an eosinophilic granular material. Necrosis is followed by infiltration of neutrophils and eosinophils into the adventitia mostly and to a lesser extent into the media. After the acute phase subsides the exudate is organised. At this time macrophages and lymphocytes infiltrate. Thrombosis may occlude the lumen. Due to weakening of the wall, small aneurysms form, giving a nodular appearance externally to the affected vessel.

Thromboanglitis obliterans or Buerger's disease : This condition usually occurs in the limbs and is met with more frequently in males of Jewish race, especially in smokers. There is acute inflammatory reaction involving all the layers of the wall, resulting in inflammatory thrombosis. There is proliferation of the endothelial cells and fibroblasts. Organisation of the thrombus and subsequent canalisation may occur.

Aneurysm : An aneurysm is a localised dilatation of an artery, vein or a cardiac chamber.

Causes : The main factor involved in the causation of the aneurysm is weakening of the wall. This weakening may arise due to damage of the media. The various causes that weaken the wall are :—

1. Syphilis in man. 2. Infected embolus may cause suppuration and destruction of the media. 3. Polyarteritis nodosa. 4. Trauma. 5. Parasites—*Strongylus vulgaris*. 6. Infection from an abscess or tuberculous lesion may weaken the wall. 7. Congenital weakness of the walls.

Varieties of aneurysms :

1. **True aneurysm** : In this variety the sac is formed by the wall of the artery.

2. **False aneurysm** : The sac in this condition is not formed by the wall of the artery but by the surrounding connective tissue. This occurs due to rupture of the vessel by trauma.

3. **Fusiform aneurysm** is one in which a long segment of the vessel is uniformly dilated around the whole circumferance. This is mostly seen in the aorta and its branches.

4. **Sacculer aneurysm** is the formation of a pouch on one side of the wall.

5. **Dissecting aneurysm** : Strictly speaking this is not a true aneurysm since there is no dilatation of the wall. In the aorta, hemorrhage occurs between the layers of media and blood circulates around within this space, dissecting the wall. Fatal hemorrhage may supervene. The condition is usually due to a degenerative lesion in the media.

6. **Cirsoid aneurysm**: These are a mass of dilated, pulsating and intercommunicating arteries and veins. usually subcutaneous in location and most are congenital. A few may be due to trauma.

7. **Arteriovenous aneurysm** : (aneurysmal varix) is an abnormal acquired communication between an artery and a vein due to simultaneous injury to both. There is pulsation in the vein since blood passes directly into it.

8. **Mycotic aneurysm** : This is due to infection by bacteria which weaken the wall, small aneurysms developing thereby. This is usually associated with vegetative endocarditis.

9. **Miliary aneurysm** This is aneurysm of minute arteries, and is usually seen in the cranium. This is congential and is also known as a *berry aneurysm.* This is a small saccular dilatation.

10. **Parasitic aneurysm** This is found in horses, in the anterior mesenteric artery due to infection by *Strongylus vulgaris.*

Sequelae.

1. Pressure atrophy of the structures around an expanding aneurysm may be seen.
2. Rupture of the aneurysm may occur with fatal results.
3. In the horse when the anterior mesenteric artery is affected colic may occur because of thrombosis of the artery or due to emboli that may emanate and occlude intestinal vessels.
4. Inflammation from the anterior mesenteric artery may spread to the neighbouring autonomic ganglia causing intestinal stasis resulting in colic.

Diseases of the veins

Phlebitis: Inflammation of the veins is usually septic in character. Infection may be;

1. In the new born animal, **umbilicus** may be infected—*Omphalophlebitis.* The usual organisms are *Shigella equirulis* in the foal and coliforms in the calves.

2. **By extension from adjacent inflammed areas** This is common in lungs (pneumonia), uterus (metritis) and the udder (mastitis). Infection passes through the thin walled veins more easily than through the thicker arterial walls.

3. **By venepuncture:** During intravenous injection if irritant chemicals are injected inadvertently outside the vein, periphlebitis and phlebitis will be set up.

4. **Foreign body**: In traumatic reticulitis, a foreign body may cause chronic phelebitis of the veins involved.

Macroscopically, the inflammed vein is enlarged, has a thickened wall with neutrophilic infiltration. Usually thrombosis develops rapidly—thrombophlebitis. An infected thrombus will get softened and disintegrated and thus septic emboli may be formed. A bland thrombus may become organised. Some times, the thrombus may be calcified—*phlebolith.* The importance of phlebitis lies in the danger of thrombosis with eventual emboli formation, causing pulmonary embolism, pyemia, septicemia or septic arthritis.

Varicose veins: Varicose veins are dilated and tortuous veins. These are not as common in animals as in man, whose leg veins and hemorrhoidal veins are most commonly affected. Stagnation of blood in the dilated vessels causes pain. In man the following causes are attributed.

11. **Exciting** causes are those that increase the pressure of blood in the veins. These may be found in the following situations.

a) Whenever there is hindrance to the return of venous blood—as in mitral stenosis, pulmonary emphysema and cirrhosis of liver; (b) Pressure on vein—by tumors, pregnant uterus, increased abdominal pressure as in straining. (c) Standing for a long time. (b) Muscular exertion as in athletes. (e) Ageing. (f) Post-inflammatory weakness of vessel wall

There may be muscular hypertrophy followed by atrophy. The elastic tissue is replaced by fibrous tissue, leading to dilatation and in these areas thrombosis may occur.

In animals the veins of limbs are not commonly affected, The affected are the scrotal plexuses in the horse and the supramammary veins in the cow.

Diseases of lymph vessels

Lymphangitis

Lymphangitis may be non-specific or specific.

Non-specific lymphangitis: The irritant may reach the lymph vessels in two ways: (i)by extension of the inflammation of the surrounding tissues through the walls of the vessel and (ii) transport by the tissue fluid through the lumen. Hence lymphangitis is common in those vessels that drain areas in inflammation.

Again non-specific lymphangitis may be **Simple lymphangitis,** which is most commonly seen in the lungs in various inflammatory diseases viz pneumonia in swine fever; contagious bovine pleuropnemonia; brochopneumonia in dogs and horses. The lesion in these diseases starts as bronchitis and the irritant spreads by peribronchial spread and enroute the peribronchial and perivascular lymph vessels are affected. These become very much dilated with leucocytic infiltration into their walls. Such vessels are easily seen on the surface of the lungs.

Inflammed lymphatics can be seen as thickened, reddish streaks and those of the subcuits appear as cords. These are painful to the touch. Occlusion of the lymphatics prevents drainage of lymph and so edema develops The nearest lymph gland that drains the area is inflammed, swollen and painful.

Purulent-lymphangitis is associated with suppurating condition of the tissue drained. There is intense infiltration by leucocytes of the wall of the vessels together with thrombosis.

Specific lymphangitis is seen as, tuberculous lymphangitis; farcy, ulcerative lymphangitis; epizootic lymphangitis; bovine lymphangitis, in John's disease and in actinomycosis. These are described under the respective diseases in the section, 'Pathology of Specific Diseases"

Tumors of vascular system

Hemangiomas and lymphangiomas have been described.

CHAPTER 15

THE HEMOPOIETIC SYSTEM

Development of blood cells
- Erythropoiesis
- Granulopoiesis

Terms used in describing anemias

Polycythemia

Oligocythemia

Dyshemopoietic anemias
- Porphyrinopathies
- Congenital porphyria
- Diminished stroma protein formation
- Diminished hemoglobin formation
- Toxic inhibition
- Aplastic anemia
- Myelophthisic anemia

Hemolytic anemias

Hemorrhagic anemias

Leucocytes
- Leucocytosis and neutrophilia
- Agranulocytosis
- Eosinophilia
- Lymphocytosis
- Leucopenia
- Leukemia

Diseases of Lymph nodes
- Atrophy
- Hypoplasia
- Necrosis
- Amyloid degeneration
- Hyperplasia
- Pigmentation
- Emphysema
- Inflammation-acute&chronic

Diseases of spleen
- Anomalies, Atrophy
- Hyperplasia
- Hyaline degeneration
- Amyloid infiltration
- Pigmentation
- Rupture
- Congestion
- Thrombosis and embolism
- Infarction
- Splenitis
- Splenomegaly
- Hypersplenism

DEVELOPMENT OF BLOOD CELLS

1. In the foetus : The primitive blood cells arise by proliferation of the endothelial cells lining the numerous blood islands. These are nucleated primitive erythroblasts, possessing basophilic cytoplasm, a large nucleus with a loose chromatin network and several *nucleoli*. These elaborate primitive hemeglobin. The intravascular formation of nucleated erythrocytes lasts upto 8 weeks in the human embryo..

2. The hepatic phase : From about the second month of foetal life, erythropoiesis occurs in the sinusoids of the liver and these remain active until a few weeks before birth. During this period, granulocytes begin to appear and by fourth month they are numerous.

Hemopoiesis also occurs in the spleen in which erythroblasts first make their appearance by the fourth month. However by the fourth month myelopoiesis no longer occurs in this organ. Erythropoiesis occurs till the end of the gestation period while lymphopoiesis takes place throughout life. Thymus, which is primarily a lymphopoietic organ, for a short period, produces erythroblasts.

Lymph nodes : Lymphopoiesis begins at fourth or fifth month and continues throughout life.

3. **The myeloid phase :** This begins approximately by the fifth month. In the beginning, granulopoiesis alone occurs (while liver is involved with erythropoiesis) but gradually, the bone marrow takes over the function of formation of blood cells.

The nucleated red cells, in the foetal blood gradually decrease in number and by the sixth month, none are present in the peripheral blood, which now contains only non-nucleated red cells.

Extramedullary hemopoiesis : This denotes formation of blood cells in organs other than the bone marrow. In times of need, eg. severe hemolitic anemia, the liver and to a slight degree the spleen, reassume their hemopoietic activity.

Hemopoiesis after birth : After birth, erythrocytes, granulocytes, monocytes and thrombocytes are formed in the bone marrow, while lymphocytes are formed in the bone marrow and the lymph nodes and spleen

ERYTHROPOIESIS :— Extravascular(Intravascular in poultry)

The following stages are noticed in the development of erythrocytes.

Undifferentiated stem cell
↓
Rubriblast (Pronormoblast)
↓
Prorubricyte (Basophilic normoblast)
↓
Rubricyte (polychromatic normoblast) } By mitotic division
↓
Metarubricyte (Acidophilic normoblast)
↓
Reticulocyte } No more division Only maturation
↓
Erythrocyte.

In the process of erythropoiesis the following changes, in general, take place in the cells from the stage of rubriblast to erythrocyte.

1. The cell size gradually decreases.
2. The nucleus becomes progressively less sponge-like but more condensed.
3. Nucleoli disappear.
4. The size of the nucleus also decreases.
5. During metarubricyte stage, the nucleus is extruded.
6. The cytoplasm gradually turns pink from an initial blue color as hemoglobin is gradually incorporated.

GRANULOPOIESIS :— Extravascular. (Intravascular in poultry)

Hemocytoblast
↓
Promyelocyte } By mitotic division
↓
Myelocyte
↓
Metamyelocyte—(Juvenile)
↓
Band form (stab) } No division but maturation
↓
Segmenter.

In the process of granulopoiesis, the following changes take place from the stage of promyeloblast to a segmented granulocyte:

1. The size of the cell becomes smaller.
2. The cytoplasm, which is intensely blue in the promyeloblast stage, becomes paler.
3. Granules begin to appear in the cytoplasm.
4. The nucleus which is large, becomes smaller and also becomes segmented.

Erythropoietin : For erythropoiesis, a humoral substance, the *erythropoietin*, appears to be of great importance. The granular cells of the juxtaglomerular apparatus appears to be an important source of erythropoietin, which is probably a glyco-protein, with a molecular weight between 60,000 and 70,000. It is present in plasma, urine and milk. Erythropoietin stimulates the differentiation of the bone marrow stem cells to rubriblast. It governs the rate of hemoglobin synthesis. Its secretion is controlled by oxygen content of renal arterial blood. *Hypoxia is a stimulus* for erythropoietin secretion. This is the reason for the polycythemia found in high altitude disease.

Anemia of chronic renal disease may mostly be due to decreased erythropoietin production by the damaged kidneys.

Androgens, adrenal corticoids, thyroxine and growth hormone stimulate erythropoiesis. The first three probably act directly by stimulating erythropoietin production, while the last has a direct effect on the marrow, stimulating erythropoiesis. Estrogens depress erythropoiesis, probably by competing with erythropoietin production or by competing with it in its action on stem cells.

Bone marrow : Macroscopical examination of bone marrow indicates:

(a) Hyperplasia :—denoting increased activity when it is red, cellular and opaque or

(b) Hypoplasia,—decreased activity when it is gelatinous and yellowish.

As the animal grows older, the red, active hemopoietic marrow is substituted by a yellow or white, fatty and inactive marrow. But in conditions of extreme necessity, this inactive yellow marrow can be converted into red, active marrow.

TERMS USED IN DESCRIBING ANEMIAS AND DISEASES OF THE BLOOD

Anisocytosis denotes variation in size of erythrocytes. In cattle blood slight anisocytosis is normal.

Annulocytes are erythrocytes that have a narrow rim of hemoglobin surrounding a large central pale area. These are also known as Pessary cells.

Basophilia—This indicates that the erythrocytes instead of taking a normal red stain take a bluish or pale-bluish stain. That means to say that ribonucleic acid, which takes a blue stain, is still retained. This condition denotes incomplete maturation and is met with in anemias. It also indicates lack or deficiency of hemoglobin.

Basophilic stippling or punctate basophilia. In this condition the erythrocyte contains blue staining granules scattered throughout—the remnants of

RNA. This is met with in conditions in which there is acute and intense erythrogenesis as in anaplasmosis of bovines and hemonchosis in sheep and in lead poisoning.

Cabot rings are bluish thread like rings in the erythrocytes and are nuclear remnants. These may be found in hemolytic and toxic anemias and are found in lead poisoning.

Crenation means abnormal notching of the erythrocytes These may be seen in delayed drying

Heinz bodies are refractile inclusions found in the erythrocytes of horses that undergo phenothiazine therapy. These are supposed to be associated with denatured protein and are seen in hemolytic anemias. Their presence indicates erythrocyte injury.

In man Heinz Bodies are noticed following treatment with primaquine, acetanilid. sulphanilamide, phenylhydrazine, phenacetin, sodium nitrite, sodium chlorate-para-amino-salicylic acid, nitrofurantin.

Heinz Bodies are not visible if the blood smear is fixed in methyl alcohol. They can, however, be seen in blood stained with supravital dyes like brilliant cresyl blue. A drop of blood is mixed with 3 or 4 drops of 5% of the dye in saline. The preparation is taken on a slide and ringed with paraffin, a cover slip is placed and then examined under oil immersion. The bodies are seen as blue bodies.

Howell-Jolly bodies are remnants of nuclear material and appear as single or double spherical bluish bodies situated eccentrically usually. Normal blood of cats and horses may contain upto one percent of erythrocytes with these bodies. They may also be seen normally in young pigs and dogs. In the bovine H-J bodies must be distinguished from Anaplasma marginale. The anaplasma is uniform in size while H-J bodies vary in size. These are seen in anemias and lead poisoning

Hyperchromasia (Hyperchromic erythrocytes) indicates intensity in staining of erythrocytes. This is not due to increased hemoglobin content but to increased thickness of the cells.

Hypochromasia (Hpyochromatic cells) indicates decreased intensity in staining of erythrocytes which may be due either to thinness of the cells or to decreased hemoglobin content

Leptocytes are thin erythrocytes with larger surface without increase in volume.

Macrocyte is an erythrocyte the diameter of which is larger than normal, having therefore, higher Mean Corpuscular Volume.

Megaloblast is an immature cell in the erythrocyte series comparable to prorubricyte stage seen in animals due to Vitamin B_{12} or folic acid deficiencies.

Meniscocytes (Drepanocytes) are crescent shaped erythrocytes characteristic of sickle cell anemia. This abnormal shape is due to the insolubility of hemoglobin S in its deoxygenated form Doubly refractile masses consisting of rodlike particles are formed which deform the erythrocytes.

Microcyte is an erythrocyte the diameter of which is smaller than normal

Normocyte } cells of normal size and staining
Normochromic } with normal intensity

Ovalocytes are elliptical erythrocytes. These are normal in the camel family. Some ovalocytes may be seen in advanced anemia with poikilocytosis.

Pappenheimer bodies(siderotic granules)are purplish coccoid granules seen at the periphery of erythrocytes in anemias due to impaired heme synthesis (sideroachrestic anemias). The granules contain iron in ferritin.

Poikilocytosis: denotes variation in shape.

Polychromasia: or **polychromatophilia** denotes the staining of the erythrocytes with many colors, red blue and intermediate color

Spherocytes: are not seen in animals. In man these occur as congential abnormality. These are dome shaped and are thicker than normal,

Target cell is one with a central rounded area of pigmented material surrounded by a clear ring without pigment outside of which is the pigmented border of the red cell (resembles bull's eye). These are more commonly seen in the dog's blood and probably are artifacts.

Polycythemia:

This is an increase in the circulating erythrocytes and the blood picture is normochromic and normocytic

Polycythemia may be

(a) **Relative:** There is reduction in the total blood volume and so increased concentration of normal number occurs whenever there is excessive fluid loss as in hemoconcentration in dehydration due to continued vomiting, diarrhoea, sweating, shock and collapse.

(b) **Absolute;** There is increase in the total number of red cells, while the blood volume remains normal.

i) Primary: — Polycythemia vera—a tumor of erythropoietic marrow, was reported among six dogs, a cat, a cow and 14 heifers (it was found to be familial in these heifers) Evythropietin levels are low.

ii) Secondary:— 1) Neonatal—cause obscure.
2) Physiological (a) Permanent—as in high altitudes
(b) Temporary:— Splenic contraction in sporting dogs and racing horses.

iii) Pathological.— Compensatory increase in prolonged anoxic states eg., cardiac and pulmonary disease. It was found in Tetrology of Fallot, encountered in dog, cat, cow and horses.

Increase in erythropoietin production causing polycythemia was found in man in the following conditions: neoplasm of the liver, cerebellar hemangioblastoma, pheochromocytoma; hydronephrosis; cysts, adenoma and carcinoma of kidney; adrenal adenoma and uterine fibroid.

Oligocythemia: This is decrease in the quantity of erythrocytes in peripheral blood

Anemia: is reduction in the quality and or number of erythrocytes below normal. Now the two terms are used synonymously.

Relative oligocythemia: There is increase in total blood volume (with

normal number of erythroeytes) and there is ultimately reduced red cell concentration; eg hemodilution.

Absolute: oligocythemia and anemia: In normal animals production of erythrocytes by hemopoiesis is equal to destruction. So the condition under 'Anemia' can be conveniently grouped as.

a) **Production low, but destruction normal**
Dyshemopoietic anemias

b) **Production normal but destruction excessive**
i) Hemolytic anemias ii) Hemorrhagic anemias

DYSHEMOPOIETIC ANEMIAS

In this are grouped all those types af anemias in which there is defect in the formation of erythrocytes The defect may lie in the formation of stroma protein or in the formation of Hb etc.. These are described as follows.

Porphyrinopathies : Porphyrins are necessary for the normal synthesis of heme. If certain enzymes are lacking, then heme is not synthesized and excessive amounts of porphyrins are found in the urine (porphyrinuria — urine turns red on exposure to light) and in the body — porphyria.

Congenital porphyria occurs in the bovines and pigs. This is a heritable disease due to simple Mendelian recessive gene. The pigment is photosensitive and so when deposited in the teeth (dentine) it takes a red color known as "pink tooth". In the bone it is responsible for the condition, "Osteohemochromatosis" In the kidneys, the pigment is deposited in the tubular epithelium and interstitial tissue. When found in urine, it imparts a red color after exposure to light. Affected animals suffer from photodynamic dermatitis if exposed to sun light— photosensitisation, but if protected from direct sun, no harm seems to occur.

The following deficiencies have been found to cause anemia After restitution of the deficient hematinic, regenerative forms appear. "Reticulocyte shower"

A. Diminished stroma protein formation: Blood picture is **macrocytic and normochromic or hypochromic.** Bone marrow is megaloblastic showing numerous megaloblasts and giant metamyelocytes.

a) Dietetic deficiency of Extrinsic factor: Cobalt—cyanocobalamin—Vit. B_{12}

Vit. B_{12} deficiency is not seen in animals other than ruminants. In ruminants this is synthesized by ruminal microoganisms provided cobalt is available. But in other animals, the vitamin is ingested as it is, and so deficiency is seen only in ruminants in areas where the pasture is deficient in cobalt. Vitamin B_{12} is necessary for the synthesis of RNA and DNA. B_{12} and Folic acid deficiency causes arrest of maturation of prorubricytes and metamyelocytes. These have larger nuclei than normal Depressed DNA synthesis causes delayed nuclear maturation but hemoglobin synthesis is not affected and so continues. When hemoglobin synthesis reaches a certain concentration in the erythrocytes, the nucleus leaves them and so macrocytes result giving rise to macrocytic anemia.

b) **Dietetic deficiency of Folic acid :** Folic acid is required for the maturation (especially the nuclei) of the erythroblasts. In its absence maturation is slowed down and so macrocytic anemia results.

c) **Deficiency of the intrinsic factor :** The intrinsic factor which is secreted by the gastric mucosa and which is supposed to be in the nature of an enzyme, helps in the absorption of the macromolecular Vit B_{12}. In its absence (gastric diseases) Vit B_{12} may not be absorbed and so will result in anemia.

d) **Failure to store the Erythrocyte Maturation Factor:** Erythrocyte maturation factor (Hematinic principle) is now known to be Vit. B_{12} which is normally stored in the liver. Hence is the efficiency of liver extracts in anemia. In diseases of liver the storage of this vitamin does not occur and so anemia results.

e) **Failure to use the Erythrocyte Maturation Factor:** The EMF may either be not utilised or may not be mobilised from the liver resulting in macrocytic type of anemia. This is known as *Achrestic anemia.* (Achrestic means failure to utilise). The bone marrow is megaloblastic and this differentiates it from aplastic anemia.

f) **Hypopituitarism:** Anterior pituitary seems to exert a potent influence in erythropoiesis, directly or through the Thyroid which influences metabolism of carbohydrates, releasing needed energy.

B. **Diminished hemoglobin formation:**

Blood picture is normocytic and hypochromic	Becoming microcytic and hypochromic

a) **Dietetic deficiency of iron:** can occur due to

i) **Deficient intake :** Milk of sows is poor in iron and so piglet anemia develops if rooting is prevented.

ii) **Defective absorption:** Excessive phosphorus and phytic acid, form insoluble complexes of iron, which are excreted through the feces.

iii) **Increased requirement :** In young, growing animal and pregnancy.

b) **Dietetic deficiency of copper :** Copper acts as a catalyst in the utilisation of iron in hemoglobin formation.

The deficiency is seen in piglets, cattle and sheep. Essentially this is an iron deficiency anemia. In iron dificiency, decreased hemoglobin synthesis leads to retention of the nucleus beyond normal number of cell divisions. Some cells undergo additional mitosis and so microcytic erythrocytes result in iront deficiency.

c) **Dietetic deficiency of ascorbic acid:** Vit. C is dietary reducing agent and so facilitates the reduction of Fe^{+++} to Fe^{++} state which is easily absorbed. Vit. C is also required for the synthesis of folic. acid and for its conversion into the more active folinic acid.

d) **Dietetic deficiency of pyridoxine:** Pyridoxine is required for the utilisation of iron in hemoglobin synthesis. So deficiency of pyridoxine results ultimately, in anemia resembling iron deficiency.

e) **Dietetic deficiency of Nicotinic acid:** Nicotinic acid is concerned in the synthesis of pyridine nucleotide which takes part in cell respiration. So, deficiency of Nicotinic acid interfers with the respiration of immature red cells. This is noticed in dogs and pigs

f) **Dietetic deficiency of Riboflavin:** Riboflavin is concerned in the metabolism and arrangement of amino acids of the protein of hemoglobin molecule and so is useful in hemoglobin synthesis. This condition is met with in dogs.

g) **Deficiency of Thyroxine:** Thyroxine along with vitamin C is required for the conversion of folic acid to folinic acid. Thyroxine is necessary for the metabolism of carbohydrates and fats (So required for energy production).

In Myxedema, the secretion of intrinsic factor is depressed (and so absorption of vitamin B_{12} is poor). So a normocytic or macrocytic anemia may be encountered.

C. Toxic inhibition Here the marrow appears to be normal and active but is unable to utilise the hematinics.

Blood—Normochromic: Microcytic.
No regenerative forms

Examples:

i) **Chemical Poisons:** Nitrogen mustard (which is cytotoxic).
Folic acid antagonists—antimetabolites—6 mercaptopurine etc.
Streptomycin, chloromycetin—Antibiotics and sulphonamides.
Metals—Bismuth, Arsenic and Gold (by injection).
Others—Benzol, hair dyes, insecticides.

ii) **Chronic interstitial Nephritis:** In advanced cases there is uremia which suppresses erythropoietic cells. Probably erythropoietin is not produced in the kidneys in this condition.

iii) **Oesophagastomiasis:** This worm causing pimply gut depresses absorption and so may cause various deficiencies, resulting in anemia.

iv) **Chronic infections:** In chronic infections like Tuberculosis, Brucellosis and Rheumatic fever (in man) a normocytic normochromic anemia is noticed. In these conditions there appears to be some abnormalities in hemoglobin synthesis for there is increased excretion of coproporphyrins. There is hypoferremia with reduction in serum iron binding capacity. Along with these, hypercupremia is noticed.

Absence of regenerative forms indicates impaired erythropoiesis. It is suggested that during infections and inflammation there is great demand for iron by the tissues affected and so it is side tracked to these areas instead of to the bone marrow and so hemoglobin is not formed and anemia results.

v) **Ionising radiation:** The hemopoietic system is highly sensitive to radiations, the leucopoietic being the most. After exposure there is lymphopenia with the spleen and lymphoid tissue becoming soft and shrunken. Decrease in granulocytes is much more sooner than development of anemia since granulocytes are short lived. Hemorrhages occur due to thrombocytopenia and damage to the vascular endothelium.

D. Aplastic anemia: This occurs due to aplasia of bone marrow, wherein there is utter inactivity. The anemia seen is **normochromic and normocytic**. No regenerative forms are present.

Aplastic anemia may be divided into.

i) **Primary or idiopathic :** rather rare.

B) Secondary:

a) Exhaustion: *Due to chronic hemorrhages*—Gastric and intestinal ulcers (rare in animals; Blood sucking worms, Neoplasms; Deficiency of vit. C. K, and prothrombin.

b) Toxic: Ionising radiation, Chemical poisoning—same as those detailed under the heading "Toxic inhibition" but in a higher dose and exposed for a longer duration.

c) Metabolic: Another form of aplastic anemia occurs in baby pigs that are born of sows which suffer from protein malnutrition during pregnancy. This can be prevented by feeding sows, during pregnancy, high protein diet containing Vitamin B_{12}, folic acid and iron. Once the symptoms are seen in the baby pigs, no treatment is of any avail.

E. **Myelophthisic anemia:** There is replacement of bone marrow by other tissues. Since in this disease immature forms of granulocytes are found in the peripheral blood, it is also known as leuco-erythroblastic anemia. This condition is found in:

1. Secondary metastasis of other tumors—lymphatic leukemia in dog and cat.
2. Osteodystrophies—where the myeloid tissue is replaced by connective tissue and.
3. Primary tumors of the reticulo-endothelial system—Nieman-Pick Disease; Hodgkin's Disease etc.

HEMOLYTIC ANEMIAS

In this condition intravascular destruction of erythrocytes occurs. Anemia is normochromic and macocytic becoming hypochromic and microcytic as the iron stores are used up. Many regenerative forms are seen. Bone marrow is active usually in this type of anemias, while erythrocytes show increased hypotonic fragility and spherocytosis

We have already studied the normal breakdown of hemoglobin. Hemoglobin breaks up into heme and globin. The iron of the heme is stored by the RE cells for future use. The pigment part is excreted as cholebilirubin and urobilinogen. The protein moiety is broken down in the liver into amino acids which are used again in the synthesis of hemoglobin. In some hemolytic anemias their breakdown of hemoglobin occurs at a faster rate. So there is

a) Jaundice—with increased bile pigments in the blood, feces and urine and
b) increased stroage of iron in the form of hemosiderin crystals.

In other types of hemolytic anemias, there may be hemoglobinuria.

Causes of the hemolytic anemias may be classed as:

1. **Abnormal auto-antibodies** the presence of which may be
   a. Primary or idiopathic, or
   b Secondary due to
      i) Malignant disease—lymphatic neoplasms; ovarian tumors, gastro-intestinal carcinoma.
      ii) Collagen diseases—disseminated lupus erythematosis.
      iii) Viral diseases – infectious mononucleosis

In this condition spherocytes, spontaneous agglutination of erythrocytes and hemoglobinuria may be found.

2. Abnormal iso-antibodies: Due to the presence of hemolysins in the plasma. produced by (a) incompatible blood transfusion (b) injection of blood products (c) pregnancy—blood group antigens of the foetus pass to the dam which does not possess these antigens Icterus neonatorum that develops in such a condition has already been studied. (Page 179)

3. Toxic (Partly also toxic dyshemopoietic)

A. Chemicals: i) Copper poisoning This condition is seen only in sheep in which excess of copper released suddenly in stress, produces hemolysis resulting in jaundice and hemoglobinuria.

Copper is a poorly excreted element and so if there is continuous ingestion of unduly large amounts of copper liver becomes loaded with this element. Such poisoning can occur by

a) ingestion of fodder treated with copper-containing insecticides and fungicides.

b) too heavy a dose of water containing copper sulphate given as a preventive and curative for stomach worms.

c) ingestion of large quantities of salt lick containing $CuSO_4$.

d) eating forage contaminated by copper from mines and dumps.

e) too much of supplemental mineral mixture containing copper sulphate.

f) eatinng forage that contains large quantities of copper due to soil having greater concentration of this element,

Stress can be brought about by: 1. Transport 2. Starvation after good feeding. 3. Excessive exercise, especially if unaccustomed. 4. Sudden stoppage of food. 5. Drenching 6 Exposure to cold 7 Loss in body weight.

Lesions seen are icterus, yellow and friable liver (which may be shrunken in later stages), distended urinary bladder(excretion of blood-colored urine). smooth kidneys, which are dark brown in color and a dark colored swollen spleen. Hemoglobinuria and dyspnoea are noticed clinically.

ii) Onion poisoning Occasionally, fatalities occur in cattle and sheep fed onions, in regions where they are extensively grown. The toxic principle is n-propyl disulphide. The symptoms are hemolytic anemia with hemoglobinuria and icterus. The carcass smells of onions.

iii) Poisoning by castor seeds: Ricin in castor seeds produces hemolysis and so ingestion of large quantities of castor results in hemolytic anemia.

iv) Phenothiazine poisoining (Drug sensitivity) : Phenothiazine, a good anthelmintic, sometimes even in therapeutic doses, has been found to be hemolytic. especially in horses. Cattle are also susceptible though to a lesser extent than horses. The symptoms are hemolytic anemia with hemoglobinuria Other lesions are hepatitis and nephritis.

v) Naphthalene used as moth balls may be accidetally ingested by pet animals and hemolytic anemia results.

iv) Lead also may produce acute hemolytic anemia.

vii) Hypersensitivity to certain drugs like sulphanilamide, quinine, paraminosalicylic acid and some anti-pyretic drugs may result in hemolytic anemia.

viii) **Snake venoms :** Snake venoms contain a lecithinase which acting on lecithin converts it into lyolecithin which is highly hemolytic.

B. Post-parturient hemoglobinuria : Also called – post parturient hemoglobinemia. This is found only in dairy animals, usually after parturition. This condition is associated with hypophosphatemia and so in such animals, signs of phosphorus deficiency are seen besides anemia : eg. pica, shifting lameness, decreased productivity, lordosis (curvature of the spinal column).

Lesions include i) a pale, slightly enlarged liver having centrilobular necrosis due probably to thrombosis of portal vein capillaries by "ghost corpuscles". ii) Black colored kidneys—due to deposition of Hb. iii) Dropsy of serous cavities. iv) Ecchymosis. v) Edema of lungs.

C. Infections : hemolysis occurs in infection by :

a) Protozoa—Anaplasmosis, Babesiosis, Hemobartonellosis ; Eperythrozoonoses, **Ehrlichia canis.**

b) Bacteria—Leptospirosis, Clostridia; Streptococci and Staphylococci.

c) Viruses – Equine infectious anemia; feline infectious anemia.

D. Hypersplenism is found in some dogs in which there is severe anemia, macrocytic or normocytic in type. Lesions include splenomegaly and icterus.

E. Cold hemoglobinuria In calves. Ingestion of excessive quanities of cold water by calves (rarely in older cattle) resulted in a mild disease, characterised by intravenous hemolysis and hemoglobinuria, associated with cardiac insufficiency and pulmonary edema. Spontaneous recovery usually occurred.

HEMORRHAGIC ANEMIAS

In this condition extravascular destruction of erythrocytes occurs. We have noted, that normally there is a balance between blood production and blood loss. But in hemorrhagic anemia blood loss is greater than production. The bone marrow can rally round to meet the situation only if necessary basic ingredients, most important of which is iron, are available in sufficient quantity.

In cases where there is a balance between blood loss and production, the picture is one of normocytic, normochromic anemia with many regenerative forms. In cases where this balance is maintained with difficulty, i. e., where the bone marrow is working at a fast rate, macrocytes will be found. In due course as iron stores become depleted, the picture turns to one of microcytic hypochromic anemia, with numerous regenerative forms. Ultimately, when the bone marrow becomes exhausted and is no more able to cope up (aplastic stage) a normochromic and normocytic anemic picture is seen but without any regenerative forms.

Therefore 1. the amount of blood lost. 2 the rate at which the blood is lost and 3. the diet controlling the balance between blood loss and production determine the nature and type of anemia that develops.

The various types are : A: Acute hemorrhagic anemia due to injury; sweet clover poisoning; Warfarin poisoning; bracken fern poisoning.

Sometimes, in scarcity and famine conditions, horses and cattle may ingest, large quantities of bracken fern when poisoning may occur. In this condition there is an acute thrombocytopenia which is the direct cause of hemorrhages.

At necropsy one finds hemorrhages in the gastro-intestinal tract under the mucosa (resulting in ulcers), in the myocardium, in the liver and kidney.

Bracken fern is a cumulative poison and there may be terminal bacteremia due to granulocytopenia, resulting in bacterial embolism, hemorrhages and infarcts of heart and kidneys. Bracken fern contains thiaminase which destroys thiamine (Vit. B_1) and so in single stomached animals it may produce thiamine deficiency. But in ruminants which can synthesise thiamine this is not a problem. There is some other unknown factor responsible for the hemorrhage in them. Acute hemorrhagic anemia may be seen in ulceration of stomach in pigs, bleeding abomasal ulcers in cattle, cocidiosis in poultry; bovine enzootic hematuria; hemonchosis and anylostomiasis.

B. Chronic hemorrhagic anemia

Due to blood sucking worms; Hemonchus, Fasciola, Bunostomum, in cattle and sheep; Strongyles in horses, Ancylostomes in dogs.

Ectoparasites—ticks, lice and fleas

Protozoa—Coccidiosis in dogs

Hemorrhagic diseases—Chronic bovine hematuria

In gastrointestinal ulcers and vascular tumors.

C. Purpura and hemorrahagic diseases

Purpura is accumulation of blood, under the skin due to spontaneous rupture of the capillaries. Hemorrhages result even due to mild damage.

Purpura is a syndrome but not a disease. The causes may broadly be divided under;

1. **Vascular disorders;**

i. Purpuric infections : Symptomatic purpura. This is found in various diseases characterised by petechial hemorrhages, eg. Hemorrhagic septicemia, Anthrax etc. Cause is injury to the vessels—capillaries and venules—by the toxins. In viremic diseases the endothelium is directly damaged due to the multiplication of the virus in the endothelial cells eg. Infectious canine hepatitis and hog cholera

ii. Allergic purpura or purpura hemorragica : This is a symptom of post-infectious toxemia as in Strangles. It is also seen in fistulous withers, poll evil and emphysema of guttural pouches. Besides the petechial hemorrhages noticed on the mucous membranes, edema of subcutis, peritoneal cavity and muscles is also seen. In this condition there is no thrombocytopenia. The defect appears to be injury to the vascular endothelium due to development of an allergy, resulting in increased capillary permeability.

iii. Congenital purpura : Purpura may develop in the foetus. The mechanism is suggested to be similar to the one found in erythroblastosis foetalis. Iso-agglutinins formed against platelets in the mother pass into the foetus via placenta and produce thrombocytopenia.

iv. Senile purpura : This is not seen in animals but is sometimes seen in old men and in very under-nourished people. The vessels of the skin are easily injured as there is no subcutaneous fat and the skin is very much atrophied.

v. Vitamin C deficiency : In avitaminosis C, there is no thrombocytopenia. Hemorrhages occur due to increased capillary permeability and capillary

fragility since cement substance of capillary wall is not synthesized. However this condition may not be met with in animals since vitmin C is synthesized in their gut.

II. **Impaired Clotting mechanism:**

A) Thrombocytopenia:

a) **Idiopathic or primary thrombocytopenia;** cause is unknown. Probably auto-antibodies against platelets are present.

b) **Secondary Thrombocytopenia:**

1. **Damage to the bone marrow:**

ly Chemicals— i) Nitrogen mustard, benzol, urethane antimetabolites

ii) **Individual sensitivity** to therapeutic doses of—sulphanilamide quinine, gold salts, Oxytetracycline, Streptomycin, P. A. S, soda salicylate , ergot organic hair dyes, D. D. T. etc.,

iii. **Animal toxins**—snake venom, extensive burns.

iv) **By Physical agents** —ionising radiation, heat stroke.

v) **Infections:** in septicemias, occasionally.

2) **Myelophthisic replacement;** In Leukemias.

3) **Hypersplenism**—destruction of thrombocytes.

4) **Aplastic Anemias**—In this condition due to causes already described there is complete atrophy of the bone marrow and so there is no production of platelets.

5) **Bracken fern poisoning;**—already dealt with

B **Other Coagulating defects**

i) **Hemophilia** is a condition in which coagulation of blood does not occur after an injury and so in some cases ends fatally. It is an inherited defect.

Two types of hemophilia, A and B, are recongnised. Type A is due to absence or reduction of anti-hemophilic globulin (A H G). In this, coagulation time is prolonged. This is sex-linked, conditioned by a recessive gene and the defect is evident only in the males passing through the females. The condition is met with in dogs and swine But in swine it does not appear to be sex-linked So it is found in both sexes.

Type B (found in man) is due to deficiency of the Christmas factor or factor IX. It is also sex-linked

ii) **Prothrombin deficiency:**

This is mostly due to impaired formation.

1) **Liver diseases**—In hepatic-disease the following, that are necessary for clotting mechanism are not synthesized; fibrinogen, factor V, prothrombin, factor VII and factor IX. Deficiency of bile that may occur in hepatic disease may lead to deficiency of vitamin K.

2) **Deficiency of Vit K.** Animals usually do not suffer from vit. K. deficiency as it is synthesized in the intestines. Only fowls with short intestines may be affected. Pigs medicated with sulpha drugs and antibiotics may also suffer as micro-organisms are eradicated by these, and Vit. K synthesis stops.

3) **Impaired absorption of Vitamin K ;** For absorption of vitamin K, bile salts are necessary. So if there is deficiency of bile due to hepatic disease or

as in obstructive jaundice, vitamin K cannot be absorbed and so prothrombin and factor VII cannot be synthesized.

Similarly, in diseases of intestines, absorption of vit. K may be interfered with eg. as in colitis, sprue etc.

4) **Poisoning by dicoumarin and Warfarin: Sweet clover disease:** Sweet clover contains coumarin, which is converted into dicoumarol (which is 3.3 methylene-bis-4 hydroxycoumarin) This is a powerful anticoagulant. It probably antagonises the activity of vit. K and so depresses the formation of prothrombin, factor VII, factor IX and factor X. Poisoning occurs among cattle and sheep in which extensive hemorrhages are seen—under the subcutis, on the serous membranes and in the viscera. Anemia results.

Swine may also suffer but horses appear to be refractory, probably because the detoxicating activity against dicoumarol is well developed.

Warfarin, which is chemically similar to dicoumarol is used as a rodenticide and so may accidentally be eaten by pets. Its action is similar to dicoumarol and causes extensive hemorrhages.

iii) **Presence of Circulating anticoagulants,** a) Heparin is a powerful anticoagulant, producing this effect by preventing the conversion of prothrombin into thrombin. Heparin is produced by mast cells. In anaphylactic shock in dogs, large amounts of heparin are liberated resulting in bleeding.

b) Some snake venoms are also anticoagulants and so bites by such snakes may result in fatal bleeding.

C. **Unknown Etiology:—**

i) **Mouldy corn poisoning in cattle and swine:** Corn spoiled by mouldy growth, if consumed by cattle and pigs, produces among other conditions, acute hemorrhages in various parts of the body, together with necrosis of the hepatic parenchyma and renal epithelium. Abortion in pregnant cows may be noticed. Lesions include centrilobular necrosis, cloudy swelling and fatty degeneration of the renal tubular epithelium with glomerular atrophy and necrosis of some tubular epithelium.

ii) **Epistaxis in horses:** In some families of horses bleeding from nose occurs during strenous exercise. It is due to a non sex linked recessive character. The walls of blood vessels are very thin and so rupture whenever distended during great exertion (as in racing)

SECONDARY EFFECTS OF ANEMIA

The secondary effects and symptoms noticed in anemia are mainly the results of anoxia, which leads to:- 1. Hyperplasia of hemopoietic tissues, evidenced by the regenerative forms, which are larger and more fragile and less efficient than the normal erythrocytes.

Extra medullary hemopoiesis may be observed in the liver and spleen,

2. Dyspnoea and tachycardia.
3. Fatty degeneration of the parenchymatous organs.
4. Rapid fatigue, due to incomplete metabolism.
5. Compensatory hypertrophy of the heart, in the early stages. If decompensation sets in, C. V. C. and resultant hydropericardium and ascties may occur.

6. Edema—due to damage to capillary endothelium, which becomes more permeable.
7. Petechiae.

Pallor of the skin, glossitis, anorexia, flatulence, constipation, diarrhoea, vomiting, albuminuria, fever and splenomegaly are other symptoms, noticed in anemia.

LEUCOCYTES

Increase in number : This may be due to :-

Leucocytosis : which is a temporary phase, useful to the animal and reversible.

Laukemia : A cancer of the leucopoietic tissue and so is progressive, irreversible and fatal.

Decrease in the number of circulating leucocytes is called *Leucopenia*.

Leucocytosis : Neutrophilia is increase in the number of neutrophils in the peripheral blood. When bone marrow is stimulated, immature neutrophils may be found in the peripheral blood, in greater numbers than normal. This phenomenon is called "shift to the left". To measure this shift to the left Schilling has proposed his hemogram in which the four following stages of neutrophils are estimated.

1. *Myelocytes*.
2. *Juveniles* in which the nucleus is indented.
3. The *band* form or *stab* in which the nucleus is curved or bent and
4. The *segmenter*. The first 3 are immature forms.

This shift to the left is of two types.

a) **Regenerative reaction :** in which because of increased activity of the bone marrow immature forms are a little more in number than mature forms.

b) **Degenerative reaction :** in which immature forms are far more in number than the mature ones. In this condition there is a depression in the maturation of leucocytes in the bone marrow and this denotes a very severe infection with unfavourable prognosis. In severe infections, neutrophils contain large toxic granules called "Dohle's Bodies". Dohle's Bodies are aggregates of rough endoplasmic reticulum. Some neutrophils may show vacuoles in the cytoplasm. These are supposed to be due to leakage of hydrolytic enzymes released from ruptured lysosomes under the influence of bacterial toxins

Causes of Neutrophilia : Physiological : may be found in new-born animals, in pregnancy, during exercise and with high protein diets.

Pathological 1. **Acute infections :** especially by cocci and also by leptospira, psittacosis organism, poliomyelitis virus, small pox virus, *E. coli* and *Actinomyces bovis*.

2. **Metabolic :** as in uremia, diabetic coma, burns.
3. Poisoning by
   Chemicals – lead, digitalis, mercury
   Organic—foreign protein; epinephrine
4. Acute hemorrhages and hemolysis
5. After surgical operations

6. Malignant diseases—leukemia and any other rapidly growing tumor. Ruptured immature neutrophils are called "basket cells".

Agranulocytosis is a condition in which there is almost complete disappearance of the granulocyte series of leucocytes from the peripheral blood. There will, therefore, be concomitant leucopenia.

The causes are either total suppression of leucopoiesis or inhibition of maturation of the granular series in the bone marrow. The causes include :

(1) toxic chemicals and drugs acting on the leucopoietic tissue—eg. benzol, arsenical preparations, barbiturates, amydopyrine.

(2) bacterial toxins—toxins of *Staphylococcus aureus, Streptococcus hemolitticus, Streptococcus viridans.*

(3) X-ray irradiation.

Myelopoiesis is totally destroyed in the Viral Feline Panleucopenia and so agranulocytosis is a symptom of that disease.

Usually there is hyperplasia of the stem cells of the bone marrow in the early stages. In chronic and prolonged cases myeloid hypoplasia supervenes.

Eosinophile leucocytosis or eosinophilia

This is seen in ; 1. Allergic diseases—asthma, hay fever, serum sickness.
2. Parasitic infections—trichinosis, helminthiasis
3. Skin affections—eczema, scabies
4. Following recovery from acute diseases
5. Chronic eosinophilic myositis of dogs
6. Following splenectomy
7. Following administration of certain poisons and drugs: arsenic, copper, sulphur drugs, chlorpromazine, digitalis
8. In certain diseases of hemopoietic system: chronic myelocytic leukemia, Hodgkin's disease
9. Following mild irradiation

Lymphocytosis

Usually absolute increase in lymphocytes is rare though relative increase is common,

Causes : 1. Certain viral infections—Mumps, influenza
2. Bacterial infections: usually chronic infections—Brucellosis, Tuberculosis
3. Thyrotoxicosis
4. Lymphatic leukemia
5. In convalescence
6. Adreno-cortical insufficiency
7. Following vaccination

Lymphocytes that are damaged during preparation of smear are called "smudge" cells.

Monocytosis

1. In protozoal diseases : Trypanosomiasis, Malaria, Kalaazar
2. During convalescence following acute diseases.
3. Rickettsial affections : Typhus

4. Hodgkin's disease
5. Chronic bacterial diseases; Tuberculosis, Brucellosis
6. Monocytic leukemia

Leucopenia This term denotes reduction in the number of leucocytes in the peripheral blood. Usually all leucocytes are affected equally.

Causes:

A. Diminished production: 1. Bracken fern poisoning—already discussed,

2. Viral diseases- Rinderpest, Distemper, Infectious Canine Hepatitis, Mucosal Disease
3. Certain bacterial infections—Brucellosis, Typhoid, Tuberculosis
4. Protozoal infections - Kalaazar -
5. Fungal diseases—Histoplasmosis
6. Ricketisial diseases—Tick-borne fever
7. Cachectic states and starvation
8. Metabolic disturbances as in hypothyroidism and hypopituitarism in which there is general lowering of body metabolism and so activity of bone marrow is decreased.
9. Chemical and physical agents that produce hypoplasia of the marrow Eg. benzol, ionising radiation, X-rays, urethane, nitrogen mustard, etc. described earlier under anemias.
10. Hemopoietic disorders: Anemias—Aplastic—Myelophthisic.
11. Of unknown cause cirrhosis of liver, primary splenic neutropenia.

B. Increased destruction: By 1. Physical agents—large doses of ionising radiation
2. Loss of leucocytes in large numbers in pus and in inflammatory exudate.
3. By bacterial toxins—toxins of *Clostridium welchii* and Pasteurella as in pasteurella pneumonia of sheep.
4. By protozoa—*Theileria parva* (East-coast fever)
5. Destruction by leucocytic antibodies, especially when amidopyrene is administered antibodies are formed which destroy leucocytes in the presence of the drug.
6. Hypersplenism:- found in certain diseases, like tuberculosis, Hodgkin's disease—cured by splenectomy.

C. Altered distribution. 1. In anaphylactic shock the leucocytes are trapped in the sinuses of the liver, spleen, and lungs.
2. In stress, liberated cortisone of the adrenal cortex produces eosinopenia and lymphopenia.

LEUKEMIA

This is a primary neoplastic disease of the bone-marrow and other reticulo-endothelial tissues. Comparatively leukemia is not so common in animals as in man

Again in animals, the neoplastic condition affecting lymphopoietic tissue is more common. The neoplastic cells often flood the blood, when it becomes *eukemic* At other times, when the blood picture is relatively normal the condition is known as *aleukemic leukemia*.

Neoplasia of granulopoietic tisssue is called *granulocytic leukemia* while if the lymphocytic type is involved it is known as *lymphatic leukemia*. Similarly, *monocytic leukemia* is also met with. Granulocytic leukemia is further divisible into neutrophilic, eosinophilic, and basophilic depending upon the type of cell involved.

In the circulating blood immature forms of leucocytes or "blast" cells are frequently encountered It is quite often impossible to determine to which type of cell this 'blast' cell is a precursor. And so by examining a blood smear alone it may sometimes be difficult to determine the type of leukemia. Examination of bone marrow smears and the presence or otherwise of hepatomegaly, splenomegaly and involvement of lymph nodes help in the diagnosis. If the lymph nodes are not affected but if the bone marrow smear reveals presence of numerous myeloblasts, and if there is, in addition, hepatomegaly and splenomegaly, often a diagnosis of myeloid leukemia can be arrived at. On the other hand, a quiescent bone marrow with involvement of all the lymph nodes is indication of lymphatic leukemia

Leukemia is always fatal.

In animals lymphocytic leukemia is variously known as lymphocytoma, malignant lymphoma, lymphomatosis or lymphosarcoma. The last named term is usually applied to the condition wherein neoplastic masses of cells are found internally as in other sarcomas. Involvement of all the lymph nodes suggests a multicentric origin. Metastases are found in almost all the organs in which the normal structure may be completely changed and replaced.

Other infrequent conditions seen *are the reticulum cell sarcoma and the giant follicular lymphoma arising from the reticulum cells.*

Incidence of different types of leukemias in animals :

Cattle : Lymphatic leukemia is the tumor most commonly met with in bovines next to squamous cell carcinoma. Tumor masses are found in almost all the organs and all the lymph nodes are invariably enlarged. Metastatic tumor tissue is found more frequently in the liver, heart, walls of the abomasum, omasum, reticulum, uterus and ureter.

Blood picture may reveal anemia. In the blood leukemia will be moderate with immature blast cells. Neutrophilia may be present. The affected lymph nodes show central necrosis,which may incite sterile inflammatory reaction.

A solitary case of granulocytic leukemia was reported in a four month old calf.

Symptoms : Enlargement of the superficial lymph glands—prescapular, sub-maxillary and precrural—is the first symptom noticed followed by pallor of the mucous membranes, laboured breathing and a gradual loss in general condition. Digestive symptoms may be exhibited if the liver and abomasum are involved. Paraplegia is found if the spinal cord or brain are affected.

Sheep : Compared to bovines, incidence of leukemia is not as frequent. Among the different types lymphocytoma is more common and all the viscera are affected. Subcutaneous lesions are more common in this species.

Swine : Lymphomatosis is common in swine, deposits occurring even in long bones. Reticulum cell sarcoma resembling Hodgkin's Disease is seen.

Horses: In the horse also generalised lymphomatosis is found though not very frequently.

Dog: Mostly lymphocytic leukemia is seen. A few cases of granulocytic and monocytic leukemias are described. In the lymphocytic variety, generalised lymphadenopathy is met with. Splenomegaly and hepatomegaly are often observed. Leukemic stage is usually absent, and so the blood examination is often misleading. Moderate neutrophilia is constantly observed. Anemia is common. Biopsy is very useful in diagnosis. Hodgkin's disease has been reported among dogs,

Cat: Lymphomatosis is common in the cat and appears as a generalised condition. Lymphadenopathy is not a feature.

Plasma cell myeloma

This tumor found rarely in man and known as Ewing's tumor, is of rarer occurrence in animals. This is found mostly in the bone marrow and some times in other organs. Multiple involvement of bone may result in fractures. Blood picture may or may not show the neoplastic cells. Normal peripheral blood does not have plasma cells. Persons suffering from this tumor excrete Bence-Jones protein in their urine.

DISEASES OF THE LYMPH NODES

Fowls do not have any lymph nodes

Function: 1. Production of lymphocytes.

2. The reticulo-endothelial system of the lymph nodes have the following functions·

a) Phagocytosis of foreign particles and worn out blood cells.

b) Rarely, extramedullary hemopoiesis in severe anemia

c) Probably production of normal and antibody globulin by plasma cells

Atrophy: Atrophy of lymph nodes is associated with

a) Some viral infections

b) Ionising radiation

c) Excessive doses of adrenal cortical hormones and sex hormones (lymphopenia also occurs) "Alarm reaction"

d) Senility

e) Starvation and

f) Chronic wasting diseases

Hypoplasia of lymph nodes is caused (together with degenerative changes) by infection and toxic agents or hormonal mechanism. In "Alarm reaction" of stress diffuse dissolution of lymphocytes is seen.

Necrosis: Necrosis of the whole or a part of a lymph node may occur when infectious agents grow locally, In anthrax and erysipelas, necrosis of the lymph node draining the affected area occurs.

Macroscopically, the necrotic areas are dry, and circumscribed. In some infections, gas bubbles may be present.

Amyloid degeneration; In general amyloidosis, amyloid may be found in the lymph nodes. Deposition of amyloid starts in the germinal centres and spreads outwards.

Hyperplasia of the lymph nodes is an usual reaction to subacute or chronic type of irritants and is met with either as a general or a local phenomenon in such diseases like canine distemper, chronic enteritis or chronic pneumonia.

Macroscopically, the affected nodes are enlarged, whitish-gray and firm but not fibrosed or calcified. Follicles are prominent.

Microscopically there is great enlargement of the germinal centres with a zone of mature lymphocytes surrounding them.

If the underlying disease is removed, hyperplasia subsides.

Pigmentation

Exogenous: Exogenous pigmentation of the lymph nodes is most common in the pulmonary and mesenteric nodes.

Anthracosis: Coal dust in the bronchial nodes is common in animals especially dogs that live in industrial areas and pit ponies. The coal particles are found in the macrophages of the medullary cords.

In ruminants mesenteric and other nodes develop a grey exogenous pigmentation of the medulla probably due to some pigments ingested with feed. In tatooed animals, the granules of the pigment used for tatooing are found in the regional lymph nodes.

These exogenous pigmentations are not of clinical importance.

Endogenous pigmentation: Hemosiderin is the most common endogenous pigment and is found in lymph nodes draining areas where hemorrhage has occurred.

Macroscopically, such nodes are brownish in color.

Microscopically, brown amorphous crystals of hemosiderin are found in the reticular and sinusoidal macrophages.

Bile pigments may be found in the hepatic lymph nodes. Melanin is found in the superficial lymph nodes of old grey horses.

Emppysema: In association with intestinal emphysema of pig, emphysema of the mesenteric lymph nodes is seen.

Emphysema of the bronchial nodes is common among cattle suffering from pulmonary interstitial emphysema.

Macroscopically, the nodes are enlarged, soft and puffy. The cut surface looks like a sponge.

Microscopically, vesicles are found in sinuses and the sinus endothelial cells become macrophages and even giant cells. These cells occurring as clusters cause pressure atrophy of the lymphoid tissue.

Circulatory disturbances; Hemorrhages are seen in lymph nodes in severe infectious diseases, hemorrhagic diathesis, local trauma and passive venous congestion.

Macroscopically, reddened areas are noticed, which may be diffuse, focal or even petechial

Hemolymph nodes must not be confused with hemorrhagic lesions.

Inflammation.

Lymphadenitis is inflammation of the lymph nodes. This may be non-specific, local or general. Functioning as a filter, the lymph node naturally is

affected by any irritant that may be present in the area it drains. The following are the irritants that may cause non specific lymphadenitis:

(a) Irritant chemicals, (b) soluble toxins from trauma and burns, and (c) bacteria. Depending on the nature of the exudate, lymphadenitis may be acute, serous, hemorrhagic, suppurative or chronic.

Acute serous lymphadenitis : This condition is common in the nodes draining lymph from acutely infected or inflammed areas. In some septicemic diseases the nodes throughout the body may be affected eg. anthrax, pasteurellosis, swine erysipelas, hog cholera; salmon disease Mesenteric nodes may be affected by the absorption of irritants from the gastro intestinal tract.

Macroscopically, the affected node is enlarged, moist and reddened.

Microscopically, hyperemia and edema are noticed. Due to proliferation of the lymphatic parenchyma and reticulo endothelial tissue, the lymph sinuses are filled with lymphocytes, mononuclears (derived from the RE, system) plasma cells and a few neutrophils

Hemorrhagic lymphadenitis occurs when the irritant is stronger than in the serous variety. The best example is anthrax The exudate in the gland is mixed with blood. Microscopically lymph sinuses contain large number of erythrocytes.

Suppurative lymphadenitis: Pyogenic bacteria cause suppurative lymphadenitis. The common organisms producing this are : *Streptococcus equi* in horses (strangles) *Corynebacterium ovis (caseous lymphadenitis* in sheep).

Macroscopically pus may be found in the nodes

Microscopically, the predominant cell of the greatly infiltrating leucocytes is the neutrophile. There is necrosis and liquefaction of the parenchyma and several small purulent foci may be present which may coalesce to form a big abscess.

Chronic lymphadenitis: the affected nodes are large, hard and dry. This is seen in Johne's disease (mesenteric lymph nodes),

Microscopically, there is hyperplasia of the R.E. system with numerous endothelial cells becoming rounded, swollen and cast off into the lymph sinuses that are much distended. To this picture is given the name of "*sinus catarrh*" Macrophages predominate, Reactive hyperplasia of the lymph nodules is also present. Fibrosis that occurs is the cause of hardness.

Specific lymphadenitis :

Lymphadenitis is a characteristic lesion of the following diseases.

(a) Tuberculosis
(b) Glanders
(c) Actinobacillosis
(d) Johne's disease

} granulomatous lymphadenitis with caseation and calcification.

(e) Salmon poisoning in dogs
(f) Strangles in horses
(g) Caseous lymphadenitis in sheep
(h) Bovine lymphangitis and lymphadenitis caused by *Pasteurella pseudotuberculosis rodentium.*
(i) Brucellosis in guinea pigs
(j) Tularemia in rodents

(k) Epizootic lymphangitis.

(l) Helminthic larvae -Pentastoma and other helminthic larvae in mesenteric lymph nodes of cattle; lungworm larvae in the bronchial nodes.

Neoplasms — Primary benign tumors of lymph nodes are not common. But primary malignant neoplasms—lymphosarcoma—are common.

Secondary tumors that are common in the lymph nodes are: carcinoma, malignant melanoma and occasionally sarcoma, which invade the lymh vessels.

DISEASES OF THE SPLEEN

Functions of spleen : 1 Production of lymphocytes.

2. Through the reticulo-endothelial system.
   (a) Phagocytosis of foreign particles.
   (b) Phagocytosis of effete erythrocytes.
   (c) Conversion of hemoglobin to bilirubin and storage of iron.
   (d) Extra-medullary hemopoiesis.
   (e) Production of antibodies by plasma cells.

3. Blood vascular system. The spleen is a "great reticulo-endothelial sponge" (Boyd) and so holds a large amount of blood. The speed of blood flow is controlled by the presence of sphincters, muscular trabeculae and a muscular contractile capsule. Blood cells can be "sequestrated" in the red pulp, so that the macrophages can act and destroy them

Anomalies : Accessory spleens are acquired and are found scattered in the gastrosplenic omentum. These are implanted pieces, produced by traumatic rupture of the spleen.

Doughnut spleen is a circular spleen with a hole in the middle, rarely seen in the horse. Sometimes a primitive lobulated spleen may be encountered.

Aplasia and hypoplasia of spleen may sometimes be met with.

Atrophy of the lymphoid tissue is similar to that seen in the lymph nodes, (causes are also similar).

In swine, cats and dogs, there is a form of atrophy of spleen resulting from induration due to chronic stasis of blood The spleen is much reduced in size and the capsule is shrivelled. The parenchyma is scanty and Malpighian corpuscles are not visible.

Hyperplasia : Focal hyperplasia of the spleen is common in old dogs and is characterised by round, soft and grey projecting nodules—*nodular hyperplasia.* These nodules comprise of newly formed hyperplastic lymph follicles, which do not contain the central arteriole.

Hyaline degeneration is seen in the walls of the arterioles.

Amyloid infiltration occurs as a part of generalised amloidosis. Two varieties are recognised:

(1) Focal: "the sago-spleen" in which the central arteries of the Malpighian corpuscles are affected. The involved foci are prominent, pale and translucent standing out against a red back ground, like boiled sago.

(2) Diffuse : "the Bacon spleen" in which the arterioles and fibres of the reticulo endothelial system are affected. The organ is enlarged with rounded edges and the cut surface is smooth and translucent.

Of these the focal is more common.

Pigmentation: Hemosiderosis is not of much consequence, unless in excessive amounts, since normally hemosiderin is stored in the spleen in the reticular macrophages. On destruction of larger number of erythrocytes, as in hemolytic anemias,there may be increased amounts of this pigment in the spleen.

Rupture of the spleen is common in dogs due to automobile accidents or due to sharp blows on the abdomen. Such rupture may divide the spleen into two. Healing may take place and scars be visible postmortem.

Splenomegaly due to congestion, amyloid disease. hyperplasia or tumors may predispose the spleen to traumatic rupture. Hemorrhages into the peritoneum may occur with fatal results.

Circulatory disturbances; Due to wide variations in size, congestion of the spleen is difficult to interpret.

Acute Congestion. This is common in acute infectious diseases and in acute bacterial intoxications as in enterotoxemia.

Acute passive Congestion: In euthanasia of pet animals by barbiturates acute congestion of the spleen is noticed since the barbiturates relax the smooth muscle and when the smooth muscle of the splenic trabeculae and capsule is relaxed blood fills into the organ.

Paralysis of the splanchnic nerve results in relaxation of the splenic musculature and hence passive congestion results.

In cardiac failure, acute congestion of the spleen may be noticed as a part of general venous congestion.

Macroscopically, the spleen is very much enlarged and soft. On section the cut surface bulges and dark blood oozes.

Chronic passive hyperemia: This is not common among animals. The cause are;

1. Partial or complete obstruction of venous return

a) Thrombosis or pressure by cysts, tumors and abscesses on veins draining the spleen.

b) Torsion of the stomach and spleen in dogs.

c) Torsion of the splenic ligament in the pigs.

2. Cirrhosis of liver, leading to congestion of portal vein.
3. Lesions of heart and lungs giving rise to general chronic venous congestion.

Macroscopically, the spleen is moderately enlarged and firm due to increased fibrous tissue in the pulp and in the trabeculae.

Microscopically. there is progessive induration of reticular stroma and trabeculae. The pulp cells and follicles are gradually replaced. Hemosiderin accumulates in the phagocytes.

Thrombosis: Thrombosis of the splenic veins is rare but occasionally met with in the following conditions;

In cattle; traumatic reticulitis and portal thrombosis,

In horses; extension of infections from parasitic abscesses.

Embolism may involve splenic artery and its branches. Emboli originate from the valvular vegetations and cause infarction.

Infarction of the spleen is common. If the splenic artery is occluded by an embolus the whole organ may undergo infarction.

In hog cholera, occlusion of the follicular branches of the splenic artery by proliferated endothelial cells results in hemorrhagic infarcts. The base of the infarct is red in the beginning but later turns pale with the diffusion of hemoglobin.

Hemorrhages;— In dog, due to automobile accidents, if rupture does not occur, blood may collect under the capsule and form hematomas.

Splenitis ; The inflammation of the spleen may be acute or chronic.

Acute Splenitis is a common feature of acute generalised infectious diseases such as salmonellosis, anaplasmosis, infectious anemia of horses, eperythrozoonosis and swine erysipelas. The infective organisms grow in the spleen.

Macroscopically the spleen is enlarged dark and soft. The pulp is fluid If infection is by pyogenic organisms, abscesses may be found.

Microscopically, necrosis of the pulp and neutrophilic infiltration may be seen in the sinusoids which are congested Certain amount of proliferation of lymphocytes is present and there is reaction of germinal centres. In the red pulp proliferation of the reticular cells and macrophages may be found. Along with the above changes, plasma cells proliferate (for production of antibodies)

Chronic splenitis occurs in such chronic diseases as tuberculosis. glanders, actinomycosis. pyemia pseudotuberculosis of sheep and histoplasmosis. The spleen is enlarged, firm and tough. Abscesses or granulomatous foci that are specific features of the diseases are seen.

Splenomegaly : Enlargement of the spleen is found in many different kinds of diseases. Since spleen is an organ of ant body production, the reason for such enlargement is quite understandable. There is marked hyperplasia of the reticuloendothelial system as well as of the white pulp with diffuse infiltration by neutrophils. The following are some of the diseases in which enlargement of spleen may be noticed It should be noted that absence of splenomegaly does not rule out the presence of the diseases.

Horses: Equine infectious anemia; metastatic melanoma; Tuberculosis; salmonellosis, Anthrax.

Cattle; Anthrax; Salmonellosis; Babesiosis; Anaplasmosis; Theileriasis Lymphocytoma; Acute congestion in bacteremic and toxemic conditions.

Pigs; Erysipelas; Salmonellosis; Eperythrozoonosis; acute congestion; Torsion.

Dogs ; While using barbiturates and chloroform; Histoplasmois; hemangiomas and hemangiosarcomas, lymphomatosis, myelogenous leukemia.

Fowls; Spirochetosis; lymphoid leucosis.

Splenomegaly is also seen in the following conditions.

a) in congestive heart failure,the spleen is enlarged due to stasis of blood and resultant fibrosis. b) when bone marrow is destroyed extramedullary hemopoiesis occurs in spleen. known as myeloid metaplasia with resultant splenomegaly (c) in conditions in which the histiocytes of the liver are saturated the e by

stimulating the splenic histiocytes to undergo hyperplasia and (d) in hepatic fibrosis in which condition the antigens from the intestines that are not detoxicated enter the general circulation and thence to spleen in which plasma cell production is stimulated for antibody manufacture.

Hypersplenism : This is a pathological condition in which there is excessive activity of the phagocytes. This condition may exist with or without splenomegaly.

In this condition there is excessive hemolysis (resulting in hemolytic jaundice), leucocytosis (resulting in leucopenia) and thrombocytolysis (resulting in thrombocytopenia and so purpura). The causes may be: 1. Depression of marrow function or inhibition of the maturation of cells of marrow by spleen through some hormonal influence.

2 Hypersequestration in which there is increased stasis in the sinuses leading to increased fragility of the erythrocytes (consequent on loss of plasma and erythrocyte potassium). Such red cells are easily phagocytised.

3. Antibody formation against the erythrocytes, leucocytes and thrombocytes leading to destruction of these elements.

Splenectomy relieves the clinical picture and the blood cells increase in number to the normal levels.

Tumors : Primary neoplasms of the spleen are rare. Fibrosarcomas, lymphosarcomas, myeloid leukemia, Hodgkin's disease; follicular lymphoma (splenoma), cavernous angioma, reticulum-cell sarcoma, leiomyosarcomas and hemangiosarcomas may be seen. Of these the hemangiogenous tumors are more frequent.

Secondary metastases are not common since spleen is a "poor soil" for the growth of the tumors.

THE RESPIRATORY SYSTEM

The main function of the respiratory system is the exchange of oxygen and carbon-dioxide between the blood and environmental air. As such to satisfactorily. discharge this function, respiratory system depends on the work of the heart. Diseases of the circulatory system are often accompanied by some abnormalities of the respiratory system.

Anomalies : ***Cleft palate or palatoschisis*** is a fairly common defect seen in the new-born animals. In this condition there is an abnormal connection between the nasal cavity and the mouth and hence milk passes into the lungs. So the animals do not survive long, dying of pneumonia and starvation.

NOSE

Congestion : Congestion occurs whenever animals are exposed to cold air. The blood vessels in the nasal passage dilate so that the air breathed in may be sufficiently warmed Secondary bacterial infection may result in inflammation and edema.

Epistaxis is hemorrhage from the nasal cavity.

Causes : 1. Trauma; 2. Convulsive expiration; 3. Parasites—*Oestrus ovis*; 4. Erosion of the vessels by pathological processes in the nasal cavity—Neoplasms; 5. Compression of the jugular veins by too tight collars in the working horses; 6. During certain infectious diseases Eg. Glanders, Anthrax, Purpura; Infectious bovine rhinotracheitis; Malignant catarrhal fever, Septic metritis; 7, Idiopathic—familial in certain race horses; 8. Neoplasms—hemangioma; 9. Uremia; 10. Poisoning by nitrates, bracken, sweet clover or mercurials

Acute rhinitis or coryza or acute nasal catrrrb

This is acute inflammation of the mucous membrane of the nose. In man this is the common 'cold'.

Causes : 1. **Irritants :**

(a) **Physical :** Dust, foreign bodies like chaff, pollen.

(b) **Chemical :** Irritating gases and smoke.

2 **Parasites :** *Linguatula serrata* in dog, larvae of *Oestrus ovis* in sheep.

3. **Fungi :** *Aspergillus fumigatus.*

4 **Bacteria :** *Sphaerophorus necrophorus, Bordetella bronchisepticus, Pseudomonas aeruginosa, Streptococci, Staphylococci.* May be found in the course of infections by *Mallcomyces mallei* (Glanders), *Pasteurella, M. tuberculosis, A bovis, A. lignieresi* and *Cryptococcus.*

5. **Viruses :** May be found in infections by viruses of :

a. Rinderpest c Equine influenza e. Laryngotracheitis (fowl)

b. Swine Influenza d Canine distemper f Fowl pox

Macrosopically, the mucous membrane is swollen and congested. Dry at first, a mucous discharge occurs subsequently, which turns mucopurulent later.

Microscopically, hyperemia, inflammatory exudate with inflammatory cells and hydropic degeneration of the epithelial cells (goblet cells) are seen. Extension to sinuses results in sinusitis.

Infectious sinusitis of turkeys is of economical importance to the turkey raisers Caused by a virus, there is an acute inflammation of the mucosa, resulting in a thick mucous exudate The infra orbital sinus is more often affected. Usually there is closure of the opening of the sinus cavity into the nasal cavity thereby preventing escape of the exudate

Chronic rhinitis : This is usually a sequel of the acute variety. There is ulceration of the mucosa, which in some places, may be thickened and congested.

Atrophic rhinitis of swine

This disease, especially of weanlings, is infectious. A virus was incriminated to be the cause, while Trichomonads were secondary invaders. At different times it was thought that *Pasteurella multocida, Hemophilus influenza suis, Fusiformis necrophorus* and a *Mycoplasma hyorhinitis* (P.P.L.O.) were of etiological importance. Though osteitis fibrosa (general osteodystrophy) is produced in swine with lesions and symptoms similar to those of Atrophic Rhinitis by feeding with calcium deficient diets producing secondary hyperparathyroidism thereby, characteristic lesions of Atrophic Rhinitis with atrophy of turbinate-bones could be produced only in those pigs exposed to nasal installation of nasal washing from natural cases Though a specific organism could not be attributed to be the cause of the disease, yet it is proved that it is infectious in nature and not just a specific effect of diet.

The disease starts with slight catarrh and swelling of nasal mucosa due to irritation. Progressive affection results in dyspnoea, anorexia and finally death due to inanition occurs. The disease is chronic and animals grow rather poorly. Lameness and fractures are often encountered. In many animals there is

deformity of the snout which bends to a side or upwards due to excessive bone resorption around the skull sutures.

Deviation of the snout is due to uneven resorption around the skull sutures. So there should be growth on one side while resorption occurs on the other so that the snout bends on the side of resorption due to growth pressure.

In the early stages there are foci of congestion of turbinate bones, which may be depressed. In 2 to 4 weeks, the turbinate bones are absorbed leaving a strip of hard tissue When the nose is sawn transversely in front of the second upper premolar, a characteristic appearance is noticed. Normally the turbinate bones completely fill the cavity at this level. But in atrophic rhinitis, a large cavity is seen. A mucopurulent exudate forms, which is discharged through the nose. Intermittent epistaxis may be present. The parathyroids may be larger in size.

Microscopically, there is rarefaction and disappearance of the turbinates. This is of the nature of osteolysis rather than osteoclasia No evidence of osteoclastic activity is present. The nasal septum also disappears leaving a fibrous band. Infiltration by lymphocytes and a few neutrophils is seen. It is postulated that "inflammation of the nasal mucosa is not a cause of atrophic rhinitis". In some places there may be metaplasia of the epithelium of the nasal mucosa to squamous type. The lamina propria is diffusely infiltrated with mononuclears There is hyperplasia of the parathyroids with increased secretion of parathyroid hormone, which is responsible for the bone lesions.

The condition can be prevented by a dietary calcium level of 1.20 per cent and a phosphorus level of 1.00 percent. Due to increased calcium intake, zinc in a concentration of 100 parts per million was recommedded to be fed to pigs.

Rhinohyperplasia or Bullnose is a chronic suppurative inflammation of young pigs, said to be caused by *Spherophorus necrophorus*, but other organisms such as *Streptococci, Pseudomonas aeruginosa, Staphylococci* and *Corynebacterium pyogenes* have also been incriminated.

Clinically, the nose is enlarged posterior to the cartilaginous rim

Lesions include. caseous or suppurative necrosis involving the bones of the nasal cavity, its lining and the skin adjacent to the nares. Being a slowly developing condition, fibrosis is predominant and this with edema is responsible for the swelling noticed. Extension of the inflammation to the sinuses and regional lymph nodes (lymphadenitis) is observed. Exaudate obstructing the nasal passage and distortion of the maxillary and nasal bones are responsible for the dyspnoea seen.

Nasal granuloma—see under "Diseases caused by Helminths".

Rhinosporidiosis : *Rhinosporidium seeberi* produces a chronic rhinitis in animals, characterised by the formation of polypi—some sessile while others are pedunculated and cauliflower-like. These are soft and jelly-like to the touch, covered by the nasal epithelium.

Microscopically, the fungus is present in loose fibrous or myxomatous tissue. The lesion bleeds easily. A few lymphocytes and epithelioid cells may be seen. (See under "Diseases caused by Fungi")

Tumors in the nasal cavity; Nasal polypi are soft moist masses. These have already been studied, (vide page 227)

The following tumors are encountered.

Benign	Malignant
Osteoma	Osteosarcoma
Chondroma	Chondrosarcoma
Myxoma	Myxosarcoma
Fibroma	Adenocarcinoma
Angioma	Squamous cell carcinoma

One interesting tumor is the adenocarcinoma arising from the glands of ethmoid and olfactory mucosa, which occurs in epidemic form in some countries Horses and cattle are affected. The tumors are highly malignant and destructive and metastasise rapidly.

DISEASES OF THE LARYNY AND TRACHEA

Roaring or laryngeal hemiplegia; In normal health the arytenoid cartilages are drawn outwards during inspiration to allow ingress of air. The important muscle that operates this is the *Cricoarytenoideus*. If for any reason there is injury to and degeneration of the nerve supplying this muscle, then the cartilage cannot open and so will stand in the way of air passing freely into the wind pipe.

In horses a condition is noticed, in which there is hyaline degeneration and fibrosis of the left Cricoarytenoideus muscle together with demyelinization and Wallerian degeneration of the left recurrent laryngeal nerve that supplies the muscle.

The cause for the paralysis of the nerve is obscure. One theory is that it is subjected to repeated trauma by the pulsation in the aorta as the nerve circles round aortic arch where the nerve is situated during its course.

Other causes are lead poisoning and pressure on the nerve by aneurysms, enlarged lymph nodes, abscesses, tumors, oesophageal divericula and other traumatic conditions.

When the nerve is paralysed, and the muscle is degenarated and replaced by fibrous tissue, the arytenoid cartilage does not open out during inspiration and so air cannot enter the treachea freely and this condition is accentuated when the animal is exercised, and a noise is heard by brushing of air with the arytenoid cartilage. This is therefore known as *Roaring*.

Laryngitis

A *mild* catarrhal laryngitis is met with which may progress to chronic form if the cause perisists.

Cause; Usually an extension of infection from the nasal cavity or the pharynx in infectious diseases. Eg. Distemper in dogs; Strangles and influenza in horses; Infectious laryngo-tracheitis in poultry; (Bacteria and viruses)

2. Irritant vapours, chemical irritants.
3. Mechanical injury,—kicks, bites, grass awns; injuries while passing probang or stomach tube.
4. Excessive barking, clergyman's throat.

5. Specific diseases; Tuberculosis, glanders and actinomycosis.

Macroscopically, the lesion is a swelling of the mucous membrane of the larynx and trachea, which is hemorrhagic and dry at first, later becoming coated with a mucoid exudate that may turn mucopurulent.

Croupous or membranous laryngitis ; The laryngeal mucosa is coated with greyish fibrinous deposits as in *laryngotracheitis of fowls* A membrane consisting of fibrin and leucocytes is formed over the necrotic mucosa. Death in fowls is due to asphyxia

BRONCHI

Bronchostenosis is a narrowing of the bronchial lumen due to obstruction or peripheral pressure

Cause : 1. Aspiration of foreign bodies; 2 Accumulation of exudate; 3) Parasites within the lumen. 4. Inflammation of the wall of the bronchus producing alteration in it—the exudate infiltrating the wall reduces the diameter of the bronchi. 5. Pressure from outside the bronchial wall – abscesses, tumors, enlarged lymph nodes and exudate of the pleural cavities; 6 Spasm of the muscles of bronchi – as in allergy (Asthma)

A partial closure of the bronchi or bronchioles results in balooning of the lung involved, since air that enters during inspiration is not expelled during expiration and so is trapped. Repeated inspirations will therefore result in balooing of the alveoli.

Complete obstruction of a bronchus results in collapse of the lung.

BRONCHIECTASIS

This is dilatation of the bronchus.

Causes: 1. This usually follows a chronic inflammation of the bronchi, in which the elastic tissue; the musculature and even the cartilages may be destroyed. Due to loss of the elastic tissue, contractile power of the bronchus is lost and so the bronchus dilates. At the place of dilatation, exudate accumulates thereby still further dilating the bronchus.

2. In chronic pneumonia, the contraction of the fibrous tissue exerts a pull thereby widening the walls. The dilatation is facilitated by a weakening of the wall in bronchitis, which, may also be found..

3. In bronchostenosis, air accumulates during inspirations and so causes dilatations of the bronchi below the level of obstruction When the bronchi are completely closed resulting in atelectasis, there is an elastic pull on the bronchial wall due to negative pressure in the pleural cavity.

Macroscopically, two forms are recognised A.-The saccular, which is less common, is an out-pouching of the bronchial wall; usually results due to localised necrotising foci in bronchitis, found in cattle and sheep in lungworm infection.

B. The cylindrical variety, which is more common, especially in cattle, is a uniform dilatation of the bronchi.

Macroscopically, the wall of the affected bronchi shows variable infiltration by chronic inflammatory tissue. The musculature, cartilage and the lining epithelium may disappear in varying degrees.

The affected lung is collapsed and is carnified. There may usually be pleural adhesion.

Results: The course is chronic and unfavourable. These animals cough persistently and become debilitated. Complications include abscesses with metastases, bronchiolitis with emphysema, bronchopneumonia, and secondary amyloidosis.

ACUTE TRACHEO-BRONCHITIS

This condition is usually encountered along with upper respiratory diseases More often it is a condition seen in pneumonias.

Though bronchitis means inflammation of the bronchial epithelium, frequently the inflammation spreads to the wall of the bronchus and from there to the lung tissue—expanding peribronchitis—or the infection may spill into the alveoli from the terminal bronchioles thereby resulting in bronchopneumonia

Causes:

1. **Inhalation of irritants**—dust, feed, industrial fumes, medicaments, smoke
2. **Infections:** a) **Bacterial.** — Pasteurellosis
   b) **Viral;**— Ranikhet disease, Infectious bronchitis of fowls, Infectious bovine rhinotracheitis.
   c) **Parasites;**— Lung worms.

Macroscopically; the mucosa is thickened, reddened and covered by an exudate which may be catarrhal, fibrinous or purulent.

In aspiration of foreign material, a gangrenous bronchitis is seen in which there is extensive necrosis of the mucosa which sloughs. The wall of the bronchus may also be destroyed.

Microscopically, there is congestion and infiltration by inflammatory cells in which neutrophils predominate. There may be increased secretion of mucus and in severe cases the epithelium may be destroyed. The lumen contains mucus, leucocytes, dead epithelial cells, lung worms and their ova.

Results; Recovery; bronchiectasis; abscess formation; bronchopneumonia; chronic bronchitis.

CHRONIC BRONCHITIS

Causes; 1. Mild, continuous irritants—smoke and dust; 2. Chronic venous congestion—as in heart disease; 3. Chronic infection of upper respiratory tracts—chronic sinusitis; 4 Bronchiectasis 5. Most common cause in animals is lung worm infection, tuberculosis and lung abscesses.

Macroscopically, the bronchial mucosa is thickened and has a velvety feel. Sometimes it may be congested but more often is pale and edematous. The exudate is mucoid or *mucopurulent and in cases of worm infection; it is* mixed with worms and their eggs. The bronchi may also be dilated.

Microscopically: there is infiltration by lymphoid cells. The ciliated epithelium is lost and replaced by a cuboidal variety The mucous glands may show atrophy There is hyperplasia of peribronchial glands which now resemble goblet cells. There is increased fibrosis of the walls, producing polypoid projections into the lumen —*bronchiolitis obliterans.* Lymphoid follicles may be formed in the walls of the bronchi.

Results : 1. *Bronchopneumonia*; 2. Atelectasis; 3. Bronchiectasis; 4. Emphysema—putting greater strain on the right side of heart—chronic venous congestion will result ultimately.

LUNGS

Atelectasis : The failure of the alveoli to open and contain air is called atelectasis. So the alveoli become collapsed. This condition may be congential or acquired.

Congenital atelectasis: Animal is born dead and has not breathed.

Causes; 1. Obstruction of the bronchi by mucus or inhaled liquor amnii.

2. Damage to the respiratory centre that may occur in injury to the brain.

The lungs are dark and reddish-blue in color due to dilatation of the alveolar capillaries. The lungs are firm to the touch and sink in water since there was no aeration. The alveoli are collapsed and the epithelium lining the alveoli is cuboidal. Sometimes the alveoli may contain fluid.

Acquired atelectasis (Pulmonary collapse)

Cause: 1. **Obstruction ;** The lumen of the bronchus is obstructed and the air in the alveoli is resorbed. The cause of the obstruction may be

a) **In the lumen :** foreign bodies, pus, mucus, masses of parasites.

b) **On the wall**—tumors, abscesses, enlarged lymph nodes, cysts.

2. **Compression ; Extrapulmonary**—hydrothorax, pneumothorax, hydropericardium, abdominal distension as in tympany of rumen and ascites.

Macroscopically, there is never a total collapse but only focal. The affected lung tissue is dark or reddish-blue in color and is depressed from the level of the surrounding healthy lung. The affected part sinks in water and is leathery in consistency. Pleura is thickened and wrinkled.

Microscopically, the alveoli are devoid of air. They may appear as small or elongated clefts or the walls may lie in apposition with each other with no lumen visible. Due to the absence of pressure of alveolar air, the capillaries become dilated and engorged with blood. In later stages the alveolar epithelium may be desquamated while the tissue of the interlobular septa may proliferate.

EMPHYSEMA

Emphysema is increased air in the lung. This is divisible into:

1. **Acute alveolar emphysema;** The alveoli are greatly distended and sometimes may rupture, orming "**Vesicles**".

Causes; a) **Compensatory** – In pneumonia or atelectasis when affected portions of lungs cannot dilate other healthy parts dilate to a greater extent to fill the space created by the expansion of the chest cavity.

b) **Over exertion**—in coughing and struggling over-ventilation occurs.

c) Feeding on lush pastures.

d) Allergic or toxic agents—feeding mouldy forage or mouldy sweeet potatoes. Parathion poisoning causes acute emphysema.

2. **Acute interstitial emphysema** often accompanies the acute alveolar emphysema. In this condition air collects in the interlobular space beneath the pleura and other interstitial tissue of the lung. This is seen more often in cattle and sheep.

Causes : a) Condition that produces dyspnoea—pneumonia.

b) Bellowing — in oestrum or when separated from calf ; common in kosher-killed animals.

c) Perforation of lung by mechanical means—foreign body through the rumen and reticulum.

d) Forced breathing as in old hunting dogs.

e) Pulmonary strongylosis.

In these cases, alveolar emphysema, when it occurs, is so severe that the alveoli rupture and air escapes into the interstitial tissue of the lung, especially in the inter-lobular septa. In severe conditions air may escape via the thoracic inlet into the subcutis of the neck and may accumulate there along the spine, from the pole to the base of the tail.

3 Chronic alveolar emphysema : Commonly called 'Broken wind' or 'Heaves' in horses, in which animal it is more often seen.

Causes: 1. Working horses immediately after a heavy meal — digestive organs are distended and so prevent the expansion of diaphragm during inhalation. So animal makes violent respiratory efforts;

2. Dusty and mouldy foods—causing coughing.

3. Obstruction of the bronchi—as in bronchiolitis.

4. Allegry to some sensitising agents (pollen, dust or fungi) may play a part

The above conditions cause severe and continuous cough.

So during violent cough and inspiratory movements alveolar walls are subjected to undue pressure. This process, repeated for months and years produces atrophy of the walls and their subsequent rupture. Pressure also interferes with capillary circulation — so the nutrients and oxygen supply to the alveolar wall is diminished with resultant fatty degeneration of the alveolar epithelium, degeneration and disappearance of elastic fibres and the inter - alveolar and the inter infundibular septa, resulting in rupture of the alveolar walls. Hence the adjacent alveoli become confluent—"bullae"

In bronchiolitis with obstruction, air passes through the pores of Kohn and so adjacent alveoli become balooned—collateral ventilation.

Owing to their diminished elasticity and permanent distension, alveoli expand with difficulty at each inspiration and also contract less easily on expiration and consequently continue to increase in size and finally rupture.

Besides the mechanical stress that produces emphysema as detailed above, it may primarily be also due to inflammation of respiratory bronchioles, alveolar ducts and alveoli. These structures becomes necrotic and weakened thereby becoming balooned due to the pressure of air that may be trapped.

Macroscopically, affected lung is voluminous and is pale due to decreased blood circulation. Lung pits on pressure easily and the indentations of the ribs are seen clearly.

Microscopically, the alveoli are over distented. Their walls are atrophied and then rupture of some alveoli with confluence of neighbouring alveoli give rise to giant alveoli.

Sequelae Hypertrophy and dilatation of right ventricle—chronic venous congestion.

Vascular disturbances of the lung

Active Hyperemia is commonly seen in some acute general infectious diseases or in acute pneumonia.

Passive Congestion occurs generally in older animals.

Causes: 1. **Cardiac lesions**: a) Myocardial weakness; b) Lesions in the mitral valve; c) Chronic pericarditis.

2. **Extrathoracic Lesions** Bloat—intra—abdominal pressure increases

3. **General vascular dilatation** : Shock.

Macroscopically, the lung is larger in size, dark red in color and is firm to the touch. On section blood oozes.

Microscopically, alveoli contain red cells and macrophages that have engulfed the red cells (Heart failure cells).

Sequelae : 1. Edema of lung—brown induration ; 2. Bronchopneumonia. 3. Dyspnoea; 4 Generalised hypoxia—fatty changes in the liver and kidneys.

Hypostatic congestion; In animals in moribund conditions blood accumulates in lung on the side on which the animal reclines. This is because the heart is too weak to maintain sufficient blood pressure.

EDEMA

Usually precedes pneumonia.

Causes : 1. Passive congestion of lungs—in lesions of myocardium and mitral valve

2. Toxic material that increases the permeability of the capillary endothelium—ANTU (Alpha Naphthyl Thiourea) poisoning; in shock
3. Hypoproteinemia
4. Acute anemias
5. After inhalation of smoke, phosgene, chlorine, ammonia, nitric oxid
6. Mulberry heart disease of swine
7. Bacterial toxins (exo or endo, produced locally or elsewhere)
8. Allergy (vaccination with strain 19 against Brucellosis and in helminthic infections)

9. Intravenous administration of fluids increasing venous pressure in pulmonary circulation.

Macroscopically, lung is large and firm. On section edematous fluid drips from the cut surface. Trachea and bronchi contain froth—due to churning action of the tracheal air on the protein-containing fluid. Edema of the interstitial tissue is present. The alveolar septa stand out prominently.

Microscopically the alveoli and bronchi contain a pink stained homogeneous material. The pink staining capacity is proportional to amount of the protein present. The greater the amount of protein present, the pinker will be the fluid stained.

The edematous fluid is a good medium for the growth of microorganisms and so pneumonia is a frequent sequel.

Hemorrhages Blood in sputum due to pulmonary hemorrhages is called *hemoptysis* This is rare and must be distinguished from the blood present in lungs by inhalation while the animals are slaughtered.

Causes: 1. Erosion of the blood vessels and rupture into a bronchus.
2. Extreme over exertion—as in young horses.
3. Extreme cardiac action as in death due to asphyxia.
4. Injury from a foreign body.
5. General hemorrhagic conditions
Bacterial diseases—Pasteurellosis, Anthrax
Viral diseases—Hog cholera
Defects in coagulating ability — hemophilia and bracken fern poisoning
Toxins—Uremia in which injury to capillary endothelium occurs.

Macroscopically, hemorrhages may be in the nature of petechiae, ecchymoses or even hematocysts. Pulmonary hemorrhages are dangerous because air is displaced and hypoxia may result. Blood in bronchi and trachea foams.

Microscopically, blood may be found in alveoli, bronchioles and bronchi.
THROMBOSIS, Often in pneumonia, due to extension of infection to the blood vessels thrombosis may occur and is frequently observed in septicaemic diseases—Pasteurellosis and Hog cholera.
EMBOLISM ; The strategic position of the lung is well suited for the arrest of emboli. Emboli emanate from;
A. Thrombi occurring in
1. Heart-worm infection in dog. 2. Mesenteric veins in horses (*Strongylus vulgaris* infection) 3. Uterine veins and pelvic veins (metritis and mastitis). 4. Posterior vena cava—extension of infection from hepatic abscess. (No 3 and 4 more common in cattle)
B. Ascarid larvae
C. Tumor cells—metastasis in malignant tumors,
D. Fat — not so common in animals as in man in whom it arises from crushing fractures of bones.

Sequelae ; Abscesses, thrombosis, infection and parasitic cysts.
INFARCTION ; For the infarction of lungs to occur there must be damage to both pulmonary and bronchial circulations.

Macroscopically, the affected areas appear dark-red, firm and solid looking. They bulge on the cut surface. They are cone shaped with base at the pleural surface and by careful dissection it is possible to disclose the thrombus and embolus at the apex of the wedge.

Microscopically, the whole area of infarction, including the alveoli, capillaries and the septa, is filled with blood. Later necrosis sets in the alveolar walls.

INFLAMMATION OF THE LUNGS

Inflammation of the lung is called pneumonia. Pneumonitis—literally meaning inflammation of the lungs—is used by different authors to convey slightly different meaning. Runnels uses it as a synonym with pneumonia; Smith and Jones apply the term for "any inflammatory disease of the lungs" preferring the use of the term pneumonia to "apply to one of the acute infectious inflammations with copious exudate filling the alveoli". Jubb and Kennedy differentiate the two terms by saying, "in pneumonitis the reaction is largely confined to the

wall of the alveolus and in pneumonia it is the alveolar lumen which reveals the most obvious changes". In other words they reserve the term pneumonitis to the specific disease in which alveolar wall is the primary site of pathology. To avoid confusion, the term pneumonia alone is used in these descriptions.

Pneumonia is a very common disease found in animals except probably the cat, in which it is rarely met with.

In man, a specific type of pneumonia called the *lobar or croupous pneumonia* caused by *Diplococcus pneumoniae* is met with, in which whole lobes may be affected, characterised by a fibrinous or croupous exudate in the alveoli. Differing from this condition is another type, called the *Catarrhal or lobular or bronchopneumonia* which is patchy and in which only parts of a lobule or only a lobule are affected, characterised by a catarrhal exudate of the alveolus

In animals, it is the lobular pneumonia that is frequently seen. There is no condition similar to the lobar pneumonia of man. Most varieties in animals may start as a lobular pneumonia but end up as a lobar variety. So all gradations may be met with in the same animal and, what more, the same etiological factors may give rise to these different grades of pneumonia.

Causes 1. By far the most common causes are the bacteria, the viruses, fungi and parasites. Again pneumonia may also be a lesion found in many specific diseases.

Bacteria :-

Corynebacterium equi
Streptococcus equi
P. mallei
} HORSES

Mycoplasma mycoides
Pasteurella multocida
Corynebacterium pyogenes
Actinobacillus lignieresi
Staphylococci
Streptococci
Mycobacterium tuberculosis
} CATTLE

C. Pyogenes
Pasteurella multocida
Staphylococci; C. ovis
Streptococci: E. coli.
} SHEEP

Streptococci; E. coli
Bordetella bronchisepticus
Staphylococci
Klebsiella
} DOG

Streptococci; C. pyogenes
Haemophilus suis
Pasteurella multocida
} SWINE

Pasteurella multocida
Coliforms
} CATS

Viruses

Equine infectious pleuropneumonia, Equine influenza, Calf pneumonitis, Canine distemper, Ranikhet Disease, Sheep pox.

Fungi : Blastomyces, Coccidioides, Histoplasma, Actinomyces, Aspergillus, Cryptococcus, Mucormycosis.

Parasites : *Metastrongylus apri* in swine; *Dictyocaulus viviparus* in cattle; *Protostrongylus rufescens* and *Dictyocaulus filaria in* sheep and goats; *Ascaris lumbricoides* var *suum* in pigs.

2. **Irritants :** Inhalation of dust, pollen, foreign bodies, smoke, hot and cold air, anaesthetics, war gases, medicinal agents.

Routes of infection :

1. **Through the respiratory passages—Bronchogenous :** This is by far the most common route. Some infectious agents like *Aspergillus fumigatus, Streptococci, Staphylococci, E coli* and *Corynebacterium pyogenes* and some viruses (Eg. viruses of Ranikhet disease and viral pneumonia of pigs.) may invade the lung through this route.

2 **Through the blood vascular system—Hematogenous :** Blood stream may carry bacteria (Salmonella, Pasteurella) and parasitic larvae to the lungs.

3. **Through penetrating wounds :** Wounds by bullets, knives, pitch forks etc., may penetrate the lungs from the exterior, usually carrying bacteria and producing pneumonia. Similarly, foreign bodies penetrating through rumen, reticulum and diaphragm may set up pneumonia.

Predisposing causes : Conditions, called predisposing factors make the animals more susceptible to diseases of respiratory system. These are fatigue, exposure to cold air, long travel by train or ship, severe hunger, malnutrition; chronic undernutrition, parasitism, exposure after dipping in winter months, cardiac weakness and recumbency for a considerable time.

The predisposing factors produce rhinitis and laryngitis by virtue of their lowering the resistance with superimposed infection. These affect or even destroy the ciliary movements and so predispose the organ for infection. Edema of the lung, that results due to cardiac lesions and other factors is an ideal condition for pneumonia to occur since the edematous fluid is a good medium for the organisms to develop and so cause pneumonia. In the recumbent animal "Hypostatic Pneumonia" may ultimately result.

Some etiological agents appear to become pathogenic only when the pulmonary tissue is suitably altered by other agencies, eg: in swine, virus of porcine pneumonia requires *Haemophilus suis :* in swine, *Pasteurella multocida* paves the way for a pleuropneumonia-like organism.

Some organisms, which are natural inhabitants in the upper respiratory tract become virulent and pathogenic, when the vitality of the animal is lowered eg. *Bordetella bronchisepticus* produces pneumonia in dogs in Canine distemper infection The virus of Distemper *per se* may not be able to cause pneumonia Similarly pasteurella will cause pneumonia in Hog cholera.

Varieties of pneumonia

It has already been observed that in animals lobar pneumonia as it occurs in man is not met with. So the clear-cut classical stages described in human pathology are not seen in animals in entirety. Since a description of the stages of human lobar pneumonia helps in the understanding of the genesis and development of pneumonia the same is given below.

1. **The stage of congestion :** This is the early stage in which there is active heperemia and edema of the alveoli.

Macroscopically, the lungs are congested and swollen. These still float in water On section, blood tinged-fluid escapes.

Microscopically, the capillaries on the alveolar walls are dilated and filled with blood. Alveoli contain a little serous exudate and often a few red blood cells. Depending on the irritant, this may develop within a few minutes (chemicals) to a few hours (infectious agents).

2. **Stage of red hepatisation : Macroscopically,** the affected portion of the lung is quite conspicuous being readily discernible from the healthy. A distinct line of demarcation is found. The affected part is red, and consolidated, solid looking resembling liver—**hepatization.** Portions of the affected parts sink in water, since all air is replaced. Over this area the pleura is inflammed and dull red in color. A membrane may form. Lymphatics are obstructed by fibrinous plugs. The pleural fluid is increased. The peribronchial and perviascular lymphatics are dilated with protein-rich fluid.

Microscopically, the alveoli reveal a fibrinous exudate containing erythrocytes, polymorphonuclear leucocytes and desquamated epithelial cells. Dilatation of lymphatics and widening of septal cells are observed.

Develops in two days.

3 **Stage of grey hepatisation : Microscopically,** the lung is still consolidated and sinks in water. The color is less red than the previous stage and some parts are grey like grey granite The redness is due to persistence of capillary hyperemia or the hemorrhagic nature of the exudate.

Microscopically, the alveolus appears to be less filled than in the previous stage. Fibrin can clearly be seen and strands may be found to pass from one alveolus to another through the pores of Kohn Erythrocytes have almost disappeared from alveoli. The greyness of the affected tissue in this stage is attributed to i) ischemia of the alveolar capillaries due to pressure of exudate on them, ii) increased infiltration by leucocytes iii) thrombosis in the alveolar capillaries and iv) lysis of red blood cells.

The liquefaction of the exudate commences and the nuclei of polymorphs become blurred and less distinct.

4. **Stage of Resolution :** At this stage liquefaction and removal of the exudate take place. The liquefied material may be absorbed via lymphatics or veins or may be expectorated.

Microscopically, the exudate is disappearing What remains is granular; polymorphs are either absent or the few that remain are degenerated. A number of macrophages derived from alveolar epithelial lining as well as from the blood

are in evidence. The epithelium, most of which has died and was desquamated is regenerated. Thus the lung returns to the normal state of functional activity.

In animals, though the classical lobar pneumonia described above is not met with, yet, in infection by *Pasteurella multocida* in cattle, sheep and swine a croupous type of pneumonia is encountered. But, even then, the quantity of fibrin in the alveoli is not as much as in the human condition Infection is usually by inhalation, the earliest lesion produced being a bronchiolitis From this place the infection spreads rapidly. If the bronchiole is obstructed., the flooding exudate infects the surrounding parenchyma And through the pores of Kohn. the edema fluid may infect the neighbouring lobule, which is not directly served by the obstructed bronchiole. Infection may also be direct and continuous from the bronchiole to the alveolus In a third manner, the infection may take place through the wall of the bronchus, namely by peribronchial path-way, when infection passes on to the alveoli lying immediately adjacent to the bronchiole by contiguity. Lastly, the infection may spread rapidly via lymph and blood.

Usually the apical, cardiac and intermediary lobes and the anterioventral portions of the diaphragmatic lobes are affected,

Due to difference in age of the lesions in different areas, one may see different stages of the disease in the same animal and hence we may see different colored areas. Again, thrombosis of pulmonary vessels, producing infarction may add difference in color of some areas.

Contagious pleuropneumonias of cattle and goats are of this lobar variety in which the interlobular septa are dilated and prominent due to a great outpouring of plasma and fibrin into them. It is the dilated septa that give the "marbling" effect to the lung in these areas.

Bronchopneumonia

This is the commonest type of pneumonia found in animals.

Causes;

Calves—is enzootic, caused by a virus.
Sheep—is enzootic? (Virus?)
Swine—i) Virus plus *Hemophilus suis*—swine influenza
ii) *Salmonella choleroe suis*—hematogenous
iii) In Hog cholera—*Pasteurella sp* plus *Hemophilus sp.*
Foals—*Corynebacterium equi* (may also be a complication of Strangles)

Primarily, infection starts as a bronchitis and bronchiolitis from where it may spread to the alveoli as described earlier viz. by flooding with exudate when there is occlusion of the bronchi, by direct extension from bronchi and bronchioles to alveoli and by peribronchial path-way. Compared to the lobar variety, extension to different parts is slow. The anterior and ventral parts of the lungs are more commonly affected because the bronchi to these parts take off vertically and so infection gravitates. Also the respiratory movement of these parts is less due to limited rotatory action of anterior ribs. As the infection spreads, fresh foci are set up and hence different stages of the disease are noticeable within the same animal

The *lesions* are patchy in distribution. One or several lobules may be affected which are red and firm, sinking in water. Areas adjacent to the affected show "compen atory emphysema".

The lymphatics are swollen and become easily visible. The lymph nodes are hemorrhagic and swollen. The bronchi contain hemorrhagic exudate. Ths pleura over the affected area shows inflammation with fibrinous exudate.

Microscopically;one or the other of the four stages described under the lobar variety may be seen

Sequelae: 1. Death—due to i) Toxemia; ii) Hypoxia and iii) Cardiac failure.

2. **Atelectasis**—when bronchiole is still obstructed, even after resolution of the alveolar exudate.

3. **Suppuration and abscess formation**—if the causative agent is pyogenic. Common in dogs, caused by *Bordetella bronchisepticus* and in foals by *Corynebacterium equi.*

4. Gangrene—this occurs when there is superimposed infection by saprophytic putrefactive bacteria. Usually occurs in aspiration pneumonia.

5 **Septicemia** when the infective organisms enter the blood,causing inflammation in other parts of the body eg. arthritis, meningitis etc.

6 **Incomplete resolution**—resulting in organisation of the alveolar exudate. Fibrosis of the lungs and pleura may result—*carnification.* (becoming like flesh).

OTHER TYPES OF PNEUMONIA

Necrotic, Gangrenous and Aspiration pneumonia

Causes: 1. **Faulty drenching** in cattle and careless passage of stomach tube in horses.

2. **Inhalation of irritant drugs, oils, anesthetics or feed** (lambs and pigs)

3. **Aspiration of** i) milk or gruel *(in pail – fed calves)*
   ii) ingesta—in paralysis of throat in parturient paresis of cattle.

4. **Hematogenous**—from gangrenous lesions elsewhere in the body. eg gangrenous metritis or mastitis.

5. **Penetration** of sharp foreign bodies through rumen and reticulum.

6. **Direct Infection** by *Spherophorus necrophorus.*

The irritants produce severe inflammation and extensive thrombosis of the blood vessels resulting in necrosis. The leucocytes and bacteria produce liquefaction of the necrotic material, resulting in cavitation. Putrefactive organisms produce gangrene.

Macroscopically, there is extensive consolidation of the anterior and ventral portions of the lung with foul smelling exudate. The affected parts are greenish or black in colour and sometimes large cavities are seen. The area around these lesions shows congestion and intense reaction.

Gangrenous pneumonia may be a sequel of choke (esophageal obstruction) Sequelae—Death in all cases.

Metastatic suppurative pneumonia

This may be acute or chronic This is due to embolic deposition of pyogenic organisms from lesions somewhere else in the body. eg Suppurative metritis, mastitis, lesions in Strangles, navel ill and heart valve lesions. (Vegetative endocarditis)

Microscopically: because the pathogen is hematogenous, there is uniformity in the distribution and size of the lesions. The diaphragmatic lobe is mostly affected and a number of foci may be found beneath the pleura. The lodged organisms produce inflammation with suppuration.

There may be several abscesses scattered with a zone of acute inflammation in the parenchyma surrounding them. There may be a capsule, the thickness of which indicates the duration of the process, (older the lesion, thicker the capsule.) Encapsulated abscesses may be organised, leaving a depressed scar.

Verminous pneumonia

Pneumonia in animals is caused by many species of parasites. They are

	Parasite	Habitat
Cattle—	*Dictyocaulus viviparus*	Bronchi
	Paragonimus westermanii	Bronchi and cysts in the lungs
Sheep—	*Dictyocaulus filaria*	Bronchi
	Protostrongylus rufescens	Bronchi
	Mullerius minutissimus (capillaries)	Alveoli and blood vessels
	Paragonimus westermanii	Cysts of lung and bronchi
Swine—	*Metastrongylus apri*	Bronchi
	Ascaris larvae	Migration
Horse—	*Dictyocaulus arnfieldi*	Bronchi
Cat—	*Aleurostrongylus abstrasus*	Bronchi
Dog—	*Angiostrongylus vasorum*	Pulmonary arteries
	Paragonimus westermanii	Cysts in lung and bronchi

The infection by strongyles, whose habitat is in the lungs, starts by the ingestion of infective larvae with feed and water. The larvae enter the portal circulation and finally reach the capillaries on the alveolar walls. Here they pierce the capillaries and enter the alveoli where they develop and become mature in about a week to 10 days. The mature worms then go up to the bronch and settle.

The pathology produced by these "lung worms" may, therefore, be described in two stages. In the first stage, the larvae enter the alveoli, develop and mature and in the second, they settle down in the bronchioles and bronchi. There may not be any clear-cut demarcation of the two stages, which may therefore overlap. In the place where the larvae enter the alveolar walls are found microscopic necrotic foci surrounded by an infiltration of neutrophils, eosinophils and macrophages. Thus thickening of the alveolar walls occurs. A few giant cells may also be seen. In massive infection, there may be severe hemorrhages.

When the parasites have become mature, they migrate, into the respiratory bronchioles and bronchi where they set up inflammation on the walls. A thick mucous exudate containing polymorphs, eosinophils and macrophages forms at the site, often occluding the bronchi. The epithelium of the bronchi and bronchioles becomes hyperplastic and thickened. Weakening of the bronchial walls because of pressure and inflammatory process results in bronchiectasis. Obstruction of the bronchioles and bronchi results in emphysema, since during inspiration air enters but which is not exhaled. So repeated trapping of the air results in emphysema. In places where there is complete occlusion of the bronchioles, atelectasis develops as the air in the alveoli is resorbed.

The lesions are found mostly in the diaphragmatic lobes and consist of focal wedge-shaped areas of emphysema and atelectasis. Secondary bronchial infection may complicate the picture.

Macroscopically, the lesion is one of bronchopneumonia. The exudate in the bronchi contains the worms with their embryonated eggs, along with neutrophils, macrophages, lymphocytes and desquamated epithelial cells. The alveoli may contain besides the inflammatory cells, worm ova.

Mullerius capillaris, a parasite of sheep and goats lives, not in the bronchi, but in the pulmonary blood vessels. The lesions produced are nodular and consist of granulomatous process—viz. fibrosis, giant cells, mononuclears and eosinophils. When the worm dies the nodule may become calcified.

Paragonimus westermanii, a fluke, is found in the lungs of cattle, sheep and dogs (also in man). These are usually found in pairs in inflammatory cysts in the lungs Most of the cysts have communication with the bronchi for the passage of eggs. Those not having such outlets become organised.

Larvae of Ascaris in their itinerary to complete their life-cycle pass through the lung, where they may cause severe damage and in some cases even death, if infection is sufficiently heavy. As in the strongyle worms, the larvae reach the lung via blood. From the capillaries, the larvae enter the alveoli through their walls thereby damaging them in the process. In heavy infection there may be massive hemorrhage when the larvae pass through capillaries. While the lung tissue is thus damaged, bacteria, normally found there migrate to the damaged areas and produce inflammation. Thus the combined action of the parasitic larvae and bacteria causes death of the animal.

Sometimes liver flukes may be found in the lungs, but this is an aberrant location. A nodule is formed with a fibrous capsule and the parasite dies as it cannot thrive in a strange locality.

Interstitial pneumonia

This is a condition in which the alveolar septa are affected. Though some exudate may be found in the alveoli, by far most of the changes, proliferative, are found in the alveolar septa. The following characterise this variety.

i) Thickening of the alveolar septa by
   a) exudate—serous or fibrinous
   b) infiltration by leucocytes and
   c) formation of new connective tissue

ii) Proliferation of the epithelial cells of the alveoli

iii) Formation of hyaline membrane in the alveoli and over the alveolar ducts and

iv) Almost complete absence of neutrophils in the exudate.

Causes are many and varied.

1. The usual cause are viruses, which may be the primary cause as in swine influenza and feline pneumonitis.

2. Or this condition may be pulmonary manifestation of generalised septicemic diseases like Erysipelas, Leptospirosis, endocarditis of the right side, Salmonellosis etc.

3. Psittacosis group of organisms.

4. Larval migration of ascarids and other intestinal parasites (not usually important)

The condition may be acute or sub-acute in intensity. Injury to the capillary endothelium is responsible for the fibrinous exudate in the the alveolar septa. Bacteria and parasitic larvae injure the endothelium directly and so make it more permeable. In the case of the viruses, increased permeability occurs in an indirect manner. The viruses affect the alveolar epithelium which proliferates and so becomes thicker preventing oxygen transfer. Hypoxia injures the capillary endothelium which therefore, becomes more permeable. This edema is partly responsible for the thickening of the septa.

Macroscopically, the lungs may be pale or reddened and do not collapse when the thorax is opened. As a whole, the lung appears edematous, with fluid dripping from a cut surface The interlobular septa are also widened. Usually interstitial pneumonia is the seat of secondary bacterial invasion and resultant bronchopneumonia.

Microscopically, alveoli may contain serous or serofibrinous exudate. which is also present in the alveolar . and *interlobular septa.* There is fibrous thickening of the septa and metaplasia of the alveolar epithelium, which is flat and pavemental normally and not easily seen, becomes cuboidal and prominent. These are called **Cells of tripier.** This change is said to be *Fetalisation* or *epilheliolisation.* Furthermore, the cells may proliferate, become rounded and desquamated, forming macrophages with phagocytic properties. In some cases, giant cells also form by the fusion of the macrophages thus formed from the alveolar epithelium or by the proliferation of their nuclei without the division of the cytoplasm. Predominance of these cells gives rise to the "giant-cell" pneumonia.

Lymphocytes and macrophages infiltrate into the alveolar septa and around the small blood vessels.

One characteristic feature of interstitial pneumonia is the presence of hyaline membranes, which are found covering the alveolar epithelium. The membrane is thought to be derived from and composed of the plasma proteins. Due to vascular damage and increased permeability of the capillary endothelium, rpofuse exudate forms, which nearly resembles plasma and hence the formation of the membrane.

Complete resolution may occur with alveoli regaining their normal histological structure and physiological function. The hyaline membrane may be removed by the macrophages. If resolution does not occur, healing by fibrosis takes place.

The fibrinous exudate is organised if it is not resorbed and remains longer than 2 to 3 weeks. This organised tissue is shrunken and grey and is like flesh—carnification.

Mycotic pneumonia

Inflammation of the lung caused by a variety of fungi is called mycotic pneumonia. Various fungi that may invade the lung and cause pneumonia are Aspergillus, Blastomyces, Mucor, Coccidioides and Cryptococcus. Of these, the most common is *Aspergillus fumigatus*.

Aspergillus fumigatus is ubiquitous and is found everywhere in nature. It is surprising that more cases of pneumonia are not caused in man and animals with the wide spread distribution of the spores of this fungus.

Though man and domestic animals are susceptible to Aspergillus and stray cases of pneumonia are encountered, but by and large, fowl is the most frequently affected.

The spores of the fungus are found in the poultry litter and mouldy grain. Infection is by way of the respiratory tract. Spores may be inhaled with the infected dust or may be aspirated from the mouldy grain. Since this affection is very common in brooder houses, it is called "Brooder pneumonia".

Lesions are found in the trachea, bronchi, and air sacs. Sometimes, larynx also may be affected. White or greenish, thick cheesy material is found in the affected areas.

The spores after entering the terminal bronchioles and alveoli, grow by budding and formation of septate hyphae. This produces a local inflammatory reaction, a nodular bronchopneumonia and there is infiltration by polymorphs and macrophages. This focus expands and more and more lung tissue is affected.

Macroscopically, the lesion is nodular. Evacuation of the central caseous material into the bronchus results in a central cavity. In lesions that are not progressive, increasing number of macrophages and epithelioid cells may be present with a fibrous capsule.

Microscopically, septate hyphae of the fungus with the inflammatory cells may be found around a central caseous material. In some cases, foreign-body giant cells may be found at the periphery. In lesions that are not progressive and are being obliterated, the hyphae are very short and the lesion resembles that caused by actinomyces—the "*asteroid body*".

Pulmonary adenomatosis

This is a disease of domestic animals, characterised by hyperplasia and hypertrophy of the alveolar epithelium, giving it a glandular or adenomatous appearance.

The disease has been described in sheep (in various parts of the world and in India), in cattle, pigs, horses and man. The causes are:—

Sheep: A filterable virus is supposed to be the cause, though it is not yet conclusively proved. A *mycoplasma* may also play an etiological role.

Cattle: Food allergy or some intoxication is thought to be the cause: feeding on mouldy foods.

Man: Nitrogen peroxide from silos is supposed to be the etiological agent.

Pigs and horses: A pleuropneumonia-like organism and toxins of *Crotalaria dura* respectively are supposed to cause a condition similar to pulmonary adenomatosis in these animals

The disease in sheep is called Jaagsiekte (Jaag=drive, siekte=sickness) or driving sickness in Afrikaans since fits of coughing are produced when affected animals are driven (exercised). The disease may occur sporadically or as an outbreak The incubation period appears to be about 7 months. After the onset of symptoms, the course is slow, death occurring in 2 to 3 months. Natural transmission is by droplet infection.

The disease can be differentiated from other pneumonic conditions by the absence of rhinitis in this condition.

The lesion starts as a small nodule, resembling that produced by *Mullerius capillaris*. But the former is softer and more friable. The nodules that are discrete to start with may coalesce to form bigger nodules and so finally, the lung is bulky and heavy, showing the imprints of the ribs. Emphysema of unaffected lungs is also noticed. Bronchial lymph nodes may frequently show metastases.

Microscopically, the characteristic change noticed is hyperplasia and hypertrophy of the alveolar epithelium. The normal pavemental epithelium, not easily discernible, becomes cuboidal or columnar with rounded nuclei. There may be papillary projection of epithelium into the lumen of the alveolus. In some places, there may be desquamation of the epithelium into the lumen. The alveoli seldom contain any exudate found in other pneumonias. The bronchial epithelium may also show papillary projection into the lumen. The bronchial lumen may contain desquamated epithelium as well as alveolar septal cells. Lymphocytes and plasma cells may infiltrate in the stromal tissue.

The thickening of the alveolar epithelium prevents the normal exchange of gases and so hypoxia is produced, causing dyspnoea. Due to decrease in the pulmonary capillary bed, dilatation of the right side of the heart may result. Fibrosis of the lung usually supervenes since the condition is not resolved completely and early.

In cattle lesions similar to the above may be seen but the condition is more acute and so fibrosis is not usually observed.

In man, pulmonary adenomatosis is common in those working near corn silos, where nitrogen peroxide is liberated. This is, therefore, called "silo-filler's disease". Death is quick occurring in six weeks after initial exposure,

In the human disease, bronchiolitis fibrosa obliterans is more in evidence due to the organisation of the bronchiolar exudate. Grossly these lesions appear as numerous uniformly distributed, firm miliary nodules.

Maeli

This means dyspnoea in Icelandic language. Maedi is found in Iceland and probably in Holland and North America. A condition similar to Maedi was reported in India.

The incubation period is long, upto 3 years. The disease is contagious and occurs as an outbreak. A virus is supposed to be the causal factor.

Maedi affects older sheep, usually those over two years. Affected animals show progressive debility, dyspnoea and inanition and may linger on for three to six months. In final stages acute pneumonia may supervene (due to pasteurellosis).

Macroscopically, the lungs do not collapse, when thorax is opened, but feel rubbery. They are greyish-blue in color, larger and very heavier than normal. The alteration in lungs is uniform in the absence of any complications. There is hyperplastic lymphadenitis and so the nodes are swollen. Except in terminal stages, consolidation, as found in other types of pneumonia is seldom met with. So, on section, no exudate oozes and the cut surface is dry.

Microscopically, the lesion is chronic interstitial pneumonia and the reason for the increased density of the lung is due to increase in the thickness of the alveolar walls. There is infiltration of walls by lymphocytes and reticulo-endothelial cells – macrophages and short reticular cells. The alveolar lumen may contain desquamated epithelial cells. There is hypertrophy and hyperplasia of the bronchiolar epithelium. Peribronchiolar and perivascular infiltration with lymphocytes is marked. There may be increase in the smooth muscle of the respiratory bronchioles, which may give a false appearance of fibrosis. No healing is observed.

Inclusion bodies, 1μ to 3μ in diameter, bluish-grey with Giemsa's stain are found in the cytoplasm of the alveolar macrophages.

Jaagsiekte	Maedi
1. Alveoli show adenomatosis	1. Adenomatosis is not seen
2. Inclusions absent	2. Inclusions present
3. Lesions focal, not diffuse	3. Lesions diffuse
4. Lymph nodes not affected	4. Lymphadenitis present
5. Course shorter	5. Course longer

TUMORS OF THE LUNGS

In the lungs primary tumors are rare. Those reported are classified as having the following cell types: squamous cell, columnar cell, mixed cell and undifferentiated cell. The exact sites of origin are still under dispute, whether from the bronchial epithelium (though most of the tumors are believed to arise from this site) or the alveolar epithelium or from the epithelium of the mucous glands.

Both benign and malignant tumors occur. A few connective tissue tumors have also been described. Lymphocytoma is the commonest.

DISEASES OF THE PLEURA

Congestion and edema (hydrothorax) are common in acute poisonings and passive congestion.

Pneumothorax is the presence of air in the pleural cavity. This may be due to entry of air into the pleura by the following ways:

1. From the lungs; when some bullae rupture,
2. Through the chest wall—by piercing sharp objects.

Pneumothorax causes atelectasis of the lungs and this is of great importance in the horse in which the right and left cavities communicate.

Pleuritis; Pleuritis is inflammation of the pleura. It is also known as **pleurisy** Mostly pleuritis is secondary to pneumonia.through primary infection of the pleura may occur. The infection may be by several routes:

1. **By direct extension** from the underlying lungs or mediastinal glands or esophagus.
2. **By blood stream** in septicemic diseases.
3. **Introduction through** the thorac wall—trauma by knives, bullets etc
4. **Introduction from** the rumen via reticulum and diaphragm.
5. **Through** the esophagus—when sharp bones or pins may penetrate the pleura and convey bacteria or in the horses when choke occurs.

Pleurisy may also be a condition noticed in specific diseases like swine erysipelas and contagious bovine pleuropneumonia. When pleura is involved in pneumonia, the condition is known as *pleuropneumonia*. But it is not necessary that pleura should be affected whenever lungs are. It is also possible to have pleuritis when the underlying lungs are healthy as in Black Quarter.

Bacteria that reach the pleura are readily spread due to respiratory movement. Bacterial growth damages the mesothelium and the endothelium of the superficial blood vessels, resulting in an inflammatory reaction, characterised by congestion and thickening of the membrane. The friction, between the parietral and swollen visceral surfaces causes intense pain during respiration The pain is relieved on the formation of inflammatory exudate which separates the two surfaces.

The exudate may be serous but more often is serofibrinous or fibrinous. Usually it is copious and may press on the lung, producing pain. Pyogenic bacteria may produce purulent exudate when the conditions is known as *empyema*.

If the exudate is purely serous, it may be absorbed. But if serofibrinous or fibrinous, complete absorption does not take place, organisation sets in producing "adhesions" in which the parietal and visceral surfaces get sewn, causing pain during respiration.

If saprophytic organisms gain entry to the pleura along with foreign bodies, gangrenous pleuritis will occur.

Tuberculosis of the pleura is most common in cattle, frequent in dogs and cats but seldom found in other animals.

Tumors of the pleura are rare. They may be (a)Primary—mesothelioma, a malignant tumor or (b) secondary,—metastasis of melanoma.

CHAPTER 17

THE DIGESTIVE SYSTEM

Teeth—caries
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- Thrush

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- Peritonitis

The Teeth

Caries is decay of teeth in which the enamel is decalcified followed by softening and discoloration. Caries is rather rare in domestic animals. This occurs occasionally in pet dogs with imbalanced and inadequate diets. The affected teeth have, usually, one or more depressed areas, which are brown or black in color. The organic acids, especially lactic acid, that are formed due to the action of bacteria on carbohydrates, dissolve the salts of the enamel. Then, the same acids corrode into the less stronger dentine, which contains in its structure 30% of

protein. The damage to the dentine is deeper and most widespread. The opposed surfaces of adjacent teeth may be more frequently affected "*Enamel Flecks*" are yellow stained spots on the enamel in the early, incipient stages. The affected teeth are shaky and are very painful and so interfere with mastication.

Causes include disturbances in calcium and phosphorus metabolism as well as dietary deficiencies of these minerals.

As we have already seen, caries is frequently seen in fluorine poisoning.

MOUTH AND PHARYNX

Malformations: Clefts of the mouth occur due to failure in embryonic processes. The most common is palatoschisis or hare lip. In this condition the ingesta is likely to enter the respiratory passages.

Stomatitis: This is diffuse inflammation of the mucous membrane of the mouth. But when confined to particular parts of the mouth it is known as:

Gingivitis for inflammation of gums.

Glossitis for inflammation of the tongue.

Lampas for inflammation of the palate.

Cheilitis for inflammation of the lips.

If pharynx alone is affected, inflammation of that part is known as Pharyngitis. If tonsils alone are affected, then the condition is known as tonsillitis.

Stomatitis is a common affection noticed in animals and more often it is a symptom of some other disease. Again, it may be a primary affection or may occur as secondary to other associated diseases viz gastritis or infectious diseases.

Cause: 1. Physical (a) Trauma by awns, thorns, burrs, wood pieces, glass pieces; sharp bits, irregular sharp teeth; sharp edged feeding utensils

(b) Heat: Hot drenches, (c) eating frozen foods

2. Chemical; caustic alkalies, corrosive acids, fertilisers.
3. Deficiency of vitamins; a) Hypovitaminosis A; especially in fowl
   b) Niacin deficiency: Black tongue in dogs—(necrotic stomatitis)
4. Microorganisms;
   i) BACTERIA; *Actinomyces bovis; Actinobacillus lignieresi; Spherophorus necrophorus; Pseudomonas aeruginosa; Corynebacterium pyogenes; Streptococci and Staphylococci*;
   ii) FUNGI; *Monilia albicans* and *Oidium pullorum* in poultry,
   iii) VIRUSES: Foot and Mouth disease; Rinderpest: Virus diarrhoea—Mucosal disease; Infectious Canine Hepatitis; Contagious ecthyma; Vesicular exanthema; Fowl pox, Blue tongue in sheep.

Macroscopically, the lesion starts as catarrhal inflammation of the mouth and pharynx with reddening and swelling of the mucosa, which is covered by small, whitish spots, raised than the surrounding. "*Aphthous stomatitis*" is the name given to the condition. These spots may later develop into small crusts or into ulcers.

Thrush: is found in birds. This is the name given to a condition in which grey or yellowish thick material tenaciously gets attached to the mucous membrane.

Vesicular stomatitis is the condition in which vesicles or blebs or blistere containing fluid are formed on the mucosa and seen in Foot and Mouth disease.

Infectious Vesicular Exanthema and Infectious vesicular Stomatitis. Rupture of the blisters results in the formation of erosions, which subsequently heal. Catarrhal and vesicular stomatitis may develop into ulcerative variety.

Fibrinous and necrotic stomatitis; are seen in infection by *Spherophorus necrophorus*. Very severe irritants may cause gangrenous stomatitis.

Fowl pox pro luces diphtheritic stomatitis and pharyingitus in which a grayish membrane is found.

Sequelae; 1. Starvation as prehension and mastication are prevented.

2. Spread of infection to other parts—esophagus, lungs, stomach etc.,

Tumors of the mouth and pharynx are common. Most common neoplasms in the dog and calves are the infectious papillomata, occurring as clusters on the lips and gums. These are supposed to be viral in origin (verruca vulgaris).

Epulis is a fibroblastic tumor, usually occurring in the gums consisting of dense fibrous tissue with varying amounts of epithelium and a few giant cells

Carcinoma, sarcoma, fibroma and melanoma are other tumors occasionally seen.

SALIVARY GLANDS

Pathological processes are very rarely found in the sailvary glands of animals because. (1) the salivary secretions have some anti-bacterial properties: (2) there is good flushing by the secretions and (3) the glands are in fairly well protected situations.

Foreign bodies are occasionally found in the ducts especially of the parotid and submaxillary glands. These are usually, awns, slivers of wood and kernels of grain, causing inflammation. Sometimes these may produce obstruction and consequent dilatation of the ducts.

Dilatation of the salivary ducts may occur when the flow of saliva is obstructed by foreign bodies, inflammatory exudates etc.,

When the dilatation occurs as cyst on the floor of the mouth it is called a *ranula*, which is smooth and rounded, containing a clear fluid and which can be easily ruptured.

Sialoliths are salivary calculi and are common in horses. These are formed by the precipitation of minerals around nuclei of foreign matter in the ducts Salivary calculi are usually single, and sometimes may be very large; preventing the flow of saliva. These calculi produce stasis, distension of ducts and finally atrophy of the gland.

Inflammation of the salivary glands; *Sialadenitis* or *parotiditis* is very rare in animals and may be due to traumatic injury or due to infection by bacteria. Stasis of saliva in the ducts facilitates infection. This may be associated with strangles in horses, mastitis in cattle and distemper in dogs.

Macroscopically, the glands are swollen and red. Abscesses may be found in glands and sometimes cystic dilatations may occur. Inflammation of the salivary glands may cause atrophy of the gland.

Neoplasms of salivary glands are not common in animals.

ESOPHAGUS

Choke; is obstruction of the esophagus, occurring in horses and cattle, but more common in the former.

Causes : 1. Impacted masses of feed due to : improper chewing, bad teeth and rapid gulping of dry feed.

2. Lesions of esophagus—stenosis or diverticulum—repeated choking may occur,

3. Old age.

4. In cattle, large objects of food—beet root, carrot, apples, potatoes, fetal membranes, sticks, wire, (large bones in dogs).

5. Enlarged lymph nodes—mediastinal and cervical.

6. Enlarged thyroids.

7. Neoplasms of adjacent tisssue—especially thymus, thymoma in new-born animals.

In the horses choke occurs in the thoracic area while in cattle and dogs the pharynx is obstructed. Choke may be complete or incomplete. In complete choke feed will be returned and water will come out of the nostril when animal is watered. Inhalation of the feed will cause secondary foreign-body pneumonia. In cattle, complete obstruction will cause dangerous tympany.

Because of pressure, ischemia and resultant necrosis develop leading to gangrene

Infection may spread to the surrounding tissues—cellulitis or to the lungs—gangrenous pneumonia Sapremia or toxemia that occurs is the cause of death in fatal case .

Partial obstruction will give rise to dilatation of esophagus above the obstruction,—the *esophageal diverticulum.*

Sequelae : 1. Death due to gangrenous pneumonia, bloat, cellulitis or asphyxiation. 2. Esophageal diverticulum. 3. Rupture of esophagus.

DILATATION (ECTASIA) The dilatation of esophagus may be *fusiform* or *cylyndrical* of which the former is the more common.

Causes : 1. Accumulation of food proximal to a stenosed area.

2. In ruminants accumulation of food during regurgitation on the distal side of stenosis.

3. Trauma from horns etc. rupturing the muscular coat.

4. Idiopathic, due, probably, to relaxation of the esophageal muscles consequent on nervous lesions.

The food gets accumulated in these areas producing pressure.

Sequelae : 1. Rupture due to pressure of the food.

2. The food may become decomposed and produce softening of the epithelium, inflammation, ulceration gangrene and death. 3. In ruminants, bloat.

ESOPHAGITIS

Inflammation of the esophageal mucosa is rare in animals because of the thick and resistant condition of the mucosa.

Causes : Trauma—probang; stomach tube, foreign bodies.

Chemicals—corrosives

Parasites—bot-fly larvae in horses, and hpoderma larvae in cattle.

Persistent vomiting, in dogs and pigs.

Avitaminosis A in Fowls.

Macroscopically, the mucosa is red and swollen. In the catarrhal variety, the exudate is mucous. The ulcerative variety is met with in conditions caused by trauma (Stomach tubes), in virus enteritis and in Mucosal Disease in cattle.

In the fowl, thallium sulphate poisoning produces esophagitis.

When pyogenic bacteria enter the place of obstruction (in choke) which had become necrotic due to pressure suppurative esophagitis occurs.

Sequelae : Usually recovery occurs. Stenosis may result if severe.

NEOPLASMS : Neoplasms of the esophagus are not common. Of those that are encountered, the connective tissue tumors of the dog must be mentioned. In the thoracic portion of the esophagus are found fibrosarcomas and osteogenic sarcomas that have some connection with *Spirocerca lupi* infection. The osteogenic sarcoma is evidently a metaplastic manifestation of the fibrosarcoma. Metastases of these tumors are sometimes found in the lungs and other tissues.

Carcinoma in cat and horse and papilloma in cattle were other tumors met with.

OBSTRUCTION OF CROP IN BIRDS

Causes : 1. Atony or paralysis of wall leading to stasis of food.

2. Ingestion of large quantities of dry grain which swell in the crop and form a hard mass.

3. Foreign bodies like wire etc.

The stagnated food gets decomposed, gas accumulates and inflammation sets in.

Sequelae : 1. Rupture due to distended food and gas or due to penetration of a foreign body.

2. Death because of (a) asphyxia, due to compersion of trachea, (b) heart failure due to pressure on heart, (c) starvation since food does not enter the proventriculus (d) intoxication due to absorption of toxins from fermented foods.

INGLUVITIS : (Inflammation of crop)

Acute catarrhal ingluviitis

Causes : 1. Trauma by foreign bodies.

2. Chemical agents : phosphorus, fertilisers.

3. Toxins from decomposed food.

4. Infectious diseases.

5 Parasites—*Acuaria sp.*; *Capillaria sp.*

Lesions include cogestion, edema and tympanites. Diphtheritic ingluviitis is found in fowl pox.

FORE STOMACHS OF RUMINANTS

TYMPANITES OR BLOAT : Normally animals get rid of gases produced in the rumen by eructation. Bloat (or accumulation of gas) can therefore occur when the gas is produced at too rapid a rate than can be eructated or when the eructation mechanism is faulty.

Bloat may be acute or chronic. The chronic variety occurs whenever there is any hindrance to eructation in the esophagus, either within or without—pressure by tumors, foreign bodies, enlarged lymph nodes, abscesses, constrictions

or diverticula. Or the lesion may be in the rumen causing decreased contractions of the ruminal wall as in atony, serosal adhesions, paresis, diffuse lymphomatosis.

Acute Tympanites; This may be due to choke in esophagus. Or it may also be due to sudden changes of feed or to excessive feeding on legumes that are wet with dew or rain.

There are various theories proposed to explain bloat but none of them are quite satisfactory. The following are some of them.

1. Some legumes contain HCN, which is toxic, causing paralysis of the ruminal or reticular musculature and so inhibits eructation.

2. Some legumes contain Phosphatase which with arsenates accelerates fermentation so that large quantity of CO_2 is produced.

3. H_2S, CO_2 and CO produced in large quantities casuse paralysis of ruminal muscles.

4. If fed excessively on green plants only, which do not contain sufficient stiff fibres, the mucosa of the rumen is not adequately scratched to elicit the reflex contraction of the musculature.

Saliva which has important antifoaming properties plays a significant part in the prevention of bloat. Mechanical stimulation of cardia, especially by roughages, increases the rate of secretion of saliva. But with ingestion of young succulent legumes, too little saliva may be secreted and so foaming is not counteracted and bloat results. Hence hay or straw must be fed liberally when animals are fed with succulent legumes. Mucin in saliva prevents formation of froth. But ruminal mucinolytic bacteria may destroy salivary mucin thereby producing bloat. Polysaccharides produced by capsulated ruminal bacteria may be another etiological factor in bloat.

Interference with the nerve pathways that are responsible for the eructation reflex may also lead to tympany. The receptors for this reflex are in the reticulum and the afferent and efferent nerve fibres are in the vagus nerve. Any lesions in this nerve may, therefore, lead to bloat, since the reflex that leads to eructation is interrupted.

Bloat is of two varieties: the dry and frothy. The former is less harmful since in this condition the gases can be more easily got rid of by eructation. On the other hand, in the frothy bloat, the gas is trapped as small bubbles in the fluid forming a foamy mass, which is not easily eructated. The following are supposed to produce frothy bloat.

1. Saponin found in plants is incriminated. Saponin is a good saponifying agent.

2. Water-soluble proteins of the legumes are capable of forming froth. This is probably of greater importance than No. 1, since bloat was observed even in animals fed with low-saponin plants.

3. Changes in surface tension and viscosity. It was found that (i) feeding times: (ii) kinds of feed and (iii) amounts of water, change the viscosity and surface tension. Those increasing the viscosity and lowering the surface tension produce froth. Normally in rumen due to bacterial activity, fatty acids are produced. These increase the surface tension. If the production of these fatty

acids is decreased then their protective action is not available and so surface tension will be lowered favouring froth production. This is the theory behind the use of vegetable oils in the treatment of bloat.

Distended rumen compresses other abdominal organs and causes passive congestion since the pressure on thin-walled veins impedes circulation. Along with this, there is forward thrust on the diaphragm, pressing on the lungs, which become smaller and sometimes atelectatic. The result of this is hypoxia and ultimate asphyxia and death.

In animals that die of bloat, besides congestion of the abdominal viscera, one may notice hemorrhages on the serous membrane of the lungs, on the pericardium, on the tracheal mucosa and in the lymph nodes of head and neck. Blood is tarry, as in Anthrax. The bronchial lymph nodes may be hemorrhagic. Liver is pale. Sometimes the rumen or diaphragm may be ruptured. If the animal is dead for some hours, the ruminal epithelium peels off.

Sequelae; If quickly relieved, acute bloat can be cured. If not, death may supervene due to asphyxia.

Impaction of the rumen and reticulum

This is a common conditon in cattle, the important feature of which is that the rumen stops functioning, the musculature does not contract and so the food ingested stagnates.

Causes: 1. Overfeeding with large amounts of highly fermentable carbohydrate feeds.

2 Penetration of the wall of the rumen or reticulum by sharp objects—wire, nail etc,

3 Lack of water.

4 Defective mastication and insalivation due to defects in teeth or lesions of the tongue.

5. Paresis of rumen which may occur due to injury to vagus by pressure from abscesses, tumors, tubercular nodules, swollen lymph glands and ruminal displacements.

Lack of exercise and debility are supposed to predispose the animal to atony of rumen. Tight packing of the rumen leaves no room for bacterial growth and normal ruminal fermentation and digestion. This leads to weak contractions of the ruminal and reticular walls and so the food does not get propelled. The stagnated food becomes putrified with the liberation of foul smelling gases. Anorexia develops and regurgitation stops.

In some animals, diarrhoea may be present if the putrid ingesta finds its way into the intestines causing enteritis. In mild cases, if the primary cause is removed, normal state may be regained. But in severe cases, toxemia will cause death At necropsy, the rumen will be found to contain hard, caked, undigested food, with evil smelling odor.

The pathogenesis of atony and impaction of the rumen after ingestion of large quantities of carbohydrate-rich feeds is as follows; The carbohydrates are fermented by gram positive organisms, notably *Streptococcus bovis*, with the formation of lactic acid, resulting in lowering of pH of the ruminal contents to as low as 4 or 4.5 from a normal 5.5 to 7. 5. Due to the production of lactic acid

the osmotic pressure of ruminal contents increases and so fluid is drawn into the rumen from the blood and so hemoconcentration, anuria, dehydration and circulatory collapse result. As the pH of the ruminal constituents falls, the motility of the rumen decreases and there may even be complete stasis. At the lowered pH, the normal microflora of the rumen are destroyed, the lactobacilli and streptococci thrive and the salivary secretion ceases so that the buffering action of the saliva is absent. Absorption of the lactate causes acidosis. Histamine may also be produced in the rumen which on absorption is toxic.

In such an atonic rumen, in which the normal microflora are lost, *Fusiformis necrophorus* and fungi of the family *Mucoraceae* (those belonging to the genera *Mucor*, *Rhizopus* and *Absidia*) invade the ruminal wall producing ruminitis and ulcers.

In animals that die of acute atony, the contents of the rumen and reticulum are thin, porridge-like and bulky. The cornified epithelium is soft and peels off easily, exposing hemorrhagic areas underneath. The blood is dark and thick. Lungs show bleeding into the alveoli and bronchi. Heart musculature is flabby. In animals that survive for three days and more, demyelination of the nervous system may occur.

Complications : Enteritis, peritonitis and ketosis.

Traumatic reticulitis

This is a very common condition in older cattle. These animals ingest and swallow, along with their feed, a wide variety of sharp objects like needles, nails, pieces of fencing wire and screws. Contraction of the rumen and reticulum during pregnancy may aid in the development of the condition

The sharp object pierces the wall of the reticulum during its contractions. Usually, it pierces the antero-ventral wall, which is near the diaphragm. The passage through it is usually slow and so the track formed by the moving nail is thickened by a dense fibrous wall. Piercing the diaphragm, the foreign body may enter the pericardium and even the heart, producing inflammation enroute. Or sometimes, it may take a downward slope and pierce the chest wall near the xyphoid cartilage forming an abscess there

At the point where the object pierces the reticulum, a localised peritonitis is formed and so adhesion of the reticulum to the diaphragm at this place occurs.

The sharp object may sometimes penetrate the lungs or the liver or the spleen. If not contaminated, only mechanical injury to the affected parts is seen. Traumatic pericarditis with serofibrinous or purulent exudate may be the result when the pericardium is affected. Other sequelae of traumatic reticulitis are : vagus indigestion, when the ventral branch of the vagus is affected by the inflammatory and scar tissue formed by the penetrating foreign body ; diaphragmatic hernia that may occur due to weakening of the diaphragm by lesions produced by the foreign body ; abscesess of liver and spleen and death due to rupture of left gastroepiploic artery.

At necropsy : The thick-walled track followed by the foreign body and adhesions of the reticulum to the diaphragm may be clearly seen. Along the track may be found abscesses and fistulae connecting one abscess to the other.

Fibrinous pericarditis with hypertrophied myocardium may be seen if foreign body has entered the pericardium.

Ulcers of the fore stomachs may be seen in cattle occasionally and are usually due to *S. necrophorus* infection. These ulcers may be found in animals that are started on heavy grain feed and also in calves kept on milk. Chemical ulceration due to corrosives may occur, though rare.

Ulcers are seen in Viral diarrhoea and Mucosal disease.

Hepatic abscesses are considered to be complications of ruminal ulcers.

THE STOMACH

MALPOSITIONS :

Diaphragmatic hernia of stomach may be met with in dogs and cats involved in automobile accidents. The diaphragm is ruptured and the stomach enters the thoracic cavity.

Abomasal displacement : Abomasum may be displaced from the normal position either to the left or to the right. But the left-sided displacement is more common, in which it comes to lie between the rumen and left abdominal wall. The greater curvature of the body of the abomasum which is more mobile slips under the ventral ruminal sac.

Causes : This condition is met with more frequently after parturition. It is suggested that during pregnancy the rumen may be lifted by the expanding gravid uterus and the abomasum may slip under the rumen. After parturition, when the uterus recedes, the rumen is dropped to its normal position, when it traps the the abomasum. Atony of the abomasum due to feeding large quantities of concentrated feeds is a contributory cause for the codition to continue without any tendency at correction. Atony may be caused by the inhibitory effects of high fat or protein feeds. Post-parturient diseases like milk fever, mastitis, metritis and ketosis may cause atony of the abomasum. Abomasal displacement has been met with in cases treated surgically for chronic indigestion. In such cases the incisions made are the weak spots where the abomasum may slip through. Violent activity like jumping in estrus, may be a cause in non-parturient cases. A hereditary predisposition may exist.

Symptoms manifested are vague and are like those of chronic indigestion—anorexia alternating with voracious appetite, abdominal pain and ruminal tympany. Animal loses weight rapidly, is listless, dull and has a tucked-up-appearance. The dung is scanty but soft (not caked as in chronic acetonemia). Mild ketonuria is present. Frequent abnormal tinkling abomasal sounds may be heard at the level of paralumbar fossa.

Due to pressure and compression the normal function of the abomasum is interfered with. Because of the abnormal position (consequent on the displaced position of the abomasum). the function of the esophageal groove may be affected.

Diagnosis : Displacement of abomasum must be differentiated from chronic acetonemia, traumatic reticulitis, vagus indigestion, diaphragmatic hernia, pyelonephritis and lymphomatosis. Laparotomy may be needed for diagnosis.

Treatment consists in correction of the condition by (1) rolling the anima, gently. If this is not successful, (2) the animal is operated on the left or both

flanks, the rumen raised, the abomasum released and replaced in its original position.

TORSION of the stomach may sometimes be seen in old dogs. There is twisting of the stomach around the esophagus. This is due to sudden movements (jumping, rolling etc.) especially when the stomach is full. With the esophagus as the pivot, the heavy stomach rotates clockwise. The twist closes both the openings of stomach and so gastric tympany develops with resultant dyspnoea. In some cases the stomach may rupture. As the blood vessels are compressed, there may be congestion and hemorrhage. The contents of the stomach are blood stained.

RUPTURE OF STOMACH; This is common in the horse and is usually due to tympanites and dilatation. Sometimes trauma and violent gastric contractions may be the causes.

ACUTE DILATATION OF THE STOMACH is due to excessive amounts of feed or gas accumulating in the stomach.

Causes; 1. Overeating; especially with grain in horse.

2. Excessive fermentation when easily fermentable foods are eaten.

The following factors act as accessory causes; Obstruction of the pylorus, (by rags or sacking in calves), reflex closure of the pylorus, atony of the gastric musculature, incomplete mastication due to poor teeth, diseases of the stomach wall and hard work immediately after feeding, external compression of the pylorus by lipoma in horses and lymphocytoma in cattle.

The accumulated feed and gas in the stomach cause such a stretching of the gastric wall that contractions are interfered with. And this stretching causes severe pain also. Dilatation of the stomach causes vomition. Loss of fluid may result in fatal dehydration and alkalosis. Rupture will result due to stretching of the muscle fibres, when death will occur due to shock.

The stomach which is dilated 4 to 5 times its normal size presses upon other organs: pressure on diaphragm and lung results in dyspnoea and congestion. Sometimes the diaphragm may rupture. Obstruction of the pylorus due to a tumor or cicatricial constriction and wind sucking may cause recurrent dilatation with resultant hypertrophy of the muscular wall of the stomach.

GASTRITIS

Inflammation of the stomach is a fairly common condition in animals. It may be primary or may be secondary to some other infections, as in canine distemper, viral diarrhoea, swine erysipelas. Gastritis may be acute or chronic. Causes may be the same for both but of different severity and acting for different lengths of time.

A. Physical: 1. Overfeeding, causing dilatation of stomach, is always accompanied by gastritis.

2. Feeding with frozen roots causing bloat, produces gastritis.
3. Feeding very coarse material (eating bedding in horses and dogs.)
4. Faulty dentition, preventing mastication is another cause of gastritis.
5. Foreign bodies may traumatise the gastric mucosa.
6. Spoiled, mouldy and fermented hay and silage.
7. Too sudden changes of feed may also be responsible.

B. **Chemicals** ; 1. Caustic and corrosive chemicals like mercury, lead, copper, arsenic and phosphorus.
2. Toxic plants.
3. Uremia; (Excretion gastritis is caused by the excretion of the poisoning material through gastric glands.)
4. Feeding easily fermentable foods liberates irritating substances which produce gastritis.
5. Feeding heavily fatigued animals has the same effect as number 4 above, since in such animals, the feed is not easily digested, stagnates, ferments and so produces irritation.
6. **Stress** ; In stress, adrenaline is produced in large quantities which is responsible for gastritis. This is seen in nervous dogs and in calves separated from their mothers

C. **Bacterial** ; In calves — enterotoxemia, colibacillosis ; pigs—erysipelas, vibrionic dysentry, salmonellosis, colibacillosis.

D. **Viruses;** Pig— hog cholera; transmissible gastro-enteritis in baby pigs *Cattle :* rinderpest, mucosal disease.

E **Fungi** ; Mucormycosis, moniliasis and aspergillosis cause *gastritis* in many animals.

F. **Parasites** ; Stomach worms—Trichostrongylus, Hemonchus, Ostertagia, larval paramphistomes in ruminants. Larvae of Habronema and *Gasterophilus equi* in horses. In pigs *Hyostrongylus rubidus, Ascarops strongylina* and *Physocephalus sexalatus*.

In general, close confinement and insanitary conditions where bacteria thrive contaminating feeds and feeding utensils are predisposing factors.

Gastritis may be acute or chronic. Acute gastritis may be catarrhal, fibrinous, suppurative, hemorrhagic or necrotic, depending upon the cause and their severity. By far the most common is the catarrhal and to a lesser extent, the hemorrhagic.

Catarrhal gastritis: Macroscopically, there is increased thickening and reddening of the gastric mucosa. A thick mucous exudate will be found covering the mucosa, which in some places may show ulceration. If severe, there may be hemorrhages and the gastric contents may be blood stained.

Microscopically, usual characteristics of inflammation—hyperemia, exudation and leucocytic infiltration in the mucosa are noticed. Some of the gastric glands may be damaged and lost.

Acute hemorrhagic gastritis is also common. Due to hemorrhage, the mucosa is bright red in color and the gastric contents are blood stained. Digested blood imparts a brownish coloration to the contents Besides, the changes described under catarrhal variety are seen Hemorrhagic gastritis is met with in acute infectious and intoxicating diesases like pasteurellosis, braxy, uremia, leptospirosis (in dogs) and in caustic chemical poisoning.

In gastritis, food does not get digested, motility of the gastric wall is retarded and irritation may produce pain and vomition.

PARASITIC GASTRITIS

Special mention must be made of parasitic gastritis, since this is very common in animals. The parasites that produce gastritis are:

Pigs:—*Hyostrongylus rubidus, Physocephalus sexalatus. Simondsia paradoxa, Ascarops strongylina.*

Cat:—*Gnathostoma spinigerum.*

Cattle:—*Hemonchus contorius, Ostertagia ostertagi, Trichostrongylus axei,*

Sheep—Same as above

Horses:—*Trichostrongylus axei, Gasterophilus equi larvae; Habronema larvae.*

The strongyles are blood suckers and they produce minute injuries on the mucosa The larvae may burrow into the mucosa for completion of their life cycle and thereby cause damage to the glands and epithelium. Heavy infestation, besides causing anemia, will produce cattrahal gastritis. Gasterophilus in the stomach may produce ulcers while Habronema larvae live in granulomatous nodules which may be infected by secondary bacteria and which produce abscesses

CHRONIC GASTRITIS in animals has usually the same causes as the acute but operating for a longer time. Sometimes it may be secondary to chronic gastric dilatation and hepatic cirrhosis. Partial anemia in the former and passive hyperemia in the latter decrease the local resistance thereby facilitating infection

This condition is usually of a hypertorphic type with thickening of the gastric wall. The mucous membrane is thickened and covered with tenacious, viscid glassy mucus. Mouths of the glands may be occluded resulting in retention cysts.

Microscopically, there is desquamation of the epithelium with increased interstitial connective tissue and this is the cause for exaggeration of mucosal foldings. Hyperplasia of gastric glands and hyperplasia of muscle fibres with infiltration of inflammatory cells and hypertrophy of basal lymphocytic nodules are other features seen. The mucosa may be thrown into polypoid folds giving rise to *polypoid gastritis.*

GASTRIC ULCERS are common among animals. Calves are more often affected. Usually gastric ulcers run an acute course, heal promptly and seldom become chronic as in man. So most often these ulcers are seen postmortem. Small superficial defects are known as *erosions* and these are common among ruminants Gastric ulcers of animals rarely perforate.

The exact causes of gastric ulcers are still obscure. The following are some of the causes cited.

1. **Trauma :** This appears to be the commonest cause of abomasal ulcers in calves. In early weaning of calves (before fourth week after their birth) when they are put on roughages before the fore-stomachs have attained their full functional development and before rumination has started, the coarse plants irritate the tender gastric mucosa producing gastritis which results in ulcer formation.

2. **Infections;** Erosions are common in the abomasum of cattle in rinderpest, mucosal disease, pox and bovine malignant catarrh.

3. **Circulatory disturbances;** In intense hyperemia (stasis) focal hemorrhages by diapedesis, that may occur through vascular nerves, the involved epithelium, because of disturbed nutrition, undergoes autolysis or even nercrosis and then is acted by the gastric juice: which digests it. Because the hemoglobin is digested by acid the area becomes brown in color. Such vascular stasis and hemorrhages are found in many infectious diseases—foot and mouth disease, canine distemper, rinderpest, rabies, purpura hemorrhagica and in uremia in which toxemia is present.

4 **Nervous effects;** The sympathetic and the vagus nerves control the secretion and blood circulation of abomasum. Any factors that disturb these nerves may produce stress, hemorrhages and increased secretion of glands Stress in pregnancy and abomasal displacement may cause such disturbances and so it is that abomasal ulcers are common in post-parturient dairy cows.

Vasoconstriction produced by adrenaline in stress is thought to be another factor. Vagus indigestion causes atony of abomasum and ulcers are thereby caused there.

5. **Obstruction of pylorus** in calves and adult cattle is another cause of gastric ulcers. In calves pylorus may be obstructed by foreign bodies or coarse feed. In adult cattle pylorus obstruction occurs in traumatic reticulitis and in abomasal displacement.

6. **Chemicals:** Corrosives and escharotics may act either directly on the mucosa or indirectly through their action on the nerves. Grazing on pastures heavily fertilised with nitrogen is associated with gastric ulcers.

7. **Parasites:** In horses larvae of *Gasterophilus* species and *Hebronema megastoma* cause gastric ulceration.

8. **Neoplasms** of the stomach, especially lymphocytoma, are accompanied by ulceration.

9 **Nutrition:** Nutritional hepatic dystrophy (due to deficiency of vitamin E) in pigs is associated with gastric ulceration. Occurring in the young pigs, there may be heavy mortality due to massive hemorrhage.

10. **Fungi :** Mucor sp. and Monilia sp. cause gastric ulcers in pigs.

Macroscopically, the erosions (in cattle) are of the size of a millet and usually affect the mucosa superficially. Slowly, by the action of the gastric juice the erosions may enlarge and become deeper to form ulcers. Sometimes blood vessels may be eroded and fatal hemorrhages may result. But this is not so common in animals as in man. The ulcers are usually demarcated, having raised borders and so have a punched-out appearance. The base of the ulcers may be the propria or submucosa or muscular coat or in some cases be even the serous coat. When the serous coat is the base, perforations are likely to occur.

Miroscopically, the erosion is covered by an exudate consisting of mucus, fibrin and inflammatory cells. The epithelium is desquamated and the sub-mucosa may be infiltrated by large number of leucocytes.

Fate of the ulcers: Ulcers develop very rapidly. The abomasal ulcers develop in three to four days. Perforation, when it occurs, may happen in six to seven days.

Most of the gastric ulcers heal by the formation of granulation tissue, which fills from the base of the ulcer. The resulting scar tissue (which contracts) is covered by the epithelium of the mucosa. Destroyed glands are not regenerated. Healed gastric ulcers have a star-like appearance.

Hemorrhages and perforation resulting in fatal peritonitis are the complications seen in some ulcers. Stress that may occur in lactation may be one of the causes of perforation of abomasal ulcers in cattle. Progressive anemia and emaciation may result in some cows.

Neoplasms : Most common is the lymphosarcoma of the wall of the abomasum in the bovine. Other tumors seen are : Leiomyoma, adenocarcinoma.

THE INTESTINE

Congenital anomalies : Atresia of the intestine may be found as a hereditary defect among cattle.

In pigs and foals atresia of rectum is an inherited lethal characteristic.

In cattle, imperforate anus at birth is a semi-lethal character.

Persistent omphalomesenteric duct is known as Meckel's diverticulum, which is sometimes met with in pigs and horses and is probably hereditary. This may produce complications since it may become obstructed or inflammed or even ruptured resulting in peritonitis.

Stenosis of the intestines sometimes occurs congenitally.

CIRCULATORY DISTURBANCES

Acute passive hyperemia occurs in conditions where there is sudden obstruction to the venous out flow as in prolapse, torsion, hernia or intussusception. The involved part is dark-red in color, and swollen. There may be effusion into the peritoneal cavity and edema of the intestinal wall. Hemorrhages are common. Due to acute passive congestion, the following may result : enteritis, necrosis, gangrene, rupture, peritonitis and death.

Chronic passive congestion is common in liver diseases or obstruction of the portal vein or may be part of the general venous congestion (with lesions in the heart and lungs). The veins are dilated and stand out prominently. The intestines become predisposed to enteritis. The intestinal wall is thickened and edematous and ascites is also found. In long standing cases, there is fibrous thickening of the wall and atrophy of the glands.

Hemorrhages are common on the mucosa and serous surface of the intestines. The causes are;— blood sucking parasites, hemorrhagic diseases, enterotoxaemia (in sheep) and specific infections in which this is a lesion.

Infarction : If circulation is obstructed either by embolus or from pressure from the out side, infarction may result. Putrefactive organisms normally resident in the bowel invade the infarcted part producing gangrene.

Thrombosis : Thrombosis of the intestinal vessels is common in the horses and is mostly due to larvae of *Strongylus vulgaris*.

The infective larvae burrow into the mucosa and travel along the intestinal arteries, crawling against blood flow along the intima and reach the anterior mesenteric artery where they settle. At that place the endothelium is damaged and a thrombus is formed. This thrombus may become organised and canalised. Complete organisation of the thrombus is prevented by the penetration of larvae

which produce irritation. The damage caused by the penetration of larvae as well as their continued presence so weakens the wall of the artery that an aneurysm may develop. Due to the replacement of the elastic tissue by fibrous tissue the resilience of the arterial wall is curtailed and so it cannot contract when stretched and so dilates (aneurysm). The wall may enlarge gradually and sometimes may rupture. Calcification of the fibrosed wall may sometimes occur but this still more weakens the wall.

Of various branches of the anterior mesenteric artery, it is in the right branch that thrombus more often occurs. This branch supplies the ventral colon and so thrombosis of the vessel produces ischemia of these parts. Ischemia of the parts results in atony of the musculature leading to decreased peristalsis and so to stagnation of and impaction by the ingesta. Gas may be produced if fodder is succulent. Accumulation of gas and impaction give rise to colic.

Emboli from the thrombus may occlude some branches of the artery resulting in infarction, gangrene, shock and death.

Mechanical obstruction

Causes : Congenital : Atresia and imperforation have already been described.

Acquired : (a) **Stenosis** may be due to pathological lesions: hematoma, neoplasms, abscesses, and chronic inflammatory scars, displacements like torsion, volvulus, intussuception, hernia.

(b) **Impaction :**

(i) Foreign bodies—Bone, stones, cartilage, rubber ball, rags, golf balls.

(ii) Hair balls—in cats.

(iii) Impacted undigested coarse food, especially in the horse; sudden changes in feeds and faulty dentition are accessory factors. May also be found in dogs—coproliths.

(iv) Impacted meconium in new born animals.

(v) Parasites—masses of round worms in pigs and fowls and tape worms in sheep.

(vi) Enteroliths.

(vii) Neoplasms—lipoma in horses.

Obstruction causes weak peristaltic movements of the bowel above the point of obstruction, resulting in dehydration of the contents at that place. Spasms with violent contractions of the gut above the place of obstruction causes intense pain (colic) and sometimes rupture may occur. Vomition is an usual symptom of intestinal obstruction in dogs and cats.

Macroscopically, the place where obstruction has occurred is found to be distended. The contents are hard, which pressing on the mucosa may cause necrosis and erosion. Ultimately stenosis may develop at this part. Rupture and peritonitis leading to death may result if the obstruction is not relieved.

TORSION is a twisting of intestines on its axis.

VOLVULUS is a twisting of the bowel on itself as occurs when it passes through a tear in the mesentery. These conditions seen in horse more frequently may also be met with in other animals.

Causes : 1. Violent movements as in rolling and struggling.

2. Violent peristaltic movements.

3 . Foreign bodies—sand or enteroliths,by their weight make the part heavy and aid in its winding around other parts.

4. Gas. Accumulation of gas makes the part bulge and twist round other viscera.

Torsion occurs more often in the small intestines, which have a long mesenteric attachment. In the horse, the right colon is fixed by ligaments and so torsion occurs in the left and transverse colon. In the cattle torsion of cecum is more common.

The changes that occur in torsion are — acute passive congestion leading to edema, hemorrhage, gangrene, peritonitis and death.

Macroscopically, the affected portion is swollen and darkened in color. The wall is very easily torn. Peritonitis may be evident in some

INTUSSUSCEPTION is telescoping of a portion of intestines into another, usually the anterior into the posterior, and occurs mostly in the jejunum and cecum in dogs and cattle. Along with the portion of intestines, its mesentery also is dragged along and so there is compression of the thin-walled veins resulting in acute passive hypermia.

Macroscopically, the affected part is dark-red or bluish and swollen. Usually gangrene and peritonitis supervene terminating in death.

In some stray cases, the invaginated portion may be. sloughed off, healing occurring by granulation tissue (Epithelium covers the scar. But at the site of scar. circular stenosis may form. Death in volvulus and other intestinal displacements is due to : 1. Acute anemia which may occur due to extensive hemorrhages into the intestine and peritoneal cavity. 2. Asphyxia and heart failure due to compression of lungs and heart by pressure on the diaphargm by excessive gas formation 3. Rupture of intestines and stomach leading to peritonitis and absorption of toxic products. 4. Toxemia due to absorption of toxins from decomposed food and bacterial growth .

INCARCERATION of the intestine is trapping of the intestine internally, from pressure on its external surface. Incarceration may occur due to.

(i) adhesion of the intestine to other abdominal organs.
(ii) the loop of intestine may pass through the epiploic foramen of of Winslow
(iii) occasionally a persistent urachus may cause incarceration.
(iv) Similarly an adhesion to the uterus may cause this condition.
(v) when the bowel passes through a fissure of the mesentery, congenital or acquired, incarceration may supervene

The changes in these conditions are similar to those found in acute passive congestion viz. stagnation of the intestinal contents followed by venous stasis due to non-return of venous blood as the thin-walled veins are compressed; edema, infarction, gangrene and peritonitis. Ultimately rupture, shock and death occur.

PROLAPSE OF THE RECTUM : Sometimes the rectum protrudes through the anus. The causes are straining, irritation, abdominal pressure, diarrhoea, increased peristalsis and constipation.

Macroscopically, the rectum, bright-red in color, will be found hanging through the anus. It may be edematous and soon becomes gangrenous. The changes are similar to those found in incarcerated intestines. If not attended to early, the prolapsed rectum will be pecked by fowls or injured by swine. Due to swelling, fecal matter cannot be voided. Antemortem prolapse can be distinguished from the post-mortem prolapse by the absence of congestion in the latter.

HERNIA of the abdominal organs is the protrusion of the abdominal viscera through a natural or artificial opening. Hernia of intestines is commonly seen in domestic animals, especially the pig and horse.

The intestines may pass through a natural opening, the internal inguinal opening which is patent in the males. The umbilicus, if not healed is another site of herina. Other causes are trauma when the abdominal muscles may rupture or even the diphragm may tear resulting in the intestines passing through the opening. *Violent straining* during parturition or defecation may also be another cause.

Depending upon the location, hernia may be *external* or *internal* (diapharg-matic, pelvic).

Among the external, are;—

The **ventral**, when the abdominal muscles are ruptured. This is common in horses (spontaneous in pregnant mares) and occasionally in cattle. Causes; Trauma:- Horn injuries, kicks, automobile accidents, laparotomy and castration scars. In pregnant ewes this may due to muscular degeneration of nutritional origin.

The **umbilical** when the bowel passes through a congenital or acquired defect of umbilicus and seen in foals, calves and pups.

The **inguinal** when the bowel passes through the internal inguinal ring. This is not so common in animals as in man, because of horizontal position: seen in colts and pigs.

Scrotal herina: the intestines slide into the tunica vaginalis along the inguinal canal in contact with the spermatic cord. The testes may undergo thermal atrophy when in contact with the intestines.

Femoral hernia may develop when the omentum and intestines pass through the femoral triangle along the femoral artery and so the bowel is found on the inner surface of the thigh.

The perineal hernia may occur in old dogs due to violent straining in cases of enlarged prostate.

External hernia consists of; — (i) a hernial sac formed by the parietal peritoneum and the covering skin, (ii) a hernial ring which is the opening in the abdominal wall and (ii) the hernial contents.

If the hernial contents can be returned into the abdominal cavity it is called a *reducible hernia*. But if it cannot be so returned is is called *irreducible*. The causes of the latter are;— (i) adhesions between the visceral mass and the hernial sac. (The adhesions arise due to inflammation of the peritoneum) (ii) accumulation of ingesta in the loop of intestines making it too bulky to be reducible and

(iii) venous stasis, due to incarceration, whereby the volume is so increased that, the bowel cannot be reduced.

If the hernia does not have a parietal peritoneal covering of the viscus, it is called a *false hernia*. In such cases, opening of the skin will reveal the bowel. The condition is called *eventration*.

Strangulated hernia is one in which the blood supply is cut off by the pressure of the hernial ring through which the intestines pass. If not relieved in time, this condition is fatal since infarction, gangrene, peritonitis and shock will develop within 24 to 36 hours.

ENTERITIS

Enteritis is the term denoting inflammation of the whole of the intestinal tract. But usually it is applied to the inflammation of the small intestines. The inflammation of the colon is called *colitis*, that of cecum *typhlitis* and of rectum *proctitis*.

Enteritis is very common in domesticated animals and fowls and is of immense economic importance.

Since enteritis occurs along with gastritis (the same irritants causing gastritis passing on to intestines produces enteritis also) gastro-enteritis is a frequent condition met with.

Causes are many and varied and they include, bacteria, viruses, protozoa, rickettsia, helminths, fungi, chemicals, disturbed metabolic processes as in ruminants, venous congestion as in portal hypertension and congestive cardiac failure, toxins of clostridia, coliforms and spoiled or mouldy feeds and avitaminosis. In enteritis, the whole length of the bowel may not be affected, inflammation localising only at one part or the other.

Based on the nature of the exudate and the changes produced in the intestinal tract, enteritis is classified, as catarrhal, hemorrhagic, fibrinous, suppurative and necrotic.

Acute catarrhal enteritis is the mildest of inflammations of the intestinal tract, occurring in a diffuse manner throughout the bowel.

Causes include mild irritants like foreign bodies, sand, coarse feeds, bites of parasites (hook worms), chemicals and drugs, *Vibrio coli* (causing winter diarrhoea in cattle). Acute catarrhal enteritis may be noticed in :—

i) Enteritis in sucklings—scours in calves, lambs, foals and piglets caused by *E. coli*, pasteurella, salmonella, proteus, vibrios and streptococci.

In calves and lambs avitaminosis A is a predisposing factor while in young pigs deficiency of animal proteins and trace elements predispose them to infections. In such a state, the organisms are able to gain a foothold and thrive causing the disease.

ii) Viral Diarrhoea-Mucosal Disease in cattle.
iii) Enterotoxemia in sheep.
iv) Virus gastroenteritis in pigs.
v) Salmon poisoning in dogs.
vi) B W.D, infectious cloacitis, pullet disease and ornithosis in fowls.
vii) Oral antibiotic therapy may cause enteritis in two ways : (1) these may

themselves be irritants or (2) they may so alter the intestinal flora that there is over growth of other bacteria (*Staphylococci, Proteus* sp; *Pseudomonas* sp) and fungi *(Candida albicans)* which are normaly kept under restraint and so enteritis results.

Macroscopically, one should be able to distinguish this condition from the normal hyperemia that occurs during active digestion. The mucosa is reddish in color and slightly thickened, covered with a mucinous exudate. The Peyer's patches are prominent being hyperplastic, outlined by a zone of hyperemia.

Microscopically, the edema of the intestines is due to exudate with leucocytes in the lamina propria and to a little extent ia the submucosa. Hyperemia is evident by the engorgement of the blood vessels. Goblet cells are numerous arising from metaplasia of the epithelial cells and produce large amounts of mucin. The tips of villi may be reddened and edematous.

The intestinal contents are watery, consisting of mucus, fibrin and desquamated epithelial cells.

Sequelae : When cause is removed inflammation may subside and the bowel returns to normal. But if irritant persists, the condition may develop into the chronic state.

Chronic catarrhal enteritis may develop from the acute condition or more usually it may arise gradually as in Jhone's Disease, intestinal helminthiasis, chronic venous congestion (due to congestive cardiac failure) and cirrhosis of liver.

Macroscopically, the wall of the intestines is greatly thickened. The mucosa is smooth (covered by thick mucus) and thickened due to infiltration by macrophages, plasma cells and lymphocytes. This infiltration makes the mucosa corrugated due to infoldment. The corrugations are at right angles to the length of the intestines ; *(Chronic polypoid enteritis)*

Microscopically, the characteristic appearance is the presence of numerous macrophages, plasma cells, lymphocytes and connective tissue cells in the lamina propria and even in the sub-mucosa. The intestinal glands are atrophied due to the pressure of the infiltrating tissue. Sometimes retention cysts due to closure of the mouths of glands are found. The mucosa is covered with tenacious mucus.

Hemorrhagic enteritis : This is a more severe form of catarrhal enteritis, characterised by the presence of erythrocytes in the exudate. Always patchy in distribution, this is mostly seen in septicemic, bacterial and viral diseases e. g. Anthrax and Rinderpest. This condition may also be found in uremia of dogs ; coccidiosis, poisoning by arsenic and croton oil, enterotoxemia, in vitamin B deficiency in dogs and pigs and in colibacillosis. Continuous feeding of dogs with horse meat causes an anaphylactic condition manifested by hemorrhagic enteritis.

Macroscopically, there is infiltration of blood in the intestinal wall which is thickened and the intestinal contents are blood stained. Blood found in the anterior portion of the intestines is digested and so is brown in color while in the posterior portion it is bright red.

Microscopically, red blood cells may be found in the exudate of the mucosa. The villi may show necrotic changes and thrombosis of some enteric vessels is evident.

Sequelae: Being very severe, death usually occurs. If treated in time, prognosis may be favourable.

Fibrinous enteritis : This is of the diphtheritic type and occurs in cattle, pigs and cats, and rarely in horse and fowl.

Causes:

Bacteria; *Salmonella cholerae suis* (necrotic enteritis in swine), *Escherichia coli*

Chemicals; salts of mercury and arsenic.

Parasites: *Echinostomum*, a fluke in turkeys

Macroscopically, the characteristic finding is the presence of strands of fibrin on the mucosa of the intestines. The wall of the intestine is edematous. In more severe conditions, a thick, grayish or whitish-gray membrane may be covering the intestinal mucosa, which is hemorrhagic and edematous The inflammation may extend into the submucosa and petechial hemorrhages may be seen. The mesenteric lymph glands are swollen, hemorrhagic and juicy.

Macroscopically, the membranous exudate consists of strands of fibrin containing in its meshes varying number of neutrophils and desquamated epithelial cells together with mucosa. Mucous membrane shows edema, hyperemia and infiltration by neutrophils. Coagulative necrosis of the epithelium occurs in some places, which along with the exudate forms the false membrane which is adherent to the intestine.

Sequelae; Being very a severe condition death is a common sequel. In those that are able to withstand, recovery with complete healing occurs.

Suppurative enteritis is not common and may result due to infection by pyogenic organisms of wounds caused by helminths.

Causes; Pyogenic organisms ; *Streptococci, Salmonella & Shigella.*

Macroscopically, the exudate contains pus.

Microscopically, the exudate contains, besides mucus, desquamated cells neutrophils and bacteria.

Necrotic enteritis : Necrosis of the intestinal epithelium and underlying tissues occurs.

Causes; Severe irritants; Chemical — croton oil, mustard gas, wood preservatives; insecticides.

Bacterial—Necrophorus organism, *Salmonella.*

Viral—Rinderpest, Viral Diarrhoea—Mucosal Disease, Hog cholera;

Protozoa—Coccidiosis, Histomoniasis.

Vitamin deficiency—Niacin deficiency in swine.

Macroscopically, patchy necrotic areas are seen. The necrosis of the mucosa extends into the sub-mucosa also. Fibrin may be found on the necrotic mucosa. When the necrotic material is removed, a red, raw, bleeding surface is seen. The mesenteric lymphatic glands are swollen and juicy. In hog cholera, the characteristic lesion is the "button ulcer", which is a spherical ulcer in the mucosa of the colon. This is circumscribed with sharp edges.

Microscopically, besides hyperemia, exudate and cellular infiltration, necrosis of the epithelium of the mucosa is seen. The ulcer reveals a demarcated zone of necrosis in the mucosa and sub-mucosa.

Actually, these button ulcers are tiny areas of infarction that arise by the occlusion of small arteries by the swollen and proliferated endothelium.

Sequelae : The condition is mostly fatal. If the condition is one of niacin deficiency, restitution of the deficiency may cure the condition.

ENTEROLITHS; sometimes stones are found in the large intestines of horses. They are formed of triple phosphates which are deposited concentrically, layer after layer, over a nucleus of sand or a metal piece or an undigested vegetable fibre.

Genesis ; When animals are fed on wheat or bran which are rich in magnesium phosphate, intestinal calculi can occur. Normally magnesium phosphate is dissolved by the gastric juice and then absorbed in the intestines. On the other hand, when excessive amounts are fed to an animal, and that too to one suffering from chronic catarrhal gastritis in which gastric juice is not secreted, much of the magnesium phosphate reaches the intestines in an undissolved state. This combines with ammonia that is formed from the decomposition of protein (which is also abundant in wheat and bran) to form triple phosphate. This triple phosphate crystalises around foreign bodies like a grain of sand, a piece of metal or undigested plant fibre Enteroliths do not form in the small intestines because (i) the movement of the food is too rapid there to allow the deposition of salts and formation of calculi and (2) bacterial decomposition of proteins to form ammonia does not take place there. The following may be contributory factors: disturbance in the colloid protection of dissolved salts; change in bacterial flora, with altered fermentation conditions and sluggish intestinal movements that occur in the dilatation of the bowel or in relaxation of intestinal muscle met with in feeding with bran. Enteroliths may sometimes attain a large size, some may weigh as much as 20 lbs. and are usually round and smooth.

Phytobezoars ; These are food balls (phyto=plant, bezoar=concretion)

These arise from plant fibres and awns which are impregnated with triple phophate and rolled into balls. These have a velvety surface, are light in weight and are brown in color. They may also be found in the crops of birds

Trichobezoars (Piliconcretions)—Hair balls.

Hair balls are found mostly in the rumen. Animals having itching skin conditions (animals infected with mange or lice) may lick each other when the loose hair may be swallowed. Similarly, calves kept together, suck and lick each other's ears, tails etc. swallowing hair. The hair is rolled into balls during ruminal contractions. Mucus of rumen may form a smooth coat over such balls.

The enteroliths, phytobezoars and trichobezoars are usually of no consequence unless they obstruct the passage, when, sometimes, even rupture may occur. Cattle may regurgitate a food ball into the œsophagus which may be choked More often these concretions are found only at postmortem.

COLI GRANULOMA IN FOWLS (Hjarre's Disease)

A granulomatous condition of the fowl intestine and liver, caused by a mucoid strain of *E coli* is reported in many parts of the world including India. Usually adult fowls are affected.

Macroscopically, a large number of grayish - white nodules varying in size from a millet seed to a hazel nut are found projecting from the serous surface of the intestines. These are distributed diffusely from the duodenum to the ceca. The large intestines are free of the lesions. In some places, the whole circumference of the bowel may be involved.

Microscopically, the lesion is a granuloma involving all the structures of the bowel wall, with desquamation of the mucosa and fibrous thickening of the serosa.

A typical nodule consists of the following structures from within out-wards: a central structureless mass which is calcified in some places; peripheral to this is a zone of caseo-necrotic material with cells in varying stages of degeneration and necrosis; peripheral to this is a zone of granulation tissue, with epithelioid cells and a few giant cells interposed in between it and the previous zone. *E. Coli* could be demonstrated in and isolated from the lesions. Similar lesions are found in the liver.

The following are the differences between the lesions of coli granuloma and tuberculosis. The lesions of coli-granuloma are single while those of tuberculosis form conglomerates; lesions of coli granuloma are not found in spleen and bone while tuberculous lesions are found in those situations; in tuberculous lesions the acid-fast organisms can invariably be found while in coli-granulomatous lesions only *E. coli* are seen.

Neoplasms of the intestines: Lymphocytoma is the most common neoplasm met with in animals. Masses of the neoplastic cells are found in the wall of the bowel.

Other tumors met with are; — adenocarcinoma, lipoma, leiomyoma, sarcoma and papilloma.

Anal glands of dogs may sometimes be inflammed and may become purulent if infected by pyogenic organisms. There may also be swelling of the glands due to retention of the secretion. These swellings may cause constipation and so need to be manually evacuated.

Adenoma of the anal glands has been met with.

COLIC

Colic means pain in the abdomen. This is a symptom manifested by animals suffering from diseases of various organs. Animals suffering from colic have an anxious look, lie down and get up frequently, roll on the ground, look towards the flank often, have polypnoea and tachycardia. Though all animals may suffer from colic it is the horse that is most often affected.

Colic is not a term used by the pathologist. But for the convenience of the students, the conditions in which colic is a symptom are listed below (after Cohrs)

Diseases of the stomach: acute and chronic dilatation of the stomach (caused by overfilling of the stomach or by pyloric stenosis), gastritis, gastric ulcers, gastric parasites.

Diseases of the intestines; Volvulus; torsion; intussception, stenosis, displacement, obstructions, impaction, retention of meconium, enterolithiasis, foreign bodies, thrombosis and embolism of mesenteric vessels, enteritis, parasites.

Acute peritonitis.

Diseases of the liver and bile passages : impaction of gall stones, acute hepatitis; sudden enlargement of the liver due to hemorrhage, rupture of the liver:

Diseases of the urinary organs : acute nephritis; renal abscess, pyelitis, displacement with obstruction of the ureter, cystitis, urethral obstruction.

Diseases of the genital organs; torsion of the uterus, uterine contractions associated with the movements of the foal, labour pains during normal parturition or abortion, enlargement of the prostate causing retention of urine.

Diseases of other organs Certain diseases of the esoghagus (dilatation, stenosis, displacement, contractions), irritation of the rectum, and the surrounding tissues by parasites (*Oxyuris, Gasterophilus)*, equine myoglobinuria associated with paralysis, hunger and extreme exhaustion.

Of all horses examined postmortem, colic has been responsible for 34 to 50 percent of deaths. The following are the most common causes of colic among horses: volvulus of small intestines, volvulus of large intestine, other forms of displacements, primary gastric dilatation, obstruction of small intestines, cecal impaction, impaction of large intestines, thrombosis and embolism of mesenteric vessels, and enteritis.

Chronic or recurrent colic, which is not so frequent may also occur and is due to embolic thrombosis, chronic impaction of the cecum, chronic dilatation of the stomach and intestines, obstruction causad by adhesions, old incarcerations, tumors, stenosis and inflammatory new growths; stones in the bowel and parasites.

LIVER

In addition to being the largest organ in the body, liver also discharges the greatest number of functions. Hepatic cells are among the highly specialised cells in the body. At any one time, 25% of the blood in the body flows through the liver.

THE FUNCTIONS OF LIVER ARE :

1. **Secretion of bile;**—Bile contains pigments and bile salts. Bile pigments are not useful to the body. On the other hand, retention of these (hyperbilirubinemia) is toxic to the body.

Bile salts are formed in the liver from cholesterol, and excreted as sodium salts of taurocholic and glycocholic acids after conjugation with taurine and glycine. These bile salts play an important role in digestion, especially of the fats.

The bile sults;— (a) activate pancreatic lipase and amylase;

(b) aid in the emulsification of fats in the intestines.

(c) aid in the absorption of fats.

(d) aid in the absorption of fat soluble vitamins (especially vitamin K).

(e) act as cholagogues and

(f) maintain a stable pH in the Intestines.

Bile contains mucin and related substances which act as stabilisers for the fat emulsion in the bowel.

Though not strictly antiseptic, bile renders the intestines uncongenial for the bacteria to thrive and so is bacteriostatic.

2. **Protein matabolism;** (a) Amino acids are deaminised.

(b) Uric acid is converted into allantoin.

(c) Highly toxic ammonium salts are detoxified by converting them into urea.

(d) The non-nitrogenous residues obtained after deamination of amino acids, are converted into glucose and ketones which are used by the body.

(e) From Amino acids are formed :

i) Plasma proteins (albumin, globulin, fibrinogen, prothrombin).

ii) Tissue proteins and

iii) Protein reserves stored in the liver.

3. Carbohydrate metabolism : Glycogen is synthesized and stored in the liver. Excessive carbohydrates ingested are converted into lipids and stored in the fat depots. With the assistance of pancreas, liver maintains a constant level of blood glucose. In times of need, gluconeogenesis from proteins and fats occurs in the liver.

4. Fat metabolism : We have already seen how bile salts assist in the absorption of fats. Fats, that are characteristic of animals, are also synthesized from fatty acids and glycerol by liver

With the assistance of choline, liver is able to transform the depot fats into tissue fats (phospholipids) so that the tissues can utilise them.

5. Erythropoiesis : In the bird, liver is the site for erythropoiesis. In other animals, during fetal life, erythropoiesis occurs in the liver In these animals under certain circumstances (in severe anemias) erythropoiesis takes place in the liver even in the adult, i.e., extramedullary hemopoiesis.

6. Iron metabolism : The reticulo-endothelial cells of the liver are capable of destroying the red blood cells and the minerals released (Fe, Cu and Co) are storted in the liver for use again by the body.

7. Detoxication : Some toxic substances, especially putrefactive products from the alimentary tract are detoxified by the liver by conjugation while bacterial toxins and hormones produced in excess of requirements are inactivated. Many drugs used therapeutically are also made harmless by the liver, eg. morphine, barbiturates, phenol, camphor.

8. Vitamin metabolism and storage : Failure of bile excretion due to hepatic damage interferes with the absorption of fat-soluble vitamins—A, D, E, and K. Vitamin A is stored in the liver and Vitamin K is utilised there for the formation of prothrombin and so these functions will be interfered with in the diseases of liver. Some members of the vitamin B group, especially Thiamine, Riboflavin and Niacin are partly metabolised in the liver where they may also be stored.

It is, therefore, obvious that with severe disease of the liver, a great many vital processes will be affected. The following are the more important pathological conditions met with :

1. Jaundice due to retention of bile.

2. Bleeding may be due to (i) failure of prothrombin formation; (ii) lack of absorption of Vitamin K; (iii) lack of formation of fibrinogen.

3. Hypoglycemia due to impairment of glucose metabolism: glycogen is not stored in the liver nor is it released into the blood. This condition makes the animals weak and irritable.

4. Hypoproteinemia—due to failure to synthesize plasma proteins. Animal becomes emaciated and generalised edema develops.

5. Anemia due to iron and protein deficiencies; liver stores iron and so in liver disease iron stores are deplete t

6. Toxemia due to failure in detoxication of proteins and intestinal toxins.

7. Renal failure—Heptorenals syndrome—in severe hepatic injury, the toxins that are not detoxified are excreted through kidneys, which are affected by these toxins resulting in renal degeneration. Renal function suffers—uremia develops.

8. Pyrexia—the heat regulating center is affected by the toxins since they are not detoxified by the injured liver.

Liver Function tests –

Several tests have been evolved to measure different functions of the liver. It must be remembered that liver has a great reserve power and it has enormous ability to recover from injury. So the tests are not adequate, clinically, to evaluate the correct state of the health of liver and hence it is not wise to put too much reliance on these tests. Since the functions of the liver are carried out by the activity of enzymes, inadequacy or absence of one particular enzyme may affect one function and so a decrease of one function does not mean that other functions are affected.

Postmortem changes : Postmortem decomposition of liver occurs rapidly since gas-forming organisms *(Cl. welchii)* invade from the intestines which are close by. Liver, particularly rich in nutrients, is a good medium for the growth of these bacteria. Gas bubbles form in the blood vessels. The parenchyma and the blood vessels adjacent to the bowel are stained by hemoglobin blush-black. The presence of gas gives the liver a foamy appearance—"Foamy-liver". Imbibition of bile by the liver tissue surrounding the gall bladder is noticed.

DEGENERATIONS

Cloudy swelling is common in the liver :—

Causes : i) Bacterial toxins : seen in all infectious diseases.

ii) Poisons : (a) Chemicals : salts of heavy metals—arsenic and lead.

(b) Plant toxins—glucosides, saponin.

(c) Drugs—carbon tetrachloride used as an anthelmintic

iii) Viruses.

iv) Hypoxia.

Macroscopically, the liver is enlarged and the capsule is tense. Consistency is softer Borders are rounded. The organ has a dull, cooked appearance. On section it bulges at the edges. Lobular marking are indistinct.

Microscopically, the cells of the liver are swollen and have a pale granular cytoplasm due to swelling of mitochondria. The nuclei may be indistinct.

Sequelae : Recovery of the cells occurs if cause is removed. But if it continues the condition may progress to fatty degeneration or necrosis.

Fatty change in the liver is common and sometimes may be of severe degree.

As explained in general pathology, all visible fat in the liver is due to fatty infiltration. The liver is too sick to metabolise the fat brought to it from the depots.

The main causes are : Toxins, poisons and anoxia.

Poisons : Inorganic—phosphorus, arsenic, antimony.

Organic : chloroform, carbon tetrachloride, tannic acid, tetrachlorethylene alkaloids of phytotoxins, Aflatoxin, senecios

Anoxia : Chronic venous congestion.

Nutritional: Inadequate choline; Metabolic — Diabetes mellitus in dogs and cats, deficiency of thyroxine and anterior pituitary hormones.

Macroscopically, the liver is enlarged, has a smooth surface and is pale or yellowish. On section it bulges on the cut surface and fat droplets are seen on the blade. Very fatty livers, as in pregnancy toxemia of sheep, float in water. They are friable.

Microscopically, the hepatic parenchymal cells contain fat droplets, either as a single large globule or as multiple small globules If single and large the nucleus may be thrust to a side. The sinusoids are compressed and so appear anemic. Usually the distribution of the lesions in the liver may be diffuse or zonal. In chronic venous congestion it is in the periphery of the central vein. But on the other hand, in poisoning, when poison is brought through the portal vein, the fatty changes are found at the periphery of the lobule

Sequelae : If the cause is removed early, the condition can be completely corrected, But in continued presence of the pathogen hepatic fibrosis—portal cirrhosis—will eventually result.

HEPATITIS

Essentially hepatitis is an alterative inflammation of liver in which the various degenerative processes like cloudy swelling, fatty degeneration and necrosis are caused by irritants which also produce inflammation. Besides, in liver these degenerative changes are accomoanied by lmphocytic and exudative infiltrations typical of an inflammatory reaction. Hepatitis is classified as *alterative inflammation* because the inflammatory process is caused by the same etiological agents that also produce degeneration and so alteration in the parenchymatous cells is produced.

Hepatitis may be either infectious or non-infectious or toxic. The latter may again be acute or chronic. The chronic variety is usually called **Cirrhosis.**

Infectious hepatitis ; This is found in various infections as detailed below.

A. Conditions in which the liver is only or primarily affected

(i) Infectious Canine Hepatitis (Ruharth's disease)
(ii) Blackhead in turkeys (Histomonas affections)
(iii) Wesselsbron disease of sheep, a viral disease found in South Africa
(iv) Leptospirosis.
(v) Viral hepatitis of ducks.
(vi) Viral hepatitis of poultry.

B Conditions in which liver is also affected along with other organs;

(i) Necrobacillosis
(ii) Suppurative conditions
(iii) Tuberculosis
(iv) Histoplasmosis
(v) Toxoplamosis

(vi) Coccidioidomycosis
(vii) Rift valley fever
(viii) Salmonellosis
(ix) Coligranuloma of poultry
(x) Pasteurellosis
(xi) Brucellosis
(xii) Glanders
(xiii) Actinomycosis
(xiv) Botriomycosis

Routes of Infection : Infection to the liver may be conveyed through several routes. The following are the more important:

1. **Portal vein :** Ingested organisms enter the portal vein and so are conveyed to the liver.

2. **Hepatic artery :** Organisms when present in the blood as emboli or in a bacteremic state, reach the liver.

3. **Umbilical vein of the new born animals:** When the umbilical vein is contaminated, organisms grow well in the partially coagulated blood which acts as a good medium and reach the liver. *S. necrophorus* and pyogenic bacteria are the commonest organisms involved producing hepatic necrosis and abscesses respectively.

4 **Bile ducts :** Infection may ascend from the duodenum. Obstruction of bile ducts causing stasis may be another source for infection.

5. **By direct extension** from neighbouring organs as in traumatic reticulitis.

Acute toxic hepatitis ; This is characterised by necrosis, which is usually preceded by degenerative changes like cloudy swelling and fatty degenaration.

Hepatic necrosis is conveniently classified, as per anatomical distribution into focal, centrilobular, midzonal, peripheral, diffuse and paracentral necrosis.

Focal necrosis In this variety, numerous microscopic necrotic areas are seen scattered in the liver and may be found in any part of the lobule.

Causes ; 1. Viral—as in equine viral rhino-pneumonitis in the foetus,

2. Bacterial—in bacteremic or septicemic affections—Johne's disease, Salmonellosis, Tularemia, Listeriosis in new-born.

3. Obstruction of biliary passages.

4. Due to parasitic migration.

Focal necrosis of the liver is not of much consequence since the function of the liver is not affected. Healed lesions show some scarring but this also disappears after some time.

Centrilobular necrosis or Periacinar necrosis.

In this condition, the cells nearest the central vein are affected.

Causes; Anoxia ; i) Acute hemorrhagic anemia; ii) Low atmospheric pressure; iii) Congestive cardiac failure; iv) In Shock—due to reduced blood pressure, reduced oxygen tension and reduced volume flow.

Toxins: Blood borne especially; Carbon tetrachloride. (See page 137)

Macroscopically the liver is enlarged and paler than normal. In severe cases the organ may be redder due to increased quantity of blood The lobular markings

are exaggerated. This is due to the difference in color at the center and periphery. When congestion of the central part is present, the periphery is paler due to degenerative changes in the cells. On the other hand, if necrosis of the cells in the center occurs, then the center will be pale while the peripheral cells are darker.

Microscopically, the cells round about the central veins have disappeared, blood taking up their places. Away nearer the periphery the cells may show fatty degeneration and those beyond these cells cloudy swelling. Infiltration of the periportal connective tissue by lymphocytes is seen after some days.

Sequelae : Single affection may heal by regeneration. Repeated attacks, however, will result in fibrosis which will ultimately reduce the size of the organ.

Pseudolobulation with proliferated bile ducts and resulting nodulation is the ultimate result found in frank post-necrotic cirrhosis.

Mid zonal necrosis : This lesion that is found in *yellow fever* of man affects the hepatic lobe, mid-way between the periphery and the central vein. This condition is not seen in animals.

Periportal necrosis : In this condition the cells adjoining the portal tract become necrotic and so the toxins should have been conveyed by the portal vein. This is more commonly seen in phosphorus poisoning. Accompanying inflammation of the portal triads results in cirrhosis, similar to portal cirrhosis.

Massive necrosis or Acute yellow atrophy : In this condition, there is necrosis of considerable number of the cells in a lobule. This may be a severe manifestation of various types described above.

Since whole parenchyma of the lobule is dead no regeneration occurs, the reticulum and fibrous frame work collapse and there is post-necrotic scarring.

The liver is yellow because of fatty degeneration and necrosis and smaller in size due to loss of parenchyma.

Causes : Virus :–In man.

Poisons :–Carbon tetrachloride, chloroform, phosphorus.

Dietetic :–Deficiency of sulphur-containing amino acids, Tocopherols and Selenium.

Sequelae :–Death

Paracentral necrosis is a peculiar type of wedge-shaped necrosis occurring only on one side of the central vein, but not around it and extending up to the periphery. This type is encountered in Rift-valley fever and in uremic conditions.

Saw dust liver : In well-fed young cattle, at postmortem, focal necrosis of the liver is common. The animals do not manifest any symtoms while alive. The foci of necrosis may be few or many, and appear to the naked eye as though saw dust is sprinkled on the liver.

Microscopically, the lesion consists of hepatic cells which have undergone coagulative necrosis and infiltration by lymphocytes and neutrophils. These spots are evidently scars resulting from inflammatory reaction. It is conjectured that the irritant is borne by the portal veins from the gut and it is for this reason that the lesions are found nearer the portal areas.

CIRRHOSIS

Cirrhosis of the liver is chronic hepatitis characterized by fibrosis, degeneration and hyperplasia of hepatic cells. The stimulus for the fibroblastic proliferation is some irritant, chronic and severe enough to produce degeneration and necrosis of the parenchymatous cells.

The irritant may reach the liver through (a) The portal vein (b) hepatic artery and (c) bile ducts.

Based on the route of infection, the cirrhosis is classified as follows :

1. **Portal or nodular cirrhosis ;**

Causes : Usually, the causes of portal cirrhosis are the same as described under acute focal toxic hepatitis. But frequently one may not be able to ascertain the cause. It should be noted that the irritant is mild and acting for a long time. In this context mention must be made of toxic plants and chemicals. Among the toxic plants known to cause cirrhosis are :—

Crotalaria saggitalis in horses; plants of *Senecio* family in horses, cattle and sheep; *Atalaya intermedia* in horses; *Amsinckia intermedia* (tar weed) in horses, swine and cattle; plants containing high selenium content—in horses. (Wheat, loco weed).

The following chemicals are found to produce this condition :—

Pitch in tar paper, repeated exposure to chloroform, carbon tetrachloride and phosphorus.

Long continued intestinal toxemia is another cause of this condition.

Pathogenesis :

When the irritant is conveyed via the portal veins, changes are noticeable first at the periphery of the lobules—area next to the portal tract. Due to the action of the irritant the following changes take place : degeneration of the hepatic parenchyma, stimulation of the connective tissue in the interlobular septa to proliferate, infiltration of lymphocytes and macrophages into the islands of Glisson: Depending upon the severity of the irritant necrosis of the hepatic tissue may also occur. Along with the new connective tissue, new blood vessels are formed. These irregular blood vessels anastamose with the network of the portal vein as well as with the branches of hepatic artery. Thus arterio-venous shunts result and so ischemia of some parts of the liver occurs leading to further hepatic necrosis.

Along with these changes, hyperplasia of the surviving cells takes place, replacing those that are destroyed. But the connective tissue, which is young and cellular in the early stages becomes mature and fibrous, then contracts, interfering with blood circulation. The decreased blood supply interferes with the proliferation of the hepatic cells and so hyperplasia does not progress further.

In the new fibrous tissue, in the portal areas especially, new bile ducts are formed. These are not functional, lacking in outlets and so stasis of bile occurs.

As the fibrous tissue grows into the liver lobule, the hepatic cells become atrophied due to pressure and lack of nutrition The central vein becomes narrowed (due to the pressure of the fibrous tissue) impeding the out-flow of blood, thereby rendering the irritant to stay longer in the liver. Growth of the fibrous tissue into the lobule divides the parenchyma into small islands of hepatic cells—pseudolobulation.

If irritant enters the liver through the hepatic artery, changes of damage are first noticed in the tissues of portal canal and inter- lobular connective tissue. The features here are: lymphocytic infiltration and proliferation of the connective tissue which slowly encroaches into the lobules producing changes described above.

One noteworthy feature is that when once the fibrous tissue is stimulated to proliferate, this proliferating fibrous tissue itself becomes an irritant. So, even if the original irritant is removed or destroyed, cirrhosis progresses with more and more fibrous tissue formation until the condition terminates fatally.

Macroscopically, the liver is hard and firm. The surface is uneven and nodular. In the early stages the organ may be large. But as the condition progresses. due to atrophy of the parenchyma and contraction of the fibrous tissue, the liver may be reduced in size. The color of the organ is tawny or yellowish-gray and it is to this color that the name "Cirrhosis" was first applied. The color is due to the stasis of bile in the liver.

The architecture of the liver is lost (the normal marking of the lobule disappears) and the hyperplasia that is present gives nodularity to the organ (Hobnail liver) The nodules lack a central vein and are usually greenish in color due to the stasis of bile, which cannot be excreted since the newly formed bile ducts lack on outlet. Stasis leads to deposition of the bile pigment.

On section, the liver cuts with difficulty due to the dense fibrous tissue formed. While cutting a peculiar grating sound can be heard.

Microscopically, the characteristic picture is the increase in fibrous tissue—within and around the lobules. In the portal area small new bile ducts and inflammatory cells (lymphocytes and macrophages) are present. Pseudolobulation is evident. Central veins in some lobules are either absent or are placed eccentrically.

The parenchymatous cells show various stages of degeneration—cloudy swelling, fatty degeneration and even frank necrosis.

Regeneration of surviving cells is evident in some places, giving rise to the nodules noticed macroscopically. These regenerating young cells are plump, robust and stain more intensely.

Multinodular or Atrophic or Gindrinker's or Laennec's cirrhosis.

This is portal cirrhosis of man and merits description here briefly, since in the dog, a similar condition is met with, though not due to similar etiology but to toxins absorbed from the intestines.

Though the exact causes of cirrhosis are still obscure, it is thought that deficiency of Vitamin B complex and lipotropic factors; especially in drunkards, produces this condition. Lack of Vitamin B complex and lipotropic factors results in a highly fatty liver, the fat globules literally occupying the cell cytoplasm pushing the nucleus to a side. Along with this infiltration, there is proliferation of the fibrous tissue which is infiltrated by chronic, inflammatory cells. The bulging cells, pressing on the sinusoids produce ischemia resulting in necrosis of the parenchyma. New capillaries form and invade the lobule and connect the central vein with the portal vessels. The penetrating fibrous tissue divides the parenchyma into smaller lobules. Some surviving cells proliferate and form nodules

(Hobnail). Contracting fibrous tissue makes the liver smaller and hence "Atrophic cirrhosis" results.

Biliary cirrhosis (Monolobular or hypertrophic cirrhosis)

In man this type of cirrhosis occurs consequent on obstruction and infection of the biliary tract. The causes are:

a) Cholangitis—the inflammatory exudate and the desquamated cells clog the bile ducts.

b) Pressure on the bile ducts from without—tumor of the head of pancreas.

c) Stone in the common bile duct.

d) Stricture of the duct.

e) Obstruction of biliary passages by flukes (*Chlonorchis sinensis)* and ascarids.

Macroscopically, the liver is enlarged and the surface is either smooth or finely granular. It is greenish in color.

Microscopically, connective tissue encircles individual lobules (hence monolobular). The bile ducts may be dilated and tortuous. There is great infiltration of the connective tissue with chronic inflammatory cells. Newly formed nonfunctional bile ducts are also found. Hepatic cells reveal degenerative changes. Jaundice is a constant symptom. Ascites is not common.

Biliary cirrhosis in animals is rare because cholangitis and cholangiostasis do not occur in them. Liver flukes that inhabit the bile ducts do not cause extensive cirrhosis but only a local fibrosis.

Effects of Cirrhosis

1. Due to disturbance in portal circulation

A. Ascites; due to

i. Increased hydrostatic pressure in portal veins—flow of blood through liver is hindered due to compression and distortion of the portal and hepatic veins as well as sinusoids by the regenerating nodules. The effect is more in portal cirrhosis since the number of such nodules is greater in this condition than in the biliary type.

ii Decreased colloid osmotic pressure—since there is decreased production of plasma proteins, particularly albumin.

iii Hormones are not inactivated by a damaged liver In health, the liver inactivates the mineralocorticoids of the adrenal and the anti-diuretic factor of the posterior pituitary. But if these are not inactivated more of sodium chloride is reabsorbed and with it more of water is also reabsorbed, resulting in conservation of more fluid in the body and so ascites results.

B. Varicosity of esophageal veins—sometimes resulting in rupture and so hematemesis occurs.

C. Splenomegaly.

D. Gastroenteritis—result of C. V C. of abdominal viscera.

E. Caput medusae in man—this is dilatation of the cutaneous veins around the navel and is seen distinctly in white skinned people.

11. Loss of Inactivation of hormones, toxins etc.,

i. Estrogens normally are inactivated in the liver in the male. But in hepatic cirrhosis this does not occur and so gynecomastia and testicular atrophy occur.

ii. Toxins—exogenous or endogenous—are normally detoxified by the liver. If this is not done, the toxins affect the brain, producing degenerative changes resulting in "walking disease" in horses.

iii. Jaundice—due to pressure on the bile capillaries by the compressed cord cells (by fibrous tissue). So there is obstructive jaundice resulting in digestive disturbances.

iv. Bleeding due to deficiency in production of prothrombin.

v. Anemia—since iron and Erythrocyte Maturation Factor cannot be stored.

vi. Vitamin A deficiency since Vitamin A cannot be stored in the liver.

Other forms of Cirrhosis

Pericellular cirrhosis; In this condition the fibrous tissue invades the parenchyma and encircles individual cells This picture may be seen in the far advanced stages of the multi and monolobular cirrhosis. In well developed aflatoxicosis pericellular cirrhosis is often found.

Pigment cirrhosis: This is the fibrotic condition of the liver that is found in hemochromatosis (bronzed diabetes of man). The macroscopical and microscopical appearances are similar to mild portal cirrhosis with nodulation The large amounts of hemosiderin deposited in the hepatic cells seem to irritate the organ causing cirrhosis.

Glissonian cirrhosis: Correctly speaking this is not a true cirrhosis since the liver as a whole is not affected. Inflammation of the Glisson's capsule (the result of regional peritonitis) extends to the adjacent liver parenchyma. Though macroscopically resembling portal cirrhosis, microscopically the fibrosis is seen to extend from the capsule to a short distance beneath it.

Cardiac or central or congestive or stasis cirrhosis In chronic venous congestion resulting from cardiac lesions the cells round about the central veins suffer—degeneration and necrosis due to pressure and hypoxia; As the hepatic cells disappear, a relative increase in the fibrous tissue is evident. Later on there may be diffuse fibrosis and alteration in the architecture in some cases. This may giverise to atrophy and granular appearance of the organ.

Parasitic cirrhosis: In this variety, the irritant enters the liver through the bile ducts. The cause is usually a chronic obstruction of the bile ducts by flukes or other parasites. In swine mature ascarids invading the bile ducts cause biliary obstruction.

In this condition the changes are localised and are usually restricted in animals to fibrosis of parenchyma for a short distance around the biliary passages. Cirrhosis may spread out due to the penetration of bile into the tissues that surround the bile ducts.

Macroscopically, the liver appears larger, hard, firm and greenish in color. The surface is smooth. The bile ducts are hard and stand out due to extensive calcification—'clay-pipe' appearance.

Microscopically, the fibrous tissue is found encircling the bile ducts and the individual lobules (hence perilobular or monolobular cirrhosis). In this type there is formation of a large number of new bile ducts that are nonfunctional. Also there is infiltration of a large number of lymphocytes into the fibrous tissue. The bile ducts may be completely occluded by the flukes, blood stained exudate, and debris.

Because of the obstruction in the bile ducts, jaundice and deposition of bile pigment in the liver are present.

Besids the flukes and round worms that obstruct the bile ducts, other parasites damage the liver and produce cirrhosis during their larval migratory phase. Wherever the larvae lodge, chronic inflammatory changes arise with resulting fibrosis. In milder infections, the lesions are usually diffuse white spots, one to three cms. in diameter. In heavy infections, advanced fibrosis may be encountered. In pigs the scars produced by ascarid larval migration are depressed while those of *Stephanurus dentatus* latvae are elevated.

In infections by schistosoma species, dense white zones of fibrosis develop around intrahepatic portal branches. The lesions produced are similar to portal cirrhosis with nodularity.

Microscopically, the ova or their remnants may be seen in the fibrous tissue.

Kupffer's cells contain brownish pigment granules.

Abscesses of the liver: Due to the entrance of pyogenic bacteria abscesses may be found in the liver. These bacteria enter the liver by way of portal veins and hepatic arteries mostly. Infection may also occur from the umbilical vein in the young animals. Though the primary umbilical site may heal, the liver may still have abscesses. In the adult and older cattle, infection may occur from traumatic reticulitis.

In countries where cattle are fattened for slaughter, abcesses are frequently encountered in the liver. The cause is *S. necrophorus* gaining entry through the portal vein. In these animals, highly concentrated grain appears to produce ruminal disturbances, resulting in ulcers. Through these ulcers *S. necrophorus* enters the portal vein and ultimately reaches the liver, where it produces first coagulative necrosis. Subsequently this lesion becomes liquefied by the R. E. cells and then gets encapsulated. Later these abscesses heal leaving fibrous scars. In time, these scars may disappear.

Neoplasms: Tumors of the liver may be primary (arising from the liver parenchyma and bile ducts) or secondary (metastases of tumors located somewhere else.)

Primary: Hepatomas arising from the hepatic cells or tumors arising from the bile duct epithelium are now considered as not rare in animals. They may be benign or malignant. The benign tumors are more common.

Another primary tumor of the liver that is found in the dogs is the hemangioma causing, sometimes, fatal hemorrhage. Primary fibroma may also be found in the liver.

Secondary: Metastases of lymphocytoma and pancreatic carcinoma are mostly seen but metastases of any malignant tumor may be found in the liver. In the cow metastases of uterine carcinoma are common. The neoplastic cells of

lymphocytoma may form nodules, or may infiltrate diffusely, replacing the parenchyma gradually.

Mammary gland carcinoma in the dog metastasizes in the liver from the secondary tumors in the lung

CHOLANGITIS: In liver fluke infection, cholangitis is met with, caused by the irritation of the spines on the cuticle of the parasites as well as the toxins liberated by them. The lumen of the bile ducts is dilated and its wall is thickened due to fibrous tissue proliferation around it. These ducts stand out as thick cords. In some cases, due to calcium deposition, these may feel hard also.

Microscopically, the mucosa is thickened and forms papillary projections into the lumen. Infiltration of the walls by macrophages, lymphocytes and eosinophils is common. The lumen contains parasites, cell debris and some mucus. The fibrous tissue that proliferates around the walls of the bile ducts may extend to a short distance into the parenchyma of the liver.

Occlusion of the bile ducts may give rise to obstructive jaundice.

CHOLECYSTITIS: This is rare in animals. Infection is usually ascending. from the duodenum. Stasis of bile by the presence of foreign bodies, parasites, concretions or by pressure on the biliary duct by pancreas are other causes since the retained bile is itself an irritant. *E. Coli* and *Salmonella* are frequently found.

Usually the catarrhal variety is noticed with congested mucosa and increased secretion of mucus by the glands.

CHOLELITHIASIS: Gall stones or choleliths are not as common in animals as in man. These are found mostly in cattle. The gall stones may be found in the gall bladder or bile ducts but unlike in man, bile ducts are more often affected because of frequency of parasitic involvement. They may arise in the bile passages of the liver also.

Gall stones are composed of a mixture of cholesterol, bilirubin, bile salts, calcium and an organic matrix. These may be dark brown or yellowish-green in color. There may be numerous small stones or a few large ones in the gall bladder. The larger ones may be faceted due to rubbing against one another. They are light and friable.

Etiology : Almost always gall stones occur as a result of cholescystitis. The dead cells or bacteria or mucus may form the nuclei around which are deposited cholesterol, bile pigments and bile salts. Sand particles and food materials that may reach the gall bladder through the bile duct from the duodenum during violent peristalsis may also form nuclei of the stones. Cholesterol is normally held in solution by loose combination with bile salts. This combination may be easily broken up. In cholecystitis, the bile salts are rapidly absorbed, leaving the cholesterol which is precipitated.

Sequelae: Most of the gall stones are "silent". That is, they cause no symtoms, being observed at autopsy only. But some may cause colic, nausea and dyspepsia.

If the bile passages are obstructed, obstructive jaundice may occur. If the obstruction of the bile duct is complete, rupture of gall bladder may occur sometimes.

THE PANCREAS

Exocrine disorders : In animals, diseases of the pancreas are not common.

Acute pancreatic necrosis (acute hemorrhagic pancreatitis, necrotising pancreatitis; acute hemorrhagic necrosis).

This condition may sometimes be met with in dogs, cats, swine and horses. Ruminants are believed to be not affected. The essential lesion is necrosis of pancreas by its own enzymes. How this is brought about is obscure. The proteolytic enzymes are the most important. How and where trypsinogen, secreted in the pancreas, is activated to trypsin, to produce this acute condition is not yet clear. Probably occlusion of the duct (by parasites) or injuries or circulatory disturbances or regurgitation of bile or bacterial infection (via blood or from the intestines by ascending infection) may be the causes.

The ezymes escaping out of the pancreatic tissue digest the surrounding peripancreatic fat first and the pancreatic parenchyma subsequently. The fats are hydrolysed with the liberation of fatty acids, which form calcium soaps in the tissues round about the pancreas. Entering lymph channels the lipase may produce fat-necrosis in different and distant organs, even as far away as anterior mediastinal region.

Macroscopically, in fatal cases, there is a small quantity of fluid in the abdominal cavity. Hemorrhages may be present in the omentum. In the mesentery and around the pancreas, whitish areas or nodules of fat necrosis with an inflammatory zone surrounding them are found.

The pancreas is swollen, and soft, yellowish or slightly hemorrhagic. The lesions may be widespread or localised. If limited to a little area, encapsulation may occur. On section, yellowish-grey, soft (pus like) areas of necrosis may be visible.

Microscopically, one finds necrosis of the parenchymatous cells and fat, edematous swelling, infiltration by a few leucocytes, hemorrhages and thrombosis of vessels. Crystals of fatty acids and bluish calcium soaps are found in the necrotic area. Foreign-body giant cells are seen at the periphery.

Results : Death in acute cases after manifesting severe abdominal pain and cardiovascular collapse in shock. Chronic inflammation may result if the episodes are repeated and chronic fibrosing pancreatitis results with atrophy of the organ which is nodular (in cats and dogs). In the horse and in some dogs, on the other hand, the organ is enlarged due to great increase in the scar tissue. Pancreatic fat will reveal a granulomatous reaction.

Steatorrhoea occurs due to loss of the pancreatic juice. In this condition the feces is fatty and foul smelling.

Diabetes mellitus with glycosuria may be seen in dogs

Neoplasms : Tumors of the pancreas are not common. Even in the few that are described, the exocrine tumors (tumors of the acini) are more common. The acinar neoplasm is an adenocarcinoma usually, while that of islets of Langerhans is an adenoma.

ENDOCRINE DISORDERS

Diabetes mellitus : It is pertinent to review here the carbohydrate metabolism since diabetes is essentially its derangement.

Carbohydrates are absorbed as glucose, which is converted into glycogen and stored in the liver. When needed by the tissues, muscles in particular, glycogen is converted into glucose—6– phosphate and then oxidised releasing energy. The by-products—CO_2 and water—are eliminated. These processes are regulated and controlled by various hormones. The most important of these is insulin.

Insulin : This is a hormone produced by the Beta cells of the islets of Langerhans. It is a protein, having a molecular weight of 6000. It has 51 amino acids arranged in two chains and these contain 17 different amino acids.

Insulin has the following functions; helping in the storage of glycogen in the liver, facilitating the entry of hexoses across the cell membrane into the cell (muscles especially); stimulation of hexokinase for formation of hexose—6—phosphate and inhibition of activity of hepatic glucose—6—phosphatase and thus preventing overproduction of glucose.

Conditions may arise when

1. Insulin may not be adequately secreted due to necrosis of pancreas. The causes for necrosis have already been described.
2. Insulin may not be liberated into the circulation though synthesized by the Beta cells. Cause is unknown.
3. Diminished production of insulin due to "work-exhaustion". This occurs when insulin-antagonists act for a long time. Under this category must be mentioned.
   a) Insulinase, a proteolytic enzyme which destroys insulin.
   b) Glucagon and epinephrine are anti-insulin by virtue of their capacity to stimulate hepatic phosphorylase and produce glycogenolysis and hyperglycemia.
   c) Growth hormone (S. T. H.) This antagonises
      i) the effect of insulin on hexokinase ;
      ii) the ability of insulin to transport glucose across the cell membrane.
      iii) by stimulating insulinase and
      iv) by probably stimulating the release of glucagon.
   d) Thyroxine—This increases the metabolic rate and gluconeogenesis.
   e) Adrenal cortical hormones—antagonise by gluconeogenesis and supporting the action of growth hormone.

The above antagonists first stimulate the islets of the pancreas, which become hyperplastic, releasing excess of insulin to arrest the hyperglycemia produced by them. In time, the cells become exhausted and atrophied.

The modern concepts of diabetes in man are ;

1. Diabetes may be present from birth as an inherent defective carbohydrate metabolism and this will be manifested later as diabetes due to various causes, viz, stress due to pregnancy, ACTH therapy, Cushing's syndrome, overeating, streptococcal infections and acromegaly. This error is due to an inherited recessive Mendelian factor.
2. Insulin in diabetes may be in the body as inactive complexes.

3. There may be autoimmunity so that patient's antibodies against insulin may inactivate the insulin in the body.
4. In diabetes there is inability to store sugar as glycogen and so it accumulates in the blood leading to hyperglycemia.

The following changes in the metabolism of carbohydrates take place in insulin deficiency.—Though carbohydrate is transported in the form of glucose, it should be first converted into glycogen in the muscle cells before it can be metabolised to CO_2 and water (by way of TCA cycle). For this, therefore, glucose has to be transported across the cell membrane, to enter into the cells.

In the absence of insulin, normal quantities of glucose molecules are unable to move across the cell membrane and so it is not utilised and hence blood glucose level rises—**hyperglycemia.** When this is above the renal threshold (in dog normal is 160 to 180mg. per 100 ml) renal tublues are unable to completely reabsorb the glucose of the glomerular filtrate and so *glycosuria* also results. Now because of glucose in the urine its osmotic pressure rises and this prevents the reabsorption of the water by the tubules and so **polyuria** also results giving rise to increased thirst, *polydypsia* and *dehydration.*

Glycogen stores of the liver are depleted due to glycolysis. So sufficient amounts of pyruvic acid and oxaloacetic acids are not formed to combine with active acetate formed from the fats. So this active acetate accumulates, condenses and forms ketone bodies, which in excess produce **ketonemia** and **ketonuria.** Being acidic, the ketone bodies neutralise the alkali reserve resulting in **acidosis** which terminates in **air hunger** and **coma.**

Since tissues are unable to utilise glucose (except nerve cells and red blood cells which do not require insulin for glucose utilisation), catabolism of proteins and fats takes place as source of energy. Since fat of fat depots has to move into liver for phosphorylation (without which it cannot be utilised in the tissues) fatty infiltration of liver occurs. Ketone bodies are therefore formed in excess (due to catabolism of excess of fats) and so ketonemia occurs. (Normally small quantities of ketone bodies are produced but these are metabolised in the body.) So ketonemia gives rise to ketonuria. The breath and urine have the characteristic sweet odor. These keto acids interact with sodium and potassium salts and so these bases are lost in the urine and acidosis develops. Acidosis, dehydration and ketonemia give rise to coma.

Protein is catabolised to amino acids from which glucose (gluconeogenesis) and fatty acids are formed. Glucose cannot be utilised and so is lost in the urine. Hence body weight decreases. Excess of amino acids are deaminised in the liver and so there is elevation of blood and urine non-protein nitrogen. With the depletion of carbohydrates, fats and proteins, body loses weight inspite of consuming considerable quantities of food.

Diabetes mellitus may be found in dogs and cats. In dogs it is a disease of older animals, especially in females, due to chronic pancreatitis. For some unknown reason, such dogs develop cataract in the eye.

In lambs diabetes is seen in those that are overfed on carbohydrates. Glycosuria is met with in enterotoxaemia in sheep.

Macroscopically, the animal is emaciated and dehydrated. The liver is highly fatty. The pancreas may either be normal or show pancreatitis and necrosis with fibrosis. Lipemia is evident with the serum appearing white.

Microscopically, lesions are not very constant and conspicuous, Necrosis and hyalinisation of Beta cells have been noticed. Vacuolation of the Beta cells and the epithelium of ducts is present and is due to glycogen infiltration. Similar glycogen infiltration of the epithelial cells of the Henle's loops, and the distal convoluted tubules of the kidney is noticed. The liver cells are loaded with fat (foam cells).

The retinal and vascular lesions of man are not met with in animals.

Hyperinsulinism: This can occur in dogs with (1) excess of insulin injections or increased production of insulin by a tumor of Beta cells. In this condition, glucose is removed from the blood by (a) glucose oxidation by insulin-sensitive tissues, (b) deposition of glycogen in the liver and (c) by lipogenesis, resulting in *hypoglycemia*. The nervous system which is dependent primarily on glucose for energy suffers and its dysfunction is manifested by incoordination, dizziness, muscular weakness, tremors, loss of consciousness and convulsions.

2. Moderate hypoglycemia (50 to 60 mg. percent) activates the sympathetic nervous system, and so epinephrine is released. This brings about glycogenolysis in liver. Similarly glucagon may be released which also causes glycogenolysis in the liver. Hypoglycemia also stimulates the release of ACTH which in its turn causes the production of glucocorticoids which raise blood glucose level by gluconeogenesis and suppressing the peripheral utilisation of glucose.

Glucagon: This is a polypeptide hormone secreted by the Alpha cells of the islets and contains 29 amino acids in a single chain. Its function is quite opposite that of insulin, namely to produce glycogenolysis of liver glycogen. The release of glucagon is brought about by hypoglycemia.

Glucagon action is to increase the activity of liver dephosphorylase kinase which activates phosphorylase and this causes glycogenolysis leading to elevation of blood sugar level. This activity similar to that of epinephrine. But glucagon does not cause glycogenolysis of muscle glycogen since it has no effect on muscle phosphorylase, while epinephrine acts both on liver and muscle phosphorylase.

THE PERITONEUM

Ascites or Hydroperitoneum is edema of the peritoneum and is common in dogs and cats but may also be encountered in sheep and cattle.

Causes: 1. Portal obstruction—due to hepatic lesions—cirrhosis, hydatidiasis, fascioliasis, neoplasia (secondary) and pressure upon the vein by neoplasms, abscesses and enlarged lymph nodes.

2. General chronic venous congestion—cardiac valvular disease or pulmonary lesions.

3. Urinary obstruction in male cattle and sheep with or without rupture of bladder.

4. Hypoproteinemia — gastro-intestinal trichostrongylosis and Johne's Disease in which there is protein loss.

5. Cachectic diseases :- anemia and starvation—in which general dropsy develops.

6. Increased capillary permeability—due to histamine release in shock or due to toxins as in Edema Disease of pigs.

7. Lymphatic obstruction—by neoplasms.

8. Carcinomatosis—primary (malignant mesotheliomas) or secondary (extensive implantation carcinomatosis).

Hemorrhages into peritoneum are common in all animals and may be due to trauma of organs or sweet clover disease. Small focal hemorrhages are common in acute toxemias (enterotoxemia) and infectious diseases—anthrax, hemorragic septicemia and infectious canine hepatitis. These hemorrhages are found on the serosa of the diaphragm, stomach and intestines.

Hemorrhages in the peritoneum are also seen in the course of certain parasitic diseases such as distomiasis, *Strongylus edentatus* infection etc.

PERITONITIS is a very common condition in most of the domestic animals and may be localised or generalised.

The pathogens may be bacteria :- *E. coli*, *Streptococci*, *Staphylococci*; *Corynebacteria*, *Clostridia; Pasteurella* group, Anthrax in pigs.

Viruses—of bovine encephalitis

Helminths

Chemicals—introduced for medication

Endogenous—Bile and pancreatic juice.

Routes of entry : 1. Externally, through surgical wounds or from truama.

2. By blood stream as in bovine viral encephalitis.
3. By rupture of an abdominal organ.
4. Extension through the walls of stomach, intestine or uterus when their mucosa is inflammed.
5. Through osteum abdominale of an infected oviduct.
6. From an infected umbilicus.
7. By way of lymphatics from scrotal infection and infection of abdominal wall.
8. Direct extension from an infected kidney [illegible]

The irritant first produces a serous inflammation which later becomes fibrinous or fibrinopurulent. The fibrin is helpful in localising the inflammation by forming adhesions. Being a very large absorptive surface of the body, toxins are speedily absorbed from the peritoneum damaging other parenchymatous organs.

In the condition known as Glasser's Disease in swine, a diffuse serofibrinous peritonitis is seen.

In visceral gout of birds, *uric acid peritonitis* occurs characterised by the deposition of urates on the serous membrane which consequently shows inflammatory changes.

Tuberculosis of the peritoneum is very frequent in cattle, less frequent in dogs and rarely met with in other animals.

One of the protective mechanisms of nature is the mobilisation and movement of the omentum which covers and sticks to the area of inflammation thereby

restricting its spread. But this has its own drawbacks, since adhesions may form between it and the inflammed parts. Fibrin that forms, if not removed within 6 to 10 days, is organised, thereby inhibiting the movements of the intestines and impeding the digestive process.

Neoplasms : the primary tumors, mesotheliomas (malignant) arise from the serosa and are common in the young and newborn animals.

The secondary tumors are metastases from the liver or uterus.

Transcoelomic implantation of ovarian tumors found in women is not common in animals.

CHAPTER 18

THE URINARY SYSTEM

THE KIDNEYS

Functions of the kidney :

1. Excretion of metabolic end products, especially end products of nitrogen metabolism—urea, creatine, creatinine, ammonium salts etc.—so as to maintain a standard chemical compsition of the blood.

2. Regulation and maintenance of acid-base balance of the extra-cellular fluids.

3. Selective reabsorption and thereby conservation of substances useful to the body : sodium chloride, glucose etc.

4. Maintenance of standard extra-cellular body fluid volume by excretion of water or its reabsorption, whenever indicated.

The above functions are performed by changes in the rate of excretion of the constituents of plasma.

The following extra-renal factors interfere with the functions of the kidney.

1. **Hemoconcentration :** In this condition the viscosity and osmotic pressure of blood are increased, resulting in decreased blood flow in the glomeruli. So there is decreased filtration of fluid resulting in reduced urine.

2. **Low blood pressure :** Causes :-Shock and cardiac decompensation.

Due to decrease in the effective filtration pressure there is complete stoppage of urine formation (Anuria)

3. **Obstruction to the out-flow of urine:** When urinary passages are obstructed by calculi or tumors, back pressure develops in the urine, which opposes the filtration pressure in the glomeruli and so urine is not filtered. Obstruction of urinary passage may also occur in cystitis.

The following intra-renal factors may affect functions ;

A. **Injury to the glomerular filter :**— This may cause (i) increased permeability of the glomerular capillary endothelium, thereby facilitating the passage

of larger protein molecules into the capsular filtrate, and so albuminuria results or (ii) reduction in the filtering surface as in (a) acute inflammation and (b) fibrosis. In condition (ii) the capacity of the glomerular filter is reduced, resulting in reduced urine formation—anuria or oliguria.

B. Injury to the tubules : This results in alteration of the tubular functions: (a) selective reabsorption does not take place and so the essential substances needed by the body like sugar etc. are lost; (b) water from the glomerular filtrate is not reabsorbed, resulting in polyuria and dehydration; (c) tubules may be blocked, thereby obstructing the formation of urine—anuria, uremia; (d) the filtrate formed in the Bowman's capsule may be completely reabsorbed by the lymphatics and veins resulting in anuria, and uremia; (e) substances that are normally eliminated selectively by the tubules are retained, eg. creatinine.

C. Alteration in the circulation of kidney : This may cause reduction in tubular function due to decreased blood and oxygen supply to the tubules. This is brought about by (a) narrowing of the arterioles when blood supply to the organ is reduced and (b) fibrosis of the renal capsule in which condition the renal parenchyma is compressed so much that the total capillary bed is decreased and so the blood supply is reduced.

Mention must be made of the peculiarity of the blood supply in the kidney. The afferent vessels of the glomeruli arise from the arteriae rectae. The efferent arteriole (having smaller diameter than the afferent) on emerging from the tuft, breaks up into capillaries, which surround the tubules and are the nutritive vessels for these structures. Hence, it is natural to find atrophy and disappearance of the tubules if glomeruli are destroyed. And it is to be known that when once destroyed regeneration of glomeruli does not take place. Once lost they are lost for ever (but tubules can re enerate very well).

ERRORS IN RENAL FUNCTION

PROTEINURIA : This is the presence of proteins(albumin mostly) in urine. Ultimately this condition will lead to hypoproteinemia and so to generalised edema (renal edema) finally.

Causes are;- (a) increased permability of the glomerular capillaries.
(b) tubular injury. (c) inflamfnatory reaction.

Albuminuria is met with in the following conditions: congestive heart failure, glomerulonephritis, renal infarction, nephrosis and amyloidosis.

GLYCOSURIA : This is the presence of glucose in urine and may be found in diabetes mellitus, enterotoxemia in sheep due to *Clostridium welchii* type D, following intravenous injection of large quantities of dextrose solution and in njection of adrenocotricotropic hormones.

KETONURIA is the presence of ketone bodies in the urine and is met with in diabetes mellitus, acetonemia of cattle, pregnancy toxemia in ewes and in starvation.

ANURIA means complete urinary failure and **Oliguria** is a condition of reduced excretion of urine. These are brought about in the following manner.

(a) Glomerulonephritis : In this condition due to (i) swelling of the capillary endothelium and (ii) infiltration of inflammatory cells, the capillaries of the

gomeruli are compressed and so blood flow through them is blocked. So urine is not filtered.

(b) **Cloudy swelling and fatty degeneration of the tubular epithelium:** In these conditions, the pressure within the kidney is raised so much by the swollen cells that it obliterates the blood vessels. The tough, inelastic capsule of the kidney does not permit any expansion of the kidney and so the pressure in the organ is passed on to the vessels, compressing them.

(c) **Stagnation of the secreted urine:** If the urine formed is not evacuated from the kidney due to obstruction, the back pressure thus exerted will oppose the filtration pressure thereby preventing the formation of urine.

(d) **Extensive destruction of tubular epithelium:** In this condition there is diffusion of the urine filtered by the glomerulus into the lymphatics and veins.

(e) **Extreme dehydration ;** Sufficient fluid is not present to be excreted.

(f) **Low general blood pressure :** This has already been dealt with above.

POLYURIA : This is increased amount of urine passed.

Causes :- (a) Diabetes mellitus.
(b) Diabetes insipidus (posterior pituitary involvement).
(c) Moderate injury to tubular epithelium and so water is not reabsorbed.
(d) Chronic interstitial nephritis.
(e) In hypercalcemia and hypomagnesemia.

PYURIA : signifies pus in the urine and is found in suppurative inflammation of the kidney or some other part of the urinary system.

HEMATURIA is blood in the urine which is therefore colored red. On centrifugation or standing of urine, the erythrocytes settle down leaving a clear supernatant fluid. This condition is due to hemorrhage from any part of the urinary apparatus—glomeruli to urethra.

Causes:- (1) Diseases of the urinary organs; acute nephritis; pyelonephritis cystitis, chronic bovine hematuria; urethritis; renal infarction
(2) Traumatism
(3) Chemical irritants: cantharides; terpentine; carbolic acid;
(4) Calculi
(5) Acute septicemic conditions :- H.S. Anthrax
(6) Neoplasms—Carcinoma of the bladder or kidney.
(7) Parasites : *Dioctophyma renale.*

HEMOGLOBINURIA : This is the presence of free hemoglobin in the urine which is brown or coffee colored. Since there is hemolysis, red cells cannot be sedimented either by standing or centrifugation. This condition arises due to hemoglobinemia, hemoglobin escaping through the glomerular filter. Hemoglobinuria is found in

(a) Protozoan infections—Babesiosis in cattle, horses, sheep and dogs.
(b) In certain infective diseases : Streptococcal septicemia; infection by *Clostridium,* leptospirosis
(c) Chemical poisoning—potassium chlorate poisoning, chronic copper poisoning in sheep

(d) conditions of unknown etiology-post-parturient hemoglobinuria in cattle.

UREMIA : Uremia is a toxemic syndrome resulting from renal insufficiency. It is associated with urea retention but is not caused by that condition. It is due partly to the retention and toxic action by non-protein nitrogenous substances including urea, creatine, uric acid, ammonia etc., and partly to the development of an acidosis.

The term 'azotemia' means an increase of non-protein nitrogenous material in the blood. This may be due to extra-renal factors such as dehydration, rapid break down of proteins or increased metabolism. Azotemia due to renal cause is uremia and can be differentiated from azotemia due to the extra-renal causes.

Uremia is a fairly common condition met with in significant derangement of renal function. It is estimated that five percent of all dogs that are examined at autopsy have some degree of uremia. This condition is more common in males than in females in all species of animals.

Urea is formed in the liver by the breaking down of amino acids. This is not toxic as is evidenced by injection of urea intravenously without any untoward effects However, the blood urea level is a good index of the toxemia that develops in the uremic condition. The uremia that develops may not be of renal origin at all. It may be:

(i) **Post-renal** when the urinary tract is obstructed by calculi. In post-inflammatory strictures, carcinoma of bladder, prostatic enlargement and congenital defects there is retention of urine, which with back pressure in the kidney opposes filtration pressure and so anuria results.

(ii) **Pre-renal** found in :

(a) Lowered blood pressure due to shock, trauma and intestinal hemorrhage. There is decreased glomerular filtration pressure. Waste products of protein metabolism which are thus retained, aggravate the condition.

(b) Diarrhoea, vomition and intestinal obstruction and excessive sweating. In these conditions there is salt deficiency, dehydration and electrolyte imbalance.

(c) Fever, large infarcts, gangrene, diabetes, high protein intake. In these conditions there is increased protein destruction.

The renal lesions resulting in uremia may be :

(a) Glomerulonephritis; b) Chronic interstitial nephritis (small granular contracted kidney, c) Toxic tubular necrosis and d) Extensive amyloidosis. These lesions cause the following disturbances in the Physiology of the kidney: (a) decrease in glomerular filtration resulting in retention of urea, phosphates and sulphates, (b) decrease in tubular reabsorption resulting in the loss of water and electrolytes. (c) decrease in tubular secretion, resulting in the upsetting of the balance in potassium, H-ion, ammonia and creatinine, leading to hyperkalemia (producing cardiac inhibition and death) and acidosis. (d) Probable decrease in the detoxifying mechanism of the kidney so that the accumulated toxins act on the hemopoietic system and cause anemia.

Normally, the blood urea nitrogen (BUN) is less than 14 mgm. per 100 ml. of blood. But in uremic-states, this is well above this figure. Besides the non-protein nitrogenous substances (uric acid, ammonia, creatinine, urea, amino-acids)

other substances, such as sulphates and phosphates of potassium and chlorides are retained. These deplete the alkali reserve and so acidosis develops. This acidosis may be due to (i) reduction of glomerular filtration of the acid substances or (ii) loss of base especially sodium. In the damaged tubular epithelium "ion exchange" does not take place With the rise in phosphates, the calcium content of the blood is diminished.

Lesions met with in uremia are :

1 Toxic degenerative changes in the parenchymatous organs: ulceration of the mouth and stomach accompanied by hemorrhagic gastro-enteritis. The toxins are presumed to be excreted through the alimentary tract, causing the inflammatory changes. (Excretion gastritis—See page 347).

2. Injury to the neurones : by the toxic materials retained in the blood.

3 Deposition of calcium urates and urea on serous membranes : These minerals produce trauma of the serous membaranes and joints and so inflammation in these places occurs.

4. Dyshemopoietic anemia due to

(a) Decreased intake of iron and Vitamin B_{12} as animals suffer from anorexia or vomition. (b) Toxic suppression of hemopoiesis in the bone marrow.

5. Hyperplasia of parathyroid : In chronic nephritis, especially in the dog with extensive tubular damage, there is phosphate retention. This produces *osteodystrophy*. The following processes are involved. The damaged tubules reabsorb the phosphate indiscriminately thereby elevating the plasma phosphorus level Excess of the phosphorus, when being excreted through the intestines combines with calcium, forming insoluble calcium phosphate. So calcium is not available for absorption and hence hypocalcemia results. This hypocalcemia in turn stimulates the parathyroid resulting in hyperplasia and increased parathyroid hormone production. This in turn produces (i) resorption of bone by osteoclasts, thereby releasing calcium and phosphorus and (ii) increased renal tubular excretion and diminished reabsorption of phosphorus

These are attempts by nature to retain the plasma calcium and phosphorus levels within physiological limits. But the damage of the kidney in chronic renal disease is irreparable and so a vicious circle is established, with more and more phosphorus retention and consequent hyperparathyroidism and osteoporosis resulting. This is clinically known as osteodystrophy. Nature tries to strengthen the softened bone by the cheaper fibrous tissue, which becomes hyperplastic and this is more evident in the bones formed by intra-membranous ossification, namely those of the head and jaw. Therefore these bones become very soft and pliable and can be bent. Hence they are known as "Rubber nose" and "Rubber jaw" respectively. Histologically, some degree of osteodystrophy can be found in all the bones.

6 Polyuria : due to excretion of large amounts of calcium and phosphorus osmotic polyuria and attendant polydipsia result.

7. Metastatic calcification : Hyperparathyroidism with consequent hypercalcemia will result in metastatic calcification of soft tissues, especially gastric mucosa, larynx, trachea, lungs, visceral pleura etc..

8. Degeneration of liver with icterus.

9. **Cardiovascular lesions :** Found mostly in dogs.

(a) Increase in pericardial fluid with small amounts of fibrin

(b) Necrosis of the endocardium of the left auricle and the intima of the aorta and pulmonary artery to their first few centimeters. Later, the necrotic areas become fibrosed and calcified in those animals that survive

(c) Cardiac hypertrophy (especially of left side), medial hypertrophy of arterioles and capillaries. These changes are attributable to hypertension that develops due to (i) resistance to flow of blood through the kidney in chronic interstitial nephr tis(leptospiral infection) consequent on fibrosis or (ii) production of excessive amounts of renin by the damaged kidney, which releases hypertensiu (a vasoconstrictor) acting on plasma hypertensinogen.

10. **Terminal pulmonary edema**

POSTMORTEM CHANGES IN THE KIDNEYS

As in the liver, in the kidney also, postmortem autolysis occurs rapidly after death So if autopsy in not conducted immediately after death, it will be difficult to differentiate between antemortem degenerative processes and postmortem autolysis. The changes are observed in the cells of tubular epithelium in which the nuclei and brush borders disappear and the Altmann's granules become clumped together. In birds there is hyperchromasia of the nuclear wall and karyorrhexis.

More rapid autolysis of the kidney parenchyma may occur if prior to death there are already degenerative changes in the renal tubules. This is especially true in "Enterotoxemia" of sheep, in which the kidney is very soft—"pulpy-kidney". To the naked eyes, the cortex appears opaque and grey in this postmortem change. Gas bubbles may occur from bacterial putrefaction.

Hypostatic congestion, imbibition of hemoglobin and pseudomelanosis are other changes noticed postmortem.

ANOMALIES OF DEVELOPMENT :

Agenesis—absence of one or both, of the kidneys may be met with,

Hypoplasia, in which the kidney is smaller, is more often seen. In such animals, the other kidney shows compensatory hypertrophy.

Persistent lobulation (normal in fetal life) may be seen in dogs, sheep and swine.

"Horse-Shoe kidney" is seen in all species of animals. This results from fusion of the kidneys at the posterior poles.

Duplication of one kidney may be seen in pigs and so in such animals three kidneys may be noticed.

Cysts ; Cysts in the kidneys are the most common congenital defects. One or more cysts may be found. Kidney with numerous cysts is known as *congenital polycystic kidney*. These arise due to lack of continuity between the nephron and the collecting duct and so urine formed in the nephron is not evacuated but collects to form a cyst.

Cysts may also be formed (acquired) after birth especially in chronic interstitial nephritis, in which the fibrous tissue compresses the tubules with resulting dilatation (and accumulation of urine) of the proximal part.

CIRCULATORY DISTURBANCES

HYPEREMIA : *Active hyperemia* is noticed in acute nephritis and in generalised acute septicemias and bacterial intoxications. *Passive hyperemia* is a feature found in generalised passive congestion. In these conditions the kidney may be slightly enlarged but due to unyielding nature of the tough capsule and the dense renal parenchyma, spectacular changes are not found. Usually, congestion is more evident in the medulla.

INFARCTS of the kidney are common due to occlusion of the branches of renal artery and are anemic in type. Renal infarcts are very common in cattle, especially cows, the commonest causes being thrombosis of the uterine veins after parturition and ulcerative thrombotic endocarditis caused by *C pyogenes* and *Streptococci*. In pigs infarcts are seen in erysipelatous endocarditis. They are wedge shaped, with base of the wedge towards the cortex and apex towards the pelvis. If the condition is not septic, healing by scar tissue will ensue, with pitting on the surface.

EDEMA of the kidney is not common because there is no place in the kidney for the fluid to accumulate. The capsule is inelastic and the parenchyma is firm. In acute interstitial nephritis some inflammatory edema may be observed.

DEGENERATIVE PROCESSES IN THE KIDNEY : Degenerative renal lesions are known as Nephrosis. This term is applied to necrotic lesions of the kidney also. Though formerly distinction was made betwen nephrosis (degenertive changes) and nephritis (inflammatory), as per our concept of alterative inflammation, the distinction is no longer tenable.

The degenerative processes affecting the tubules mainly, include cloudy swelling, fatty degeneration and even necrosis. It is the highly functional and specialised epithelium of the proximal convoluted tubules that is greatly susceptible to the irritants. Next in order that are affected are the Henle's loops and the distal convoluted tubules

HYDRONEPHROSIS : In this condition there is dilatation of the renal pelvis due to obstruction to free flow of urine. The obstruction produces stasis of urine, which with its back pressure causes atrophy of the renal parenchyma.

The obstruction may be any where in the urinary tract—from the urethra to the renal pelvis. For hydronephrosis to develop, the obstruction must be partial leading to gradual stasis. If it is complete, atrophy of the corresponding kidney results. Hydronephrosis may be congenital or acquired Among the causes for the acquired are :- (a) calculi; (b) hemorrhagic cystitis; (c) enlargement of the prostate in the dog ; (d) tumors of renal pelvis, ureter, bladder and urethra ; (e) compression of the ureters by surrounding inflammatory tissue, neoplasms, gravid uterus, ovarian or uterine masses ; (f) displacement of the bladder in perineal hernia.

Hydronephrosis may be unilateral or bilateral. Extreme degree is observed only in the unilateral affection with partial obstruction proximal to the bladder. Here the whole kidney may be converted into a bag with a paper-thin capsule. In the not-so-severe cases, there is atrophy of the tubules, some of which may be widely dilated. The cortex will be thinner and grayish in color.

Sometimes, the fluid in the kidney may be pus due to supervening pyelonephritis, when the condition is known as **pyonephrosis.**

Results : In bilateral—death due to uremia
In unilateral—in uncomplicated cases, hypertrophy of the other kidney.
Pyonephrosis if infection supervenes as in pyelonephritis.

TOXIC NEPHROSIS : The toxins are conveyed to the kidney by blood The toxins are:-

Chemical : Inorganic : salts of ; mercury (used as calomel; mercuric perchloride; mercurial fungicides ; uranium (uranium nitrate); chromium (potassium dichromate); copper (copper sulphate used to eradicate molluscs, parasites and fungi); bismuth (in B.I P.P. as dressing for wounds); cadmium, arsenic and phosphorus

Organic : Carbon tetrachloride and tetrachlorethylene (anthelminitics), insecticides containing chlorinated hydrocarbons, oxalates and oxalic acid (from plants): sulphonamides, turpentine, iodoform, cantharides, phenol.

Mercury poisoning is common in cattle, horses and swine. Mortality is high. This is a chronic type of cumulative poisoning, a single dose of acute poisoning not being common. The kidneys are enlarged, pale and bulge on cut surface.

Microscopically, in the acute disease there is coagulation necrosis and desquamation of the epithelium of the proximal convoluted tubules. Granular and hyaline casts are formed. There is moderate dilatation of the lumen of the tubules Infiltration of edema fluid and lymphocytes is found in the intertubular tissue. If death does not occur within about a week, regeneration of the epithelial cells occurs. These new cells are flat and dark staining. A notable feature is the deposition of calcium in the basement membrane, on the necrotic epithelium and the debris. By the third week, regeneration is complete (See page 133).

Oxalate nephrosis occurs in dogs and cats by swallowing ethylene glycol (used as an antifreeze) and in sheep that eat plants rich in oxalic acid or oxalate. The kidneys are slightly enlarged and are greyish brown in color. The doubly refractile, rectangular crystals of calcium oxalate are deposited in masses in the tubules, especially in the proximal convoluted tubules The tubular epithelium reveals vacuolar degeneration. There may be focal necrosis but damage is mild. Anuria is explained by the obstruction of the tubules by crystals.

Sulphonamide nephrosis may be met with in calves subjected so sulphonamide therapy but with inadequate sodium bicarbonate and water. In the acidic urine, the crystals are deposited in various parts of the kidney.

The epithelium of the proximal convoluted tubules and the Bowman's capsule undergo hydropic degeneration. Swelling and proliferation of the epithelium of the distal tubules is also evident. Anuria may result due to mechanical blockade of the tubules.

Endogenous toxic nephrosis occurs in ketosis, icterus and degenerative changes found in severe septicemic and toxic diseases. There is at first cloudy swelling and then early necrosis of the tubular epithelium.

Among the above varieties, special mention must be made of *Cholemic nephrosis*, seen in the jaundiced animals. The nephrosis is believed to be due to the action of the substances retained in the bile on the tubules. This condition, therefore, is more pronounced in obstructive jaundice. In these conditions the kidneys are swollen, pale and opaque and are brown in color due to the deposition of blood pigment in the renal tubules. The pigment is found intracellularly.

Hepato-renal syndrome When there is damage to and necrosis of liver parenchyma, severe degenerative changes and necrosis may be found in the renal parenchyma also. Normally liver detoxifies various toxic substances formed somewhere in the body But when it is damaged, this function of the liver is deranged and so the toxic substances reach the kidney and damage it.

Anoxic nephrosis : When adequate supply of oxygen is lacking, degeneration of renal tubules may result. Anoxia may be found in (i) urine retention producing increased intra-renal pressure; (ii) shock—due to severe burns; (iii) intestinal obstructions. The kidney is pale and soft.

Lower nephron nephrosis—the crush syndrome—hemoglobinuric nephrosis;

When large quantities of hemoglobin are excreted through the kidneys, lesions are found in the lower portions of the nephron, viz, the ascending loop of Henle and the distal convoluted tubule. This condition, therefore, occurs whenever there is hemoglobinemia as in (1) excessive hemolysis—by poisons or protozoa or incompatible blood transfusion; (2) severe burns; (3) equine Azoturia and (4) in crushing injuries—automobile accidents and air raids. In acidic urine the pigment is precipitated, which blocks the tubules resulting in anuria. Selective damage seems to be caused in the lower nephron with hemoglobin casts in these places. Tubular epithelium contains fine particles of hemoglobin and may show hyaline droplet formation.

Macroscopically, the kidney is swollen and pale with reddish streaks in the medulla.

NEPHROCALCINOSIS

Three types of disturbances in calcium metabolism are met with. White streaks or spots may be seen macroscopically denoting the places of calcium deposition. Calcium is deposited as phosphates and carbonates,

1. Dystrophic calcification. *(Primary epithelial calcification):* In this condition the epithelial cells of the tubules are affected. As explained earlier (page 142) this occurs whenever there is necrosis of the cells and so may be encountered in nephrosis. Actually calcification is met with in sub-acute mercury poisoning. Due to poisoning by the mercury salts, the tubular epithelial cells are degenerated and necrosed and calcium salts are deposited on them.

2. Calcium casts : When the urine is concentrated and inspissated in the distal convoluted tubules, the ascending loops of Henle and the proximal part of the collecting tubules, the calcium in the urine is precipitated on albumin casts These calcium casts may destory the tubular epithelium, which may also, on the other hand, be calcified. Later there is inflammatory reaction in the interstitial tissue, evidenced by infiltration of histiocytes and fibrosis.

3. **Deposition of calcium in the interstitial tissue:** This condition is seen in dogs between the ages of 6 months and 4 years and is usually found in such affections in which there is reaction in the interstitial tissue. Therefore, this is seen in leptospirosis. The process starts with the deposition of calcium salts in the basement membrane of the tubules and later it may extend to the interstitial tissue The capillary vessels may also reveal calcium masses in their walls. Sometimes this condition may be found in association with deposition of calcium in other organs

INFLAMMATION OF THE KIDNEYS

The following classification of nephritis, among animals, appears to be the simplest :

1. Suppurative :— i. Pyaemic—hematogenous
   ii Pyelonephritis—urinogenic—ascending
2. Non-suppurative ;— i. Interstitial
   ii. Tubular (already dealt with under nephrosis)
   iii. Glomerulonephritis.
3. Specific :— Tuberculosis, etc.

PYAEMIC NEPHRITIS OR EMBOLIC NEPHRITIS is a focal suppurative nephritis arising from infection with pyogenic organisms which are blood borne.

Causes are :— Cattle—coliforms in calves.
Corynebacterium pyogenes and *Streptococci* in adults.
Foals : *Shigella* and *Streptococci*
Swine : *Streptococci* and *Staphylococci*

Infection may occur as a secondary to suppurative processes elsewhere—the umbilical vein, mammary gland, uterus, the pericardium or the lung In the foal, the infection may be acquired *in utero*

The bacteria may reach the kidney in clumps or in the emboli from cardiac vegetation. These are arrested in the glomeruli or in the intertubular capillaries where abscesses are formed

Macroscopically, there may be numerous tiny abscesses, literally studding the kidney. These may be visible on the cortex through the capsule. Abscesses in the cortex are circular while those in the medulla are elongated. All the abscesses are of the same size, being of same age.

Microscopically, abscesses with leucocytic infiltration are found. Bacterial emboli are found in the glomerular loops and in capillaries between the tubules

Sequelae: Mostly terminate with death.

PYELONEPHRITIS : This is a term used to indicate the inflammation of all parts of the kidney—involving the pelvis and parenchyma of the kidney.

Usually, this condition is met with in cows, but may also be found in sheep and swine

Infection in most of the cases, is thought to be an ascending one from the lower regions of the urinary tract. For such an infection to occur stasis of urine is an essential predisposing factor. When stasis occurs, the bacteria, especially the motile ones, ascend to the pelvis.

Causes of stasis : In young animals - anomalies of the urinary system—pervious urachus in calves and kinking of ureters in pigs.

In mature animals :—In pregnant females gravid uterus presses upon the bladder and ureters; in males (dogs especially) enlargement of the prostate and obstruction by urinary calculi. Tumors, abscesses and fibrosis along the urinary excretory tract may prevent evacuation of the urine. Similarly, cystitis and ureteritis may also hinder speedy emptying of urine.

The causative orgnisms :

Cattle : *Corynebacterium renale* mostly and *C pyogenes* sometimes.

Sow : *Coliforms*, *Proteus*, *Streptococci*, *Pseudomonas* and *Salmonella*.

C. renale appears to have selective affinity for the pyramids of the pelvis and is the organism seen in most of the cases in the cows. The condition occurs more frequently in the post-parturient period because at that time there may be infection of the uterus and vagina from which infection spreads to the pelvis of the kidneys by way of the short and broad urethra. Infection may sometimes be hematogenous also.

At first there is pyelitis from where infection spreads to the kidney parenchyma by way of large uriniferous tubules.

Macroscopically, bladder is enlarged and ureters are dilated. The pelvis is widely dilated with pus; beneath the capsule on the cortex are visible irregular spots, grey in color, indicating tiny abscesses. Gray streaks may be found in the medulla in the early stages. The calices are widely dilated and filled with purulent material containing calcium particles. The walls of these are red and ulcerated. The papillae are either absent or if they are present are dirty-grey in appearance with erosions and a zone of hyperemia around. The renal pelvis is filled with purulent material which contains triple phosphates. The ureters are thickened and their mucosa is roughened. Due to increasing accumulation and retention of urine, back pressure may produce pressure atrophy of the parenchyma so that in some instances only a thin rim of cortex may be found.

Microscopically, the lesion is a purulent process with neutrophils and a few lymphocytes, which may be found as streaks among the tubules. Glomerular loops and Bowman's capsules are filled with leucocytes and bacteria. Tubules may contain cell-casts as well as bacteria. The walls of the collecting tubules, as well as the interstitial tissue, may become necrosed and this is demarcated from the healthy tissue by a dense zone of leucocytes and hyperemia. The epithelial lining of the pelvis may be necrosed and there may be leucocytic infiltration underneath.

Urine is bloody and contains pus.

The condition is fatal.

NON-SUPPURATIVE NEPHRITIS

INTERSTITIAL NEPHRITIS : Among animals, this is the most common type of nephritis seen. In dogs, it is an important disease. Bloom believes that at least 55% of all dogs autopsied have some form or other of interstitial nephritis. The condition is found more frequently in older and male dogs. The disease is sometimes observed in horses, swine, sheep and cattle.

In a normal kidney, no tissue is found between the tubules (interstitial space). But in interstitial nephritis there is infiltration of inflammatory cells and exudate as well as proliferation of the fibrous tissue.

Causes : In most of the cases, the cause is obscure. Usually the condition is associated with retention of urine. Lesions of the lower regions of the urinary tract causing hindrance to free passage of urine are conducive to the development of the condition. Though infection is mostly hematogenous, ascending infection is not ruled out.

Possibly tubular damage is a starting point and a factor in many cases, damage occurring while toxins are excreted by the kidney. Interstitial nephritis has been seen in the following conditions:

Dogs Leptospiral infection; infections Canine Hepatitis; metritis; pyometre; bronchopneumonia and other respiratory affections; cystitis; prostatitis, chronic peritonitis and chronic infections.

Cattle and swine : Leptospiral infection. In pigs this condition is a metastatic infection and the organisms responsible are. organism of erysipelas, corynebacteria, *E. coli* and non-hemolytic staphylococci and streptococi, besides leptospira. Though the condition is frequent in these animals, it is benign and is encountered at autopsy in normal looking animals,

Fowls : The condition is usually seen in infectious diseases notably *Pullorum disease*. It is focal in distribution.

Young calves : Pneumonia and enteritis (White spotted kidney).

The disease may be acute, subacute or chronic. Again it may be diffuse or focal.

DIFFUSE INTERSTITIAL NEPHRITIS

Macroscopically, in the acute type, the kidney may be of normal size or slightly enlarged. The capsule strips off easily. The cortex shows a mottling of red and gray. The gray areas are the places of infiltration by inflammatory cells and are present in the cortex and may also occur in the outer medulla. In the normal yellow kidney of cat these changes are usually not easily observed.

The subacute and chronic types of interstitial nephritis merge imperceptably. The kidney is smaller in size, pale-grey in color, is hard and cuts with difficulty. The thickened capsule peels with difficulty and when stripped some portion of cortex is torn. The cortex is shrunken and very narrow. The surface is uneven due to irregular contraction of the fibrous tissue—the "Small granular contracted kidney".

It is not easy to distinguish kidney with chronic glomerulonephritis from that of chronic interstitial nephritis. The following differences may, however, be noticed.

Chronic glomerulonephritis	Chronic interstitial nephritis
1. Surface finely granular	1. Surface coarsely granular
2. Inflammatory process begins in the glomeruli	2. Inflammatory process begins in the interstitial tissue
3. Involvement of interstitial tissue secondary	3. Involvement of glomeruli is secondary
4. Diffuse involvement of glomeruli.	4. Glomeruli not involved or only few affected.

Similarly difficulty in differentiating between the kidney with interstitial nephritis and that with pyelonephritis may be felt. But the lesions in the latter are

more irregular and asymmetrical and lesions may also be found in the pelvis and bladder.

Retention cysts of varying numbers and sizes are present Sometimes they may be so many as to resemble the polycystic kidney.

Microscopically, the acute diffuse interstitial nephritis presents a picture that is essentially a true inflammatory reaction, consisting of exudation infiltration and proliferation, affecting the interstitial tissue. The infiltration, consists mainly of lymphocytes and plasma cells, with fewer number of neutrophils. The infiltration of the leucocytes is found mostly in the cortex and outer medulla and is diffuse and wide spread. Heaviest infiltration is found at the cortico-medullary junction Early in the lesions fibroblastic proliferation is evident, especially in a case of leucocytic infiltration. Glomeruli, however, are found to be normal The epithelium of the tubules shows degenerative changes (especially in Canine leptospirosis), which may be so severe and extensive as to cause death by uremia. These changes are more pronounced in the proximal convoluted tubules.

The acute episodes may pass off with resolution and healing. But the condition may also progress to the chronic phase, with scarring, especially with repeated acute attacks.

The gradual increase in the fibrous tissue in the chronic diffuse interstitial nephritis produces atrophy and disappearance of tubules, by pressure. Some of the tubules may show cystic dilatation in the parts proximal to constriction by the fibrous tissue. Granular and hyaline casts are found in such dilated tubules. As the fibrous tissue increases, the leucocytes diminish in number but do not completely disappear denoting thereby that the inflammatory reaction still exists. The Bowman's capsule may show rings of fibrous tissue around them. Eventually, the fibrous tissue replaces much of the renal parenchyma. Collagen that forms may contract, producing the granularity on the surface. In some places hyaline changes can be seen in the fibrous tissue and some glomeruli may be represented by hyaline nodules only.

In the cortex may be found hyperplastic and hypertrophic changes in those tubules, which are still normal, with tall columnar epithelium, having an "adenomatoid" appearance but which is purely compensatory. Calcium may be deposited, especially in the glomeruli. The walls of blood vessels become thickened and the lumen narrowed.

As in the liver, after a certain stage of fibroblastic proliferation, the fibrous tissue appears to continue to proliferate and is self-perpetuating even though the causative factors may no longer be present. The stimuli for such proliferation appear to be (1) the constituents of urine that may leak out of the damaged tubules; (2) the degenerative changes in the tubules due to encircling fibrous tissue and (3) the alteration in the blood vessels and circulation caused by the fibrosis.

FOCAL NON-SUPPURATIVE INTERSTITIAL NEPHRITIS

The causes for this condition may be those enumerated for the diffuse variety. But the lesions are focal and sparse. Incomplete resolution of diffuse interstitial nephritis may result in focal type, while coalescence of numerous foci in the focal interstitial nephritis may be the cause for diffuse variety.

The best example for focal interstitial nephritis is the "White spotted kidney" seen in calves, which do not seem to suffer much of this condition and show no symptoms, since, when they grow old, complete resolution and obliteration of the lesions ensue. It is only incidentally that the lesions may be observed in calves that die of some other disease. *E. Coli* is incriminated to be the etiological factor in these animals.

Macroscopically, the lesions are small. Pin-point-sized, grey-white, circumscribed areas that are found scattered on the cortex under the capsule and also deep into it, are seen on section. Outer medulla also may show these lesions. Some foci may bulge on the cortex and are clearly noticeable through the capsule.

Microscopically, there is inflammatory reaction of the interstitial tissue only while the glomeruli are free. There is edema of the interstitial tissue with infiltration by moderate number of lymphocytes and plasma cells Fibrous tissue proliferates causing degeneration of the tubular epithelium and later atrophy and disappearance of some of them. As in the diffuse variety, some tubules may be dilated and cystic. Granular and albuminous casts are present in the tubules.

Clinical correlation; In the early stages of interstitial nephritis, may be found marked albuminuria, casts and either oliguria or polyuria.

In the chronic stage is found polyuria with low specific gravity, because the damaged tubules are not able to concentrate the glomerular filtrate.

With progressive loss of renal parenchyma, due to replacement by connective tissue the renal function fails and uremia supervenes with ultimate death. In such animals there may be calcification of laryngeal mucosa, left atrial endocardium, pleura and pulmonary artery (due to metastatic calcification that occurs consequent on secondary hyperparathyroidism that develops on phosphate retention occurring in this condition)

GLOMERULONEPHRITIS

In glomerulonephritis, the glomeruli are chiefly affected. This condition is not as common in animals as it is in man. It may sometimes be found in dogs, cats, swine, horses and mink. Horses used for antisera production suffer from this disorder. Acute glomerulonephritis may sometimes be seen as an enzootic among minks vaccinated against distemper.

The exact cause of glomerulonephritis is not yet known but it is believed to be an allergic (antigen-antibody) reaction to foreign proteins The glomerular capillaries become sensitised to the foreign protein and get damaged due to deposition of antigen-antibody complex. Two mechanisms by which the antigen-antibody reaction is brought about in the kidney have been identified. In one, there is production of antibodies by the animal to the glomerular basement membaranes. In this variety, deposition of antibody, complement and fibrin beneath the endothelium on the basement membrane is demonstrable by microimmuno—fluorescent technique. In the second variety, there is deposition of antigen-antibody complex (which is not of renal origin) in the basement membrane beneath the epithelium. In this condition it is thought that the antigen antibody complexes circulating in the blood react with the cells releasing histamine, which increases

the permeability of the glomerular capillaries, thereby facilitating deposition of such complexes on the basement membrane.

Glomerulonephritis is often seen as a sequel to bacterial or viral diseases elsewhere in the body.

The primary changes are in the glomeruli. The tubular and interstitial lesions are secondary to the glomerular affection since their blood supply depends on the efferent arteriole emanating from the glomerulus. Among animals though acute diffuse glomerulonephritis as occurring in man is not observed, focal glomerulonephritis is met with in various acute septicemic infections such as acute swine erysipelas and infections by *Coliforms, Leptospira, Streptococci, Staphylococci, Pasteurella* and *Salmonella.*

Acute glomerulonephritis

Macroscopically, both the kidneys are enlarged and pale. The capsule which peels easily, is tense. On the cortex, red dots indicating the congested glomeruli, are seen. Sometimes small hemorrhages are also observed on the cortex. On section, the kidney slightly bulges on the edges.

Microscopically, there is hyperemia of the glomerular capillaries, soon followed by proliferation of the endothelial and epithelial cells This proliferation blocks the capillary lumens and so glomerular ischemia results. Infiltration by inflammatory cells aggravates the ischemic condition. Hence the efferent arteriole becomes ischemic, and so nutrient blood supply to the tubules is diminished. So now there is "increased cellularity" of the glomeruli. The capsular space, thus appears completely occupied by the swollen tuft, leucocytes, precipitated protein and a few erythrocytes. The subepithelial basement membrane becomes thickened. Electron microscopical examination reveals swelling of the basement membrane and deposition of electron dense material between the membrane and endothelium, within the membrane and between the membrane and epithelial cells. The changes in the basement membranre are evidently due to the increased permeability of the membrane. Fibrin-thrombi forming in the golmerular capillaries may cause hemorrhages. Collagen-like material may also be deposited between the capillaries. The ischemia that results is the cause of degenerative changes in the tubular epithelium which may contain hyaline droplets. Casts of protein, leucocytes and erythrocytes are seen in the tubules.

Sequelae : The condition may heal and resolve in mild cases. If not, it may progress to subacute and chronic phases.

Subacute glomerulonephritis: *Macroscopically* in the subacute glomerulonephritis we find the "Large white kidney". The kidney is enlarged, pale and smooth with non-adherent capsule Cortex may reveal a few hemorrhages The capsule is tense. The cortex is wider than normal and is yellowish in color and so there is distinct color contrast between it and the medulla.

Microscopically, the proliferation of epithelial and endothelial cells is more pronounced. The proliferation of the epithelial cells of the parietal layer of Bowman's capsule results in a crescent-shaped tissue, several cell-layers thick—"epithelial crescents".

Due to deposition of collagen-like material between the crescents and the tufts, adhesions develop. Hence the tufts are obstructed and subsequently destroyed.

The tubular epithelium undergoes fatty degeneration, which progresses to hyaline droplet degeneration and necrosis. The tubules reveal casts of protein, leucocytes and necrotic epithelial cells. The interstitial tissue is edematous and contains infiltrated inflammatory cells together with some amount of collagenous tissue. The basement membrane becomes diffusely thickened and permeable to proteins.

Chronic glomerulonephritis: The subacute glomerulonephritis may imperceptably merge with the chronic phase. It is not necessary, it must be understood, that all cases of acute glomerulonephritis should culminate into chronic form. Only a small proportion may do so.

Microscopically we find in this stage, a shrunken and contracted kidney with a finely granular surface. The capsule is adherent and when removed some of the cortex is peeled off. On section, the cortex is found to be narrower and markings are obscured. Small retention cysts, due to obstruction of the tubules, are seen

Microscopically almost all the glomeruli are found to be affected and most of them are fibrosed. Some show hyaline changes while quite a few are atrophied and may disappear altogether. Still others may show adhesion between the tufts and the capsular epithelium. The inflammatory changes in the interstitial tissue is more pronounced. Greater number of lymphocytes and more marked fibrosis are evident. In the scarred tissue, many tubules have disappeared. Arteries show thickening due to proliferation of the media and intima and so are narrowed. Some of the tubules which are still connected to functional glomeruli, are dilated

Clinical Correlation

Acute stage :

Urine ; Oliguria—due to ischemia of the glomeruli no blood is available for filtration.

High specific gravity of urine : some of the still normal and functional tubules concentrate the urine. Albumin, erythrocytes, leucocytes, renal cells and casts seen.

Blood ; Raised B U N content of blood.

Chronic stage: Urine : Polyuria with low specific gravity—later urine decreases in volume, albuminuria, casts, red and white cells.

Blood ; Progressive anemia. B U N abnormally high.

Filtration rate and renal plasma flow are reduced.

MAIN TYPES OF NEPHRITIS IN DIFFERENT SPECIES OF ANIMALS

Species.	Type.	Cause	Frequency of incidence.	Source.
Cattle.	Pyelonephritis	*C. renale* *C. pyogenes*	Common	Associated with metritis and retained placenta following parturition

Species.	Type.	Cause	Frequency of incidence.	Source.
Cattle	Pyemic	*C. pyogenes* *Staphylococcus*	Rare	
	Specific	*M. tuberculosis*	Rare	Miliary
Calves	Focal interstitial.	*E. coli.*	Common	From umbilical infection White spotted kidney.
Sheep.	Focal interstitial.	—	Fairly Common	—
	Pyelonephritis.	*Streptococcus* *Staphylococcus*	Rare	Associated with calculi.
Horse.	Glomerulonephritis	not known (antigen-antibody reaction?)	Rare	Associated with recent streptococcal infections of the upper repiratory tract.
Foal.	Pyemic.	*Shigella equirulis*	Common	Associated with joint ill
Pig.	Focal interstitial	*E rhusiopathiae* *E coli, Corynebacteria, nonh molytic strepto & staphylo,*	Common	Associated with vegetative endocarditis.
	Pyelonephritis	*E coli.* *Streptococcus* *Staphylococcus*	Common	Probably an ascending infection.
Dog.	Interstitial	*Leptospira canicola*	Very Common	—
		L. icterohemorrhagiae	Not so Common	Associated with rats
	Tubular.	Poisons. P, As, $HgCl_2$	Common	—
	Pyelonephritis	*Streptococcus* *Staphylococcus*	Rare	Associated with calculi
	Specific.	*M tuberculosis*	Common	Miliary.
Cat.	Interstitial	not known	Seen frequently	Leptospirosis occasionally.
	Glomerulonephritis.	not known	Rare.	
	Specific.	*M. tuberculosis*	Common	
Fowl.	Interstitial.	*S. pullorum.*	Common	Associated with pullorum disaese.

NEOPLASMS

Primary neoplasms of the kidney are not very common. Two neoplasms deserve special description They are "Embryonal Nephroma" and "Hypernephroma". These have been described under "Neoplasms" (Vide pages 261 and 262).

Secondary tumors may be found more often than the primary. The most common is the lymphosarcoma. Masses of the neoplastic cells may be found in the kidney as discrete nodules of varying sizes or the cells may infiltrate diffusely into the intertubular tissue. Then it may present a problem to distinguish these neoplastic cells from inflammatory infiltrating cells. But a careful examination reveals that in the neoplasm only the lymphoblastic cells are present while in the inflammatory lesion, besides the smaller lymphocytes, other inflammatory cells are also present.

DISEASES OF THE URINARY BLADDER

Anomalies: Persistent urachus may sometimes be seen in foals but very rarely in other animals. Urachus is the tube that connects the bladder to the umbilicus in the foetus. Just before birth, this is severed from the umbilical cord and becomes obliterated But in some instances, it is still patent after birth and is said to be "pervious". This is a problem needing surgery, since infection of bladder may occur through a pervious urachus.

Diverticula of the bladder wall and division of the bladder into several cavities are other anomalies rarely observed.

Rupture of the bladder may occur due to(a) Trauma—automobile accidents, gunshot wounds and faulty catheterisation (especially when bladder is full) or (b) obstruction of the urethra—by calculi, enlargement of prostate, neoplasms of urethra or inflammatory debris.

Prolapse of the bladder may occur through the urethra into vulva in cows, mares and sows due to straining during parturition. The short, broad urethra facilitates this condition. If not corrected, necrosis and gangrene of the bladder will supervene.

Perineal herniation of the bladder may occur in male dogs during straining in prostatic enlargements.

Calculi in the bladder form due to causes described under the subject "Urolithiasis". (See later) The calculi may be small and numerous and have a smooth surface. Or they may be large, when due to mutual rubbing against each other during contractions of the bladder, smooth facets develop.

Calculi are of consequence only when these occlude the urethral passage resulting in stasis of urine and attendant sequelae:- Viz, cystitis catarrhal or fibrinous developing into hemorrhagic); rupture of the bladder or uremia.

BOVINE ENZOOTIC HEMATURIA OR CHRONIC BOVINE HEMATURIA

This is a chronic disease of cattle that is observed in all parts of the world. It occurs as an enzootic only in certain localities In India this is mostly seen in the hilly regions of Darjeeling, Ooty, Kodaikanal. Garwhal, Kumaon and other Himalayan regions.

Though the exact cause has not yet been definitely established some toxic principle in the plants ingested may ultimately prove to be the cause.

Various agents have been incriminated from time to time. These are:—

Coccidia; liver flukes; piroplasms; schistosomes; *Aspergillus kamala;* high oxalic acid content of the food; deficiency of minerals in the soil (calcium, phosphorus, manganese etc.), feeding on bracken fern and a virus. In Russia and Poland, plants of the genus *Ranunculaceae* have been found to damage the kidney and bladder

The course of the disease is chronic and may last for months or one or two years. The early symptoms are the presence of blood in the last few drops of urine. As the disease progresses the blood content increases and in the later stages, pure blood may be passed. Secondary anemia supervenes with degenerative changes in various organs of the body. Animal becomes emaciated and finally dies

Macroscopicaliy, the lesions may be found on the mucosa of the bladder. In the early stages, petechiae or ecchymoses may be found, which enlarge and become confluent as the condition progresses. The mucosa becomes thickened and red cauliflower-like tumor masses develop on the walls.

During excretion, the irritant produces inflammation of kidneys which therefore manifest hemorrhages. Later due to mechanical obstruction to passage of urine cystic dilatation may be found.

Microscopically, in the early stages may be seen hemorrhages on the bladder, followed by hyperplastic proliferation of the epithelium. Metaplasia to squamous or columnar (mucin producing) epithelium is frequent. The hyperplasia may be of neoplastic dimensions and in some cases may be a precursor to carcinoma (precancerous stage).

The capillaries also proliferate and growths similar to hemangiomas are observed. These are of two varieties. One is arranged as cavernous hemangioma, with thin walled, large dilated blood spaces, The second is capillary hemangioma in which masses of endothelial cells invade the surrounding structures, even into the muscular coat. Lesions of glomerulonephritis, tubular degeneration and interstitial nephritis may be seen.

INFLAMMATION OF THE BLADDER—CYSTITIS

Causes : The most important predisposing cause is retention of urine, especially if associated with trauma. Hence paresis of the bladder is a chief contributory cause.

Infections by bacteria in the majority of cases is by the ascending route and so this is more frequent in the female animals with short urethra—cow, sow and mare. (from suppurative endometritis and vaginitis). In some cases, infection may be descending also (from suppurative nephritis and pyelonephritis).

Pervious urachus in the foals and calves is yet another route of infection while catheterisation is also found to be a factor in the causation of cystitis.

Infection may also occur by expansion from neighbouring organs. The infective organisms are : *Escherichia coli, Corynebacterium renale* (especially in cow and sow). *Proteus vulgaris, Staphylococci* and *Streptococci*

Cystitis may be acute or chronic

Macroscopically, acute catarrhal, fibrinous, purulent or hemorrhagic types may be found. In the catarrhal form, the mucosa, is thickened, edematous and

reddened. The urine is cloudy. In the hemorrhagic form due to hemorrhages in and on the wall of the bladder, the urine is coloured red, while in the purulent form the urine is opaque. In the fibrinous variety, flakes of fibrin are present in the urine

Microscopically, the epithelium is degenerated and desquamated. There is leucocytic infiltration of the mucosa and submucosa, and form dense sheaths around blood vessels. In the purulent variety the infiltration of leucocytes is heavy extending even to the muscular coat. Congestion of the blood vessels is seen. In severe conditions, ulceration of the mucosa supervenes.

In *chronic catarrhal cystitis*, (usually due to calculi) the mucosa is very much thickened due to fibrosis and frequently there is epithelial desquamation. There may be hypertrophy of the muscular layer with infiltration by lymphocytes.

Sometimes one may encounter chronic polypoid cystitis in cattle, in which the mucosa is thrown into folds. In this condition, there is dense infiltration of the proliferated connective tissue by mononuclear leucocytes.

In the dog, a follicular form is frequently seen in which the mucosa is studded with small, grey nodules, resembling lymph nodes consisting of aggregations of lymphocytic cells

Neoplasms of the bladder are seen occasionally. The most common is the metastatic lymphosercoma. in which the neoplastic cells infiltrate the wall of the bladder, forming diffuse or nodular thickening. The primary tumors include, leiomyoma, papilloma, transitional-cell carcinoma and squmous-cell carcinoma.

The Urethra : Affections of the urethra are rare since the lining mucosa is sufficiently tough to withstand infections.

Obstruction of the lumen by calculi is the most common cause of *urethritis*. In this condition, if the calculi are not removed, necrotic or hemorrhagic urethritis may result. When healing from urethritis takes place, stenosis of the urethral lumen may sometimes occur.

UROLITHIASIS

Urolithiasis means the presence of calculi in the urinary system. They may be found in the urinary tubules, in the ducts of Bellini (microconcretions), in the pelvis of kidney (nephrolithiasis), in the ureters, in the bladder (cystic calculi) or in the urethra

Urinary calculi are found in all animals and they are of greater importance in the ox, since the stone may be arrested at the sigmoid curve and cause fatal obstruction. The groove in the *os penis* of male dog is a frequent site for the lodgement of the calculi.

The calculi differ in their chemical composition in different species of animals. This difference is largely governed by the pH of the urine. In alkaline urine (herbivores) the calculi are mostly carbonates and phosphates of calcium, magnesium and ammonium. In the acidic urine (carnivores and omnivores) oxalates, urates, xanthine and cystine predominate.

With infection the nature of the urine may change depending upon the infecting organism. Staphylococcal infection renders the urine alkaline while infection by coliforms renders it acidic. So even in herbivores, in coliform infection of the urinary passages, oxalates and urates may be found.

The following types of calculi are common among animals:

Horses : Calcium carbonate; triple phosphate (ammonium-magnesium phosphate); magnesium phosphate; magnesium carbonate.

Ox and other ruminants : Phosphates and carbonates of calcium, magnesium and ammonium; silicates; oxalates.

Pigs : Triple phosphates; carbonates of calcium and magnesium; magnesium phosphate; oxalates; silicates

Dogs and Cats ; Oxalates, urates, uric acid, cystine, triple phosphates, calcium carbonate and phosphate.

The uric acid calculi as uratic calculi (composed of ammonium or sodium urates) are small, hard and brown in color. They show concentric rings on section These are common in Dalmatian dogs.

The oxalate calculi are very hard and the surface is rough and spiny (mulberry calculus). So it produces severe irritation, inflammation and hemorrhage and hence these stones are dark in color as they contain blood. These may also show concentric rings, but not as well defined as in uric acid calculi. These are mostly found in acid urine and may be solitary and large in the bladder.

Phosphate stones consist usually of calcium phosphate, magnesium phosphate or triple phosphate and are white, smooth, chalky and easily broken. These are formed in alkaline urine and may contain small amounts of urates, oxalates and carbonntes.

Xanthine stones are brownish-red, often laminated concentrically and are easily broken.

Cystine calculi are small, irregular, friable and yellow, becoming greenish on exposure to air. These are more common in cats.

Size : The size of the calculus depends on its location. The renal calculi may be microscopic in the tubules. The pelvic calculi attain to a size upto 8 cms and are hard and ovoid in shape.

The cystic calculi may vary from a grain of sand to that of a tennis ball. They may be smooth or rough,circular or ovoid and laminated or smooth on section. Some calculi develop smooth facets by the rubbing of one with the other.

They may be white or if containing blood, dark.

Formation of calculi . For the formation of the calculi there must be a nidus or nucleus around which salts may be deposited. Such a nidus may be organic material that may be found in the following conditions ;

a)	casts	From the injured nephrons in nephritis
b)	bacteria	
c)	leucocytes and	
d)	degnerated cells	
e)	keratioised desquamated epithelial cells	In vitamin A deficiency and in stilbestrol administration
f)	mucoproteins	

Causes : How urinary calculi form is not very clear. The following factors singly or in combination, may be the causes :

1 **Vitamin A deficiency :** In vitamin A deficiency, the transitional epithelium of the urinary tract undergoes metaplasia into keratinised stratified squamous epithelium. The keratinised epithelial cells get exfoliated and may form the nidus of calculi

2. **Infection :** Infection of the urinary tract by *Streptococci, E Coli* and *micrococci* may occur when formation of calculi may be facilitated because the exudate and bacteria may not only form the nidus but the reaction of the medium may be suitably altered for the deposition of salts But it is not always possible to detect infection in urolithiasis.

3 **Concentration of salts :** The organic and inorganic salt content of the food and water has an influence on formation of calculi. If the feeds consist of concentrates, with inadequate water, then calculi may be formed. Also if drinking water contains a high precentage of minerals, formation of calculi is facilitated, because in these circumstances, the mineral concentration of urine is increased. Hypervitaminosis D may cause hypercalcemia and so hypercalcuria. Curtailment of water, excessive sweating and ingestion of plants with high oxalic acid content increase the salt concentration of urine

An inborn error in the metabolism of these salts, probably predisposes formation of calculi.

4. **Deficient green feed:** It was found that when animals were maintained on dry concentrates without alfalfa or green forage, incidence of urinay calculi was greater This was due to the excretion of muco proteins in the urine in the absence of the green fodder These mucoproteins act as nuclei for the calculi.

5. **Sulfonamide medication :** Formerly, in the early days of sulfonam de therapy, calculi were common. If sulfa dugs were used without sodium bicarbonate and sufficient water acetyl salts were formed and precipitated in the renal tubules, pelvis and ureters, forming calculi.

6. **Hormones ;** (a) For fattening lambs diethly stilbestrol pellets are implanted under the skin. This has a metaplastic action on the urinary epithelium, which is transformed into keratinised squmous epithelium. The desquamating cells, form nuclei for the deposition of mineral salts. The calculi that form in the bladder may cause obstruction of the urethra.

(b) In man tumors of parathyroid, in which there is hypercalcemia, are associated with urolithiasis

7. **Prolonged confinement :** In man when patients are confined to bed for long periods with inability to move their limbs, the bones are decalcified and phosphatic calculi are formed in the bladder.

Sequlae : Calculi are harmful in two ways :

1. They may irritate the urinay passages and cause inflammation.
2. Obstruction of the passages may occur.

The results depend upon the place of obstruction.

a) If the obstruction is in the urethra, there is retenion of urine with attendant uremia and dilatation of the bladder. In some extreme cases the bladder may rupture with fatal results.

b) if the obstruction is in the ureter :

i) Atrophy of the corresponding kidney if obstruction is complete.

ii) Hydronephorsis if the obstruction is partial.

URINARY CASTS

Casts are supposed to be products of albuminous exudate into the tubules from the blood vessels. Presence of casts in the urine is known as *clyindruria*. Casts may also contain tubular epithelial cells that may become swollen, destroyed and desqumated. Usually urine containing casts gives positive test for albumin.

Presence of casts in urine, in most cases, is indicative of some type of nephritis and is of great diagnostic value.

Casts have been classified according to their microscopic appearance as follows :

Hyaline casts are pale, often colorless, homogeneous and cyindrical in shape. They have straight parallel sides and rounded ends. These casts dissolve in acetic acid (but fatty casts do not), Hyaline casts are found in congestion and in inflammation of the kidney.

Granular casts may be either coarsely granular or finely granular. They may be curved or straight with rounded or broken ends. The granular material is composed of albumin, fat, epithelial cells or disintegrated leucocytes or erythrocytes.

Epithelial casts contain epithelial cells of the tubules and indicate acute nephritis. They may be yellowish due to imbibition of blood pigment. When the condition changes to subacute or chronic, these casts may become fatty or waxy.

Waxy casts are more opaque than the hyaline casts and are yellowish in color. These are found in chronic nephritis, but seldom in acute. They are invariably found in amyloid degeneration of the kidney.

Blood casts are found in acute nephritis, renal hemorrhage and in acute congestion of the kidneys. They are formed of erythrocytes and so may have a reddish color.

Fibrin casts are found in cases where there are hemorrhages. They may be yellowish due to altered blood pigment.

Pseudocasts have no connection with renal disease but occur due to conglomeration of various substances on mucous threads. Urates and phosphates may aggregate together to resemble casts The following are examples of pseudocasts :

Fatty casts : These contain numerous fat globules which can be stained by Sudden III or Osmic acid. The fat is probably derived from the degenerated epithelial cells. The fat globules are not soluble in acetic acid

Pus casts consisting of pus cells that have been kneaded together are found in renal suppuration. They may contain a few fat globules also.

Cylindroids are not casts but consist of mucus and some fat globules. They are striated.

Bacterial casts occur due to proliferation of bacteria in stagnant urine but are not true casts.

CHAPTER 19

THE NERVOUS SYSTEM

By Dr. J. L. Vegad

GENERAL CONCEPTS :

The general laws of pathology remain fully applicable to the nervous system but certain peculiar characteristics of the nervous tissues have to be recognized. For example, when the central nervous system is attacked, the functional disturbances in turn may be widespread, and could affect the entire body. This becomes all the more important when we realize that once a neuron is destroyed, it cannot be replaced, The functional distrubances of the neurons (nerve cells) in the brain are therefore of supreme importance because they govern so many vital activities of different organs. When irritants alter the colloids of the nerve cells and their fibres, the functions of the nervous system may be directly disturbed, indirectly affecting the activities of the organs. The animal may not only be affected mentally, but its body movements, its gland secretions and its reflexes are also affected. Various activities of the brain can be upset by diseases. These are usually manifested by loss of consciousness, nervous depression, and increased nervous irritability. Another important feature of the central nervous system is that it is often possible to diagnose, from a careful

study of symptoms such as paralysis or other disturbances of function, as to in what particular portions of the brain or spinal cord lesions may be located. Actually, many symptoms noticed in diseases of other systems or in generalised diseases are due to either malfunction of or injury to the nerve tissue. In diseases of the nervous system the following symptoms are exhibited :—muscular tremors, ataxia, convulsions, dullness, coma, fever, anorexia, sleepiness, mania, stupor and paralysis. Though cytological and macroscopical changes can be demonstrated in the nervous system in some of the diseases showing the above symptoms, in some others, functional disturbance is not accompanied by morphological changes. For example, in Tetanus, though profound functional disturbances take place, yet no macroscopical changes occur in the nervous system

Loss of consciousness :- results from the effect of various toxic agents upon the brain. Complete loss of consciousness is said to be the state of coma. In this animal lies outstetched and motionless, its reflexes are gone, the pupils are dilated, respiration is slow and irregular, heart beat weak, and skin cool. It usually ends fatally. **Nervous depression** results from pressure upon the brain, such as the one brought about by hemorrhage within the cranial cavity a brain tumor, or even collection of fluid within the ventricles. There is loss of feeling, sleepiness, and muscular incoordination. A good example of this functional disturbance is equine encephalomyelitis. **Nervous excitement** results from congestion and inflammation of the brain and its coverings. There is delirium and mania, and even convulsions. The best example of this functional disturbance is rabies.

Disturbances in nervous functions also affect muscles in two ways. In the first case, there is increased activity (**spasm**), and in the other loss of contractility (**paralysis and paresis**). In **muscle spasms** there are sudden, violent involuntary contractions. They may be continuous (**tonic spasms**), or intermittent (**clonic spasms**). When the spasms are mild and are confined to groups of muscles, they are called **tremors**: a very good example of this being epidemic tremor (avian encephalomyelitis) of chicks. If the muscle spasms are widespread and involve the whole body, including the limbs, they are called **convulsions**. They are often seen in puppies infested with ascarids. When tonic and clonic spasms alternate, and are accompanied by loss of consciousness, they are termed **epilepsy**. In the second form of neuromuscular functional disturbance **paralysis** (the complete immobility of a muscle) and **paresis** (the incomplete loss of motion), the underlying cause is the defective innervation of the muscle. The defect may lie in the motor centres or in the conduction paths. It prevents the flow of motor impulses and immobility results. **Hemiplegia** is the paralysis arising in the brain cortex and in the peripheral nerves and is unilateral. Bilateral paralysis of the posterior parts of the body and hind limbs resulting from injury to the cord is called **paraplegia**.

REACTION OF NERVOUS TISSUE TO INJURY ;

Nervous tissue is very susceptible to injury. Further, when a neuron is destroyed, it cannot be replaced. When the nervous system is sick and fails to

perform its functions, the structural changes may be of three types: 1. macroscopic alterations. 2. microscopic alterations, or 3. the changes may be of a biochemical nature, and nothing may be visible even microscopically. It would be helpful to examine separately the reactions of the neurons and the glial cells to injury.

Neurones: These are the cells that are responsible for carrying the impulses The neurone consists of a cell body and one or more processes—the axon or dendrites. Degenerate neurones are normally found in healthy brain but more so in the young animals. Since autolysis occurs very early after death in the nervous system it is essential to know how to distinguish between autolytic and degenerative changes. The following are the changes noticed postmortem:—

Imbibition of large amounts of fluid giving a spongy, wet appearance to the tissue; Neurones and glia shrink, leaving a clear space between the cell and the surrounding parenchyma; Shrinkage and condensation of the nucleus (P. M. pyknosis); Fragmentation, fading and disappearance of the nucleus and cytoplasm and Nissl granules; Axis cylinders, which are normally unstained, take stain diffusely.

The neurones being highly specialised are easily susceptible to injury by hypoxia or toxic materials, evidenced by degenerative and necrotic changes. The following reactive changes are noticed.

1. **Shrinkage** characterised by cells becoming very irregular, nucleus pyknotic, clumping and condensation of Nissl substance and tortuousness or sclerosis of the processes. This is seen in senility and chronic infections.

2. **Swelling of the nerve cells:** Here the cytoplasm stains very faintly and only the cell outline may be discerned with fragmentation of the processes. This is a reversible condition and occurs in severe intoxications and systemic infections.

3. **Vacuolation** of the nerve cells may be seen in toxic conditions and in viral encephalomyelitis.

4. **Chromatolysis:** In this condition the Nissl substance becomes fine and dispersed and later may disappear. The nucleus may be eccentric. This change is seen in injury to the axon. It is suggested that the Nissl substance being a ribonucleoprotein is actively involved in the synthesis of axoplasm and so chromatolysis may denote an exhaustion phase. When axon regenerates and is repaired, Nissl substance is restored.

In viral infections and other severe intoxications chromatolysis occurs when the Nissl substance disappears. The cytoplasm also shows degenerative changes: swelling and rounded contours. Unlike in the axonal trauma, in viral infections and injury to central axons chromatolysis is irreversible since the neurones re destroyed.

5. **Neuronophagia:** when the nerve cell dies, microglia and oligodendroglia invade the cell and remove it by phagocytosis.

6 **Satellitosis:** Normally every neurone has one or two oligodendroglia near them. They are called satellite cells in this location. Whenever a neurone is damaged, oligodendroglia and microglia crowd around such cells, without actually invading them, and this phenomenon is known as "satellitosis". Both satellitosis and neuronophagia are indications of necrosis of a neurone.

The following are some of the causes that bring about the degenerative changes described above :—

Inorganic salts : Lead, arsenic.
Organic : Anesthetic agents.
Metabolic : Toxic products of uremia.
Infectious agents : Neurotropic viruses.
Nutritional deficiency : Deficiency of B_1, Copper, Cobalt
Vascular : Interference with blood supply—causing anoxia.

Effects of vaccination : Allergic encephalitis occurs after vaccination with vaccines containing brain tissue. There is destruction of myelinated tracts in the white matter

Toxic agents : a) In liver disease, toxic agents, exogenous or endogenous, are not detoxified and so these pass on to the brain producing degenerative changes

b) In mercury poisoning, neural degeneration and necrosis of the brain and demyelination of the nerve tracts extending to the spinal cord are noticed.

c) In *lathyrus* poisoning there is degeneration of neurones in the spinal cord accompanied by gliosis and ultimate atrophy of the spinal cord.

d) In poisoning, by chlorinated hydrocarbons, there is Nissl degeneration and necrosis of neurones, especially of the ganglia and brain stem.

Astrocytes, which are star shaped, are the supporting cells found throughout the central nervous system. These cells react to injury and proliferate and this process is known as "gliosis", which may be uniformly diffuse or may be focal.

Apart from the function of forming a supporting matrix, astroglia probably actively participate in the transport of fluids and solutes between the blood vessels and the nerve cells.

When nervous tissue is destroyed, repair does not take place by fibrosis The fibrous tissue from the adventitia of the blood vessels may repair sometimes. But, if a cavity arises due to softening and absorption of the brain, it is not filled in but a cyst is formed with a thin fibrous capsule around which the astrocytes proliferate. Inclusion bodies may be present in the astrocytes also. (gemistocytes in Canine Distemper).

Oligodendroglia are cells containing dark round nuceli and are found mostly in the white matter, in long rows between the fibres. They may also be found in small numbers as satellite cells around the nerves and blood vessels. Though their exact function is obscure, oligodendroglia are supposed to be connected with the maintenance of myelin sheaths. In places where medullated nerve fibres are destroyed oligodendroglia are found to disappear. The cells invade the dead neurones and engulf them (neuronophagia).

Microglia are mesodermal in origin (while astroglia and oligodendroglia are of neuroectodermal in origin) and so belong to the reticulo-endothelial system. They are the phagocytes of the central nervous system and are found in both white and gray matter. With ordinary stains, only their oval dark nuclei are noticed. Their branching cytoplasmic processes require special stains to be seen.

Microglia are amoeboid and phagocytic and become hypertrophied and proliferate. Hypertrophied cells that engulf the dead tissue are rounded and the

cytoplasm is foamy containing lipids and are called "*Gitter cells*" (from *Gitter-zellen*, German). "*Compound granular corpuscles*" or *fat granule cells*". Often these gitter cells migrate to the perivascular spaces (space of Virchow-Robin).

The meninges : The covering of the central nervous system consist of the dura and pia-arachnoid. While the dura in the cranium is attached to the cranial periosteum, in the spinal column it is separated widely from the vertebral periosteum. Pia closely follows the brain and is separated from the arachnoid by the subarachnoid space. The space contains the cerebrospinal fluid and has the spongy network of arachnoid trabeculae.

The meninges are composed of cells that are mesodermal in origin. Their reaction to injury is comparable to that of other tissues elsewhere in the body. Reaction to injury is by inflammation and fibrosis, unlike that found in the response of glial tissue.

Blood vessels :— The blood vessels of the brain have some peculiarities. These are : (1) The arterioles and venules are very thin walled, devoid of elastic and muscular tissue. (2) Veins do not have valves. (3) The blood vessels acquire a meningothelial sheath as they pass through the subarachnoid space and a second outer sheath derived from the pia. So a perivascular space is formed between these sheaths—the space of Virchow-Robin, which is continuous with the perineural and interstitial space of the C. N. S. It is in this space that the cells accumulate and give rise to "*Perivascular Cuffing*". Depending on the nature of the pathogen, the cells vary. In baterial infections, neutrophils predominate while in viral, lymphocytes and in allergic encephalitis macrophages, lymphocytes, plasma cells and eosinophils are found. In infections of the brain, inflammatory exudate collects in the space of Virchow-Robin. The adventitial wall is the source of macrophages (in some instances) and the fibrous tissue elements that compose the capsule in abscesses that arise in some bacterial infections.

Cerebrospinal fluid is found in the ventricles, spinal cord and the subarachnoid space and probably serves the function of lymph (which is absent in the C. N. S.). Its function is not only to serve as a medium for metabolic exchange but also to serve as a protective cushion for the delicate C. N. S.

Cerebrospinal fluid is formed by secretion and dialysis from the blood by the choroid plexus in the lateral ventricles. From the lateral ventricles the fluid passes into the third ventricle through the foramina of Monro and from there, through the aqueduct of Sylvius into the fourth ventricle. From the fourth ventricle, the fluid passes through the foramina of Luschka into the subarachnoid space. Mention has already been made that the subarachnoid space is continuous with the perivascular space. So the fluid is found in this place. Since perineural space also communicates with the subarachnoid space, the fluid is also found in the perineural space.

Most of the fluid is drained into the venous sinuses found in the dura through the activity of arachnoid villi, which project into these venous sinuses. Some fluid may also diffuse into the blood vessels from the space of "Virchow-Robin".

Normally, the cerebrospinal fluid is colorless and clear with Sp. gr. never exceeding 1009, pH. is 7.4 to 8. Leucocyte count is very low, fewer than 10-12 cells (mostely monouclears) per cubic millimeter being present. The globulin content

doses not exceed 10mg/100ml. But in infections, the C. S. F. may show considerable change. It may become cloudy or even bloody and the globulin content may be very high, 130 to 1500 mg / 100 ml. and the leucocyte count also being very high. Normal sugar content is 35 to 70 mg%.

Paths of infection :

Infection may reach the central nervous system by way of the blood stream or the lymph stream, or it may pass along the axis cylinders of motor or sensory nerves.

Blood-brain barrier :

It is now well established that the cerebral blood vessels differ from other vessels in their permeability. It has been demonstrated, for example, that when, large molecule dyes are injected intravenously they appear in the reticuloendothelial cells of liver, spleen and lymph nodes, but not in the microglia of brain, which also belong to the reticuloendothelial system, and are the brain histiocytes. Thus, blood-brain barrier is an obstacle that exists between circulating blood and the brain which effectively prevents a wide variety of toxic substances of large molecules from reaching the brain. The blood-brain barrier, thus is essentially a defence mechanism against noxious agents. In pathology, its importance lies in the fact that it successfully prevents the entry of most bacteria and viruses from the circulating blood into the brain. And, it is only when certain toxic substances, including bacteria and viruses, break down the barrier that they are able to enter into the brain tissue and set up the infection, or induce other pathological changes. Histologically, the blood brain barrier is composed of the vascular endothelium of the cerebral blood vessels, the basement membrane, and the perivascular glial membrane which is the close application of the cytoplasmic foot processes of innumerable astrocytes to the endothelium of the capillaries.

CONGENITAL ANOMALIES :

Anencephaly is the absence of most of the brain, and is seen in most animal species.

Acrania is the complete failure of cranial development.

Amyelia is the absence of spinal cord.

Cranioschisis is congenital fissure of the cranium.

Encephalocele is the protrusion of meninges, alone or with part of the brain, through a defect in the cranium.

Exencephalus is the absence of cranial vault exposing the fully developed brain.

Microcephaly is the presence of an abnormally small brain.

Meningocele is hernia of the meninges, which protrude through an opening of the skull or spinal column.

Rachicele is hernia of the spinal cord.

Spina bifida is a congenital defect in walls of spinal canal caused by lack of union between the laminae of the vertebrae. Through this defect the spinal cord may herniate. The condition has been repor-

ted in cattle, dogs and sheep. Sometimes there is no protrusion and swelling to indicate the defect This means the condition is hidden, and is then called **spina bifida occulta** (hidden.)

DISTURBANCES OF CIRCULATION

Hyperemia :

Acute general active hyperemia is present when bacterial or viral diseases involve the entire central nervous system (rabies, viral equine encephalomyelitis, and hog cholera).

Acute focal active hyperemia is seen in the vicinity of abscesses, tumours and infarcts.

Chronic general passive hyperemia : This occurs when there is a passive hyperemia due to lesions in the heart or lung or an obstruction to the flow of blood from the brain such as thrombosis of both jugular veins. Histologically, there are increased number of glial cells throughout the brain and spinal cord, which indicates chronicity of the condition.

Chronic focal passive hyperemia : This occurs when a tumour or abscess presses upon a vein, or a thrombus forms within a vein causing a reduction in the flow of blood from a local area of the brain.

Anemia :

General anemia : This occurs when anemia involves the entire individual. This may be seen in parasitic anemia as in gastrointestinal parasitism, excessive hemorrhage and in anemias associated with deficiency of iron, copper and the vitamin B complex.

The brain and spinal cord are whiter than normal, and the blood vessels contain decreased amount of blood and are therefore less prominent. Histologically, areas of liquefactive necrosis as well as gliosis and neuron degeneration may be present. These result from oxygen deficiency.

Local anemia or Ischemia : This occurs from a deficiency of arterial blood in a local area of the brain or spinal cord, the two main causes being thrombosis and embolism.

Thrombosis and embolism of carebral arteries are rare in animals and may occur in the brain and spinal cord. Emboli may be (i) detatched vegetations from the cardiac valves or may arise from lesions of the lungs, left atrium or coronary artery, (ii) clumps of bacteria, (iii) tumor cells, (iv) Parasites (larvae of ascaris, onchospheres of tapeworms, young trichinella etc.) or (v) agglutinated erythrocytes.

Thrombus can arise from leisons of cerebral vessels (atheroma) or can occur in diseases that damage the vascular endothelium :— trauma causing fractures of the skull, invasion of the vessel wall by neoplastic cells, abscesses and hog cholera. If collateral blood supply is inadequate infarction results The infarcted area is finally liquefied, a cyst being formed

If the blood supply is not adequate to maintain the nutritive and oxygen requirements of the area, **infarction** occurs. The infarcted area is pale or red depending upon the blood supply. Infarction ends up in liquefactive necrosis of the involved area

Hemorrhage

Petechiae are common in acute septicemic diseasees (Anthrax, Hemorrhagic septicemia, Hog cholera, Leptospirosis) or in infections by pyogenic organisms. These also occur after thrombosis or in degeneration of the vessel walls or in general hemorrhagic diseases as bracken fern poisoning

Rupture of an artery will give rise to large areas of hemorrhage with clots causing apoplexy. Rupture may occur in injuries—automobile accidents, gun-shot wounds, diseases of wall of blood vessels (atheroma) with hypertension as in arteriosclerosis, chronic-nephritis, bursting of an aneurysm as in parasitic aneu*rysm in horses.*

The first symptom in cerebral hemorrhage is shock, later passing on to coma and terminating in death. Animals that survive the first shock suffer from some degree of paralysis due to pressure on and damage to neurones. Hemorrhages may be found subdurally. They may also occur in the substance. of the brain. When hemorrhage is present in the ventricles, the cerebrospinal fluid may be blood tinged.

The blood clot in the brain first contracts separating the serum which is absorbed. The clot that remains is liquefied and a cyst is formed with a clear fluid—the "apoplectic cyst". The capsule of the cyst is formed by the neuroglia.

Hyperemia of the brain and meninges together with petechial hemorrhage and edema are found in the following conditions: (a) Electrocution, (b) Lightning stroke and (c) Sunstroke.

Edema of brain : *Edema of brain may be focal, caused locally by local lesions such as :*

(a) Neoplasms ; *(b)* Trauma accompained by hemorrhage and laceration; (c) Meningitis ; *(d)* Focal necrosis ; (e) Cerebral and meningeal hemorrhages.

Generalised edema of brain may be found in; i) Diffuse meningitis; ii) Viral encephalitis ; iii) Enterotoxemia caused by *Clostridium welchii* iv) Lead poisoning; v) Poisoning by organic mercury compounds; vi) Shock; vii) ANTU poisoning; viii) Sunstroke ; ix) Causes that give rise to general edema of the body ; x) Salt poisoning in pigs.

Macroscopically the brain appears more moist and heavy. The gyri are widened while the sulci are narrowed. Swollen gyri that press against the skull appear flattened.

On section. the gray matter appears wider while the internal white matter is softer. The ventricles appear narrowed.

Microscopically, the white and gray matter appear to have a loose texture and the interfibrillar space is widened. Neurones and glia appear swollen. Edematous fluid accumulating around the space of Virchow-Robin (Perivascular space) widens these areas.

Note :— Swelling of the brain occurs as a post-mortem change (Autolytic change) especially in the young animals and so care is necessary in interpreting the condition.

HYDROCEPHALUS is the condition on which there is abnormal accumulation of cerebrospinal fluid in and around the brain.

In animals this is a congenital condition due to some error in development, obstructing the pathways of fluid passage. Vitamin A deficiency during intrauterine life may cause internal hydrocephalus in calves and pigs.

If the accumulation is in the ventricles, the condition is called *internal hydrocephalus*. But if fluid accumulation occurs in the sub-dural space or pia-arachnoid, it is called *external hydrocephalus*.

Internal hydrocephalus can arise whenever there is obstruction to the free passage of the cerebrospinal fluid. The obstruction can occur at the foramen of Monro, the aqueduct of Sylvius or the foramina of Luschka. Cysts, (hydatids and coenurids) tumors or inflammatory exudate are the usual causes for the blockade. Congenital narrowing of the lumina may also be a contributory cause.

Due to pressure of the accumulating fluid the ventricles dilate and the adjoining nerve tissue atrophies The cranium is greatly enlarged causing foetal dystocia. If hydrocephalus develops before the cranial sutures fuse, the cranial bone may grow to a large size.

External hydrocephalus results due to either too much fluid formed and not rapidly drained by the arachnoid villi or to hindrance to the drainge of normally produced fluid as occurring in congenitally constricted tentorial aperture. It is usually the result of rupture of the thin dorsal wall of the third ventricle which allows the fluid to escape into the subarachnoid space between the cerebral hemispheres and the cerebellum The external hydrocephalus is called the "Communicating hydrocephalus" which is less common than the internal variety. The accumulated fluid exerts pressure on the surface of the brain. So there is a general atrophy of the brain and widening of the sulci between the convolutions. Usually, the exrernal hydrocephalus is "acquired". The result of hydrocephalus is pressure atrophy of the surrounding nervous tissue causing depression, incoordination, ataxia, and death.

DISTURBANCES IN GROWTH :

Aplasia : Aplasia of portions of the brain and spinal cord are observed in young animals.

Hypoplasia : This is relatively more common than aplasia; certain important examplesbeing-congenital posterior paralysis in calves and swine,spastic paresis in cattle and cerebellar hypoplasia in pigs, dogs, cats, lambs, goats, and calves.

Cerebellar hypoplasia: This anomaly is seen in calves and cats mostly but may also be found in other animals. In the Jersey calves and cats, this defect is inherited. Animals may usually die shortly after birth. Those that survive for a short while, show locomotor disturbance and incoordination. At necropsy, cerebellum may be found to be rudimentary or even absent. Cerebellar hypoplasia was encountered in calves born of cows which were affected with Virus diarrhoea-Mucosal Disease while pregnant. Similarly modified hog cholera virus causes, (when used as a vaccine, in the dam) cerebellar hypoplasia in the fetal pig.

Microscopically, molecular and granular layers are reduced in size, There is a relative reduction in Purkinje cells.

Hypertrophy : This may result from increase in size of the glial cells, microglia showing the greatest degree of hypertrophy. The neuron does not increase in size.

Hyperplasia : This results from an increase in the number of glial cells. Glia, especially the microglia, increase in number under conditions of hypoxia. Hyperplasia of the neurons does not occur.

Metaplasia : This does not occur in the nervous tissue proper. It may occur in the connective tissue of the meninges and blood vessels, in which case cartilage and bone may be found.

Atrophy ; Atrophy of the cerebrum may occur in hydrocephalus. Pressure atrophy also occurs in the vicinity of tumors, abscesses, haematocysts, and depression fractures of the skull.

DISTURBANCES IN CELL METABOLISM ;

Cloudy swelling : The neurons and the glia undergo ,cloudy swelling as a result of hypoxia for irritation produced by toxic substances or infectious agents. The cells become larger, cellular outline more round, and cellular structures indistinct

Fatty degeneration ; This appears as fat droplets in the cytoplasm of the neurons.

Hpdropic degeneration: It is a continuation of cloudy swelling: In this droplets of edematous fluid are observed in the cytoplasm of the neurons and glia

Amyloid infiltration . This is uncommon in the central nervous system of most domestic animals.

Glycogen infiltration : This does not occur in the central nervous system.

Pigmentation : In cattle and sheep, melanin is most frequently encountered in the pia mater of the anterior one-fourth of the brain, Focal areas of melanin may be found in other portions of the meninges and even within the brain and spinal cord.

Calcification : This is more commonly found in the meninges than in the brain and spinal cord proper. It occurs in the presence of dead tissue and faulty circulation, examples being abscesses, infarcts, parasitic lesions, sites of old hemorrhage and in necrotic neurons.

Traumatic injury the Nervous system : The brain being soft, is susceptible to shock that emanates from impact, especially from fast moving objects.

A sudden blow on the cranium may result in fracture of the cranial bones which may not be depressed, On the other hand a blow on the vertebral column ,results in fracture ,or dislocation. Fracture of the skull causes considerable damage to the meninges and brain. Hemorrhage may occur and nerve fibres disrupted. Hemorrhage aggravates the condition by the pressure of the accumulated blood on the brain tissue.

Concussion occurs when the skull receives a sharp blunt blow suddenly, not accompanied by fracture. There is loss of consciousness. The condition is not fatal and no morphological changes are present. Recovery is the rule. Lesions may consist of small hemorrhages in the brain and under the skin at the site of injury

Laceration : In this condition there is discontinuity of the tissue and usually occurs in automobile accidents. Blunt objects may cause laceration and *contre-coup* lacerations occurs on the brain on the side opposite to that on which the injury is struck. This is due to striking of the brain on the skull on the opposite side, since normally the brain is smaller than the cranium and is slightly movable. In such places hemorrhages are common Penetrating wounds, usually caused by

gun-shot wounds, are followed by severe hemorrhage. Fractures are also common in such injuries. Penetrating wounds are usually followed by secondary infections and are fatal.

NECROSIS

1. Coagulative necrosis :

This involves the neurons and the glia The causes are severe injury to the cells brought about by hypoxia, chemical poisons, bacterial toxins and viruses.

No changes are seen macroscopically. Microscopically, the cells are swollen and become more globular in shape. The Nissl substance may eventually disappear (chromatolysis or tigrolysis), the cytoplasm stains more intensely with eosin, and the nucleus shows pyknosis, karyorrhexis, or karyolysis. Microglia accumulate around the necrotic neurons, the process being known as **satellitosis**. When the microglia phagocytose the necrotic neuron, the process is called **neuronophagia**

2. Liquefactive necrosis :

This is the most common type of necrosis encountered in the brain and spinal cord compared to coagulative and caseous types because the nervous tissue contains little coagulable albuminous material but is rich in lipoids In fact, necrosis of the brain is almost always liquefactive in nature. It so happens that when necrosis occurs in the nervous system, the autolytic enzymes released from lysosomes of the dead cells, cause disintegration of myelin into a liquid mass that consists mainly of lipoid

Infarction is one of the common causes of liquefactive necrosis. It may also occur when the central nervous system is invaded by pyogenic bacteria. The lysosomal enzymes released from neutrophils induce liquefaction of myelin, neuroglia, and other structures, and this is known as *encephalomalacia*. Softening of gray matter is known as **poliomalacia** and that of white matter **leucomalacia**. Encephalomalacia is commonly seen in the following conditions :

Deficiency of Vit E. in young chickens (crazy chick disease); Mouldy corn poisoning in horses (Cornstalk disease); Acute pancreatitis in all animals; Antenatal copper deficiency in lambs (sway back); Cobalt deficiency (Enzootic marasmus); Enterotoxemia in lambs; Mulberry heart disease in swine; Vitamin B deficiency (Chastek paralysis) in fur-bearing animals and in calves and sheep, when it is called cerebro-cortical necrosis; Blue tongue in sheep; Rift valley disease of Kenya; Distemper of dogs; toxoplasmosis and lead poisoning; Infarction due to an embolus consisting of tumor cells or parasites or a piece of a thrombus or due to thrombosis of an artery; poisoning by mecuric salts.

The lesions seen are:-thickening of the blood vessels, endothelial hyperplasia and liquefaction of brain substance. Thrombosis and hemorrhage may be found in some cases. Around the area, there may be proliferation of capillaries and the formation of a capsule by the cells of meninges. Astroglia proliferate and surround the area of encapsulation. The involved tissue undergoes liquefaction and a serous fluid is present.

3 Caseous necrosis :

The cause of this type of necrosis is infection of the brain by **Mycobacterium tuberculosis**. The necrotic area is seen as a dry, crumbly, yellowish - white mass. It may even contain areas of calcification. Microscopically, all cellular or architectural structures are lost, and the necrosed area is surrounded by a zone of inflammation.

Necrosis of nerve fibres of the peripheral nerves, the tracts and central nervous system is first indicated by fatty degeneration of the myelin sheaths of the nerve fibres affected. This change occurring in the brain and spinal cord is called demyelination. Ultimately the axon may disappear, but demyelination alone can render the nerve fibre non-functional. If demyelination alone has taken place, regeneration is possible with restoration of function.

Gangrene :

This could occur if brain is invaded by saprophytic microorganisms, as if the case of traumatic injuries of the skull or as septic emboli from areas on gangrene in the lungs.

INFLAMMATION

Terminology :

Encephalitis — is inflammation of the brain
Myelitis — is inflammation of the spinal cord
Encephalomyelitis — is inflammation of the brain and spinal cord
Meningitis — is inflammation of the meninges
Pachymeningitis — is inflammation of the dura mater
Leptomeningitis — is inflammation of the pia mater
Meningoencephalomyelitis — is inflammation of the meninges, brain and the spinal cord.
Polioencephalitis — is inflammation of gray matter in brain
Poliomyelitis — is inflammation of gray matter in the spinal cord.

The same general laws of inflammation apply to the brain and spinal cord as elsewhere. Since there are no mucous membranes, catarrhal inflammation does not occur. Serous inflammation also probably does not occur: if it does it resembles edema. Hemorrhagic exudates are rarely met with and fibrinous inflammation is limited practically to the meninges. Purulent, lymphocytic and proliferative inflammations are the types which are regularly encountered in the central nervous system.

Fibrinous encephalitis, myelitis and meningitis :

These are seen in cattle and sheep during pasteurella infection of the central nervous system. They are characterized by cardinal signs of inflammation and increased fibrin content in the sub-arachnoid and Virchow-Robin space.

Suppurative (Purulent) encephalitis, myelitis and meningitis:

These are observed in all species of animals. There are the usual cardinal signs of inflammation ; the principal constituent of the exudate being pus. The inflammation may be focal or diffuse.

The pyogenic organisms responsible are staphylococci, streptococci, corynebacterium, pasteurella, listeria, and pleuropneumonia-like organisms.

Routes of infection : 1. By direct extension from suppurative conditions of the middle ear, nasal passage, cribriform plate or from meninges.

2. Through blood stream (in septicemic diseases) and lymphatic vessels accompanying nerves (Listeriosis).

3. Infection of penetrating wounds of the skull. Suppurative myelitis will result due to infection of the wound made while docking the tail.

The lesions may be microscopic and consist of focal collection of neutrophils and lymphocytes. The accumulation of pus causes pressure and destruction of the local tissue If an important area in involved, severe effects follow. The abscesses do not have well developed capsules as mesodermal cells that form it are few Astroglia proliferate and form a poorly defined capsule around the cerebral abscess.

Listeriosis (Listerellosis)

This is the most frequent cause of a purulent reaction in the brain of farm animals. Infection by Listeria monocytogenes produces suppurative meningoencephalomyelitis in cattle, sheep and goats. How actually this organism reaches the central nervous system is not known. The disease is characterized by the presence of multiple microabscesses which contain the organism.

Lymphocytic meningencephalomyelitis :

This is the most important form of inflammation of central nervous system in animals. In this the lymphocyte is the principal constituent of the exudate : the cells being trapped in the Virchow-Robin spaces as they leave the vessels—**perivascular lymphocytic infiltration or perivascular cuffing.** This type of inflammation is caused mainly by viruses.

The viruses may be (1) Neurotropic. That means to say, those that affect almost only the nervous system. Examples of these are the Rabies in dogs and Borna disease in the horses.

(2) Organotropic. The viruses that affect other tissues may also infect the nervous tissue by chance. Examples : Canine distemper; Hog cholera; Epidemic tremor of fowls ; Malignant catarrhal fever and Rinderpest.

Encephalitis is said to be due to allergic causes, when no other obvious etiological factors can be discerned. Under this category are listed those conditions that result after vaccination (Post-Vaccinal encephalitis) with vaccines containing nerve tissue. Others that may produce encephalitis are the causal agents of psittacosis and ornithosis, PPLO, rickettsia and trypanosomes.

Routes of entry: 1. Blood stream—Hog cholera,

2. Nerves—Rabies.

3. Neurolymphogenous—infection ascending along the lymph pathways of cerebrospinal nerves.

The virus entering a neurone kills it. Demyelination of the nerve fibres may be present. Petechiae also may be found.

Macroscopically, no gross lesions of significance are noticed. Hyperemia and edema of the pia-arachnoid may be the only lesions seen. Occasionally localised areas of softening may be found.

Macroscopically, the following are the characteristic features noticed :

1. Congestion and hemorrhages.

2. Cuffing of blood vessels : i.e. accumulation of lymphocytes in the space of Virchow-Robin. Later plasma cells and macrophages may also be found in these places. This lesion is found both in the white and gray matter.

3. Edema.

4. Gliosis : diffuse proliferation of the astrocytes throughout the brain giving the tissue a dense and cellular appearance.

5. Satellitosis—appearance of scavenger cells or "Hortega cells" or "Gitter cells" around the necrotic area. These are the microglia and oligodendroglia which remove the dead neurones and debris by neuronophagia. The gitter cells contain lipid and are the only cells seen in the area where neurones were situated previously. Some times in more chronic cases, the microglia increase in number, their nuclei become elongated and their cytoplasam contains iron deposits and such cells are known as "Rod cells"

6. Neuronophagia.

7. Rarely proliferation of blood capillaries may be seen.

8. Inclusion bodies may be found in the neurones or astroglia in a number of diseases ; rabies, canine distemper, infectious canine hepatitis, Borna disease

SPECIFIC INFLAMMATIONS OF BRAIN AND SPINAL CORD :

Rabies :

It is an acute viral disease of domestic animals characterized by a very severe lymphocytic inflammation of nervous system. There is diffuse and severe meningoencephalomyelitis. A characteristic feature of the disease is the presence of intracytoplasmic inclusion bodies (Negri bodies) in the cells of the hippocampus and cerebellum.

Pseudorabies :

It is an infectious viral disease of cattle, pigs, dogs and cats. In pigs there is diffuse lymphocytic meningoencephalomyelitis which does not seem to occur in cattle.

Hog cholera (swine fever) encephalitis :

In hog cholera 80 to 90 per cent of the animals suffer from an acute diffuse lymphocytic meningoencephalomyelitis.

Canine distemper encephalitis :

The virus of canine distemper also produces a typical diffuse lymphocytic meningoencephalomyelitis. However, not all the affected dogs develop lesions in the central nervous system.

Infectious viral equine encephalomyelitis :

This is an acute viral disease of horses and mules and is also characterized by a typical diffuse lymphocytic meningoencephalomyelitis. It terminates fatally in about 50 per cent of the cases.

Borna disease :

This is an acute diffuse viral meningoencephalomyelitis of the horses that occurs in Europe, especially in Germany. It has not been described in India.

Louping ill :

This is an acute diffuse viral lymphocytic meningoencephalomyelitis of sheep in Scotland, England, and Ireland.

Avian viral meningoencephalomyelitis or Epidemic tremors :

It is an infectious viral disease of chicks. Microscopically, the disease is characterized by a diffuse lymphocytic inflammation of the entire central nervous system.

Ranikhet disease (Pneumoencephalitis of poultry) :

It is a viral disease of chickens which is also characterized microscopically by diffuse meningoencephalomyelitis.

Chronic meningoencephalomyelitis :

The only important example of this in domestic animals is that of tuberculous meningoencephalomyelitis. The lesions in the central nervous system consist of a central area of caseous necrosis which may be partially calcified. This indicates that a generilized tuberculosis is present.

PARASITIC ENCEPHALOMYELITIS

Myiasis :

Hypodermo bovis larvae may be found in the fat of the vertebral canal in cattle. At times they may invade the spinal cord or the brain. The larvae of *Oestrus ovis* have also been reported to invade the brain.

Cestodiasis:

Tapeworm cysts are found in the central nervous system of the domestic animals, examples being the cysts of *Multiceps multiceps*, *Taenia pisiformis* and *Taenia echinococcus*. The ova of these tapeworms are ingested by the animal. In the intestine, the hexacanth embryo comes out, pierces the intestine, and is carried by blood stream to various places in the body. Some of the larvae, particularly those of *Multiceps multiceps*, reach the brain where they encyst. The path of migration of the larvae in the brain is macroscopically visible as red streaks due to the persence of hemorrhage.

The larval stage of *Multiceps multiceps*, a dog tapeworm is known as *Coenurus cerebralis*. It causes a rather uncommon disease of the central nervous system of sheep, known as '*gid*' or '*sturdy*'. The symptoms depend on whether the bladderworms are located in the brain or spinal cord. In both the places, they form cysts reaching 50 mm in diameter or more. Each cyst is filled with clear fluid and contains even up to 500 scolices. As the cyst enlarges there is pressure atrophy of the surrounding nervous tissue. So the convolutions may be flattened and cortex becomes thinned Even the cranial bones may be subjected to pressure atrophy and some may be punctured even. The chronic irritation induces a chronic lymphocytic meaingitis, encephalitis, or myetitis, depending on the location of the parasite. In severe infection, an acute diffuse lymphocytic meningoencephalomyelitis is produced, and the animal dies. Cysts usually involve the lumbar portion of the spinal cord, which results in such symptoms as inco-ordination and paralysis of the posterior extremities. Death of the larvae will result in calcification of the cyst.

Nematodiasis :

Various nematode larvae are found in the central nervous system. Strongyle larvae are found in the horse. The larvae of ascarids, strongyloides, hookworms and microfilaria of *Dirofilaria immitis* may be found in the capillaries or in the nervous tissue of the brain, spinal cord or meninges. Their lesions are characterized by a chronic lymphocytic inflammation.

Cerebrospinal nematodiasis (Neurofilariasis; Kumri);

This is found in sheep, goats and horses, and is caused by *Setaria digitata*. In horses it is known as Kumri (Hindustani for 'weakness of the loin') in our

country, whereas in sheep and goats it is known as 'lumbar paralysis'. Besides India, the disease also occurs in Srilanka, Burma, Korea and Japan

Setaria digitata is a natural parasite of cattle. But when the microfilaria find entry into heterologous hosts, like sheep and goat, they wander away to the central nervous system. Symptoms are basically neuroparalytic and include motor weakness, inco-ordination and loss of balance. Severe cases exhibit paresis of one or all limbs, the hind limbs being most frequently and severely affected. The cases may terminate fatally, or recovery may follow.

Macroscopically, lesions are found in the brain and spinal cord and the severity depends on the number of parasities present. Narrow tortuous tracks of hemorrhage and softening may be found denoting the path taken by the parasite.

Microscopically, the lesions consist of a central space (where the parasite was) surrounded by a degenerated and necrotic tissue The necrosis is liquefactive in type. Hemorrhages may be present in this area Due to damage by the larvae the axis cylinders in the affected area are swollen and degenerated and appear enlarged and fragmented. The myelin sheath becomes swollen and distorted, accompanied by glial proliferation.

Lymphocytes, eosinophils and microglia infiltrate around the area. Perivascular cuffing of nearby vessels is observed. The larvae may not be visible in these lesions as they might wander off. Careful microscopic examination of the tissue and cerebrospinal fluid is neceesary to see the larvae.

The condition known as "Wobbles" among young horses and mules may be a manifestation of cerebrospinal nematodiasis, Animals one to two years of age are affected. Suffering animals move with difficulty and on motion sway (or wobble) from side to side. They may fall frequently and show difficulty in rising. Trauma of the spinal cord at the cervical region is suspected to be the cause by some authors. Weakness inherited genetically is also suggested. Lesions are found only in the cervical portion of the cord where tracts of hemorrhagic necrosis and bilateral symmetrical areas of malacia are found

Microscopically, the following are noticed liquefactive necrosis, hemorrhage, degeneration and demyelination of the peripheral nerve tracts, perivascular cuffing, satellitosis, neuronophagia and gliosis.

In some cases, though parasites may be found in the central nervous sytem neither symptoms nor lesions are observed.

Toxoplasmosis :

Toxoplasma gondi, a protozoan parasite is found in the central nervous system of domesticated animals. Lesions consist of a central area of coagulative necrosis, surrounded by microglia and neutrophils. There is also lymphocytic meningitis, lymphocytic perivascular cuffing, and gliosis

'Allergic' encephalitis (Postvaccinal encephalitis) :

This sometimes occurs in dogs following rabies vaccination, It occurs 2 to 3 weeks after vaccination, and is characterized by a lymphatic meningoencephalomyelitis. There is motor paralysis of one or more limbs, which may later involve most of the body. Death is the usual outcome.

MENINGITIS

Pachymeningitis: The inflammation of the dura mater is usually secondary to infection of the middle ear or adjacent bone. It may be suppurative or non-suppurative In the suppurative variety which is more common, local abscesses may be found on the dura and the peridural spaces. Subsequently chronic fibrosis may develop when the dura is thickened with local adhesions Infection may spread to the arachnoid causing leptomeningitis.

Leptomeningitis is the inflammation of the pia-arachnoid. When associated with inflammation of the brain, which is usually the case, the condition is known as meningoencephalitis

Leptomeningitis may be suppurative or non-suppurative. The causes are:-

1. Extension from adjacent tissues—as in viral encephalitis (swine fever, rabies etc).
2. Mechanical injuries—fractures.
3. Bacterial infection from neighbouring areas—middle ear, nasa cavity and sinuses (Usually *Streptococci* and *Staphylococci*: other bacteria that may cause leptomeningitis are :— *Listeria, C. pyogenes, Pseudomonas*, coliforms, Pasteurella ; Chronic meningitis is produced by *Toxoplasma, Mycobacterium tuberculosis* and *Cryptococcus* In swine *Leptospira pomona* causes non-suppurative meningitis).
4. Hematogenous infection in septicemic conditions—navel ill; enzootic pneumonia, Colibacillosis, purulent pneumonia, metastasis from infections such as mastitis, metritis or peritonitis.
5. Parasitic invasion as in multiceps infection in sheep.
6. Hemorrhagic meningitis is seen in acute lead and copper poisonings. Due to the movement of the C. S. F. the inflammation is usually diffuse.

Hyperemia is severe in meningitis. The causative organisms grow on the surface of the pia-arachnoid and in its spaces. Injury to the blood vessels is responsible for the inflammatory exudate. In suppurative meningitis, the exudate which is yellow or greenish accumulates in the pia-arachnoid space. Pus may also be found in the spinal fluid. When it accumulates in the lateral ventricles, the convolutions tend to be flattened. Due to gravity the inflammatory fluid collects at the base of the brain. Suppurative inflammation is characterised by the infiltration of neutrophils while mononuclears (lymphocytes and macrophages) predominate in the non-suppurative variety.

Examination of the spinal fluid collected from a lumbar puncture gives, valuable information as to the nature of infection.

Myelitis is inflammation of the spinal cord. Usually myelitis is found along with encephalitis when the condition is known as encephalomyelitis Myelitis may be suppurative or non-suppurative and the lesions are comparable to those of the brain. Sometimes non-suppurative myelitis may occur without any attributable cause. In such cases trauma (automobile accidents) are believed to be the cause. Fractures of spinal column and protrusion of inter-vertebral disc may be other causes.

Macroscopically, there may be congestion of the pia, petechiae on and inside the spinal cord and in advanced cases, softening of the nervous tissue.

Microscopically, congestion, infiltration by inflammatory cells and degenerative changes of the nerve cells are seen. The nerve cells are swollen. Nissl substance disappears and the nucleus assumes an eccentric position. Degenerative changes of the nerve fibres are also observed

THE PERIPHERAL NERVES

Degeneration : When a nerve cell undergoes degeneration due to action of an irritant, the degenerative process also affects the nerve fibre of that cell. This is known as descending degenaration. Degeneration can also begin in the nerve fibre and progress towards the nerve cell (ascending degeneration). **Microscopically**, both axis cylinder and myelin sheath are simultaneously involved (total degeneration). Loss of the myelin substance is called **demyelination.** Lipoid of the myelin can be stained and demonstrated by Marchi's method.

When a nerve fibre (axon) gets severed from its cell body, the distal part of the nerve fibre undergoes characteristic degenerative changes known as **Wallerian degeneration.** The axis cylinder disintegrates and disappears, the myelin sheath (medullary sheath) also degenerates and is transformed into a chain of lipoid droplets which can be stained black by Marchi's method. The cells of the sheath of Schwann proliferate and get converted into phagocytes which remove the remnants of axis cylinder and the lipoid droplets Similar change occur in the proximal part up to the first node of Ranvier.

Regeneration: In a degenerated nerve fibre there are also attempts at repair. Nerve fibres in the central nervous system cannot regenerate, but the peripheral nerves regenerate fairly rapidly. Schwann cells play a leading role in the healing of nerves. If the sheath of Schwann (neurilemma) is intact, the Schwann cells proliferate and arrange themselves in both proximal and distal ends in the form of a tube. Along this tube new axis cylinders grow and unite the two severed ends. They fail to heal the gap if it is more than one inch. In such cases, the gap is filled in by granulation tissue which originates from the three connective tissue coverings of the nerve and its bundles. The Schwann cells proliferate at both ends. In case of amputation, the axon fibrils coil up and form a nodule called *amputation neuroma* which is covered by fibrous tissue

When a peripheral nerve is cut degenerative changes occur in the neurons. These changes are called *Nissl's Degeneration* in which the cells become enlarged and the nucleus is pushed to a side (eccentric) Chromatolysis of Nissl's substance occurs after breaking up. When regeneration of the nerve fibre starts the neuron tends to return to normal. Nissl granules reappear, nucleus takes up a central position and the cell becomes smaller. Repair in a nerve fibre is a prolonged process requiring 10 to 12 months for complete healing. Repair of the nerve fibres of central nervous system, lacking a sheath of Schwann, does no occur

Neuritis is inflammation of the peripheral nerves Inflammation of the nerves is usually accompanied by degenerative changes.

Causes: 1. Trauma.
2. Toxins—bacterial mostly. Neuritis occurs in infectious diseases as in strangles, protozoal—dourine, viral—rabies and distemper
3. Chemical poisons—lead, mercury, arsenic, alcohol.
4. Plant poisons—*Lathyrus sativus.*

5. Nutritional deficiency—deficiency of the members of vitamin B group.
6. Allergic factors.
7. Viruses—Marek's disease, Ranikhet disease.

Macroscopically, the nerve may be swollen and reddened. More often no naked eye changes are noticed.

Microscopically, degenerative changes, even leading to Wallerian degeneration are found. Edema and infiltration by inflammatory cells of *interstitial connective tissue* can be seen. The exudate may be serous (serous neuritis) or purulent (purulent neuritis). The latter variety may destroy the nerve completely.

Gross evidence of any abnormality may be completely absent. In some cases, however, nerves may reveal mild congestion or they may be unusually swollen, soft or flabby. Microscopically, inflammatory changes are present in the connective tissue and degenerative changes in the nerve fibres. All degrees of changes are met with, the more severe forms leading to destruction of the nerve fibres and Wallerian degeneration in the distal portion of the fibres.

Marek's disease:

It is a lymphoproliferative disease of the domestic fowl which has an unusual predilection for peripheral nerves. It occurs in classical and acute forms. The classical form is characterized by peripheral nerve enlargement, and paretic and paralytic symptoms. The nerves that are commonly affected are the brachial and sciatic plexuses, coeliac plexus, abdominal vagus and intercostal nerves. Microscopically, the nerves are infiltrated with lymphoid cells, which include primitive and activated reticular cells, lymphoblasts and small, medium and large lymphocytes.

CONGENITAL MYOCLONIA OF PIGS (TREMBLES)

New born pigs are affected by this condition, which is characterised by clonic convulsive movements due to hyperirritability of muscles. After a few weeks the symtoms may disappear and the pigs may then thrive. In severe cases they die of inanition as the piglets are unable to suckle properly.

Cause: Definite cause is not known. The following are suspected: heredity factors, hypothyroidism, virus infection, defective care and deficiencies of the sow during pregnancy. Many workers feel that the last two may be important.

Macroscopically, no visible changes may be noticed. Edema, thickening and hemorrhage in the cerebellum may be noticed. Congestion and hemorrhage may be observed in the brain, lymph glands, liver, kidney, lungs, spleen, thymus and ocular muscles.

Microscopically, there may be delay in the myelin sheath formation in the spinal cord. Ganglion cells of corpus striatum may reveal changes of shape and vacuole formation. Vasculitis affecting small arteries (sometimes obliterating them) is seen in various organs.

EPILEPSY

Epilepsy is a sudden brief (petit mal) or prolonged (grand mal), loss of consciousness usually preceded by convulsions.

Symptomatic epilepsy: may occur in animals due to organic brain lesions such as neoplasms or inflammation or trauma; disturbances in brain metabolism

due to visceral pathology or metabolic diseases or poisons; cerebrospinal nematodiasis ; verminous infestations or profound toxemias.

True or Idiopathic epilepsy is an inherited condition in Brown Swiss cattle and Cocker spaniels. The inheritance is through a recessive factor. Between attacks the animals are perfectly well and the condition persists for life.

Symptoms · A true grand mal epileptiform seizure is manifested by an early period of alertness, followed by a state of tetany, which gives way after a few seconds to a clonic convulsion with padding, opisthotonus. champing of jaws and salivation. The clonic convulsions are followed by a period of relaxation. The convulsion may spread from the initial area to the rest of the body, which is referred to as Jacksonian epilepsy. The animal is unconscious throughout the seizure. Evacuation of the bladder or bowel or both is common during the seizure. The animal may quickly regain its normal state after the seizure or act dazed or uncoordinated for a few minutes The temperature may be elevated or normal. The pulse is frequent and respiration rate is increased. The blood, cerebrospinal fluid and urine are normal.

The attcks are always recurrent and the animals are normal in the intervening periods.

Symptomatic treatment may be administered. Sedatives are usually resorted to.

Control : The animals should not de used for breeding.

SWAYBACK OR ENOZOOTIC ATAXIA

The disease is seen in new born lambs in certain parts of the world. The symptoms noticed are severe ataxia, locomotor disturbance, paralysis and inability to walk. Affected animals may be blind and so are unable to move. Death may also be due to broncho-pneumonia (exposure).

Swayback is attributed to a deficiency of copper. The ewes which are maintained on a copper deficient diet or grazed on lands with molybdenum-rich grasses may manifest anemia and produce "Steely" wool. Lambs of such ewes show demyelination (see page 149) and suffer from "Swayback".

Macroscopically, lesions are not prominent in mild cases. But in severe cases, cavities containing gelatinous material may be found in the white matter due to liquefactive necrosis with secondary internal hydrocephalus. The lesions are bilaterally symmetrical. Flattening of cranial bones occurs due to cystic degeneration and increased intracranial pressure.

Microscopically, diffuse symmetrical destruction of the white matter in the cerebrum is noticed, which is liquefactive necrosis. There is destruction of descending myelinated tracts. Gitter cells are numerous in the area. There is reduction in the cytochrome oxidase activity of neurones.

Tumors (Neoplasms)

Primary tumors of the brain and spinal cord are rare. However, tumors of the central nervous system are most common in dogs and least common in the pig and sheep. Primary tumors include those of the neuroglia (gliomas

astrocytoma, oligodendroglioma), nerve cell and fibres (neuromas), ganglion cells (ganglioneuroma), ependymal cells (ependymomas), and of meninges (meningiomas). Central nervous system is also prone to secondary tumors, which are metastatic, their primary sites being the lung or some other organ.

The brain tumors are of limited malignancy, metastases not occurring elsewhere. Pressure on the brain by the developing neoplasms produces various symptoms depending upon the part of the brain involved and the functional disturbances in turn are dependent on the neurons of that part which are responsible for them. Death is the invarible outcome.

CHAPTER 20

THE REPRODUCTIVE SYSTEM

(Revised by Dr. P Rama Rao)

THE FEMALE GENITAL SYSTEM

- Developmental anomalies
  - Freemartin
  - Arrests in the Mullerian duct system
    - White heifer's disease
  - Intersexes
- Ovary
  - Developmental anomalies
  - Disturbances in growth
    - Aplasia
    - Hypoplasia
    - Polyoogonia
  - Hemorrhage
  - Perioophoritis
  - Oophoritis
  - Cystic ovaries
    - Follicular cysts
    - Leutin cysts
  - Tumors
- Bursa
  - Bursitis
  - Adhesions of bursa
- Fallopian tubes
  - Malformations
  - Hydrosalpinx
  - Salpingitis
  - Pyosalpinx
  - Tuberculosis
  - In birds
- Uterus
  - Malformations
  - Rupture
  - Malposition
    - Torsion
    - Hernia
    - Prolapse
  - Circulatory disturbances
    - Hyperemia
    - Hemmorrhage
    - Thrombosis
  - Atrophy
  - Hypertrophy
  - Metritis
    - Acute catarrhal (endometritis)
    - Chronic non-suppurative
    - Acute suppurative
    - Chronic suppurative
    - Sclerotic
    - Perimetritis
  - Tuberculosis
  - Brucellosis in pigs
  - Mucometra/Hydrometra
  - Uterine abscesses
  - Endometriosis
  - Mummification of fetus
  - Maceration of fetus
  - Abortion
- Cervix
  - Malformation of cervix
    - Double External Os
    - Hypoplasia of cervix
    - Tortuosity of cervical canal
    - Cervical dilatation and diverticula
    - Prolapse of cervical rings
  - Cervicitis
  - Mechanical injuries
  - Cysts
  - Neoplasms
- Vagina
  - Developmental anomalies
  - Cysts
  - Rupture
  - Vaginitis
    - Granular
    - Vesicular
    - Epivag
  - Pneumovagina
  - Tumors
- Oviducts of birds
  - Abnormal eggs
- Mastitis
  - In bovines
  - In ewes
  - In pigs

DEVELOPMENTAL ANOMALIES

Freemartin

The bovine freemartin is a genetic female born cotwin with a normal male with which it has exchanged whole blood. The structural modifications of female genitalia are supposed to result from the influence of androgenic hormones produced by the male foetus

The gonads are undifferentiated. Ovaries are small. The Mullerian duct system is not differentiated fully. There are usually portions of tubular system, which do not develop and often the uterus is small and incomplete. The vulva has frequently long tufts of hair. The clitoris is quite prominent The vagina is fairly developed and the cervix is usually absent. One feature of the reproductive tract, which is very useful in distinguishing this condition from severe cases of aplasia of Mullerian ducts is the presence of seminal vesicles. Epididymis may be present or absent. The histologic appearance of the gonad is one of a quite undifferentiated structure. There are small tubular structures resembling primitive seminiferous tubules with lining cells similar to the sertol. cells. There are interstitial cells, which in the new born freemartins resemble fibroblasts. In the freemartin which is allowed to live the age of one or more years, the interstitial cells develop and resemble luteal cells in the ovary or Leydig cells of the testis. In the older animals, these develop into multiple large masses of orange or tan colored masses which resemble both interstitial cell tumor or corpora lutea on gross examination. Most freemartins do not develop ovarian follicles. Endometrial glands are present and produce fluid resulting in cystic distension of vestigeal remnants

The seminal vesicles are usually small and have abundant fibrous stroma. The epithelium resembles that of seminal vesicles of a castrated bull.

Arrests in the Mullerian duct development

These defects are of significance only in cattle and swine.

White heifer's disease

This condition is seen more commonly in short horn cows due to arrest in the Mullerian duct system and consists of a number of abnormalities.

Depending on the intensity of arrest in the development it may be classified into three groups.

Group A — Hymenal constriction; absence of anterior vagina, cervix, uterine body; cystic dilatation of uterine horns. Presence of well marked Wolffian bodies and occasional submucous vaginal channels.

Group B — Uterus unicornis, the abnormal horn being present as a flat muscular band. Hymenal constriction may or may not be present.

Group C — Essentially hymenal constriction and the rest of genitalia comparatively developed. If constriction is complete, gross uterovaginal distension results.

In addition to aplastic or hypoplastic defects of Mullerian ducts, there are common anomalies which results from failure of fusion of caudal portions of the ducts. Complete failure of fusion results in double vagina and double cervix. The more common failures of fusion occur in or adjacent to the cervix. The anterior vagina may be partitioned by a dorsal septum in conjunction with

a double cervix. A dorso-ventral band may be present across the external os (double external os), the cervix and vagina being properly fused. The failure of fusion may involve only a part of the cervix, chiefly the caudal part, so that there is one uterine body and bifurcated cervical canal with duplication of external os. The cervix and uterine body may be completely divided, a condition known as **uterus didelphys**.

Intersexes. The intersex is an individual with congenital abnormality, where the diagnosis of the sex is confused. Intersexes may be of two types; 1) True hermaphrodites in which gonads of both sexes are present; 2) Pseudohermaphrodites having gonads of one sex only but possessing reproductive organs with some characteristics of the opposite sex. Male and female pseudohermaphrodites depending on the gonads present are recognised.

Pseudohermaphroditism is very common in goats and studied in detail from genetic point of view. It is caused by a recessive gene. The incidence of hermaphroditism in Sannen breed of goats is high and mostly present as male pseudohermaphroditism.

Intersexes are common in pigs but not to the same degree as in goats. In bovines intersex seems to be a rare condition.

THE OVARY

Placed in the abdominal cavity, the ovaries are well protected from exogenous causes.

The functional activity of the ovary is under the control of the anterior pituitary through the two hormones, Follicle Stimulating Hormone (FSH) and the Luteinising Hormone (L H) Hence any pituitary endocrine disurbance affecting the gonadotropin levels affect the ovaries considerably. Placenta also elaborates certain hormones which have considerable effect on the ovarian function. Ovary produces two hormones, estrogen and progesterone, which have very important functions on the development of tubular female genital organs and control of estrus cycles.

DEVELOPMENTAL ANOMALIES

Supernumerary and accessory ovaries are very rarely seen in cow. Supernumerary ovary is an extra gonad which is entirely separate from the normally placed gonad and appears to arise from a separate analogue. An accessory ovary is situated near the normally placed gonad and may be connected to it giving the impression that it developed from a normal ovary.

Anomalies in the position of the ovaries occur and vary according to the length of the broad ligament. It is not unusual to find cows with a short broad ligament on one side resulting in the ovary being located closer to the body wall than normal In cows with uterus unicornis the gonad of the affected side is generally located near the body of the uterus. On the other hand in bitches with uterus unicornis, the ovary may be located some distance from the uterus. In such cases, the ovary may be overlooked during routine ovariohysterectomy.

DISTURBANCES OF GROWTH

Aplasia or absence of ovaries is occasionally seen, especially in swine and sheep

Hypoplasia of the ovary is considered to be due to the failure of migration of primordial germ cells from the yolk sack to the developing gonad during embryonic stage. Thus the developing gonad (i.e. the ovary) becomes devoid of germinal epithelium, which is the precursor for the follicular system Both ovaries may be affected, or sometimes only a single ovary or a part of the ovary may be affected In the Swedish Highland breed af cattle, hypoplasia is determined to have been caused by a single recessive autosomal gene.

The ovaries are small and rudimentary in the form of a thin band with wrinkled rough and irregular surface similar to that of new born calves.

In ovarian hypoplasia gonads consist of predominantly medullary tissue. Tunica albuginea is thick and covered by low cuboidal epithelium. Follicles are completely absent The stroma is dense and made up of thick fibrous tissue with several anovular cords of Type I and Type II, blood vessels and rete tubules.

Polyoogonia is a condition in which each follicle, which normally contains only one ovum, may contain several ova without disturbing the function of genital organs

HEMORRHAGE occurs : 1, during ovulation (of no consequence) 2 while enucleating corpus luteum manually in the cow, 3. while expressing ovarian cysts in the treatment of sterility and 4, in hens affected with avain leucosis complex, pullorum disease and fowl pest.

PERIOOPHORITIS

Usually chronic in nature and is more common than oophoritis. It is more commonly a localised serositis which is seen as red fibrin and serosal tags containing numerous leucocytes attached to the surface of the ovary. Fine bursal adhesions may result but are too delicate to interfere with ovulation.

Serosal granulomas may occur in bovine peritoneal tuberculosis or porcine brucellosis or in setariasis in bovines. These appear as reddish nodules or tags. These infective granulomas remain strictly localised to the surface of the ovary and do not penetrate its substance.

Grossly the ovarian surface is shaggy and often encapsulated with adhesions of bursa

Microscopically, the tunica albuginea is thickened by fibrous tissue with occasional lymphocytic and plasma cell infiltration.In tuberculosis, ovaries may be covered by tuberculous granulomatous tissue with Langhans' type of giant cells In Setariaris, granulomatous foci with sections of larvae along with neutrophils and surrounded by mononuclears, epithelioid cells and fibrous capsule may be seen.

OOPHORITIS : (Inflammation of the ovary) is rare in animals. Sometimes abscesses may be found in or about the capsule. They are usually the result of an ascending infection from the oviduct or uterus.

Peritonitis may involve the ovaries when adhesions may occur. This is especially true in the case of bovine tuberculosis of the peritoneum and brucellosis of swine, when infective granulomata may be found on the surface of the ovaries.

CYSTIC OVARIES

The ovaries contain one or more cysts of varying size. Though met with in all animals, cysts of the ovary are more frequent in the cows, sows and mares.

Cysts are found in high milk yielding cows more frequently and so endocrine disturbance is the main cause. Great enhancement in the milk yield by selection has resulted in more and more cows suffering by this disorder.

Follicular cysts: In this condition, the graffian follicle does not rupture as it should normally and so liquor folliculi accumulates and so the cysts enlarge upto as much as 11 cms. in diameter.

The granulosa cells which are normally of several layers degenerate as atretic bodies. In most of the follicular cysts a single layer of granulosa cells are left appearing as a string of pearls lining the antrum. In some, even the single layer of granulosa cells disappears leaving the membrana propria to line the antrum. The cumulus oophorus and the ovum degenerate. However, the glandular cells of Theca interna continue to be secretory adding to the estrogenic pool.

The incidence of cystic ovary is high in high lactating animals immediately after parturition. Defective feeding and faulty animal husbandry practices may be some of the contributory factors. There may be an involvement of hereditary predisposition.

The cause of the cystic ovaries is considered to be failure of release of LH or failure of release of LH in sufficient quantities to cause ovulation. It may also be due to imbalance of FSH and LH.

Changes have been noticed in the basophil cells of Hypophysis which are believed to elaborate the gonadortophic hormones. The cells assume a bigger and bizarre shape, with large nuclei and large nucleoli. The basophilic granules are at first large and numerous. Later, the cytoplasm becomes clear, homogeneous and later even becoming acidophilic. Side by side with these changes, the acidophils become hypertrophied and densely laden with granules which produce growth hormone and prolactin.

The following extra-ovarian lesions are observed:

1. Relaxation of the sacro-sciatic ligament.
2. Uterus enlarged and edematous
3. Cervix—enlarged with patent os
4. Endometrium—cystic endometrial hyperplasia, "swiss-cheese" type. The hyperplastic glands secrete excess of mucin, producing retention cysts.
5. Vagina and vulva are edematous.
6. In the dog mammary tumors and uterine fibroids
7. Enlargement of the thyroid gland and so hyperthyroidism
8. Increase in the width of the zona fasciculata of the adrenal gland.

Clinically, nymphomania (persistent sexual desire) is observed in cows and bitches.

Lutein cysts: Normally, after ovulation, corpus lutuem forms from the proliferation and luteinization of the cells of theca interna and the follicular

epithelial cells. A small central cavity is present. But in lutein cysts, there is abnormala ccumulation of fluid in this cavity. These are more common in cows and sows than in other animals.

The cause is probably non-release of adequate quantities of luteinising hormone by the anterior pituitary.

Increased production of Progesterone by these cysts renders the uterus susceptible to infection, pyometra being the usual sequel.

The ovary is large, round or oval and soft. The corpus luteum is not discernible on the surface as in the case of normal one. The cyst has a narrow internal lining of yellowish-brown luteal tissue and contents are opalescent, light yellow and gelatinous.

Microscopically, the cyst wall comprises of three layers surrounding the central cavity containing homogneous contents. The inner layer consists of a thin band of loose connective tissue separating the adjacent luteal tissue from cystic contents. The middle layer has varying thickness of lutein tissue. The outer layer consists of concentrically arranged dense bands of connective tissue merging with ovarian stroma.

Intra-follicular luteinisation

Formation of luteal tissue within the follicles even before rupture is called as intrafollicular luteinisation. Usually the ovaries are of medium size.

This condition is noticed only on histopathology. The follicle is located in the centre of ovarian stroma. Tunica theca is thick and lined by several layers of granulosa cells. In the centre there are irregularly arranged lutein cells with large vacuolated cytoplasm and large nucleus having scanty chromatin.

It may probably be due to hormonal disturbance between FSH and LH and requires further investigation.

Embedded corpus luteum :

Small encapsulated yellowish-brown lutein tissue is located in the middle of cortical stroma and associated with endometritis.

The embedded corpus luteum has lutein cells of normal appearance but the fibroblastic proliferation forming irregular masses is conspicuous

The embedded CL seems to be significiant by way of inhibitting the oestrum resulting in anoestrous condition.

Small and Sclerosed ovaries :

This is the commonest abnormality noticed in ovaries of buffaloes. The ovaries are small with smooth surface. Neither follicle nor corpus luteum is apparent on the surface. The cut surface reveals dense stroma with no developing follicles.

Microscopically : The surface epithelium is usually absent. Tunica albuginea is thickened with dense fibrous tissue. The cortex is reduced in thickness. Developing follicles are completely absent and a few atretic follicles may be seen. The stroma is dense with thick fibrous strands running in different directions. Aggregates of thick walled, closely packed capillaries are seen in the stroma.

Different views are put forth for the development of this condition Subactivity or inactivity could be due to hypofunction of thyroid as evidenced by low blood levels of thyrotropic hormone in buffaloes with small ovaries. An imbalance of gonadotrophic hormones is suggested. Nutritional error might have a significant role.

Epoophoron : These consist of intercommunicating, short, closely packed, acinar structures in the loose connective tissue of mesovarian attachment at either poles of ovary. The lumen is narrow and lined by cuboidal to columnar epithelium with large lightly stained nucleus. These have the origin from anterior mesonephric tubules.

Rete ovarii : Consists of tubular net work of anastomosing canals, separated by thick bands of connective tissue at the hilus. These tubules are lined by low cuboidal to columnar epithelium with round or elongated lightly stained nucleus. At times the epithelium may be hyperplastic and assume adenomatous appearance. Rete ovarii is seen prominently in the old ovaries. The origin is from mesonephros.

Anovular cords : These are seen in the ovaries of bovines as scattered or in groups in the ovarian stroma. Anovular cords are probably originated either from groups or nests of epithelial cells which never had oocytes from normal follicles in the early stages of development replacing the follicles.

Three types of anovular cords are recognised.

Type I anovular cords : These are elliptical and surrounded by a thin layer of PAS positive membrane. The cords are filled with 3 to 4 rows of irregularly arranged epithelial cells with no ovum. The nucleus of the cells adjacent to basement membrane is oval with diffuse chromatin. The cytoplasm is stained light and contains a net work of very thin eosinophilic fibrils.

Type II anovular cords : These are elliptical or round and slightly larger than type I cords. The cells are arranged in one or two layers of cells. The lumen contains a moderate amount of PAS positive material.

Type III anovular cords : These are larger than Type II, the diamater reaching 200 microns. There are two layers of epithelial cells with PAS positive amorphous substance in concentric layers in the lumen. The connective tissue around the anovular follicles is arranged in several circular layers and in these epithelioid and eosinophilic cells are often found.

Presence of anovular cords is directly proportional to the severity of hypoplasia of ovaries. In very severe cases Type II and Type III forms are seen. In less severe cases Type II and Type I cords and in early cases of hypoplasia Type I cords are predominantly seen.

Folliculoids : These are seen in ovaries on microscopic examination and are present in one or both the ovaries of aged bovines. Two types of folliculoids are recognised.

1) Trabecular type
2) Colloid type

Trabecular type : These have distinct connective tissue PAS positive capsule with the invaginations of septa into the lumen dividing it into smaller cavities. The septa are lined on either side by single or double layers of granulosa-like

cells. These cells are elongated having large vesicular nucleus and scanty cytoplasm. Several rossette-like structures consisting of eosinophilic irregular bodies surrounded by radially arranged single or double layers of cells are present in the cavity. These structures have a resemblance to Call-Exner bodies, characteristic of Granulosa-cell tumor but the origin and morphogenesis of rossettes is different

Colloid type : A few irregular shaped PAS positive colloid bodies are characteristically seen in the lumina of solitary folliculoids. The cellular elements are few Two types of colloid bodies are seen. One type is large, irregular in size and shape with a laminated appearance. The other type is small and spherical with homogenous structure. Both the types are surrounded by a single layer of granulosa-like cells.

The common association of anovular cords with folliculoids and their close morphological resemblance suggest that the anovular cords might be precursors of the folliculoids. Probably under constant stimulation of gonadotrophin, particularly in aged animals, the anovulatory follicles proliferate to form folliculoids.

Parovarian cysts : These occur frequently in most species of domestic animals in the vicinty of ovary in mesosalpinx and vary in size from a few mm. to 1 cm. or more in diameter. The cysts are lined by a single layer of cuboidal epithelium. The wall of the cyst contains smooth muscle.

Parovarian cysts arise from the remnants of either Mullerian or Wolffian ducts.

TUMORS OF THE OVARY : These are comparatively rare in animals.

Of the primary tumors, cystadenoma and cystadenocarcinoma are the most frequent and met with in the bitch and hen These may be unilateral or bilateral and may be unilocular or multilocular, the cavities containing clear fluid and lined by either cuboidal or low columnar cells. Sometimes cilia may be noticed in these cells. More frequently, there may be papillary projections from the lining, filling the cavity. Usually, such papilliferous tumors are malignant. Peritoneal implantation of the ovarian carcinoma may be observed. Metastasis is by way of lymphatics.

Tumors consisting entirely of the epithelial cells mentioned above are known as solid carcinomata and are seen in fowls and rarely in bitches. Transcoelomic spread occurs in this tumor.

Other tumors, called endocrine tumors of the ovary that may be rarely seen are :- granulosa-cell tumor, theca-cell tumor arrhenoblastoma and dysgerminoma Of these, the granulosa-cell tumor is more often encountered in the cow and bitch.

The following secondary tumors may be found; lymphosarcoma; mammary tumor of the bitch, intestinal carcinoma of the cow

Teratomas and dermoid cysts may be found in the ovaries of animals and birds

AFFECTIONS OF BURSA

Bursitis is generally due to the extension of inflammation from the peritoneum or from the infundibular end of the oviduct. The ascending infection from the oviduct is common in cases of retained placenta and septic metritis. Perimetritis

may also contribute to bursitis. Excessive pressure during enucleation of corpus luteum may cause inflammation of ovarian bursa

Adhesions : Occur as a consequence of bursal inflammation or due to hemorrhage caused by ovulation or enucleation of corpus luteum. The bursal adhesions cause infertility by interference with the transport of gametes The adhesions may be in-between the membranes.

DISEASES OF THE FALLOPIAN TUBES

The disease of the Fallopian tubes (oviducts) are not common in animals. Affections of the oviduct are of importance since the ovum is transported to the uterus via these tubes and any disease of the tubes, therefore, will interfere with pregnancy and reproduction.

Malformations : absence of fallopian tubes or segmental aplasia may be met with. Accessory tubes, reduplication of the tubes are other malformations seen.

Of importance in animals are the following conditions of the oviducts :- hydrosalpinx salpingitis and pyosalpinx. These conditions are more important in the cow and sow.

Hydrosalpinx denotes a cystic dilatation of a part of the oviduct, containing clear fluid This condition arises due to some obstruction in the oviduct. It is usually a result of salpingitis in which, occlusion of the lumen may arise.

Based on gross changes and histopathology two types are recognised :

1) Hydrosalpinx simplex and
2) Hydrosalpinx follicularis or multilocularis.

It may be seen affecting one or both the tubes.

In the simple form, the fallopian tube is considerably distended, elongated and tortuous forming several coils in the mesosalpinx. The wall is thin, translucent and distended with varying amounts of clear fluid. The distension is more often located in the ampulla.

In the follicular form, unlike the simple form, the tube is distended with a little fluid but is hard, tortuous and irregularly beaded. On cross section the lumen presents a multilocular appearance.

Microscopically, the mucosal folds in the simple form are considerably atrophied and lined by low cuboidal to columnar epithelium devoid of cilia. The lamina propria and muscular coat are thin.

In the follicular form, the fibrous septa are usually thin, but in some places, thickening is marked. The trabeculae are lined by low cuboidal or flat epithelium. Infiltration of lymphocytes, a few plasma cells and eosinophils are seen in the lamina propria.

Salpingitis : This is the most common disease of the oviduct and which is usually not diagnosed while the animal is alive. This is of great economic importance since salpingitis is one of the causes of sterility.

The organisms that are incriminated are : *Streptococcus viridans, Staphylococcus aureus,* M, *tuberculosis* and *Brucella suis.*

The organisms may enter the oviduct

1. By way of the blood stream—a generalised infection as in tuberculosis.

2. Through the osteum abdominale—spread of peritonitis (descending)
3 Through the osteum uterinum—extension of endometritis (ascending)

Sometimes irritants may be introduced by uterine insufflation or surgical operations

Macroscopically, there may not be visible changes in the tubes except for slight enlargement and congestion of the mucosa. In the milder forms, there may not be any exudate in the lumen In more severe cases catarrhal or fibrinous exudate may be present, consisting of dead cells and debris

Microscopically, there may be mononuclear infiltration besides congestion of the mucosa desquamation of epithelium and proliferation of stromal elements Plasma cells are particularly abundant. These microscopical changes may be evident even in normal looking (grossly) tubes

The mucosa of the fallopian tubes does not possess much of regenerative capacity and so when once the epithelium is lost it is not restituted.

Sterility occurs in salpingitis for the following reasons :- (1) The ciliated epithelium and contractile muscle necessary for transport of ovum, are destroyed, preventing the movement of the ova to the uterus. (2) The inflammatory exudate is toxic to the spermatozoa, causing their death, (3) Exudate or proliferating cells may occlude the lumen of the tubes. (4) Fibrosis in chronic salpingitis may cause occlusive stenosis.

Pyosalpinx ; This is pus in the salpinx and occurs in suppurative salpingitis, which is usually a sequel to suppurative metritis. Pus accumulates in some segments of the tube due to occlusion of the lumen in certain places by inspissated exudate or inflammatory thickening or by chronic granulation tissue.

The wall of the oviduct is infiltrated by neutrophils, lymphocytes and plasma cells, which are also found in the exudate that collects in the lumen. Metaplasia of the epithelium to squamous variety is common.

Pyosalpinx invariably ends in sterility.

Tuberculosis : Tuberculosis of fallopain tubes is common in cows. Two varieties are seen 1. *Caseous tuberculous salpingitis*, in which the tube is very much thickened and swollen. The mucosa is much thickened and caseated. The lumen contains the disintegrating tissue masses. Adhesions may be present between the ovary and the tube and between the individual coils of the tube.

2. *Nodular tuberculous salpingitis* in which miliary tubercles are found in the mucosa, as a result of generalised tuberculosis.

In birds, salpingitis is a common affection.

Causes : Primary, bacteria : — *Salmonella pullorum*, (descending infection from ovary), *E. coli*.

Parasites : *Prosthogonimus macrorchis*

Secondary : Pullorum disease, vitamin A deficiency.

Macroscopically, the oviduct is swollen and distended. The body cavity contains masses of yolk material and fibrin strewn about, which may cause loops of intestines to become adherent. The mucosa of the oviduct is red and edematous. The cloaca and parts round about are soiled.

Microscopically, catarrhal, fibrinous, hemorrhagic or purulent salpingitis may be encountered. Ascending infection may cause ovaritis or peritonitis.

Neoplasms : The most common neoplasm in the oviduct of fowls is the leiomyoma. It is also frequently met with in the right mesosalpinx

THE UTERUS

Malformations : The following malformations may sometimes be seen :— Aplasia, hypoplasia, duplication of cornu, longitudinal division of the uterus by a septum.

The malformations of the uterus are due to the failure of development of Mullerian duct system. The conditions are bilateral in the case of infantile genitalia and freemartins and unilateral in the case of uterinum unicornis. Sometimes cystic dilatations are noticed in the undeveloped segments.

Rupture of the uterus may occur during parturition due to violent contractions or due to obstetrical manipulation in dystocia. Rupture may involve only the mucosa in which case healing will occur. If the whole wall of the uterus is involved, death may supervene, due to (1) hemorrhage, (2) or inflammation of the uterus spreading to the peritoneum ; or (3) entry of the placenta into the abdominal cavity.

Rupture may occur in prolonged dystocia and torsion due to weakening of the wall Another rare cause may be over distension of the uterus with introduced fluids

Malpositions : Torsion of the uterus is most common in the cow, especially during the terminal stages of pregnancy. Minor twists are self corrected. The condition assumes importance only if the twist is 180° or more. The veins of the broad ligament and ovarian ligaments are compressed while acute hyperemia occurs in the arteries. If the condition is not corrected the dam will die of gangrene, sepsis and peritonitis As the cervix is tightly closed in the twist, parturition cannot take place unless the disorder is corrected. The uterus is liable to rupture easily in this condition as the walls become weakened and friable.

Hernia : Displacement of the uterus, especially uterus in advanced pregnancy, through a ruptured diaphragm into thoracic cavity may occur in dogs and cats (possessing sufficiently long broad ligaments) as a result of automobile accidents

Displacement of the uterus in abdominal and ventral hernias is also met with. In the bitch perineal hernia is also seen. Herniation into the inguinal and femoral canals may occur, when it is called a metrocele if a peritoneal lining of the sac is present.

Prolapse of the uterus through the vulva is most common in the cows but may also be seen in other animals. It may be due to strong uterine contractions for expelling the fetus, the placenta or the exudate. Forced traction during distocia, post-parturient hypocalcemia and retained placenta are the predisposing causes. The sequelae are similar to those found in intussusception viz. acute congestion, hemorrhage, necrosis, infection, gangrene and death. Sows and poultry may injure everted uterus.

CIRCULATORY DISTURBANCES

Physiological hyperemia and edema of the endometrium are found during estrus.

Hemorrhage occurs during estrus, analagous to the menstrual discharge. The source of the blood is capillaries of the endometrium. This is more often found in heifers and bitches. Ecchymoses on the serosa and musculature are normal in heifers during estrus.

Pathological : Acute hyperemia is present in metritis.

Chronic general venous congestion is found with cardiac and pulmonary lesions hindering normal blood flow through these organs.

Hemorrhage can occur during parturition and dystocia. One of the common causes is manual intervention in dystocia when rupture of the uterus with hemorrhage may take place.

Torsion of the uterus as well as prolapse may also be responsible for hemorrhages. Hemorrhage from the arteries of the broad ligament, especially in the sow, is a surgical hazard during cesarean operation, if too much traction is applied on the uterus. In dogs metrorrhagia may be the result of hormonal disturbances. There may be severe lowering of folliculin.

Lastly, massive hemorrhages may be met with in cattle in sweet clover poisoning.

Thrombosis of the uterine vein may be observed in septic metritis, torsion or prolapse. The affected veins are dilated and tortuous and do not collapse at death.

ATROPHY OF UTERUS : Causes;- 1. Senility

2. Oophorectomy after a normal full growth of uterus is attained.

3. Hypopituitarism : (a) due to wasting disease or (b) primary lesion of the pituitary.

HYPOPLASIA may be observed in oophorectomised young animals, the uteri of which have not attained full size.

HYPERPLASIA : Hyperplasia of the endometrium is observed in all species of animals but is more often met with in the dog. It is also known as *cystic hyperplasia* of the endometrium.

The cause appears to be increased estrogen and or progesterone secretion, under the influence of which the endometrium undergoes hyperplasia.

This condition has been noticed in animals having granulosa-cell tumor, papillary cystadenoma and persistent corpora lutea and ovarian follicular cysts.

Feeding on pasture legumes—lucerne and clover—containing substances having estrogen activity has been found to cause hyperplasia of the endometrium in the ewe and cow.

Though a variety of organisms, including *E. coli*, *Staphylococci* and *Streptococci*, have been isolated from the affected uteri, they must be considered as secondary invaders of the already existing hyperplastic endometrium.

Macroscopically, endometrium of both the horns contains cysts of different sizes some microscopic and some others as big as 4 or 5 mm in diameter. The cysts may completely fill the lumen giving it a "swiss-cheese" appearance. They

contain clear fluid. The lumen of the horns may also contain, in some cases, mucus or pus (if infection by pyogenic organisms has occurred) which may flow out of the vulva.

Microscopically, no inflammatory changes may be present in the uncomplicated cases but only cyst formation of the glands which are increased in number and irregularly distributed (unlike the normal orderly arrangement). These cysts contain a single layer of epithelium enclosing clear watery fluid. In some animals there may be plasma cell infiltration of the lamina propria. The endometrium may show thickening due to polypoid proliferation. In those animals in which infection has taken place, there will be pus in cystic glands and neutrophilic infiltration of the lamina propria. In this type, the uterine horns show alternating constrictions and dilatations which appearance mimics pregnant uterus

Clinical features: 1. Abnormal uterine bleeding.

2 Disturbances in estrus cycle: (a) irregularity (b) longer or short duration (c) diminished or enhanced characteristics of the different phases. 3. Sterility; 4. Aborton 5. Long or pregnant lactation. 6. Development of secondary infection, characterised by high leucocytic count with a shift to the left.

INFLAMMATION OF THE UTERUS

Metritis is inflammation of the uterus and is found in all animals. If the inflammation is restricted to the endometrium alone the condition is known as *endometritis.* On the other hand, if the whole thickness of the wall is involved *metritis* is the term used. Inflammation of the serosa is known as *perimetritis.*

ENDOMETRITIS

Causes 1. Infection

(a) *Trichomonas fetus, Vibrio fetus, Brucella,* weaker strains of pyogenic *cocci* and *coliforms* Infection may occur during coitus or during artificial insemination or during manual handling of the uterus for therapeutic purposes.

(b) A severe metritis might have subsided leaving a low grade inflammation of the endometrium.

2 **Irritants :** Introduction of too hot fluids or too irritating chemicals into the uterus, thereby injuring the delicate mucosa.

Macroscopically, no gross lesions are evident. An increased secretion of tenacious mucus may all that may be visible. The mucosa may be swollen, red and rough instead of having a smooth surface and covered with fragments of necrotic material.

Microscopically, in the mild catarrhal variety there may be slight but diffuse infiltration by lymphocytes, plasma cells and macrophages. The blood vessels are engorged.

More severe forms invariably involve all the layers of the wall and must be considered as metritis

Clinical consideration: Though mild in appearance, endometritis must be attended to promptly or else conception may not occur. The inflammatory exudate being toxic is lethal to the ovum whether fertilised or not. Besides, the condition may progress to the chronic stage when permanent sterility may supervene.

Causes. Metritis is caused by bacteria, infection being facilitated by the following, which may cause an initial injury: parturition, dystocia, mechanical injuries by the obstetrical instruments, projecting fetal bones after embryotomy, excessively warm irrigating fluids, chemical antiseptics and disinfectants introduced into the uterus.

The organisms that invade the injured uterus are: *Corynebacterium pyogenes, Streptococci, Staphyylococci, E. coli, Sphenophorus, Clostridium sp.*

Tuberculosis and other chronic granulomatous infections may also invade the uterus.

The parturient uterus, with its lochia is a good medium for the growth of bacteria. Retained placenta and albuminous exudate are also ideal for the propagation of microorganisms and so infection is common in those animals in which lochia is plentiful and involution is delayed. The latter condition may result due to weakened and injured uterine musculature in prolonged dystocia.

Nutritional deficiencies and disturbances and hormonal and endocrine disturbances are considered as predisposing factors by preparing the soil for infection.

Routes of infection

1. **Ascending infection** from the vagina. This is the most important route.
2. **Descending infection** from the abdominal cavity through the fallopian tubes.
3. **Through lymphatics** from the peritoneum.
4. **By way of blood :** this is important in tuberculosis.

Depending upon the virulence and nature of the organisms, metritis may be acute catarrhal, acute suppurative and chronic suppurative.

Acute Catarrhal Metritis : This is acute endometritis and has been described above. This condition is difficult to be differentiated from an uterus during estrous cycle.

Chronic non-purulent endometritis can be met with in which there is heavy infiltration of leucoytes, mostly plasma cells, in the mucosa, which, therefore becomes thickened. This thickening is not uniform because of the fixed position of the outlet ducts of the gland and so assumes a polypoid appearance. This is known as *Chronic Polypoid Endometritis*.

Because of the proliferation and the subsequent contraction of the superficial connective tissue, the mouth of the glands may be closed rendering them cystic. This condition is known as *Chronic Cystic Endometritis.*

If the causes of the endometritis is removed, the leucocytes are replaced by fibrous tissue which on contraction produces atrophy of the glands and the mucosa and so *Chronic atrophic endometritis* results.

In cows the mucosa in chronic endometritis may undergo degeneration and subsequently be calcified and the condition then is known as *endometritis calcificans*

Acute suppurative metritis : This condition which usually arises from infection by pyogenic organisms is a frequent complication of dystocia, retained placenta or abortion.

Microscopically, the mucosa of the uterus is very much reddened, thickened, rough and is covered by a purulent, often reddish exudate. This may frequently contain shreds of disintegrated fetal membranes. The uterine wall is thickened and friable. The muscle fibres atrophy and disappear or show Zenker's degeneration. Subserosa is edematous and infiltrated with leucocytes. Serous coat also shows inflammatory changes

Microscopically, there is infiltration of the endometrium by large number of neutrophils. After several days, macrophages, lymphocytes and plasma cells infiltrate the endometrial stroma.

Infection extending into the uterine veins results in thrombosis of those vessels.

Chronic Suppurative metritis Pyometra: Pyometra literally means pus in the uterus. But usually this term is applied to chronic suppurative metritis. This condition is seen in dogs, cats, cows and swine.

In cattle, pyometra is encountered as a result of retention of placenta. The placenta putrifies since it is a very good medium for bacteria to thrive. Incomplete involution may be an associate factor.

The pathogenesis in the dog is different. In this animal, increased progesterone activity seems to be the prime cause.

Pyometra arises due to infection of the hyperplastic endometrium met with in "Pseudopregnancy" of bitches, which is caused by persistent corpus luteum releasing large amount of progesterone.

Corynebacterium pyogenes, E coli, Proteus sp. and Staphylococci have been isolated from the uterine exudate.

In the cow *Trichomonas fetus* infection is a common cause.

Macroscopically, in the cow, thin cream-like pus may be discharged through the vulva, soiling the tail and the perineum When the animal lies down, due to pressure on the abdomen, pus which may stagnate due to gravity during a standing position, may flow out. The uterus is dilated, and involution may not be complete.

In the bitch, the exudate is always retained as the cervix is completely closed. The horns are dilated and thin-walled and contain chocolate-colored fluid. The abdomen is enlarged as in a full pregnancy. The serosal surface may show congested vessels and evidence of inflammation. The mucosa may be thickened irregularly and in some places it may be ulcerated and hemorrhagic and covered with necrotic shreds of membarane appearing as though bran is sprinkled. Retention cysts may sometimes be seen.

Microscopically, in the cow the appearances are similar to those of endometritis. viz, congestion of the blood vessels, infiltration by inflammatory cells especially neutrophils and lymphocytes and plasma cells. These cells accumulate under the epithelium, leading to its purulent softening and separation of the necrotic area of tissue and these appear as bran-like material.

The condition in the dog is more acute and so greater infiltration of neutrophils and lymphocytes occurs. There is hyperplasia of the endometrium, producing pseudostratification or papillary proliferation. Sometimes squamous metaplasia may be noticed.

Extragenital lesions : These are found in the dog and cat and may be due to the 'toxic' effect on other organs as well as to periodical bacteremia that may occur. The lesions are : 1. Anemia due to depression of the bone marrow.

2. Extramedullary leucopoiesis, especially in liver, spleen, kidneys, adrenals, lungs and lymphnodes.
3. Lesions of the kidney—glomerulonephritis, tubular degeneration, hemorrhages in the medulla, infarcts, pyelonephritis.
4. Congestion and degenerative changes in the liver.
5. Adrenals—necrosis of the cortex and hemorrhages in the medulla.
6. "Sinus Catarrh" of the lymph nodes
7. Intense leucocytosis. The total white cell counts vary from 30,000 to 160,000 per c.mm. An extreme shift to the left and toxic granulation of neutrophils are found.

Sclerotic metritis is characterised by complete destruction of endometrium as a result of severe chronic endometritis. A thick dense connective tissue layer replaces the endometrium. The foci of infection in the connective tissue layers is responsible for purulent exudate in the uterine cavity. The uterine caruncles and endometrium are destoryed resulting in permanent sterility. The uterus on rectal examination appears hard and firm and the cervix thickened. Cow is usually anestrus and the corpus luteum is found deeply embedded.

Perimetritis and Parametritis are characterised by varying amounts of adhesions between uterus and broad ligaments, with other pelvic and abdominal organs. The adhesions are reusltant of severe metritis, douching with strong irritant solutions, perforation of rectum with leakage of its contents, torsion of uterus vaginal and cervical lacerations curing difficult birth, excessive bleeding, following enucleation of corpus luteum or vigonrous massage of infected uterus. The condition may also be due to peritonitis or tuberculosis of the genital organs.

In women uterus may be infected with organisms producing gas gangrene while abortion is induced. The muscles and peritoneal coat are involved with inflammatory changes and gas formation. This condition is known as **Physometra.** Among animals a similar condition may be met with in infection of the uterus by the bacillus of Black Quarter.

Necrobacillary metritis that may occur in cows due to puerperal infection by *Spherophorus necrophorus* is always fatal.

Tuberculosis of Uterus : Infection may be descending from tubercular peritonitis or it may be hematogenous as occurs in generalised tuberculosis. Two forms are seen : *(1) Disseminated miliary tuberculosis* in which the tubercles are found uniformly in the mucosa. *(2) Diffuse caseating endometritis* in which the body or the cornua are diffusely thickened. The lumen contains large quantity of serous or purulent exudate containing large caseating masses. The mucosa is very much thickened and caseated.

Actinomycosis : Incidence of actiuomycosis is not common. Large swellings with extensive pelvic adhesions are produced. Prognosis is generally poor.

Brucellosis of the uterus of pigs : Some aged pigs reveal pea sized, miliary yellowish-white nodules in the uterine mucosa, caused by *Br. suis* These nodules may be single or may occur in groups. The mucosa which may be raised

may sometimes be ulcerated. In the centre, the nodules contain a little pus. Histologically the nodules are granulomas. Animals having these lesions give positive test for brucellosis.

Mucometra/Hydrometra

The two conditions are considered together as the difference is probably only in physical properties and depends on the degree of hydration of mucin, which in turn may be related to the relative activity of estrogenic hormone.

The accumulation of thin or viscid fluid in the uterus is concurrent with the development of endometrial hyperplasia or is proximal to an obstruction of the lumen of the uterus, cervix or vagina. In the first instance the amount of the fluid may be several litres and the greater the volume of the fluid the less viscous it is. Small amounts of mucin gives the mucosal surface a gummy stickiness. In cows with cystic ovaries, the large volumes of fluid is usually associated with functional cysts of the follicles.

In the second instance, that of obstruction to the lumen, the volume of fluid depends on the site of obstruction. The fluid is slightly cloudy and watery.

Animals with mucometra are sterile. If affected uterus becomes infected, an intractable pyometra results.

An abnormally long and tortuous cervix may result in a form of mucometra caused by the retention of uterine secretion.

Microscopically : The endometrium is thin and lined by a single layer of cuboidal to low columnar cells. The uterine glands are reduced in number. The endometrial stroma is edematous.

Uterine abscesses are usually man made lesions. These are commonly present on the dorsal wall of the uterus at the anterior extremity of the body and produced either by an insemination pipette or some instrument used for uterine medication.

These are of variable sizes containing thick yellow pus.

Endometriosis : Means presence of endometrial glands and stroma in places other than endometrium. If these are seen between the muscle bundles of myometrium, the condition is called **endometriosis interna** or **adenomyosis**. On the other hand, if these are seen in places other than uterus such as mesosalpinx, ovary, cervix or intestinal serosa, it is called **endometriosis externa**.

Endometriosis is occasionally noticed in bitches and bovines. The endometrial glands show changes in response to ovarian activity and often there are hemorrhages. In women these appear as chocolate coloured cysts. Histogenesis of endometriosis is still controversial.

Mummification of fetus :

Mummification of a dead fetus is seen occasionally in any, but usually in multiparous species and most commonly in the cow. In the mare it is typically one of twin fetuses which is mummified.

A prerequisite for mummification is absence of infection unlike in maceration The fluids are reabsorbed and the membranes become closely applied to the desiccated fetus. The whole fetus becomes brown or black and rather leathery, moist on the surface with sticky mucus without odour.

The time required for complete mummification depends on the size of the fetus but probably requires as long as 6 to 8 months. In uniparous animals, the mummified fetus is usually retained indefinitely or if aborted may only be delivered into vagina. In the case of multiparous animals, it may be delivered along with viable fetuses. Animals which had and recovered from mummified fetus usually breed normally on subsequent occasions

In bovines, haemic mummification is seen while in mare and sows it is or paperaceous type.

Maceration of fetus : Depends on the presence of infection in the uterus. If the early embryo succumbs to uterine or embryonic infection maceration is usually followed by resorption from the uterus or expulsion along with a small amount of purulent discharge. If the fetus is about three months, complete foetal maceration does not occur and bones resist maceration. These may be discharged or be retained in the pus of pyometra indefinitely, often near the cervix.

Advanced uterine lesions accompany the macerated foetus. The uterine wall is thickened and the reaction within it varies from the acute exudative inflammation of pyometra to more or less complete sclerosis and replacement by granulation tissue in long standing cases.

ABORTION

Expulsion of a dead fetus prior to the normal full gestation period is called abortion. Abortion is mostly due to infection of the fetus, placenta or the uterus since these conditions cause death of the fetus. A dead fetus is a foreign body and so is expelled from the uterus .

The following are the causes :

1. Specific infections :

A. Brucellosis : *Brucella abortus, melitensis* and *suis* affect the cow, sheep goat and pig. *Brucella ovis*, a new species, affects only the sheep These strains can be differentiated by means of biochemical methods.

Bovine brucellosis : The typical abortion occurs at about the 7th month of gestation in the cow The organism has special affinity for the pregnant endothelium A few weeks after abortion or parturition, the organism can no longer be detected in the uterus.

Routes of infection :

1. Alimentary canal—ingestion of feed or water contaminated by fetal membranes, fetus or uterine discharge.
2. Vagina—coitus, artificial insemination.
3. Conjunctiva.
4. Skin.
5. Contamination of healthy udder from an infected one during milking.

The organism produces abortion in the following manner :

1. First placentitis is produced by the invading organism.
2. Sero-purulent exude accumulates between the endometrium and chorion

3. Edema and infiltration of the chorion by macrophages, lymphocytes and plasma cells
4. Necrosis and hyalinisation of chorio-allantois.
5. Thus the membranes become separated from the uterine endometrium.
6. Fetus dies.
7. Severence of blood supply to fetus.
8. Dead fetus is a foreign body and so expelled—abortion.

In milder cases, a live fetus may be born, which is usually weak and may succumb soon after. In the chronic cases, there is fibrotic adhesion of the placenta to the endometrium, resulting in retention of placenta. In these cases, calves may be born alive.

In the aborted fetus may be found croupous or catarrhal pneumonia; edema of the pericardium, umbilical cord and skin and serosanguineous exudate in the serous cavities. Suppurative or hemorrhagic gastro-enteritis is present. Hyperplasia of lymph nodes and spleen are prominent lesions.

Brucellosis of swine has a course similar to the bovine but is more acute and severe, abortions occurring between the 2nd and 3rd months of pregnancy. Coital infection is more frequent.

Brucellosis in dogs. Brucella canis causes abortion in dogs. This is a highly infectious disease and transmission may be by contact, through infective discharges and also by venereal transfer. In male dogs epididymitis, testicular atrophy and complete sterility may be caused

No fever is noticed. Abortion occurs between the 7th and 9th weeks of gestation.

Ovine Brucellosis (Br ovis); Infection is probably by ingestion.

In rams after an initial bacteremia and mild systemic reaction, the organisms localise in the epididymis causing sterility. Semen is of poor quality and contains leucocytes and brucellae. There may be acute inflammation of the scrotum with edema. The condition may become chronic with enlargement of epididymis, thickening of the scrotum and atrophy of testes.

In ewes abortion may occur in late pregnancy or still births may also result due to placentitis There is purulent exudate on the placenta and edema of allantois. There may be elevated, firm, yellowish-white plaques in the intercotyledonary areas and the cotyledons are enlarged and edematous.

Brucella melitensis abortion in goats : Infection is by ingestion. Abortion may occur, but sometimes live kids may be born. Viable kids are infected and infection persists in a latent form and at maturity clinical symptoms are manifested. In the goats an acute systemic reaction develops and later localisation of the organisms in the placenta causes placentitis and thus abortion results. After abortion the uterine infection persists for over 5 months and the mammary glands remain infected for many years. In some cases, spontaneous recovery may occur.

B. Vibriosis : Vibrio abortion occurs in cows and sheep and infection is by ingestion in sheep and coitus or artifical insemination in cows.

Vibrio causes acute catarrhal endometritis, cervicitis and vaginitis.

The pathogenesis of abortion is similar to that of brucellosis, the initial lesion being a placentitis followed by exudation, necrosis, vasculitis, separation of the placenta from the endometrium, death of the fetus and abortion.

In the cow abortion occurs between the 5th & 7th months of pregnancy while in the sheep at 2 months. Usually placenta is not retained.

In the fetus may be found edema of the subcutis, serofibrinous pleurisy, peritonitis, pericarditis, fatty degeneration of the liver and kidney and hemorrhages in the renal cortex Infection causes repeat breeding.

C. Trichomoniasis :

Trichomonas fetus is transmitted to the cow through coitus, the bull harbouring the flagellate in the mucous membrane of the penis, terminal portion of urethra and prepuce.

In the cow, within three days after infection, vulvitis and vaginitis develop, from where infection spreads producing cervicitis and endometritis and placentitis. There is copious greyish-white thin exudate and abortion will occur within about 16 weeks of pregnancy. Sometimes the dead fetus may be macerated in the exudate. Or the fluid may be absorbed (if not infected by other bacteria) and the fetus may be *mummified* If pyogenic organisms invade the uterus *(Corynebacterium, Staphylococci* and *Streptococci,)* pyometra will result. In chronic infection with fibrosis, placenta may be retained. Infection causes repeat breeding.

D. Listeriosis.

Listeria monocytogenes, which primarily affects the brain, may sometimes infect the pregnant uterus and cause abortion in cattle and sheep, The organisms become septicemic in the fetus and cause its death. Abortions usually occur during the last trimester of pregnancy.

The fetus shows hemorrhage in the kidneys; anasarca; areas of necrosis and granulomas in the liver, spleen, lungs and kidneys; catarrhal gastro-enteritis, cardiac vegetations and hemopericard.

E. Epizootic bovine abortion :

A virus of the family psittacosis-lymphogranuloma group is found to cause abortion in cows and ewes (In sheep it is called enzootic abortion).

An arthropod vector is probably important in transmission Animals rarely abort more than once, due probably to development of immunity. In an outbreak 75% of affected may abort.

Abortion occurs during the last trimester.

The virus causes death of the fetus. The characteristic lesion found in all the organs is a focal inflammation consisting of neutrophils, lymphocytes and macrophages. Injuring the vascular endothelium, the virus is responsible for petechiae found on the skin and internal organs.

Macroscopically, the fetus shows subcutaneous edema, hemorrhages on the conjunctiva, on the mucosa of ventral surface of the tongue and on the tracheal mucosa. Skin at the groin shows erythematous patches. Body cavities are filled with serosanguineous fluid. The pathognomonic lesions are found in the liver, which is enlarged, friable, pale red to reddish-orange in color and has a coarsely granular surface (due to chronic venous congestion). Placenta is not retained.

Microscopically, the liver may either show changes consequent on chronic venous congestion (dilatation of the central vein and sinusoids and consequent pressure on the hepatic cells causing necrosis) or granulomatous lesions in the hepatic capsule, in the portal triads or in the adventitia of the central veins. Meningitis, focal encephalitis and mild degenerative changes in the kidney, pancreas and lung may be noticed. There is hyperplasia of the reticulo-endothelial tissue of the spleen, thymus and lymph nodes and so these organs are enlarged. Infiltration of the adventitia of meningeal and parenchyma cells of the brain by pleomorphic mononuclear cells, arranged concentrically is a characteristic appearance.

The granulomas wherever they are seen (liver, kidney, spleen, lymph node) consist of central necrotic areas surrounded by neutrophils, epithelioid cells and lymphocytes, surrounded by fibrous tissue.

Diagnosis : Is difficult because of inconstancy of lesions. It is difficult to isolate the virus. Serology is not useful. Only symptoms must guide one to arrive at a diagnosis.

F. Leptospirosis in cattle

In cattle various strains of Leptospira produce abortion, after 6th month of pregnancy. The placenta is avascular with collapsed blood vessels. Cotyledons are atonic, yellow-brown in color and leathery. No inflammatory infiltration is noticed.

In the fetus are found edema of the subcutis, peritoneum, umbilical cord pericardium ; focal interstitial nephritis with round-cell infiltration, glomerulonephritis, infiltration of eosinophils into the cortex and round-cell infiltration into the periportal tissue of the liver.

Abortion is due to fetal death.

G. Abortion in mares by Salmonella abortus equi :

The organisms produce a purulent hemorrhagic placentitis. Allanto-chorion is edematous and exhibits hemorrhages and necrosed areas with a wall of hemorrhagic reaction separating it from the surrounding tissue. Abortions occur late in pregnancy. Infection is followed by development of immunity.

H. Equine viral abortion

Two viruses of horses are incriminated : (1) The virus of equine rhinopneumonitis (influenza). The fetus shows edema of the subcutis, jaundice and edema of the lungs. The lungs are heavy and voluminous. Liver shows focal necrotic areas. Visible under the capsule are grayish-white foci. Such necrotic areas are seen in the spleen and lungs also. Petechiae are found throughout the body. Abortion occurs in the 9th or 10th month. Acidophilic nuclear inclusions are found in the bronchial and alveolar epithelial cells.

(2) The virus of equine arteritis brings about the death of the fetus. The fetus shows hemorrhages in the splenic capsule and respiratory mucosa.

I. Abortion caused by the virus of Infectious Bovine Rhinotracheitis

Abortion in cows may be caused by the IBR virus when the animal suffers from respiratory affection caused by this virus. Vaccination of cows with the IBR virus vaccine also brings on abortion.

Symptoms: Abortion occurs during the last trimester of pregnancy. No symptoms are noticed prior to abortion. There may be history of vaccination by IBR vaccine or of a respiratory affection. Fetus is usually decomposed when aborted since abortion occurs only 24 to 36 hours after its death. Animals which abort do not become sterile.

Macroscopically, fetus shows petechiae on the heart. Serous cavities contain serosanguineous fluid There is edema of lungs and placenta.

Microscopically, there is focal necrotising hepatitis and placentitis. Renal cortex shows hemorrhagic necrosis. No inclusion bodies are seen.

Diagnosis : 1. By symptoms.
2. Affected animals are serologically positive.
3. Virus isolation from cotyledons.

J. Mycotic abortion : Cattle and sheep.

Abortion in cattle may occur due to infection by fungi of the following species : *Aspergillus, Absidia, Mucor* and *Rhizopus* The infection is a secondary one, the primary lesions being in the lungs, abomasum (ulcers) and the intestines. Infection is by the blood stream.

Abortion in affected cows occurs during the later half of the gestation period between 6th and 8th months and placenta is retained.

Macroscopically, the fetus may show only circumscribed grayish plaques on the skin resembling ring worm lesions. Internal organs are free

In the cow, lesions are present in the placenta The Chorion-allantois is thick and leathery. Infection occurs first in the placentomes which show necrotic plaques and the fungus can be demonstrated in these locations.

Microscopically, the typical lesion consists of focal collection of inflammatory cells macrophages predominating. Extensive necrosis of the placentomes occurs. In the uterine wall, the intercaruncular areas show red patches covered in places by a thin yellowish-grey pseudomembrane Thrombosis and perivascular necrosis occur in these places and hyphae are found both in the tissues and over the mucous surface Some degeneration of circular muscle is noticed and small arteries are hyalinised throughout the uterine wall. Hyperemia and hemorrhages are common in the affected area. Separation of the placenta from the cotyledons causes death of the fetus.

2. Abortion may occur in infections by various organisms which first produce metritis, followed by placentitis and abortion or birth of weak fetus. The following are noteworthy :

Cattle : *Salmonella sp; Corynebacterium pyogenes; Streptococci; Staphylococci; M. tuberculosis; Actinobacilus; Pasteurella*

Mares : *Streptococcus zooepidemicus; Klebsiella genitalium; Shigella equirulis; E. coli.*

Ewes : Virus of Ovine Abortion; *Salmonella abortus ovis.*

3. Poisoning on administration of Ergot: Ergot being an ecbolic produces violent contractions of the uterine muscle resulting in abortion.

4. Neutralisation of the effect of progesterone by estrogens: Progesterone maintains pregnancy while estrogen terminates it by inducing uterine contraction.

5. **Poisons :** (a) Chlorinated naphthalenes (which are anti-Vitamin A and so may produce metaplasia of the uterine epithelium, infection and separation of the placenta); (b) Purgatives; (c) nitrates through ingestion of plants containing large quantities of this chemical,

6. **Faulty nutrition of the mother:** Deficiencies of minerals and vitamins.

7. **Vaccination of mother during pregnancy against bacterial and viral disesses.**

8 **Severe and acute septicemic diseases of the mother :** Leptospirosis, dourine, viral diarrhoea, hog cholera, erysipelas, infectious rhino-tracheitis In these diseases. abortion may frequently occur.

9. **Hereditary predisposition.**

10. **Torsion of the umbilical cord** (rare).

11. **Traumatic injury to the placenta** (very rare).

NEOPLASMS of the uterus : In the domestic animals neoplasms are neither common nor important as in man.

The most common uterine tumor is the lymphosarcoma, a local manifestation of a generalised condition. In the uterus the neoplastic cells may aggregate as nodules or may diffusely infiltrate the organ.

Adenocarcinomata of the uterus are encountered with metastases in the lungs and liver

In the bitches, uterine fibroids (leiomyomas) are most frequent.

CERVIX

Malformations

These occur more frequently in the cervix than in other parts of the reproductive tract.

Varying degrees of persistence of the median wall of the Mullerian ducts which are destined to develop into cervix result in the formation of a complete or partial duplication of cervix.

Incomplete double cervix occurs much more frequently than a complete duplication and usually involves the portion of the cervix adjacent to vagina. In the case of both incomplete or complete double cervix, if the insemination is done pregnancy may occur but it may result in dystocia.

Absence of external os may be commonly encountered. In this case, the expulsion of uterine secretions cannot occur resulting in accumulation of fluids in uterine horns—hydrometra.

Double external os : Presence of a dorso-ventral band adjacent to external cervical os giving an impression as though two cervical openings are there. It may not interfere with conception or pregnancy but may cause dystocia occasionally. The fetal membranes may be caught on this dorso-ventral band This condition is inherited and conditioned by a single recessive gene with low penetrance.

Hypoplasia of cervix : The cervix may be very small and there may be deficiency in number of cervical rings. Such a cervix is usually defective in protecting the uterus against bacterial invasion from vagina.

Tortuosity of cervical canal : Extreme degrees of tortuosity of the cervical canal may be a cause of inferility in heifers. There may be S-shaped kink and insemination pipette cannot be inserted into cervix.

ABORTION DISEASES OF CATTLE : CAUSES AND DIAGNOSIS

Disease	Clinical features	Abortion		Lesions		Laboratory diagnosis	
		Rate	Time	Placenta	Fetus	Isolation of etiological agent	Serological
Brucellosis (*Br. abortus*) reported in India.	Abortion and repeat breeding	High, upto 90% in susceptible cattle	Late abortion, after 6 months	Placentitis, necrosis of cotyledons	Pneumonic lesions, fetal diarrhoea may be present	Culture of material in 10% Co_2 tension. Chromogenic on potato medium,	A) *Individual tests* 1) Blood plate agglutination with colored antigen. 2) Serum agglutination (tube agglutination 1 in 40 positive). 3) Vaginal mucus agglutination test. 4) Semen agglutination in bull. B) *Herd tests* 1) Milk ring test 2) Whole milk and whey plate agglutination test.
Trichomoniasis (*Tr. foetus*) Reported in West Bengal. Uttar Pradesh Madya Pradesh	Repeat breeding due to embryonic death. Post-coital pyometra in 10% of the cows	Moderate 5 to 30%	Early. within 2-4 months	Placenta is edematous Uterine discharge with flocculent material, clear and serous.	Fetus slighily maccerated.	Cultural examination of fetal stomach. uterine exudate and preputial washings	Serological test with vaginal mucus not popular.
Vibriosis (*V. foetus*) No confirmed reports in India.	1. Repeat breeding 2. Prolonged and irregular estrus cycles	Low, 5% to 20%	5—6 months	Placentitis, semi-opaque, small thickenings; patechiae, localised avascularity and edema	Flakes of pus on visceral peritoneum	2, Demonstration of the organisms in the above. Culturing of organisms under 10% Co_2 tension. Pathogenic strains are catalase positive.	Cervical mucous agglutination tests (1/50 positive) Mucus to be collected after 40 days of suspected service
Leptospirosis (*Lep. pomona*) Other strains are incriminated in A.P.	Repeat breeding due to endometritis; Abortion may occur during febrile condition; later due to fetal death	25—30%	After 6 months	Avascular placenta, atonic, yellow brown cotyledons. Brown gelatinous edema between allantois and amnion.	Fetal death common	Culturing fetal stomach, placenta and uterine exudate	Serum agglutination and microagglutination lysis test. against all leptospiral antigens. (21 days after febrile condition). Positive cases in brucellosis- free animals reported in A.P.

ABORTION DISEASES OF CATTLE : CAUSES AND DIAGNOSIS

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Mycosis—*Mucor, aspergillus* and *absidia* species.	Abortion	6-7% unknow abortions	3-7 months	Necrosis of maternal cotyledons. Adherence of necrotic material to chorion causes—soft yellow cushion-like structure, small yellow raised leathery lesions on inter-cotyledonary areas.	Small raised grey but soft lesions or diffuse white areas on skin resembling worm lesions.	Direct examination of cotyledon, fetal stomach contents for presence of hyphae; cultural examination can be undertaken.	Gel diffusion test using the srum from aborted cases and fungal mycelium as antigen

Cervical dilatation and diverticula; Dilatation and diverticula usually occur in heifers at the level of third and fouth cervical rings. The cervical canal is usually very small anterior to the defect so that it may be difficult to insert insemination pipette. With age tenacious mucus tends to accumulate in the area of the defects.

Prolapse of cervical rings: is a condition which usually develops with age following repeated parturition. Lacerations and hemorrhages which occur during parturition, results in the formation of excess fibrous stromal tissue, enlargement of cervical rings, vascular embarrassment and occasionally squamous metaplasia of the affected rings. The first and sometimes the second cervical rings prolapse into the vagina.

Cervicitis is the inflammation of the cervix and normally follows abnormal parturition such as abortion, premature birth, dystocia, retained placenta, post partum metritis, pneumovagina and vaginitis The organisms responsible for cervicitis are the same as those of metritis.

Cervicitis, always occurs whenever metritis or vaginitis is present, since cervix is located between these two. Causes include :

1. **Mechanical injuries :** (a) during parturition,
   (b) copulation and
   (c) phooka—criminal stimulation of the vagina or os for higher milk yield.
2. Diseases of uterus and vagina.

Cysts ; Retention cysts of the cervix are seen in cows. These are usually small. Bigger ones may partially occlude the cervical canal.

Neoplasms Squamous cell carcinoma may be encountered.

VAGINA

Developmental abnormalities :

Double vagina due to persistent median septum along the vaginal passage.

Median vertical bands connecting the floor with the roof at the hymenal border. This condition is more common.

Heterotopic vulval opening may be located in the inguinal region behind the udder instead of below the anus in the perineum

Cysts : Dilatation of the Gartner's canals (which are remnants of Wolffian ducts) may be noticed in the cow poisoned with highly chlorinated naphthalenes and in those having ovarian follicular cysts. Multiple cysts are located on the floor of the vagina as parallel rows. These contain a thin clear fluid and are lined by simple cylindrical epithelium.

Rupture of vagina may occur during parturition or during coitus (especially in sows). Infection of the rupture may result in abscess, phlegmon, gangrene and peritonitis.

Vaginitis and Vulvitis

Causes include : Physical—Trauma; Chemical; Nutritional deficiencies. Bacterial and Viral agents that are the same as for metritis and abortion

Appearances are similar to those of inflammations of other mucous membranes.

Granular vaginitis is otherwise called *nodular venereal disease.* This dis is described as one of the causes of infertility and is believed not to c abortion.

The causative agent is not clearly established, Many organisms have b listed : hemophilus, pleomorphic rods and viruses. The incidence of the di is highest in naturally served herds.

Raised orange-red areas about 3 mm. in diameter are noticed in the po rior part of the vagina upto the urinary meatus. The lesions are most comm seen in the region of the clitoris, below the lips of the vulva. Sometimes les extend up to the dorsal commissure. The raised areas or granules are isolat and in severe cases may coalesce. The granules are lymphoid follicles or ly phoid accumulations. The epithelium over the granules is easily injured bleeding may occur.

"Vesicular venereal disease", "vesicular vaginitis", "Coital exanthe The cause is supposed to be a virus, which is considered to be the same ca infectious rhinotracheitis and infectious keratoconjunctivitis in cattle, an epith liotropic virus.

This is a highly contagious disease, frequently transmitted by coitus. T course of the disease, is about 10 days. Recovery is the rule with tran immunity. **Incubation period is 1 to 3 days**

Macroscopically, pustular lesions may be found only in the vagina and vul Early fever and leukopenia may be seen during viremic phase. Starting w hyperemia of the mucosa of this part hemorrhages may be observed later in t submucosal lymphoid follicles. The mucosa is covered with thick muc Within 24 hours, there is mucopurulent discharge from the vagina. There m be pustules over the lymphoid follicles. Rupture of these lesions results in ul ration. Extension of infection to cervix and uterus results in cervicitis metritis.

Microscopically, the epithelial cells undergo hydropic degeneration acidophilic intranuclear inclusion bodies are found in them, since the virus epitheliotropic. Neutrophilic infiltration is present near these lesions. In th lamina propria, infiltration by lymphocytes and plasma cells may be found to ther with edema and hyperemia

Resolution occurs in about 8 days.

Specific bovine venereal epididymitis and vaginitis (Epivag) is a chr viral disease of cattle, transmitted by coitus and is found in Africa. The disea characterised by mucopurulent vaginal discharges in females and causing per nent adhesions of fallopian tubes. In the bulls the disease causes swelling epididymis.

Pneumovagina is common in mares and is due to the deformities or injur to the vulva and its suspensary apparatus It is also due to the vice of cri biting In this condition, the mare makes an inspiratory effort holding so thing hard in its mouth. Because of this effort, and because of the already e ting negative pressure of the uterus, and due to the inability of the vulva keep back the external air from entering the vagina, air, enters the vagina cause ballooning of the vaginal wall. Along with air, urine and dung also

entrance into the vaginal cavity. This contamination causes vaginitis, cerviciti- and endometritis. Klebsiella can generally be isolated from the exudates of the vagina.

Tumors : Fibromas may be found, which may be soft or hard, pedunculated or sessile.

Leiomyomas are also seen. Some have fair amount of collagen fibres when they are known as fibromyomas. These are comparatively harder than the pure leiomyomas.

Transmissible venereal tumor of the bitch is frequently seen in the vagina.

THE OVIDUCT OF THE BIRD

The oviduct is divisible into five portions.

The infundibulum : This is thin walled and sucks in the ovum released from the ovary.

2. **The magnum** is very glandular and has a good muscular wall The yolk during its passage through has a rotatory motion due to the spiral arrangement of the outer muscular layer. It is in this part that the yolk gets its albumin layer.

3. **The isthmus :** Here the two egg membranes are added to the albumin covered yolk.

4. **Shell gland or uterus :** Here the shell is formed.

5. **The vagina :** This is the terminal portion. Between shell gland and the vagina is a sphincter.

The formation of a normal egg depends upon :

1. The speed with which it passes through the several portions of the oviduct and 2. The normal function of various parts. So abnormal eggs may be encountered if :

i) the yolk is held too long in one section—excessive amounts of the substance that is normally added, will be deposited.

ii) the ovum is driven back by anti or reverse peristalsis. In this condition more or less of the substance already deposited will be added.

iii) there are disturbances of secretion of the parts. In this condition more or less of the substances may be added.

iv) if the movement of the ovum is too rapid, the quantity of the material to be deposited, may be too thin or even no material may be added.

v) if there is any inflammatory exudate in the oviduct. This will also be enclosed in the egg formed.

The following types of abnormal eggs may be encountered.

1. **Double yolked eggs:** In this condition two yolks enter the oviduct simultaneously and so are enveloped in a single layer of albumen, membrane and shell.

2. **Ovum in ova :** In this condition there is fully formed smaller egg within a large one. A fully formed egg, due to reverse peristalsis reaches anterior parts and comes in contact with another ovum and gets attached to it. Then this combined mass while moving downwards is enclosed with albumin and shell.

3. **Yolk-less eggs :** In this variery, the fully formed egg does not contain yolk. The explanation offered is the yolk that is released does not enter the

oviduct due to closure. But at the same time stimulates the oviduct to produce various layers of albumin, membrane and shell.

Or, a nidus of exudate may be coated by albumin etc. to from a yolk-less egg.

4. **Soft shelled eggs :** This variety is also known as *leathery eggs*. In this a shell is lacking. The causes are various and include deficiency of calcium, vitamin D or there may be disturbances of secretion of calcium by the shell gland. This disturbance may be due to:

(a) poisons – chemicals such as Zinc sulphide used as sprays on trees.

(b) infective agents as in infectious bronchitis; or

(c) too rapid peristalsis at that part.

5. **Layered eggs :** In this the various layers are duplicated. This is due to forward and backward movement of the egg mass so that various substances are deposited again and again

6. **Egg concretion:** When there is some inflammatory process of the oviduct the egg may be retained and so more and more clayers of albumin, membrane and calcium are deposited. There may be deposition of fibrin also and the whole mass may be dehydrated and calcified to form a huge mass of concretion.

7. **Foreign bodies within eggs :** Eggs may contain various foreign bodies These may be :

a) Feed, feathers, feces, small pieces of wood etc. that have reached through the cloaca, upper parts of the oviduct by antiperistalsis and get incorporated into an egg .

b) Parasites – *Prosthogonimus macrorchis* which is found in the Bursa of Fabricius normally, may go up the oviduct and be incorporated in an egg. So also *Ascaridia galli* may go up the oviduct from rectum and be incorporated in an egg.

c) Blood clots may be found when hemorrhage occurs due to rupture of vitalline membrane.

Abnormal location of eggs

Sometimes eggs, in various stages of development may be found in the abdominal cavity. They may be there because :

1. Yolk does not get sucked by the infundibulum and so is found in the abdominal cavity.

2. By antiperistalsis, partially formed egg may be thrown out through the infundibulum into the abdominal cavity.

3. The oviduct may rupture so that the egg is found in the peritoneal cavity.

The eggs and their contents acting as foreign bodies produce peritonitis and death. Sometimes, if secondary infection were to occur, the organisms multiply quickly since the egg-contents are good medium, death resulting.

Egg bound condition in the fowl is that in which the egg is lodged tightly in the oviduct or cloaca and is not laid. This gives rise to local irritation, inflammation, peritonitis and death if not relieved in time.

This condition may be due to :

(a) narrowing of the oviduct as a result of inflammation, thereby making it difficult for the egg to be laid.

(b) Far too large an egg like a double yolked egg.

(c) Paralysis of the muscles of the oviduct. In this condition the egg is not propelled further towards the cloaca.

THE MAMARY GLAND

MASTITIS (mammitis)

Mastitis or inflammation of the udder, may theoretically be caused by trauma of various kinds. But by far the most common causes are the infectious agents. All domestic animals suffer from this condition, but it is in the cow that mastitis is of importance because of the economical loss the owner may suffer.

Bovine Mastitis : Causes : The bacteria that have been found to cause mastitis, are *Streptococcus agalactiae : Streptococcus dysagalactiae; Staphylococcus aureus and albus; Corynebacterium pyogenes; E. coli; Pseudomonas aeruginosa; Pasteurella multocida; Brucella abortus; Mycobacterium tuberculosis; Actinomyces bovis ; Actinobacillus lignieresi ; Nocardia; Mycoplasma and Cryptococcus neoformans.*

Though formerly *Streptococcus agalactiae* was the commonest organism causing mastitis, after the advent of antibiotic treatment *Staphylococcus aureus* has been found to be the major cause of mastitis in cows.

In India, *Staphylococcus aureus* and *pyogenes* have been isolated from large number of mastitis cases. In some herds, on the other hand, gram negative organisms (*E. coli* and *Aerobactor aerogenes*) have been isolated.

Since the pathology of *Streptococcus* mastitis has been well studied, the same is described here When once udder becomes infected with *Streptococcus agalactiae*, it is said that it never becomes free of this organism. Though some kind of equilibrium develops between the udder and the organism, at times acute exacerbations may occur when the organisms multiply and increase in great numbers.

In a herd all cows are not equally affected. The route of infection appears to be through the teat canal. Wounds that occur in cow pox or those caused by suckling calves, facilitate infection. Contaminated cups of milking machines, milkers hands and farm utensils are other sources of infection.

The teat canal is lined by the same type of epithelium that covers the teat, but this epithelium seems to secrete a type of smegma, (rich in fatty acids) and this inhibits the streprococci.

The development of mastitis can be described under three phases : (1) The invasion phase in which the bacteria are able to enter the teat orifice and be present in the teat canal and cistern; (2) The infection phase in which the organisms are able to overcome the resistance and multiply and lastly (3) The inflammatory phase in which the organisms invade the udder.

When the streptococci invade the epithelium of the ducts inflammation results and due to the rapid development of the granulation tissue beneath the epithelium it is thrown into folds of polypoid thickening.

The organisms that penetrate the interstitial tissue cause edema and infiltration by neutrophils which destory some of the organisms. The lymphatics in the stroma become widely dilated due to infiltration by leucocytes that migrate from the regional lymph node. The epithelium of the acini becomes vacuolated and

desquamated. Streptococci are numerous in the ducts Milk being a good medium for the growth of bacteria infection is much more serious in a lactating udder than in a dry one. The exudation process gives rise to pathological fibrosis and involution of the acini. Subsequently macrophages and fibroblasts increase in number while neutrophils decrease. There may be stagnation of secretion in the smaller ducts and at this stage the udder may be firm and indurated due to the inflammed interalveolar tissue and retained secretion. When acute stage passes off and the damage caused is slight regeneration of the acini may occur. But if there is large scale destruction, regeneration is not possible and so the acini collapse and are replaced by granulation tissue. Interstitial spaces are infiltrated by lymphocytes. Such a gland is reduced in size and becomes hardened in consistency—"the shrunken quarter".

The acute systemic symptoms are due to the action of the bacterial toxins that diffuse into the general circulation.

Macroscopically, one or more quarters may be affected The secretion may be serous or may contain floculi and sometimes it may be purulent.

The gland is swollen and slightly hard. On section, it dose not possess the silky pink color of normal udder but is red or white. Lobulation is distinct. When fibrosis has set in it can easily be seen surrounding the lobules and the ducts.

Mastitis caused by *Streptococcus dysgalactiae* is more severe than that caused by *Str agalactiae* and more destructive leaving non-functional udder.

Streptococcus uberis produces a mild and chronic mastitis

Staphylococcus aureus usually affects younger animals; especially after parturition. Infection is supposed to be contagious and through the teat canal. There may be a peracute and fulminating type or more commonly a chronic type. Treatment is not very satisfactory. Differing from the stretococcal variety, in this type organisms persist in the interstitial tissue producing the granulomatous lesions described hereunder.

In acute cases mortality may be high due to toxemia and the udder is hard, swollen and very painful. Secretion of milk is very little and that too blood stained. Uninfected quarters are also swollen because of the action of the toxin (alpha) that has diffused into them. Gangrene may supervene when infection spreads to the blood vessels causing thrombosis. The udder then becomes cold and greenish or blue. Pitting edema is seen in the flank, inguinal region and the ventral aspect of the abdomen anterior to the udder. Gas may be present in the affected gangrenous area producing crackling sound on pressure. In such cases death may supervene or if the animal survives, the udder is totally lost.

In less severe cases necrotic foci are found surrounding which is a zone of leucocytes and this in turn is enveloped by fibrous tissue This granulomatous lesion is known as *botriomycosis*, in the centre of which can be seen gram positive cocci. The udder tissue contains numerous such granulomata. Fibrosis ultimately occurs resulting in the shrunken quarter described under streptococcal mastitis.

Staphylococcus pyogenes produces a very acute type of mastitis accompanied by severe systemic disturbances and fever. Animals die in a few days. Gangrene may supervene.

Corynebacterium pyogenes is the cause of the so called "Summer Mastitis' affecting both immature and lactating glands. The organism being pyogenic large amount of pus is produced, resulting in abscesses. There may be fistula discharging the pus to the exterior as well as large scale necrosis and sloughing. The latter are evidently due to thrombosis. Fibrosis with loss of function results in those animals that survive.

Coliform organisms, *E. coli* and *Aerobactor aerogenes,* produce, sometimes, an acute inflammation of the udder. Infection is supposed to be by blood stream though galactogenic infection cannot be ruled out. The affected quarter is hot, painful and edematous. Clotted milk, sometimes blood tinged, may be seen. Infection may subside, with involution of the quarter or it may become chronic with acute exacerbations developing later. Sometimes severe general toxic symptoms may be noticed with death following due to the potent toxins.

Gangrenous mastitis : In severe cases of mastitis caused by virulent strains of organisms thrombosis of the mammary vessels occurs resulting in infarction and gangrene. *Staphylococcus aureus* and *E coli* with *Clostridium welchii* produce this condition. Usually all the four quarters may be affected. The udder becomes cold, and bluish within 3 to 4 days after infection. In many cases death may supervene.

Cryptococcal mastitis is a surgical hazard that may be encountered in repeated intra-mammary infusions. *Cryptococcus neoformans* produces an acute inflammatory reaction. The gland becomes hard. One or more quarters may be affected. The milk turns to a watery, flaky secretion. The gland is fleshy and interlobular septa are distended with edema. There is large-scale destruction of the glandular tissue and the alveolar and ductal epithelium is liquified to form a viscid, mucoid material. In sections, the double refractile fungus can be seen in large numbers, some of which are found englufed by the histiocytes. In some isolated chronic cases granulomatous nodules with interlobular and intralobular fibrosis occurs together with infiltration by histiocytes and lymphocytes.

Organisms may be found in the supra-mammary lymph gland. Metastases, in some cases, may be found in the lungs.

Brucella mastitis :

In infection by Brucella, udder is the reservoir of the organism, which is excreted through the milk. The udder may not show any changes or only scattered lesions may be observed which are not very characteristic.

Histologically the lesion is a granuloma which is intralobular. It consists of lymphocytes, plasma cells, histiocytes and a few Langhans' type giant cells.

In some cases, instead of the granuloma, an interstitial mastitis is seen with infiltration of lymphocytes, histiocytes, plasma cells and fibrous tissue. Regional lymph nodes may or may not be swollen.

Mycoplasmal mastitis : This is identified to be caused by several strains of *Mycoplasma sp.* Cows of all ages are affected. All four quarters may be involved, with sudden drop in milk yield Milk becomes abnormal grossly. There is cessation of lactation and the animal will not be useful again for dairy purposes. Faulty milking machines and unsterilised teat syringes, contamination of teats during milking and inhalation are the sources of infection

The condition is a purulent mastitis and the organism may also invade the blood and then affect the joints and other tissues causing systemic symptoms. There is arthritis with swelling of the joints and lameness.

Milk is thick and cheesy and may be tinged with blood. There may also be clots or granular material in some specimens. The udder is swollen and later it gets atrophied. Treatment so far has not been of any avail.

The following organisms also may cause mastitis : *Pseudomonas aeruginosa; Pasteurella multocida; Nocordia asteroides; Candida sp,* and *Mycobacterum tuberculosis.*

Mastitis ine wes

The organisms responsible for mastitis in ewes are : *Staphylococcus aureus, Pasteurella hemolytica; Corynebacterium pyogenes; Streptococci and Coliforms.*

Infection is usually ascending through the teat canal. Injuries made by suckling lambs provide a route of entry for the organisms.

Staphylococcus aureus produces a more acute disease than in the cow. Morbidity is 25% and mortality is greater than in the cows, being 25 to 50%. There are severe systemic disturbances with intense edema of the udder, which may extened upto the belly and gangrene may supervene, when the udder assumes blue color and so the condition is named "blue bag". There is serous or sero-fibrinous or purulent exudate in the acini, interlobular septa and in the interacinar septa. Alveolar exudate contains desquamated epithelial cells and leucocytes. Large scale necrosis may occur with abscess formation. Abscesses may rupture on the skin. Ultimately, the gland becomes fibrosed and functionless. Mastitis caused by other organisms is less severe than that produced by *Staphylococcus aureus.*

Contagious agalactiae of goats and sheep :

This is a disease primarily of goats but slightly infective to nearby sheep also The causal agent is *Mycoplasma agalactia.*

Though the udder is mostly affected, the disease may run a septicemic course in which mortality is heavy (10 to 33%). Later, infection localises in the eyes, the joints and the udder.

Infection is probably by ingestion, though it may also occur by way of teat-canal and the conjunctiva. The infective agent is eliminated in the secretions and discharges.

Both adult goats and kids are susceptible. Pregnant animals may abort. If live kids are born, they may be found infected.

The lesions noticed are :

Mammary gland : The inflammation commences in the interstitial tissue, with fibrosis. Later acini may be involved and they may be atrophied as the fibrous tissue increases and encroaches on the acini. Ultimately the udder is completely fibrosed and so lost. Organisms are voided in the milk.

Eye : In about half the number of cases mucopurulent conjunctivitis and keratitis complicated by ulceration are noticed.

Joints : Mostly the carpal and tarsal joints are affected showing arthritis and periarthritis, manifested by lameness. The peri-articular tissues appear swollen due to inflammatory edema.

Mastitis in pigs.

Staphylococcal mastitis. Stphylococcus aureus causes sporadic cases. The condition is chronic with the formation of large fibrous nodules in the gland. The nodules may open out through sinuses and then pus containing small granules may be discharged. The udder is lost.

Mastitis caused by Coliform bacteria: This is usually found in sows heavily fed with concentrates and in those which farrow in unhealthy pens. Heavy losses are encountered.

The udder is swollen, hard, discolored and painful. There may be severe systemic reaction. The sow does not get up and does not allow piglets to suckle. So the piglets die of starvation. If they suckle, they suffer from enteritis. There may be concomitent metritis. The udder is lost and even if the pig survives, it will not be useful for breeding.

Mastitis caused by C. pyogenes.

This is very frequent in pigs. The infection may be *primary* or it may be *secondary* from metastatic involvement from a focus somewhere else. In the udder there are absecesses with central collection of greenish pus. Fistulae may be present opening out on the skin from the abscesses. Such animals are no more useful for breeding.

Mammary neoplasms: As described earlier, tumors of the mammary gland are common only in the canine species in which the mixed tumor (fibrochondroadenocarcinoma etc.) is frequently seen. (pages 255, 256.)

THE MALE GENITAL SYSTEM

Testes
- Cryptorchidism
- Hypoplasia
- Hematocele
- Hydrocele
- Testicular degeneration
- Orchitis

Epididymis

Spermatic cord
- Funiculitis

Seminal vesicles

Prostate
- Hypoplasia

Penis and prepuce
- Posthitis
- Balanoposthitis
- Phimosis and Paraphimosis
- Neoplasms

TESTES

Cryptorchidism: In the fetal life the testes are located in the sublumbar region, from where they descend into the scrotum during later stages of pregnancy. Sometimes the testes do not descend and so are retained in the abdominal cavity. This condition is known as cryptorchidism.

Cryptorchidism may be unilateral or bilateral. The bilaterally cryptorchid animal is sterile The condition is more often seen in swine and horses. In horses it is believed to be hereditary, being a sex-linked dominant factor.

Because of the higher temperature in the abdominal cavity, the testes do not develop normally Spermatogenesis, therefore, is arrested. The interstitial cells of Leydig may be normal or hyperplastic. Testes are smaller and softer than normal.

Cryptorchid stallions sometimes develop increased sexual urge (Satyriasis) and become vicious and difficult to control. In dogs, cryptorchidism appears to predispose the testes to tumors. Seminoma and Sertoli-cell tumor are met with more often in such animals.

Hypoplasia of testes :

This condition, though seen in all animals is more frequent and of greater importance (economic) in the bull In the Swedish Highland cattle, hypoplasia of the testes is a problem, being hereditary. Hormonal disturbances, vitamin deficiencies and some poisons may be the other causes.

Hypoplastic testis is smaller in size and is harder in some cases. **Microscopically** the picture, varies with different degrees of hypoplasia. In severe conditions the tubules are narrowed and have only one layer of cells, which do not show mitotic activity, denoting cessation of spermatogenesis. The basement membrane may be hyalinised and thickened. Increase in the peritubular connective tissue is responsible for the hardness. No change may be noticed in the cells of Leydig which may be increased in number.

In milder cases varying degrees of spermatogenesis may be found, some showing giant spermatids.

Hematocele is the presence of blood in the tunica vaginalis. Apart from trauma locally, this condition is found whenever there is hemoperitoneum. In leptospirosis and Infectious Canine Hepatitis hemolysed blood is seen in tunica vaginalis

Hydrocele is the condition in which clear serous fluid accumulates in the tunica vaginalis. This condition may be an accompaniment of generalised edema or ascites. It may also be due to local inflammation as a result of trauma, when the fluid may be turbid or blood stained.

TESTICULAR DEGENERATOIN

Degeneration of seminiferous epithelium is the commonest type of bull infertility encountered. This condition may be unilateral or bilateral.

Causes : The causes of testicular degeneration are many and it is not possible to pin-point the primary cause, because the factors responsible for degeneration may have ceased to exist before the degeneration is noticed.

Failure of thermo-regulatory mechanism which is responsible for the maintenance of testicular temperature lower than that of the body is proved to be one of the influencing factors for testicular degeneration. Excess of scrotal fat, short Cremaster muscle, inguinal or scrotal hernia, periorchitis and dermatitis and edema of the scrotum, interfere with thermo-regulatory mechanism.

Physical : Excessive heat, freezing, trauma, hematoma and laceration of scrotum may cause testicular degeneration. The condition may also be noted in the animals recently transported.

Localised and systemic infections are also common causes of testicular degeneration. Fever, toxemia, inflammatory changes of tunica vaginalis, orchitis, inflammation of the scrotum and epididymitis, contribute to the testicular degeneration.

Belgian workers have found an enteric virus to be a cause of this condition.

Nutritional factors : deficiency of : Vit. A, phosphorus, protein and energy requirements.

Avitaminosis A causes testicular degeneration through inhibition of release of gonodotrophins

Vascular lesions: torsion of testes, testicular tissue biopsy, spermatic cord compression (while controlling hemorrhages), inflammation of testicular artery, especially in the horses due to migrating strongyl larvae may cause testicular degeneration.

Hyaline degeneration of arteriolar walls and thrombosis of testicular arteries which are associated with age also cause testicular degeneration.

Obstructive lesions of the head of the epididymis interfere with the flow of spermatozoa and secretions of the tubular system of testes. The back pressure thus caused produces degeneration of the germinal layers of the seminiferous tubules.

Toxic substances that cause testicular degeneration include a number of chemicals (arsenical dips), metals, rare earth salts, and radiation.

Injection of Cadmium chloride causes testicular degeneration through lesions of vascular endothelium resulting in thrombosis

The degeneration caused by highly chlorinated napthelenes is generally reversible. Spermatogonia-B and late spermatogonia-A are highly sensitive to radiation.

Hormonal imbalance of the FSH and LH and improper administration of hormones.

Auto immunisation with autologus or isologus spermatozoal materials.

Macroscopically, the gross lesions include soft and flabby consistency in normal sized or smaller testes. The tunica albugenia does not bulge on the cut surface. In chronic cases fibrosis of the testes may be recognised. In cases of extensive fibrosis the testis is firmer and smaller. Calcium deposition may be noticed as yellowish white flakes in the degenerated products of the tubules

In acute degeneration deposition of calcium is observed in the connective tissue.

Microscopically, the tunica albugenia is condensed, thickened and wrinkled Sometimes it becomes impossible to differentiate the hypoplasia from testicular degeneration. Histological changes vary with the severity and stage of degeneration. The degenerative changes may not necessarily involve the tubules uniformly. The entire length of some tubules may be affected, while in others only partial. During early stages of degeneration, failure of maturation of spermatozoa, and degeneration of spermatids, are evident. Many spermatids are observed to be necrotic. Some of these give rise to multinuclear phagocytic giant cells. During advanced degeneration cytoplasmic vacuolation and nuclear pyknosis are observed

in the precursors of spermatids. As the condition further advances denudation of layers of germinal epithelium occurs leaving the basement membrane exposed. Lastly even the very resistant sertoli cells may become denuded. The tubules then collapse and are replaced by connective tissue.

In some cases when the degeneration is not rapid, along with the changes in the germinal epithelium, thickening and hyalinisation of the basement membrane and an increase in the interstitial connective tissue are noticed. Finally the the tubules become replaced by dense hyaline connective tissue In the lumen of stenotic tubules large polyhedral mononucleate and binucleate cells with granular eosinophilic cytoplasm are observed These may contain within their cytoplasm a golden yellow pigment—"wear and tear" pigment . The nature of these cells is not known but are probably altered spermatogonia with an unusual capacity for survival.

Stenosis of a portion of tubules leads to stagnation of spermatozoa and tubular secretions and ultimately calcified foci desquamate and the basement membrane fragments. Contact of degenerated sperm, with the connective tissue causes granulomatous reaction.

The semen picture is of great diagnostic value in testicular affections. The semen volume is not generally affected but the density may tend to be poor. The sperm mobility is affected. Sperm count may be low. The number of abnormal sperms increases ranging from 10% to 50%. Detatchment of heads, variation of the size and the shape of the heads are common abnormalities. Presence of proximal protoplasmic droplets, looped middle piece and tail, tight coils of middle piece and tail, may be other abnormalities encountered.

ORCHITIS

Inflammation of the testes occurs more frequently in sheep, cattle and swine.

Causes :

1. **Trauma :** This is more common in rams, in which the testes are pendulous
2. **Bacteria :** A variety of bacteria are responsible. Infection may be hematogenous or may be extension from the lower genital organs through the epididymis.

The most common bacteria causing orchitis are :- *Brucella abortus* in bulls, *Brucella suis* in boars.

Salmonella abortus equi and P. *mallei*—in equines.

Corynebacterium pyogenes, Corynebacterium ovis—in rams.

Orchitis produced by *Brucella abortus* in bulls, is mostly an acute condition with swelling of the scrotum, which is hot and painful. Since the testis is located compactly in tunica albuginea, a tough fibroelastic membrane, swelling of the testis to any appreciable degree does not occur. It is only the accumulation of the inflammatory exudate in the tunica vaginalis and the scrotum that is responsible for the swelling seen. The exudate is fibrino-purulent and sometimes hemorrhagic Adhesion of the parietal and visceral layers of tunica vaginalis may occur. Due to pressure by the tunica albuginea, and the action of the pathogen, necrosis of the testes occurs with suppuration and abscess formation. Abscess may open out on the scrotum.

Microscopically, there is mycrocyst formation, degeneration and desquamation of the tubular epithelium. Infiltration of lymphocytes, macrophages. and plasma cells occurs in the interstitial tissue and the organisms can be seen in large numbers in the epithelial cells and necrotic areas In many cases, an accompanying epididymitis is present.

In chronic cases, in the initial stages, miliary tubercle-like granulomas form (as in mastitis) in the tubules and intertubular connective tissue. Because of pressure *of the infiltrating tissue* and action of the pathogen, degenerative changes and atrophy of tubular epithelium occur along with fibrosis and shrinkage. Finally the testes are hard, shrunken and very much smaller in size.

In the boar, the characteristic appearance is the formation of multiple abscesses, consisting of a central necrotic and caseated material surrounded by epithelioid cells, Langhans' type giant cells, plasma cells, lymphocytes and a connective tissue capsule.

Salmonella abortus equi produces an acute suppurative orchitis in donkeys and horses The seminiferous tubules rupture and the sperms spill into the interstitial space, and so a foreign-body reaction is set up. General systemic and febrile reaction is usually seen in this condition.

Pasteurella pseudotuberculosis rodentium produces suppurative orchitis in rams. Pathogen is transmitted from the rodents by tick bites.

Tubercular orchitis : The lesion is usually a secondary hematogenic infection from a primary focus somewhere else. Lesions noticed may be calcified miliary nodules in which the typical histology of a tubercle nodule may be noticed or it may be in the form of a diffuse caseating lesion radiating from the rete testes. Very often the epididymis is also affected.

Straus's test used for diagnosis of diseases caused by *Malleomyces mallei* (glanders), *Malleomyces pseudomallei* (melioidosis). *Cryptococcus neoformans* (epizootic lymphangitis), *Corynebacterium avis* (ulcerative lymphangitis and ovine lymphangitis), *Brucella abortus* and *Pseudomonas aeruginosa,* is essentially a suppurative orchitis and peri-orchitis in a male guinea-pig that results in 2 or 3 days following an intraperitoneal injection of a small amount of the culture of the organism.

Tumors : Described under the chapter "Neoplasms". (Page 259)

EPIDIDYMITIS

Inflammation of the epididymis is usually seen along with orchitis.

Of special importance is the epididymitis in rams caused by *Brucella ovis.* In this condition no lesions may be found in the testes

Usually the tail of the epididymis is affected, where the lesion produced is a granuloma. The pathogen incites an inflammatory reaction at the place of its localisation with inflammatory edema, infiltration by lymphocytes and macrophages. Neutrophils may appear a little later. The epithelium first shows papillary hyperplasia and later undergoes hydropic degeneration accompanied by fibrosis of the interstitial tissue, rendering the organ to become hard Epididymis is enlarged to 3 or 4 times.

Occlusion of the lumen by the debris and exudate results in spermatocyst formation. Should this cyst rupture into the tunica vaginalis, a foreign body reaction is set up with dense adhesions between its visceral and prietal layers.

Though primary orchitis is not found in this condition, secondary degenerative changes, with calcification may be seen in the seminiferous tubules due to stasis of sperm.

Spermatic cord : The inflammation of the spermatic cord which is usually seen after castration is called **Funiculitis.** This may be acute and necrotizing as seen in the pig or it may be chronic—**scirrhous cord**—in horses and cattle. It is common in the pig because in this animal contamination can occur very easily due to the proximity of cord to the ground and to the general insanitary conditions of the environment.

In the scirrhous-cord, there is excessive formation of granulation tissue in the stump of castrated cord due to infection, usually by staphylococci. (*S. aureus*) Abscesses with thick walls may be present in this tissue. Leucocytic infiltration is common. A characteristic appearance is the presence of granules in the inflammatory tissue consisting of colonies of bacteria surrounded by a zone of clubs and inflammatory cells as in actinomycosis. This is known as **Botriomycosis.** In the spermatic cord and testes of horses, verminous granulomas caused by wandering larvae of *Strongylus spp* may occasionally be found.

SEMINAL VESICLE

Segmental aplasia of ampulla and seminal vesicle usually occurs in association with segmental defects of the epididymis.

Duplication of seminal vesicle on one or both the sides may be occasionally seen.

Seminal vesiculitis : Inflammation of seminal vesicle is usually rare but this condition is of serious concern when it occurs in a bull which is used for artificial insemination. In these cases, the pathogenic organisms may be transmitted to a wide population.

Causes : *Brucella abortus, Corynebacterium pyogenes, PLV* and *Mycoplasma* are some of the organisms incriminated.

Macroscopically, seminal vesicles are enlarged and tender on palpation in acute stages. There is a tendency for loss of lobulation.

Microscopically, the acute stage of the disease is characterised by infiltration of alveoli and interstitial tissue with neutrophils. In chronic cases, lymphocytes, plasma cells and histiocytes become numerous in the interstitial tissue.

Not all cases of seminal vesiculitis can be diagnosed by mere rectal palpation. In some cases, the seminal vesicles may not be noticeably enlarged. For clinical diagnosis of vesiculitis, palpation of the vesicles is useful if they are considerably enlarged. If they are not enlarged, massage of the vesicles and stripping of the ampullae will force inflammatory cells into the urethra and exudate will drip from the penis. Semen collected following this procedure has a marked increase in leucocytes and there may be clumping of exudate so that the sample will have the appearance of curdled milk.

THE PROSTATE

Hyperplasia of the prostate : This condition is seen only in dogs. Dog above five years of age and especially housebred, are commonly affected.

Causes : The precise cause of prostatic hyperplasia is obscure. It is thought that excessive testosterone may produce this condition which yields to castration. Forced retention of urine in housebred dogs in sometimes thought to be one of the factors.

Macroscopically, the gland is very much enlarged, with either a smooth or nodular surface. Sometimes the biloted appearance of the gland is lost. Fluctuating cysts are palpable under the capsule.

Microscopically, the picture is one of hyperplastic adenoma in which the acinar cells are increased both in size and number. The supporting tissue is also increased. The epithelium is tall and is frequently thrown into folds as papillary projections into the lumen. There is always infiltration of lymphocytes and plasma cells in the interstitial tissue Some acini may be cystic with increased amount of secretion which pressess upon the epithelium, flattening it. The interlobular connective tissue may be increased. Bladder may show compensatory muscular hypertrophy.

Clinically, due to pressure on the rectum by the enlarged prostate constipation may be produced. Difficulty in micturition is attributed not to the pressure on or narrowing of urethral lumen, but to paresis of the bladder resulting from pressure of the enlarged gland on the parasympathetic nerves.

PENIS AND PREPUCE

Inflammation of the prepuce is called **posthitis** and that of glans penis **balanitis.** Usually both occur together as **balanoposthitis.** In the dog it is a common condition. Cause may be trauma or bacteria. There is catarrhal exudate with infiltration of leucocytes into the degenerated epithelium. Mucosal lymph follicles may be enlarged. In other animals balanoposthitis is associated with various organisms inculding *Pseudomonas aeruginosa;Corynebacterium pyogenes* and *C. renale.* This condition is also met with in bulls that cross cows suffering from "Infectious pustular vulvovagintis". In the last named condition (known as infectious pustular balanoposthitis) pustules form on the preputial lining and glans penis, giving them a granular appearance. Infection does not extend into urethra. Edema of penis and prepuce may cause paraphimosis

Phimosis is a condition in which the penis cannot be extended from the prepuce, due to inflammatory swelling.

Paraphimosis is the opposite condition in which the extended penis due to inflammatory enlargement, cannot be withdrawn into the prepuce.

Neoplasms : In the bull, transmissible fibropapilloma is encountered. These are multiple and cauliflower-like.

In the horse squamous cell carcinoma may be met with

In the dog transmissible venereal tumor is common.

CHAPTER 21

THE MUSCULOSKELETAL SYSTEM

THE SKELETAL SYSTEM

The following terms indicate certain anomalies and abnormalities in the skeletal system.

Abrachia :—Absence of anterior limbs.

Amelia :—Absence of limbs. The scapula and pelvic girdle may be intact or rudimentary.

Apodia :—Absence of posterior limbs.

Micromelia :—All parts of limbs are present but are of smaller size.

Perodactyly :—Absence of some of the toes.

Adactylism :—Absence of all the toes in a limb.

Brachydactylism :—Abnormal shortening of toes.

Polydactylism :—More number of digits; seen in horse and pig.

Syndactylism :—Fusion of toes, seen in cattle and pigs.

Prognathism :—Having a long jaw,—*pig-mouth* condition in horse.

Brachygnathism :—Having a short jaw. —*parrot-mouth* in horse.

Lordosis :—Is the curvature of the spine with a ventral convexity due to heavy loads or heavy abdominal organs; terminal parts of the thoracic spine and the lumbar spine are involved. The spinous processes rub against each other and so periostic osteophytes develop.

Kyphosis :—Abnormal curvature and dorsal prominence of spine—hump-back. This is rare in animals

Scoliosis:—Abnormal lateral curvature of the spinal column—may be congenital and sometimes inherited. May be due to diseases of bones like achondroplasia, osteodystrophy.

Torticollis :—Wry neck—twisting of the neck with an unnatural position of the head.

Various factors govern bone formation. Briefly these are :—

1. **Minerals:** Sufficient amount of calcium and phosphorus must be supplied in the food in correct proportion and the intestinal tract must be healthy and of correct pH for their absorption.

2. **Proteins :** Sufficient amount of protein must be fed for the formation of the ground substance.

3. **Vitamins :** Vitamins A, D and C control bone formation. Vitamin A is necessary for the proper stimulus and bone growth. Its deficiency produces inanition and growth rate is retarded. It is concerned with the metabolism of endothelial cells and so is required for the proliferation of endothelial cells of the capillaries, for their transformation into osteoblasts and for the erosion and removal of the calcified cartilage. Vitamin D controls absorption and utilisation of calcium and phosphorus How these are brought about is not clear. Vitamin C controls the formation of osteoblasts and so controls deposition of osteoid

4, **Endocrines :**

(a) **Parathyroid** controls calcium and phosphorus metabolism. The action of the parathyroid hormone is two fold (1) It increases the phosphate diuresis and (2) it produces hypercalcemia through its action on the osteoclasts which withdraw calcium from the bone.

(b) **Anterior Pituitary:** The growth hormone influences the growth of connective tissue, especially bone. Gigantism occurs where there is increased secretion of growth hormone and the bony growth is enormous.

(c) **Thyroid :** Thyroxine controls the metabolism of carbohydrates and fats and so energy production is under its control. Indirectly therefore, bone formation is influenced by the thyroid as energy prduction is controlled by it. In hypothyroidism there is retardation of endochondral bone formation and osteporosis which occurs due to negative metabolism balance.

(d) **Gonads and Adrenal cortex:** Bone, growing and mature, is affected by estrogens and androgens. Their mode of action is not clear. These hormones accelerate the epiphyseal closure and maturation of the bone. In deficiency of these hormones there is disproprotionate elongation of immature long bones.

The following three types of cells are found in the bone :—

1. **Osteocytes** are the ordinary bone cells that are found in the lacunae. These are old cells that cannot divide.

2 **Osteoblasts** are bone producing mesodermal cells and line the deep layer of periosteum, the endosteum and the Haversian canals These cells, like fibroblasts, have great power of proliferation and produce alkaline phosphatase. Osteoblasts secrete precursors of collagen and mucopolysaccharides. The latter act as the cement substance and in this is embedded collagen. These form the matrix of the bone called osteoid

3. **Osteoclasts** are the phagocytes af bone and are multinucleated Foreign body giant cells can be formed from them. These are under the control of parathyroid and under its influence remove bone.

Phosphatase : The alkaline phosphatase found in bone is formed by the osteoblasts It is believed that this enzyme splits the organic phosphate compounds liberating excess of phosphate which upsetting the local calcium phosphate balance leads to the precipitation of calcium salts. It is in this manner that mineralisation of the osteoid (the organic matrix of the bone) is believed to take place. Evidence is now available that the phosphatase of bone is concerned in the elaboration and secretion of protein—the organic matrix.

Osteodystrophy denotes a disturbance in the growth of bone. Osteodystrophies may be acquired or congenital. Causes are many and varied.

1. Lack of minerals and vitamins (Rickets and osteomalacia etc.)
2. Excessive hormones—gigantism, acromegaly. osteoporosis.
3. Unknown causes.

RICKETS

This is a condition seen in growing young animals in which there is a failure of adequate calcification of bones. Similar condition in adult mature animals, in which growth of bones has stopped, is known as *osteomalacia*—literalty meaning softening of bones.

Essentially, rickets is a deficiency disease—deficiency of calcium, phoshporus or vitamin D. The deficiency of these may arise in several ways.

Deficiency of calcium :

1. Deficiency of calcium in the diet : Inadequate calcium in diet may not occur in animals.

2. Improper balance of calcium and phosphorus : Excess of phosphorus in the ration (feeding too much of bran etc.), may combine with calcium and form a relatively insoluble $Ca_3(PO_4)_2$ which is excreted in the feces.

3. Failure of absorption of calcium : Calcium is mostly absorbed as $CaH PO_4$ and for this the medium must be acidic. If the intestinal contents are excessively alkaline, calcium cannot easily be absorbed.

4. Formation of insoluble complexes : (a) Oxalates and phytates present in some green leaves and grains respectively may form insoluble compounds in monogastric animals and are lost in the feces.

Excess of oxalic acid in leaves and excess of lactic, tartaric, and malic acids in silage bind calcium in large quantities. Similarly acid breakdown products of proteins bind calcium if food is too rich in protein. In too coarse a food greater amounts of hippuric acid is formed from cellulose. Too rich or poor fat reduce the utilisation of calcium. Reduced body movement may also be a contributory cause

(b) When sulphur is fed to chicks as a coccidiostatic, it combines with calcium to form insoluble compounds which are lost in the feces.

5. Steatorrhoea : In dogs, fatty acids from fats that are not assimilated may combine with calcium, forming calcium-soaps which are lost in the feces. This is common in man in *Coeliac disease*. Since Vitamin D absorption is conditioned by absorption of fat, in steatorrhoea, Vitamin D also is not absorbed and this still further affects the absorption of calcium, since vitamin D is not only necessary for calcium absorption but it also increases calcium absorption by the intestinal mucosa.

6. **Renal Disease :** In nephritis, phosphorus is not excreted as it should be and so accumulates in the blood and body. The excess phosphate ions are excreted through the intestinal tract, where they combine with calcium to form insoluble compound which is lost in the feces.

7. **Increased requirements in growing animals :** If adequate quantities are not allowed in the ration deficiency may arise in growing animals in which the needs for calcium and phosphorus are great.

Deficiency of Phosphorus :

1. **Inadequate amounts of phosphorus in diet :**—In certain parts of the world soil is deficient in phosphorus and so animals maintained solely on the plants from such soils develop phosphorus deficiency. which is clinically manifested as osteophagia (pica). (See page 145)

2 **Change of reaction of intestinal contents :** As explained above an acid medium is required for the absorption of $CaH\ PO_4$. But if the reaction changes absorption cannot occur.

3 **Steatorrhoea :** Vitamin D is necessary for absorption of phosphorus also. In steatorrhoea Vitamin D is not absorbed and so is not available to the body and so phosphorus is not absorbed.

4. **Formation of insoluble complexes :** Excess of calcium, iron and aluminium form insoluble phosphorus compounds and so phosphorus deficiency results.

5. **Increased requirements :** In growing animals, if adequate quantity of phosphorus is not allowed in the ration, deficiency will result.

Deficiency of Vitamin D :

Insufficiency of vitamin D may occur in young animals.

Causes : I. Steatorrhoea : As mentioned earlier. vitamin D is not absorbed in the absence of fat.

2. **Diseases of liver :** If sufficient bile is not secreted, absorption of Vitamin D may be interfered with.

3. **Deficiency of sunlight :** Since vitamin D can be formed in the skin by the action of ultroviolet rays on ergosterol, deficiency of sunlight may cause deficiency of vitamin D. Smoke and smog in industrial places filter the ultraviolet rays and so rickets may supervene.

The essential defects in rickets are :

1. The cartilage cells are not calcified.
2. The cartilage cells are resistant to degeneration.
3. Failure of the blood vessels to invade and corrode the cartilage.
4. Overgrowth of the osteoid on the persistent and growing cartilage.
5. Increased growth of fibrous tissue in the osteochondral zone and
6. Absence of calcification of osteoid.

While describing the normal formation of bone it was mentioned that the cartilage cells nearest the diaphysis should become degenerated, must be calcified and then the capillaries nearby should invade and corrode the cartilage, on the remnant trabeculae of which the osteoblasts that arrive there form the organic matrix. the osteoid, over which calcium salts are deposited. In rickets, due to

the deficiencies enumerated above these events do not take place. So the cartilage cells persist and grow and the zone of cartilage is wider and longer. Therefore chondrocostal and osteochondral junctions are widened and enlarged. Since osteoid is not calcified, the osteochondral zone is softer than normal. So, on pressure and due to weight the bones bend

Symptoms noticed are stunted growth, bowing of the limbs. ("bow legs") "Pot-bellied" appearance of the abdomen, kyphosis and scoliosis, enlargement of ends of bones and joints; bending of knees and fetlocks; overextension of pasterns with overgrowth of hooves, deformity of the pelvic bones (which later may cause maternal distocia), cranium is more dome shaped and the fontanels are wide; "Rickety-rosary", (enlarged chondrocostal joints appearing as a string of beads) and crooked sternum in birds. Teeth may be poorly formed and irregular; jaws cannot be closed. Shortening of bones results In rachitic dwarfism

Macroscopically, the epiphyseal cartilages are abnormal, wider and soft and so can easily be cut.

Microscopically, epiphyseal cartilage is wide and the osteochondral junction is irregular Tongues of surviving and resistant cartilage cells appear to be arranged in a disorderly and crooked manner Osteoid which is pink staining is abundant while the blue staining bony trabucule are few and widely separted.

Overgrowth of fibrous tissue occurs at the osteochondral zone and in the marrow. There is, therefore, reduction of myeloid cells.

Restitution of the deficiencies corrects the disorder. But deformities persist eg. "bow legs" remain,

Diagnosis: X ray shows enlargement of epiphyseal plate, enlargement of epiphyseal line, and bending of bones. There may be decrease in serum calcium and phosphorus and increase in alkaline phosphatase.

OSTEOMALACIA

This condition is othewise known as adult rickets and occurs in animals in which endochondral ossification has ceased. Like rickets, this condition is characterised by failure of calcification of matrix and appearance of excess of osteoid Atrophy of the bone substance is also present due to excessive resorption in bone consequent on negative mineral balance. Because of either excessive demands of the body for or deficiency in the dietary intake of the minerals, to meet the demands of the mineral requirements by the body, bone resorption occurs.

The medullary cavity is enlarged. Compact bone becomes spongy, In extreme cases all that is left is a membranous sac covered over by the periosteum containing traces of bone.

Because of lack of mineralisation the skeleton becomes soft and fragile and so fractures and deformities occur. The articular heads of some of the bones may sometimes separate. Kyphosis and lordosis are frequently seen together with narrowed pelvis.

The causes for osteo nalacia ave similar to those of rickets :

i) deficiency of calcium and phosphours, ii) deficiency of Vitamin D and iii) chronic nephritis in which phosphorus excretion is diminished.

Osteomalacia may also be seen in pregnancy when maternal calcium is drained to the fetus and in high yielding cows in which large amounts of calcium are excreted through the milk.

Microscopically, the following may be observed : active resorption of bone by osteoclasts, reduction in size and number of the trabeculae of spongiosa and presence of excess of osteoid

Osteodystrophia fibrosa : This is a condition of the bone dependant upon disturbance in metabolism and frequently occurs in animals It is noticed mostly in animals imbalanced in calcium and phosphorus contents. Normally, the Ca:P ratio in food should be 2:1. But if this ratio is reversed and becomes 1 : 3 or wider, osteodystrophia fibrosa results.

In the horses, osteodystrophia fibrosa is known aa "Bran Disease" or "Big Head" or "Millers' Disease" and is common among horses maintained by millers. Since bran is a cheap by product in the milling of wheat, horses of the millers were maintained exclusively on bran which has a high phosphorus content This phosphorus combines with calcium of the food and forms insoluble $Ca_3\ PO_4$ in the intestines and is excreted in the feces. Therefore sufficient amount of calcium and phosphorus is not available to the body and so hypocalcemia results. This in its turn causes osteomalacia. Hypocalcemia stimulates the parathyroid, which becomes hypertrophic, producing excess of parathormone. This hormone acting on the bone (through the osteoclasts) decalcifies it producing osteomalacia. All bones are not equally affected. This affection is first noticed in the facial bones. The bones that are most active are affected. The bones become soft as calcium is withdrawn from them. So as to strengthen the bones, fibrous tissue proliferation occurs. Since soft bones bend and twist irritation is produced and this causes inflammation to occur which ultimately is responsible for fibrosis. This fibrosis is most apparent under the periosteum hence the bone appears larger than normal. The bone marrow may also be replaced by the fibrous tissue. The facial bones of the horse appear swollen and hence is the name "Big Head".

To start with, the disease is manifested by abnormal gait, stiffness and shifting lameness. These symptoms are later followed by anorexia, anemia and cachexia. The anemia is myelophthisic in origin—due to the replacement of marrow by the newly formed fibrous tissue. There may be swelling of the jaws, dyspnoea due to narrowing of the nasal passages, difficulty in mastication, loosening and loss of teeth. Fractures are common.

Microscopically, large masses of fibrous tissue are seen in which are found remnants of bony trabeculae. Sometimes the fibrous tissue may be of such proportion as to resemble a fibroma.

Osteitis Fibrosa Cystica : (von Recklinghausen's disease) This condition is a form of osteodystrophia fibrosa The essential nature is decalcification of bone, substitution by fibrous tissue and formation of cysts.

Csuses are :

Hyperparathyroidism :

A. **Primary :** As in a tumor of the parathyroids.

B. **Secondary :** i) Dietory calcium insufficiency, ii) Dietory phosphorus insufficiency, iii) chronic renal disease.

It has already been noticed that the function of parathyroid is two fold.

i) to facilitate excretion of phosphorus in the urine.

ii) to remove calcium from bone through the mediation of osteoclasts.

The parathyroids are sensitive to blood calcium level. Any decrease in blood calcium stimulates the parathyroids and hyperplasia occurs with increased production of parathyroid hormone. Acting on the bone, this hormone is responsible for withdrawal of calcium through the activity of osteoclasts.

Normally glomeruli filter phosphates some of which are reabsorbed by the tubules. But in renal disease the phosphate excretion is much reduced and so the phosphate level of the blood rises – hyperphosphatemia. To compensate for the rise and to keep the Ca:P ratio constant, calcium is withdrawn from the bones. Besides, the retained phosphate is excreted through the bowel where it combines with calcium and forms an insoluble compound and so is lost to the body, resulting in hypocalcemia, which stimulates parathyroid, liberating excess hormone and this withdraws calcium from the bone to maintain normal blood calcium level.

The function of parathyroids is therefore homeostasis, to maintain the optimum Ca : P level in the blood.

Under the circumstances described above when calcium is removed from the bones, they become soft and weak and so to strengthen them, there is fibrous tissue proliferation. This change, though found in all the bones, is more prominent in the bones of the head. In the dog, the lower jaw becomes so soft that it is as pliable as rubber. "Rubber-Jaw syndrome". Since the newly formed connective tissue is poorly supplied with blood, degeneration, softening and cyst formation occur—hence the name Osteitis fibrosa cystica.

Microscopically, osteoid and fibrous tissue are more in evidence. Attempt at formation of new bone in some places is evident by the presence of osteoblastic activity. In others, osteoclasts are seen nibbling away spicules of bone. Cysts of varying sizes and hemorrhages are seen.

OSTEOPOROSIS

In this disorder, there is reduction in the bony matrix. But what is present is fully mineralised (whereas in rickets and osteomalacia tissue matrix is formed but inadequately mineralised). The bones become porous and brittle, as in this condition destructive processes exceed the productive in the remodelling of bone. Biochemically, the blood levels of calcium and phosphorus are normal.

Causes :

1. **Senility :** causes not fully known. Probably due to decreased osteoblastic activity or decreased sex hormones (see below).

2. **Lack of protein** as in loss of protein (renal disorders) or decreased production as in liver disease or defective absorption due to intestinal disorders. Protein is essential for the formation of osteoid, without which bone cannot be formed.

3. **Deficiency of vitamin A.**

4. **Deficiency of Vitamin C :** osteoblasts and osteoid are not formed.

5. **Local pressure** on bones may cause atrophy :- for example tumour *Coenurus cerebralis,* hydatid cysts and pulsating arterial aneurysms in contact with vertebrae.

6. **Disuse:** For proper healthy condition of the bone to be maintained exercise is necessary. If a part is immobilised for a long time bone of the part

becomes thinner and porous due to increased activity of the osteoclasts and inctivity of osteoblasts (due to lack of normal stimulus of stresses and strains).

7. **Loss of nerve supply** to the part results in paralysis and so the part cannot be moved and osteoporosis will result.

8 **Deficiency of trace elements**—copper deficiency in dogs, manganese deficiency in pigs and zinc deficiency in fowls.

9. **Hyperthyroidism**: Osteoclastic activity is probably increased.

10. **Hyperparathyroidism**: Increased resorption of bone occurs.

11 **Cushing's Syndrome**: Excess of glucocorticoids probably suppresses the osteoblastic activity. Bodies of vertebrae are severely affected.

12. **Lack of either androgens or estrogens**: In human pathology, osteoporosis is frequently observed after menopause. The sex hormones appear to have some, influence over the osteoblastic activity.

13. **Poisons**—For example lead posioning in sheep and goats causes osteoporosis.

Macroscopically, the bones appear lighter and thinner—atrophied. The cortex is thinner but the marrow cavity is wider. Bones become brittle and so are prone to fractures.

Microscopically, the bony trabeculae are thinner with decreased number of osteoblasts. Osteoclastic activity denotes destruction of bone.

Alkaline phosphatase of serum is normal. Can be diagnosed by Izuka's test.

PULMONARY OSTEOARTHROPATHY

This condition is known as Marie's disease in humans and found in all animals, though of greater incidence in dogs. The lesions are found in the lungs. The bones of the limbs are affected, while the joints are frequently not.

There is formation of new bone, mostly under the periosteum, which is therefore pushed out (Periosteal hyperostoses). As the osteophytic formation is not even, the bony surface is rough. Osteophyte means a bony excrescence. The articular surfaces are free Joints may be swollen due to periarticular proliferation

In the lung, foci of new bone formation are seen.

Causes : The exact causes are not known. This disease is noticed in the following conditions :

1. Chronic disease of heart and lungs—bronchiogenic carcinoma, bronchiectasis, emphysema, chronic tuberculosis and congenital heart disease.
2. When there is interference in the vascular supply to the extremities.
3. Passive congestion of the affected parts.
4. Neoplastic condition of the lungs.
5 *Spirocerca lupi* infection.
6 *Dirofilaria* infection,

It is surmised that anoxia, probably with some obscure toxins, is the causative factor. It is also thought that the skeletal changes are the result of reflex vasomotor disturbances in limbs secondary to circulatory disturbances in the lungs. A familial predisposition is noticed in man.

OSTEITIS AND OSTEOMYELITIS

Inflammation of the bone is called **osteitis**, and that of periosteum is **periostitis** Inflammation of bone marrow is known as **osteomyelitis**. Inflammation of vertibrae is **spondylitis**.

Osteitis and Osteomyelitis may be acute or chronic. Acute purulent osteomyelitis is always caused by bacteria which gain entry into the bone in the following ways.

1 **Direct** :
   (a) through compound fractures
   (b) gunshot and other wounds.

2 **By lymph vessels** in draining neighbouring purulent areas such as :
   (a) purulent arthritis
   (b) purulent periostitis
   (c) gathered-nail wound
   (d) suppurative otitis media.

3. **By blood stream** from a suppurative lesion elsewhere and in pyemia.

This condition is not so frequent in animals as in man and the organisms that cause it are *Pyogenic bacteria S necrophorus, Erysipelothrix rhusiopathiae, Salmonella* and *Cryptococcus neoformans*.

Acute periostitis may be non-suppurative. usually caused by trauma (concussion) and is seen in horses as "sore shins" (due to working on hard roads).

Macroscopically, in periostitis, the usual inflammatory reaction is seen in the periosteum, hyperemia with purulent exudate accumulating between the cortex and periosteum. The exudate may separate the periosteum from the bone and necrosis of the cortex results. Periosteum may be ruptured, liberating the pus into the nearby tissue. Since periosteum is in continuity with the endosteum and medulla,pus may pass on to these structures. In such an event,necrosis of the bone occurs due to separation of both periosteum, and endosteum on which the nutrition of the bone depends.

In suppurative osteomyelitis,pus is found in the medullary cavity and it may burst through the cortex But more often, such a drainage is difficult and the condition progresses to a chronic stage.

In the young growing animals abcesses are found at the chondrocostal joints and in the epiphyseal plates.

The necrosed bone is separated from the healthy bone by the action of osteoclasts and a *sequestrum* is formed. Osteoblasts nearby are active and produce new bone, which forms a case, as it were, around the sequestrum and this is known as *involuctum*. Pus is discharged to the outside from the sequestrum through small openings in the involucrum called *cloacae*.

Sequelae :

1. Pathological fracture due to extensive destruction of the bone.
2. Chronic osteomyelitis.
3. If suppurative osteomyelitis is extensive and present for a long time amyloid degeneration may occur.
4. Resolution and healing with timely treatment.

5. Suppurative arthritis may occur due to extension of infection to the neighbouring joint; metastatic abscesses.

6. Death due to pyemia and septicemia.

Chronic osteomyelitis :

Causes : 1. May be a sequel to osteomyelitis.

2. Repeated injury or concussion, in horses especially.

3. Bacteria—of low virulence—*Actinomyces, Brucella, Mycobacterium tuberculosis, Salmonellosis*

4. Fungi – *Coccidioidomycosis.*

In the case of chronic trauma and concussion **exostosis** results. This is the formation of granulation tissue of the bone, Just as **fibrosis** occurs in chronic inflammation of soft tissues, so also, in the bone, chronic inflammation results in the formation of new bone. Essentially this is a result of chronic ossifying periostitis.

In the horse special names are given to exostoses occurring in certain locations

1. **Ring bone,** if the exostosis is found on the 1st or 2nd phalanx. This is a painful condition causing lameness.

2. **Splint :** Exostosis at the end of metacarpal or metatarsal bones; not usually painful and so no lameness seen

3 **Spavin :** Exostoses on the medial portion of the distal tarsal bones, causes lameness as the bony growth pinches the cunean tendon, which passes over it.

The exostoses or osteophytes have the structure of a compact bone, but do not have haversian system.

The lesions produced by bacteria in chronic osteomyelitis are granulomas. **Microscopically,** in chronic osteomyelitis, centres of pus are surrounded by granulation tissue and inflammatory cells, consisting mostly of mononuclears and a few giant cells. Due to activity of the osteogenic layer of the periosteum new bone is formed and so the shaft is thickened and marrow narrowed—**osteosclerosis** In actinomycosis and tuberculosis there is rarefaction of bone—**rarefying osteitis.** In tuberculosis, there is extensive destruction of bone with the formation of caseous material but new bone is not formed.

FRACTURES

A fracture is a break in the continuity of a bone and is usually due to trauma.

Varieties of the fractures :

1. Simple fracture : Fracture of bone without an opening over the overlying skin.

2. Compound fracture : Fracture with an opening on overlying skin.

3 Comminuted fracture : The bone is splintered into many pieces

4. Impacted fracture : When one fragment of a fractured bone is firmly driven into the other.

5. Greenstick fracture : Here one side of the bone is broken while the other is intact as occurs when a green stick is bent.

6. Pathological fracture : The fracture is not due to trauma only but due to some bone disease existing. Eg. osteosarcoma.

7. Articular fracture : When joint surface of a bone is involved.

8. Depressed fracture : In the skull where the involved bone is depressed below the surface

9 Linear fracture . Here bone is split lenghthwise

10. Transverse fracture : Fracture at right angles to the axis of the bone

11. Multiple fracture : Here are two or more lines of fracture of the same bone but not communicating with each other.

12. Oblique fracture ; Break extends in an oblique direction.

Healiug of fracture :

Along with fracture of bone, there is hemorrhage as the blood vessels near by are torn and ruptured. Moreover, the capillaries of the haversian canals also contribute to the hemorrhage. Because of ischemia. (due to cessation of local circulation) bone cells die and these incite an inflammatory reaction. The accumulated blood clots and in twenty four hours this clot is invaded by fibroblasts and capillaries from the periosteum and is organised. This fibro-vascular tissue is strong enough to keep the two broken ends together and is known as a **soft tissue callus.** (Callus, Latin for a hard substance).

Osteoblasts dervied mostly from the deeper layer of the periosteum invade the blood clot along with the capillaries and within 4 or 5 days trabeculae are formed around central spaces which become Haversian canals. This is the osteoid laid down by the osteoblasts. This osteoid is well formed by the end of second week Osteoblasts are also formed by metaplasia of the fibrous tissue. Later calcium salts are deposited on the osteoid to form bone The newly formed bony tissue unites the two ends of the fractured bone and is known as **provisional callus.**

The callus formed by the periosteum and locatad sub-periosteally is called *external callus.* that present in the medullary region is called *internal callus* and that between the ends of the shaft the *intermediate callus* or *in-line callus*

The callus formed is larger than the outlines. of the bone and so bulges on the periosteal side In the beginning there is no orderly arragement of the trabeculae and haversian systems. Later the provisional callus is removed by osteoclasts and remodelled by osteoblasts into regular bone This is called definitive callus It may take several months for this definitive or hard callus to form. Finally, during the remodelling processes, excess of the callus is removed

If the gap between the two ends of a broken bone is too wide, the fibroblasts of the provisional callus may become cartilage cells by metaplasia and this is later converted into bone—endochondral ossification.

Factors that interfere with healing

1. **Non-alignment of the two ends of the bone :** Due to this, deformity excessive callus formation and displacement may occur

2. **Infection** This is common in compound fracture, leading to necrosis and osteomyelitis, which retard the process of healing

3 **Deficiency of calcium, phosphorus, vitamin D and proteins:** These may occur in dietary deficiency, starvation, metabolic or infectious diteases eg. renal

disease, malabsorption diseases due to gastro-intestinal pathology, parathyroid disorders ; excessive loss of protein as in albuminuria or heavy stomach worm infections.

4. **Presence of foreign bodies** hinder normal and rapid healing. These may be bullets, muscle, fat or clothing.

5. **Fragments of necrotic bone :** This is more common in comminuted fractures where, the necrotic bone acts as a foreign body, producing inflammation and preventing healing.

6. **Inadequate immobilisation : A false joint** or pseudoarthrosis may occur if the fractured ends are not firmly immobilised. The provisional callus is not sufficiently mineralised and so permits bending at the fractured area.

7. **Senility :** In older animals, healing is slow due to decreased vascularity and retarded metabolic processes.

8. **Pathological :** presence of osteodystrophy or neoplasms prevents healing of fractures

Tumors of bones :

Primary : Fibroma, myxoma, lipoma, chondroma, osteoma, chondro sarcoma, fibrosarcoma, osteogenic sarcoma, and Giant-cell tumor

Secondary: Metastatic carcinoma and sarcoma from other parts of ths body.

Chondrodystrophia foetalis (Achondroplasia).

In certain breeds of cattle, the Dexter and Norwegian Telemark, some calves do not attain full development of cartilage growth. So they become dwarfed. The exact cause is not known, though endocrine deficiency is suspected. But the condition is known to be hereditary, transmitted by a lethal factor.

Since endochondral ossification is retarded and even stopped, the bones of the limbs and extremities are shortened. There is brachygnathia and so the animals have a bulldog appearance of the head (Bulldog calf). The skin is thick and much folded due to subcutaneous edema. The calf has a vaulted skull. Growth of the skull being by intramembranous ossification, the head is disproportionately large. Such calves are usually aborted between their third and eighth months of intrauterine life.

JOINTS

Inflammation of the joints is called **arthritis.**

Inflammation of hip joint is called **Coxitis** while that of stifle joint **gonitis.** Arthritis may be acute or chronic.

Acute Arthritis :–

Causes :

1. Contusion or strain in which there is stretching of the joint capsule.

2. **Bacteria :** Routes of infection may be (a) via blood stream (b) by extension from neighbouring tissue and (c) by puncture wounds.

Trauma usually produces a *serous type of* inflammation in which there is increased production of synovia, distending the joint capsule. The condition is mild showing a slight hyperemia of the articular cartilage and the synovial membrane.

Bacterial arthritis may be conveniently classified as :

1. Non-suppurative and
2. Suppurative

In non-suppurative arthritis, there is acute serous or serofibrinous exudate. Causes are :—

Erysipelothrix rhusiopathiae (serous polyarthritis) in sheep and pig
Hemophilus influenza suis in pig.

Macroscopically, exudate contains yellowish flakes, which are often compressed into flat structures which float in the joint fluid The synovial membrane is thickened and studded with hemorrhages,

Microscopically, hyperemia and neutrophilic infiltration are common. Articular cartilage may be eroded.

Suppurative Arthritis: This condition is usually associated with Navel ill. The bacteria localise in the joints because of the rich blood supply there and also probably to the weak defences in that region,

The following organisms are incriminated.

Species of animal affected	Causative bacterium	Nature of lesions
Calf	1. *E. coli*	Septicemia and acute arthritis, cloudy synovia- Many organisms in the joint,
	2. *Corynebacterium pyogenes*	Purulent arthritis with destruction of joint. Organisms may be found in pure culture or mixed with other organisms.
Colt	1. *Shigella equirulis*	Swollen joints
	2. *Streptococci*	Purulent exudate
Sheep	1. *Staphylococci*	Purulent exudate with
	2. *Corynebacterium*	joint destruction
Swine	1. *Streptococci*	
	2. *Brucella abortus*	Purulent exudate

Infection may be

a) Primary—penetrating wounds of joints.
b) Secondary—i) Extension of suppurating process from neighbouring lesions ii) metastatic lesions in pyemia.

Macroscopically, all the symptoms of an acute inflammation are seen notably swelling of the joint. White, yellow or green pus may be present in the joint depending on infecting organisms. In mycoplasma infection it is thin and colorless.

In suppurative arthritis the articular cartilage is destroyed and infection may spread to the underlying bone Suppurative osteomyelitis, necrosis and caries of bone result Particles of disintegrated bone are found in the pus, like grains of sand. Sometimes, the pus may be discharged through a break in the skin

resulting in an open joint. The articular cartilage may be inflammed and eroded. Synovial fluid which is increased, is purulent. There may be inflammation of periarticular tissue.

Microscopically, there is infiltration by neutrophils.

If the condition becomes chronic, there is excessive fibrosis of the joint.

Sequelae: The condition has unfavourable prognosis in young animals. Due to pain, they will not be able to move about and in chronic condition fibrosis and ankylosis of the joint will result.

Chronic arthritis

Causes: Sequel to acute arthritis.

Primary

(a) Chronic traumatic.

(b) Bacterial, Tuberculosis in ox and pig, Fowl Cholera in fowls

Chronic serous arthritis: Due to destruction of the articular cartilage, there may be fibrous adhesion between the two articular surfaces. Subsequently the two bones may fuse together producing **ankylosis** of the joint.

Tuberculous arthritis is characterised by the granulomatous inflammation. It is manifested in 3 forms :

i) Miliary form in which miliary nodules are found in the synovial membrane. The neighbouring tubercles may coalesce and project into the joint cavity as 'pearls'. This form is seen in pigs

ii) Infiltrating tuberculosis is seen in cattle, characterised by diffuse tuberculous granulation tissue, containing epitheliod cells and giant cells (Chronic organ tuberculosis.)

iii) Caseating tuberculous synovitis with caseation but without specific granulation tissue.

In chronic stages of Fowl cholera *Pasteurella avium* may get localised in the joints and tendon sheaths, where there is an accumulation of a cloudy or cheesy material, thus giving the structures a swollen appearance.

Mycoplasmal arthritis in swine

Mycoplasma granularum is a common cause of arthritis in 100 to 200 pound swine. Heavy muscling, genetical background and stress act as predisposing factors.

The disease is usually an acute one, with sudden onset of lameness. The course runs for 3 to 10 days. Subsequently flare ups cause longstanding chronic arthritis.

Macroscopically, in the acute form, there is increased serosanguineous synovial fluid in the femuro-tibial, coxo-femoral, cubital or scapulohumoral joints. The synovial membranes are swollen, hyperemic and discolored but the joint capsule and articular surfaces appear normal.

Microscopically, hyperplasia of synovial lining cells, villous hypertrophy and extensive mononuclear infiltration are noticed.

Degenerative arthropathy Osteoarthritis– deformans

This condition should be distinguished from the conditions of joint resulting from arthritis. In arthropathy, no inflammation occurs initially, but is an

ageing process Normally, the young cartilage is white and translucent. As it ages it becomes opaque, yellowish and less and less elastic. To start with, the cartilage cells undergo hydropic degeneration and fatty changes The fibrils of the cartilage become visible. Subsequently fissures form on the cartilage followed by fibrosis of the ground substance. The cartilage soon becomes separated and eroded, exposing the bone underneath, The older cartilage has lost its power of regeneration and growth and so repair does not occur.

The cartilage being avascular depends on the synovial fluid for its nutrition. So any changes that may occur in the synovial fluid as a result of ageing process may contribute to the degeneration of the cartilage. When ulceration of the cartilage occurs, the bone is exposed and subjected to stress, and becomes polished and hard (eburnation). Granulation tissue grows from the exposed bone of the two articular surfaces and thus fills the articular cavity. This tissue subsequently becomes ossified resulting in ankylosis At the margin or edge of the joint are formed periostitic exostoses.

The synovial membrane becomes fibrous and thickened The villi become thickened, fibrous and long and contain fatty tissue. Occasionally near the area of degeneration, some cartilage cells proliferate and form into small nodules which may be calcified following degeneration. These nodules may become detached into the articular cavity.

Causes are obscure The following are noteworthy :

1. Probably it is an ageing process.
2. Repeated trauma—as in concussion sustained by working horses on hard roads; sprains.
3. Obesity
4. Faulty circulation.
5. Absorption of products of faulty digestion

Ring bone : This is a condition of degenerative arthropathy affecting the inter-phalangeal articulation of horses, resulting in ankylosis and lameness

The articular cartilages may be destroyed resulting in ankylosis due to union of the articular ends of the bones by granulation tissue which becomes ossified. So ankylosing arthrosis results—*articular ring bone*.

More often there is chronic inflammation of the periosteum and the ligamental apparatus due to repeated concussion and this results in **periarticular ring bone**, in which the exostoses occur as a ring round about the ends of bones Sometimes these periarticular exostoses may fuse bridging the joint and fixing it.

Spavin : This is arthropathy of the tarsal joint affecting its distal and medial parts. Ankylosis may result. The condition first starts with degeneration of the cartilages of the second and third tarsal bones. Subsequently other tarsal and metatarsal bones may be involved

The normal white or bluish cartilage undergoes degeneration, becoming opaque and fibrous. It breaks down and ulcerates. Granulation tissue from the exposed bone grows and fuses with that growing from the opposite end When this becomes ossified, the joint becomes ankylosed No periarticular change may be noticed So such a condition is known as occult spavin.

In some cases, the synovial membrane may become thickened due to irritation. The fibrous layer of the articular capsule proliferates and then becomes ossified, resulting in large exostoses, which can be easily seen on the internal and medial aspects of the hock joint. These exostoses pinch the cunean tendon and so pain may be caused, resulting in lameness.

Ankylosing spondylosis : In this condition, the small vertebral articulations become ankylosed.

In the dog it is due to the protrusion of the nucleus pulposus irritating the periosteum and the ventral spinal ligament, resulting in exostoses which may subsequently fuse and join the vertebrae.

In bulls also this condition is met with in those that are used for stud. Due to frequent trauma attendant on their work, there is constant irritation. The lumbosacral region is more often affected causing paralysis or ataxia.

Protrusion of inter-vertebral discs

This condition is met with in man and dog.

Normally, the intervertebral disc consists of a central *nucleus pulposus* which is semisolid mucoid connective tissue. This is enclosed in a thick fibrous covering, the *annulus fibrosus*. Due to violent trauma and degenerative changes in senility, there may be a rupture in the annulus, from which the nucleus pulposus escapes and becomes displaced. The susceptibility of the disc to degeneration is inherited. Usually two forms of displacement occur: (i) dorso-lateral prolapse of the nucleus pulposus into the spinal canal. (ii) ventral prolapse beneath the spinal ligaments. In this variety due to formation of osteophytes ankylosng spondylosis results.

In the chondrodystrophic breeds (Dachshunds, Pekingese, French bull dogs), at a very early age, the nucleus pulposus becomes cartilagenous, which later becomes degenerated and calcified. So the nucleus pulposus, which is normally a gel and so able to withstand shocks and transmits pressures uniformly to the annulus fibrosus, becomes transformed into a cheesy mass which crumbles easily. This material transmits pressure to localised portions of the annulus, which also undergoes degeneration. Its lamellae become hyalinised and later split.

In other breeds, the above changes occur in mid or later life.

The displaced nucleus pulposus presses upon the spinal cord producing nervous lesions. The protrusion of the disc may occur at any level, but occurs more frequently in the lumbar region or in the posterior thoracic region. Complete paralysis of the posterior region may be noticed. Pressure on the spinal cord may produce hemorrhage and necrosis in the involved area. Wallerian degeneration of the nerves may be noticed in the spinal nerves arising from the affected region as well as demyelination of nerve tracts

Symptoms include: (a) pain with exaggerated reflex movements which may be intermittent or occur over long or short periods (b) Partial paralysis of the limbs (c) Violent reaction to stimuli—spastic type. (d) Rapid progressive paralysis and early death due to respiratory failure.

BURSITIS

Inflammation of the bursa over the joint is bursitis. This is of frequent occurrence in animals. Examples of these are :

Hygroma of the carpal joint in cows and ' capped elbow or hock joints of horses.

Causes :

1 Trauma, especially, if repeated.

2. Over-use

3 Infection (brucella infection in cows produces Hygroma, and in horses "Fistulous withers." and "Poll evil").

Macroscopically, the inflammation may be serous, serofibrinous or purulent. Trauma produces serous type and one example is the serous bursitis of hock joint in the horses. This is called Bog spavin. Here the joint is filled with serous fluid

Pole evil is the inflammation of the bursa between ligamentum nuchae and atlas and axis

Fistulous withers is the affection of bursa between the ligamentum nuchae and the thoracic spines. The inflammation is a suppurative granulomatous one in which fistulae open on the surface of the skin

Causes may be traumatic, parasitic *(Onchocerca cervicalis)* or *Brucella abortus* and *Actinomyces bovis*. The suppurative and granulomatous reaction is attributed to the two organisms, infection occurring hematogenously

Navicular disease : This is bursitis and arthritis involving the distal sesamoid or navicular bone in the horse. Usually the fore limb is affected:

First there is serous inflammation of the lining membrane of the podotrochlear bursa, with hyperemia. This is followed by erosion and ulceration of the articular cartilage, over which the flexor tendon passes. Due to the changes in the cartilage, the tendrills of the tendon become frayed and ultimately rupture of the tendon may occur. Later the bone is inflammed, becomes rarefied and may fracture.

Infectious synovitis : This is a chronic disease of chicks. Morbidity and mortality are low. The characteristic lesion is a purulent synovitis of the leg joints

Causes : *Mycoplasma synoviae* and *Mycoplasma gallisepticum.*

Infection occurs in chicks, 12—14 weeks of age, by ingestion and the incubation period is 24 to 80 days.

Symptoms seen are :— emaciation, retarded growth, pale, comb, distended hock joints and swollen foot pads and lameness.

Macroscopically, in the early stages is found a creamy exudate in the synovial membrane of the joints, especially those of the hock and foot . This material becomes caseous as the disease progresses The surface of the affected joints becomes yellow or orange.

In the early septicemic stage, the spleen, liver and the kidneys may be swollen.

Microscopically, brain may show gliosis and degeneration of purkinje cells. In the liver and spleen there is proliferation of the reticular cells of the reticuloendothelial system. Bile duct proliferation may also be seen.

With timely treatment using antibiotics the disease can be cured.

Sometimes a focal infiltration by mononuclears and necrosis of the myocardium and fibrinous pericarditis may by noticed. Thymus and bursa af Fabricius may be atrophied due to degeneration of lymphoid tissue.

DISEASES OF SKELETAL MUSCLE

Atrophy :

Etiology :

1. **Senility :** In old age there is gradual atrophy of all muscles. But cattle and swine are slaughtered young and so atrophy is not seen in them. In dogs, milch cows, horses and ewes which are allowed to grow old, atrophy may be observed. It is likely that there may be under nutrition or the animal is not able to metabolise available nutriments in old age. The muscle cells are not able to assimilate the nutrients and so catabolism exceeds anabolism.

2. **Disuse :** This is seen in fractures of bones when the parts are immobilised for long periods and the muscles therefore are not utilised. Disuse of the limb may also occur due to pain as in rupture of a tendon, acute arthritis, ankylosis and diseases of the bones and muscles.

3. **Starvation :** Sufficient food is not available to make up for the catabolism that takes place.

4 **Atrophy of wasting diseases, cachexia and malnutrition :** In chronj wasting diseases like tuberculosis and Johne's disease; in debilitating conditions like neoplasia; in cachexia and in malnutrition, the food digested is either not effectively metabolised or is not used by the body and so atrophy results.

5 **Denervation :** When a nerve is injured or severed, the muscles supplied by it become paralised and atrophied. Examples are; a) Atrophy of laryngeal muscles when the recurrent laryngeal nerve is injured (Roaring). b) Atrophy of supraspinatus muscles when the suprascapular nerve is injured. (c) Atrophy of muscles in lesions of the central nervous system—poliomyelitis, protrusion of intervertebral disc, tumors etc.

6. Pressure; Continuous pressure on the muscle, producing ischemia locally and interfering with movement will cause atrophy The cause of pressure may be tumors, abscesses, cysts, ill-fitting, collars and saddles as well as infiltrating lymphoid cells in neoplasia of these cells.

Macroscopically, the muscle which is normally pink, loses this color and turns pale, grey or brown. It is firmer due to replacement by fibrous tissue. Due to uneven atrophy of different muscles, disfigurement may occur. Skeleton becomes prominent.

Microscopically, the size of the muscle fibres is reduced. Sarcoplasm may become so reduced and in some places may even disappear, that the sarcolemmal nuclei become prominent. There may be deposition of "wear and tear" pigments at the poles of nuclei giving the muscle a 'brown color—'brown atrophy' The cell nuclei may proliferate and fill the empty sheath.

As the etiological factors for atrophy also cause degenerative and necrotic changes, cloudy swelling, fatty degeneration and coagulative necrosis may be encountered.

In later stages there may be infiltration of fat in some areas *(atrophia lipomatosa)* and fibrosis in others.

Inflammation of muscles :

Myositis : Inflammation of the muscle is called myositis. This may be acute or chronic.

The routes of infection and causes are :

1) Trauma. 2) By direct extension from lesions of neighbouring arthritis, osteitis or periostitis. 3) In pyemia, hematogenously. 4) By parasitic infection.

Acute myositis may be non-suppurative.

Best example of acute non suppurative myositis is Black quarter in cattle and sheep. In this condition, the organisms, *Clostridium chauvoei* causes inflammation and necrosis of the muscles with production of gas. The muscle fibres are torn by the gas bubbles. Local hemorrhage is present and the area is black due to formation of black iron sulphide. Regional lymph nodes are actively congested and parenchymatous organs show fatty changes. Serous cavities contain blood stained serous fluid.

Microscopically, there is necrosis of the muscle, infiltration by neutrophils and clumps of the anaerobe.

Suppurative myositis : Hematogenous infection may occur from other foci as in Strangles and Glanders. Infections may occur in lacerating and penetrating wounds or by extension from adjoining areas.

The usual changes of suppurative inflammation are found, viz. abscess or phlegmon Microscopical appearances are typical of any other suppurative inflammation with a great outpouring of netrophils. The muscle fibres undergo liquefaction following coagulative necrosis.

Sequelae : As there is loss of muscle tissue, healing is by means of fibrous tissue proliferation and scar formation. If severe, septicemia may result.

Chronic myositis : Examples are found in infections by Actinomycosis and Actinobacillosis. The muscles of the tongue, cheek and throat are affected The lesions consist of chronic suppurative myositis in which the "Sulphur granules" are noticed in a mass of inflammatory granulation tissue. There is infiltration by large number of lymphocytes, neutrophils and plasma cells. Muscle fibres are destroyed.

Parasitic myositis : The following parasites are found to infect the muscles of animals.

i) **Toxoplasma.**

ii) **Trichinella spiralis** is found in man, pig and other animals. The larvae are encysted in many muscles, especially those of diaphragm, intercostal muscles and tongue. The cysts are parallel to the muscle fibres which undergo granular degeneration of the sarcoplasm. Intense infiltration by eosinophils, plasma cells, histiocytes and lymphocytes occurs. Sarcolemmal nuclei proliferate. The encysted larvae may be alive for as long as 20 years.

iii) **Sarcosporidia** are present in the skeletal and cardiac muscles of all species of animals. No specific disease has been attributed to these parasites though light infections cause no perceptable symptoms, heavy infections may

be responsible for lameness, weakness, paralysis, emaciation and sometimes even death. Parasitised muscle fibres are destroyed by the parasite and the adjacent cells undergo pressure atrophy.

iv) Cysticercus, (Measles).

a) **Cysticercus cellulosae :** The bladder worm of *Taenia solium*, a tapeworm of man, infects the muscles of pig The muscles of the shoulder, neck, diaphragm, tongue, intercostals, abdominal and cardiac muscles are affected. Heavy infection may result in fatal anemia and cachexia.

b) **Cysticercus bovis** is the intermediate stage of tape worm *Taenia saginata* of man, found in the muscles of cattle. All muscles may be a affected but especially those of tongue, mastication and heart are more often infected.

c) **Cysticercus ovis** is the intermediate stage of dog tape worm *Taenia ovis* and is found in the muscles of sheep.

Diseases of unknown origin

Eosinophilic myositis :

A: Cattle and Sheep :

Very rarely,at slaughter, yellowish green areas may be noticed in the lingual, oesophageal, cardiac and diaphragmatic muscles of cattle and sheep.

The green color fades on exposure to light.

Microscopically, large numbers of eosinophils, histiocytes, plasma cells and lymphocytes, are found between the muscle fibres and in tissue spaces. Though extensive degeneration of the muscles may not be noticed, in some places necrosis and invasion of muscle fibres by the eosinophils are observed. In more chronic cases fibrosis is evident.

There is some suspicion that the condition may be a manifestation of allergy and sarcosporidia are mentioned in this connection as the sensitising factor.

B. Dogs.

Clinically, the condition involves the masseter, temporal and pterygoid muscles chiefly. Other muscles may be affected. The muscles of mastication are enlarged bilaterally so that opening of the mouth is painful and mastication is interfered with. The eyes bulge out, resulting in keratitis and corneal ulceration, since eyelids cannot close completely. German shepherds and Alsatians are more often affected. As the animals cannot eat, they die finally, of inanition.

The local tonsillar and mandibular lymph nodes are also swollen. Blood picture reveals high eosinophilic count upto as much as 90%. Temporary remissions may occur but are followed by repeated attacks and the animal finally dies.

Macroscopically, the affected muscles which are swollen and hard to the touch, show grey and red streaks and white and yellow spots Hemorrhage is present The regional lymph nodes are congested and swollen.

Microscopically, there is heavy infiltration of eosinophils, lymphyocytes, plasma cells and macrophages into the muscle, producing atrophy, and hyaline necrosis, vacuolar degeneration and lysis of muscle fibres. Hemorrhage is common. The necrotic muscle is removed by macrophages and fibrosis follows. In the liver is found periportal lymphocytic infiltration.

Cause : Unknown. Some kind of allergy is suspected... Probably a nutritional problem, Vitamin E deficiency, may have something to do with the condition

White muscle disease : Stiff-lamb disease.

This is essentially a coagulative necrosis of the muscles due to various causes. The disease occurs in calves and lambs and can be produced in rabbits and guinea-pigs. The clinical picture is classified into three main types.

1 **The stiff type ;** The head is carried low and has a drooping posture. Animal experiences difficulty in rising and walking. While walking, the gait is stiff The weight-bearing and active muscles, for example, muscles of the croup and quarters, diaphragm, heart and intercostals, are mostly affected In lambs this is the form encountered and the animals are always recumbent and do not like to move. On forcible movement, they have stiff gait and wobble. (Stiff lamb disease)

2 **The respiratory type :** Here the muscles of respiration (diaphragm and intercostal muscles) are affected and the animal may show symptoms of respiratory distress

3, **The cardiac form :** In this type, animals show considerable weakness, inability to stand, rapid pulse and low blood pressure. Since the heart is affected and weakened, exertion brings on respiratory distrees and even death. In animals with cardiac involvement alone, sudden death occurs without any other symptoms.

Macroscopically, those muscles, which are continuously active, viz , diaphragm and intercostal muscles, show the changes. The muscles are bilaterally affected and are pale like fish flesh The whole muscle bundle may not be affected but only a part of it will show the change. The muscles become hard and wooden. The paleness is due to loss of myoglobin which is excreted in the urine. The change in color is also due to changes in optical characterirtics of the muscle protein when it becomes coagulated. Pneumonia, edema, hydrothorax, C.V.C of liver and hydropericardium, will be found when heart is involved. Heart shows yellowish or grey streaks or patches. The left ventricle is more often affected

Microscopically the muscle fibres are swollen with loss of striation and with widespread hyaline degeneration. This progresses to coagulative necrosis. Fibres are fragmented and may completely disappear. Marked sarcolemmal proliferation is present Some fibres may be calcified, infiltration by macrophages and lymphocytes is seen. Similar lesions may be found in the heart. In some places healing by fibrous tissue is evident.

One noteworthy feature is that the nerves and C. N. S, are normal without showing lesions.

Clinically the serum glutamic oxaloacetic transaminase (SGOT) level will be more than 300 units while the normal is less than 100 units.

Causes :

1. **Vitamin E deficiency :** Vitamin E is an antioxidant and in its absence oxidation in the muscles is increased to 400 times the normal and so degeneration and necrosis occur.

Vitamin E deficiency may occur in the following manner.

a) Dietetic deficiency.

b) Feeding too much of cod liver oil. The unsaturated fatty acids in th 4od liver oil antagonise Vit E.

2. **Selenium deficiency :** Selenium is required in minute quantittes. In its absence muscle necrosis occurs. Selenium deficiency can occur in the following manner.

a) Deficiency in the soil;animals that are grazed on fodder grown on soils deficient in selenium suffer from the diease.

b) Excess of sulphur, used in fertilsers, inhibits the uptake of selenium by plants,

3. **Vitamin B deficiency :** It is found that Thiamine deficiency, especially, produces cardiac necrosis. Deficiency can occur in animals when the ruminal flora are not active to synthesize the vitamin as occurs in cobalt deficiency.

4 **Abnormal ruminal fermentation :** Some toxic products are probably produced in the rumen that cause muscle necrosis

5. **Deficiency of choline** produces muscle necrosis in rabbits (experimentally)

6. **Vitamin A deficiency :** Probably vitamin A deficiency produces this disease in swine

7. **Multiple deficiencies :** Lastly, in starvation as occurs during drought and malnutrition, multiple deficiencies of vitamins and minerals (phosphorus) may occur and muscle necrosis may be encountered. Similar lesions are seen in hypothyroidism.

AZOTURIA (equine myoglobinuria, Monday-morning sickness; Paralytica hemoglobinuria)

This disease in the horses, literally means "nitrogen in the urine."

Azoturia is found to occur suddenly in horses going to work after complete rest for a few days but maintained on full work-rations. The animals suddenly stop, sweat, shiver and show great suffering from pain in the lumbar region. The affected muscles, which are those of gluteal, lumbar and femoral regions, are swollen and board-like. Soon the animal passes coffee colored dark-brown or black urine since it contains large quantities of mygolobin. Animals lie down and soon die. Those that survive, are weak and it takes a long time for them to recuperate and for the atrophied muscles to regain their normal state.

Pathogenesis

In normal muscle contraction, muscle glycogen is converted into pyruvic acid ($CH_3COCOOH$) Due to inadequate oxygen, only 1/5 of this is oxidised to CO_2 and H_2O to liberate energy. The rest is converted into lactic acid ($CH_3CHOHCOOH$) which is converted into glycogen in the liver and used again When the animal is at rest but well fed, the muscles are well stored with glycogen. When it is put to work suddenly much of this glycogen is converted to lactic acid in the muscles and large amounts of this stimulate extreme contraction of the muscles, which become hard (board-like) In the contracted state of muscles, blood circulation is poor and so oxygen supply is reduced. Under this hypoxic condition more of lactic acid is formed (from pyruvic acid) which

still further contracts the muscles and so greater curtailment of blood flow occurs leading to still greater reduction of oxygen supply.

Thus a vicious circle is established, the net result being that the muscles do not get sufficient amount of oxygen and nutrition, and so necrosis results. Necrosed muscle liberates myoglobin which is excreted in the urine. Large masses of myoglobin in the urine appear to produce renal blockade, renal ischemia and lower nephron nephrosis, wherein the epithelium of the distal convoluted tubules as well as that of Henle's loops are degenerated, some of which become necrosed and desquamated. Renal vasoconstriction, that may be caused by the same factors responsible for the hemoglobinuria, produces renal ischemia. This condition causes degenerative changes in the tubules and so anuria and fatal uremia result

Macroscopically, the affected muscles are swollen, pale and have increased amount of interstitial fluid.

The affected kidneys are swollen and on section the cortex is brownish and medulla has reddish streaks.

Urine shows granular reddish casts and a few hyaline casts.

Microscopically, the changes in the muscle are those of Zenker's degeneration, in which the muscle becomes a homogeneous hyaline mass without striation The fibres may be fragmented There may be disappearance of all the constituents of the muscle fibre excepting the sarcolemma and fibrous stroma This is the cause of atrophy noticed in surviving animals. In animals that survive regeneration may occur, but it is a very slow process

In the kidneys, lesions are found mostly in the tubules. The epithelium of the proximal tubules may be degenerated and all stages from cloudy swelling to necrosis are encountered Desquamation of epithelium occurs. Similar changes may be noticed in the epithelium of Henle's loops and distal convoluted tubules. The lumens of the tubules may contain, besides the desquamated cells, masses of myoglobin. These form granular pigmented casts. A few hyaline casts may also be found.

Death is due to renal insufficiency leading to uremia.

TUMORS

Primary:

Rhabdomyoma, rhabdomyosarcoma, lipoma, liposarcoma, fibroma, fibrosarcoma; myxoma

Secondary

Metastases of carcinoma and sarcoma, are not frequent since muscle does not afford a suitable 'bed' or 'soil' for them to grow. The following may be found occasionlly:

Lymphosarcoma, adenocarcinoma, melanoma, and angiosarcoma.

CHAPTER 22

THE ENDOCRINE GLANDS

(Revised by Dr. A. Rajan)

The endocrine glands are ductless glands and are of vital importance to the body since, through their hormones, they act as chemical regulators. These glands have widespread and specific influence on various processes connected with metabolism, growth and reproduction. For example, the parathyroids control the metabolism of calcium and phosphorus, the thyroid that of iodine, adrenal cortex that of sodium and pancreas (Islets of Langerhans) that of carbohydrates. The anterior pituitary controls the growth. Gonads and the anterior pituitary control repoduction.

General outline of hormonal relation :

The anterior pituitary controls the activities of most of other glands and is called "the conductor of endocrine orchestra". Through neurohumoral pathways via the hypothalamus, the anterior pituitary is connected to the central nervous system and so to a certain extent, the endocrine system is under nervous control. In most cases, the anterior pituitary doses not act directly on tissue cells but through the mediation of another endocrine gland which is called "Target gland". The hormones affecting these target glands are "tropic" in character. Direct influence of tropic hormone on tissue cells is exemplified by the action of lactogenic hormone of the anterior pituitary on mammary glands in which milk secretion is stimulated. (Trophic is also spelled as tropic).

Adrenal medulla has no tropic hormone for its stimulation, but it is under the direct control of central nervous system, eg liberation of adernaline in fright.

In the case of parathyroids, neither tropic nor nervous influences are present since they respond directly to the body tissues. eg. hyperplasia of parathyroids in hypocalcemic states.

The hormones of the target glands have an inhibiting action of the anterior pituitary. This is a "*feed back mechanism*" or *servomechanism* This may be brought about by the action of the hormones of the target glands directly on the pituitary or indirectly through inhibition of the "releasing factors" in the hypothalamus. Hypothalamus stimulates the anterior pituitary through certain factors called 'relasing factors' to produce the corresponding hormones by it For example, corticotropin releasing factor or harmone for ACTH (CRF), thyrotropic hormone releasing factor (TRF) and similar releasing factors for STH, LH and FSH Hypothalamus may be stimulated for the release of the factors by the hormones of the target glands or by stimuli arising from higher centers. This servomechanism acts as a safety valve so that over-production of tropic hormones is prevented and thus the energy metabolism is kept under control. Therefore, continuous administration of an excess of target gland hormone will produce atrophy of the corresponding tropic-hormone-producing gland.

Hypothalamus produces some inhibitory hormones like prolactin inhibiting harmone (PIH) in mammals and growth hormone inhibition hormone otherwise called SOMATOSTATIN

Compensatory atrophy : This may be seen in a gland if there is prolonged administration of its own hormone: eg. tumor of one adrenal will produce atrophy of its pair.

Exhaustion atrophy : If there is continuous stimulation of a target gland by its tropic hormone, exhaustion atrophy of the stimulated gland occurs (after an initial period of hyperplasia and over-production by this gland).

An animal may be affected by diseases of the endocrine glands and such disease processes may be the result of :

1. **Hypofunction** of the gland. This may be due to :
   (a) congenital hypoplasia
   (b) destruction of the tissue by
      i) Disease processes ; inflammation, tumors
      ii) Atrophy—of unknown origin.
      iii) Thrombosis and embolism.

2) Hyperfunction :
   i) Tumors
   ii) Non-neoplastic hyperplasia.

THE THYROID

Thyroid is situated in the cervical region, and consists of two lobes connected by a narrow isthmus. The gland is composed of follicles (the thyroid follicles) which are closely apposed to each other. These are supported by a delicate connective tissue stroma. The gland is provided with a rich blood supply, A few lymphocytes are present.

The follicles are lined by a layer of epithelial cells. The gland is very labile. That is to say, it is susceptible to various influences and demands for thyroxine, and hence the histological structure varies with function, breed, age, season, metabolic activity etc.

At stress, when thyroxine is most needed, the epithelium assumes a tall columnar shape and the colloid is resorbed But at rest, the colloid accumulates and the epithelium assumes its normal size and shape (involution).

The anterior pituitary through its thyrotropic hormone controls the function of the thyroids as follows : (1) The thyrotropic hormone stimulates the trapping of the iodides (2) Two iodinated tyrosine molecules are coupled to form the tetraiodotyrosine under the influence of the thyrotropic hormone. (3) Thyroxine after formation is combined with a protein to form thyroglobulin (molecular weight 680,000). This compound is hydrolysed by protease, when needed, under the influence of the thyrotropic hormone and thus free thyroxine is liberated into the blood.

Hypothalamus, which is the controlling center for emotional activity releases thyrotropic hormone releasing factor (TRF) which acting on the anterior pituitary causes the production of the thyrotropic hormone. Thyroxine may inhibit production of the thyrotropic hormone by "feed back mechanism" or "servomechanism". This check is needed, for otherwise, due to continued stimulation of the gland, exhaustion atrophy of the thyroid may result. Emotional disturbances through hypothalamus may play a part in the production of thyroid hyperactivity. (See exophthalmic goitre later).

Goitrogenic substances : In the synthesis of thyroxine, iodine must first be absorbed by the gland as iodide, which is later converted into an organic compound by combination with tyrosine. Those substances, therefore, that inhibit these two processes, viz. uptake of iodide by the thyroid and its subseqent conversion into an organic compound, prevent the formation of thyroxine. A low level of thyroxine is a stimulus for the production of thyrotropic hormone which causes hypertrophy and hyperplasia of the follicular epithelium. This is manifested as goitre. So these substances that are responsible for the production of goitre are called goitrogenic substances. Examples of these are :—Phenothiazine; thiocyanates, cabbage, linseed, soya bean, sulphonamdies.

The uptake or trapping of iodine by thyroid is blocked by thiocyanate and perchloride while thiourea, sulphonamides and phenothiazine may prevent or inhibit the synthesis of thyroxine. So these substances are known as *antithyroid substances*. Ultimately these substances may also produce goitre.

Functions of the thyroid and thyroxine.

The function of the thyroid through its hormone thyroxine, is to maintain a high rate of metabolism in the animal (that means, it raises the basal metabolic rate, BMR). The rate of metabolism is affected by the hormone acting at one or more points in the citric acid cycle. Since the enzymes that are involved in this cycle are located in the mitochondria, it is postulated that it increases the permeability of the cell membrane or the membranes of the mitochondria. The hormone increases also the number of mitochondria. So more metabolites are exposed to more enzymes of the mitochondria, thereby increasing the metabolism.

When thyroxine is administered, a number of enzymes, especially cytochrome oxidase, cytochrome C and succinic oxidase increase in the tissues. Oxygen utilisation is stimulated. Thyroxine causes increased utilisation of carbohydrates, increased catabolism of proteins and increased oxidation of fats as shown by loss in weight. Protein synthesis is enchanced by thyroid hormones, through the mediation of mitochondria.

Central nervous system requires thyroxine. If it is deficient the nerves are permanently damaged in the young growing animal and so it is lethargic, dull and stupid. Increased thyroxine stimulates the activity of central nervous system and so the animal is jumpy, nervous, irritable and hyperactive.

Thyroxine causes hepatic glycogenolysis and so hyperglycemia is produced. It also affects the normal reproductive functions. In hypothyroidism, androgens may not be produced and so thyroxine is required for libido. In the female the litter size may be reduced, milk production is lowered and cysts form in the ovary leading to sterility. It is necessary for estrus and so in its deficiency, silent heat occurs.

Lower environmental temperature causes release of the thyrotropin releasing factor (TRF) of the hypothalamus and so thyroxine is released which acting on the thyroid causes thyroxine production. This raises the basal metabolic rate and so temperature of the body is raised.

Therefore, thyroid controls the body metabolism and so is necessary for proper physical, sexual and mental development and function.

Cysts : Cysts may be ultimobranchial duct cysts or thyroglossal duct cysts or parenchymal cysts. The former are seen either attached to the thyroid gland or embedded in the gland and are seen in the dogs The Cyst contains clear fluid and histologically the presence of ciliated columnar epithelial lining differentiates it from parenchymatous cysts

Atrophy : This may occur secondary to lesions in hypophysis or due to certain unknown causes. Glands become smaller. Sections show fatty metaplasia of interstitial tissue Follicles become smaller and they are filled with basophilic colloid and corpora amylaceae. Connective tissue stroma is prominent and there is condensation of reticular fibres

Hypothyroidism : This occurs in areas where there is incidence of endemic goitre due to deficiency of iodine in the water and soil. Due to deficiency of thyroxine, the basal metabolic rate is abnormally low and so all the vital processes are slowed down The condition found in young growing children is called cretinism.

Cretinism may be sporadic or endemic.

Sporadic cretinism occurs in young of healthy parents. There appears to be a genetic defect by which there is an inability to produce thyroid hormone. Low blood level of thyroxine stimulates pituitary to produce thyrotropic hormone in excess with resultant formation of goitre.

Endemic cretinism occurs in areas where the incidence of goitre is common in man and animals (due to iodine deficiency).

Symptoms in man : The cretin is a dwarf physically, sexually and mentally. Growth is arrested, bones are brittle, abdominal muscles are flabby leading to pendulous abdomen; skin is dry and cold; lips and face are swollen (mouth is half open always) and the tongue is large. The patient is extremely lethargic and has a vacant, idiotic look and the gonads are ill developed. "What was intended to be created in the image of God has become what has been called the pariah of nature, and all for want of a little iodine" (Boyd)

Calves usually are either born dead or die within a day or two Animals have myxedema and alopecia, The fetal placenta is retained. There is swelling of the throat (goitre) and this may be so large that fetal dystocia may develop. Growth of cranial, body and limb bones is arrested Eruption of teeth and second dentition are retarded. Deafness, idiocy and hypoplasia of the pituitary may be noticed. Similar symptoms are seen in colts, lambs, piglets and kids.

The young pigs show deficiency of hair, cyanosis of skin and subcutaneous tissue, edema of skin, shortening of neck (thick neck) and limbs.

Hypothyroidism in adults is called **myxedema** and occurs in man at about the age of 40 years and is more frequently encountered in females.

Causes . 1) Thyroidectomy. 2) Following an earlier severe hyperthyroidism. 3) Atrophy and fibrosis of unknown etiology. 4) Sequel to thyroiditis 5) Radio iodine therapy. 6) Hypopituitarism—decreased production of thyrotropic hormone.

Clinically, the patient is lethargic, heavy and has no inclination to move. She is cold and feels cold. The skin is dry and rough and hair is lost. The face is puffed up and broad. The basal metabolic rate is low and heart rate lower than normal (bradycardia). Serum cholesterol level is high. In the subcutaneous and other connective tissues, there is an accumulation of mucoid or myxomatous substance, which gives the puffed up appearance to the face. Females are frigid and become sterile while males are impotent The thyroid gland is atrophic and hard. In some places it is just a mass of fibrous tissue. The glandular parenchyma is widely separated by fibrous tissue. Lymphocytes infiltrate the tissue and may also form, in some places, nodules.

Myxedema does not appear to occur in animals other than dogs, in which the symptoms are obesity, alopecia, thick skin and lethargy. Serum cholesterol level is very much increased, but serum protein-bound iodine is low.

Hypothyroidism in cattle is manifested by sluggishness with agalactia, silent heat, retained placenta, still births and a tendency to purulent endometritis.

In general, we find the following changes in hypothyroidism

Decrease in : BMR; oxygen utilisation in the liver, kidney and muscles; number of mitochondria; blood flow; cardiac output; blood pressure; cardiac

rate (so bradycardia); nervous function and myelination (so animal is dull, stupid, sluggish and sleepy); gut motility; absorption of glucose; phagocytic activity of leucocytes; serum proteins (so myxedema develops due to decrease in colloidal osmotic pressure of blood); egg production in fowls.

Increase in ; body weight; circulation time (due to weak heart) susceptibility to infection.

Other changes noticed : constipation; hypophagia; weakness and hypotonia of muscles; skin is dry and brittle; loss of hair; thickened skin; dermatitis and retarded feather development

GOITRE

Goitre is non-inflammatory and non-neoplastic enlargement of the thyroid.

I. Parenchyamatous goitre.: This is also called the hyperplastic goitre or goitre of cretinism and is congenital. This condition occurs in areas the soil of which is deficient in iodine and this results in iodine deficiency in animals and man of the locality As explained under cretinism, there is increased production of thyrotropic hormone with resultant hypetrophy and hyperplasia of the thyroid epithelium

Macroscopically, the affected thyroid gland is enlarged, meaty, and firm. In very severe cases, cysts may be present The color is darker than normal due to increased vascularity

Microscopically, hypertrophy of the follicular epithelium is seen to start with. The cells become tall and plump and so encroach into the lumen that is either reduced in size or even obliterated. So there is reduction in colloids Later, there is hyperplasia of these cells and so one finds papillary projection into the lumen, completely filling it. There may be formation of new follicles in the midst of the newly formed clusters of the epithelial cells. Later when exhaustion phase supervenes, the epithelial cells are degenerated and desquamated and fibrosis occurs. Himalayan goitre is characterised by prolonged hyperplasia and mild involutionary changes. There is pronounced stromal hyperplasia and when there is involution the stromal reaction persists indicating previous hyperplasia.

Microscopic picture may simulate a papillary carcinoma but in the absence of anaplasia of the cells and invasion of the basement membrane this can be ruled out.

II Colloid goitre :

This is also called *simple goitre* and is the more frequently seen form among animals, especially dogs. The thyroid glands are much swollen.

Causes : 1. Low levels of iodine in soil and water. 2. Excessive demands- adolescene, pregnancy. 3. Diseased conditions interfering with assimilation of iodine—gastro - enteritis. 4. Ingestion of goitrogenic substances; thiouracil, soya beans

The glands are enlarged Cut section is transluscent.

Microscopically, the acini are widely dilated and contain watery, faintly staining colloid. The epithelium is flattened Colloid is deficient in iodine and thyroxine. Sometimes neighbouring acini may coalesce to form cysts Leakage of the colloid into the interstitial space may occur due to rupture of cysts.

Since this condition is supposed to be an involutionary phase of hyperplastic goitre, here and there some acini may show papillary projections of the epithelium.

Usually no other symptoms except difficulty in swallowing and dyspnoea due to pressure of the enlarged gland is noticed. This is not congenital.

III Nodular or adenomatous goitre:

Nodules of varying sizes, microscopic to several centimeters in diameter, are found very often in old and senile horses, dogs and cattle. Some consider that this is an outcome of alternating hyperplasia, hypertrophy and involution affecting the gland. On section, the nodules are translucent and may contain cysts or vesicles filled with gelatinous colloid.

Microscopically, the picture is variable. All gradations may be found in different nodules, from the picture of a colloid goitre to that of hyperplastic goitre. But in a nodule, the picture is constant. In some nodules dilated acini filled with colloid and having flattened epithelium may be present In other places, acini containing papillary projections of the epithelium obliterating the lumen, may be found Retrogressive changes, leading to necrosis with subsequent softening and liquefaction are responsible for cyst formation (*pseudocyst*). The connective tissue which is increased undergoes hyalinisation. Calcification (*calcareous goitre*) and metaplasia into bone (*osseous goitre*) of the connective tissue may also be encountered.

Cystic goitre results due to formation of the cysts by the confluence of smaller colloid-filled follicles. These are lined by epithelium. (Pseudocysts are just spaces without an epithelial lining).

Hemorrhage into follicles due to erosion of vessels causes *hemorrhagic goitre*. The conglutination of the fibrin produces *rubber colloid*.

Is nodular goitre neoplastic? This question has not been conclusively settled. Some consider the nodules as adenomas while others as a hyperplastic involution process only. The regularity of structure in the nodules and want of evidence of expansion by absence of compression of the adjacent tissue make one consider goitre as non-neoplastic.

In some cases, the nodules are inert and functionless. But in some others thyroid hormone may be actively secreted when toxic adenoma is applied to the condition. This condition is met with in horses and dogs with signs of hyperthyroidism

A hereditary predisposition (*endogenous factor*) to goitre is suggested. But there should be some exogenous factor (like deficiency of iodine etc.) to precipitate the overgrowth Sexual excitement and pregnancy are also factors to be considered. Fluorine containing compounds have goitrogenic effects.

VI Exophthalmic goitre (*Grave's disease, Basedow's disease*) Primary thyrotoxicosis

This disorder of the thyroid gland found in man is probably not seen in animals. Women are more often affected than men.

Causes: The exact cause of this condition is still obscure. Iodine deficiency is definitely not a cause Some kind of shock, probably psychic, connected with sex, is suggested as being causative. There is genetic predisposition of

the condition. Lesions of the anterior pituitary is suggested by some. A long acting thyroid stimulator (LATS), probably a gamma globulin, for the formation of which lymphocytes and plasma cells may play a part, is suggested to be of etiological significance by some workers.

Clinically there may not be enlargement of the thyroid. The following are the characteristic symptoms: 1. Exophthalmus 2. Tachycardia 3 Muscular tremors 4. High B. M. R. and other symtoms of hyperthyroidism—weight loss, sweating etc.

Macroscopically, the gland is meaty, darker in color and on section is not translucent but fleshy.

Microscopically, the follicular epithelium is tall columnar and hyperplastic Papillary projections into the acini are often seen. Colloid is scanty in the acini and is thin and watery, vacuoles may be present in the colloid near the epithelium suggesting the resorption of colloid by the cells. Throughout the stroma is found lymphocytic infiltration, sometimes to the extent of lymphoid follicle formation with active germinal centres. Increase in vascularity is marked

Other lesions : 1. Lymphoid hyperplasia in thymus, Peyer's patches, lymph nodes and tonsils. 2 Lymphocytosis. 3 Myocardial degeneration and fibrosis. 4, Muscular weakness. 5. Increased amount of fat and water in the eye and the extra-orbital muscles are swollen and firm. Retraction of eye lids occurs so that the sclera are visible These lesions are supossed to be due to the action of an *exophthalmus producing substance* (EPS) which is distinct from the thyroid stimulating hormone but produced by the pituitary. Ultimately, exhaustion atrophy occurs with symptoms of myxedema supervening.

In general, we find the following changes in hyperthyroidism.

Increase in : BMR; size of the skeleton in the young; oxygen utilisation in the liver, kidney and muscles; oxidation of enzymes; number of mitochondria (with swelling of mitochondria); blood flow and cardiac output; blood pressure; cardiac rate (and so tachycardia); blood volume (due to vasodilatation); nervous function (so animal is alert, quick, irritable, anxious, wakeful, restless and fatigued); motility of bowel (so diarrhoea and polydipsia); glucose absorption;

Decrease in : body weight; fat depots; serum cholesterol level.

Other changes seen : Muscle weakness and tremors; mobilisation of calcium from bones and increased loss of this element in urine and feces (but blood Ca level is not altered) and hence fractures occur easily and frequently.

THYROIDITIS may be classified into.

- Infectious thyroiditis
  - Acute
    - Non-suppurative
    - Suppurative.
  - Chronic
    - Specific
    - Non-specific
- Thyroiditis due to physical agents
  - Trauma
  - Irradiation
- Due to undetermined etiology
  - Hashimoto's struma
  - Subacute granulomatous (Quervain's thyroiditis)
  - Riedel's struma

Thyroiditis is less common in animals. But in generalised infections it may also get involved. Poor development of the reticulo endothelial system, small arterial branches and smallness of the organ have been attributed as reasons for this. As an extension of a subcutaneous suppurative inflammation, lesions may spread to the thyroid.

Hashimoto's struma (struma lymphamatosa). It is relatively common in dogs and there is a genetic predisposition. Incidence is high in Beagle breed of dogs. The cause is an autoimmune reaction. Thyroglobulin which is a secluded antigen leaks out into the blood due to defect in the basement membrane and stimulates antibody production. These antibodies are being formed by plasma cells and lymphocytes which infiltrate the gland. Three specific antigen-antibody- systems have been described: (1) Microsomal. (2) Thyroglobulin and (3) An altered colloid

Gross lesions are not characteristic. Histologically there are multiple isolated lymphoid nodules with well formed germinal centres replacing the parenchymal tissue. The interstitial tissue and the parenchyma show dense streaks or collections of lymphoid cells, plasma cells and large mononuclears. Larger or oval oxyphilic cells are occasionally seen in the follicles (Askanazy cells).

Subacute granulomatous (Quervain's) Thyroiditis. The disease follows an acute respiratory infection caused by a virus. It is associated with epithelial necrosis, disappearance of epithelial cells and infiltration by histiocytes, mononuclears, fibroblasts and multinucleated giant cells.

Riedel's struma: The gland is very firm, hard and is adherent to the surrounding tissue. Histologically there is severe fibrosis and diffuse, moderate infiltration with lymphocytes and mononuclear cells. The involvement is unilateral compared to the Hashimoto's struma. There is no giant cell reaction.

Thyrocalcitonin: This is a hormone produced by the parafollicular or 'C' cells of the thyroid. These cells are independent of the thyroid secreting follicular epithelium. The 'C' cells are larger and paler and are not incontact with the thyroid colloid. The cells are argyrophilic, stain metachromatically with toluidine blue after hot hydrolysis. Cytoplasmic granules can be stained by strong basic dyes after oxidation with performic acid.

Thyrocalcitonin is a single chain polypeptide, consisting of 32 amino acids. Its molecular weight is 3000. It is now synthesised.

The function of hormone is to reduce bone resorption and to maintain Ca level thereby inhibiting osteoclastic activity Hypercalcemia stimulates the release of the hormone. Its action is opposite that of parathormone. It is thought that in postparturient hypocalcemia (milk fever) there is sudden release of thyrocalcitonin. This is used as a therapeutic agent in hypercalcemic state and in demineralising bone disease. There is significant rise in thyrocalcitonin leve in the blood in thyroid medullary carcinoma.

Calcitonin enchances the excretion of sodium, phosphates and calcium.

Neoplasms of the Thyroid

Primary tumors.

Adenoma in the horse while adenocarcinoma and adenoma in the old dogs are more common. These tumors are found in areas of endemic goitre.

Microscopically, the adenoma resembles the parent tissue and may be difficult to differentiate. The neoplastic acini are smaller with very little colloid and their cells are hyperchromatic. A connective tissue capsule encloses the tumor.

The cells of the adenocarcinoma are highly anaplastic and it is difficult to identify them. Acinar formation is minimal or may not be present but only solid sheets of cells which are cuboidal or cylindrical in shape may be present. Sometimes papillary arrangement is noticed. The nuclei are central, large and hyperchromatic. Nucleoli are large and mitotic figures are numerous Where acini are found colloid is absent in them. Capsule may not be present.

Metastases are common in the malignant variety and secondary deposits are found in all organs, especially in the lungs

Secondary tumors of the thyroid

Metastases from mammary tumors and lymphosarcomas are common in the thyroid.

THE PARATHYROID GLANDS

After birth, two pairs of parathyroid glands are present. In the dog one is situated on the thyroid at its antero-lateral aspect and the other is found in the areolar tissue just anterior to the thyroid. Unless looked for carefully, the parathyroids are usually missed being translucent and merging with the areolar tissue In size, these are small. and weigh about 0.1 gm.

Histologically, the gland cells are of two types

1 **Chief cells or water-clear cells** which are vacuolated.

2 **Oxyphil cells,** the cytoplasm of which is granular and stains red.

The cells are arranged as islets or clusters in a vascular fibrous stroma.

Function : The main function of the gland, through its hormone, parathormone, is to maintain the calcium-phosphorus balance. This is brought about in two ways.

1. by influencing the renal excretion of phosphorus by depressing the renal resorption of phosphate, causing thereby phosphate diuresis and
2. by regulation of the osteoclastic activity in the bone—demineralisation of bone.

Parathyroid hormone helps in the absorption of calcium and magenesium from the intestines.

Parathormone is a protein having a molecular weight of 8500. It has a single chain of 84 amino acids.

Hyperparathyroidism !

May be primary or secondary

Primary hyperparathyroidism is due to an adenoma, in which increased parathormone is secreted. Changes consist in osteopathy, nephrocalcinosis and urolithiasis. The osteopathy has already been described under renal osteodystrophy (Page 467) In nephrocalcinosis there is diffuse calcium deposition in the renal tubular epithelium. (Page 386).

Secondary hyperparathyroidism is found in :

1. Inadequate intake of calcium in diet; or imbalance of calcium : phosphorus ratio.
2. Hypocalcemia due to vitamin D deficiency.
3. Steatorrhoea
4. Chronic renal failure—phosphorus excretion is interfered with.
5. Pregnancy and lactation—calcium is side-tracked to the fetus and milk respectively.

In these conditions there is a lowering of blood calcium or rise in the level of blood inorganic phosphorus leading to hyperplasia of the parathyroids. Osteitis fibrosa cystica and Rubber Jaw syndrome are the sequelae in these conditions which have already been studied (Pages 467 and 468).

THE PITUITARY GLAND

The pituitary gland or the hypophysis is located in the sella turcica. The gland is divided into adenohypophysis and the neurohypophysis.

Adenohypophysis: This arises from Rathke's pouch, which is an invagination of the pharynx This glandular part is divided anatomically into ;—

i) Pars distalis – anterior lobe.
ii) Pars intermedia – the intermediate lobe.
iii) Pars tuberalis, which surrounds the infundibular stem.

Neurohypophysis : arises as a downward growth from the floor of the third ventricle

Structure : Pars distalis is composed of three types of cells, as distinguished by the staining affinities of their granules.

1. The Chromophobes, which do not have stainable granules and are supposed to be immature cells. These form 50% of the cells.
2. The Acidophils. forming 40% have granules taking up acid stains (red with eosion). These are PAS negative.
3. The Basophils, forming 10% have granules taking up basic stains (blue with hematoxylin). The basophils are again divided into the larger **beta cells** and the smaller **delta cells**

Neurohypophysis : The neurohypophysis is connected to the hypothalamus by nerve tracts. This contains modified glial cells called "pituicytes".

Hormones : The adenohypophysis produces the following hormones.

By acidophils :

1. Growth hormone—STH
2. Prolactin.
3. Adrenocorticotropic hormone—ACTH (Doubtful)

By basophils :

1. Thyroid stimulating hormone ; T.S.H (Thyrotropin)
2. Follicle-stimulating Hormone – F.S.H.
3. Leuteinising hormone—L.H. (In the male it is called Interstitial-cell stimulating hormone, I C.S H.)

These are glycoproteins and contain a carbohydrate. The thyrotropin is believed to be secreted by the beta cells while the gonadotropins (FSH and LH) by the delta cells.

The intermediate lobe secretes the melanophore stimulating factor, M. S. F. or hormone (M.S H) See page 175.

The neurohypophysis produces : 1. The oxytocin and 2. The Antidiuretic hormone—A.D.H, or Vasopressin These hormones are produced in the hypothalamic nuclei and then pass along the axons of neuro-secretory cells to be discharged into the small blood vessels of pars nervosa, which is outside the blood-brain barrier.

The Somatotropic Hormone (STH): STH is specific. It acts on all the cells (ectodermal, mesodermal and endodermal) and influences the metabolism of proteins, carbohydrates and fats. This is a complex protein having 396 amino acids. *Its functions are :* (1) increasing the growth of soft and osseous tissues. This is brought about by increasing the cell permeability to amino acids thereby favouring a build up of the muscle mass of the body and nitrogen retention. Growth hormone (STH) acts by stimulating the production of somatomedins in the liver and kidney and these are then released into the circulation These somatomedins act on the epiphyseal cartilage of bones, and help in the transport of amino acids into cells for growth purposes It brings about protein synthesis by (i) interfering in protein breakdown, (ii) inhibiting coversion of amino acids into urea and (iii) accelerating protein synthesis from amino acids. (2) Probably STH causes the release of glucagon, which in its turn raises the glucose level of the blood by glycogenolysis in liver. (3) It probably prevents the entry of glucose into the cell and also inhibits the action of hexokinase So through (2) and (3), STH is antagonistic to insulin and so is 'diabetogenic'. This hormone has profound effect on lactation, which can be induced and enhanced by STH injections.

Prolactin : This is a protein with a molecular weight of 32,000. This hormone also is necessary for intiation of lactation Besides it has a corpus luteum stimulating effect.

Adrenecorticotropic hormone (ACTH) : This is a protein having 39 amino acids The regulation and output of this hormone is intimately associated with the hypothalamus and a servomechanism exists. The adrenal steroids act on the hypothalamus influencing the amount of corticotropin releasing factor (CRF) discharged. Besides external stress stimuli like hemorrhage, temperature, toxins and emotional states influence the release of ACTH by affecting the release of CRF

ACTH stimulates the adrenal cortex but not the medulla. It increases the parenchyma of the cortex as well as its hormones.

ACTH stimulates the phosphorylase activity of the adrenal cortical cells thereby bringing about a series of enzyme-catalised reactions which lead to production of energy needed for corticoid biogenesis

Thyrotropic hormone (TSH). This a glycoprotein with a molecular weight between 10,000 and 28,000. Its functions are (1) to control the uptake of iodide by the thyroid (trapping), (2) to control the formation of iodine-organic compound and later thyroxine and (3) release of thyroxine from the thyroglobulin compound TSH also causes the hypertrophy and hyperplasia of the chief cells of thyroid.

Follicle stimulating hormone(FSH). This is a glycoprotein, with a molecular weight varying between 29,000 and 67,000 among various species of animals. Its secretion is under the control of hypothalamus with a servomechanism Its release is also influenced by environmental conditions such as changing seasons and the length of daylight. Probably visual stimuli through hypothalamus influence it production

The function of FSH is to control spermatogenesis in the male and development of ovarian follicles in the female. It does not stimulate the Leydig cells or cause the production of estrogen.

Luteinising or Interstitial cell Stimulating Hormone (ICSH). LH is in the female while ICSH is in the male. It is a glycoprotein with a molecular weight varying in different species of animals, from 40,000 to 100,000.

LH output is also regulated by the hypothalamus and there is a servomechanism.

The function of the LH is to control the development of the ovarian follicle towards maturation, cause the production of estrogens and ovulation (if FSH has already acted) It may also control the development of corpus luteum. In the male ICSH stimulates the cells of Leydig for secretion of testosterone, causing steroid synthesis.

Oxytocin : Is a peptide containing 9 amino acids (with two sulphur molecules.) It acts on (1) the smooth muscle of the uterus and (2) on the myoepithelial cells of the mammary gland, causing their contraction and hence is useful in the contraction of the uterus (at the end of parurition) and in ejection of milk respectively

Antidiuretic hormone (ADH) or Vasopression, This is also a peptide having 9 amino acids with two sulphur molecules. Its main function is to promote reabsorption of water from the urine by the distal convoluted tubules and collecting tubules This is brought about by increasing the size of pores or channels.

Regulation of ADH production depends on the water content of the body. Increased water content of the blood inhibits the release of ADH and so water is not absorbed by the renal tubules and so excess of water is got rid of. Whenever there is decrease in body water or increase in the electrolytes in blood, ADH is released and this mediates the reabsorption of water from urine, increasing the blood and body water, which dilutes the electrolytes and thus matintains homeostasis The osmoreceptors are located in the supraoptic and paraventricular nuclei.

Pain favours ADA release leading to oliguria while ethyl alcohol inhibits ADH release and so we find diuresis after drinking alcoholic beverages.

Pituitary is subjected to various types of morphological alterations due to normal physiological processes and one should know these to interpret effectively the pathological processes.

Ageing is characterised by accumulation of herring bodies and wear and tear pigments in pars nervosa and proliferation and hyalinisation of sinusoidal reticulum in pars distalis causing splitting up of parenchymal acini. (Herring bodies are globules of amorphous acidophilic colloid-like material).

Aplasia : Aplasia of the hypophysis has been observed in Holstein-Friesian cattle, controlled by an autosomal recessive gene. Histologically there is evidence of failure of differentiation of acidophils.

Cysts : Cysts are relatively common in dogs. They may be developmental cysts (Rathke's cleft cyst; Craniopharyngeal cyst: Evagination cyst) or acquired cysts (common in cattle and horses due to nutritional deficiency of Vitamin A or due to senility) Craniopharyngeal end of Rathke's pouch may persist resulting in the formation of pharyngeal hypophysis, a cystic non-glandular structure

Cysts are common in short muzzled dogs and cross breeds. Cysts may be single or multiple and may be unilocular or multilocular. These are seen commonly in the periphery of pars distalis and pars tuberalis. Cysts are lined by cuboidal, columnar, ciliated columnar or squamous epithelium. Though cysts may be encountered only occasionally, they cause functional disturbances.

Atrophy may be caused by the pressure of cysts or Tumors. Atrophy may be present to a great extent without signs of hypophyseal deficiency. Basophils are more sensitive to pressure than acidophils.

Pituitary lesions in disorders of other organs :

Hypothyroidism : Adenohypophysis is enlarged and the large Beta basophils are hypertrophied. The granules of these cells become finer and disappear eventually In severe cases, these Beta cells become vacuolated giving a spongy appearance to the medulla, where these cells are found in large numbers.

Gonadal deficiency : Gonadotropin-producing basophils become degranulated and are transformed into hypertrophic aminophils, a change characterised by enlarged nucleus, thinness and irregularity of nuclear membrane.

Cystic ovaries: This has already been considered.

Stress : In chronically stressed animals basophils of both types store granules This accounts for gonadal inactivity in such animals. The acidophils are hyperactive and may be completely degranulated or a few granulated cells persist adjacent to sinusoids

Hyperpituitarism : This condition is manifested by overgrowth and proliferation of bone. In man this is called "**Gigantism**" in young growing individuals in whom the ossification of bone has not yet stopped and "**acromegaly**" in adults in whom no more growth occurs.

Gigantism: Gigantism is due to increased secretion of somatotropin in the young. The individual grows very tall and the skin and subcutaneous tissues show fibrous hyperplasia. Since STH is diabetogenic, glycosuria is a symptom. As this condition occurs in adenoma of the acidophils and since the neoplastic growth produces pressure atrophy on the basophils impotence in the male and amenorrhoea in the female are observed. If the patient lives beyond the age of epiphyseal fusion, acromegaly results.

Acromegaly is the condition that is observed in the adult. Since no growth in the bone is possible, the bones become thicker and broader. The hands and feet are abnormally large (akros = extremity; megale = enlarged) and the fingers are crooked and knotty. The facial bones become long and thick, especially the jaw, resulting in prognathism. Viscera are enlarged (splanchnomegaly or macrosplanchnia) and fibrous hyperplasia of skin and subcutaneous tissue is common. Nose, lips and ears become large. Kyphosis is also seen in some. Impotence in the male and amenorrhoea in the female are other symptoms. Diabetes mellitus occurs due to diabetogenic action of the hormone. Eye lesions may be noticed due to pressure on the optic chiasma by the tumor.

Cushing's syndrome : Basophil adenoma may produce the disorder. Conversely, hyperfunction of the adrenal cortex may cause changes in the pituitary. Details of this condition will be studied under adrenal gland.

Hypopituitarism: Since the pituitary is enclosed on three sides by hard bone, even slight enlargement of some part will cause pressure atrophy on others and so corresponding decrease in the activity of the cells results.

Causes of pituitary hypofunction: 1, Pressure by: (a) Tumors, (b) Cysts: 2 Inflammation, sclerosis; 3. Infarction, necrosis.4. Hydrocephalus—bulging of the floor of the ventricle, 5. Abnormal development. 6 Tuberculosis.

Hypofunction in the young:

Pituitary dwarfism or infantilism: Not seen in animals.

In children, though stature is small, yet the build is proportional. Examples are the midgets of circuses These subjects are normal mentally. Sexual development may be retarded.

Symmond's disease (Pituitary Cachexia) or Sheehan's Syndrome; This is found only in females, due to postpartum necrosis of the pituitary consequent on thrombosis following hemorahage. Hence severe hypopituitarism develops. The characteristics are;— severe cachexia, loss of sexual function; weakness, low metaboiic rate, loss of hair and pigmentation, mental apathy and drowsiness microsplanchnia and extreme dehydration and emaciation.

Since trophic hormones are not secreted, there is atrophy and fibrosis of the thyroid, adrenal, ovaries and parathyroids together with the symptoms and lesions consequent on the deficiency of the hormones secreted by these glands and structures.

This condition was described in dogs.

Frohlich's Syndrome—Dystrophia adiposogenitalis: This develops probably due to the pressure by a tumor or hydrocephalus and is mostly found in ladies. The characteristic features are:

(1) Obesity: There is disproportionate and excessive accumulation of fat on the abdomen, buttocks and thighs, while other parts are thinner. (2) Genital hypoplasia and decreased sexual function. (3) Idiocy or mental retardation. (4) Thin skin and hair. (5) Reduced sweat secretion.

In man it is feminising with the characteristic distribution of fat as in a female.

Diabetes Insipidus: Normally under the influence of the antidiuretic hormone of the neurohypophysis, 80% of the water in the glomerular filtrate is reabsorbed by the epithelium of the Henle's loops and distal convoluted tubules. But if the secretion of the ADH is interfered with due to failure of the hypothalamic-hypophyseal system, reabsorption of water from the glomerular filtrate does not occur and so large quantities of urine with low specific gravity are passed and this condition is known as *diabetes insipidus.*

Lesions of the pars nervosa or any causes that injure the hypothalamus will produce diabetes insipidus. The secretion of ADH by the pars nervosa is under the control of stimuli from the hypothalamic nuclei The causes that may produce this situation are:

1. Trauma—surgical or fractures. 2. Pituitary tumor or metastases from bronchogenic carcinoma or mammary carcinoma, 3. Meningitis:—pressing on the stalk, 4. Encephalitis

Tumors of the pituitary

Chromophobe adenoma : This tumor was reported very rarely in horse and dog The tumor consists of cells grouped with an alveolar arrangement. The cells have non-granular cytoplasm. Not producing any hormone, no endocrine disturbances are noticed due to this tumor. Secondary hypopituitarism may develop due to pressure atrophy of the acidophils or basophils, manifested as Frohlich's Syndrome and visual disturbances

Acidophile adenoma : This tumor comprises of cells having an acidophilic granular cytoplasm. Results are gigantism or acromegaly.

Basophile adenoma consists of cells having a basophilic granular cytoplasm Result is Cushing's Syndrome.

Carcinomata of the above three types of cells may be noticed. But these are only locally invasive.

THE ADRENAL GLANDS

The adrenal consists essentially of two glands, differing in structure and function. The cortex, which is external is of mesodermal origin, being derived from the urogenital ridge, from which the gonads and the urinary organs also are derived and so is closely related to them. The medulla, on the other hand, is derived from the neural crest from which the sympathetic nerve cells also arise

Structure

The cortex : The cortex is conveniently divided, histologically, into three zones. The zone of cells lying just under the capsule and arranged as small nests of cells is called *Zona glomerulosa*.

A zone wider than the above and situated beneath it, and in which the cells are arranged as parallel cords, is called *Zona fasciculata*. The zone of cells lying between *Zona fasciculata* and the medulla (the innermost zone) in which the cells are arranged as interlacing cords and containing sinusoids of reticuloendothelial system, is called the *Zona reticularis*.

The cells of all the zones appear similar, polygonal in shape and containing lipids, which give the yellow color to the gland. The cells are also rich in vitamin C content which is probably useful in the synthesis of hormones.

The adrenal cortex is under the control of the anterior pituitary. The adrenocorticotropic hormone (ACTH) controls the form and function of the cortex. ACTH may increase the production of cortical hormones and the parenchyma of the cortex is also increased. Decrease or increase of ACTH is followed by atrophy (regressive transformation) or hypertrophy (progressive transformation) of the adrenal respectively.

When ACTH is lacking, atrophy of all zones occurs. Differentiation of the cells is lost. The cytoplasm and nuclei become smaller and the storage capacity of the lipid is lost. The capsule becomes thickened and the fibrous tissue of the gland is increased. Continuous ACTH administration causes hypertrophy and hyperplasia of the cortex, which, therefore, becomes broader. The cytoplasm and nuclei of the cells become increased in size and the storage capacity of the cells for lipid is increased.

The medulla consists of nests of large cells containing brown granules, nerve fibres, sympathetic ganglion cells, and a rich vascular supporting tissue The

chief cells are large, do not contain any fat but have granules staining brown with chromic salts and so are called chromaffin cells. It is thought that these granules indicate adrenaline content of the cells

Hormones ; The three zones of the cortex probably produce three different physiologically active hormones. But in chemical composition, all have the basic sterol nucleus.

On the basis of physiological activity, three groups of steroids are recognised.

1. **Glucocorticoids.** These hormones are secreted by the Zona fasciculata. The most important glucocorticoids are the *hydrocortisone* or *cortisol* (compound F) and *cortisone* (compound E). The former is more potent and active physiologically The functions of the glucocorticoids are :

1. Conversion of amino acids into glucose—gluconeogenesis from protein—antianabolic

2. Inhibition of peripheral glucose utilisation and so increasing the blood glucose level (hyperglycemia). This is probably brought about by inhibiting the hexokinase reaction and so antagonises insulin action.

3. Decreasing tissue stores of glycogen, especially in the liver

4. Cause catabolism of proteins leading to negative nitrogen balance So urinary excretion of nitrogen and uric acid is increased. Anabolism of proteins is discouraged and so growth in the young ceases, wounds heal more slowly and because of this there is inhibition of antibody production.

5. Because of breakdown of proteins, there is greater blood level of amino acids. And these are converted into glucose as mentioned above. While the permeability of the membrane of extrahepatic cells is decreased for amino acids the permeability of the hepatic cells is increased and so there is increased formation of plasma and liver proteins Hence there is greater deamination in the liver.

6. Shifting of body fat stores. Circulating fatty acids are more and these are used for energy. Some are converted into glycogen of the liver, sparing glucose.

7. Sodium retention and potassium diuresis So continued administration of cortisol leads to edema, metabolic alkalosis, hypokalemia and hypochloremia.

8. Suppression of ACTH production.

9 In excess, due to potassium depletion, muscular weakness.

10. Prolonged use causes increased elimination of Ca, P & N leading to osteoporosis and fractures

11. Suppression of connective tissue response to injury, that means to say, it is anti-inflammatory.: suppression of the activity of the fibroblasts, depression of vascularisation and granulation tissue formation and intercellular ground substance. All these are probably related to protein catabolism. Due to altered permeability of the capillaries, there is decreased exudation of plasma into the tissues

12. Reduction and even absolute disappearance of eosinophils in the blood

13 Causing lysis of lymphocytes in the blood and lymph nodes.

14. Decreasing the secretion of pepsin and HCl.

15. Decreasing the hyaluronidase activity.

16 Interference of antigen-antibody reaction.

17. In man euphoria is produced—that is a sort of well being is experienced by man in such painful cases as cancer and in depressive states.

Control of production : The cortisol level of the blood inhibits the ACTH releasing factor of the hypothalamus Cortisone may also directly inhibit the pituitary from producing ACTH. The hypothalamus may also be influenced by impulses coming from sites of injury or burns and by impulses coming from the cortex of the brain and by "stressors". Hypothalamus, as already explained, is influenced by the epinephrine levels.

Mechanism of acton of glucocorticoids : These stimulate the critical enzyme production by activating a DNA-dependent synthesis of RNA which in turn accelerates the formation of specific enzymes in the cells.

Therapy with glucocorticoids or ACTH: ACTH can be used only if the adrenal cortex is healthy and responsive: Synthetic glucocorticoids like *prednisone etc.* are 3 to 5 times more potent than cortisol and prolonged use of these cause adrenal atrophy

DOG : Primary adrenal cortisol insufficiency has been met with in dogs and they manifest: anorexia, diarrhoea, asthenia, polydipsia, azotemia, hyponatremia, cardiovascular effects, eosinophilia, dehydration, anuria, hair loss, emesis hyperkalemia. Cortisone 1 mg/lb body weight daily cured the symptoms. *Arthritis* in dogs may be usefully treated with cortisone in the dose mentioned above, Acute conditions respond while chronic ones do not *Asthma* may be treated with an initial dose of 1 mg per pound body weight and later maintained with reduced doses.

Purulent dermatitis may be treated with the combination of cortisone and an antibiotic.

Otitis externa shows moderate response to cortisone therapy. In *eye conditions*, cortisone is beneficial in inflammation. **DO NOT** use when there are ulcers of the cornea. After healing of the ulcers cortisone can be used to alleviate inflammation if still present.

HORSE : Glucocorticoids have been useful in lameness of horses. It is particularly useful in the acute stages provided adequate rest is given after treatment. It is not so useful in chronic cases. Injection into the tendon sheath at 50 to 250 mg. of cortisol. a total of 4 injections, each 2 to 3 days apart has given good results. This therapy is useful in acute carpitis, gonitis and in metacarpophalangeal arthritis.

In laminitis in the early stages, 50 to 150 mg can be given intravenously with advantage

For treatment of enlargement of bursa, capsule or sheath, aspirate the fluid with a sterile needle and syringe and then with the same needle but a different syringe inject cortisol and if bacterial infection is present (as evidenced by turbidity or discoloration) add an antibiotic.

BOVINES: Theoretically ACTH can be given in ketosis but intravenous injection of calcium-boro-gluconate is so effective that none probably thinks of treatment with ACTH.

Shipping fever : A condition of hypoadrenalism can arise due to stress of transport, cold, hunger, fatigue, lack of water and food, fright and infectious agents. This stimulates the production of ACTH, which in its turn stimulates the cortex which finally becomes exhausted. Infectious organisms may then invade and cause pneumonia In such situations one can treat with cortisone and antibiotics

Note : Do not use cortisone in the later stages of pregnancy for abortion may occur and the palcenta may be retained

SWINE : In animals that are unable to get up with arthritis, 50 to 75 mg. of cortisone per day enables them to get up and move about.

The following points may be remembered *in* cortisone therapy :

1. Prolonged cortisone therapy inhibits ACTH and so atrophy of the adrenal may result. Hence, if you stop cortisone abruptly, non-functional adrenal may result. So discontinue slowly and gradually.

2. Because of its anti-inflammatory effect cortisone is not useful in specific diseases. It is indicated as a replacement therapy in Addison's disease only.

3. Be careful to check if no infectious disease in present. Because if one is present, the organisms will quickly spread and overrun the body since the defences of the body are diminished

4. The natural walling processes of diseases as in tuberculosis are destroyed by cortisone If you have to use it, then include a specific antibiotic against the infection.

5. Cortisone or ACTH may be useful in chronic diseases as in arthritis.

6. When you desire slow healing, you can use cortisone as a topical application. For example, in surgical or traumatic wounds of the prepuce you would like to have a slow healing instead of a rapid one resulting in strictures. In such cases you can use this topically.,

7. Remember that synthetic glucocorticoids are metabolised slowly and so they are longer acting than cortisone. So their dose should be suitably adjusted.

II Mineralocorticoids :

These hormones, supposed to be secreted by the cells of Zona glomerulosa, control the electrolyte and water balance The chief hormone is the aldosterone. Desoxy-corticosterone acetate (DOCA) is the synthetic hormone having similar properties.

The functions of aldosterone are :—

1. Promotion of excretion of sodium and water by the renal distal convoluted tubules.

2 Promotion of excretion of potassium. So in aldosterone administration there will be muscle weakness due to loss of potassium.

3. Regulation of the extracellur fluid (ECF) volume. Aldosterone promotes reabsorption of sodium—so osmotic pressure of serum increased—supraoptico—hypophyseal system stimulaled—more of ADH released-promotes reabsorption of water from urine—lowers the osmotic pressure of serum—ADH production inhibited—ECF volume kept in balance

In aldosterone therapy, due to increased reabsorption of water (along with sodium)extra-celluar fluid is increased—so blood volume is increased, thereby increasing the cardiac output and blood pressure.

In deficiency of aldosterone, sodium, chloride, bicarbonate and water are lost—extracellular fluid is decreased, thereby minimising the cardiac output and lowering the blood pressure - this leads to failure of circulation, culminating in shock, coma and death Retention of potassium is toxic and the acidosis due to loss of bicarbonate and sodium is harmful to the body aiding in the genesis of shock and death.

Anterior pituitary has no control over the production and activity of aldosterone.

Mode of action of aldosterone : Aldosterone first stimulates the genes or DNA molecules and so specific messenger RNA is synthesised, which in its turn acts to form the specific enzymes that are necessary to supply the energy for sodium transport

Control of production of mineralocorticoids: 1. The sodium and potassium content of blood directly stimulates the adrenal cortex.

2. Aldosterone stimulation can be induced by sodium deficiency or hemorrhage in which conditions there is lowered blood pressure. In this condition the juxta glomerular cells (J.G. cells) situated on the afferent arteriole of the glomerulus act as stretch receptors. So when blood pressure is lowered, renin which is a proteolytic enzyme is released. This enzyme splits angiotensin from alpha serum globulin. Angiotensin acts on the cells of zona glomerulosa and aldosterone is released and this acts on the distal tubule cells of the kidney and sodium is resorbed. Along with it water also is resorbed—hypernatremia causes thirst and so more of fluid is ingested—this with reabsorbed water increases the blood volume thereby increasing the blood pressure. This acts as a servomechanism since the J G. cells are stretched and renin release is shut off and no more aldosterone is released ultimately.

Clinical use : Mineralocorticoids may be useful in the treatment of chronic interstitial nephritis in the dog, because in this condition there is potassium intoxication, sodium loss and uremia. 2 to 5 mg. of DOCA intramuscularly for several days with glucose, fluids and antibiotics is indicated when potassium is eliminated and symptoms will abate

III Sex hormones : These hormones are produced mostly by the Zona reticularis, are anabolic and play a part in protein synthesis. Most of these hormones are antagonistic to the glucocorticoids in this respect. Most of these hormones are androgens (masculanising) and a few only estrogens (feminising).

Medulla : The hormones of the medulla are adrenaline and noradrenaline, which are produced in the ratio of 4 : 1.

The main functions of these two hormones is the maintenance of blood pressure and changes in carbohydrate matabolism and other adjustments so as to meet stressful conditions. Noradrenaline is responsible primarily for circulatory adjustments while epinephrine is for metabolic changes necessary to meet emergencies. Both are powerful stimulants of the heart and increase its force, frequency, and amplitude of contraction So blood pressure is raised Coronary arteries are dilated. Skin vessels are constricted. The rate and depth of respiration is increased.

THE THYMUS GLAND

Thymus is an unpaired organ situated in the thoracic cavity, in the anterior mediastinum, in close association with the pericardium and the great veins at the base of the heart.

The endocrine gland is of vital importance in the young animals. Histologically thymus consists of lymphocytes and an epithelial reticulum. The gland undergoes involution, which commences in animals attaining maturity, viz., between 4 and 6 years in cattle, between 2 and $2^1/_2$ years in horses, between 1 and 2 years in goats, sheep, pigs and dogs; between 6 months and 1 year in cats and between 10 and 15 years in man.

Though considered to be a lymphoid organ, the thymus does not contain any germinal centers the epithelial reticulum probably acts as a blood—thymus barrier; blocking the penetration of antigens.

Functions : The exact functions of the thymus are not yet clear. But from the mass of experimental data, the following appear to be significant.

1. Thymus is an important site of lymphopoiesis in the embryo and the new born It plays a very significant role in specific resistance. Thymectomised animals when stimulated with an antigen after whole body irradiation do not produce detectable antibody, but thymus graft or injection of thymus cells alters the picture with a significant antibody rise. Clones of thymic cells are believed to migrate to the lymphoid tissues in Payer's patches, spleen and other lymph nodes, colonise and enable these organs in later life to give an antibody response to specific antigenic stimulation. The antibody making cells of adult life originate from the clones of antibody making cells of the thymus.

2. Thymus produces a hormone which has an immunotrophic effect, making those cells that have the immunologic potenital, immunologically competent.

Thyroid, adrenal and gonads antagonise the action of thymus. Involution of thymus begins at the time of sexual maturity. After castration or adrenalectomy, the thymus is enlarged and its involution delayed.

Status thymolymphaticus : Sometimes, men die suddenly without any obvious cause and in such people it was found that the thymus and the lymphoid tissue throughout the body were enlarged. In these cases, the hyperplasia of the thymus and other lymphoid tissues is accompanied by the underdevelopment of adrenals and the cardiovascular system, leading to lowered resistance of the patient, who succumbs to various infections. Adrenal insufficiency in such persons renders them unable to adapt themselves to stress This condition has been reported in dogs, cats and cattle

Myasthenia gravis : This is a very peculiar disease of man, characterised by great muscular weakness. The muscles of the face are more severely affected. All the voluntary muscles are involved. No histological changes are noticed either in the muscles or in the motor end plates. Lymphocytic infiltration may occur later.

The cause is still obscure but the condition is associated with hyperplasia of and presence of lymphoid germinal centers in the thymus, which appear to produce some kind of antibody, which prevents the transmission of impulses at

the motor end plate. This is an example of auto-immune mechanism. Anti nuclear antibodies are demonstrable in the blood of such patients. This condition has not been met with in animals.

Tumors : The neoplasms of the thymus are known as thymomas, which are rare among domesticated animals. Unlike in man, these tumors are not accompanied by myasthenia gravis These tumors have been described in goats cattle, sheep, dogs, horses, pigs and rabbits. Most of the tumors were encountered among adult animals.

Site : The tumors are found in the thoracic cavity, anterior to the heart. They may extend to the neck

Macroscopically, thymomas are encapsulated, soft in consistency and gray ish in color. Many tumors may contain cysts and hemorrhagic foci.

Microscopically, the tumors consist of broad sheets of epithelial cells. These cells are derived from the epithelial reticulum of the gland. The cells may sometimes be arranged as whorls or rosettes. Large number of lymphocytes are present

Clinical . Usually thymomas do not metastasize. Some may spread to the lungs, pericardium and regional lymph nodes.

Lymphosarcoma or Hodgkin's disease may sometimes be met with in the thymus and these are usually seen in younger animals. They are similar to such conditions occurring elsewere in the body.

THE PINEAL GLAND

This is a tiny gland placed above the posterior extremity of the third ventricle. In structure it is composed of epithelial cells in a loose connective tissue stroma. Some gila cells and lymphocytes may be found.

The pineal gland secretes a hormone, *Melatonin* (5-methoxy-N acetyltryptamine). Serotonin which is found in the gland is transformed into melatonin by the action of the enzyme, hydroxyindole-O-methyl transferase (HIOMT) which is found in large quantities only in the pineal gland Melatonin antagonises the action of the melanocyte stimulating hormone (MSH) of the posterior pituitary Melatonin acts on the brain to depress the rate of gonadal maturation and to interfere with subsequent gonadal function and cyclicity. Pineal hyperfunction is associated with delayed puberty while hypofunction with precocious puberty. In man complete destruction of the gland by tumors cause cachexia, trophic disturbances, adiposity, premature development of genital organs, premature spermatogenesis and growth of interstitial cells. Melatonin inhibits thyroid hormone secretion rate and the secretion of adrenal steroids.

Tumors : Three types of tumors may arise from the pineal gland.

1. **Pinealomas :** These consist of islands of large epithelial cells, with acididophilic cytoplasm and enclosed in a fibrous stroma Among the large cells are scattered small cells. believed to be lymphocytes These tumors have been described in a goat, horse, dog, silver fox and in a cow.
2. Gliomas comprising of glia cells.
3. Teratomas, from the totipotential cell rests.

CHAPTER 23

THE SKIN AND ITS APPENDAGES

The following terms are used while studying skin lesions.

Acanthosis is thickening of the epidermis due to hyperplasia of the cells of Malpighian layer. This condition may or may not be associated with hyperkeratosis and parakeratosis

Ballooning degeneration is characterised by intracellular edema (hydropic degeneration) which is the early stage in vesicle formation. The cells are swollen and prickles disappear (acantholysis) and the cells become isolated from one another. This is seen in viral diseases.

Bulla (Bleb) is a space, containing fluid situated intra—epidermally or sub-epidermally These are larger than vesicles.

Dyskeratosis is a faulty development in which the cells of the Malpighian layer undergo abnormal, premature or imperfect keratinisation. The changes may suggest developing malignancy since the following characteristics of malignancy are found: hyper-chromatism, loss of polarity and large number of mitotic figures.

Erosion is loss of superficial epithelium. It is also called **excoriation** and is usually caused by continuous discharges.

Fissure or Rhagades is a deep linear defect in the epidermis,often extending to the dermis and occur in dry, crusty skin in which the elasticity is lost.

Hyperkeratosis is the abnormal thickening of the stratum granulosum.

Lichenification is thickening of the skin in irregular areas with exaggerated markings of the skin and is seen usually in chronic dermatitis.

Makula is a discolored spot of the skin, which is not elevated above the skin and may be seen in hemorrhages and focal hyperemia.

Pachyderma is thickening of the skin, all the layers of which are affected. The individual cells are normal The condition is due to nonspecific dermatitis. There may be hyperplasia of the connective tissue. This is also known as elephantiasis and may be seen in the hind limbs of horses and scrotum of old dogs.

Actinomycotic pachyderma is a special form, often seen in the ears of swine. The ear is very much enlarged and hard. This is an actinomycotic granuloma.

Papule or pimple is a small circumscribed solid elevation of the skin resulting from an infiltration of the deep corium.

Parakeratosis is a condition in which the keratin layer is imperfectly formed but in which the nuclei of the horn cells are retained. The stratum granulosum is reduced in size. In this condition, there is production of dandruff.

Pseudoepitheliomatous hyperplasia is a severe acanthosis in which there is deep downward growth of rete pegs, resembling a carcinoma. The lesion is seen at the margins of burns, indolent ulcers and other chronic focal inflammations.

Pustule is a vesicle filled with pus.

Scales are bran-like, thin flakes, consisting of imperfectly keratinised superficial layers of the epidermis and are usually seen in chronic dermatitis.

Spongiosis is inter-cellular edema of the epidermis. This is seen in inflammation. A severe spongiosis will result in vesicles or bullae.

Ulcer is a break in the continuity of the epidermis, exposing dermis and so is deeper than erosion.

Vesicle is a small bulla in the epidermis containing serum, plasma or blood, covered by a thin rim of epithelium and raised above the surface of the skin. These may coalesce to form bullae.

Wheal (Urtica) is a sharply circumscribed, flat, edematous elevation of the skin and is found in urticaria. Several may coalesce to form large plaques.

Anomalies

Epitheliogenesis imperfecta is an inherited skin defect in calves, piglets, pups, lambs, kids, and foals due to an autosomal recessive character. The skin fails to develop around the feet, nose and ears. Infection occurs and septicemia results quickly.

Hypotrichosis congenita : (congenital alopecia) This is congenital absence of hair and is seen in calves, foals and dogs (Dachshunds). Such animals are easily susceptible to cold and sunburns. (Congenital hairlessness in calves is considered to be a Mendelian recessive trait).

Alopecia : This is lack of hair, wool or feathers. The old hair falls out and further growth does not occur. This is a symptom of many skin diseases such as dermatitis, eczema, mange etc.,

In fowls congenital lack of feathers is known as *apennosis* and may be found throughout the body or in some parts.

Causes :

1. **Inflammatory skin disease :** There is irritation and itching to relieve which animals rub themselves. Due to friction, hair is denuded. This is usually seen in mycotic infections or infestation by ectoparasites.

2. **Chemical agents :** (a) **Thallium poisoning :** In chronic poisoning dermatitis is produced in which the fibres break and the shaft is weakened. (b) In chronic poisoning by arsenic and selenium, alopecia is noticed.

3. **Following severe febrile disease :** This is noticed in sheep, especially those that have recovered from blue tongue. There may be a partial or complete loss of wool.

Adrenaline : 1. It elevates blood glucose level by (i) glycogenolysis of liver glycogen, (ii) breaking down muscle glycogen into lactic acid which in the liver is synthesised into glucose (latic acid can be utilised by cardiac muscle to yield energy); (iii) by stimulating ACTH release which is its turn through gluco-corticoids causes gluconeogenesis and glyconeogenesis ; (iv) depression of utilisation of carbohydrates in the tissues.

2. It raises oxygen consumption and so BMR is raised.

3 From fat of depots, free fatty acids are released and these are utilised for energy production.

4 The output, rate and force of heart is increased. Therefore systolic blood pressure is increased.

5. Cutaneous arterioles constricted.

6. Visceral arterioles dilated.

7. Coagulability of blood is increased.

8. Dilatation of respiratory passages.

9. Rate and depth of breathing increased.

10. Erection of hair.

11. Increased sweating.

Noradrenaline : 1. Generalised vasoconstriction. So both systolic and diastolic blood pressure is increased. 2. Increase in sweating. 3. Erection of hair.

Adrenaline acts on the phosphorylase of liver and activates it for rapid glycogenolysis. In the muscles it is able to convert the glycogen into latic acid but not into glucose since muscles do not have phosphatase to split the glucose-6- phosphate and hence blood glucose level is raised by virtue of muscle glycogenolysis. The lactic acid has to go to the liver for conversion into glycogen and glucose (some of it can be metabolised by the heart since it is rich in lactic dehydrogenase).

Reaction of Adrenal glands to Strees : Adrenal cortex reacts to stress by depletion of lipids. Three major patterns occur based on variation of lipid content. 1. Focal lipid depletion with alteration in cell type; 2 focal depletion of lipid with alteration in cell type and degenerative changes and 3 lipid reversion. In the first type, cells are hypertrophic and compact In the second, degenerative changes are characterised by cytoplasmic degeneration. Solid cords of cells in zona fasciculata break up to from pseudotubules and tubules with lumina formation. The lumen of the tubules contain detached cells, pale staining fluid or erythrocytes. Occassionally infarcts affecting mid zona fasciculata may be seen.

Lipid reversion is local depletion; i.e. lipid is absent in outer fasciculata, scanty in reticularis but abundant in the remaining fasciculata. This picture is characteristic of conditions in which the adrenal recovers from stress.

Adrenal in systemic infections :

Acute infections stimulate adrenal cortex and cause lipid depletion . The inflammatory reaction in the adrenal when there is primary granulomatous inflammation is modified due to influence of cortisol and is predominently caseating and organisms are abundant. Generally, the adrenal is spared of infectious process

Colloid formation : Hyaline eosinophilic bodies having a laminated appearance varying in size from 2 to 25 μ consisting of phospholipid materials are seen as inclusions in the adrenal cortical cells. Their significance is not known These have also been reported in patients treated with spironolactone (Aldosterone antagonistic). *Note*: The adrenal glands in mammals can be divded broadly into two groups on the basis of lipid content of the cortex:—glands rich in lipid in man, monkey, rat and rabbit, and glands poor in lipid, in cattle, sheep, camel and horse. A prominent zona glomerulosa rich in lipid is separated from the rest of the cortex by a thin layer of compact cells, the sudanophobic zone. Next to this zone is zona fasciculata, rich in lipid and beneath this zone the zona reticularis, which is poor in lipid. Mitotic activity is greatest in the zona reticularis.

Senile changes in the adrenal are charecterised by narrowing and flattening of zona glomerulosa, thickening of the capsule, formation of irreguiar conglomeration of lipids in the zona fasciculata and hyalinisation of thickened arterioles Arteriosclerosis and atherosclerosis are common in the capsular arteries associated with ageing. Striking presence of thick longitudinal muscle bundles of the the adrenal vein indicates systemic hypertension

Myeloid metaplasia in association with extramedullary myelopoiesis in other organs is common. Myeloid elements arise by metaplastic differetiation of reticuloendothelial cells lining the cortical sinusoids.

Dystrophic calcification following necrosis is common in the cat and dog. Extensive necrosis and calcification may cause symptoms suggestive of adrenocortical insufficiency.

Hypocorticalism can be classified into :

Primary hypothalamic-	**Primary adrenal.**
Symmond's Disease.	Addison's Disease.
Sheehan's syndrome.	Waterhouse-Friderichsen syndrome (Adrenal hemorrhage)
Iatrogenic (Steroid induced)	Contralateral adrenal atrophy
Congenital adrenal hypoplasia (Anencephalic)	Congenital adrenal hypoplasia
	Adrenal cysts.

Congenital hypoplasia of the adrenal gland has been reported in dogs and a hereditary predisposition has been indicated. Adrenal hypoplasia associated with maldevelopment of hypophysis is relatively frequent in calves.

Hypofunction of adrenal cortex

1. Acute hypofunction (Extirpation), rare
2. Chronic hypofunction (Addison's disease).

The manifestation of Addison's disease may occur only if there is bilateral destruction of the glands. This condition is exceedingly rare in animals, but may rarley be met with in man.

Causes : The causes are those conditions causing atrophy and destruction of the adrenals. These may be : 1. Tuberculosis, 2 Atrophy, 3. Amyloid disease, 4. Secondary tumors, 5. Histoplasmosis, 6 Hypopituitarism, 7. Drug allergy.

Symtoms 1. General weakness;anemia; 2 Low blood pressure, feeble heart, 3. Brown pigmentation of skin, 4. Vomiting and diarrhoea, nausea, loss of

appetite, slowed absorption, 5. Atrophied thyroid, 6. Loss of water along with sodium leading to hemoconcentration, 7. Acidosis due to loss of bicarbonate, 8. Inability of liver to convert amino acids to glycogen at usual rates.

These symptoms are explainable by the absence of cortical hormones. The mineral and glucose metabolisms are deranged, leading to elevated potassium level and lowering of sodium; hypoglycemia, hypotension, higher blood urea and anemia. Pigmentation is due to increased melanin production as tyrosine is side tracked to the skin when adrenaline is no longer synthesised from this amino acid by the destroyed adrenals. Another explanation is that with low glucocorticoid level, production of ACTH is stimulated. The first 13 amino acids in the structure of ACTH and MSH are the same and hence their actions are also similar so far as melanin production is concerned. Hence high ACTH levels cause increased formation of melanin giving rise to the bronzed coloration of the skin. Lymphoid tissue of thyroid and lymph nodes is increased. In anterior pituitary there is decrease of basophils and increase of chromophobes. Atrophy of thyroid and heart is noticed.

Adrenocortical hyperplasia. Cortical hyperplasia is relatively common, particularly in dogs. Hyperplasia may be of zona glomerulosa, zona fasciculata or zona reticularis. It may be focal or diffuse and may or may not be associated with clinical manifestation. Hyperplastic reaction of zona fasciculata is a manifestation of adaptation syndrome in stress reaction. Progressive and regressive transformations have been described in stress reaction. The former is characterised by cellular hypertrophy, increased formation of new cells and storage of fat, while the regressive transformation is characterised by depletion of fat and hypofunction of cortex. Regressive transformation is seen in acute diseased conditions. In severe degree of progressive transformation, there may be formation of accessory cortical nodules (extra capsular extrusions). There is proliferation of reserve cells in the sudanophobic zone and transformation of these cells into zona fasciculata type. In the hyperplastic zone acini formation and secretory activity may be seen and this is a morphological expression of severe hyperactivity, wherein the gland takes recourse to exocrine type of secretion. Accessory cortical nodule formation is characterised by formation of nodules on the capsule of the adrenal. Histogenesis of the cells which form the nodule is from subcapsular blastema. The reserve cells in the subcapsular blastema proliferate and invade the capsule which gets split up and later the proliferating cells get encapsulated by fibrous tissue. The small size of the lesion, absence of encapsulation and acini formation are all features of cortical hyperplasia while adenoma is characterised by large-sized nodules, encapsulation, acini formation and the type cells are columnar, lipid-depleted cells.

Results of hyperfunction of adrenal cortex—Cushing's syndrome:

This condition seen in man, is due to excess of circulating hydrocortisone which again may be due to basophile adenoma of the anterior pituitary. Zona fasiculata is increased and the adrenals are yellow in color. Hypercorticalism may be observed in cases of tumors of non-endocrine tissue (ectopic ACTA syndrome), unassociated with regulation of adrenocortical function, like bronchial

carcinoma, pancreatic carcinoma and thymoma. These tumors produce ACTH-like substances and induce Cushing's syndrome.

The effects are : 1. Painful adiposity of neck and trunk (limbs not affected) the buffalo type of obesity ; moon face. 2. Wasting of muscles and weakness 3. In females and pre-adolescent males, hairsultism 4. Amenorrhoea in females. 5. Osteoporosis and kyphosis. 6 Peculiar striations on the abdominal wall. 7 Atrophy of the skin 8. Hypertension, 9 Hyperglycemia and diabetes, 10, Sodium retention, polydipsia, polyuria, urine with low Sp. gl. 11. Slow wound healing 12. Depression of lymphoid tissue and so lymphopenia 13. Susceptibility to infection. 14. Pot belly. 15. Thinning and atrophy of skin . 16. Dermatitis.

Hyalinisation of the basophile cells of the anterior pituitary together with disappearnce of basophile granules is a frequent finding in Cushing's syndrome

Adrenogenital syndrome; Adrenal virilism

A syndrome in which "little girls become little boys and little boys .little men" In this condition there is an excess of androgens—masculinising hormones In the female fetus, if the excess of hormone occurs during the first few weeks of intrauterine life, pseudo-hermaphroditism results. But if it occurs later in females or in boys, precocious puberty results.

Clinically the following symptoms are noticed : 1. Rapid growth with great muscularity in children (infant Hercules) 2. Hairsuitism. 3 Virilism. 4 In girls—enlargement of clitoris, hairsuitism on chest. 4. Impotence in boys (testes are atrophied). 6. In women: amenorrhoea, deep voice, hairsuitism on face and body. 7. Urinary 17 ketosteroids increased. 8 There may be deficiency of hydrocortisone—so hypoglycemia

Neoplasms : Tumors of the adrenal cortex : Adenomas may be seen in old cows and rarely in dogs and horses

The tumors may press upon and protrude into the aorta nearby. These tumors have thin capsules and are yellow in color due to fat content.

Microscopically, the neoplasm consists of cells of normal adrenal cortex. Encapsulation may be present. Cysts and areas of calcification may be seen.

Adenocarcinomas with limited metastases have been reported. Affected animals show virilism, increased libido and weakness.

Pheochromocytoma : Medullary chromaffinoma :

Tumors of the adrenal medulla have been seen in dogs, cattle, horses and sheep and are usually solitary and unilateral These tumors may attain a size of 10 cm in diameter. The tumors are friable and brown in color.

Microscopically, the cells are pleomorphic and are large and ovoid, with a large central vesicular nucleus. The cytoplasm is granular and acidophilic Mitoses are not common . The cells may be arranged in rosette formation. Cells take a dark color with chromium salts and hence the name pheochromocytoma (a tumor of dark staining cells).

Pheochromocytomas in animals are benign and do not metastasise. In most cases, no clinical symptoms are noticed. Rarely symptoms associated with hyperepinephrinism and hypernoradrenalism may be noticed These are paroxysmal attacks of hypertension with tachycardia, sweating, pallor and glycosuria

Injury by ionising radiation : *Though, comparatively speaking,* skin is radioresistant, it is radioresponsive. The beta particles appear to be more pathogenic. The radiant energy seems to alter the permeability of the cell membrane after injuring it, so that the passage of fluids, electrolytes, oxygen and nutrients is so altered that vacuoles appear in the cytoplasm.

The skin may be severely exposed in therapeutic use of ionising radiation. Within 24 to 48 hours, erythema occurs. This may fade and return within 10 to 21 days due to vascular changes. In radiation, vascular endothelial cells are damaged which become swollen Thrombosis occurs obstructing the vessels The fibrous tissue on the walls of the blood vessels becomes hyalinised.

In severe exposure, dermatitis with edema develops, followed by loss of hair (epilation) and necrosis of the epidermis. A highly keratinised, dry and scaly epidermis results—parakeratosis. If the hair follicle is damaged, hair may not grow again. So also, sweat and sebaceous glands may be destroyed If the necrotic area is shed and ulcer forms, secondary infection may occur by saprophytes leading to gangrene.

The changes noticed in the dermis, following damage to the epidermis are : hyperemia, edema. hyalinisation of the fibrous tissue and infiltration by lymphocytes and neutrophils.

If the dermis is not affected, complete healing may occur, But if there is wide spread necrosis with involvement of the dermis, scar tissue may be formed. But this scar is very weak and the epidermis is thin and inadequately keratinised. Carcinomata are supposed to arise from such scars of old X-ray burns.

Urticaria : This is an allergic condition and is characterised by the appearance of wheals (or urtica) on the skin.

Causes : Primary : Insect bites, contact with nettles and caterpillar hairs, unusual food, larvae of warbles; or drugs, eg Penicillin.

Secondary : This occurs along with other diseases in which allergic manifestations are present Eg.: Strangles in horses and Erysipelas in swine, Chronic fowl cholera (wattle disease) in fowls.

The lesions are those found in an allergic reaction. First there is erythema due to vascular dilatation, followed by exudation.

LAMINITIS (Founder)

The inflammation of the sensitive laminae of the hoof is called laminitis This occurs usually in horses but may sometimes be seen in cattle.

In horses, the onset is sudden and acute with high temperature, rapid pulse and accelerated respirations. The animal shows distress and does not like to bear weight on the affected limb.

In the hoof, acute congestion with venous stasis is seen Rarely in very severe condition, after some days, suppuration may occur. As the foot is enclosed in a very tough and unyielding hoof the pressure of the increased blood and edema on the sensitive tissue causes extreme pain which may be severe enough as to cause systemic disturbances Hemorrhages into the tissue may occur. There may be separation of the sensitive laminae from the inner laminae after a few days in some cases Due to weight of the body, the *os pedis* takes a more

perpendicular position and it may actually, sometimes, pierce the sole. Due to the abnormal position of the *os pedis*, weight is borne by the heels and so the toe grows exceedingly long As the keratogenic lamellae are affected and some destroyed, formation of the hoof is uneven.

There is evidence to show that the basic defect is not vascular but degenerative changes in the "keratinogenic layers" of the hoof. These degenerated lamelle do not produce sufficient keratohyaline. There is loss of eleidin granules and "Onychogenic fibrills". Probably some toxic factor destroys some substances important in the formation of hoof. The following hypothesis explains the pathogenesis of this condition, The absorbable toxins, indol and phenol, are formed from tyrosine and tryptophan. These toxins become bound to the free sulphate ions of blood and so the balance between the blood serum sulphur and the sulphur in chondroitin sulphuric acid is disrupted. Hence the collagen loses its strength and elasticity. Consequently the keratinous material loses its supporting tissue, laminitis developing thereby.

Causes :

Laminitis is met with in the following conditions in the horse

1. After excessive ingestion of concentrates, causing enteritis and diarrhoea
2. Drinking very cold water after the animal is over-heated
3. Following the use of irritating purgatives like aloes.
4. Concussion on hard roads—long drives
5. Standing for a long time.
6. Chronic laminitis develops in fat ponies that are kept at pasture without much exercise,
7. In certain septicemic conditions—metritis, retaind placenta, mucosal disease. (In cattle)
8. In cattle certain feeds are supposed to cause allergic laminitis. Cotton seed cake, mouldy hay and barley have been incriminated,

In cattle and sheep that are overfed with concentrates, laminitis might develop.

It is reported that treatment with methionine was very effective, one treatment being sufficient in acute and subacute cases. Medication was continued in chronic cases till symptoms abated

Sequelae: If the condition subsides in 72 to 96 hours no permanent damage results. But if it persists permanent damage to hoof ensues manifested by dropped sole, convexity of the hoof, elongated toe and concentric rings on the hoof parallel to the coronet.

Non-neoplastic cysts

Epidermoid cysts : These are mostly found in the dog. The cysts are rounded or oval and located subcutaneously and so are easily movable But if found in the dermis, they become fixed. These cysts enlarge slowly.

Epidermoid cysts may be solitary or may occur as clusters or sometimes be even generalised. Their size may vary from a few millimeters to a few centimeters in diameter. The cysts are well demarcated, with a thin wall The contents are gray or brownish and may be semi-solid or dry in consistency and contain hair or wool

Usually, epidermoid cysts are benign and can be removed surgically. Rarely do they ulcerate and become infected.

If the contents escape into the tissues, a foreign body reaction is set up. Occasionally, epidermoid cysts may manifest malignant transformation.

Microscopically the cyst wall consists of a collagenous capsule surrounding squamous epithelium Contents consist of keratin, deposited concentrically. The wall of the cyst does not have any skin adnexa. The cyst probably arises by the occlusion of the mouth of hair follicles with resulting trapping of the epithelium, which continuously desquamates keratin into the lumen.

Dermoid cyst . This is similar to the epidermoid cyst but differs from it in that the wall of the dermoid cyst contains skin appendages. The dermoid cysts are located at the junction of the dermis and the subcutis and so are moblie They are soft and round or oval. Occasionally, a cyst may communicate with the surface through a tiny pore. The cyst contains keratinised, greasy substance together with hair and desquamated cells. Calcium and cholesterol may be deposited in the contents

This is a benign lesion and has no connection with congenital "Dermoid", of the eye or the teratomatous "Dermoid" of the ovary.

Sebaceous cyst: Dilatation of sebaceous gland or its duct gives rise to a sebaceous cyst, which contains the greasy sebum and cholesterol.

Sudoriferous cyst: Occlusion of the ducts of sweat glands gives rise to cysts which have a thin capsule, lined by a single layer of columnar or cuboidal epithelium and containing a watery fluid

Calcinosis circumscripta : These are commonly seen among dogs as raised elevated or bulging masses under the skin, one to ten cms. in diameter. On section, white, granular masses are present separated by thin connective tissue septa On incision, chalky paste-like material may be enucleated.

Microscopically, the lesion consists of masses of granular material, staining blue with hematoxylin, evidently a calcium salt, surrounded by granulation tissue with lymphocytes, plasma cells, foreign body giant cells and macrophages

TUMORS OF THE SKIN

Epidermal : Squamous cell carcinoma, Papilloma, Melanoma (Benign and Malignant). Basal-cell carcinoma.

Dermal : Mastocytoma, Fibroma, Fibrosarcoma, Histiocytoma, Myelocytoma, Venereal tumor of dogs, Keloid of horses, Equine sarcoid.

Adnexa : Hair matrix tumor, Basal-cell carcinoma, Adenoma and adenocarcinoma of perianal glands, Adenoma and adenocarcinoma of sweat glands Adenoma and adenocarcinoma of sebaceous glands.

In the subcutaneous tissue: Hemangioma, Hemangioendothelioma, *Lipoma* Lymphangioma

CHAPTER 24

THE EYE AND EAR

THE EYE

Though detailed anatomy of the eye is beyond the scope of this book, a brief description is necessary to understand the pathological conditions that may be met with.

The eyeball is located in the bony cavity of the skull known as the orbit. It is protected by eyelids, which have skin on the outside and startified squamous epithelium (the conjunctiva) lining the surface that comes into contact with the eyeball. The borders of the eyelids have eyelashes containing hair. Just behind the eyelashes are a row of tiny sebaceous glands, the Meibomian glands, the secretion of which serves to lubricate the eyelashes, preventing their adhesion. Movement of the eyeball is brought about by various muscles that are attached to the orbit and the eyeball.

Membrana nictitans or the third eyelid is situated near the inner canthus and consists of elastic cartilage covered by conjunctiva.

The conjunctiva is a membrane with a fibrous tissue stroma covered by epithelium and lines the inner surface of the lids and the third eyelid and is also reflected on the eyball—on the sclera and cornea. The conjunctiva also contains small glands similar to lachrymal glands.

4 **Iodine deficiency in the new born :** Animals born of mothers fed on iodine-defiicient food, are hair-less.

5. **Endocrine imbalance:** Hyperestrogenism in male dogs (testicular tumors produce symmetrical alopecia).

In cryptorchidism (either unilateral or bilateral) bilateral alopecia develop due to deficiency of testosterone.

In bitches, hypoestrinism produces alopecia on the posterior part of the abdomen, inside the thighs and under the tail.

In hypothyroidism (due to atrophy of the thyroid gland, either primary or secondary to disease of pituitary) bilateral alopecia, hyperpigmentation of the skin (diffused or localised), followed by hyperkeratosis are noticed

6. **Vitamin deficiency:** In Biotin deficiency, alopecia is believed to be produced.

Vitamin C deficiency in calves causes loss of hair and cracking of thickened skin

Vitamin E deficiency in cats produces loss of hair on the head and extremities as well as dryness of the skin.

7. **Plant poisons:** When sheep and goats feed on *Tamarindus indica;Senecio* and *Chrysocoma tenuifolia*, alopecia develops.

8. **Injury to nerves:** Peripheral nerve injury is followed by alopecia.

9. **Psychic disturbance-:** In man *alopecia areata* is believed to be due to psychic nervous stimuli There have been many cases of alopecia coincidental with business failure, death or departure of beloved persons and the ordeals of getting married or divorced. Capillary blood supply is not affected.

10 **Other Causes:** In chronic interstitial nephritis in dogs partial alopecia develops. Partial alopecia and hyperkeratosis are found in cattle and dogs suffering from functional disorders of the liver such as cirrhosis.

Feeding of excessive whale oil, palm oil and soy oil as milk replacers causes alopecia. The fibres break easily. Alopecia may de due to the variation in the blood supply to the hairs or due to variation in the nutritive quality of the blood supplied.

Inherited symmetrical alopecia is sometimes met with among cattle and is due to a single autosomal recessive character. Calves begin to lose hair between 6th week and 6th month of their postnatal life. The condition commences on the head, back and hindquarters and progressively extends to other parts of the the body, It is symmetrical Affected parts are completely bald.

Congenital Ichthyosis: Ichthyosis means scaly skin of fish. Some calves are born which have a skin, usually devoid of hair, but which is composed of hard, horny plates, with fissures seperating them The fissures follow the normal skin folds, Affected animals do not usually live longer than one or 2 days.

Macroscopically: there is severe hyperkeratosis and some acanthosis This condition is supposed to be hereditary, conditioned by a simple recessive lethal gene

Acanthosis nigricans : This condition in dogs results from hormonal imbalance—(due to decrease in thyroid uptake). In this condition there is hyperkeratosis with increased pigmentation. This is seen mostly in dogs. The lesions

are bilateral and are small, poorly circumscribed patches, blacker than normal and found on the skin of the abdomen, axilla, innerside of the thigh, inguinal and circumanal region. . The skin is thickened and folded and usually devoid of hair

Microscopically, there is elongation of the dermal papillae. Congestion may be noticed. Prickle cell layer may be increased in thickness with prominent rete pegs Pigment is not confined to the basal layer but is present in cells of all layers. The glands and hair follicles may be atrophied. The cause of acanthosis nigricans is obscure This condition is found in association with certain visceral tumors like adeno-carcinoma of the liver. Dogs affected with sertoli-cell tumor and hypoplasia of the pituitary show this condition

ECZEMA

This is an inflammatory skin condition (dermatitis), charcterised by vesicle formation, infiltration by inflammatory exudate, watery discharge and development of scales and crusts These are the several manifestations of an illunderstood condition. The term eczema is a very general term and is not very descriptive There is no valid reason to retain in since *dermatitis* can be better described specifically according to the nature of exudate; as serous, papular, suppurative, necrotic or parasitic.

Eczema may. however, be applied to the allergic condition of the skin though in animals the nature of allergin cannot usually be determined. The allergins may be exogenous or endogenous. The exogenous allergins may be chemicals (medication of the skin), fungal or parasitic. The endogenous allergins may be ingested proteins or they may be formed in the intestines due to over eating or stasis of food as in constipation; or they may be digested parasites. In some animals there may be inherited predisposition. Continued sweating may also predispose the animal

Macroscopically, in the acute or moist eczema, vesicles, bullae and infiltration by inflammatory cells can be noticed First the vesicles start as spongiosis and later edema of the dermis may develop. Pustules arise which may later dry up. Vesicles may rupture causing weeping on the surface. Dermis may show inflammatory changes.

The chronic or dry eczema : there may be scratching and rubbing leading to thickening of the skin with scale formation, lichenification and formation of scabs and fissures

Microscopically, there is hyperkeratosis and acanthosis. The pegs are prolonged. The dermis shows fibrosis and infiltration by lymphocytes.

DERMATITIS

Severe inflammation of the skin is called dermatitis.

Causes of dermatitis include the following :

1. **Physical :**
   a) Pressure—exemplified by decubital ulcers.
   b) Trauma—abrasions, scratches etc.
   c) Beta irradiation.
   d) Photosensitization.
   e) Heat and cold—if excessive
   f) Sunburn—in unpigmented animals especially.

g) Excessive wetting causing maceration of the stratum corneum. This is easily invaded by baceria.

2. Chemical irritants—acids, bases, thallium poisoning.
3. Bacterial—Swine erysipeleas, Authrax, Tuberculosis of skin, *Streptococcal* and *Staphylococcus aureus infections.*
4. Fungal—various types of ring worm, moniliasis *(Candida albicans,* Epizootic lymphangitis).
5. Parasitic—Nematodes, *Stephanofilaria*; ectoparasites;
6. Viral—Pox (Variola), contagious ecthyma and in Foot and Mouth disease, rinderpest, mucosal disease etc.
7. Allergy—allergic dermatitis of horses.
8. Dermatitis due to nutritional deficiencies—as in deficiency of B vitamin complex in pigs.
9. Protozoa—Leishmaniasis.

Lesions : These vary with cause.

Serous dermatitis : This is the mildest type seen and occurs in sunburns and mild friction due to ill-fitting saddle and harness as well as mild chemicals, heat and cold.

Macroscopically, the skin is red and raised due to spongiosis. Sweat and sebum may be secreted in large quantities.

Microscopically, hyperemia, edema and infiltration by a few leucocytes are seen.

Result : If the cause is removed, the condition resolves quickly and no permanent damage is done. If the cause is more intense and persists, acute dermatitis will follow :

Acute Vesicular Dermatitis :

Causes :

1. More intensive sunburn
2. Stronger chemicals
3. Hotter and colder applications.

} Than in the previous variety

4. Photosensitisation
5. Specific diseases—pox

Macroscopically, besides redness and heat of the locality, there is edema of the dermis leading to swelling of the part. Blebs form in the epidermis containing clear fluid. The roof of the vesicle is formed of stratum corneum.

Microscopically, the lesion starts as an erythema, (morbid redness of skin), with edema in the dermis and infiltration by lymphocytes and histiocytes. With increasing exudate, spongiosis occurs Hydropic degeneration of the prickle cells occurs, which may rupture due to pressure from fluid inside. Due to coalescence of adjoining ruptured cells, vesicles from, which later may enlarge to form bullae. As the fluid accumulates in this vesicle, the superficial stratum corneum is pushed outwards to form the roof of the vesicle and this layer ultimately dies. If the pressure is great, rupture of the stratum corneum will occur, revealing a red base consisting of hyperemic stratum germinativum. Intact stratum corneum prevents infection. Presence of leucocytes in the vesicles changes them to pustules. Later the pustules may rupture and a crust is formed

consisting of the coagulated exudate that forms at the site. Beneath the crust infiltrating neutrophils protect the area while epithelium regenerates and healing occurs

Seborrhoea is increased secretion of sebum and is usually met with in dermatitis The common examples are the greasy heel of horses and seborrhoea in dairy cows. In the former, there is excoriation and soreness on the back of the pastern and may be due to standing in unhygienic barns The part appears greasy and is sensitive to the touch, resulting in lameness Similar condition of the legs may also occur in cattle that stand in mud.

In the seborrhoea of dairy cows, that have newly calved the lesions are commonly found in the groin between the udder and the thighs or in the udder itself between the two halves There is voluminous sebum secretion and the skin may become necrosed Bad smell exudes The affected skin may be shed.

Impetigo : This is pustular dermatitis usually caused by *Staphylococci* and rarely by *Streptococci*

Infection may occur through bite wounds in pigs (bites by dogs or other pigs). Lesions are mostly found on the face—on the snout, ears and over the eyes

In Canine distemper lesions are found under the belly.

In sheep wounds caused by thorns are infected and dermatitis develops.

Dirty litter and humid places are predisposing factors Skin that is continuously drenched by discharge, either from a wound or a natural opening becomes soft and easily infected.

On the udder small pustules are found at the base of teats, Other parts of the teat and uddder may also be affected. Spread to other animals occurs during milking This condition is noticed in association with staphylococcal mastitis.

Lesions : **Macroscopically,** initially erythematous patches may be observed which soon become vesicles and pustules. A scab forms on removal of which a red weeping area is seen. Healing occurs quickly.

If dermis is also affected, a diffuse inflammatory area is seen—**phlegmon.**

Microscopically, the pustule contains serofibrinous exudate with neutrophils The dermal vessels are congested.

Folliculitis is the inflammations of hair follicles. Inflammation of sebaceous glands is called **acne. Boil or furuncle** is an abscess of the hair follicle. **Carbuncle** is a cluster of boils situated close to each other, opening on to the skin through several pores.

Causes : Usually *Staphylococci*; *C. Pyogenes* may also be found. Contributing causes are sweating, contamination by filth; decreased vitality.

It may also result from ringworm, mange and as a complication of distemper.

Lesions ; The earliest lesions are small raised papules, which may either subside or progress to pustules or abscesses Discharge that occurs at the roots of hairs dries to a scab, removal of which results in loss of hair, which, however, regenerates. Local lymph nodes may be enlarged.

Microscopically, the follicle contains inflammatory exudate with leucocytes.

Lachrymal glands, producing tears, are situated below the supraorbital processes. The duct draining the tears is situated near the nasal canthus and opens into the nostrils.

Harderian glands are located in a cavity below the membrana nictitans. They secrete an unctuous material that is useful to lubricate eyelids and membrana nictitans

The **sclera** or sclerotic coat is a fibrous membrane lined externally by the conjunctiva. Though it contains many blood vessels, normally they are not seen, but come into view when they become "injected". The functions of the sclera are : (a) to protect the vascular tissues underneath and (b) by affording rigidity to the eyeball, its shape is maintained constant. Otherwise, by the pressure of the eyelids and muscles, the shape is likely to be altered and vision will be affected.

The **cornea** is the transparent portion of the eyeball It consists of 5 layers: (1) stratified epithelium, which is continuous with that of the sclera; (2) a structureless elastic layer—the Bowman's membrane; (3) the substantia propria consisting of transparent connective tissue fibres; (4) Descemet's membrane, which is very elastic and hyaloid This is deposited by the endothelial cells that line the posterior surface of the cornea This membrane is very tough and is useful when ulceration of cornea occurs. Though the substantia propria may be ruptured, it is this membrane that prevents the herniation of the iris and (5) the endothelial cells that line the Descemet's membrane This forms the anterior boundary of the anterior chamber that contains the aqueous humor. The cornea has no blood vessels and so it derives its nutrition from (1) aqueous humor; (2) from the capillaries of the sclera surrounding it and (3) from tears.

During wakeful hours, oxygen supply is obtained by the cornea directly from the air and when closed by eyelids from the blood. Vitamin A is necessary for the health of the cornea. In its deficiency xerophthalmia results.

The **choroid coat** is a pigmented, vascular membrane that lines the sclera. The retina is situated in its internal face.

The **iris** is a muscular structure with an opening in the centre. This opening is called the pupil. The iris functions as the diaphragm in a camera by virtue of the muscle fibres.

The **ciliary body**, which is a ring of tissue is joined to the iris in front and to the choroid behind. This structure consists of the ciliary processes which secrete the aqueous humor and the ciliary muscle, which helps in the "accomodation" of the eye.

Dilatation of the pupil is known as **mydriasis**. This can be brought about by various drugs: atropine, hyocyamine and stramonium (all of which are parasympatholytic); cocaine, adernaline, and amphetamine which are sympathetic stimulants

Mydriasis may be seen in the following conditions : hypertension, injury to the third cranial nerve (oculomotor), strychnine poisoning and in the later stages of chloroform anesthesia

Constriction of the pupil is known as myasis. This can be brought about by pilocarpine, physostigmine and ergotamine.

Contracted pupil is the tell-tale appearance in morphine addiction.

Myasis may also be met with in the following conditions : keratisis, ulceration of the cornea, inflammatory conditions of the uveal tract and meningitis.

The lens is a peculiar structure composed entirely of epithelium. It has neither stroma nor vascular tissue In front it is bathed by the aqueous humor and is nourished by it. Actually the anterior surface of the lens forms the posterior boundary of the anterior chamber. Its anterior surface is in contact, partly, with the iris. Its posterior surface fits into the depression of the vitreous—the hyaloid fossa. The lens has a tough hyaline capsule outside, which is impermeable to bacteria and leucocytes.

Aqeous humor : The aqueous humor (intraccular fluid) is secreted by the ciliary process into the anterior chamber from where it is drained at the filtration triangle into the veins through the spaces of Fontana.

The functions of this fluid are :

1. It nourishes the lens and cornea which are avascular.
2. It serves as a refractive medium.
3. It helps in the maintenance of the shape of the globe For proper vision it is essential that the shape of the globe is not altered.
4. It helps in the removal of waste products of metabolism.

There should be a constant turnover of the fluid to achieve the above functions or otherwise the fluid may become cloudy and the nutrition of the lens may be affected leading to cataract.

The **vitreous body** is a jelly-like, transparent structure lying between the posterior surface of the lens and the retina and is enclosed in a delicate capsule, the hyaloid membrane

Of the structures enumerated above, the cornea, aqueous humor, the lens and the vitreous, constitute the refractive media and so to a large extent perfect vision depends on their perfect transparency Should they become cloudy or opaque due to pathological processes (to be described hereafter) vision will be impaired and blindness may ensue.

Perfect vision is also dependent upon the health and proper function of the photosensitive nervous receptors in the retina and optic nerve.

The retina is connected to the brain by the optic nerve. It santerior (inner) surface is in close association with the hyaloid membrane of the vitreous and the outer surface related to the choroid.

The histological structure of the retina is very complex and consists of ten layers The rods and cones of the last layer (i e the layer in contact or near the choroid) are the most important in vision

The **optic nerve**, which is the second cranial nerve, enters the globe at the optic papilla and the fibres get distributed to the retina

Branches of the third, fourth, fifth and sixth cranial nerves innervate different parts of the eyeball and the fibres get distributed to the lachrymal glands,

Congenital anomalies of the eye

Anophthalmia congenitus This is complete absence of one or both eyes. Instances of this condition have been reported in foals, pups, calves and piglets Histological examination may reveal remnants of some ocular structures

Microphthalmia is a condition in which one or both eyes are small. Most of the cases have been reported in swine (result of hypovitaminosis A of the dam) and dogs (a hereditary condition caused by sub-lethal genes)

Cyclops is a condition in which there is only one eye due to fusion of the two orbits and is seen in monsters

Ankyloblepharon is a condition in which both the eyelids are fused together.

Strabismus is squint of human beings. In animals the condition is bilateral with the two eye globes turning inwards. Squint is seen in Siamese cats in which it is congenital. This has also been seen in calves of beef breed and in collie dogs

Entropion is turning in of the eyelids and is a hereditary congenital condition seen in sheep, dogs and foals.

Ectropion is turning out of the eyelids. Usually the lower eyelid is affected. This is also a hereditary condition.

Coloboma is a congenital anomaly due to failure of the closure of embryonic ocular cleft and so the eylids, ciliary body, lens or iris may be affected showing fissures or gaps in their continuity In the eyelids a small wedge shaped portion may be missing or even larger area may be lacking. Similarly small areas of iris and lens may be absent.

Dermoids of cornea: These tumors occur as a congenital condition due probably to a sublethal factor. The cornea of one or both eyes is partly covered by skin containing hair. Actually the cornea at the place affected is replaced by the skin Because of constant friction and irritation by hair, conjunctivitis, pannus formation, keratitis or ulceration may result.

Congenital anterior synechia is a condition in which there is adhesion between the iris and the posterior surface of the cornea.

Congenital opacity of cornea may be met with, sometimes, as a result of anterior synechia In certain breeds of cattle (Holstein-Friesian, Swiss, Norwegian, Red poll) hereditary opacity of cornea has been met with

Microphakia— the lens is small and is spherical. It may be opaque or transparent.

Displacement of the lens or luxation of the lens *(ectopia lentis)* may be met with in some animals. The dislocated lens is opaque. In certain breeds of dogs (Sealyham and terriers) it is a developmental defect.

Cataract is a condition in which the lens becomes opaque. In some animals it is a congenital condition.

Congenital aplasia of the retina and hypoplasia of the *optic nerve* may be met with in calves and they are born blind.

EYELIDS

Trichiasis is the turning in of the eyelashes and therefore cornea is irritated by the hair. Keratitis and attendant lesions may be encountered.

Blepharitis is inflammation of the eyelids. This may be a part of generalised dermatitis. But it is usually a complication of (a) distemper in dogs and cats, (b) trauma, (c) conjunctivitis or (d) inflammation of the lachrymal glands. Complications of blepharitis are: (i) **ankyloblepharon**, when adhesions between eyelids take place, (ii) **Symblepharon**, where there is union between the conjunctiva lining the lids and that covering the eye balls.

Hordeolum or stye is the inflammation or even abscess formation of the follicles of an eyelid and is a very painful condition.

Chalazion is the abscess formation of the Meibomian glands. Sometimes a cyst may form in these glands.

Edema of the eyelids : This usually results due to (i) trauma, (ii) infection, (iii) conjunctivitis, (iv) allergy and (v) fracture of the orbital ring. The following condtions are accompanied by edema of eyelids—*Horse:* Influenza, pink eye, purpura and allergic conditions

Cattle : malignat catarrhal fever, distomiasis, stomach worm affection, traumatic pericarditis and penetration of a foreign body into the eye like husks etc.

Pig :gut edema, chronic hog cholera.

Dog and Cat : distemper, allergy, stings by nettles and ants.

Neoplasms : Squamous cell carcinoma which may arise from the conjunctiva or the skin of eyelid or from the membrana nictitans is very common among cattle

Papillomata, basal-cell carcinoma and angiomata (cavernous or capillary) may be found. Adenoma of the Meibomian glands may also be met with.

ORBIT.

Exophthalmos : Protrusion of the eyeball

Enophthalmos sinking of the eyeball into the orbit.

Orbital cellulitis : is the inflammation of the orbit. Is rare.

Causes : Nutritional—in cats.
Foreign bodies
Extension from periodontitis of posterior molars in dogs.
Orbital contusion complicated by fracture of the ring of the orbit.
Extension of ophthalmitis.

This condition is usually suppurative in nature. Non-inflammatory edema with slight exophthalmos occurs in edema disease and Mulberry heart disease of swine, congestive edema of head, purpura hemorrhagica and urticaria.

Lachrymal apparatus:Dacryoadenitis is the inflammation of the lachrymal glands and is rarely met with as a complication of conjunctivitis or trauma. There is diffuse congestion and enlargement of the gland, causing protrusion of the membrana nictitans. An abscess may form rupturing on the upper eyelid. The ducts are dilated with inflammatory exudate.

Xerophthalmia due to vitamin A deficiency has already been studied (Page 157)

Occlusion of lachrymal canal : This may be congenital or acquired. In dogs of certain breeds (Sealybeams and Poodles) there may be congenital absence of the puncta.

The canal may be occluded in the following conditions. (a) entropion, (b) inflammatory swellings found in conjunctivitis and rhinitis, (c) atrophic rhinitis in swine, (d) miller's disease in horses and (e) neoplasms of the nasal passage.

Adenoma of the lachrymal gland is very rare.

CONJUNCTIVA

Conjunctivitis is commonly met with among animals and the causes are :—

(a) Bacteria : Some bacteria can penetrate the intact conjunctival mucosa—*Brucella*, *Listeria* and *Pasteurella tularensis*

Usually, the conjunctival mucosa is free of bacteria either due to the flushing action of the tears or to the bacteriostatic property of the lysozyme.

Secondary infection by *Staphylococus aureus*, *Pseudomonas aeruginosa* and *E. Coli* may occur following trauma or debilitating viral diseases (distemper in dogs).

(b) Chemicals : Disinfecting fluids, lime in white washing material, irritant gases such as formalin vapour and sulphur, smoke, acids, alkalies, sheep dips, parasiticides, skin dressing, iodism.

(c) Foreign bodies : Awns, oak husks, mud, dust and sand.

(d) Parasites : *Thelazia lachrymalis* in horse, *T rhodesii* in the ox, *T. callipeda* in dog and *T. leesi* in camel.

(e) Allergy to pollen, horse serum etc.

Symptoms : There is congestion of the conjunctiva and increased production of tears which flow over the face as the lachrymal cannal may be closed due to swelling of the membrane. The tears are clear at first but soon become turbid and thick due to the presence of leucocytes and mucoid material. It may also contain flecks The eye lids may be glued by the sticky material.

Infections by pyogenic organisms as occur in distemper of dogs and periodic ophthalmia of horses produce purulunt conjunctivitis. Croupous or diphtheritic conjunctivitis is mostly encountered in fowls. In cattle infection by *S. necrophorus* causes croupous conjunctivits. In this condition there is gray or chocolate colored membrane covering the eyeball.

Infection may spread to the cornea and keratitis may, therfore, result. In the purulent and croupous varieties, keratitis and ulceration of the cornea ar very commonly seen.

CORNEA

Pannus is a condition in which vascular granulation tissue is found betweee the corneal epithelium and the Bowman's membrane. Calcification of the granulation tissue may sometimes occur.

Keratitis. The causes of keratitis are the same as detailed for conjunctivitis. Symptoms inculde photophobia and blepharospasm (in which the eyelids are tightly closed). The cornea may, in the initial stages, show edema of the epithelium followed by opacity due to infiltration by leucocytes. In the acute stage ulceration may occur. Vascularization of cornea may be seen.

Corneal ulceration occurs during acute or chronic conjunctivitis. It may also occur as a result of suppurative conjunctivitis or due to trauma (thorns, nails, barbed wire, cat scratches, horn gores). Nutritional imbalance due to deficient proteins and vitamins and impaired nerve supply may also cause ulcers. Virulent organisms can cause ulcers due to the activity of their toxins.

If the Descemet's membrane is also perforated, aqueous humor is lost. There may be prolapse of the iris through the rupture (Staphyloma), followed by dislocation of the lens. Secondary infection of an ulcerated cornea can infect the whole globe (panophthalmia) and the eye will be completely lost.

Healing of a corneal ulcer is slow and is similar to healing of an open wound. The scar tissue contracts and a tiny scar, which is opaque, is left, which never completely disappears. Depending on its density, the corneal scar is known as nebula, macula or leucoma.

Infectious keratoconjunctivitis in cattle : Pink eye : This condtion may occur as an enzootic in many parts of the world and is common in summer and autumn Probably flies transmit the disease from animal to animal. The causative organism is *Moraxella bovis*, which is gram negative and is found in the tears. An endotoxin that causes necrosis of the skin is produced by this organism. Blood stream is not infected.

Symptoms include conjunctivitis, copious lachrymation, photophobia and blepharospasm A slight elevation of temperature accompanied by anorexia occurs. Within one to two days following the onset of the above symptoms, corneal

opacity develops in the centre followed by ulceration in two more days. Ulceration in the young expands and vascularization may occur. The cornea may become completely opaque. There may be purulent discharge from the eyes. As the condition subsides, opacity decreases and complete recovery occurs in three to five weeks. Recovery is followed by immunity which lasts for a year. Immunity is local and may be due to peristence of the organism in the conjunctival sac

A mild conjunctivitis is also caused by the virus of Infectious Bovine Rhinotracheitis characterised by congestion of the conjunctiva with increased lachrymation which is serous. Cornea may become slightly cloudy. Cornea is not ulcerated

Infectious keratoconjunctivitis in sheep—contagious ophthalmia: In sheep this is also known as pink eye, *caused by Rickettsia conjunctiva* and occurs as outbreaks in various parts of the world in summer. Though the condition is mild and not fatal, animals become temporarily blind and become weak and undernourished as they cannot graze properly and adequately.

Symptoms include conjunctivitis, keratitis, increased lachrymation, blepharospasm and opacity of the cornea with vascularization. The discharge which is watery at first becomes purulent subsequently. Recovery starts within three to four days and by the tenth day is complete. In some animals, certain amount of opacity of the cornea remains. Recovery is followed by partial immunity and the recovered animals are carriers for a year.

Among goats, the condition is mild.

Infectious keratoconjunctivitis in fowls

This condition is found in Africa, Russia and Denmark among fowls. A rickettsia is said to be causative. It is known as *Ricloasia conjunctivae.*

Birds under three months are usually affected, the incubation period is 4 to 6 days. The condition is transmissible

Symptoms : Usually one eye is affected. In a few days the other eye is also affected due to spread of infection. The eyelids are glued together and opacity of the cornea develops. If no secondary infection occurs, recovery in 2 weeks is the rule. Rarely growth of the bird may be retarded. Morbidity is 20% to 30% while mortality is less than 1%

No organisms are seen in the conjunctival sac. Inclusion bodies are seen in the conjunctival epithelium. Since antibiotics are not useful in treatment a virus is suspected to be the cause.

THE LENS

Because of the peculiar structure of the lens, the changes that could occur in this structure are of limited range. These are : (a) changes in its position and (b) degenerative or metabolic changes in which the transparency of the structure is altered.

Luxation of the lens : The lens is anchored by the zonula of Zinn or the suspensory ligaments to the ciliary body. If these ligaments are ruptured, the lens may be displaced into the anterior chamber or into the hyaloid fossa or into the vitreous If it is displaced into the anterior chamber, opacity of the cornea occurs due to the pressure of the lens on the endothelium. In such a situation,

the nutrition of the lens is altered and it becomes opaque. Glaucoma may develop due to hindrance in the filtration of the aqueous humor (consequent on the abnormal position).

Inflammatory changes may be produced by the unnatural position of the lens and so adhesions between the cornea, lens and iris may be brought about.

When the capsule of the lens is ruptured, the lens may be liquefied and resorted

Causes: 1. The condition may be a congenital anomaly

2. Trauma is the most important cause in the acquired variety and found mostly in dogs, especially in Sealyham and terrier breeds. Excessive barking may traumatise the ligaments.

3. Glaucoma which may be the result of cyclitis, may cause secondary luxation.

4. Since this condition occurs in older dogs (after the age of 3 years) degenerative changes(causes unknown)in the suspensory ligaments may be a cause.

5 A predisposing genetical factor may play a part.

Cataract : Opacity of the lens is cataract. Obviously, therefore, blindness or impairment of vision develops. Cataract may be partial or complete depending on its situation . It may be congenital or acquired, the former being the most common

Incidence : Cataract is common in dogs and rare in other animals. Among dogs, there is hereditary and breed susceptibility.

Pathogenesis : The lenticular tissue is capable of the following changes :— (1) proliferation of the capsular epithelium, (2) necrosis of the fibres of the lens and (3) increased sclerosis of the fibres forming the nucleus. So depending on the nature of the changes, cataract is classified as follows:

1 **Subcapsular cataract :** This is seen in horses, dogs and birds. In this condition there is abnormal proliferation of the lens epithelium Usually the proliferation occurs at the front surface of the lens, when it is known as *anterior polar cataract* Sometimes the proliferation of the cells may extend beyond to the posterior surface due to the degeneration of the lens. It is known as *posterior polar cataract* The cells, because of proliferation, become disorganised and form thicker layers producing opacity. This condition may be a result of posterior synechia or due to repeated attacks of periodic ophthalmia in horses and is usually associated with cortical cataract.

2 **Cortical cataract:** This is the most common form and involves the lens fibres, either at the front (anterior cortical cataract) or at the back (posterior cortical cataract). Due to accumulation of interstitial fluid consequent on the altered metabolism of the epithelial cells, the fibres become disintegrated and disorganised The cataract is stellate, spreading from the centre to the periphery. It is a progressive condition. Usually this type follows a corneal ulcer

3 **Lamellar cataract :** This occurs in young animals. It is non-progressive and is located between the nucleus and the cortex It may be congenital or acquired and results due to some injury during development and is seen in puppies following an attack of distemper or in those that have suffered from rickets (due to vitamin D deficiency).

4. **Nuclear cataract** are probably the results of senile changes in which the fibres at the centre become more dense thereby making the nucleus dull or hazy.

Causes:

1. **Congenital:** (a) failure of the byaloid artery (which is present in the embryo entering at the optic papilla and extending to the posterior surface of the lens and nourishing the vitreous humor) to regress and disappear completely, leaving remnants of its wall or its small branches.

(b) Impairment of translucence of the lens due to abnormal arrangement of the lens fibres or there may be fluid or droplets of fluid between the nucleus and cortex of the lens.

(c) A hereditary predisposition may precipitate the occurrence of cataract in later life.

(d) Deficiency of vitamin E: cataracts are found in chicks born of fowls fed vitamin E deficient diets.

2. **Acquired:** Degeneration of the lens due to:

(i) luxation.

(ii) impaired nutrition, as occurs in ophthalmitis and affections of the uveal tract.

(iii) degenerative ocular disease as in retinopathies and retinal detachment in dogs, as found in Pekingese.

(iv) Trauma.

(v) Senility—common in old stallions.

(vi) Diabetes mellitus: This is seen occasionally in dogs. Probably increased sugar content alters the osmosis in the aqueous humor.

(vii) Nutritional disease—deficiency of vitamin D; deficiency of vitamin C in the lens; deficiency of cystein, which plays great part in the oxidation-reduction processes

(viii) Toxins: (a) toxins circulating in diseases like influenza and periodic ophthalmia in horses and distemper in dogs may cause degeneration of the lens, b) toxins of uremia as occurs in chronic interstitial nephrits.

(ix) Poisons: ergot in cattle and pigs.

(x) Absorbed radiation—(see page 131).

THE UVEAL TRACT

Anterior synechia is the condition in which there is adhesion of the iris to the posterior surface of the cornea. This results due to iritis. Anterior synechia causes glaucoma.

Posterior synechia is the adhesion of the posterior surface of the iris to the anterior surface of the lens capsule. This condition also results due to iritis. In this condition pupil cannot dilate.

Iridocyclitis. This is the inflammation of iris and ciliary body and is also known as **anterior uveitis**. This condition is best exemplified by specific condition in horses known as Periodic ophthalmia

Periodic ophthalmia or Equine recurrent iridocyclitis or Moon Blindness

This is a disease of equidae, in which one or both eyes may be affected The idease may abate only to reappear (in the same or the other eye) in a mor

severe manner causing greater damage to the eye, which finally becomes blind on repeated attacks.

Causes : The exact cause is obscure. The following are suggested.

1. Deficiency of riboflavin,
2. Infection by leptospira,
3. Hypersensitivity to leptospira (?)
4. Virus (?), transmitted by flies.

Symptoms and lesions: The disease starts with photophobia, belpharospasm, lachrymation and tightly contracted pupil, which does not dilate even in darkness The conjunctiva and sclera are congested and the cornea may show vascularization. The iris is dull and yellowish but not the normal brown. The pupil becomes clouded due to the presence of the exudate and leucocytes in the anterior chamber. Particulate matter settles at the lower border of the iris and posterior surface of the cornea forming the **keratic precipitates.**

Within a week or ten days, the disease subsides so that photophobia vanishes and the animal is able to see. Though the disease appears to subside, the eye is never completely normal since some amount of anterior synechia and posterior synechia are present.

Repeated attacks cause posterior synechia, subcapsular cataract (due to the alteration in the nutrition of the lens consequent on inflammatory changes of the uveal tract), gradual absorption of the lens after the capsule is ruptured, liquefaction of the vitreous humor, shrinkage and atrophy of the eye. The choroid and retina separate due to accumulation of the exudate. The eye is finally lost

THE RETINA

Progressive retinal atrophy in dogs : This condition is found in Irish setters due to a recessive inherited factor. The retina in both the eyes becomes atrophied, manifested in the early stages as night blindness; finally resulting in total blindness. The degeneration first starts in rods. Gradually the outer and inner nuclear layers become discharged and atrophied and finally the ganglion cell and nerve fibre layers become atrophied. It may take some years even for complete blindness to develop.

Detachment of retina: Retina lies just in contact with the inner surface of the choroid but not attached to it. So the retina can be lifted (detached) by accumulating edematous fluid or exudate or blood. This occurs in inflammatory conditions of the choroid or even of retina. Inflammatory exudate pours between the choroid and retina thus seperating them In injuries to the eye, especially thorn pricks in cattle, there may be hemorrhage resulting in the separation of the two structures.

Retina may be pulled away by the traction of shrinking fibrous band of the vitreous. In ophthalmitis, organising exudate may cause traction on the retina which may be datached from its normal position. Liquefaction or loss of vitreous humor may be another cause for detachment of the retina.

When the retina is detached, the rods and cones first become atrophied and disappear, followed by the nuclear layer and the optic fibre layer . Gliosis may also occur. Finally cataract, glaucoma and shrinkage of the eye result.

OPTIC NERVE

Atrophy :

Causes :

(a) Congenital.

(b) Acquired : retinitis, papilledema, retinal degeneration, glaucoma; choroidoretinitis, trauma on occiput, hemorrhages; poisons: morphine, filix mas, arecanut; deficiency of vitamin A.

The optic papillae become thinned with disappearance of the interstitial capillaries. Retinal degeneration follows atrophy of the optic nerve. Total blindness results

GLAUCOMA

Glaucoma is a condition in which there is increased intraocular pressure leading to secondary changes in the eyeball. Increased intraocular pressure may result from (a) too excessive a secretion of the aqueous humor, or (b) hindrance in its drainage. Among animals excessive production does not usually occur. It is obstruction to drainage that is most commonly the cause of glaucoma.

If the causes that give rise to obstruction of the flow, leading to glaucoma cannot be determined with certainity, the condition is known as **primary glaucoma**. Probably congenital glaucoma is of this category. If the causes for such obstruction (see below) can be determined, the condition is known as **secondary glaucoma**

Causes of secondary glaucoma : The pathological lesions causing obstruction may be due to (i) occlusion of the pupil, which may occur as a result of posterior synechia. In this condition due to organisation of the inflammatory products, the iris becomes adherent to the lens and so the fluid accumulates in the posterior chamber, iris is pressed forward and the filtration triangle and the spaces of Fontana are blocked by this protruding iris.

(ii) Occlusion of the filtration angle by the products of inflammation, which may be acute or chronic Majority of secondary glaucomas occur this way. So obstruction may occur in : iridocyclitis in which anterior synechia occurs; inflammation following trauma of the eye ball; luxation of the lens; intraocular hemorrhages: detachment of the retina and intraocular neoplasms.

Symptoms : Glaucoma may be unilateral or bilateral. The globe is enlarged and exophthalmos may be noticed (**buphthalmos**). Cornea may be edematous and opaque. Corneal vascularisation and pannus may result due to chronic corneal edema. In man pain of the eye, headaches, toothache and earache are felt.

Lesions and sequelae : The cornea is flattened and the iris is displaced anteriorly. There is opacity of the cornea, the lens and vitreous humor. Anterior synechia may occur. Degenration of the lens may occur Blood vessels become sclerosed. Atrophy of the choroid to a thin membrane may ensue. Due to pressure a depression is excavated in the optic disc, which assumes the shape of a cup (*Cupping of the disc*) The nerve fibres become atrophied Due to atrophy of the nerve fibres, retina becomes degenerated and atrophied. Ganglion cell layer disappears and blindness results

Ophthalmitis is seen as a symptom and lesion in various specific diseases :

Species of animal.	Disease.	Lesions seen in the eye.
Cattle	Mucosal Disease.	Conjunctivitis, keratitis and corneal opacity. In calves born of affected cows, optic neuritis, cataract, microphthalmia and retinal dysplasia seen.
	Infectious bovine rhinotracheitis (IBR)	Purulent conjunctivitis, keratitis and corneal ulceration.
	Malignant catarrhal fever.	Exudative non-suppurative retinitis, optic neuritis, iridocyclitis and acute conjunctivitis.
	VIRAL DISEASES	
Dog.	Infectious canine hepatitis.	Edema of cornea seen in convalescent stages.
	Rabies.	Negri bodies in ganglion cells.
	Canine distemper.	Retinitis and optic neuritis resulting in blindness when the optic tract is destroyed. Intranuclear and cytoplasmic inclusions seen in the ganglion cell layer; choroid loses its pigment and becomes thin. Pigment-laden cells invade the retina and the perivascular spaces.
Horse.	Borna disease.	Inclusion bodies in ganglion cells of retina, Retinitis and optic atrophy may occur.
Pigs.	African swine fever.	Blindness.
	Swine fever.	Inflammatory changes in retina and uveal tract; congestion, edema and hemorrhages in iris and infiltration by mononuclears of the ciliary body.
	Swine encephalomyelitis.	
Fowl.	Marek's disease	Iridocyclitis
	BACTERIAL DISEASES	
Cattle.	Listeriosis.	Suppurative ophthalmitis and secondary corneal changes.
	Morexella bovis.	Infectious keratoconjunctivitis.
	Tuberculosis	Nodular uveitis affecting the iris or the choroid or both.
Calves.	Streptococcal meningitis; Polyarthritis; Coliform infection.	Purulent ophthalmitis
Sheep & goats	*Mycoplasma agalactia*	Opacity of cornea, keratitis with ulceration. conjunctivitis, turbidity of aqueous humor, glaucoma; exophthalmos, iridocyclitis; anterior synechia; luxation of lens and loss of vitreous.

Species of animal.	Disease.	Lesions seen in the eye.
Cattle, pigs, cats, fowls and parrots.	Tuberculosis.	Hematogenous from lesions elsewhere.
	RICKETTSIAL DISEASES	
Sheep.	*Rickettsia ovis.*	Infectious keratoconjunctivitis.
	MYCOTIC DISEASES	
Dogs.	Coccidiodomycosis. *(C immitis)*	Uveal tract affected—detachment of retina occurs. Destruction of iris and ciliary body, infiltration of cornea by inflammatory cells; glaucoma.
	Blastomyces dermatidis	Diffuse uveitis—detachement of cornea
	Cryptococcus neoformans	Choroid affected; retina detached-
Horse.	*Cryptococcus neoformans.*	Keratitis and conjunctivitis.
Fowls.	*Aspergillus fumigatus.*	Panophthalmitis — fungus grows in the vitreous.
	PROTOZOAL DISEASES	
Dog.	Toxoplasmosis.	Inflammation of retina with infiltration of mononuclears in its layers; perivascular cuffing; hemorrhages and inflammatory exudate with mononuclears and plasma cells in vitreous and around the ciliary body.
	Leishmaniasis.	Conjunctivitis and keratitis.
Horse.	Trypanosomiasis.	Diffuse interstitial keratitis,
	PARASITIC DISEASES	
Dog.	Dirofilariasis.	These parasites, found in the aqueous humor cause keratitis
Horses	Larvae of Habronema	Conjunctivitis. (The above are not natural inhabitants of the eye but occur only by chance).
In various animals	Thelaziasis.	This is a natural parasite of the eye and lives in the conjunctival sac and lachrymal ducts and cause mild conjunctivitis If the number of the parasites is more than 6, ulcerative conjunctivitis may be caused.
Fowl.	*Oxyspirura mansoni.*	Keratitis, conjunctivitis and ulceration of cornea.

Neoplasms of the eye :

Primary : Squamous cell carcinoma, especially in the bovines, is the most common neoplasm.

Adenomas and adenocarcinomas of the lachrymal gland and Harderian glands may be met with.

Adenoma and adenocarcinoma of the ciliary epithelium and iris may occur.

Secondary Matastases of carcinoma, sarcomas, melanoma, lymphosarcoma, meningioma and the venereal tumor may be met with.

DISEASES OF THE EAR

The external ear: This consists of the concha, the external auditory meatus and the ceruminous glands.

Otitis externa :

Causes : 1. Foreign bodies like awns may lodge in the ears of dogs and cause irritation and inflammation. This is a chronic condition in which there is hyperplasia of the epidermis, hyperkeratosis of the hair follicles and the infiltration of inflammatory cells. The skin becomes very much thickened and the sebaceous glands contain eosinophilic material. The foreign body may sometimes rupture the tympanum.

2. Ectoparasites :

a) *Psoroptes communis*—causes profuse exudation into the meatus, which thus contains tenacious brown material. This is seen in sheep more often.

b) *Otodectes cynotis* causes otitis in dogs and cats. Due to irritation the dogs may shake their heads often and this leads, in the long-ear breeds (Dachshund) to hematomas. Secondary infection by bacteria may produce profuse exudate and tympanum may be ruptured.

c) *Otobius megnini* or the *spinose ear tick* causes otitis in cattle. Though only lymph is sucked by the larvae and the nymphs, secondary bacterial infection of the wounds caused results in otitis.

d) *Fungi* that produce dermatomycosis may also cause otitis.

e) *Stephanofilaria zaheeri* causes dermititis of the ears in buffaloes and may cause otitis.

3. Specific disease : In swine, *Actinomyces bovis* causes a typical actinomycotic granulomatous condition of the ears, which become thick and indurated.

The characteristic symptoms are the presence of thick pus in the external auditory meatus (Otorrhoea) and the thickening of the lining of the meatus. Shaking of the head is an important symptom of the presence of pus in the meatus. Stagnant pus may lead to rupture of the tympanum with subsequent occurrence of otitis media and even otitis interna.

Neoplasms of the ear are rare : Adenoma of the ceruminous glands may occur. Sarcoid in the equines and chondroma and chondrosarcoma may rarely be met with.

Middle ear consists of the tympanic cavity, the ossicles and the eustachian tubes. In horse, guttural pouches are diverticula of the eustachian tubes. The epithelium lining the tympanic cavity is continuous with the nasal mucosa through he eustachian tubes and so infection from the nose and pharynx can extend into the middle ear.

Otitis media : Infection can occur through the external auditory meatus through the eustachian tubes. Normally, there is no communication between the external and middle chambers as, the tympanum seals the passage. But in conditions in which there is profuse exudate in the extrenal auditory meatus, ear drum can be ruptured by pressure and infection of the middle ear occurs.

The inflammatory exudate that accumulates in otitis media, unless drained becomes inspissated and organised, especially around the ossicles, immobilising them and so deafness may ensue.

As alresdy observed, infection can occur via the eustachian tubes from the nasal passages and the pharynx. The organisms found in such cases are *C: pyogenes* in calves, swine and sheep; *Pseudomonas aeruginosa* and *Streptococci* in swine; *Pasteurella* in cats and *Staphylococci* and *Mycobactertum tuberculosis* in different animals

Other sequelae of otitis media are : extension of infection into the inner ear (**otitis interna**), deafness, paralysis of the 7th cranial nerve, meningitis and encephalitis due to extension of infection into the cranial cavity through the 8th cranial nerve with resultant death.

CHAPTER 25

IMMUNOLOGICAL DISEASES

By Dr. B. B. Mallick,

Introduction

Whether pursued in Jenner's time or in our own, the immune process is clearly one of the body's defense mechanisms against infection. Ideally, therefore, the body should respond immunologically only to antigens (Ag) of pathogenic microorganisms, viruses, and parasites. However, it is not so since animals, including man will respond to injection of harmless Ag such as foreign serum proteins. Some persons also respond, often to their ultimate disadvantage, to otherwise harmless Ags of pollens, housedust and animal danders.

It used to be taken for granted that the body would at any rate never make the much graver mistake of responding immunologically to its own Ags. The mechanisms which prevent the formation of antibodies (Ab) to its own tissue components may on occasion break down. That is, body can, and in many cases does, produce an immunological response to its own Ags. This phenomenon is known as "Autoimmunity" and has rapidly assumed outstanding importance and is responsible for many of the immunological diseases. Thus autoimmunity is the general term used to describe an immune response, either Ab or cell mediated against normal body constituents. Earlier, the term immunological diseases was used to refer to this kind of conditions. But hypersensitive reactions caused by Exogenous allergens might well claim admission to the group.

ETIOLOGY

It may be postulated that the autoantibodies (AAb) may be manufactured under the following circumstances.

1. Alteration in antigenecity of tissue proteins :

This may be due to (a) degenerative lesions e.g., lens proteins in cataracts and skin in burns, (b) Attachment of hapten e.g., Allergic contact dermatitis.

CONTENTS

PART III
INFECTIOUS DISEASE