1 ASCARIASIS (COMMON ROUND WORM)

a CAUSE AND TRANSMISSION	b DISTRIBUTION, SPECIES AND AGE GROUP AFFECTED	c DURATION AND MORTALITY	d CLINICAL SYMPTOMS
Ascaridia galli. Site-intestinal tract. Life history is simple and direct. Eggs are passed out in the faeces and germinate in litter. Chickens as they feed ingest the infective stage eggs.	Most common intestinal parasite of chickens observed throughout the world. Problem is not severe in cage reared chickens. Young chickens of 2-3 months old most susceptible. A few ascarid worms in mature birds are not harmful but heavy infection may lead to serious losses.	Mortality never goes high but losses are due to general weight loss and stunted growth.	General signs are retarded growth, progressive anaemia, droopiness, emaciation and diarrhoea. Performance of the bird is adversely affected.

ASCARIASIS (Continued)

e POSTMORTEM LESIONS	f TREATMENT	g PREVENTION AND CONTROL	h SPECIMEN REQUIRED FOR DIAGNOSIS
Catarrhal enteritis may be observed, Generally there may be minute haemorrhages and ulceration at the point of attachment of worms to mucosa. In severely affected cases haemorrhagic enteritis may be observed. On occasions a low grade peritonitis has been seen.	Piperazine compounds are used widely. It is given as one day treatment in drinking water. 10 g/gallon of drinking water can be given. The flock should be dewormed at 5 wks of age and then at 30 day interval to 21 wks in case of floor raised pullets.	Control is best accomplished by coordinating medication and management. The ascarid worm is not a problem in birds reared on wire. In cage rearing deworming may be done at 2 stages; at the time the birds are taken from floor to grower cages and at the time of shifting from grower to layer cages.	1. Worms in 70% alcohol or in normal saline solution. 2. Fresh faeces packed over ice or saturated with 5 to 10% formalin. 3. Dead bird packed over ice.

2 BUMBLE FOOT

DISEASE DUE TO FAULTY MANAGEMENT

CLINICAL SYMPTOMS AND PATHOLOGICAL FEATURES

This localised infection of the foot causes bulbous swelling of the foot pad and surrounding tissues. It appears usually after an injury to the footpad and causes lameness in one or both feet. Infection by various bacteria commonly occurs and superficial wounds may or may not be present. Sometimes ulcers are formed on the planter surface of the foot. The layers become lame and stop laying.

DISEASE DUE TO FAULTY MANAGEMENT

POSSIBLE CAUSES AND REMEDIES

The condition is more common in heavy breeds. Early surgery and antibiotics therapy may control the condition. Removal of high roosts reduces the incidence.

3 CANNIBALISM

DISEASE DUE TO FAULTY MANAGEMENT

CLINICAL SYMPTOMS AND PATHOLOGICAL FEATURES

Many forms of cannibalism occur in birds. The main types being vent pecking, feather pulling, toe pecking, head pecking, and wing and tail pecking.

There are signs of injuries particularly on the vent or the region of the abdomen, head, wattles, eyes, toe and comb. The area of injury becomes swollen and dark blue or black with subcutaneous haemorrhage. In wing and tail pecking and feather pulling, there is continuous oozing of blood from follicles.

DISEASE DUE TO FAULTY MANAGEMENT

POSSIBLE CAUSES AND REMEDIES

Predisposing causes are: feeding only pellets, excess hard grains in the ration, insufficient feeder/drinker space, being without feed too long, insufficient light, over crowding, too much heat, dietary deficiency of protein and minerals and irritation by external parasites. Once the birds have tasted blood they will continue the cannibalistic habit unless prevented. Many remedies have been used to stop cannibalism: hanging cabbage and sugar beets in the pen, painting the windows red, using red light bulbs, applying pure tar to pecked areas of birds, adding salt in rations and feeding oats. Debeaking is the most widely accepted means of prevention and stopping cannibalism.

4 CHILLS IN CHICKS

DISEASE DUE TO FAULTY MANAGEMENT

CLINICAL SYMPTOMS AND PATHOLOGICAL FEATURES

This is seen mostly in the first three weeks of life. The chicks huddle together. There is white diarrhoea with pasting up of cloaca. It could be confused with pullorum disease. Postmortem lesions include unabsorbed watery yolk, distended gall bladder, congestion of lungs, nephritis.

DISEASE DUE TO FAULTY MANAGEMENT

POSSIBLE CAUSES AND REMEDIES

Failure of heating system. This can be prevented by construction of brooder houses so as to maintain required temperature.

5 COCCIDIOSIS

a CAUSE AND TRANSMISSION	b DISTRIBUTION, SPECIES AND AGE GROUP AFFECTED	c DURATION AND MORTALITY	d CLINICAL SYMPTOMS
Disease caused by one or more of the many species of coccidia. There are 9 species of Eimeria reported from chickens. All coccidia of the domestic fowl involve the digestive tract, except for renal coccidiosis in geese. Both clinically infected and recovered bird shed coccidial oocysts in droppings which contaminate feed.water, litter and soil. Oocysts are also transmitted mechanically by equipment, attendants, clothing and such carriers. Oocysts sporulate to become infective. Ingestion of large number of sporulated oocysts results in clinical disease.	Disease has a world-wide distribution. A disease of young chicks but outbreaks seldom occur at less than 11 days of age. More common at 4-6 weeks of age. Non exposed adult birds remain highly susceptible and have serious outbreaks.	Mortality depends on strain of the species, age of chickens and genetic susceptibility of the host. It varies from 1 to 40% in severe outbreaks.	Clinical signs and course of the disease are greatly influenced by degree of acquired immunity by the flock. Affected birds sit around and have little or no interest in feed and water. In acute cases birds have ruffled feathers, blocdy droppings, high temperature with increased mortality. In chronic infections signs include weakness, weight loss, increased number of culls and decreased egg production. Survivors may recover in two weeks time.

COCCIDIOSIS (Continued)

e POSTMORTEM LESIONS	f TREATMENT	g PREVENTION AND CONTROL	h SPECIMEN REQUIRED FOR DIAGNOSIS
Lesions depend on the species of coccidia. E-tenella: produces enlargement of ceca with thickening of its wall, accumulation of clotted blood, tissue debris and oocysts. E-necatrix: produces lesions in anterior and mid portion of small intestines. Wall of the intestine is thickened and in severe cases lumen may be filled with blood. Small white yellow plaques usually intermingled with bright-red petecheae or dull spots of various sizes seen on serosal surface are pathognomonic. E-maxima: lesions are produced in middle intestine characterised by haemorrhagic enteritis with thickening of the intestinal wall and ballooning. Lumen contents are grayish brown or pink to red brown. E-acervulina: parasitises the duodenal loop, but may extend to lower part of intestine, Lesions of E. acervulina are whitish gray transverse ladder like areas in duodenum and upper part of jejunum. Heavy infection causes coalescence in the lesions and thickening of the mucosa.	A large variety of drugs for use as feed additives for preventing coccidiosis are available. Examples are Furazolidone, Nitrofurozone, Sulphamezathine, 3-Nitro, Amprolium Sulphaquinoxaline, Sulphamethoxine, Nicarbazine & Buquinolate. Preventive treatment with these drugs is being widely done. Occasionally in the face of an outbreak of coccidiosis treatment may be beneficial. These anticoccidial drugs at a therapeutic level may then be used for treatment. Products are provided with instructions of use.	Good litter management is a prerequisite. Treat outbreaks to stop mortality. Allow chickens to develop a low level infection for acquired immunity. Vaccines are also available (more literature may be consulted on coccidiosis immunity).	1. Faeces saturated with 5-10% formalin, or in 2.5% solution of potassium dichromate. 2. Sick or freshly dead chickens over ice or wrapped in formalin soaked cloth. 3. Portion of intestine in 10% formalin.

a CAUSE AND TRANSMISSION	b DISTRIBUTION, SPECIES AND AGE GROUP AFFECTED	c DURATION AND MORTALITY	d CLINICAL SYMPTOMS

COCCIDIOSIS (Continued)

e POSTMORTEM LESIONS	f TREATMENT	g PREVENTION AND CONTROL	h SPECIMEN REQUIRED FOR DIAGNOSIS
E-mitis, E-mivati E-hagani and E-praecox parasitising upper half of the small intestine are less pathogenic and produce poor growth and more culls. E-brunetti occurs in lower small intestine, rectum, ceca and cloaca It is rated as a severe pathogen after E-tenella and E-necatrix. In moderate infection there is cattarrhal enteritis with thickening of intestinal wall. Severe infection causes extensive coagulation necrosis with complete sloughing of mucosa with only the basement membrane remaining.			

6 CROP IMPACTION

DISEASE DUE TO FAULTY MANAGEMENT

CLINICAL SYMPTOMS AND PATHOLOGICAL FEATURES

Over distention of crop. The crop becomes dilated and filled with foul smelling feed stuff and foreign materials. Birds with impaction may survive for days, but gradually become emaciated and die of inanition.

DISEASE DUE TO FAULTY MANAGEMENT

POSSIBLE CAUSES AND REMEDIES

Consumption of large quantities of fibrous and bulky material. Cases of impaction are also seen after eating of sand, straw, sawdust and long blades of grass. Paralysis of muscles and nerve supply to crop may also result in impaction.

Early detection of condition can be corrected by surgery.

7 EGG EATING

DISEASE DUE TO FAULTY MANAGEMENT

CLINICAL SYMPTOMS AND PATHOLOGICAL FEATURES

It is a similar habit to cannibalism and spreads quickly throughout the flock.

DISEASE DUE TO FAULTY MANAGEMENT

POSSIBLE CAUSES AND REMEDIES

Predisposing conditions are: inadequate nesting facilities, failure to collect eggs frequently, soft and thin shelled eggs, dietary deficiencies particularly of minerals.

This can be prevented by controlling predisposing factors.

Debeaking should be done to prevent this habit.

8 FOWL CHOLERA

a CAUSE AND TRANSMISSION	b DISTRIBUTION, SPECIES AND AGE GROUP AFFECTED	c DURATION AND MORTALITY	d CLINICAL SYMPTOMS
Bacteria: Pasteurella multocida spread by contact. Bowel, nasal and mouth discharges contaminate feed and water. Spreads rapidly.	Worldwide in distribution. Reported from most avian species. Prevalent in late summer, fall and winter. Affects all age groups.	Acute form lasts 1-2 weeks with high mortality. Chronic form is long lasting with mild losses.	Symptoms vary. Depends on acuteness and localisation. Chicks often die suddenly without showing symptoms. Listlessness, diarrhoea, purplish comb with difficult breathing may be seen in acute form. Chronic cases localise in joints, wattles, eyes and foot pads with swelling and abscess formation.

FOWL CHOLERA (Continued)

e POSTMORTEM LESIONS	f TREATMENT	g PREVENTION AND CONTROL	h SPECIMEN REQUIRED FOR DIAGNOSIS
Acute cases may have no lesions. Petechial haemorrhages on mucous membranes and visceral organs. Small greyish foci of necrosis in light coloured liver and swollen congested spleen. Chronic cases have localised infection which generally becomes suppurative. They occur often in respiratory tract. Pus may be present on joint and tendon sheaths.	Sulpha drugs and antibiotics in drinking water/feed.	Vaccinate flock. Vaccination after exposure is not satisfactory. De-population and clean up most effective method of control.	1. Smears from heart blood, liver impression smears. 2. Bone marrow, heart, liver or localised lesions packed over ice. 3. Sick birds.

9 AVIAN INFLUENZA (INCLUDING FOWL PLAGUE)

a CAUSE AND TRANSMISSION	b DISTRIBUTION, SPECIES AND AGE GROUP AFFECTED	c DURATION AND MORTALITY	d CLINICAL SYMPTOMS
Avian Influenza Virus-Type A. Fowl plague virus has been shown as a highly pathogenic member of the Avian Influenza group of Viruses. Spread within the flock by contact; Nasal and mouth discharges contaminate water and feed. Rate of spread is very rapid.	Fowl plague virus has been reported from Britain, Russia, North Africa, Middle East, Far East, North and South America. Subsequently members of Type A. Influenza virus have been isolated from many countries including Germany, England, U.S.S.R., South Africa and Czechoslovakia. Disease mainly affects chickens but viruses have been isolated from various other avian species with different disease syndromes.	Morbidity and mortality rates are variable. It depends largely on strain of the virus and condition of exposure. Mortality varies from 0 to 10%. Infection with fowl plague and some other pathogenic member of this group results in high mortality (100%) and high morbidity.	Signs of this disease are ill defined. General signs include decreased feed consumption, emaciation and lowered egg production. Respiratory signs vary from mild transitory symptoms to moist rales, coughing and sneezing. There may be oedema and cyanosis of head, face and unfeathered skin with excessive lacrimation. Nervous disorders characterised by shaking of the head with closed eyes, turning the head on the back, lameness or paralysis of legs may also be observed, in some outbreaks. In some outbreaks disease may be so acute that chickens are found dead without any signs and symptoms.

AVIAN INFLUENZA (Continued)

e POSTMORTEM LESIONS	f TREATMENT	g PREVENTION AND CONTROL	h SPECIMEN REQUIRED FOR DIAGNOSIS
Inflammatory changes are first observed in the skin, comb and wattles. Necrotic foci may be observed in liver, spleen, lung and kidneys. Yellow gray exudate on the air sacs and a mild peritonitis may be observed in fatal outbreaks. Haemorrhagic or fribrinopurulent pericarditis may also be noticed. Haemorrhages may be observed on heart, proventriculus, duodenal part of the intestine, and subcutaneous fat. Postmortem lesions resemble those seen in Fowl Cholera, Newcastle Disease, infectious Bronchitis, infectious Laryngotracheitis, chronic Respiratory Disease and Phosphate Poisoning.	No specific treatment. Treatment of affected flock with antibiotics may help control mortality.	No solid recommendation can be made for prevention and control of this disease. General sanitory precautions may be of help. Vaccines are still on trial.	Same as for Newcastle disease.

10 FOWL POX

a CAUSE AND TRANSMISSION	b DISTRIBUTION, SPECIES AND AGE GROUP AFFECTED	c DURATION AND MORTALITY	d CLINICAL SYMPTOMS
Fowl Pox Virus. (a large DNA Virus): Spread by contact with infected birds. Also carried by flies, mosquitoes and wild birds.	Disease is prevalent worldwide. Affects chickens of all ages.	Morbidity varies from infection of a few birds to involvement of entire flock. In uncomplicated outbreaks the course is 3-4 weeks. Mortality is usually low but in serious outbreaks involving young chicks may go as high as 50%.	Typical pox signs may be in one of the three forms or in a combination. (a) cutaneous form: raised wart-like lesions on the unfeathered areas (head, vent and legs) are seen, (b) diphtheritic form: lesions seen in the mouth. (c) Oculonasal form: involvement of nasal chambers resulting in coryza like symptoms; swelling of head, nasal discharge, lacrimation, caseous exudate in conjunctival sac. Unthriftiness, emaciation, poor weight gain and slow spread through the flock are typical signs of the disease. If layers are infected egg production is temporarily retarded.

FOWL POX (Continued)

e POSTMORTEM LESIONS	f TREATMENT	g PREVENTION AND CONTROL	h SPECIMEN REQUIRED FOR DIAGNOSIS
Cutaneous lesions appear as small white foci which increase in size and become yellow. This progresses through inflammatory process and finally forms a dark-brown scab in approximately 2 weeks time. Lesions usually coalesce to form bigger scabs. Lesions in various stages of development can be seen on the same birds. Scab falls off as it heals in about 2 weeks. If the scab is removed before complete healing, it leaves beneath a moist haemorrhagic, granular surface. The diphtheritic form develops as elevated opaque nodules, which rapidly become yellow necrotic caseous pseudo-membranes or diphtheritic membranes. These lesions usually appear on buccal mucosa, trachea and larynx. The process may extend to nasal sinuses causing swelling of sinuses. Cheesy membranes in the pharynx usually suffocate the bird.	There is no specific treatment. Remove false cheesy membrane from the mouth and give a course of antibiotics in drinking water to prevent complication.	In areas where pox is prevalent and in flocks where pox has been observed in the previous year, vaccination with fowl pox vaccine is advisable.	1. Affected live bird. 2. Scab in 10% formalin and 50% glycerine separately.

11 HEAT STROKE

DISEASE DUE TO FAULTY MANAGEMENT

CLINICAL SYMPTOMS AND PATHOLOGICAL FEATURES

The birds in production are highly susceptible to high temperatures. The symptoms comprise of sweating, wet feathers, laboured breathing, weakness, excessive thirst, high temperature, sometimes nervous symptoms followed by complete prostration. The egg production falls rapidly. Postmortem shows white parboiled appearance of musculature, acute congestion of lungs with pneumonic lesions, congestion and haemorrhages in the brain.

DISEASE DUE TO FAULTY MANAGEMENT

POSSIBLE CAUSES AND REMEDIES

Faults in heating systems, over crowding, bad ventilation, closed nests and insufficient water supply are some main factors causing heat prostration. An attempt to overcome this problem can be made by increasing air circulation, dipping the birds or spraying the surroundings, putting additional drinkers and building cheap artificial shade.

12 INFECTIOUS BRONCHITIS

a CAUSE AND TRANSMISSION	b DISTRIBUTION, SPECIES AND AGE GROUP AFFECTED	c DURATION AND MORTALITY	d CLINICAL SYMPTOMS
Infectious Bronchitis Virus (IBV). Spread through air by mechanical means. Rate of spread is rapid.	Disease has been reported from most parts of the world. It affects chickens of all ages.	All the birds in the flock become infected but mortality is observed in young chicks and may be as high as 60%. Mortality is usually low in adults (5-10%).	Typical signs in young chicks are coughing, sneezing and rattling. There may be occasional facial swelling. These respiratory signs cease in 2-3 weeks. In adults, in addition to respiratory signs feed consumption and egg production drop sharply. A decrease of 20 to 50% in egg production with many mishapen thin soft shelled eggs of poor internal quality is not uncommon.

INFECTIOUS BRONCHITIS (Continued)

e POSTMORTEM LESIONS	f TREATMENT	g PREVENTION AND CONTROL	h SPECIMEN REQUIRED FOR DIAGNOSIS
Main lesions of the respiratory tract are catarrhal condition in trachea and nasal sinuses; cheesy plugs in lower trachea, bronchi or air sacs. Gross lesions are reported in the reproductive tract of infected chickens. Oviduct weight and length are markedly reduced in layers infected at early age. Isthmus and magnum are the portions most severely involved. Nephrosis has also been described with outbreaks of infectious bronchitis with some strains of virus.	No specific treatment. Keep disease from spreading by strict isolation. Raise brooder temperature and stop drafts. Supportive treatment with antibiotics may be tried.	Strict isolation, sound management practices coupled with vaccination are best preventive measures.	1. Affected live bird. 2. Lung, trachea, kidney in 50% Glycerine and 10% formalin separately. 3. Serum samples from recovered birds.

13 INFECTIOUS CORYZA

a CAUSE AND TRANSMISSION	b DISTRIBUTION, SPECIES AND AGE GROUP AFFECTED	c DURATION AND MORTALITY	d CLINICAL SYMPTOMS
Bacteria: Hemophilus gallinarum. Recovered birds in the flock serve as the main reservoir of infection. Transmission takes place by airborne infective droplets and by contamination of drinking water, feed and equipment.	Found wherever chickens are raised. Affects all age groups, but the course tends to be longer in older birds.	Rate of spread is rapid with high morbidity. Virulence of organism alters the course of the disease. The course in a flock may vary from 2 weeks to several months. Disease is more severe and prolonged with high mortality when complicated with other pathogens.	There is serous to mucoid nasal discharge with oedema of face and conjunctivitis. Oedema of the face may extend to intermandibular space and wattles, particularly in males. In chronic cases sinuses become distended with yellow caseous exudate. In complicated cases foul odour comes in the flock. Egg production drops.

INFECTIOUS CORYZA (Continued)

e POSTMORTEM LESIONS	f TREATMENT	g PREVENTION AND CONTROL	h SPECIMEN REQUIRED FOR DIAGNOSIS
Acute catarrhal changes in the mucous membranes of nasal passages and sinuses with oedema of face and wattles are observed. Pneumonia may also be seen.	Sulphanamides, antibiotics and other antibacterial drugs are of value.	Isolation rearing from old stock is desirable practice for prevention. Replacement should be made from day old chicks unless the source is known to be free of this disease.	1. Live chick in acute state of disease. 2. Head, trachea, air sac exudate preserved over ice.

14 KEEL BURSITIS (BREAST BLISTERS)

DISEASE DUE TO FAULTY MANAGEMENT

CLINICAL SYMPTOMS AND PATHOLOGICAL FEATURES

This occurs mostly in heavy rapidly growing birds. A large abscess usually found on the sternum contains blood tinged fluid or thick yellowish caseous pus. This causes downgrading of the carcases and serious financial loss.

DISEASE DUE TO FAULTY MANAGEMENT

POSSIBLE CAUSES AND REMEDIES

The aetiology is not well known. Pressure of the keel on roosts and direct trauma with sharp objects causes inflammatory lesions. Bacterial contamination results in serious losses.

15 MAREK'S DISEASE

a CAUSE AND TRANSMISSION	b DISTRIBUTION, SPECIES AND AGE GROUP AFFECTED	c DURATION AND MORTALITY	d CLINICAL SYMPTOMS
Virus: B group of herpesvirus. Transmitted by direct contact. Infectious virus matures in feather follicle epithelium and is shed in the dander. Infection of new host is by inhalation of infected dander.	Disease has been observed throughout the world. Chicks get infection in early age. Disease is most commonly manifested in 8-24 weeks of age but may be seen as early as 3 weeks and as late as 18 months.	Duration of the disease is short in acute form and long lasting in classical form. Mortality is usually high in acute form seen in young chickens and is low but regular in classical form. Occurence and severity of the disease depends on many factors related to host and/or virus.	Young chickens with acute form may only show depression and die without any sign. In general there is progressive lameness or paralysis of legs, wings, neck, eyelids, or other parts of the body depending on the involvement of a particular nerve. Blindness may be seen in birds where eyes are involved. Vagus nerve involvement results in paralysis of crop and respiratory trouble. Survivors of an outbreak become emaciated and stunted.

MAREK'S DISEASE (Continued)

e POSTMORTEM LESIONS	f TREATMENT	g PREVENTION AND CONTROL	h SPECIMEN REQUIRED FOR DIAGNOSIS
Most constant finding is the enlargement and thickening of one or more of the peripheral nerves. Affected nerves lack striations, become thickened, oedematous and are grayish in colour. The brachial sciatic and vagus nerves are most frequently involved. Lymphoid tumours may be present in any organ. Gonads are most commonly affected but lymphomas may be present on liver, lung, heart, skin muscle, kidney, intestine and proventriculus. These visceral lesions appear similar to lymphoid leucosis.	No treatment.	Live virus vaccines to be used at one day old. Isolation rearing may be advocated for control of M.D. Breeding for resistance may be adopted.	1. Affected live birds. 2. Affected organs (Nerves, brain, spinal cord, gonads, lung, heart, liver, bursa, intestines, piece of skin from femoral and crural feather tracts) in 10% formalin. 3. Serum from several birds in the flock.

16 OMPHALITIS OR MUSHY CHICK DISEASE

DISEASE DUE TO FAULTY MANAGEMENT

CLINICAL SYMPTOMS AND PATHOLOGICAL FEATURES

The chicks usually appear normal until a few hours before death. The chicks huddle together, go off feed, deaths occur within 1-2 days, sometimes prolonging up to 14 days after hatching. Navel is found inflamed with a pasty substance over the vent. Sometimes scab may be present. The yolk sac is not absorbed and is congested. Occasionally peritonitis with foul smell is present. The chicks which survive are stunted and emaciated.

DISEASE DUE TO FAULTY MANAGEMENT

POSSIBLE CAUSES AND REMEDIES

The disease is associated with bad hygiene in the incubator and hatchery.

Careful control of temperature, humidity and sanitation in the incubator will prevent the disease. There is no specific treatment for chicks. Antibiotics may be of some help.

17 NEWCASTLE DISEASE

a CAUSE AND TRANSMISSION	b DISTRIBUTION, SPECIES AND AGE GROUP AFFECTED	c DURATION AND MORTALITY	d CLINICAL SYMPTOMS
Newcastle Disease Virus (NDV): a myxovirus. Spread within the flock is by aerosol. Nasal and mouth discharges contaminate feed and water. Spread by equipment.	Reported from all poultry producing nations of the world. Affects all ages, mainly chicks.	Outbreak of the disease may be so acute as to kill up to 100% of the chicks. In older birds the mortality is 30% to 40%. Mortality depends on virulence of the strain of virus and immunological status of the birds.	Signs are respiratory or nervous or both. Respiratory signs are mainly coughing and gasping. Nervous signs usually follow the respiratory and may appear in a day or two sometimes until after a week. These include spasms, muscular tremors, dropping of wings, paralysis of legs, twisting of neck and head, circling and walking backwards. There may be complete or partial cessation of egg production in laying flocks.

NEWCASTLE DISEASE (Continued)

e POSTMORTEM LESIONS	f TREATMENT	g PREVENTION AND CONTROL	h SPECIMEN REQUIRED FOR DIAGNOSIS
Changes are variable. There are petechial haemorrhages on the serous membranes. Dark red haemorrhagic lesions are seen on intestinal mucosa. These may vary from small red areas to approximately more than one centimeter large. On occasions diphtheroid necrotic areas on intestinal mucosa may be observed. Haemorrhages may be present on glandular mucosa of the proventriculus and on cecal tonsils. Congestion and mucoid exudate may be present in respiratory tract. Occasionally trachea may show haemorrhages. Airsacs may be cloudy and at times may contain a catarrhal or caseous exudate.	No specific treatment.	Basic precautions by sanitary management are necessary to exclude the disease from poultry operations and to prevent its spread. Vaccination of the flock at scheduled intervals is a must.	1. Freshly dead birds packed in ice. 2. Lung, trachea, liver, spleen, brain and spinal cord in 10% formalin and 50% glycerine. 3. Serum from flock.

18 SALMONELLOSIS (A) PULLORUM DISEASE

a CAUSE AND TRANSMISSION	b DISTRIBUTION, SPECIES AND AGE GROUP AFFECTED	c DURATION AND MORTALITY	d CLINICAL SYMPTOMS
Bacteria Salmonella pullorum. Spread (1) chiefly by egg transmission from infected hen to chick. (2) May also spread by direct contact. Bowel discharge from infected birds contaminate surroundings.	Disease is common in all poultry producing areas of the world but organised control measures have resulted in virtual eradication from many countries. Mainly disease of chickens but infection is observed in Guinea fowl, ducks, turkeys and other avian species. Affects all age groups.	Mortality is high in young chicks and moderate to high in older birds. Duration is prolonged with repeated outbreaks.	Egg or hatchery transmitted infection results in mortality during first few days of life and continues to 2-3 weeks of age. Chicks huddle near the heat source, appear sleepy with loss of appetite, drooping wings, commonly develop accummulation of chalk white excreta pasting near the vent. Survivors may be greatly stunted in growth, and become carriers without any symptoms and harbour infection in the ovary. In adults the disease does not manifest an acute infection as a rule. Disease spreads within the flock for a long period without any signs. Eggs laid by them produce infected progeny. Carriers are less resistant to concurrent flock diseases. Losses result from a decrease in egg production, fertility and hatchability.

SALMONELLOSIS (Continued)

e POSTMORTEM LESIONS	f TREATMENT	g PREVENTION AND CONTROL	h SPECIMEN REQUIRED FOR DIAGNOSIS
In chicks un-absorbed yolk sac, the contents of sac may be yellowish with creamy and cheesy consistency. Liver and spleen may be enlarged. with focal necrotic foci. Greyish necrotic foci are present on lung, gizzard, heart, large intestine and cecum. The lesions in adults include mis-shapen discoloured cystic ova. Quite frequently pericarditis and peritonitis are observed. Acute infections are indistinguishable from fowl typhoid.	Sulphanamides, antibiotics and other antibacterial drugs will reduce mortality but will not eliminate the infection from the flock. Treatment always leaves behind carriers.	Eradicate adult carriers from the flock by salmonella blood testing programme. Destroy visibly sick and all carrier birds. Complete clean up of the farm is always advisable to get rid of the problem.	1. Blood and serum sample from adult birds packed in ice. 2. Freshly dead chicken on ice. 3. Piece of liver and intestine in 10% formalin.

19 SALT POISONING

DISEASE DUE TO FAULTY MANAGEMENT

CLINICAL SYMPTOMS AND PATHOLOGICAL FEATURES

Excessive amounts of salt in the ration is toxic to chickens. The lethal dose is 4mg/kg of body weight. The signs of salt poisoning are increased thirst, difficult respiration, pronounced muscular weakness with inability to stand and convulsive movements before death. Postmortem shows congestion and haemorrhages throughout the digestive tract, congestion and blood stained appearance of muscle, liver, kidneys and lungs. There may be generalised oedema and pericarditis.

DISEASE DUE TO FAULTY MANAGEMENT

POSSIBLE CAUSES AND REMEDIES

Feeding excess salt in the ration, salted vegetables etc.,

are the main cause of this condition.

20 VITAMIN DEFICIENCY:

VITAMINS	CLINICAL SYMPTOMS AND PATHOLOGICAL PICTURE OF DEFICIENCY
Vitamin A	Vitamin A is essential for normal development and repair of all epithelial structure and the bones. Lack of vitamin in young chicks results in poor growth, ruffled feathers; lack of yellow pigment in shanks and beaks; keratinisation of skin; lachrymation, swelling around the eyes and deposition of cheesy material under the eyelids; pustules in mouth, oesophagus, crop and respiratory tract. Deficiency in mature chickens develops more slowly and results in drop in egg production, decrease in hatchability and high mortality of chicks during the first two weeks.
Vitamin D	Vitamin D is required for normal absorption and metabolism of calcium and phosphorus. Deficiency produces rickets and osteoporosis. Signs of rickets in growing chickens are disinclination to walk, leg weakness with a lame stiff legged gait, retardation of growth; enlargement of hock joints; swelling and beading at the end of ribs, marked softening of skeleton including beak, bending of sternum and spinal column and thickening of extremities of long bones. In laying chickens prompt reduction of both egg production and hatchability, thin or soft shelled eggs, rubbery breast bones and beading at the end of ribs.
Vitamin E	The deficiency of vitamin E produces encephalomalacia, exudative diathesis and muscular dystrophy in chicks. It is also required for normal reproduction and embryonic development. Encephalomalacia is a nervous derangement, seen usually between 2nd and 5th week of the chicks life characterised by ataxia, sudden prostration with legs outstretched and toes flexed, lack of coordination in movement, walks in a drunken fashion, lateral twisting of head with paralysis, death soon follows. In mature chickens no outward signs of deficiency appear even after a prolonged period. However, deficiency leads to loss of fertility in males and poor hatchability in breeding hens.

VITAMIN DEFICIENCY (Continued)

SOURCE OF VITAMINS	AMOUNT RECOMMENDED PER KG OF FEED
Berseem, legumes, Alfalfa or other grass meal, fish oils, yellow corn, palm oil, commercial vit A concentrate.	Chicks, growing pullets 1500 I.U. Laying and breeding hens 4000 I.U. Turkeys (All age groups) 4000 I.U.
Fish oils, vitamin D3, sunlight, Irradiated animal sterols.	Baby chicks and growers 200-500 I.C.U. Laying and breeding hens 500 I.C.U. Turkeys (All age groups) 900 I.C.U. (I.C.U. International chick unit)
Cotton seed oil, peanut oil, wheat germoil, soyabean oil and grains. Only stabilised fat should be used in the feed. Bad storage of feed causes destruction of vit E. The requirement of Vit E will vary depending upon the type and level of fat in the diet and level of selenium. The role of this vitamin is interrelated with selenium for prevention of exudative diathesis. In another role it is interrelated with selenium and cystine for prevention of muscular dystrophy. (see selenium)	Chicks, growers and hens 10-15 I.U. Turkeys (All age groups) 10 I.U.

VITAMIN DEFICIENCY (Continued)

VITAMINS	CLINICAL SYMPTOMS AND PATHOLOGICAL PICTURE OF DEFICIENCY
Thiamin (Vit BI)	Deficiency of thiamin leads to extreme loss of appetite, polyneuritis and death. The onset is sudden in young chicks and gradual in mature birds. The signs progress by paralysis of muscles of legs, wings and neck. Symptoms include retraction of head, unsteady gait, and dropping wings. The chicken sits on its flexed legs and draws back the head in a typical "Stargazing pose".
Riboflavin	Riboflavin is essential for normal functioning of the nervous system of the growing chick. The characteristic sign in affected chicks and poults is "curled toe paralysis". They sit on their hocks with the aid of wings. Atrophy of leg muscles, encrusted eyelids and mouth may be seen. In severe deficiency marked swelling and softening of brachial and sciatic nerves are observed. There is distinct inward curling of toes on one or both feet. Other signs are stunted growth and diarrhoea with a high mortality approximately after 3 weeks. A deficiency in hens results in decreased egg production, increased embryomic mortality, dead in shell chicks with curled toe and clubbed down. Some of the embryos are oedematous. The down feathers in the areas of neck and vent fail to emerge properly causing feathers to coil; this typical abnormality is called "clubbed down".
Pantothenic acid	Deficiency of this vitamin results in dermatitis, broken feathers, perosis, poor growth and mortality. Chicks are emaciated and crusty scale like lesions appear in the corner of the mouth and the margin of eyelids. The latter are frequently stuck together by a viscous exudate. There is loss of feathers from head and neck, with slow sloughing and keratinisation of the skin. Small cracks and fissures develop between the toes. In some cases wartlike protuberances develop on the balls of the feet. A pus like substance in the mouth may be observed. In breeding hens egg production and hatchability are affected. A few hatched chicks show slow growth and their mortality is high.
Biotin	In chicks typical signs of deficiency include broken feathers, bending of metatarsus, dermatitis of feet, scab like lesions in corners of mouth and edges of eyelids as seen in pantothenic acid deficiency. In laying hens dificiency causes reduced hatchability. Many embryos which fail to hatch have bone deformities characterised by reduced size, a parrot beak, crooked tibia and twisted tarsometatarsus.

VITAMIN DEFICIENCY (Continued)

SOURCE OF VITAMINS	AMOUNT RECOMMENDED PER KG OF FEED
Grains specially grain by-products, oil cake meal. Practical rations contain adequate amounts and deficiency of this vitamin rarely occurs. Chickens respond in a matter of a few hours to oral administration of this vitamin.	Chicks, growers hens and all ages of turkey 1-2 mg.
Milk products, fresh greens, Alfalfa meal, yeast, fermentation products and synthetic riboflavin.	Chicks growers and laying hens 2-4 mg.
Peanut meal, yeast, liver meal, Rice bran, molasses. The signs of pantothenic acid deficiency are difficult to differentiate from those of biotin dificiency. Thus in making differential diagnosis, it is necessary to examine the composition of the diet. This can be checked by feeding the diet to two groups of chicks supplementing for one group biotin and the other with pantothenic acid.	Chicks, growers and laying hens 5-10 mg. Poults and turkeys 10-15 mg.
Molasses, grains and grain products, fresh green and alfalfa meal.	Chicks and growers 0.1 mg. Laying hens 0.15 mg. Turkeys 0.3 mg.

VITAMIN DEFICIENCY (Continued)

VITAMINS	CLINICAL SYMPTOMS AND PATHOLOGICAL PICTURE OF DEFICIENCY
Choline	In addition to being necessary for the prevention of perosis, it has a role in growth and fat metabolism. Its deficiency in spite of adequate amount of manganese, biotin and folic acid, results in perosis and retardation of growth. Perosis, a condition seen in growing chickens is characterised by pin point haemorrhages in the hock joint, flattening of tibiometatarsal joint, twisting and bowing of metatarsus resulting in slipped tendon. When this condition exists, the leg cannot support the weight of the bird. In adult chickens and turkeys the deficiency is rare. However, choline is required for good egg production and high hatchability.
Pyridoxin (Vit B6)	The deficiency of this vitamin in chicks results in poor growth, perosis and characteristic nervous signs. Nervous signs are characterised by jerky movement of legs, extreme spasmodic convulsions usually terminating in death. These signs are of greater intensity compared to those seen in encephalomalacia. In adult birds deficiency causes loss of egg production, hatchability, loss of weight and death.
Vit B12 (Cobalamin)	The deficiency of this vitamin hardly occurs in those chickens who have free access to their droppings. No specific signs for deficiency of this vitamin have been demonstrated. General signs of deficiency include loss of feed consumption, lack of growth and poor feathering in chicks and growers. In adult birds the deficiency causes decreased feed efficiency, reduced hatchability and high embryonic mortality.
Folic acid	In chicks the signs are retarded growth, very poor feathering, anaemia, perosis, feather depigmentation in coloured breeds and excessive mortality. Deficiency in breeding birds causes reduced egg production, low hatchability and marked embryonic mortality. Embryos hatched from eggs deficient in vitamin show mandible defects and bending of tibiotarsus.
Niacin (Nicotinic acid)	Chicks and poults fed a diet deficient in this vitamin develop an enlargement of hock joint and bowing of legs similar to perosis. The tendon of Achilles rarely slips from its condyles. This is a differentiating feature of this condition from perosis seen in manganese or choline deficiency. In laying birds deficiency causes loss of weight, lowered egg production and hatchability.
Vitamin K	Signs include accumulation of blood in the subcutaneous tissues, haemorrhages on the legs, breast and wings and may be seen on any part of the body, either spontaneously or as a result of injury.

VITAMIN DEFICIENCY (Continued)

SOURCE OF VITAMINS	AMOUNT RECOMMENDED PER KG OF FEED
Soyabean meal, wheat bran, yeast, fish meal, liver meal, meat scrap, oil cake and synthetic choline.	Chicks, growers and hens 1500-1900 mg. Poults and turkeys 1800 mg.
Cereal grains, grain products, Alfalfa meal, animal products.	Chicks, growers and laying hens 2-5 mg. Poults and turkeys 4-5 mg.
Fish meal, animal protein, milk products, dried cow manure and fermentation products.	Chicks and turkeys of all age groups 5-10 microgram (ug).
Greens, Alfalfa meal, wheat bran and synthetic folic acid.	Chicks and turkeys (all groups) 0.5-1 mg.
Corn, soyabean meal and synthetic niacin.	Chicks 10 mg. Hen 30 mg. Turkeys 50-80 mg.
Greens, Alfalfa meal and cereal grains. Alfalfa meal at the rate of 2.5% in the feed usually prevents the deficiency.	Chicks 0.6-1 mg. Turkeys 0.5-0.7 mg.

21 MINERAL DEFICIENCY:

MINERALS	CLINICAL SYMPTOMS AND PATHOLOGICAL PICTURE OF DEFICIENCY
Calcium and Phosphorus	In growing birds the signs are very similar to those of Vitamin D deficiency. Birds become lame with a stiff-legged gait and show retardation of growth and ruffled feathers. The bones are rubbery and the joints tend to be enlarged. Rickets develop and some of the birds may show paralysis. In laying birds the skeleton is gradually depleted of these minerals becoming osteoporotic. In marked deficiency this may lead to paralysis. The bones are thin, deformed and more prone to fractures. A few thin shelled eggs with lowered production and hatchability are produced.
Manganese	The deficiency in young chickens causes perosis which is malformation of the hock joint. The main features, as already described under choline deficiency are swelling and flattening of the hock joints, twisting and bending of the distal end of the tibia and of the proximal end of the metatarsus and finally slipping of the gastrochenium tendon from its condyles. A shortening of the legs, wings and spinal column is apparent. In adult chickens a diet deficient in manganese tends to show reduced egg production, hatchability and viability of chicks. The shells of their eggs become thinner and less resistant to breakage due to poor calcification of the shell. Embryonic mortality usually starts after the 10th day of incubation and dead embryos show bone abnormalaties characterised by short thickened legs, short wings, parrot beak, a globular contour of head, protruding abdomen.
Zinc	In growing chicks deficiency causes retardation of growth and poor feathering. Long bones and hock joints become thickened and enlarged. The skin particularly on the foot becomes dry and thickened. In mature chickens Zinc deficiency affects egg production and hatchability. Embryos show wide range of skeletal abnormalities, such as curvature of spines, shortened and fused thoracic and lumber vertebrae, sometimes no viscera and skin.
Selenium	Deficiency in growing chickens causes exudative diathesis, usually between 5 and 11 weeks of age. The subcutaneous oedema results in weeping of the skin seen on the inner surface of thighs and wings. Haemorrhages on the musculature, intestine and other visceral organs may also be noticed. Oedematous fluid in the subcutaneous part and abdomen may be blood tinged. In laying hens deficiency affects egg production.

MINERAL DEFICIENCY (Continued)

SOURCE OF MINERALS	AMOUNT RECOMMENDED PER KG OF FEED
Soluble grits (Limestone grits containing chiefly $CaCo_3$) which is soluble in acid medium of crop. It differs from insoluble grits (gravel, granites, sand etc) which acts only mechanically in the gizzard.	For growing chicks Ca 1% P. 0.6% Ca/P ratio 2:1 For poults Ca 2% P. 1% Ca/P ratio 2:1 For laying hens and Turkeys Ca 2.25% P. 0.75% Ca/P ratio 3:1
Wheat bran and other wheat products, Alfalfa meal, soyabean meal and manganese compounds.	Chicks and poults 55 mg. Hens and Turkeys 30-35 mg.
Meat, fish meal and Zinc compounds (Zinc oxide and Zinc carbonate).	Chicks and poults 50-70 mg. Hens and Turkeys 65-70 mg.
Feed grown on high selenium soils are good sources of selenium. Dried brewer's yeast and a chemical compound (sodium selenite) are also good sources (see also Vit E).	Chicks and poults 0.15 to 0.2 mg. Caution: as little as 10 mg of selenium per kg of feed is toxic.