DISEASES OF POULTRY
(INCLUDING CAGE BIRDS)

BY

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SECOND EDITION

BRISTOL: JOHN WRIGHT & SONS LTD.

1969

Distribution by Sole Agents:
United States of America: The Williams & Wilkins Company, Baltimore
Canada: The Macmillan Company of Canada Ltd., Toronto

First edition, Published in Ceylon, 1966
Second edition, 1969

SBN 7236 0223 9

PRINTED IN GREAT BRITAIN BY JOHN WRIGHT AND SONS LTD.
AT THE STONEBRIDGE PRESS, BRISTOL, BS4 5NU

PREFACE TO THE SECOND EDITION

THE favourable response given to the first edition published in 1966 encouraged the author to prepare the second edition within two years of the first. In the second edition, the author has rectified errors, and included omissions to bring the book up to date, taking into consideration the criticism made of the first edition. A special effort was made to include data on published papers that reached the author's hands till the end of September, 1968.

The book has been revised to meet the requirements of the veterinary student and the field veterinarian and as such does not deal with the details of the laboratory procedures of diagnosis which are given in the standard reference work on the subject edited by H. E. Biester and L. H. Schwarte and also in the U.S. National Academy of Science Publication No. 1038.

The chapters on the avian leucosis complex and avian mycoplasmosis have been completely rewritten in view of the important developments made during the last two years, and Marek's disease has been treated as a separate entity. The avian sarcomas have been dealt with under the leucosis complex to indicate the close aetiological relationship of the former to the latter. Several other chapters especially those on coccidiosis, avian encephalomyelitis, and fowl cholera have been revised extensively. Since the first edition was written, several diseases have been described. In the second edition, these diseases, e.g., virus conjunctivitis of chicks, turkey haemorrhagic enteritis, turkey syndrome 65 (TS 65), goose septicaemia, goose plague, and infections with C.E.L.O. virus, avian entero and reo viruses, and turkey respiratory virus have been included.

As in the first edition there are four separate sections in the book, dealing with the diseases of the domestic fowl, the turkey, the duck, goose, and some other birds, and cage birds. The section on diseases of the domestic fowl is more comprehensive than the others.

The chapter on the anatomy and physiology of the domestic fowl has been deleted and one on post-mortem examination added. It is hoped that the data given in the tables of this book will provide a good source of easily assimilable information required by the veterinary undergraduate and the field veterinarian.

Peradeniya, Ceylon,
November, 1968. P.S.

ACKNOWLEDGEMENTS

IT was possible to publish the first edition of this book largely because of Professor C. A. McGaughey, Dean of the Faculty of Agriculture and Veterinary Science, University of Ceylon, from 1952 to 1965, who gave facilities for the study of poultry diseases, and useful advice based on his experience in the United Kingdom. The major part of the first edition was completed in the University of California, Davis, California, during 1963–4, when the author was on leave and was holding a Fulbright Research Fellowship. The second edition was prepared while the author was at the University of Ceylon, and it is a pleasure for me to thank my colleagues in the Department of Veterinary Science, University of Ceylon, for their co-operation and for going through the proofs, and Dr. P. Kulesegaram of the Veterinary Research Laboratory, Peradeniya, Ceylon, for helpful suggestions.

I also wish to thank the following: Messrs. John Wright & Sons Ltd., Bristol, and Dr. W. A. Pool for permission to reproduce *Table* 4; the Editor, *Veterinary Record*, and the late Dr. W. P. Blount for permission to reproduce *Table* 15; the Editor, *Avian Diseases*, and Dr. R. F. Gordon for permission to reproduce *Table* 2; the British Veterinary Association for permission to use extracts from three tables in the *Handbook on the Treatment of Exotic Pets, Part I;* Dr. J. van Zandt of the Hy-line Poultry Farm, Iowa, U.S.A. for *Figs.* 6, 8–10, 20, and Messrs. Bayer Ltd., Leverkusen, Germany, for *Figs.* 1, 2, 11, 15, 16–19.

I am indebted to Mr. L. G. Owens and Messrs. John Wright & Sons Ltd., Bristol, for their ready co-operation and the very useful suggestions given in the preparation of the second edition, and to Miss S. Dissanayeke for the secretarial assistance and typing.

LIST OF ILLUSTRATIONS

LIST OF TABLES

DISEASES OF POULTRY

SECTION A

DISEASES OF THE DOMESTIC FOWL

CHAPTER I

GENERAL

ACCORDING to zoological classification, the domestic fowl (*Gallus domesticus L.*) belongs to the family Phasianidae of the order Galliformes in the class Aves.

Being a bird it is naturally designed for flying, therefore the whole organization, both internal and external, is designed for flight.

The domestic fowl developed from its early ancestral forms in South or Central India. The other related species are:—

1. *Gallus gallus* (Linné) or *Gallus bankiva*, the Red Jungle Fowl.
2. *Gallus lafayettei* (Lesson), the Ceylon Jungle Fowl.
3. *Gallus varius* (Griffith), the Javan Jungle Fowl. All exist in South East Asia. The Ceylon Jungle Fowl is found in Ceylon only, while the other species show a slightly wider distribution.

Because of the higher body temperature, which varies from 105 to 107° F. (40·6–41·6° C.) and other physiological factors, it is not susceptible to quite the same organisms as are the mammals, and therefore, a vast majority of the diseases of the domestic fowl are peculiar to the avian species.

DISEASE AND CAUSES OF DISEASE

Disease is an injurious deviation from normal and is in most cases manifested by clinical signs and symptoms, decreased production, reduced growth rate, and higher mortality. When the changes due to a disease are not clearly evident, the condition is referred to as subclinical disease. In poultry disease, such conditions exist often, as in certain forms of infectious bronchitis in the adult, or certain types of Newcastle disease where the only clinical evidence is a decrease in egg production or the presence of haemagglutinins in a flock which has not been vaccinated against Newcastle disease.

A disease may be peracute, acute, subacute, chronic, or subclinical according to the severity of the manifestations of the disease. In poultry practice, it is not customary to examine individual birds in detail clinically. However, the flock history, mortality and morbidity patterns, the post-mortem lesions, and bacteriological and other laboratory findings are of great significance in the diagnosis of a

disease. Therefore it is desirable in most instances to sacrifice a few acutely ill birds in order to perform a post-mortem examination and make a specific diagnosis rather than attempt to treat them.

The specific causes of disease are variable. The preventive and therapeutic measures largely depend on the specific causes. Disease may be caused by a variety of organisms or factors. They include viruses and virus-like organisms (psittacoids, mycoplasmata, and PLV organisms), bacteria, fungi, parasites, deficiencies, poisons, miscellaneous conditions and changes in environment, hereditary conditions, and neoplasms.

Virus Infections.—The most widespread infections are caused by viruses. These include Newcastle disease, fowl pox, infectious bronchitis, and others. There is no specific treatment for any of them. Control of these diseases depends on vaccination and good hygiene. Sometimes diagnosis of these infections is difficult and can be made only in a fully equipped laboratory.

Bacterial Infections.—These include salmonelloses, fowl cholera, and others. Most of these infections can be treated but often it may not be economical to do so. Prevention is more important than treatment. Diagnosis is not difficult: in most cases it is possible to isolate the offending bacterium, identify it, and prove its pathogenicity. It may be necessary in some instances to establish Koch's postulates before it is possible to incriminate a bacterium as the specific cause of a disease.

Fungal Infections.—These include aspergillosis, moniliasis, and others. Treatment is often difficult and not worth while except in moniliasis. Prevention is far more important than treatment. In the tropics, fungal infections are more important. In the past decade, mycotoxicosis caused by fungal toxins has become an important problem. This was pin-pointed when it was proved that aflatoxin in the fungus *Aspergillus flavus* which grows on groundnut meal, can produce marked toxicity and even neoplasms, chiefly in the liver.

Parasitic Infections.—These may be caused by protozoa or metazoa. The most important protozoan infection is caused by various species of coccidia. These infections can be prevented or treated; the more satisfactory method is to control the infection by the use of coccidiostats and permit natural immunity to develop in the birds.

The metazoan infections may be caused by the nematodes or round worms, the trematodes or flukes, the cestodes or tapeworms, and the arthropods, which will include insects, ticks, mites, and lice. None of them are serious or widespread infections except that due to the nematode *Ascaridia galli.* This parasite can be controlled by good hygiene, a properly working litter, and the strategic dosing of the bird with a piperazine compound.

Deficiency Diseases.—These may be caused by a deficiency of vitamins or minerals. The most common deficiencies encountered in poultry are those due to deficiencies of vitamins A and B_2. These deficiencies vary from country to country. Thus, in the U.K. and other temperate countries, vitamin D deficiency is very

common and vitamin E deficiency has become a widespread problem in most countries, especially in broilers. These can always be remedied by a proper diet, and addition of excess of vitamins and antioxidants to prevent the oxidation of labile vitamins.

Poisoning.—In poultry reared in confined spaces poisoning is not common. However, modern insecticides, weed-killers, and grain preservatives, if used carelessly, and also fungal toxins, can cause poisoning.

Miscellaneous Conditions.—These include diseases of doubtful aetiology or diseases caused by multiple factors such as haemorrhagic syndrome, chick oedema disease, and systemic diseases like peritonitis, ascites, and gout. Very little can be done to control them as their exact cause is not known.

Hereditary Factors.—These do not play an important role in poultry disease due to the rigorous culling practised. However, owing to intensive inbreeding which may be practised to establish certain lines, recessive heterozygous factors may become homozygous and become manifest in lethal conditions. It is important, therefore, to eliminate all these heterozygous individuals so that these factors may not be transmitted. Over 26 lethal or semilethal factors have been described in the domestic fowl. None of them is of importance to the average poultry keeper. However, the breeders should be aware of them, and some of them have been described in the appropriate chapter (*see* p. 146).

Neoplasms.—It is definitely proved that some neoplasms, i.e., those of the avian leucosis complex, are caused by a group of closely related viruses. This disease complex can be controlled by breeding from resistant or disease-free stock. However, the causes of the various other tumours of the domestic fowl are not known. Therefore, no definite method can be laid down to reduce the incidence of these neoplasms.

SIGNIFICANCE OF DISEASE AND DISEASE PREVENTION

If poultry rearing is carried out properly under good systems of management, the stock good, and the rations adequate and well balanced, a major factor that determines the margin of profit is the presence or absence of disease.

The principal poultry diseases reported in the North Central Poultry Disease Conference area in the U.S.A. in 1966, in decreasing order of incidence were, visceral lymphomatosis*, coccidiosis, chronic respiratory disease and its various manifestations, paratyphoid, and paracolon infections. This pattern of disease incidence is probably true of most parts of the world.

The total estimated annual loss to the poultry industry in the U.K. through disease in 1966, was about £48,480,000 (Gordon, 1967). It has also been estimated that the value of anticoccidial drugs alone, used in the U.K. in 1966, was £1,500,000 while the annual cost of Newcastle disease vaccines was about £1,000,000.

* Includes lymphoid leucosis and visceral Marek's disease.

The total annual loss in the U.S.A. due to leucosis which is responsible for about 30 per cent of the total losses due to non-parasitic diseases was $65,000,000, and that due to *Mycoplasma gallisepticum* was $125,000,000 (Kesteven, 1965).

The importance of the different diseases varies considerably in the different parts of the world, depending on the systems of management and the types of poultry reared. Thus, with the intensive broiler industry in the United States and other advanced countries, mycoplasmosis (C.R.D.) has become a very important disease problem; on the other hand coccidiosis is a very widely prevalent disease wherever birds are intensively housed. However, with the use of modern coccidiostats, the incidence of the disease can be controlled.

In South East Asia, Newcastle disease (Ranikhet disease) decimated the poultry population formerly, and rearing poultry on an economic or intensive basis was difficult due to the high mortality caused by this disease and its high incidence. However, with modern vaccines which can be easily administered on a large scale with a minimum of effort and risk, this disease has been controlled satisfactorily in many parts of the developing countries. Another disease which is becoming increasingly important is avian leucosis complex and the death-rate due to this disease in most countries varies from 5 to 15 per cent. In Russia the reports indicate that it is even higher. In two areas in the United States the condemnation of broiler carcases due to leucosis accounted for 15–34 per cent of birds killed. Very little has been done in the past to reduce its incidence: treatment is of no avail; no vaccine is available at present. Again, due to modern methods of selective breeding, aimed at breeding birds with increased resistance from flocks where disease incidence was low, it has been possible to establish flocks where the incidence is minimal or even non-existent.

The effects of disease may be dramatic, such as the high mortality that may occur in the virulent forms of Newcastle disease in Asia and Europe. On the other hand, some diseases may be more insidious such as the very mild forms of Newcastle disease in Australia or infectious bronchitis in layers. Both these diseases may cause dramatic reduction in egg production. Other diseases such as mycoplasmosis retard growth and production without any appreciable mortality. Certain diseases are associated with bad management—helminthiasis (i.e., *Ascaridia galli* infection) is one such problem. This is usually seen in birds which are crowded together, or are reared on litter which is highly contaminated and moist, thus producing ideal conditions for the development of the egg to the infective stage.

Certain diseases such as bird malaria and *Leucocytozoon* infections may be restricted to certain geographical areas such as the tropical or sub-tropical regions. Parasitic infections such as those due to *Argas persicus* may be highly localized and restricted to certain flocks. These infections can be easily eradicated.

Thus it is clear that it is not possible to give generalizations on the effect of diseases. The figures from a poultry clinic in Germany for a 10-year period from 1953 to 1962 are fairly representative (*Table* 1). However, conditions may vary and the diseases and their effects may vary from one area to another. The figures in *Table* 2 give the analysis of post-mortem reports at the Houghton Poultry Research Station, England, for 1964 and 1965. The figures in these two tables will indicate the differences in the incidence of poultry diseases in the two countries.

Table 1.—Common Causes of Poultry Mortality over a 10-Year Period at the Berlin Institute and Clinic for Poultry Diseases*

Cause of Death	Mortality (per cent)	
	Large Poultry Enterprises	Small Holdings
Chicks and Pullets		
Faulty nutrition and husbandry	27	30·7
Coccidiosis	30	3
Pullorum disease	22·8	27·7
Fowl paralysis	6·4	2·7
Ascaridiosis	3	2
Gout	2·5	2·5
Ectoparasites	0·6	3·8
Hens		
Salpingitis and peritonitis	12·5	10·8
Leucosis	11	9·5
Faulty nutrition and husbandry	10·7	6·5
Fowl pox	7	2·4
Fowl paralysis	6·8	2·7
Tuberculosis	6·6	8
Infectious coryza	6·3	2
Ascaridiosis	5·3	1·8
Gout	4·3	2·4
Unknown causes	2·8	16·4

* These figures include a total of 6127 chicks and pullets and 8375 hens. The figures are those given by Prusas (1964) and abstracted in *Vet. Bull.*, **34**, Abstr. No. 3845.

Table 2.—Analysis of Post-mortem Reports of the Houghton Poultry Research Station, England, for 1964 and 1965*

Cause of Death	Chickens					
	0–4 weeks old		4–20 weeks old		Adults	
	1964	1965	1964	1965	1964	1965
Bacterial Diseases						
Pullorum disease	3	1	—	2	—	—
Salmonelloses	165	109	—	1	—	—
Fowl typhoid	—	1	1	—	5	—
Erysipelas	—	—	—	—	—	—
Tuberculosis	—	—	2	—	13	10
Pasteurellosis	—	—	—	—	—	—
Infectious sinusitis	—	—	—	—	—	—
E. coli septicaemia	—	—	10	4	2	1
E. coli granuloma	—	—	3	—	13	4
Staphylococcal arthritis	—	—	24	18	13	5
Chronic respiratory disease and coryza (undifferentiated)	1	—	36	14	42	34

* Data from *Avian Diseases*, **10**, 377.

Table 2.—Analysis of Post-mortem Reports of the Houghton Poultry Research Station, England, for 1964 and 1965 (*continued*)

Cause of Death	Chickens					
	0–4 weeks old		4–20 weeks old		Adults	
	1964	1965	1964	1965	1964	1965
Viral Diseases						
Newcastle disease (reported)	9	2	37	17	36	22
Newcastle disease (isolated)	2	1	8	1	7	2
Infectious bronchitis	3	1	37	16	74	48
Infectious laryngotracheitis	—	—	—	1	7	3
Fowl pox	—	—	—	—	2	—
Avian encephalomyelitis	31	15	14	3	4	—
Avian leucosis complex						
Lymphoid	1	—	3	3	124	32
Myeloid	—	—	2	—	5	2
Marek's disease (neural)	1	3	160	101	154	78
Marek's disease (visceral)	—	—	12	16	27	27
Marek's disease (ocular)	—	—	—	—	2	3
Marek's disease (with leucosis)	—	—	—	—	2	—
Osteopetrosis	—	—	2	—	2	—
Duck virus hepatitis	—	—	—	—	—	—
Infectious synovitis	—	1	2	—	4	—
Gumboro disease	38	31	19	10	1	—
Fungal Diseases						
Aspergillosis	48	14	5	1	1	—
Moniliasis	—	—	—	—	—	—
Parasitic Diseases						
Coccidiosis						
E. tenella (caecal)	7	5	36	17	6	2
E. necatrix	4	—	42	15	15	7
E. acervulina	19	6	57	27	52	17
E. maxima	2	1	16	11	6	1
E. brunetti	8	2	19	9	3	—
E. adenoides	—	1	—	5	—	—
E. meleagrimitis	—	—	1	1	—	—
E. meleagridis	—	—	—	—	—	—
Mixed infections	4	—	6	1	4	—
Helminths (worms)						
Ascaridia	—	—	25	9	71	50
Heterakis	—	—	—	—	6	—
Capillaria	—	—	8	2	12	7
Syngamus	—	—	—	—	—	—
Tapeworms	—	—	—	—	1	1
Other protozoa						
Histomoniasis	1	1	14	7	3	5
Ectoparasites						
Lice	—	—	—	—	3	1
Mites	—	—	—	—	1	—
Tumours						
Unclassified	1	—	6	3	62	40
Haemangioma	—	—	—	—	18	7

Table 2.—Analysis of Post-mortem Reports of the Houghton Poultry Research Station, England, for 1964 and 1965 (*continued*)

Cause of Death	Chickens					
	0–4 weeks old		4–20 weeks old		Adults	
	1964	1965	1964	1965	1964	1965
Nutritional Diseases						
Vitamin A deficiency	—	—	2	—	8	—
Riboflavin deficiency (curled toe paralysis)	—	3	1	—	—	—
Vitamin D deficiency (rickets)	2	4	15	5	1	—
Perosis	—	1	3	2	7	—
Osteopetrosis	—	—	5	1	61	33
Encephalomalacia (crazy chick disease)	17	—	17	3	—	—
Poisoning						
Sulpha drug poisoning	2	—	25	4	2	—
Other suspected poisons	1	1	5	10	1	—
Physiological Disorders						
Reproductive, egg peritonitis	—	1	9	—	100	50
Prolapse	—	—	1	—	36	10
Nephritis, visceral gout, kidney disorders	8	5	21	3	41	34
Digestive disorders and impactions	15	5	75	27	61	25
Non-infectious pneumonia and suffocation	2	1	6	1	1	1
Circulatory disorders	1	1	16	6	82	38
Liver disorders	1	—	8	—	25	—
Miscellaneous Conditions						
Pullet disease	1	—	—	—	17	5
Round heart disease	—	—	—	1	2	—
Haemorrhagic disease	—	—	5	6	3	1
Ruptured gastrocnemius tendon	—	—	2	2	6	1
Crooked toes	2	—	6	3	5	2
Hepatosplenomegaly	—	—	—	—	—	—
Environmental faults	124	46	16	8	12	6
Yolk-sac infection	28	16	—	—	—	5
Badly healed navels, poor vitality, and developmental faults	80	23	39	14	45	5
Cannibalism and pecking	—	1	5	3	23	18
Sundry conditions, including accidents, abscesses, cataracts, obesity, nonspecified enteritis, dehydration, and starvation	64	40	98	66	208	134
Undiagnosed and indefinite	84	32	60	41	136	68
Decomposed	16	6	17	8	27	6
Age-group totals	796	381	1083	530	1708	857
Totals 1964 3587 1965 1768						

The control of disease is centred basically round six cardinal principles:—

1. The selection of stock.
2. The practice of good hygiene and incorporation of an adequate and well balanced diet.
3. The adoption of a prophylactic programme in keeping with the requirements of the flock and the diseases prevalent in the area.
4. Measures adopted to control a disease outbreak.
5. Sound hatchery practice, especially regarding the selection of eggs and the disinfection of the incubator.
6. General preventive measures and rigorous culling.

1. Selection of Stock.—This is of prime importance, not only in connexion with control of disease but also as regards virility, ability to survive, and productivity. It is essential to see that the newly purchased birds are obtained from stock free from pullorum disease and other *Salmonella* infections. Apart from these diseases one should make sure that the parent stock are free from pathogenic *Mycoplasma* infections (i.e., *M. gallisepticum*, C.R.D., and infectious synovitis), and epidemic tremor, and that the incidence of leucosis in the parent stock is low or negligible. The ideal would be to select stock from flocks free from leucosis complex. Such an ideal is difficult to attain as yet, since this disease complex is widely prevalent in varying degrees in different countries and different flocks.

All salmonelloses including pullorum disease, *Mycoplasma* infections, and the virus of epidemic tremor are egg-transmitted (vertical transmission).

2. Good Hygiene and an Adequate and Well Balanced Diet.—Poultry should be housed in hygienically constructed buildings where adequate floor space, ventilation, and light are provided. Clean water and an adequate number of feeding and watering devices should be available. If they are housed in a deep litter building, it is of prime importance to see that the litter is working satisfactorily and that there is no build up in the litter of viable oocysts of coccidia and infective helminth eggs particularly those of *Ascaridia galli*. The litter should be dry and regularly stirred. Moist litter should be replaced with clean dry litter. It is best to rear chicks during the first 2–3 days on wire netting or on fresh dry litter. In rearing broilers the litter may be changed every time a new batch of chicks is introduced. A foot bath containing a suitable disinfectant should be kept at the entrance to every poultry farm. The disinfectant solution should be changed daily if necessary. Vermin, ants, and other insects should be controlled. Vermin play an important role in the transmission of salmonelloses; some ants and insects can act as intermediate hosts of certain tapeworms. Ectoparasites such as lice should be controlled by the use of safe insecticides. Stress factors such as draughts, shortage of water, and undue excitement of birds should be avoided.

Details of the construction of poultry houses are outside the scope of this book. However, whatever type of poultry house is built, adult birds should be provided on the average with 2 sq. ft. of floor space for smaller breeds and 3 sq. ft. for the larger breeds. They should be provided with about 12 hours of lighting, free ventilation, and a clean supply of drinking-water and food round the clock. Today most feed manufacturers provide good and well balanced rations. A well tried ration is the best one. However, for optimum results and economy, different

rations are used for chicks, growers, layers, and broilers. It is possible that even a well tried ration may fail to produce the desired results due to defective or old ingredients having been used. Therefore it is up to the manufacturer of the ration to safeguard against such defects. To overcome these defects most manufacturers add either ready-made premixes of minerals and vitamins with excess of these ingredients. Today most poultry feeds contain, in addition to the raw ingredients, vitamins and minerals, antioxidants, antibiotics, arsenicals, or other growth promoters, and specific agents such as coccidiostats or antibacterial agents used for specific purposes.

3. A Prophylactic Programme.—This would naturally vary according to the country, geographical area, and the type and extent of the flock. However, it is generally agreed that all chick rations should contain a specific coccidiostat. The most widely employed coccidiostats are zoalene and amprolium. (For intestinal coccidiosis of more mature birds sulphaquinoxaline is very useful.) The coccidiostat should be incorporated in the ration from birth to at least 3 months. It appears that the addition of broad-spectrum antibiotics as disease preventives has little value though they appear to have some action in stimulating growth in the first 10 weeks. Their beneficial effect is more noticeable in the presence of bad management. Better results can be obtained by improvement of hygiene and management.

It is advisable to immunize all chickens in the first week against Newcastle disease. However, if maternal immunity is present (i.e., if the adult stock are immune) vaccination should be postponed and performed between 2 and 4 weeks. It is an advantage in most instances to vaccinate birds against infectious bronchitis simultaneously if this disease is endemic. Several combined commercial vaccines are available. Depending on the type of vaccine used, it is desirable to give one or more booster doses before the birds come into production. Usually the first booster dose is given 1 month after the first vaccination and the second booster dose about 3 months after the first booster dose. This practice and the number of booster doses required to immunize birds for their productive life depends on the type of vaccine used. In the case of broilers, a single vaccination may be sufficient as they are invariably marketed before 3 months. Vaccination against fowl pox may be performed at about 1 month and then repeated, if necessary, before the birds come into production. Fowl pox virus vaccine of chick embryo origin is the one of choice and it has been found that this may be combined with Newcastle disease vaccine without impairing the efficacy of either. Thus, birds may be vaccinated simultaneously against both diseases.

Vaccination of birds against infectious laryngotracheitis, fowl cholera, and other diseases may be considered if they are endemic. If these diseases are not endemic, vaccination is not recommended.

The presence of mycoplasmosis, coccidiosis, and worms may interfere with the development of immunity, and may also cause some adverse reactions to vaccination. Therefore before a vaccination programme commences one should make sure that these infections have been controlled. In other instances the presence of maternal immunity may interfere with the production of natural immunity. Maternal immunity lasts 2–3 weeks. Further, older birds show a better immune response to vaccination. Therefore, it is not advisable to vaccinate very young

chicks unless it is essential. The selection of a vaccine should be based on its effectiveness and mildness. Vaccines can be administered by many routes. Administration through water is very convenient. However, some birds may not drink the water and therefore may not be immunized. Individual vaccination is very effective but is quite laborious and time-consuming if large numbers of birds are involved. Often, booster doses of vaccines have to be given to maintain life-long immunity.

It appears therefore, that it is essential to immunize all birds against Newcastle disease, fowl pox, and perhaps infectious bronchitis, irrespective of the locality and the type of flock, if these diseases are endemic in the area. An effective and safe coccidiostat must be incorporated in the ration from birth to at least 3 months. A tentative prophylactic programme to be followed is outlined in *Table* 3.

4. Measures Adopted to control a Disease Outbreak.—This will entail detection of all sick birds, isolating them, disinfection of premises, vaccination wherever possible, and treatment if this procedure is justified.

It is not possible to examine birds individually in a large flock. The best indication of the presence of acute disease is a marked loss of appetite. Invariably, acutely ill birds will refuse to eat and often isolate themselves from the rest. All sick birds should be isolated. In general in poultry practice, it is not economical to treat sick birds and in diseases such as Newcastle disease they should preferably be destroyed and the carcases buried or burnt. Treatment should be done only if they can be treated on a mass scale, i.e., in coccidiosis. Often such treatment may not be very economical, as in mycoplasmosis. In others such as salmonelloses it may be a salvage operation where the acute infection is controlled by furazolidone or a similar drug and the birds then marketed for table. If these birds are saved some of them may remain carriers and thus disseminate infection. In mycoplasmosis (C.R.D.) when the majority of the flock is affected, it is better to depopulate the flock and restock the farm after a month with healthy birds from disease-free stock.

In the event of a serious epidemic such as Newcastle disease, it may be necessary to disinfect the premises. On the other hand if the litter is heavily contaminated with worm eggs or coccidial oocysts it is necessary to replace the litter with new, dry, and non-contaminated litter.

The method of disinfection and the disinfectants used would depend on the type of poultry house and the availability of the disinfectant. The most economical and widely used disinfectants are 4 per cent sodium carbonate or 5 per cent sodium hypochlorite solutions. The other disinfectants which are popular are the coal tar derivatives or proprietary preparations made from them such as Cresol, Lysol, and Jeyes' fluid. Quicklime, lye, and formaldehyde are also widely used. Modern disinfectants such as quarternary ammonium compounds (Cetavlon and Savlon, I.C.I.) have the advantage of being non-irritant, detergent, and odourless (Cetavlon). These may be used for the disinfection of eggs (if necessary) and the incubator. Formaldehyde, caustic soda, and quarternary ammonium compounds have a virucidal and bactericidal action, while the chlorinated phenolic derivatives are active mainly against bacteria, especially *Salmonella* and the moulds. Most of these disinfectants are not effective against the oocysts of coccidia, against which 1 per cent ammonia solution is very effective. Disinfection should be done wherever

Table 3.—A PROPHYLACTIC PROGRAMME *

AGE	PROCEDURE	REMARKS
1st week to 4th week	Vaccinate against Newcastle disease and infectious bronchitis using live or dead virus vaccines	This procedure can be adopted in areas where the two diseases are endemic and where the use of live virus vaccines is permitted. The type of vaccine against Newcastle disease varies considerably (*see Table* 4). If there is likely to be maternal immunity from Newcastle disease in the chickens, vaccination may be postponed until 14–21 days. The vaccine is most conveniently given in drinking-water. B_1 strain of vaccine is recommended for the purpose. In the United Kingdom use of live virus is not permitted
1st day to 12th week	Use approved coccidiostat in mash, preferably one that does not interfere with establishment of immunity	In certain flocks especially in the United States, a coccidiosis vaccine is used, in which case it is necessary to use excess of vitamins A and K and the coccidiostat trithiodol continuously in the feed. This procedure cannot be recommended for widespread use in all countries until more information is available regarding the relative merits of the vaccines. Most manufacturers today use a coccidiostat in all chick mashes. The widely used coccidiostats are amprolium, zoalene, and metichlorpindol
4th week to 12th week	Revaccinate against Newcastle disease and infectious bronchitis *or* revaccinate against Newcastle disease and vaccinate against fowl pox for the first time using chick embryo vaccine	This procedure depends on the type of vaccine used earlier. The vaccine is most conveniently given in drinking-water A combined vaccine for this purpose can be obtained in some countries. Vaccination is done by the wingweb method or by the intramuscular or subcutaneous routes. The immunity established is usually of longer duration if the vaccines are given by these routes
6th week	Treat for round worms using a piperazine compound. If I.L.T. is endemic, an I.L.T. vaccine may be used a few days after worming	Worming need not be done if the litter is not infected and is quite dry. I.L.T. vaccine should not be used if the disease is not endemic
12th week	Revaccinate or vaccinate against Newcastle disease and fowl pox for the first time	Use vaccines that are given parenterally and induce immunity during the productive life of the birds
16th week	Treat for worms with piperazine compound	After the 16th week no further vaccination need be done if vaccines of adequate potency have been used. If vaccination against Newcastle disease was done for the first time at 12 weeks, revaccination may be necessary
20th week	Worming may be repeated if necessary	

* It is difficult to lay down a prophylactic programme which will be applicable under all conditions and in all countries. The above programme is the general pattern to be adopted and will differ in details from country to country according to the legal requirements, the diseases prevalent, and the vaccines available. In vaccinating, one should carefully follow the manufacturer's recommendations. In the European Economic Community vaccination is recommended against Newcastle disease, infectious bronchitis, and infectious encephalomyelitis.

possible in the interval between depopulation and restocking. Disinfection should be preceded by dry and then wet cleaning processes.

Whatever disinfectant is used it is desirable to see that it is an approved disinfectant and that the manufacturer's recommendations are followed.

Before the application of a disinfectant it is necessary to clean incubators and other utensils by thorough scrubbing.

Choice of disinfectants depends on the disease and the availability. Thus, addition of 200 lb. quicklime to 1 ton of litter or manure will destroy some of the pathogenic *Salmonella*, *Pasteurella multocida*, and also prevent the development of oocysts and the embryonation of *Ascaridia galli* eggs. Treatment of litter thus will prevent the breeding of flies and maggots and also reduce vermin infestation. If poultry are reared in open pens, the infected ground may be limed at the rate of 2 tons per acre.

In the event of a disease outbreak it is desirable to make an accurate diagnosis. The epidemiology, the morbidity, the mortality patterns, the symptomatology, and the post-mortem lesions taken together may, in most cases, help one to diagnose a disease. However, it may be necessary in viral infections to get the expert opinion of a poultry pathologist or a virologist in a recognized laboratory before a specific diagnosis is made. The exact methods of control including vaccination procedures depend considerably on the disease. Further, in certain countries such as the United Kingdom some diseases like Newcastle disease and fowl plague (both collectively termed 'fowl pest') are notifiable diseases and appropriate measures have to be taken according to the laws of the land.

FUMIGATION AND DISINFECTION OF INCUBATORS, BROODERS, AND CLOTHING.—For fumigation 10 per cent formalin is used as the preparation of choice. It is necessary to follow the instructions given by the manufacturer while fumigating an incubator. It is desirable to scrub the incubator with detergent or washing soda before fumigation is done. Fumigation will be most effective if the room where this is done is made air-tight and the temperature and humidity are raised to as high levels as are practicable. Contaminated clothing can be fumigated in this room.

Formaldehyde is generated by mixing 1·2 oz. commercial formalin with 0·6 oz. potassium permanganate for each 100 cu. ft. of air or incubator space. The mixing is done in a shallow but wide vessel and the gas generated is allowed to act for at least 10 minutes. The concentration of the gas may be increased two- to three-fold by proportionately increasing the ingredients. This should be done if infections such as omphalitis and those due to *Salmonella* are present. It is not advisable to fumigate the incubator while eggs are being hatched. The embryos are most susceptible to fumigation during the first 3 days of incubation. Fumigation may be done by the use of formalin alone. It is used as an aerosol at the rate of 1 fl. oz. per 150 cu. ft. As an alternative paraformaldehyde may be vaporized at the rate of 3 oz. per 1000 cu. ft.

The choice of a disinfectant depends on the effect required. Quarternary ammonium compounds are the disinfectants of choice, because they act simultaneously as detergents. The quarternary ammonium compounds, formaldehyde, and caustic soda have an anti-virus action, whereas phenolic disinfectants have a broad spectrum of activity against bacteria, including *Salmonella*, but do not have much action against viruses. Coccidial oocysts are resistant to most disinfectants but are killed easily by 1 per cent solution of ammonia.

5. Sound Hatchery Practice.—The disinfection of incubators has already been referred to. It is always best to follow the manufacturer's instructions in this matter unless they are varied due to some special circumstances. Custom hatching is not a desirable feature and is to be discouraged. It is essential to see that all eggs for custom hatching come from pullorum-free stock (whether legislation exists or not). It is also desirable to see that the eggs are from flocks free from other *Salmonella* infections. It is advisable to ascertain the health status of a flock before accepting eggs for custom hatching. One should make certain of the absence of mycoplasmosis and coli-bacillosis which may be transmitted via the egg or in the incubator to the hatched chicks and thus disseminate infection. It is desirable for hatcheries in all instances to have their own self-contained flocks free from infectious and contagious diseases, especially those likely to be transmitted through the eggs or during incubation.

6. General Preventive Measures.—On no account should new stock be introduced from unknown sources or from flocks where the health status is not definitely known. All birds which have been taken to shows should be quarantined for at least 2 weeks. Poultry shows are, unfortunately, places where diseases are frequently disseminated.

Unauthorized persons should not be allowed to enter premises where birds are kept. Birds and vermin should not be allowed to have access to the poultry runs. In Asia the itinerant vendor carrying sick birds for sale has been found to disseminate infection. Often these birds are sold cheap and therefore are bought for table by the unwary small breeder of poultry. The offal of such birds is carelessly thrown out and is thus responsible for spreading infection. Newcastle disease has often been found to have been disseminated in this way in India and Ceylon.

Contaminated feeds will also disseminate infection. This has been particularly so with *Salmonella* infection. A few years ago *Salmonella bareilly* infection was reported in Ceylon in dessicated coconut and later it was found to be a widespread infection in poultry in Ceylon. It is likely that the coconut poonac used in poultry feed was contaminated in the mills and thus spread the infection.

Any good breeder should practise rigorous culling and often it is not advisable to keep any sick or ailing birds. They should be killed and, if found to have suffered from an infectious disease, the carcases should be destroyed or buried.

Thus, in order to control disease the following principles must be strictly adhered to:—

1. All birds whether for replacement or new stock should come from healthy flocks certified free from pullorum disease and fowl typhoid. It is advisable also to make certain that such flocks do not show any evidence of *Mycoplasma gallisepticum* infection, salmonelloses, and that the incidence of avian leucosis complex is low or negligible. Once a healthy flock is established, wherever possible no purchases should be made from outside except from healthy sources.

2. Vermin and wild birds, as well as animals, should never be allowed into the building where birds are housed. Visitors should be discouraged from entering buildings.

3. The poultry houses should be sited at least 30 m. from public highways and 50 m. from other poultry houses.

4. There should be a well-planned prophylactic programme which is carried

out with every new batch of chicks. There should also be a cleansing and disinfecting programme using an approved disinfectant. Usually this is done systematically whenever a new batch of chicks arrives or in the case of incubators, according to the manufacturer's instructions.

5. The feed used should be from reliable sources, free from contamination, of good quality, and adequately balanced.

6. Finally the most important considerations that will ensure a healthy flock are the vigour, vitality, and health of the initial stock, coupled with good nutrition, adequate ventilation and housing, and sound management.

CHAPTER II

DISEASES CAUSED BY VIRUSES AND RELATED ORGANISMS

NEWCASTLE DISEASE

(Ranikhet Disease; Pseudo Fowl Plague; Fowl Pest; Doyle's Disease)

THIS is a highly infectious and sometimes fatal disease of the domestic fowl and some other birds, caused by a virus and characterized by respiratory symptoms and high mortality in some outbreaks. In others there may be nervous signs, sudden cessation of egg yield, while the mortality is low. Recent evidence indicates that the severity may vary from an acute disease causing nearly 100 per cent mortality to one which is asymptomatic. There appear to be several mutants of the original virus.

History.—In 1926 an apparently new disease was described in the Dutch East Indies. In the same year Doyle investigated a new disease in fowls in Newcastle on Tyne which he proved was caused by a virus. Doyle named the disease after the locality, and hence it is now known as Newcastle disease. About the same time a similar disease was seen to occur in Ranikhet in the United Provinces in India which was investigated and later identified as Newcastle disease. The name given to the disease in India is Ranikhet by which name it is still known in some Asian countries. Later the disease spread to other countries in the world and it was first reported in the United States of America in 1944. As the course of the disease in the United States was not similar to the classical form of the disease, it was not initially identified as Newcastle disease, and therefore it was referred to as pneumo-encephalitis, since the disease primarily affected the lungs and the brain. However, the disease is now known in the United States as Newcastle disease.

Distribution.—At present the disease has a nearly world-wide distribution, including Australia where it was reported in Queensland in 1966. The various strains of the virus isolated from Australia appear to be of very low virulence (French, St. George, and Percy, 1967). New Zealand appears to have remained free of Newcastle disease up to now. It is probably the most important poultry disease in the world. It is certainly the main disease that has always hindered the progress of the poultry industry in most Asian countries. The loss from mortality alone in the United States in 1957 due to this disease, where it runs a mild course, amounted to 21 million chickens.

Cause.—The disease is caused by a virus, named *Myxovirus multiforme* and belonging to the group of myxoviruses. It is characterized by multiformity both of the clinical picture in fowls and of the virus particles. It agglutinates chicken erythrocytes and is approximately 0·001 mm. in size. It is fairly resistant to adverse conditions. The various properties of the virus are discussed in the book edited by Hanson (1964). When compared with other viruses it is comparatively resistant

and may live up to 255 days in the egg, 288 days on egg-shells, and 255 days on the down under conditions obtained in hen houses (Temp. 11–30° C.). In man it may cause conjunctivitis and even lymphadenitis but recovery is rapid. Recently another virus related to that of Newcastle disease but differing in some characters has been isolated from the domestic fowl by Bankowski and Corstvet (1961) which they tentatively named *Myxovirus yucaipa.*

Clinical Signs and Course of the Disease.—Four forms of the disease are generally recognized:—

THE VELOGENIC FORM (the virulent form).—This is the acute form, and mortality may reach 90 per cent; the average duration of the disease is 3–4 days, and in some cases it may be as short as 1 day. The typical signs are respiratory râles, prolonged gasping respirations with outstretched neck and head, partially opened beak, nasal discharge, profuse diarrhoea, temperature elevated by 2–3° F., followed later by subnormal temperature and nervous signs such as paralysis, tremors, or torticollis. This form of the disease is characterized by very high morbidity and mortality.

THE MESOGENIC FORM (the less virulent form).—This form of the disease is less severe; the mortality varies from 5 to 15 per cent and is characterized by respiratory distress, greenish diarrhoea, and marked loss of egg production which may drop to zero. The shell is often discoloured, imperfect, or misshapen; the eggs become smaller. Nervous signs are more common and are more frequently seen in chicks. There may be paralysis of wings and legs or torticollis.

THE LENTOGENIC FORM.—This is the mild form and is characterized by mild respiratory symptoms and a rapid drop in egg production which is the characteristic sign. This may return to normal in one week. Mortality in adults may be negligible but it may reach 50 per cent in young chicks. The only lesion seen may be a mild tracheitis.

THE ASYMPTOMATIC FORM.—No clinical signs are seen in this form of the disease, but it can be recognized by serological tests when it is found that the haemagglutination inhibition titre fluctuates considerably in a given flock. This may be due to the presence of carriers. The incidence of this form of the disease appears to be increasing.

Usually an epidemic follows the pattern of one of the four forms described. An acute epidemic will remain acute until it has run through the flock or the neighbouring flocks.

Post-mortem Lesions and Diagnosis.—Details of diagnosis will not be considered here. However, in the field, the high mortality pattern with the symptoms described would enable one to suspect the presence of Newcastle disease. These symptoms are also suggestive of fowl plague, a virus disease of poultry which runs a similar acute course and also causes lesions similar to those of Newcastle disease. About 50 per cent of the birds may not show any significant changes in the internal organs on post-mortem examination. However, some of the birds will show pin-point areas of bleeding or haemorrhagic splashes in the proventriculus (*Fig.* 1). Haemorrhages will also be seen under the horny lining of the gizzard. In many such instances there may also be haemorrhagic areas or ulcers in various parts of the intestines, or haemorrhagic enteritis. Haemorrhagic areas in the colon and cloaca

are also common in some outbreaks. Varying numbers of birds will also show a mucous exudation in the trachea and nostrils. These lesions are seen in fowl plague as well as in Newcastle disease, but in countries where fowl plague has not been reported the lesions described can be taken to be very suggestive of Newcastle disease. In other cases no lesions will be visible. The lesions vary considerably according to the type of virus and the tropism it exhibits.

However, to diagnose the disease definitely, one or more of the following laboratory tests will have to be used. As these tests are fairly elaborate no description of

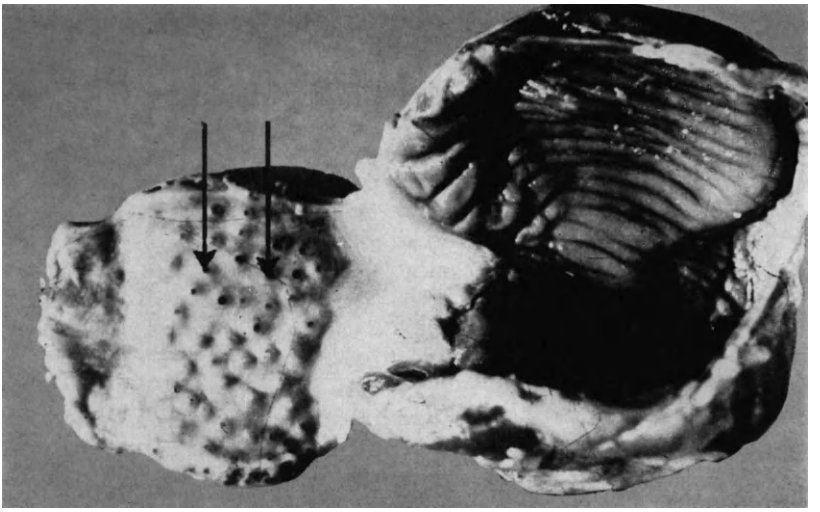

Fig. 1.—The proventriculus of a chicken with Newcastle disease showing petechial haemorrhages over the glands (arrowed).

them will be given here. Details of the tests are described by Chu (1960) and Lancaster (1963). The tests are: virus isolation and identification tests normally made by inoculating embryonating chicken eggs or tissue cultures; virus neutralization tests; haemagglutination and haemagglutination inhibition tests; immunity tests on living birds; gel diffusion tests; cross immunity tests; histopathology of trachea, and others. Further details of the diagnosis of the disease are discussed by Lancaster (1963). At least 15 new agents which belong to the myxovirus group have been isolated from chickens, turkeys, and ducks and these may complicate the diagnosis of Newcastle disease by the haemagglutination test. The serum neutralizing antibodies remain at appreciable levels for well over 8 months after natural infection, whereas the haemagglutinating antibodies reach very low levels at the end of this period.

Treatment.—There is no known treatment that is effective. However, in the event of an outbreak of the disease, the healthy birds may be vaccinated with a vaccine containing a mild strain of Newcastle disease virus. This procedure is at times likely to produce increased mortality initially. However, most authorities consider this procedure useful in a majority of cases as the efficacy of vaccination

at this stage is due to the 'interference phenomenon', where the mild virus in the vaccine competes with the virulent virus causing disease and successfully displaces it from the host cells. There is no specific treatment; immune serum may be used, but this is far too expensive for general use.

Epizootiology, Prevention, and Control.—It is important to remember that this virus is more resistant to adverse conditions than most other viruses. Further, there are strain differences and 'tropisms' (affinity of the virus for certain tissues). Under Asian conditions the most frequent source of infection is the bird that is purchased either from the itinerant vendor or from an infected or unknown source. Therefore, under no condition should such purchases be made even for table purposes.

The less frequent sources of infection are crows and the carrion they carry, visitors, and rats and mice. Visitors can carry several important disease agents of poultry.

It is worth bearing in mind that over 100 cases of infection of human beings with the virus of Newcastle disease are on record; in most cases there was only a conjunctivitis and in a few cases there was also involvement of the respiratory tract.

Newcastle disease can affect a large variety of birds, but usually only the domestic fowl and the turkey are affected seriously under natural conditions. The crow, the sparrow, the parakeet, and the owl can be naturally affected. Some carnivorous birds are entirely resistant. Under these circumstances it is important that extraneous birds are not allowed to enter the poultry yards. Infection is spread from bird to bird by direct contact or by air-borne infection. Therefore, the sick birds should be isolated, or the whole flock destroyed in the event of an outbreak, if it is a small flock. Premises may be disinfected with any of the following disinfectants: potassium permanganate 1: 5000 (1 teaspoonful in 10 gallons of water), or *N*/50 sodium hydroxide (1 teaspoonful in 1½ gallons of water), or 1 oz. of sodium hypochlorite in 4 gallons of water.

Under very adverse conditions such as in very hot weather, the virus dies in about 7 days, but under optimum conditions it may live up to a month or more. Thus once the disease has been diagnosed and controlled in a flock, it is not advisable to restock the farm except with immune birds until the lapse of one month. Recently it has been shown that high temperature and high humidity predispose birds to infection with Newcastle disease.

The conventional procedures for the fumigation of incubators such as the use of formaldehyde are satisfactory for disinfecting the incubators in the event of an outbreak of Newcastle disease.

Control can be effected by preventing the contact of the virus with a susceptible bird (this can be done on the lines indicated above) and by vaccination or by increasing the resistance. Two types of vaccines are generally employed: killed virus vaccines and live virus vaccines. The types of vaccines usually available are listed in *Table* 4. It must be stressed that in spite of considerable progress in the preparation of these vaccines, further advances are desirable.

a. KILLED OR INACTIVATED VACCINES.—These are vaccines in which the virus is killed or inactivated by the addition of formaldehyde, crystal violet, or beta-propriolactone. These vaccines are safe but the period of immunity is generally of short duration. Recent evidence indicates that formalinized oil adjuvant or aluminium

hydroxide precipitated vaccines induce immunity lasting 1–2 years if given at the recommended intervals. These vaccines are not as widely used as the live virus vaccines. All dead or inactivated vaccines have to be given parenterally. Though safer, their administration involves more labour and in the case of some, the immunity established is of shorter duration. The use of killed vaccines in the U.K. has reduced the number of outbreaks from 2176 in 1964 to 495 in 1965.

Jacotot, Reculard, and Valée (1967) have developed and tested a formalized vaccine suspended in paraffin oil which when used on chickens 10–12 weeks old produces immunity lasting for 2 years.

b. LIVE VACCINES.—These are vaccines which contain the live virus but in a weakened form. These vaccines can be given by the wingweb method, intramuscularly, intranasally, into the conjunctival sac, in drinking-water, or as inhalations (as aerosols or as dust). Usually viruses of low virulence are used to prepare these vaccines. A vaccine prepared from the F strain of virus has been found to be comparatively mild and does not cause any appreciable stress (Churchill and Blaxland, 1966). This strain of virus may prove to be the best available for the production of vaccines to be used on broilers. The Department of Agriculture, Ceylon, uses the Komarov strain of virus to prepare a vaccine. The vaccine containing this virus is meant to be used on older birds, i.e., those about 3 months old and the immunity established is expected to last over 1 year, perhaps 2 years or more. Chickens with egg-yolk containing antibodies have immunity up to 4 weeks.

Besides these, there are several other strains which have been used chiefly in the United States with very great success. These are the Haifa, van Roekel, La Sota, and F strains to mention only a few. The B_1 strain of vaccine is available in most countries and can be given by one of several routes. Roakin, Mukteswar, and Haifa strains are stronger strains of virus used in vaccine preparation and have to be used with care and under supervision. F strain is one of the mildest strains while the Haifa strain is considered to be one of the more virulent. The La Sota strain of virus appears to be capable of inducing a stronger immunity.

The type of vaccines used and the principles adopted by various countries in controlling this important disease have been listed by Pool (1963; *see Table* 4). Whatever vaccine is used, it is to be remembered that when using live vaccines the virus lives in the bird for some time and that the virus is theoretically capable of becoming pathogenic again. Further, the instructions given by the manufacturers have to be carried out carefully, and wherever possible birds in poor condition, layers, those having other intercurrent diseases, or those that are heavily infected with worms should not be vaccinated. If this is done there may be an appreciable mortality. The commercially available vaccines are mild, but the immunity is considered to be of shorter duration. However, Bankowski and Corstvet (1962) have shown that maternal immunity and residual immunity at time of vaccination with B_1 strain vaccine can markedly affect the immunity induced. Raggi and Lee (1962) have shown that intranasal vaccination with B_1 strain of vaccine at 3 months can induce immunity for over 16 months. Thus it would appear that the duration of immunity established by a vaccine is quite variable. Vaccination by intramuscular route, the wingweb method, or intranasally induces immunity of longer duration.

The relative merits of the various types of vaccines have been discussed by Gordon and Garside (1965). It would appear that the use of live vaccines is not

Table 4.—SOME COMMON NEWCASTLE DISEASE VACCINES*

VACCINE	ROUTES OF ADMINISTRATION	INTERVAL BETWEEN VACCINATION AND MAXIMUM IMMUNITY	DURATION OF IMMUNITY		DURATION OF EXCRETION OF VACCINE VIRUS AFTER VACCINATION	DURATION OF EXCRETION OF VIRUS AFTER EXPOSURE TO INFECTION	STRESS ON VACCINATED BIRDS	VIRULENCE OF VACCINE VIRUS
			Birds over 4 weeks of age	Birds under 4 weeks of age				
Dead Vaccines								
Formalin treated	Intramuscular injection	14 days	1st injection: 2 months 2nd injection: 4 months	Little response in birds under 18 days old	None	3–5 weeks	None	—
Proprio-lactone treated	Intramuscular or subcutaneous injection	6–12 days	8 months	12 weeks in chickens 10–14 days old	None	3 weeks	None	—
Live Vaccines								
B_1 strain	Intranasal, intra-ocular, dust, spray, drinking-water, or intramuscular	6–12 days	4–5 months†	3 weeks in birds under 21 days old	2 weeks	2 weeks	Present	Lentogenic

Strain F	Intra-ocular, intranasal, or drinking-water	6–12 days	4–5 months†	3 weeks in birds under 21 days old	2 weeks	2–3 weeks	Present	Lentogenic
Roakin and MK 107 strains	Intramuscular or wingweb	2–3 days	3–5 months†	Not suitable for use	3 weeks	2 weeks	Marked stress plus mortality	Mesogenic
Muktes-war	Intramuscular	2–5 days	1 year	Not suitable for use	2–3 weeks	None	Marked stress plus mortality	Mesogenic
Komarov	Intramuscular	2–5 days	3–5 months†	Not suitable for use	Some days	1 weeks	Marked stress plus mortality	Mesogenic
Bankowski tissue culture	Intramuscular	5 days	1st injection: 2–3 months 2nd injection: 10 weeks	17 weeks in susceptible birds over 5 days old	None	No inform-ation	Very slight	Lentogenic

* This table is reproduced from 'Newcastle Disease and Fowl Plague' by W. A. Pool in *The Veterinary Annual* 1962, (1963).

† Used in a vaccination programme at prescribed intervals; immunity of longer duration achieved before birds come into lay.

Note: The periods shown here do not apply in every instance; they may vary considerably under certain conditions.

justified unless the disease is endemic or widespread. In Britain the use of killed vaccines only is permitted because the disease is sporadic. The slaughter policy was discontinued in that country in 1963 except in cases of acute infections and fowl plague. Live virus vaccines of chick embryo origin may transmit pullorum disease or leucosis complex or trigger off respiratory disease. However, this disadvantage may not arise if Bankowski's attenuated live virus vaccine grown in tissue culture is used.

FOWL PLAGUE

This is an acute highly infectious disease of fowls and sometimes of water birds caused by a virus which in some ways resembles Newcastle disease. However, it generally runs a more acute course.

Cause.—The virus is called *Myxovirus pestis-galli* (*Myxovirus influenzae* A, avian strain of Andrewes, 1964), and it resembles that of Newcastle disease in some ways, but in addition to causing agglutination of chicken erythrocytes; most strains also agglutinate the red blood-cells of the horse, guinea pig, pig, and rhesus monkey. It is closely related to influenza A virus of man. It appears to be more pathogenic than that of Newcastle disease. Pigeons also are susceptible. Unlike the virus of Newcastle disease it is killed easily, and it appears to be closely associated with the erythrocytes. There are many strains of the virus and the differences between the viruses of fowl plague and Newcastle disease have been discussed by Barry, Cruickshank, and Wells (1964).

Distribution.—It appears to be widely prevalent in the Mediterranean littoral, from where it has spread to certain parts of Asia, including India and Africa. Only sporadic and occasional outbreaks of the disease have been reported from Britain and the United States. The disease has not been reported from Ceylon so far, though it is likely to be present in the island.

Clinical Signs and Course of the Disease.—The incubation period is normally 3–5 days and death may occur in 24–48 hours. The symptoms resemble those of Newcastle disease and, in addition, there may be oedema of the head, consisting of exudation of serum into the subcutaneous tissues. Necrotic areas may be seen on the comb and wattles.

Post-mortem Lesions and Diagnosis.—Generally speaking the post-mortem lesions resemble those seen in Newcastle disease; in addition, the visceral surface of the sternum, the epicardium, and surrounding fat may show pin-point haemorrhages. The changes are those of a septicaemia, and the haemorrhages are widespread and are usually present in the proventriculus, abdominal fat, and muscles. Haemorrhages in the proventriculus resemble those seen in Newcastle disease.

Diagnosis is based partly on the symptoms and post-mortem changes, but mainly on virus isolation and identification and the demonstration of haemagglutinins in extracts of infected lungs after freezing and thawing.

Treatment, Prevention, and Control.—There is no known treatment. Vaccines are available but they are not widely used. Slaughter policy and general principles

of hygiene if adopted are probably the most useful methods to control this disease. Eradication of the disease should be the ultimate aim rather than control. The general principles outlined in Chapter I (Significance of Disease and Disease Prevention, p. 3) will be useful to control the infection. Mickail (1962) prepared a highly antigenic vaccine for a strain of virus isolated in the Sudan. More recently a live virus vaccine has been prepared using a strain of fowl plague virus isolated from the pea-fowl in Sudan by Daubney and Ishak.

INFECTIOUS BRONCHITIS

This is a highly infectious and contagious virus disease of the domestic fowl which can affect birds of all ages. Generally speaking, it runs a more acute course in the chicks, and a mild course in the adult. In the young it is characterized by respiratory distress and gasping symptoms, whereas in the adult a marked reduction of egg output is a prominent symptom.

Cause.—The disease is caused by a virus which has been given the name *Tarpeia pulli*. It causes stunting and curling of the chicken embryo while some strains cause its death. These characteristics become more marked usually after serial passage. The structure of the virus has been recently described by Barry and others (1964). There appear to be different strains of the virus which appear to be serologically different.

Clinical Signs and Course of Disease.—The disease has a short incubation period of 18–96 hours and is characterized by high infectiveness, respiratory symptoms, and low mortality in adults. These signs are more marked in chicks which exhibit gasping, coughing, râles, and moist eyes. In chicks, mortality in very severe outbreaks may reach 40 per cent. This is rare. In some outbreaks in chicks it is 10–20 per cent. In adults the symptoms are less severe and they may exhibit gasping movements, tracheal râles, and marked impairment of egg production. There is also reduced fertility and hatchability. The eggs laid by infected birds are misshapen, thin-shelled, and small. The eggs also show poor interior quality, with watery white and running yolk. The course of the disease lasts 10–14 days. Some strains of the virus are responsible for a nephritic–uraemic syndrome of fowls in Australia and the United States (Winterfield and Hitchner, 1962; Cumming, 1963) where there are striking kidney lesions and dehydration coupled with history of mild respiratory symptoms. Sometimes the disease is referred to as infectious avian nephrosis. Feeding birds on meat meal is said to predispose them to infection.

Post-mortem Lesions and Diagnosis.—The nasal passages, trachea, and bronchi contain mucus, and in well-established cases cheesy plugs can be seen in the main bronchi or below the bifurcation of the trachea. Air sacs may show cloudy swelling and accumulation of cheesy material. In the adult female, ovaries may show regressive changes and there are uratic deposits on the peritoneum. The characteristic lesions are seen in the kidneys which are swollen with marked uratic deposits in the tubules. The ureters are distended with urates. The kidney tubules also show degeneration.

A definite diagnosis can be made by virus isolation and serum neutralization tests. For virus isolation, lung tissue and mucous membrane scraped from the trachea and bronchioles are used. The presence of the virus is indicated by the dwarfing and curling effect it shows on 9–10-day chick embryos, followed by an irregular slow mortality in 5–7 days. For the serum neutralization test, birds that have recovered from the disease should be sent to a laboratory. This is done on the same principles as in the diagnosis of Newcastle disease. The gel diffusion precipitin test may also be used in the diagnosis of this disease, and characteristic changes can be detected in the tracheal mucosa on histological examination for 4 days after infection. A tentative diagnosis of infectious bronchitis may be made if the symptoms and lesions described previously are seen. The sudden onset of the disease, the high infectivity and low mortality associated with respiratory signs are fairly characteristic of the disease. However, it has to be differentiated from other respiratory diseases.

Treatment.—No specific treatment is available.

Epizootiology, Prevention, and Control.—Though the disease was first described in the United States, it now appears to have a world-wide distribution. The disease is spread by air-borne transmission and is perhaps the most infectious poultry disease. Chicks hatched from eggs laid by infected birds may show passive immunity up to 3 weeks. The epizootiology of the disease is not fully understood. Cook and Garside (1967) have shown that birds giving a positive reaction to serological tests but not showing positive clinical signs will not transmit the disease to the progeny. Bad management can predispose birds to infection.

Live virus vaccines of chick embryo origin are used to induce active immunity. There are basically two types of viruses used in bronchitis vaccines. They are the Connecticut (L-2) and the Massachusetts strains. The former is considered to be mild and the latter virulent. New developments in bronchitis vaccines have combined these two basic types in the belief that these combined vaccines will stimulate protection against a variety of field strains of the virus. They can be given in the form of aerosols, sprays, dusts, or in drinking-water. Highly susceptible chicks tolerate the vaccine better if given intranasally after 3 weeks. However, it is not advisable to use these vaccines in laying flocks and very young chicks below 2 weeks that do not possess antibodies. Sometimes these vaccines are combined with those of Newcastle disease. Vaccinated chickens should be protected for a few days after vaccination. Susceptible chickens should be raised in clean surroundings away from the adults.

The use of live autogenous vaccines is deprecated. Because this virus shows antigenic variation, polyvalent vaccines should be used in the field.

INFECTIOUS LARYNGOTRACHEITIS
(*I.L.T.*)

This is an acute or subacute highly infectious virus disease of the domestic fowl with localization in the respiratory tract, affecting mainly adults and young adults, characterized in acute cases by marked respiratory distress and haemorrhagic tracheitis. The literature on I.L.T. has been reviewed by Jordan (1966).

Cause.—The disease is caused by a virus termed *Tarpeia avium*. The virus is easily destroyed by the common disinfectants. It belongs to the group of Herpes viruses (Andrewes, 1964). There appear to be different strains of the virus with different degrees of virulence (Pulsford, 1963). The detailed structure of the virus has been described by Watrach, Hanson, and Watrach (1963). At least 4 serotypes have been identified by using fluorescene-tagged hyperimmune serum.

Clinical Signs and Course of the Disease.—The incubation period varies usually from 6 to 12 days, and the disease spreads rapidly through the flock. The older birds are more susceptible. The outstanding symptoms are gasping, râles, and coughing. The typically affected bird stretches its neck and inspires air with difficulty through a half or fully open beak and makes gasping movements. There may be expulsion of blood and mucus from the trachea. In addition there may be rattling and wheezy respirations. The disease lasts an average of 2 weeks and mortality may reach a peak of about 70 per cent, though usually it is lower. Occasionally baby chicks may be affected and in the chronic cases a yellow caseous exudate adheres to the rim of the larynx and may also obstruct the tracheal opening.

Post-mortem Lesions and Diagnosis.—The lesions are confined mainly to the trachea and the larynx. In acute cases there is haemorrhage in the tracheal mucosa, and in the chronic cases there is a yellowish caseous exudate with little or no blood. There may also be cheesy plugs or yellowish false membranes in the trachea.

Haemorrhagic tracheitis in acute cases is diagnostic, but the chronic form of the disease must be differentiated from other respiratory diseases, chiefly Newcastle disease, fowl plague, infectious bronchitis, coryzas, avitaminosis A, and the diphtheritic form of fowl pox. Laboratory diagnosis is made by isolating the virus in the chorio-allantoic membrane of 11–13-day embryonating eggs. The growth of the virus is indicated by the presence of pock-like lesions on the third day on the membrane. These are necrotic in nature. It is advisable to use pooled material from several cases to establish a diagnosis by this method. In this disease there are intranuclear inclusion bodies in the tracheal mucosa. Cross immunity tests may also be used for diagnostic purposes.

Treatment.—There is no specific treatment. However, in the chronic cases the removal of the caseated masses may give relief to the birds.

Epizootiology, Prevention, and Control.—The disease was first reported in the United States in 1925 and it now appears to have a fairly wide distribution. The epizootiology of this disease is discussed by Pulsford (1963). Infection occurs usually by the respiratory tract and recovered birds act as carriers for long periods. Indirect transmission may occur by means of rats, humans, dogs, crows, and by a variety of other animals and birds. Besides the domestic fowl, the pheasant is affected naturally. There appear to be different strains of the virus and some appear to be milder than the others. Parental immunity is passed on to the progeny.

Where the disease is endemic, live virus vaccines of chick embryo origin may be used. Vaccination is done into the cloaca of older birds and into the bursa of Fabricius in the case of younger ones at 10 weeks. If parental immunity is absent vaccination may be done as early as 7 days. A take is indicated by the presence of

an oedematous swelling in the cloaca on the fourth day. Immunity lasts about 1 year. As the vaccinated birds are potential carriers, vaccination should never be undertaken in areas where the disease is not endemic. The dangers of vaccination are discussed by Pulsford (1963).

Vaccination may also be done by the feather follicle method by using a brush. In some instances this vaccine is mixed with fowl pox vaccine. Work by Raggi and Lee (1965) indicates that the duration of immunity induced by most vaccines wanes after 15 weeks and that a vaccine can be given in drinking-water with satisfactory results. A live vaccine for intra-ocular administration has been developed recently. It does not impart a carrier state to vaccinated birds but confers marked immunity (Churchill, 1965). A modified tissue culture vaccine has also been used for conjunctival instillation for immunizing birds over 2 weeks. Recently Sinkovic and Hunt (1968) have shown that strain SA-2 vaccine can be used successfully for the immunization of day-old birds.

FOWL POX

This is usually a chronic and mild disease which has a very widespread distribution caused by an epitheliotropic virus and which may assume one of the two forms:—

1. The cutaneous form, which is characterized by the production of wart-like nodules on the comb, wattles, and eyelids, and less often on other parts of the body.

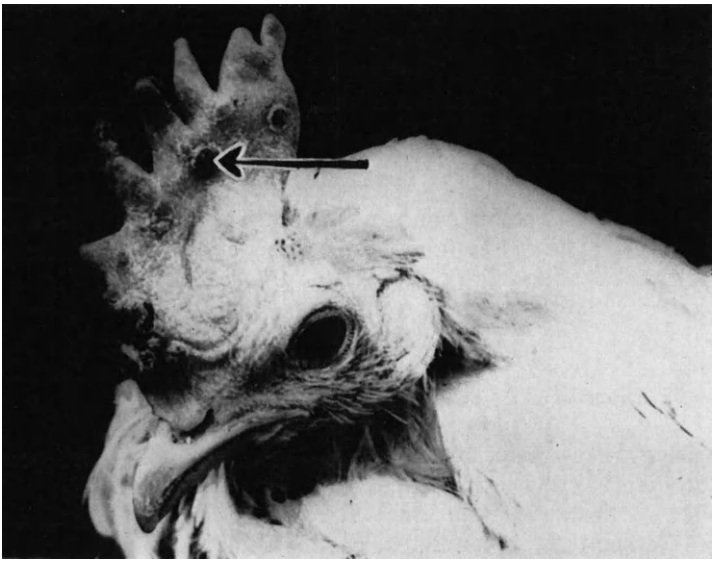

Fig. 2.—The head of a fowl showing typical lesions of fowl pox on the comb.

2. The mucomembranous form, which affects the mucosae of the mouth, nostrils, eyes, and larynx with the formation of a diphtheritic membrane, oculonasal catarrh, and cheesy deposits in sinuses. This form is more serious than the first.

In a proportion of cases the two forms coexist. The virus affects fowls, turkeys, and certain other species of birds (*Figs.* 2, 3).

Cause.—The virus causing fowl pox is called *Borreliota avium*, while the typical turkey pox virus is called *Borreliota meleagridis*, the canary pox virus *Borreliota fringillae*, and the pigeon pox virus *Borreliota columbae*. All these viruses belong to group IV of the pox viruses and are characterized by large virus particles contained in a matrix probably of lipoprotein, are ether-resistant, and are often transmitted by insects (Andrewes, 1964). Each virus is infective to the type host, and sometimes to the heterologous host as well. Pigeons are generally resistant to infection with fowl pox virus, but the pigeon pox virus causes a very

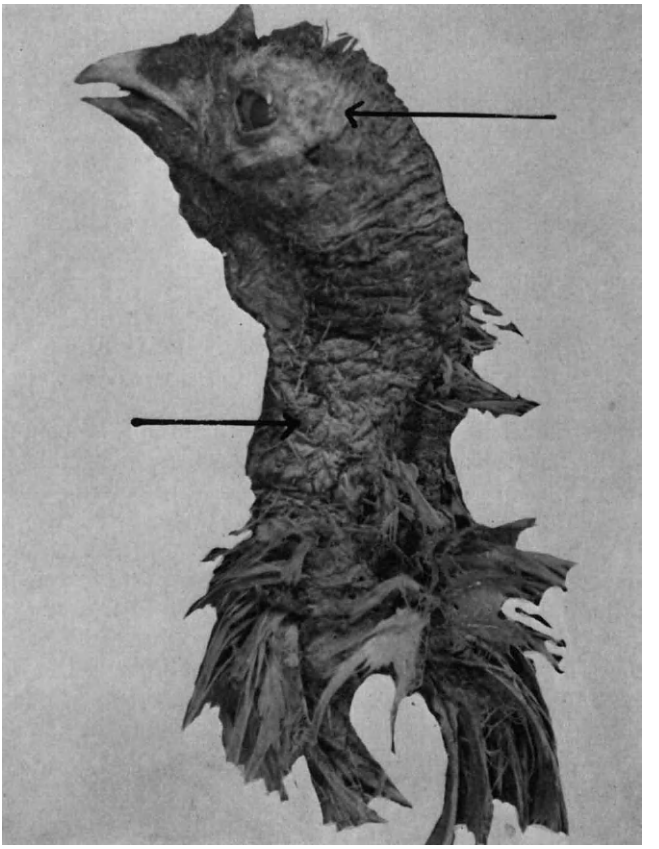

Fig. 3.—The head of a turkey hen, showing acute and extensive turkey pox.

mild reaction in fowls and renders them mildly immune to fowl pox virus for a short period. The four viruses are interrelated. Vaccination with either turkey or fowl pox virus gives almost complete protection against all the four viruses.

The virus of fowl pox is one of the largest, being about 0·25 μ in size. The virus is extremely resistant to dessication and when properly dried it may live up to 10 years. However, it is inactivated by a 1 per cent solution of potassium hydroxide in 5 minutes, or by a 1:1000 solution of mercuric chloride or crystal violet.

Clinical Signs and Course of the Disease.—The incubation period varies from 6 to 14 days and in the uncomplicated cases the disease lasts 3–4 weeks. The cutaneous form is characterized by the appearance of warty growths on the comb, wattles, face, and eyelids. This is the milder form. In the mucomembranous form, the mouth, conjunctivae, and nasal sinuses are affected. There is usually formation of diphtheritic membranes or cheesy deposits. The bird may go blind and the sinuses may be distended. Sometimes coryza-like symptoms are seen. Mortality in this form may at times reach 10 per cent or higher, depending on the complications, whereas in the cutaneous form it is negligible. In a fair proportion of cases the two forms coexist. In turkeys the lesions may be extensive (*Fig.* 3).

Prasad, Verma, and Srivastava (1967) have reported a very severe form of fowl pox in India causing 100 per cent mortality and characterized by warty growths on feet, legs, claws, and beak.

After 2–3 weeks the scabs fall off leaving white scars which also disappear in 1–2 weeks.

Post-mortem Lesions and Diagnosis.—Apart from the lesions which are described under 'Clinical Signs', many of the cells at the base of the warty growths or the diphtheritic deposits show large cytoplasmic inclusion bodies about 2–3 μ in size called Bollinger bodies. Within these large inclusion bodies are smaller Borrel bodies which are about 0·25 μ in size and which probably represent the virus particles.

Diagnosis is made from the presence of the typical warty growths, by demonstrating Bollinger bodies histologically, by cross immunity tests, and by isolating the virus on the chorio-allantoic membrane of the chick embryo. The virus produces pock-like lesions on this membrane, and can be differentiated from the I.L.T. virus by the presence of inclusion bodies (Bollinger bodies) in the infected membrane. Further, the lesions are hyperplastic and oedematous. Eleven to thirteen-day-old embryonating eggs are used for the purpose and the lesions are usually seen after 3 days.

Treatment.—There is no specific treatment. Painting the warts with tincture of iodine is advocated by some, but its efficacy is doubtful. Others use 1:1000 mercuric chloride to paint the lesions. When secondary bacterial invasion has taken place, or if there are coryza-like symptoms, antibiotics are useful. Generally speaking, treatment is not necessary and if there are complications it is often not worth while.

Epizootiology, Prevention, and Control.—The disease is prevalent in most countries. All breeds and all ages of fowls are susceptible. In temperate countries it is most often seen in autumn and winter, whereas in the tropics it is prevalent during the insect vector season. The virus cannot pass through intact skin, and can enter the body only through damaged skin. Certain mosquitoes of the genera *Aedes* (*A. aegypti*), *Culex* (*C. pipiens*, *C. tarsalis*), *Anopheles*, *Theobaldia*, *and Stegomyia fasciata* act as mechanical transmitters. In these mosquitoes the virus may live up to about a month.

Fowl pox can cause septicaemic disease and high mortality in carrion and hooded crows (*Corvus coronae* and *C. cornix*).

Control of the disease is effected chiefly by vaccination. Two types of vaccines are available: fowl pox vaccine and pigeon pox vaccine.

Modern fowl pox vaccines are egg-propagated vaccines made from mild strains of fowl pox virus and in some cases may also be mixed with Newcastle disease vaccine. Fowl pox vaccine induces durable immunity over 1 year and perhaps even for the productive life of the bird. Most manufacturers now produce two types of fowl pox vaccine, a mild reacting one and one that causes a relatively stronger reaction during the immunizing process. Layers should not be vaccinated with the stronger vaccine. Vaccination may be done by the 'stick' method or the 'feather follicle' method. In the 'stick' method a sharp pointed instrument is used and vaccination is done on the inside of the wing in the web. In the 'feather follicle' method a few feathers are plucked, usually from the outer side of the thigh, and the vaccine is rubbed on with a brush.

The flock should be examined for 'takes' after a week. If the birds have reacted, there is usually a distinct scab at the site of vaccination by the stick method, and a nodular swelling on the feather follicles and scab formation, if vaccination was by the feather follicle method. All birds that do not show a positive reaction should be revaccinated.

When using fowl pox vaccine, the birds should be at least one month old and they should be at least one month from point of lay. The ideal age for vaccination is 6–12 weeks. Maximum immunity is usually attained at the end of the fourth week and birds are still susceptible in the first 2 weeks of vaccination.

If pigeon pox vaccine is used, younger birds or even layers may be vaccinated during an outbreak of disease. However, the immunity established is mild and lasts only a short period. The 'takes' are indicated by the presence of swellings at the sites of vaccination, from the fifth day onwards. Scabs are not formed.

As the flock has to be examined for 'takes' about the seventh day irrespective of the vaccine used, it is advisable to vaccinate one particular spot on all the birds. Sometimes there may be a severe reaction to vaccinating the birds with fowl pox vaccine.

Usually full directions are enclosed with the vaccine by the manufacturer. A lay person can undertake vaccination against fowl pox without risk. Birds may be vaccinated even after the disease has occurred provided this is done fairly early in the outbreak; vaccination at this stage helps to control the outbreak.

AVIAN ENCEPHALOMYELITIS
(Epidemic Tremor)

This is a viral infection which affects clinically, chiefly the young chicken, and is characterized by low morbidity, ataxia, and tremors especially of the head and neck muscles.

Cause.—This is caused by a virus which is present in the brain of the infected chickens and appears to be excreted in the faeces of these birds. It belongs to the group of picornaviruses containing ribosenucleic acid.

Clinical Signs and Course of the Disease.—The incubation period varies from 9 to 21 days and the disease may last several weeks. The morbidity varies from 10

to 20 per cent and all the severely affected chickens die. The characteristic symptoms are ataxia due to incoordination of the leg muscles and tremors of the head and neck. The disease may first appear when chicks are 1 day old. When the eggs are infected there is a high embryo mortality especially in the last 3 days of the incubation period. Chicks hatched from infected eggs show ataxia during the first 6 days after hatching. Death occurring in chicks over 7 days old is the result of contact infection. If the infection occurs in the incubator, those birds that survive the clinical attack may show an opacity or bluish colouration of the lens, causing blindness.

The development of encephalomyelitis is not enhanced by a deficiency of vitamin E. Laying hens if infected will not usually show any clinical signs. However, there will be a drop in egg production by 5–20 per cent which may last for a period of 10 days. During this period they are usually viraemic and the virus is shed in the eggs. Infected eggs will show a high mortality especially during the latter stages of incubation. Some hatched chicks will show signs of tremor during the first few weeks of life. As a rule the disease in chicks 1–14 days old can be attributed to egg transmission.

Post-mortem Lesions and Diagnosis.—The lesions are usually microscopic in nature and are caused by focal or diffuse gliosis, perivascular infiltration, neuronal degeneration, and hyperplasia of the lymphoid follicles, primarily of the myocardium, the proventriculus, and the pancreas.

Diagnosis can be based on the flock history and the typical symptoms. However it has to be differentiated from Newcastle disease, encephalomalacia, and riboflavin deficiency. A histological examination should be made to establish a diagnosis. A specific diagnosis may be made by the serum virus neutralization test or the embryo susceptibility test.

The presence of lymphocytic infiltration, particularly the follicular type of ymphoid reaction in the atrial myocardium and the tunica muscularis of the gizzard are of diagnostic value (Springer and Schmittle, 1968).

Treatment.—No specific treatment is available and it is not advisable to treat even symptomatically. All the badly affected birds should be destroyed.

Epizootiology, Prevention, and Control.—This disease was first reported from the United States and is now being recognized in other countries such as Canada, the Union of South Africa, and Europe. Its incidence is decreasing in the United Kingdom as the result of vaccination. It is present in many other parts of the world. The disease can be reproduced in a proportion of experimental chicks by intracranial inoculation with suspensions of the brain and spinal cord of infected chickens. Chick embryos may be similarly infected by the intra-ocular route.

The infection is primarily egg-borne and usually only 10–20 per cent of the flock of chickens are affected. Transmission may also occur by direct contact. The infection apparently establishes itself in the intestinal tract of the infected bird and the virus is shed in the faeces from the fifth to twelfth day after infection. Control is effected on general principles of hygiene. It is possible to use vaccines to control the disease. Live virus vaccines and betapropriolactone inactivated vaccines have been used on a fairly extensive scale. Immunity will last for a period of 4 months or

more with the latter vaccine. Live vaccines should not be used on birds under 10 weeks old or when in lay. If used they may either be ineffective due to parental immunity or they may induce signs of disease. The live virus vaccines are usually given in drinking-water when the birds are between 6 and 16 weeks old.

Breeder hens that have been vaccinated before production will transmit immune bodies to the chicks to protect them up to 6 weeks, well past the period when they are most susceptible.

AVIAN LEUCOSIS COMPLEX

This group of diseases is characterized by autonomous proliferation of the precursors of the blood-cells, the type of reaction depending on the cell affected, the agent inducing the reaction, and a variety of other factors.

It is one of the most widespread poultry diseases in the world and causes an annual loss of $65,000,000 to the poultry industry in the U.S.A. (Kesteven,1965).

The classification of this disease complex has undergone considerable modification due to increased knowledge of the aetiological agents causing the disease complex. It is now known that several different viruses can elicit a similar response in various tissues and that one virus can elicit different responses in different tissues or in the same tissue, depending on a variety of factors (Rubin, 1962; Vogt, 1965). This has caused some confusion in the classification of this disease complex. In the past the classification was based largely on the nature of the pathology and the type of cell affected, but such a classification is not satisfactory because it does not consider the aetiology.

However, recently there has been considerable agreement on the fundamental issues, due to the work of Biggs and his co-workers in U.K. and Sevoian and others in the U.S.A.

According to Biggs and Payne (1964) and Biggs (1966) the two main entities of the disease complex are:—

1. Leucosis sarcoma group caused by a virus (or viruses) which gives a positive reaction to the complement fixation test with avian leucosis viruses (COFAL test) and induces resistance to infection with Rous sarcoma virus (R.I.F.+: resistance inducing factor positive).

2. Marek's disease caused by a virus which gives a negative reaction to the COFAL test and is R.I.F. negative.

Sevoian (1967) classified the avian leucosis complex taking into consideration the type of cell affected, the nature of the cellular response, and the aetiological factors. He considered that histogenesis was a basic criterion for tumour classification. Thus his classification was 'pathologically oriented then aetiologically tempered'.

Sevoian's classification was as follows:—

A. i. Lymphoid leucosis type I caused by R.I.F. positive and COFAL test positive virus (type I virus) and its sub-types.

ii. Lymphoid leucosis type II caused by JM type virus which is R.I.F. and COFAL test negative (type II virus) (Marek's disease).

iii. Lymphoid leucosis type III caused by T-type virus (type III virus) which is R.I.F. negative but antigenically different from type II virus. This causes marked proliferation of the cells of the reticulo-endothelial system (Olson and Kerr, 1967).

B. Myeloid leucosis type I caused by type I virus and its sub-types, as in lymphoid leucosis type I.

C. Erythroid leucosis type I caused by type I virus and its sub-types as in lymphoid leucosis type I.

D. Non-leucotic tumours type I caused by type I virus.

In this classification, types and sub-types are added as the different aetiological agents are identified and classified.

It will be noticed from this classification that the type I virus can induce different types of reaction while type II and type III viruses can induce the same type of reaction (lymphoid leucosis) even though they are different antigenically.

Since under different conditions the same virus can induce different reactions it is more appropriate, at least until further information is available, to follow the classification suggested by Biggs and his colleagues who consider the aetiology more important than the pathology. A comparison of the different systems of classifying avian leucosis complex is given in *Table* 5.

Leucosis Sarcoma Group

This is a group of neoplastic conditions of the domestic chicken caused by a virus or related viruses which have been reviewed by Vogt (1965). Some degree of confusion exists as some of these viruses can undergo modification after serial passage to produce different responses. Thus the virus causing lymphoid leucosis in the field, after serial passage can cause erythroid leucosis, sarcomas, and osteopetrosis.

This disease complex may appear in different forms described below depending upon the type of cell affected, and the cellular response to the aetiological agent. These are:—

A. Lymphoid leucosis (visceral lymphomatosis).

B. Erythroid leucosis (erythroblastosis).

C. Myeloid leucosis (myeloblastosis, myelocytomatosis).

D. Osteopetrosis (thick leg disease, osteodystrophia fibrosa).

E. Rous sarcoma and other non-leucotic tumours.

A. Lymphoid Leucosis (Visceral Lymphomatosis).—This disease is seen in chickens 4 months of age or older and there are no characteristic clinical signs. In a few birds there may be ascites and evidence of an enlarged liver (*Fig.* 4), a distended abdomen, or birds may adopt the penguin sitting posture.

Aetiology.—This disease and its various forms are caused by a virus (or viruses) of which two sub-groups, A and B, exist. Churchill (1968) demonstrated the existence of distinct serotypes among the viruses of the sub-group A. The virus induces resistance to Rous sarcoma virus (R.I.F. positive) and gives a positive reaction to the complement fixation test for avian leucosis (COFAL test). Unlike the sarcoma viruses, the virus (or viruses) of avian leucosis will produce neoplastic changes predominantly in the haemopoietic system.

Post-mortem Lesions.—Typical cases are characterized by great enlargement of the liver and spleen due to infiltration by lymphoid cells. Occasionally the kidneys, mesentery, lungs, and other organs may be similarly affected. The lesions may be diffuse (diffuse form) or there may be discrete nodules varying in size from

Table 5.—THE DIFFERENT SYSTEMS OF CLASSIFICATION OF AVIAN LEUCOSIS COMPLEX

After Cottral. Based on pathology alone	After Biggs and Payne (1964) and Biggs (1966). Based on aetiology	After Sevoian (1967). Based on pathology and aetiology
Neural lymphomatosis	Caused by an R.I.F. negative and COFAL test negative herpes B group virus and characterized by lymphoid lesions in peripheral nerves and other tissues. Marek's disease (acute and chronic forms; the chronic form is considered the classical form of the disease)	Lymphoid leucosis, type II*. All forms of lymphoid leucosis type II are caused by JM type virus which is R.I.F. and COFAL test negative
Ocular lymphomatosis	Marek's disease (uncommon atypical form of manifestation)	Lymphoid leucosis, type II*
Visceral lymphomatosis	Leucosis sarcoma group. Caused by a virus (or viruses) of the Myxo virus group which are R.I.F. and COFAL test positive of which there are subgroups A and B. Lymphoid leucosis	Lymphoid leucosis, type I*. All forms type I are caused by R.I.F. and COFAL test positive virus
Erythroblastosis	Erythroid leucosis	Erythroid leucosis, type I and sub-types*
Myeloblastosis Myelocytomatosis	Myeloid leucosis (diffuse and discrete forms)	Myeloid leucosis, type I and sub-types
Osteopetrosis	Osteopetrosis	Lymphoid leucosis, type I
Nephroblastoma and other tumours	Sarcoma, endothelioma, and kidney tumours	Non-leucotic tumours, type I*
		Lymphoid leucosis, type III caused by R.I.F. negative virus differing from JM and Rous sarcoma viruses

* Types and sub-types added as more causal agents are determined.

a pea to a golf ball (discrete form). It is now believed that initially, the malignant change occurs in the bursa of Fabricius from where malignant cells metastasize to other organs. Histologically the disease is characterized by massive accumulations of lymphoid cells in the various organs especially in the liver and spleen. There is lymphoid hyperplasia in the spleen. Horiuchi (1961) recognized three types of histological reactions in the liver: the nodular type characterized by fibroid cells which are continuous with the sinusoidal epithelium; the diffuse type in which cellular proliferation occurs along the spaces of Disse; and the infiltrative type

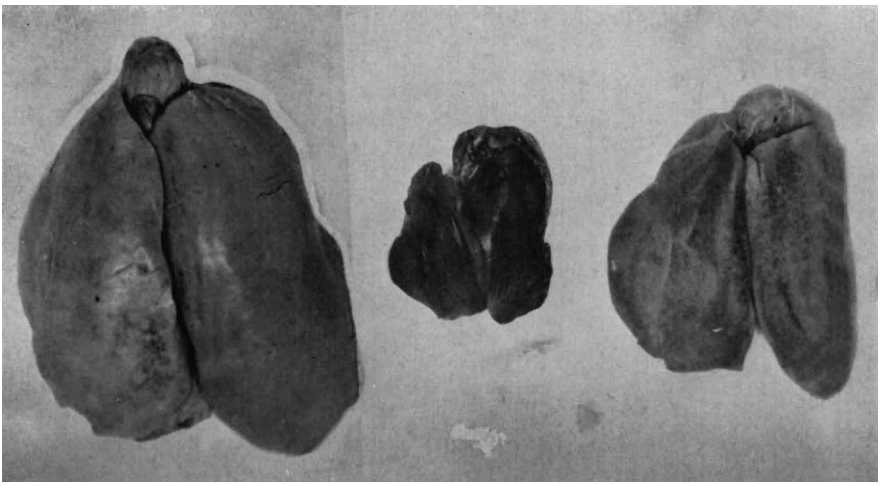

Fig. 4.—Livers of fowls with visceral lymphomatosis. The two livers on either side are those of adult hens with visceral lymphomatosis. The liver in the centre is from a normal hen for comparison.

which consists primarily of a network of sinusoidal endothelium. There is an increase in the number of immature lymphocytes in the blood.

B. Erythroid Leucosis (Erythroblastosis).—This is a transmissible condition caused by type I virus and is at times characterized by severe anaemia and later by petechial haemorrhages. This condition is not uncommon and is usually seen after a month of illness. Post-mortem examination reveals gross enlargement of the liver and spleen which are cherry-red in colour. The long bones contain a currant-jelly–like substance. The blood sinusoids are dilated and the capillaries are filled to capacity with primitive blood-cells of the erythroblastic series and the bone-marrow quite regularly shows microscopic accumulations of erythrogonia which however, fail to invade the blood-stream. The plasma leucocytic erythrocytic ratio is 88:1:11 as against a normal ratio of 55:1:44. There are large numbers of basophilic erythrocytes and erythrogonia characterized by a nucleus which stains violet-red with Romanowski stains. Mitotic erythrocytes or precursors of erythrocytes are not uncommon.

C. Myeloid Leucosis (Myeloblastosis; Myelocytomatosis).—The clinical signs of this condition resemble those seen in erythroid leucosis, but younger birds are affected and the type of cellular response is also different. There are usually two

forms of the disease, the diffuse form and the discrete form (myelocytoma). In the diffuse form the liver and spleen are enlarged and the former has a greyish mottling giving it an appearance similar to morocco leather. There are massive accumulations of myeloblastic and promyelocytic elements in the parenchymatous tissues which are located extravascularly. Intense myeloblastic activity is also observed in the extrasinusoidal areas of the bone-marrow. The blood-picture is

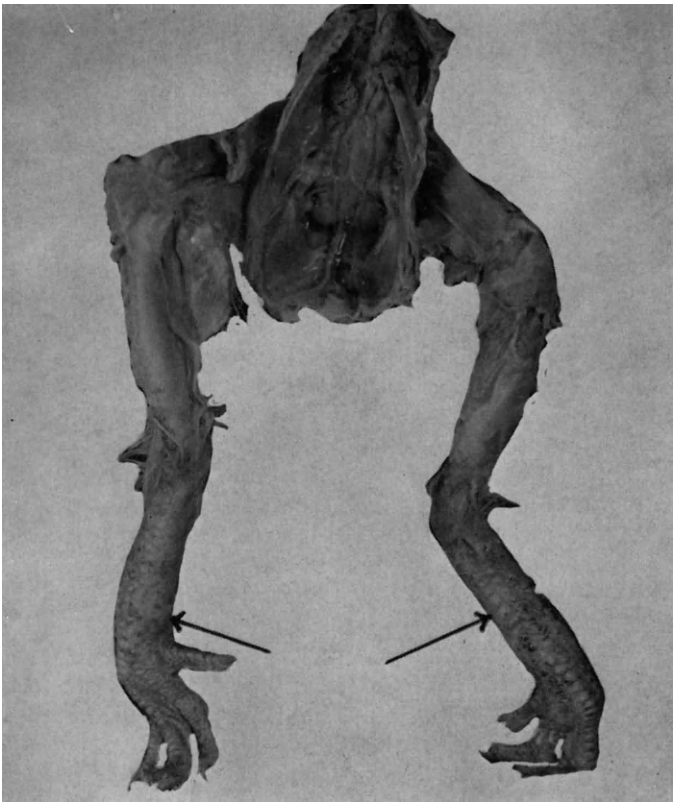

Fig. 5.—Osteopetrosis in a 3-month-old white leghorn. The arrows indicate thickened metatarsi.

characterized by primitive cells of the myeloblastic series, especially myeloblasts. In the discrete form there are chalky or cheesy white friable tumours in the liver and spleen and cauliflower-like growths in the periosteum of the keel, ribs, and pelvis.

D. Osteopetrosis (Thick Leg Disease; Osteodystrophia Fibrosa).—The virus causing this condition appears to be related to that causing lymphoid leucosis. It is characterized by increased osteoblastic activity in birds about 2–3 months old and is more common in the male than in the female. The long bones of the extremities, especially the metatarsi, are thickened, deformed, and boot-like which is characteristic of the disease (*Fig.* 5). The affected portions are hot and insensitive.

The disease is slowly progressive and ends fatally in about two months. It has been transmitted from chicken to chicken and from the turkey to the chicken. In more advanced cases, lesions similar to those observed in visceral lymphomatosis may also be seen. Affected birds usually show secondary anaemia.

The lesions can be distinguished from those of rickets where the bone is deformed without thickening, (in rickets the keel bone is often bent) and from those of perosis where there is twisting and flattening of the hock joint.

DIAGNOSIS OF AVIAN LEUCOSIS.—This is based on the macroscopic lesions and the histological appearance of these lesions. The COFAL (complement fixation for avian leucosis virus) test and the R.I.F. (resistance inducing factor) test using the Rous sarcoma virus give positive reactions. It can also be diagnosed by the fluorescent antibody test and the application of the knowledge that non-producer (NP) cells infected with Rous sarcoma virus (R.S.V.) can be activated by avian leucosis viruses to produce infectious R.S.V. This test appears to be more sensitive than COFAL and R.I.F. tests.

E. Rous Sarcoma and Other Non-leucotic Tumours.—Avian sarcomas are probably uncommon manifestations of the activity of an ubiquitous virus. They appear sporadically in fowls, more in some flocks than in others. A variety of birds are susceptible to the virus. These include turkeys, pigeons, and ducks.

AETIOLOGY.—Several closely related viruses are responsible for sarcomas. The virus causing chicken tumour No. 1 of Rous (1911) will produce adenocarcinomas when inoculated into the kidney of young chicks. It can also be spread by direct contact (Burmester and Fredrickson, 1966). Amongst other viruses in this group those causing the Murray-Begg endothelioma and the Fuginami myosarcoma are closely related to Rous sarcoma virus (Rubin, 1962). The sarcoma viruses primarily cause tumours of the connective tissue whereas the virus (or viruses) of lymphoid leucosis produce neoplastic changes in the haemopoietic system.

PATHOLOGICAL LESIONS.—The growths are soft and contain much mucinous material and blood pigment, but the slow growing ones are firm and white. These growths are circumscribed, pinkish, and fleshy, are seen in the lungs, liver, and heart, and are usually progressive and fatal. However, they may occasionally regress. In very young chicks the virus may cause multiple haemorrhages and rapid death without evidence of neoplasia.

The other non-leucotic tumours include fibrosarcoma, osteogenic sarcoma, renal sarcoma, endothelioma, and haemangioma. These have been classified mainly on the histopathological appearance. Therefore, their exact relationship to the sarcomas caused by specific viruses is not known.

DIAGNOSIS.—As sarcomas can be produced by an ubiquitous virus (or viruses) with a potential for eliciting different responses in different tissues, or in the same tissue under different conditions, identification or characterization of this group of viruses has been difficult. The exact relationship of these viruses to those causing avian leucoses has also not been established, particularly because viruses of this group may alter their onchogenicity or even their antigenic structure after serial passage.

Epizootiology.—This is a widespread disease complex. The disease is spread via the eggs or by contact. The virus does not appear to survive for long outside

the host. Infected birds develop antibody to the virus and this neutralizes Rous sarcoma virus of the same sub-group. Though chickens are susceptible to infection during hatching and the first few weeks of life, chicks hatched from eggs laid by immune hens have maternal antibody and are protected. Some immune birds carry the virus and pass it to a proportion of their offspring through the egg, and these infected embryos become immunologically tolerant. When these embryos hatch they are viraemic and are unable to develop an active immunity, but show no signs of infection. The T-type virus producing avian reticulo-endotheliosis can be transmitted by the reduvid bug *Triatoma infestans* (Klug) which can harbour the virus for at least 72 hours (Thompson, Fisher, and Luecke, 1968).

Egg transmission of lymphoid leucosis virus from carrier adult hens has been demonstrated and this appears to be the common mode of transmission although horizontal spread may occur. Studies utilizing the R.I.F. have shown the existence of three main categories of adult birds.

a. Viraemic hens which shed the virus in their eggs. Chicks congenitally infected are immunologically tolerant and do not produce antibody.

b. Birds with circulating antibody. These may occasionally shed virus in their embryos.

c. Non-infected birds. These have no antibody or viraemia.

Control.—This is largely based on selection of stock which is genetically resistant to infection. Susceptibility or resistance is controlled in each case at a single genetic locus, with the gene for resistance dominant over the gene for susceptibility. The genes for resistance to sub-group A viruses in heavy breeds and for sub-group B viruses in all breeds are more common. There is some doubt as to whether a trait based on a single gene is suitable for the development of genetically resistant stock, since mutation at this locus or mutation of the virus may result in complete loss of resistance. However, it is most likely that resistance to tumour formation is based on multiple gene interaction. Therefore, until more is known of the genes controlling resistance to tumour formation, eradication of the causal agents by testing and eliminating the carriers is probably the method of choice for control of this disease, though it is rather expensive.

MAREK'S DISEASE
(*Neurolymphomatosis; Range Paralysis*)

This is a lymphoproliferative, highly contagious disease of the domestic chicken affecting certain peripheral nerves and sometimes other tissues like the gonads, liver, heart, and iris. Chickens having this disease do not show resistance to infection with Rous sarcoma virus (Witter, Calneck, and Levine, 1966). The disease may be either acute or chronic, and shows both inflammatory and neoplastic changes. This disease alone is responsible for a loss of at least £7150,000 per annum in the U.K. (Biggs, 1967). The primary cell affected with the neoplastic change is the lymphocyte or its precursors. Infection with Marek's disease may increase the susceptibility of certain strains of chickens to coccidiosis.

Cause.—The disease is caused by a virus which belongs to the Herpes B group of viruses (Churchill and Biggs, 1967) and is strictly associated with the cells of the affected nerves, tumours, and blood. The virus does not give a positive

reaction to the complement fixation test for avian leucosis viruses and does not induce resistance to Rous sarcoma virus.

The following isolates of Marek's disease have been studied: JM, HPRS-B14, HPRS-16, GA, Conn A, and C-1. Different isolates produce different responses in the same host. Likewise, the same isolate may produce different responses in different hosts.

The infectivity of the virus is destroyed at a temperature of 56° C. for 30 minutes outside a *p*H range of 5·5–8·4. The infectivity of blood and tumours is closely cell-associated.

Clinical Signs.—The disease may be acute or chronic (this form was earlier termed neurolymphomatosis or range paralysis). Less frequently it may affect the iris where large numbers of lymphocytes may be deposited to produce the condition often called fish-eye, grey-eye, or wall-eye (ocular lymphomatosis).

The acute form of the disease is seen in birds between 1 and 5 months of age and has an incubation period of 7–28 days. There are no characteristic signs other than increased mortality which may reach 50 per cent. Nervous signs are not usually encountered in this form of the disease.

Post-mortem examination of acute cases will reveal tumours of proliferating lymphoid cells in the gonads, liver, spleen, lung, heart, kidney, muscles, and other visceral organs (Purchase and Biggs, 1967; Biggs, 1967). Marek's disease is characterized by abnormal multicentric aggregation of lymphoid cells observed chiefly in growing birds. There are nonpurulent encephalitis and perivascular lymphocytic infiltrations in the brain and meningoradiculomyelitis in the spinal cord.

The Marek's disease agent has been propagated in chicken and duck embryo fibroblasts and in chicken kidney cells.

In the chronic form (neural lymphomatosis) the clinical signs appear in more mature birds. Usually those over 3 months are affected and the incubation period is longer. The typical clinical signs are progressive asymmetric paresis or paralysis of wings, legs, or neck which may be either spastic or flaccid. One leg may be stretched forward while the other is bent. An affected wing usually droops. There may be incipient torticollis. If the vagus is affected there may be respiratory distress or distension of the crop. The disease is gradually progressive and ultimately fatal.

Post-mortem examination of the chronic cases reveals slight to moderate or marked swellings, usually either on the sciatic nerves or the brachial plexus. Usually the swellings on one nerve are more marked than the other. Sometimes no macroscopic changes are seen. The affected nerves show lack of cross-striation (*Fig.* 6) and are smooth and yellowish. Microscopically they show infiltration with mononuclear cells. Occasionally tumours may be seen in the feather follicles (skin leucosis).

The pathological changes occurring in the nerves can be classified into three types. The A-type lesions have an unusual type of cell termed Marek's disease cell (Biggs and Payne, 1967) which has a basophilic, pyroninophilic, and frequently vacuolated cytoplasm which tends to obliterate nuclear detail. The B-type lesion is characterized by interneuritic oedema and light infiltration, principally by small lymphocytes and plasma cells. The C-type lesion shows a light scattering of lymphocytes and plasma cells.

The ocular form (grey-eye, fish-eye, pearl-eye, etc.) is seen in older birds, usually those over 6 months and only 1 or 2 birds in a flock are affected. This may be regarded as an uncommon manifestation of Marek's disease. It is characterized by diffuse fading of the iris of one or both eyes, or by spotty depigmentation of the iris. The pupil is reduced considerably, loses its ability to respond to light, and the bird may not be able to see with the affected eye. The iris usually becomes greyish and hence the names fish-eye, grey-eye, or pearl-eye. The

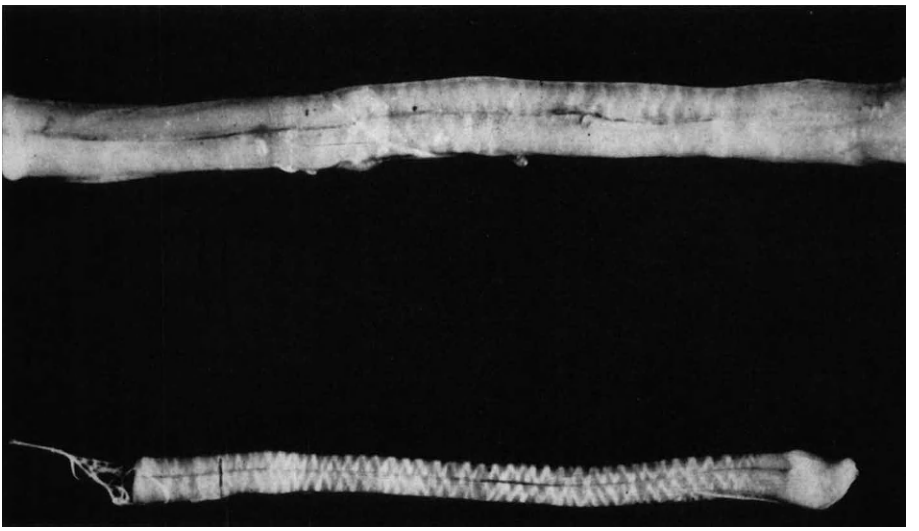

Fig. 6.—The thickened sciatic nerve of a fowl with Marek's disease (above). Note the lack of striation. Normal sciatic nerve (below).

changes in the iris are due to infiltration of the iris with lymphocytes and lymphoblasts.

Diagnosis.—This is made from the nature of the lesions. The changes in the nerves and the gonads are of diagnostic value. Marek's disease gives negative results with R.I.F. and COFAL tests.

Epizootiology.—Marek's disease is highly contagious and is spread easily by direct and as well as indirect contact and can be disseminated by the air-borne route. Litter may be infective after 6 weeks storage and the virus may survive for a number of weeks outside the body and in the darkling beetle *Alphitobius diaperinus* (Edson, Schmittle, Lal, and Goode, 1965). The disease is also said to be disseminated by the fowl tick *Argas persicus*. Young birds are very susceptible to infection and the female is more susceptible than the male. Genetic and breed susceptibility, coccidiosis, parasitism, nutrition, and other environmental factors may play an important part in determining resistance or susceptibility.

It may occur as latent infection in some flocks. It appears to have a seasonal incidence, reaching a peak in spring. Occasionally there may be unexpected high losses in the first broods in a new poultry house—'new-house syndrome'.

Prevention and Control.—Control can be affected largely by selecting stock which are genetically resistant to the disease. Biggs, Thorpe, and Payne (1968) found that both sires and dams had a significant effect on the susceptibility of the progeny to Marek's disease and that females were significantly more susceptible than males.

As the disease is highly contagious in the field, contact with infected birds should be avoided, and it is of particular importance to keep chicks isolated from adult stock.

The following principles of hygiene are recommended for the control of this disease:—

1. Hatch, hold, transport, and brood chickens in facilities which have been completely disinfected.
2. Hatch and maintain chickens of different ages and of different origins in separate, isolated areas.
3. Avoid rearing chickens in areas where the disease is enzootic and where there is a high chicken population.

ORNITHOSIS AND PSITTACOSIS

Ornithosis is a disease primarily affecting the respiratory system of a large variety of birds, caused by a virus of the psittacosis-lymphogranuloma venereum (PLV) group of organisms. The disease in man and psittacine birds (parrots, parakeets, the Psittacidae, and the Fringillidae) is called psittacosis, whereas the disease in other species is referred to as ornithosis. These diseases are important zoonoses. In man, psittacosis produces atypical pneumonia. Infection may occur after brief contact and many infections may be sub-clinical.

Species Affected.—Pigeons, ducks, and turkeys may be severely affected though mortality is usually low. The domestic fowl can become infected though usually no symptoms are seen. Infections have been reported in pheasants, geese, a large number of psittacidae, and wild or captive birds.

Aetiology.—Ornithosis and meningopneumonitis in birds is caused by *Miyagawanella ornithosis* whereas psittacosis in psittacine birds and atypical pneumonia in man is caused by *M. psittaci* which is closely related to *M. ornithosis*. Both these organisms belong to the PLV group of organisms which show small elementary bodies, and larger initial bodies about 0·8 μ in diameter in plaque, cluster, or vesicle formation. The agents are primarily spread by air-borne infection on dust or as virus particles.

Epizootiology.—Pigeons are more commonly infected than other species of birds and the disease is severe in pigeons below 16 weeks of age. Ducks and turkeys can be affected naturally and mortality could be high in young birds. In turkeys it appears to be widely distributed, the infection being often latent as the virus in this species has a low virulence. Mortality if present is usually caused by virulent mutants of the virus. Most infected turkeys appear to recover.

Infection from bird to bird and from bird to man usually occurs during raising and dressing of domestic poultry or from household Psittacidae such as parrots and parakeets.

In the case of psittacosis, single house infections usually occur from carrier Psittacidae, infection being carried by soiled feathers and dust or as virus particles.

Clinical Signs and Post-mortem Lesions.—These vary according to the species of birds affected. In pigeons the morbidity is high and mortality may be low. The young are more susceptible. There is usually diarrhoea, unilateral or bilateral serous conjunctivitis which is a very important sign, rattling respirations, and nasal discharge. Post-mortem examination reveals fibrinous plastic exudate in the pericardium and over the liver and mesentery. The liver and spleen are swollen and haemorrhagic. In the adult, the liver is markedly enlarged and may have pinpoint necrotic areas. In ducklings the lesions are similar to those seen in pigeons, but usually they do not show any clinical signs.

In the domestic fowl the liver and spleen may be enlarged. In most cases no clinical signs are seen during the course of infection. However, Storz, Call, and Miner (1963) have shown that it can cause meningoencephalitis in young chickens. Turkeys may show clinical signs in ornithosis infection. The more important clinical signs are diarrhoea, weakness, respiratory distress, conjunctivitis, and necrosis of the eye. The lesions resemble those in pigeons but pulmonary oedema or pneumonia, distension of gall-bladder, and yellowish-green discolouration of the vent are seen more commonly.

Diagnosis.—Ornithosis in turkeys and fowls has to be distinguished from *Mycoplasma* and *Escherichia coli* infections which produce lesions in the respiratory tract and the pericardial sac. This can be done by laboratory tests. Exudate from pericardium, liver, and other organs may be used to make impression smears and these can be stained with Giemsa to show the small elementary bodies which appear as coccoid structures in colonies, each less than $0{\cdot}5\,\mu$ in size. These are the causative organisms.

Bacteria-free filtrates can be used to inoculate white mice intraperitoneally, intranasally, or intracranially. Death may occur in 5–10 days, and post-mortem examination reveals fibrinous exudate in which the causal organisms can be demonstrated. The pathogenicity to mice depends on the virulence of the strain. Inoculation can also be done into the yolk-sacs of 6–10-day-old chick embryos and the agent may be demonstrated by suitable methods in the yolk-sac after 48 hours when the embryo usually dies. The infection can also be identified by complement fixation, cross-immunity, and agglutination tests. The pathogenicity of the various isolates of the virus differs considerably. The isolates from pigeons, chickens and parakeets are generally more virulent.

Treatment.—Chlortetracycline (Aureomycin) is the drug of choice and other tetracyclines are also effective. These may be given in water or food: 200–400 g. of a tetracycline may be given in a ton of feed for 3 weeks. However, this treatment does not cause complete elimination of the virus which may become drug resistant.

Many recovered birds are immune but some may be carriers of infection. No practical method has been used to immunize birds.

Prevention.—To prevent the spread of infection from one bird to another is a difficult matter, as in many instances it often goes unnoticed. However, when the

infection has been found in a flock, the flock should be culled. No regulations regarding the disposal of culled birds can be uniformly laid down. It is generally accepted that the ingestion of poultry meat, even of infected birds, does not cause psittacosis provided the meat is well cooked. The disease is a potential hazard to all those dealing with poultry or psittacine pet birds and to laboratory workers investigating poultry diseases. If the psittacosis agent is suspected to be present in a carcase when a post-mortem examination is done it is desirable to moisten the feathers of the carcase with a suitable disinfectant such as Lysol and keep it for several hours. Wearing gloves and face-masks may also help considerably in reducing the possibilities of infection occurring. No vaccine is commercially available for the prevention of psittacosis.

VIRAL ARTHRITIS AND SYNOVITIS OF CHICKENS

Olson and Kerr (1966; 1967) isolated and studied a viral agent closely related to the pox group of viruses, which was capable of causing arthritis and synovitis in chickens. This condition is characterized by swelling of the tendon sheaths, petechiae in the synovial surfaces, and lameness.

Specific arthritis in chickens may also be caused by *Mycoplasma gallisepticum*, *M. synoviae*, and several types of bacteria, chiefly *Pasteurella*, *Streptococci*, and *Staphylococci*.

EQUINE ENCEPHALOMYELITIS IN BIRDS

As the name indicates this disease is caused by a group of viruses which were initially found to cause disease in horses in the United States. Often the infected birds do not show any clinical evidence of illness. The viruses are transmitted by certain mosquitoes in which there is multiplication of the virus. Other biting insects may also transmit the disease mechanically.

Only chickens below 4 weeks of age show evidence of illness when infected and the chief signs are those of diarrhoea and prostration.

So far this infection has not been reported from Asia, and it has no great significance in the poultry industry. Its greatest significance lies, however, in the fact that man is susceptible to infection.

RABIES

This is a well known virus infection of dogs and other mammals including man; the fowl also is susceptible.

The incubation period varies but may be as short as 40 days. Birds usually get the infection by being bitten by a rabid dog. Once they are infected they may attack other birds and even man. Unlike mammals, infected birds may sometimes recover. There is no known treatment for the condition.

FOOT-AND-MOUTH DISEASE

This is a virus infection in the fowl caused by the same agents that cause foot-and-mouth disease in cattle and other ungulates. It is possible that it is more common in Asian countries than is generally recognized. In cattle, the disease is

caused by seven serological types known as O, A, C, S.A.T.1, S.A.T.2, S.A.T.3, and Asia 1. There are variants and sub-types within the main types.

As in the domestic animals, small vesicles appear on the comb and wattles, inside the mouth, and on the toes. The most common site is the dorsal surface of the tongue. These erode and leave ulcers which heal in about 2 weeks.

Some of the cases of naturally occurring foot-and-mouth disease in chickens reported in the literature are open to question, and it is likely that foot-and-mouth virus infection in the domestic fowl is not of practical interest to the poultry farmer.

GUMBORO DISEASE

This is a disease of growing birds between 3 and 6 weeks old, first described by Cosgrove (1962) in chickens in the Delmarva area in the United States, and which was named after the locality where the disease was first seen. The disease produces a nephritis-nephrosis syndrome in chickens and is essentially an infectious lymphocidal disease characterized by necrosis of lymphoid elements, chiefly the bursa of Fabricius, spleen, caecal tonsil, and thymus (Helmboldt and Garner, 1964; Cheville, 1967).

Winterfield and Hitchner (1962) found that this disease complex was caused by a variant of the infectious bronchitis virus (I.B.V.V.) and another termed the infectious bursal agent (I.B.A.). This agent appears to be spread by contaminated food and water and it may remain infective in contaminated premises for up to 122 days. The disease caused by the latter virus is now known as the Gumboro disease. The I.B.A. is comparatively resistant to moderately high temperatures, is 10–50 mμ in size and is not affected by a *p*H of 2 (Benton, Cover, Rosenberger, and Lake, 1967).

There are depression, diarrhoea, and muscular and visceral haemorrhages. Mortality may vary from 3 to 6 per cent. A characteristic feature is the distension of the bursa of Fabricius which may contain haemorrhages or a caseous core. Histologically the lymphocytes are replaced by heterophiles.

It is characterized by the enlarged bursa of Fabricius in the early stages, later leading to atrophy. Mortality occurs suddenly, and reaches a peak around the middle of the period during which the disease prevails, and drops equally suddenly to pre-infection levels (Parkhurst, 1964). The first clinical signs appear when the birds are about 20 days old. The affected chickens are markedly depressed, huddle together, and have soiled vent feathers.

Post-mortem examination reveals an enlarged bursa of Fabricius with a caseous core or haemorrhages. There may be haemorrhages on the thigh and breast muscles and the kidneys may be pale and have uratic deposits in some cases. Various control methods have been tried, but the best results appear to be obtained when the poultry houses are thoroughly cleaned out, the floors sprayed with lye, and the ceiling, walls, curtains, and equipment sprayed with a disinfecting detergent (Parkhurst, 1964) such as a quarternary ammonium compound.

The I.B.A. is highly contagious and is spread rapidly by contact. Transmission takes place through contaminated feed and water (Benton, Cover, and Rosenberger, 1967). Formalin may be used to disinfect the premises. The virus has been isolated from the lesser mealworm *Alphitobius diaperinus* (Panz.) and a vaccine has been prepared by passing it in the chick embryo (Snedekar, Wills, and

Moulthrop, 1967). The value of this vaccine has to be evaluated in the field as it produces immunity only up to 9 weeks.

VIRUS CONJUNCTIVITIS OF CHICKS

Lyabin, Pirog, Vinokhodov, and Lymbine (1966) reported an infectious disease in chicks 10–140 days old which caused follicular monocytic conjunctivitis, sinusitis, and tracheitis. There was high morbidity and mortality of growing chicks and retardation of growth of survivors. The morphology and pathology of the virus was studied and it was 80–90 mμ in size and was proved to be different from the viruses causing I.B. and I.L.T.

AVIAN UVEO-ENCEPHALITIS

An infectious avian encephalitis, probably caused by a virus different from that causing avian encephalomyelitis, has been described in Alsace, France (Gayot, Lavillsureix, Gruner, and Brini, 1962). The virus was isolated in day-old chicks, embryonated eggs, and cell cultures. The morbidity and mortality is high. This disease has not so far been reported from other countries.

CHICK EMBRYO LETHAL ORPHAN VIRUS INFECTION (*C.E.L.O.*)

Though this virus does not usually produce any clinical signs under natural conditions, when inoculated intravenously into chickens it can cause degeneration and necrosis of liver cells with the production of greyish-white necrotic foci or diffuse areas of necrosis of the liver (Kawamura and Horiuchi, 1964), and if it is inoculated into chicks by intracerebral inoculation it will produce nervous signs and death. If baby chicks are inoculated orally with the virus it will produce sneezing.

This virus is considered to be the cause of quail bronchitis, which is an acute contagious disease of bobwhite quail causing 50 per cent mortality (Dubose, 1967).

GAL VIRUS INFECTION

Pereira, Huebner, Ginsberg, and van der Veen (1963) have described a cyto-pathogenic adenovires (GAL) virus from fowls. A similar virus has been found to be associated with the virus of infectious laryngotracheitis in Australia.

AVIAN ENTERO VIRUS 1394

Dutta and Pomeroy (1967) have isolated an entero virus capable of producing cytopathic effects on chick embryo cells, necrotic foci in the liver of chick embryos, and large plaques on the chorio-allantoic membranes. Though the virus was isolated from chicks which showed cloacal pasting, it was not possible to reproduce the condition experimentally in other baby chicks.

AVIAN REO VIRUSES

Several reo viruses of birds have been isolated during the last decade. The exact pathogenicity of these viruses has not yet been determined. These include

the Fahey-Crawley virus, isolated from the respiratory tract of chickens with chronic respiratory disease. This virus causes focal necrosis of the liver in experimentally inoculated day-old chicks and produces syncytia in chick embryo and hamster cells *in vitro*.

Another virus isolated by Krauss from chicken with ulcerative enteritis causes degenerative foci and cellular infiltrations in the liver of experimentally inoculated baby chicks.

INCLUSION-BODY HEPATITIS

An inclusion-body hepatitis causing moderate mortality, depression, and diarrhoea has been described from Italy. The exact cause is not known; however, it may be due to a virus.

CHAPTER III

BACTERIAL DISEASES

SALMONELLA INFECTIONS

THESE are widespread infections caused by various species of *Salmonella* and they may be broadly classed into three main groups:—

1. Pullorum disease (bacillary white diarrhoea; B.W.D.) caused by *Salmonella pullorum.*
2. Fowl typhoid caused by *Salmonella gallinarum.*
3. Fowl paratyphoid caused by various other species of *Salmonella* which number over 850 types of which only about 20 are important.

PULLORUM DISEASE
(*Bacillary White Diarrhoea; B.W.D.*)

This is a very widespread infectious disease which usually runs an acute course in chicks and can also infect adults. In the adult, it usually runs a chronic course though acute outbreaks have been recorded. Due to extensive control methods adopted in the past several decades the incidence of this disease has decreased considerably, and it was eradicated in Scotland in 1965. However, the disease has recently become widespread in Australia where it is caused by variants of *S. pullorum.*

In chicks, it is characterized by heavy mortality in the first few weeks of life, which usually reaches a peak in the second week. Eggs laid by the carrier hens show decreased fertility and low hatchability, and chicks hatched from infected eggs show a high mortality. Further, the egg production in infected birds is lower than in the case of non-infected ones.

Cause.—The disease is caused by *Salmonella pullorum* isolated by Rettger in 1900. This is a non-motile *Salmonella* which when isolated from acute cases in MacConkey agar plates, produces small translucent colonies which are fairly characteristic. *Salmonella pullorum* ferments glucose and mannitol producing acid and some strains may produce small amounts of gas. Microscopically they appear as long slender rods measuring 3–5 × 1–1·25 μ with slightly rounded ends. They are Gram-negative and they have a definite antigenic structure. The antigenic components of the organism are IX, XII_1, XII_2, and XII_3. The XII_2 factor is variable and the standard strains contain only small quantities of this factor. Standard and variant types of *S. pullorum* have been found.

Epizootiology and Mode of Dissemination.—The most important source of infection appears to be the infective egg laid by the carrier hen. It has been found that on an average about 10 per cent of the eggs laid by the carrier are infected. Thus, these infected eggs and the chicks hatched from them transmit the disease in the incubator to the newly hatched healthy chicks. The excreta of the infected chicks are highly infective, and so are the shells and the dead embryos. Infection of

chicks and adults can also occur when they are fed infertile infected eggs. In this way, adult flocks may get acute attacks of the disease. Infection may also occur from contaminated litter, where *S. pullorum* may survive from 3 weeks in old litter to 11 weeks in new litter (Tucker, 1967).

Under natural conditions, turkeys, ducks, and guinea fowls are susceptible, though the latter two species are comparatively resistant. There are reports of man being infected with *S. pullorum*. Some workers claim that white leghorns are more susceptible to infection than the other breeds and that the male of the species is more resistant than the female.

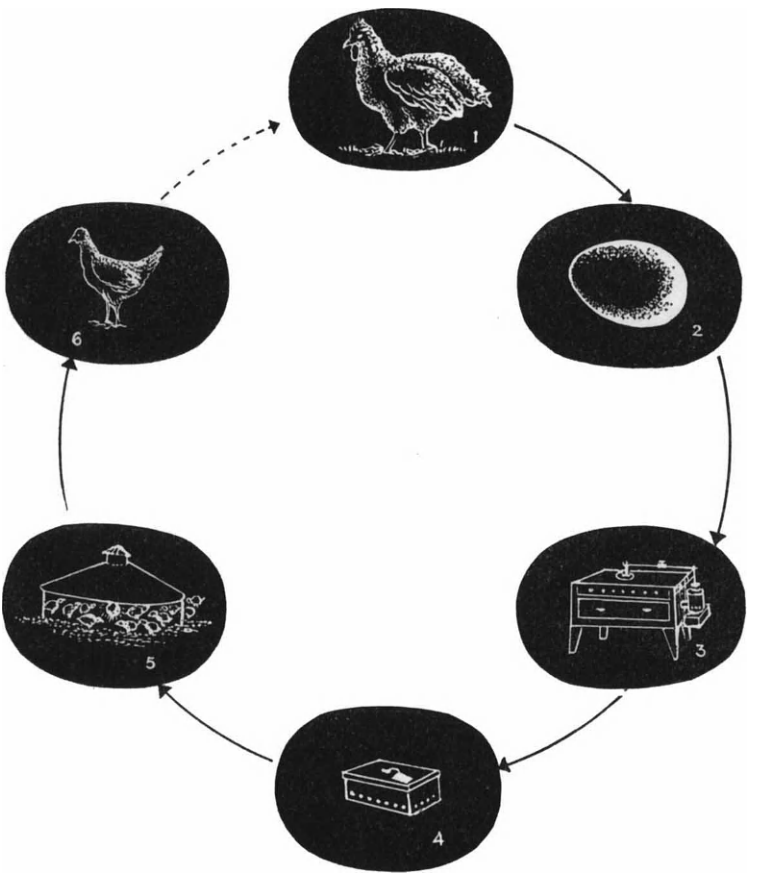

Fig. 7.—Mode of dissemination of pullorum disease. 1. Infected hen. 2. Infected egg laid by infected hen. 3. Incubator containing infected egg. 4. Chick box in which infected chicks may be present. 5. Brooder. 6. Surviving infected pullet which may be a carrier.

Infection through the infected chicken can thus occur in the incubator, chick shipping-boxes, and brooder houses. On the range, mechanical transmission via the shoes and poultry equipment can also occur. Carrier birds may infect others via the faeces, or when they are pecked at by other cannibalistic birds. Contaminated premises and infected flies can also transmit the infection. The more important modes of transmission are shown in *Fig.* 7.

Clinical Signs and Course of the Disease.—The incubation period averages 5–7 days. The disease usually takes an acute form in the chick which is more susceptible than the adult.

Moribund and dead chicks may be seen among those that are hatched from infective eggs in the incubator, or within a short time of hatching. Among chicks that are infected in the first few days of hatching, the symptoms are vague and they consist of drooping, ruffled feathers, laboured breathing, and a tendency to huddle together. Some will show white diarrhoea with a pasting of feathers round the vent. (Hence the term bacillary white diarrhoea.) Others may show a 'dipped-in-water' appearance.

The mortality pattern is fairly suggestive of the presence of infection. Lowered fertility and hatchability, together with some deaths soon after hatching, or many dead in shells, are suggestive of the presence of infection in the eggs. If infection occurs after hatching, such as will occur if a few chicks from infected eggs hatch out, mortality will be very high in the first 4 weeks of life and will reach a peak in the second week. Survivors may appear under-developed and poorly feathered.

In the adult, the disease does not usually manifest itself as an acute infection, though acute infections caused by the feeding of infected eggs are on record. As a rule, infected individuals cannot be detected by clinical examination. Some infected birds may show vague symptoms such as depression, listlessness, complete inappetence, paleness of comb, and diarrhoea. Acute cases usually run a septicaemic course and will show an elevated temperature.

Post-mortem Lesions.—Chicks having the very acute type of infection or born of infected eggs will not show many lesions. In these the liver may be enlarged and congested and the normal yellow colour may be streaked with haemorrhages, and the internal organs may show hyperaemic changes. Lungs may show changes of haemorrhagic pneumonia.

In the more protracted cases the absorption of the yolk may be poor, and the contents of the yolk-sac may be creamy or cheesy-white. Necrotic foci are seen, in descending order of frequency, in the liver, lungs, heart, gizzard, caeca, and large intestine. These foci may vary in size from that of a pinhead to that of a pea or larger. The lesions in the liver are usually small and pinhead in size, while those in the heart muscle and lungs are larger, being about the size of a pea. Sometimes pericarditis is seen and the spleen is enlarged. Kidneys are congested and the ureters distended with urates, and the caeca may contain a cheesy core.

In the adult carrier hen, discoloured cystic ova, some of which are misshapen and pedunculated, are characteristic. The oviduct may be impacted causing extensive peritonitis and adhesions in the various abdominal viscera.

In the male, the testes may be atrophied with thickening of tunica albuginea and multiple small abscesses. Both sexes often reveal pericarditis, a distorted heart with greyish-white nodules, an enlarged yellowish-green granular liver coated with fibrinous exudate, friable spleen with focal necrosis, and a fibrinous exudate of the abdominal viscera. These changes are suggestive of acute infection in the adult.

Diagnosis.—Though the mortality pattern in chicks and the lesions described may indicate the presence of pullorum disease, a definite diagnosis can be made only by isolating *S. pullorum* and identifying it.

In the adults, any one of the agglutination tests described below may be used. However, in the rapid whole blood slide agglutination test, false positives and doubtful reactions may be obtained in a number of instances.

In case of chicks dying of acute infection it is best to culture the heart blood or material from the liver in MacConkey agar and if *S. pullorum* is present, fairly characteristic colonies which are small and translucent may be seen in 24 hours. They may be later identified by the slide agglutination test and the fermentation and motility tests. *S. pullorum* is non-motile. It ferments glucose and mannitol with the production of acid and occasionally small amounts of gas. It produces hydrogen sulphide and it does not ferment dulcite and maltose. Rabbits are highly susceptible, while rats and pigeons are resistant to infection. These characteristics will enable the organism to be identified.

THE AGGLUTINATION TESTS.—The birds should be wing-banded or leg-banded and the number of each bird written on the test-tube. About 0·5–2 ml. of blood is collected into a clean dry test-tube from the wing vein by incising it with a lancet knife or a needle. The tube is laid on its side allowing the blood to clot in a long slant and is despatched to the laboratory with proper precautions to avoid haemolysis. In the laboratory varying dilutions of the serum are tested against standard antigen. If there are a fair number of doubtful reactions in a flock it is advisable to perform a bacteriological examination to eliminate typhoid and paratyphoid.

The organism may be isolated from the organs such as the heart, liver, ovaries, or blood by plating the material directly on MacConkey or S. S. agar. The plates should be dry before inoculation.

For isolation of the organisms from faeces it is advisable to inoculate faeces first with an enriched medium such as tetrathionate broth or selenite F. medium which suppresses the coliform bacilli, and then sub-culture on MacConkey agar. Examination of the MacConkey agar next morning shows that the colonies of non-lactose fermenters appear colourless, while those of coliform bacilli and other lactose fermenters produce pink colonies.

The Stained Antigen and the Rapid Whole Blood Test (*Rapid Test, Slide Test*).—This is the most widely used test in the field, because of its simplicity and the rapidity with which the results can be read. This consists of mixing 1 part of blood, usually a platinum-loopful (about 0·2 ml.) with 2–3 parts of antigen (about 0·5 ml.) on a plate, spreading it in a circular fashion over approximately 1 sq. in. and mixing the antigen and the blood with the loop or by rapidly rotating the blood. Generally speaking, a distinct agglutination in 1 minute is considered positive, and in 2 minutes doubtful, whereas if agglutination occurs after 3 minutes it is regarded negative.

The antigen used for these tests must be prepared according to standard techniques, and the details of the tests may vary slightly according to the antigen prepared. Therefore, it is best to rely on the instructions issued by the laboratory in the details of the procedure and the interpretation of the tests.

A positive agglutination test is indicated by the definite clumping of the bacterial cells within 1 minute, and should not be confused with the clumping of the erythrocytes.

A more recently developed antigen is of a polyvalent type, containing the standard and the variant strains, stained with crystal violet and produced according to the K formula which uses a colloidal sulphur medium.

The Rapid Serum Test.—This test is conducted in a manner similar to the rapid whole blood test, but instead, serum and unstained antigen, the turbidity of which has been standardized, are used. This test is not widely employed, and does not appear in any way superior to the whole blood test considering the longer time taken to obtain serum from a bird.

Other Tests.—Various other tests such as the intradermal, precipitin, and complement fixation tests are available but they are not widely employed, but a test called the spot test described recently may be of practical value though it is not widely employed.

A routine programme of testing should consist of the following procedures:—

1. All birds, especially layers, should be tested annually, particularly if they are breeding stock.

2. If infection is present, retesting should be carried out every 2–4 weeks until no reactors are found. Two consecutive tests which indicate absence of infection should be taken as the criteria for freedom from infection.

3. All reactors should be culled and sold for slaughter. It is advisable to cull the whole flock if the percentage of reactors exceeds ten.

4. After removing reactors the premises should be disinfected thoroughly. A satisfactory disinfectant for this purpose is 3 per cent solution of compound cresol compositus (liquor creosotis compositus). It is preferable, wherever possible, to transfer the non-reactors to separate non-infected premises.

5. Fresh stock, either adult, growers, day-old chicks, or eggs for hatching should be purchased from pullorum-free flocks.

6. Eggs should be saved for hatching only when the entire flock has been declared free from pullorum disease.

7. Fresh or infertile eggs, incubator waste, and uncooked swill should not be fed to poultry, particularly if they are from infected sources.

8. All incubator waste should be destroyed by incineration or by deep burial; offal of infected birds dressed for market and birds unfit for human consumption should be disposed of in a similar manner.

9. Owners of pullorum-free stock should not custom hatch for owners of infected or untested stock, nor should any of their own hatching be done in premises where infected stock may be found.

10. Birds taken to shows or newly purchased stock should be isolated for a few weeks and tested before they are taken into the farm.

11. The fumigation of incubators must be carried out as described on p. 12. For practical purposes fumigation may be done using 355 ml. of formalin and 17·5 g. of potassium permanganate for every 100 cu. ft. of incubator space, allowing the formaldehyde gas to act for 1–3 hours; the incubators should be thoroughly cleaned before fumigation.

12. All contaminated material should be disinfected or destroyed. For this purpose 4 per cent washing soda or 5 per cent hypochlorite solution could be used.

13. Eggs used for the preparation of live virus vaccines should be obtained from pullorum-free stock.

Treatment.—Treatment of the disease in chicks is not advisable. However, in acute disease outbreaks in adults, furazolidone may be used. The drug is administered in feed at levels of 0·04 per cent for 10 days for treatment and 0·01–0·015

per cent continuously for prevention. One hundred g. furazolidone per ton of feed given for 10–14 days should be adequate to stop mortality. However, treatment is to be regarded as a salvage operation and as there is the likelihood of treated birds becoming carriers, they should not be kept for egg production. In most cases treatment is not recommended. Furaltadone given at 0·5 g. per gal. drinking-water for 2 weeks and then at 0·25 g. per gal. for the next 6 weeks will protect chicks exposed to pullorum infection. This drug has the advantage of being given in drinking-water.

Fowl Typhoid

Fowl typhoid may be regarded as an acute septicaemic disease which sometimes runs a chronic course. The disease is more often seen in growers and adults, though birds of any age may be affected. Day-old chicks appear to be susceptible to infection.

Cause.—The disease is caused by *Salmonella gallinarum* which is a Gram-negative, relatively short plump rod about 1·0–2·0 μ wide. It ferments dulcitol and maltose with the production of acid alone and may produce hydrogen sulphide in nutrient broth. It is non-motile and has the somatic *Salmonella* antigens I, IX, XII. In contrast to these characters, *S. pullorum* is also non-motile but it does not ferment dulcitol and maltose. Like *S. pullorum*, *S. gallinarum* can also be easily grown on MacConkey's agar plates where small and translucent colonies appear. The causative organism does not live for long outside the bird's body, being destroyed in a few minutes by direct sunlight. It is easily destroyed by disinfectants. However, under certain conditions the organism can live for several months.

Clinical Signs and Course of the Disease.—The average incubation period is 4–5 days and may vary widely according to the pathogenicity of the organism. The average duration of the disease is about 5 days, but in chronic cases it may be much longer.

The symptoms of fowl typhoid are rather vague. However, a fair proportion of birds show a greenish-yellow diarrhoea, with pasting of the feathers around the vent. They show an elevated temperature, listlessness, and marked thirst. In the acute cases the comb is congested, whereas in the chronic cases the comb is pale and shrivelled. In some cases there may be arthritis and pericarditis.

At the commencement of the outbreak the mortality may be high but later it decreases. Mortality and morbidity vary considerably.

Post-mortem Lesions and Diagnosis.—In very acute cases, no gross changes may be seen, but in acute cases, the liver and kidneys are enlarged, and on exposure to air the liver presents a greenish-brown or bronze colouration which is characteristic. In chronic cases, small white foci may be seen in the liver, and the other changes as seen in chronic pullorum disease in the adult may be commonly encountered.

A fair proportion of cases may also show catarrhal haemorrhagic enteritis of the upper third of the intestine and petechial haemorrhages in the fat and muscles. In chronic cases the spleen is markedly enlarged. There is severe hyperplasia of the reticulo-endothelial system.

The characteristic bronze colouration and the enlargement of the liver are regarded as being diagnostic of the disease. However, when this change is not present it has to be distinguished from fowl cholera, which is usually more acute, avian monocytosis, Newcastle disease, and fowl plague. A specific diagnosis can only be obtained by cultural examination of the blood or organs and when *S. gallinarum* can be isolated and identified by the characters described before.

Pullorum antigen will give a positive reaction with blood or serum of a bird infected with fowl typhoid. Diagnosis may also be made by animal inoculation. Rabbits are susceptible to *Pasteurella multocida* the causative organism of fowl cholera, whereas they are resistant to *S. gallinarum*. This fact may be made use of to distinguish fowl typhoid from fowl cholera.

Epizootiology, Prevention, and Control.—Infection usually occurs via the infective bird, through the egg, and mechanically through man, contaminated equipment, food, water, litter, and through carrier birds. Generally speaking it is spread by the same means as is pullorum disease, but mechanical transmission appears to be more important than egg-borne transmission.

Successful eradication of fowl typhoid is based on good hygiene, such as proper disinfection and proper management, hatching eggs from non-infected flocks, and obtaining replacement stock from disease-free hatcheries.

Several types of vaccines have been used. However, no single vaccine can be generally recommended. A vaccine using the 9R strain developed by Dr. William Smith has been in use in the U.K. for some time. The immunity established by these vaccines is not strong and durable. The use of vaccines may cause the flock to react positively to the pullorum test.

Fowl typhoid may also be prevented by the continuous feeding of furazolidone at the rate of 50 g. per ton. However, this is not recommended for universal practice.

Treatment.—Several sulphonamides have been used with varying degrees of success in the treatment of this condition. Of these, sulphaquinoxaline appears to have given good results. This can be given in drinking-water to secure a concentration of 1: 2500 (or 1 oz. of a 3·4 per cent aqueous solution in 1 gallon of water) until mortality is controlled.

Furazolidone given at a level of 100 g. per ton of feed for 10–14 days or until mortality has stopped, appears to be superior to the sulphonamides. When the mortality is arrested the strength may be reduced to 50 g. per ton and given continuously until the birds are marketed. This drug has the added advantage that it prevents the excretion of the *Salmonella* in faeces. Furazolidone may also be given as in *S. pullorum* infection. Recently, strains of *S. gallinarum* resistant to furazolidone have been isolated in the U.S.A. Nihydrazone in concentrations of 0·011 per cent is reported to be very effective in the infection (Freedman, Johnson, and O'Connor, 1965). Whatever drug is used, it is not possible to eliminate the *Salmonella* from the ova, and for this reason the treated birds should not be used for breeding purposes.

The disease may be eradicated on the lines adopted for the control and eradication of pullorum disease. The birds have to be blood-tested and the carriers eliminated. For this purpose pullorum antigen may be used. In very severe or

extensive outbreaks, depopulation, thorough disinfection, and restocking the premises after a short interval may be the only satisfactory method, even though it may be an expensive procedure.

SALMONELLOSES
(*Paratyphoid*)

Salmonelloses in poultry in the present context is used to designate infection of poultry caused by various members of the genus *Salmonella*, other than infections caused by *S. pullorum* and *S. gallinarum* which are given the specific names of pullorum disease and fowl typhoid respectively, and which have been described already.

The disease is usually seen in chicks below a month old, in which it usually runs an acute course. It may be also seen in the adults. Adults are more resistant to infection, but generally speaking they remain carriers quite often.

This disease is increasing in incidence in most parts of the world (Buxton, 1957; Wilson, 1962). Severe mortality has been reported in chickens in Australia due to *S. newington*.

Cause.—Any of the various species of *Salmonella* of which there are over 850 serological types may cause infection. But the most important of these is *S. typhimurium* which appears to have a world-wide distribution. This species alone is responsible for more than half the number of cases of salmonelloses in poultry. The other important species responsible for the disease are *S. thompson* and *S. enteritidis*. The relative importance of the various species of *Salmonella* varies according to the conditions in the country and the locality. Recently, acute outbreaks of salmonellosis have been reported in Ceylon due to *S. bareilly* and *S. typhimurium* (Kulasegaram, 1963). In a 5-year survey on the incidence of *Salmonella* in avian species in Massachusetts, U.S.A., it was found that of a total of 4454 consecutive consignments submitted for diagnostic investigation, 245 were positive for *Salmonella* and that they consisted of 34 serotypes among which *S. typhimurium* had the highest incidence (Faddoul and Fellows, 1966). *S. typhimurium* causes widespread infection in most countries and is associated with focal necrotic lesions in the liver, lungs, and spleen of affected birds. A characteristic feature appears to be the presence of stalked and angulated ova in the affected hens (Kashiwazaki Aoki, Horiuchi, Shoya, and Namiaka, 1966). *S. menston* appears to be an important pathogen in the United Kingdom and it may be transmitted via the egg. The *Salmonella* are Gram-negative, non-sporing bacilli 0·4–0·6 × 1–3 μ in size. On agar cultures the colonies are round, slightly raised, glistening, with smooth edges, and are 1–2 mm. in diameter. They ferment dextrose, mannitol, maltose, and sorbitol with the production of acid and gas. The identification of various species of *Salmonella* is usually entrusted to a central laboratory where facilities for serological work are available, and even so, only few experts are capable of determining all the *Salmonella* serotypes that have been described.

Chicks below 1 month of age and turkey poults are particularly susceptible. Older birds, especially those subject to stress factors such as malnutrition, may show an increased susceptibility.

Clinical Signs and Course of the Disease.—The symptoms of paratyphoid in young birds are vague, and in many ways resemble those of pullorum disease. The highest losses are usually experienced between the sixth and tenth day of infection. If eggs are infected there is usually a high proportion of pipped and unpipped eggs containing dead embryos.

The affected chicks may huddle together and show loss of appetite, droopy wings, increased thirst, and watery or white diarrhoea with pasting up. Mortality in some cases reaches 80 per cent though usually it is around 30 per cent. These symptoms are seen in other chick diseases, and therefore only a bacteriological examination will determine the cause. In the adult, the symptoms are vague and in the acute cases there is diarrhoea and dehydration. Mortality is usually less than 10 per cent.

Post-mortem Lesions and Diagnosis.—In acute cases in chicks, lesions may be entirely absent, but in less acute cases lesions similar to those encountered in pullorum disease are seen, but nodules are seldom found in the heart and lungs. The prominent lesions are unabsorbed yolk-sac, congested liver with haemorrhagic streaks and pinpoint necrotic foci, duodenitis, and caecal cores. Nodules are often present on the small intestine. *S. stanley* may produce suppurative necrotic lesions in the ovaries and peritonitis as seen in pullorum disease.

A definite diagnosis can be made only by bacteriological examination as outlined in pullorum disease, and the identification of the *Salmonella*. Unlike *S. pullorum* and *S. gallinarum* the *Salmonellae* causing salmonellosis have flagella and are motile and unlike them they produce acid and gas in maltose and dulcitol. They differ from *Escherichia coli* in that they do not ferment lactose and produce not indole but hydrogen sulphide. *Pasteurella multocida* differs from *Salmonellae* in that it ferments sucrose and forms indole and does not produce gas from sugar.

Epizootiology, Prevention, and Control.—The *Salmonellae* live in water and vegetables for considerable periods and humidity favours viability. *S. thompson* may survive in the litter from 5–20 weeks. They survive longer in new litter (Tucker, 1967). They are easily destroyed by lye and by formaldehyde fumigation.

The adult bird serves as a carrier and pigeons and canaries may also carry the organisms. The organisms are shed in the faeces, and eggs may thus be contaminated. They are capable of active penetration of the egg-shell, which usually occurs in the first 7 days of incubation, and they appear to concentrate in the yolk. Incubation temperature and high humidity appear to enhance their penetrating properties. Unlike in pullorum disease, infection of the eggs rarely occurs in the ovary. However, direct ovarian transmission appears to be quite common in ducks.

Rats and mice are frequent carriers of these organisms and so are flies. Birds can also be infected by contamination of feed, coconut cake, and water. Food, especially if containing meat scraps has been found to contain *Salmonella*. Human beings and most animals are susceptible to infection and therefore may act as carriers. Repeated passage of organisms through a single species causes an increase in their virulence. Because of the complexity of the problem, well tried and specific control or eradication programmes are not available. It is advisable to use an initial serologic test using *S. typhimurium* or *S. pullorum* antigen. If the flocks are

small they may be culled and the premises thoroughly disinfected and restocked with healthy birds.

The incidence of the disease can be reduced by flock sanitation and good management practices. These include the following—(1) known infected flocks should not be used as a source for hatching eggs; (2) early fumigation at the commencement of incubation (before the 24th hour) using three times the amount of formalin–potassium-permanganate mixture recommended earlier; and (3) rodent and fly control and the separate rearing of ducks and turkeys. Apart from taking these measures, flocks must be tested regularly, and precautions must be taken to reduce the contamination of incubators, eggs, food, and water from infective faeces. This is done by getting rid of the carriers. Vitamin A appears to increase the resistance of birds to *Salmonella* infections and to reduce the mortality and the percentage of carriers.

As a fair proportion of outbreaks of salmonellosis are transmitted by contaminated feeds, it is advisable to use pelleted feed which has been subjected to a higher temperature (200–350° F.) and moisture (25–35 per cent) range than non-pelleted feed. At these higher temperature and moisture ranges, the *Salmonellae* are destroyed.

After a confirmation of an outbreak of salmonellosis has been made, the pens should be depopulated and the litter destroyed, preferably by burning. Before the litter is removed for destruction it should be moistened to prevent *Salmonellae* being distributed via dust particles.

Treatment.—Sulphonamides are partly effective but furazolidone and furaltadone (which is a water-soluble nitrofuran) are superior. However, none of these drugs can completely eliminate the organisms from the birds. Therefore, most of them remain as carriers, and shed the organisms in faeces. Treated birds should not be used for egg production or for breeding purposes, but they should be sold for slaughter as soon as they reach the proper market size. Nihydrazone may also be used in the treatment as in *S. gallinarum* infection.

The main purposes of treatment are to reduce mortality and make the birds marketable.

Of the sulphonamides, sulphadimidine and sulphamerazine are useful. Sodium sulphamerazine is given in drinking-water as a 0·4 per cent solution while 0·5 per cent sulphamerazine can be given in mash. Furazolidone is given at the rate of 50 g. per ton of feed (0·0055 per cent) in mash on a continuous basis as a preventive, to birds over 2 weeks of age, or at the rate of 100 g. per ton of feed (0·011 per cent) as a preventive to birds in the first 2 weeks after hatching. This dosage may be doubled for treatment. Furaltadone when used at 0·04 per cent in drinking-water was effective in controlling mortality due to *S. gallinarum* and *S. typhimurium* (Tucker, 1963). This drug is considered superior to others. However, the carrier state may not be eliminated.

Other antibiotics such as the tetracyclines have been tried with varying results. Organisms causing salmonelloses are more resistant to antibiotics and furazolidone than *S. pullorum* and *S. gallinarum.* Most *Salmonellae* are susceptible to chloramphenicol (Chloromycetin), though the degree of susceptibility varies considerably with each species and the strain. However, this drug is not widely used in poultry practice.

PARACOLON INFECTIONS

These infections are caused by a group of bacteria belonging to the family Enterobacteriaceae occupying a position intermediate between typical coliforms and the paratyphoids. They ferment lactose only on prolonged incubation.

The infections are seen most often in chicks less than 1 month old, and in such cases stress factors usually predispose the birds to infection. Such factors are cold, rain, unfavourable changes in environmental temperature, bad environment, overcrowding, and bad food.

Cause.—The pathogenic paracolons are placed in a group known as Arizona paracolons, and they have as wide a distribution and host range as the *Salmonellae*, and in other cultural and biochemical aspects they resemble *Salmonellae* of the paratyphoid group. However, they ferment lactose with the production of gas after 7–10 days, and they do not ferment dulcitol but liquify gelatin after 7–10 days.

Clinical Signs and Course of the Disease, Post-mortem Lesions, and Diagnosis.—These resemble those seen in paratyphoid and pullorum disease. Usually only chicks less than 1 month old are affected, in the presence of certain stress or predisposing factors. The mortality may vary from 10 to 50 per cent. There are usually signs of diarrhoea and pasting-up. In addition to this, various nervous signs such as ataxia, tremors, and convulsions may be seen. Eyes may show an opacity and this may finally lead to blindness.

The principal lesions are a mottled yellow liver, a discoloured heart, peritonitis, and an unabsorbed yolk-sac. Unlike in pullorum disease and fowl typhoid infections, the infected birds do not remain carriers and the infection is usually overcome in 1 month.

A diagnosis can only be made by cultural examination of the various organs and heart blood, and the isolation and identification of the organisms concerned. Unless extended incubation up to 10 days is practised, the organisms isolated may be discarded as being of no significance.

Epizootiology, Prevention, and Control.—The disease may be considered to be one resulting primarily from bad management. The same methods used in the control of paratyphoid infections are useful to control this infection. However, if the shells of eggs to be incubated are likely to be infected, fumigation should be done with 3 times the amount of potassium-permanganate–formalin mixture used to control pullorum disease. Fumigation should be done, as soon as the eggs are placed in the incubator and its working conditions have been set. Fumigation should not be done between the 24th and 96th hours of incubation.

Treatment.—Treatment of these infections is usually more difficult than of those due to *S. pullorum* and *S. gallinarum*. However, unlike these infections, treatment is advocated as the birds do not usually remain carriers. Sulphonamides particularly sulphamerazine and furazolidone may be given as in paratyphoid infections. Furazolidone is given at the rate of 100–200 g. per ton of feed for 7–10 days as a curative or at least 100 g. per ton of feed for one week each month as a preventive.

FOWL CHOLERA

This is usually an acute septicaemic disease of the domestic fowl and various other species of birds including turkeys, pigeons, and pheasants caused by *Pasteurella multocida* (*P. aviseptica*). Less often it runs a chronic course particularly in the endemic areas, or in more resistant birds.

Cause.—The disease is caused by *Pasteurella multocida* (*P. aviseptica*). The organism is 0·6–2·5 × 0·25–0·4 μ in size, and in the blood and tissues of the acutely infected bird and in a recently isolated culture, the organisms are distinctly bipolar if stained with Leishman's, Giemsa, or methylene blue stains. It produces acid in saccharose and sorbitol, forms indole, and is pathogenic to the white rat (Soltys, 1963). Several types (usually 4) are recognized according to immunological and other reactions. Four types of colony variants are also encountered. These are the mucoid, smooth (a), smooth (b), and the rough colony variants (Parry, 1966). Dorsey (1963) has studied the details of the biochemical, serological, and immunological nature of avian *P. multocida*. The organisms do not appear to remain in the yard for more than a few weeks after the last death due to fowl cholera has occurred. However, they may remain alive for at least 1 month in the manure and for 3 months in infected decaying carcases.

Rabbits and white mice are very susceptible to infection by any of the usual routes while guinea pigs are more resistant. Man may get a localized infection.

Clinical Signs and Course of the Disease.—The disease is not generally seen in fowls below 4 months. However, in the young it may be seen as foot abscesses or arthritis. Usually older birds are affected and the incubation period varies from 4 to 9 days but in experimental cases it may be as short as 2 days. In the peracute cases death is seen without any symptoms. This usually occurs at the commencement of the outbreak, after which the acute cases are seen. In such cases there is greenish-yellow diarrhoea, purplish comb and wattles, elevated temperature, and difficult breathing. Death occurs in a few days. Turkeys are often affected with the peracute form of the disease. The incidence of this disease in turkeys appears to be increasing in Texas (Grumbles, 1965).

In the subacute cases, there may be swelling of the wattles (*Fig.* 8), enlargement of the joints, râles or rattling respirations, catarrhal discharge from the nose, and torticollis due to the localization of the organisms in the ear or the base of the brain. These chronic cases are seen at the end of an epidemic or in the more resistant flocks. In Great Britain chronic forms are seen more frequently.

Post-mortem Lesions and Diagnosis.—In the peracute cases no lesions are seen. However, in the less acute cases, petechial haemorrhages are seen especially on the myocardium, gizzard, proventriculus, peritoneum, coronary and abdominal fat, and other organs (*Fig.* 9). The duodenum is inflamed and contains viscous mucus. In turkeys there is extensive purulent pneumonia, often resulting in almost complete consolidation of one or both lungs. The cranial form of fowl cholera in the turkey is often manifested by torticollis, and there are extensive yellowish caseous exudates in the air spaces of the bones of the head.

The liver is often enlarged and shows parenchymatous hepatitis. In the less acute cases there are pinhead-like greyish necrotic foci in the enlarged liver (*Fig.* 10),

which may also contain petechial haemorrhages and be streaked with light areas. In some cases there may be serofibrinous pericarditis and catarrhal tracheitis with excess of mucoid exudate. In the chronic cases there may be joint and wattle

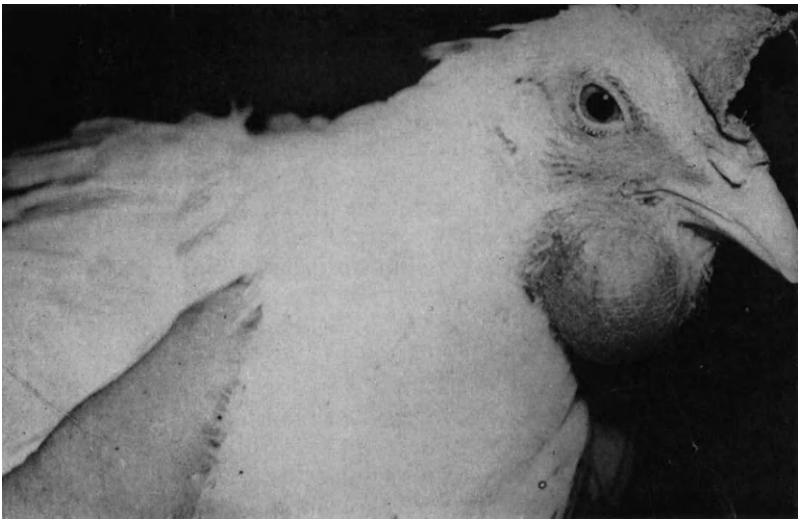

Fig. 8.—A fowl with a chronic form of fowl cholera, showing oedema of the wattles.

lesions containing cheesy material. In others the abdominal cavity may contain this yellow material. Similar lesions may be seen in the auditory canal and the brain.

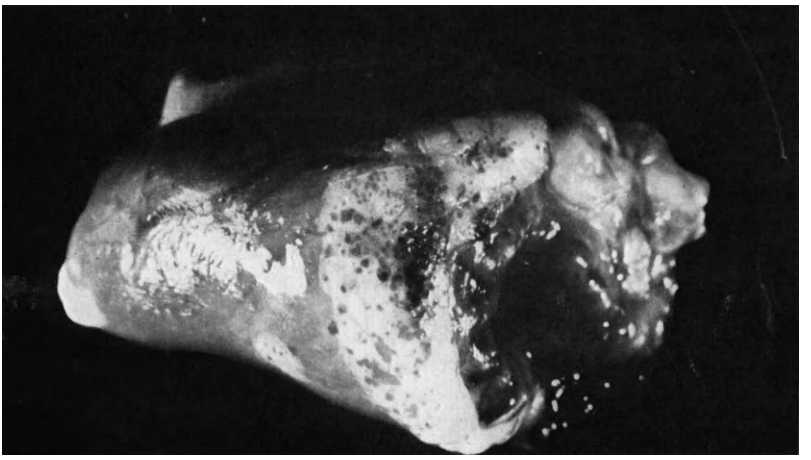

Fig. 9.—The heart of a fowl with fowl cholera, showing petechial haemorrhages.

The disease must be differentiated from fowl typhoid, Newcastle disease, fowl plague, and other respiratory infections. A tentative diagnosis can be made when bipolar organisms are seen in smears from the heart blood or impression smears

of the liver, and this can be confirmed by cultural methods and by identifying the organism by its biochemical characters and pathogenicity tests. *P. multocida* can be isolated readily by using a basic medium of tryptic digest beef agar, Difco tryptose agar with Fildes' digest sheep blood, sodium sulphide, neomycin, and potassium tellurite (Soltys, 1963).

Epizootiology, Prevention, and Control.—The organisms live in the upper respiratory tracts of healthy carrier chickens, and in the presence of some exciting factors such as unfavourable weather conditions, sudden changes in management,

Fig. 10.—The liver of a fowl with fowl cholera, showing enlargement of the organ and multiple pinhead greyish necrotic foci.

etc., they assume pathogenicity. When they become virulent the disease starts and spreads. Thus the disease is usually seen in autumn in the temperate countries and during the periods of heavy rainfall in the tropical countries. Infection usually occurs via the respiratory tract or less often via the digestive tract. Perhaps wild birds and other species of domestic poultry play an important part in the dissemination of the infection.

Recently, vaccines have been produced and used in Russia. These have been produced from an avirulent strain (AV) and the Krasnodar strain (K). In U.S.A., multivalent vaccines of the water-in-oil emulsified type are available. However, in most cases, it is advisable to prepare autogenous vaccines to deal with individual outbreaks. Good management, and resting the contaminated yards for at least 3 months, will reduce the incidence of the disease. Flock treatment with sulphonamides will reduce mortality. Separate the different species of birds and discourage the entrance of crows, sparrows, and other wild birds into the poultry yard by shooting them or scaring them away.

For a considerable time, various bacterins have been used to control this disease, especially in certain parts of the U.S.A., but the results obtained with these vaccines have not been very satisfactory (Grumbles, 1965).

Treatment.—Certain sulphonamides, streptomycin, and the tetracyclines are effective against the organisms causing fowl cholera. Of the sulphonamides, sulphadimidine, sulphamerazine, and sulphaquinoxaline have been proved to be of value. Of this group probably sulphaquinoxaline is the most useful and may be given in drinking-water or in mash in concentrations of 0·033 and 0·04 per cent respectively. The drug may be given in mash for several weeks without ill effects. Mortality may occur once the treatment is discontinued.

PSEUDO-TUBERCULOSIS

This is caused by *Pasteurella pseudo-tuberculosis*. The disease is seen more frequently in ducks and turkeys than in the domestic fowl. It may also be seen in wild or game birds, cage birds, and pigeons. It frequently affects rodents.

The disease is usually associated with bad management and is characterized by an acute septicaemia followed by a chronic infection giving rise to tubercular lesions in various organs (Parry, 1966). There are multiple greyish-white foci in the liver, spleen, kidneys, and breast muscles. A severe enteritis is common. The symptoms and lesions resemble those seen in fowl cholera. Treatment is more difficult. Chloramphenicol and neomycin are effective against this infection.

AVIAN TUBERCULOSIS

This is a chronic infectious granulomatous condition of the fowl caused by *Mycobacterium tuberculosis* (avian type). It is a disease primarily seen in older birds (usually those over 1 year) which are kept in unhygienic environments and poor conditions of management. The incidence of the disease has decreased considerably, and in the tropics does not appear to be a problem at all.

Cause.—*Mycobacterium tuberculosis* (avian type), the causative organism, is alcohol and acid fast, measures 1–3 μ long, grows most luxuriantly on media containing glycerin, and, unlike the human and the bovine tuberculosis bacilli, grows readily on egg medium, the colonies being oily and moist. In liquid media it forms a pellicle with a crumbly growth at the bottom. It is pathogenic to the fowl, rabbit, swine, sheep, and rarely, to man. It is only slightly pathogenic to guinea pigs. When cattle are sensitized there will be an increased number of doubtful reactions to the tuberculin test. There have been one or two cases of cattle infected with avian T.B. including uterine infection, but it is rare.

Clinical Signs and Course of the Disease.—The incubation period is long, being several months. Only old birds show symptoms. These are vague and include emaciation, a pale and shrivelled comb, lameness, a decrease in egg production, and diarrhoea. The keel becomes very prominent and often deformed. Mortality is low but is extended over a period.

Post-mortem Lesions and Diagnosis.—There is emaciation and dehydration, and greyish-white or yellowish granulomatous caseous nodules of variable size are

seen in the intestines, mesenteries, liver, spleen, and bone-marrow. Liver and spleen are enlarged and ascites is seen frequently. The larger nodules are nobbly and they contain soft yellow centres surrounded by a fibrous capsule. The nodules in the gut wall usually open into the intestines, discharging large numbers of bacteria, and they readily shell out. At times, there may be haemorrhage into the abdomen due to a rupture of the liver, spleen, or a blood-vessel. Lesions in the lung are uncommon.

The history of the flock and the lesions seen in the dead birds are suggestive of tuberculosis. Impression smears from the lesions particularly from the periphery will reveal acid and alcohol fast bacilli when stained by the Ziehl-Neelsen method. Cultural examination and animal inoculation will confirm the diagnosis.

A tuberculin test may be used in infected flocks to determine carriers or other infected birds. In this test, 0·05–0·1 ml. of avian tuberculin is injected intradermally. A positive result is indicated by a warm oedematous swelling which appears in about 24 hours and lasts till 72 hours. The test should be read at 48 hours.

Epizootiology, Prevention, and Control.—Infection is introduced into the premises by an infected bird. Many species of wild birds are susceptible to infection thus acting as reservoirs and sources of infection. Once it is introduced into a farm it may remain there for years. Infection usually occurs by ingestion, and the bacteria are usually eliminated in the droppings. Tubercle bacilli have been transmitted in a few cases in the eggs of poultry (Blaxland, 1961b).

Prevention is effected by good sanitation and by avoiding introducing birds from unknown sources, or where the disease status is not known.

If the disease is seen in a flock it is advisable to test the flock and destroy the reactors, while the others may be removed to a clean area, the birds tested again in a month, and the reactors destroyed. This may be repeated until there are no reactors. However, where the economic loss would not be great, it is advisable to get rid of the whole flock, i.e., destroy the reactors and sell the others for table purposes. Then the houses are thoroughly cleaned with washing soda, disinfected with 3 per cent compound solution of cresol, limed with 2 tons of lime per acre, and the land rested for at least 2 months. Animals such as swine, sheep, and cattle should not be allowed to come into the infected premises.

Treatment.—Treatment should not be undertaken, all positive birds must be destroyed, and if the flock is small it may be advisable to cull the remainder.

COLI GRANULOMA
(*Hjarre's Disease*)

This is a chronic granulomatous condition of the domestic fowl and turkey, where granulomatous growths, resembling those of tuberculosis, are seen in the organs associated with the digestive tract, caused by a mucoid strain of *Escherichia coli.*

Clinical signs are usually vague and consist of emaciation and unthriftiness. The disease is usually diagnosed at autopsy when lesions similar to those seen in tuberculosis are found, chiefly in the caeca and the liver; the latter shows enlargement and irregular areas of necrosis. Similar granulomas may be seen in the spleen and all along the small intestines.

The incidence of the disease is low. It also spreads very slowly. Infections occur by ingestion of *E. coli*. Though the disease has no great economic significance its importance is due to the fact that it may be confused with tuberculosis.

The absence of acid fast bacilli in the lesions and the isolation of mucoid colonies of *E. coli* which can reproduce the disease in experimental chickens settles the diagnosis.

The disease has to be distinguished from avian tuberculosis and nodules caused by certain types of tapeworms. There is no satisfactory treatment, and control may be affected by applying general principles of hygiene.

Other granulomatous conditions are caused by certain staphylococci, by various tumours probably viral in origin, and by certain species of tapeworms.

INFECTIOUS BACTERIAL CORYZA

This is an acute coryza of short duration caused by *Haemophilus gallinarum*, characterized by a serous discharge from the nose which is associated in many cases with the swelling of the eyelids and conjunctivitis. The disease has a short incubation period. This is Nelson's coryza type 1.

Cause.—This is a haemophilic, pleomorphic, Gram-negative bacterium which exhibits bipolar characteristics. The organism requires only the V factor for growth and does not live long outside the body. Hence, its incidence is decreasing. The principal mode of transmission is by drinking-water contaminated with infective nasal discharge. This organism may quickly disappear from the infected birds after which many bacteria especially *Pasteurella multocida* may be found in the infected tissues. The exact role of these organisms has not been established.

Clinical Signs and Course of the Disease.—The disease is more often seen when the weather conditions are unfavourable. There is usually an acute coryza with swelling of the face, and conjunctivitis, and when it becomes chronic the eyes may be distended with cheesy material. The incubation period is usually 1–2 days and unless it is complicated the disease lasts only about one week. However, when complications arise, it may last 1–2 months or longer. It may be associated with avitaminosis A, C.R.D., and fowl pox. The virulence of the disease may vary and it appears to be seasonal in incidence. The disease may be spread by direct contact, by the air-borne route, or by water.

Diagnosis.—This can be made by the isolation of *H. gallinarum* and the reproduction of the disease. It has to be distinguished from mycoplasma coryza, C.R.D., avitaminosis A, and other diseases likely to produce coryza-like symptoms. There are no significant lesions except the distended sinuses and conjunctival sacs, and the nasal sinuses containing cheesy material, which are usually seen in the complicated cases.

Treatment.—Sulphonamides are useful. Sulphathiazole when given in mash or drinking-water is effective. The dosage is ½ lb. of sulphathiazole per 100 lb. of mash. Streptomycin in doses of 0·1–0·2 g. intramuscularly is also effective in the uncomplicated cases. However, this drug is not recommended for field use as treatment of

individual birds is not worthwhile. Sulphadimidine (Sulphamezathine, I.C.I.) is also effective in the treatment. Erythromycin is also effective.

MYCOPLASMOSIS

This is caused by infection with members of the genus *Mycoplasma* and causes considerable economic losses. In the U.S.A. alone, the annual losses due to *Mycoplasma gallisepticum* have been estimated to be $125,000,000 (Kesteven, 1965). The disease may be divided into two forms: respiratory mycoplasmosis and infectious synovitis.

Respiratory Mycoplasmosis
(Chronic Respiratory Disease, C.R.D.; Nelson's Coccobacillary Coryza; Nelson's Mycoplasmosis)

History.—Nelson (1936) described a coryza of slow onset and prolonged course which he termed 'type II coryza' in his classification of coryzas of the domestic fowl. He consistently recovered coccobacillary bodies from the infected tissues of his experimental birds and hence this was termed coccobacillary coryza. Delaplane and Stewart (1943) described an apparently new respiratory disease in the U.S.A., which they called chronic respiratory disease (C.R.D.). Later it was established that these two diseases were caused by the same organism, *Mycoplasma gallisepticum*. Since this organism affects primarily the respiratory tract, the disease caused by it is now called respiratory mycoplasmosis.

Aetiology.—The uncomplicated form of the disease is caused by *Mycoplasma gallisepticum*, a minute coccoid filtrable organism about 0·5 μ in diameter, which can infect turkeys and ducks as well. It requires for its growth a medium enriched with serum. Most strains agglutinate washed chicken erythrocytes. At least 20 serotypes have so far been described from the respiratory tracts of birds; not all are pathogenic to the domestic fowl.

M. gallisepticum multiplies in the mucous membrane of the nasal chambers and trachea, and the air sacs, even in the presence of humoral antibody. Its multiplication is for the most part extracellular (Corstvet and Sadler, 1966). It can be grown on special media or in chick embryos using the yolk-sac route of inoculation. Growth on artificial media is usually slow and may take 7 days or longer to produce distinct colonies.

M. gallisepticum may produce disease by itself or its action may be enhanced by stress or any one or more of the following agents:—

1. *Escherichia coli.*
2. Virus of infectious bronchitis.
3. Virus of Newcastle disease.
4. *Haemophilus gallinarum.*
5. Virus of Fahey and Crawley.
6. C.E.L.O. virus.

Clinical Signs and Course of the Disease.—In the absence of other respiratory infections, *M. gallisepticum* infection may not cause detectable respiratory symptoms, but in laying flocks the usual sequelae are poor egg production, reduced

hatchability, and egg transmission of infection. The incubation period averages 11–18 days and the disease is most frequently seen in broilers and birds between 4 and 8 weeks of age. The most prominent signs are those of a cold—nasal discharge, distended sinuses, lowered egg production, poor carcase quality, and decreased feed conversion. The symptoms are usually confined to those manifested in the respiratory tract. The disease may last from 3 weeks to about 2 months and it spreads through the flock slowly and insidiously, so that, at the end of a month or two, nearly the whole flock is infected. The disease takes a longer course under adverse environmental conditions, such as in winter or in monsoon conditions in the tropics. The mortality is usually below 3 per cent but it may be much higher if there are complications. In India, certain outbreaks have caused mortality up to 30 per cent (Rao and Agarwal, 1966).

Post-mortem Lesions and Diagnosis.—There is usually a thick tenacious mucus in the nostrils and trachea, and tracheitis. Nasal and sometimes other sinuses of the head contain cheesy material. The walls of the air sacs and trachea are thickened and the former contain large amounts of cheesy material which may also be deposited over the heart, lungs, liver, and in the abdominal cavities. Skeletal muscles, especially those of the breast, may show whitish striae.

The histopathological lesions are splenitis, myocarditis, hepatitis, sinusitis, tracheitis, and pneumonia. The mucous membrane of the turbinates shows large discrete lymphoid follicles, lymphocytic infiltration, hyperaemia, and sometimes epithelial desquamation. Lymphocytic infiltrations or accumulations of lymphocytes are seen in the trachea and bronchi. A lymphofollicular reaction is a marked feature in the respiratory tract (Kerr and Olson, 1967).

Specific diagnosis can be established by isolating *M. gallisepticum*, but this is a difficult technique and negative results are unreliable. Serological procedures such as haemagglutination inhibition test, plate and tube agglutination tests using serum, and the rapid slide agglutination test using whole blood, are also useful in diagnosis. The rapid slide agglutination test using stained antigen is a simple yet useful test that can be used in the field for the diagnosis of mycoplasmosis. Characteristic histopathological changes can be demonstrated in the mucosa of the turbinates and trachea.

Ando, Matsui, Sato, Yoshida, Kato, and Kuniyasu (1965) described in detail the tests that may be used to diagnose this infection, including those given above. The whole blood plate test appears to be the most useful test in the field as it can be performed with a pullorum disease control programme. They also discussed in detail the method of manufacture of the antigen and the procedures to be followed in carrying out the test. If the antigen is preserved by formalin it will cause the loss of haemagglutinating activity of the antigen (Kuniyasu and Ando, 1966).

The pathogenic *Mycoplasmata* will agglutinate chicken erythrocytes, whereas the non-pathogenic *Mycoplasmata* will not. The serum plate test is conducted routinely and periodically in certain farms in Australia to detect reactors.

Epizootiology.—The disease, first reported in the U.S.A., is now a world-wide problem causing heavy losses in the broiler industry. It is primarily spread by egg transmission (vertical transmission) and a small proportion of infected pullets transmit the organism to the eggs. This usually occurs from the eighth to the

fifteenth week after experimental infection (Roberts and McDaniel, 1967). Usually less than 1 in 50 eggs laid by infected hens carry the infection. Thus, the disease is spread from flock to flock by vertical transmission, and within a flock by horizontal transmission. Though infection takes place from one bird to another within a flock by contact it is rarely transmitted from one flock to another by this means.

Management plays an important part in precipitating and aggravating the disease. Clinical signs may not become evident in carriers unless stress factors appear. In the presence of these factors or other agents listed under aetiology the disease will cause considerable economic losses.

M. gallisepticum does not live long outside the bird. Usually they are destroyed outside the host within 2 weeks and very rarely do they live longer than one month in a poultry yard. However, they may live up to 119 days in the mucosa of the turbinates of the infected birds and in the trachea of vaccinated birds up to 436 days.

Prevention and Control.—Any defects in hygienic conditions have to be remedied and if a few birds are affected they must be culled. If most of the birds are affected, depopulation is the best procedure. Restocking, preferably with day-old birds from a disease-free flock, should be done a month after thorough disinfection of the premises.

Experimental results indicate that vaccinating 5-week-old chickens with virulent strains of *M. gallisepticum* and thereby inducing early infection, could be useful in preventing egg transmission and consequently producing chickens free of this organism (Fabricant and Levine, 1963). Control may also be affected by vaccinating 1–14-day-old chicks with a live vaccine which will reduce egg transmission. However, as the vaccinated birds may harbour *M. gallisepticum* in the trachea for up to 436 days after exposure, vaccination is not a reliable means of eliminating egg transmission. Chute, Cuozzo, Stauffer, and McDonald (1965), describe methods for the experimental production of *Mycoplasma*-free chickens. Birds may be treated with tylosin, spiramycin, or erythromycin, or eggs may be dipped in a solution of these antibiotics. As none of these methods is entirely satisfactory, eradication of infection is desirable in every flock especially if they are breeders.

Eradication of Infection.—Osbaldiston and Wise (1967b) discussed the various methods available for the eradication of infection. There are two broad approaches to eradication, depending on whether or not drug therapy is employed.

ERADICATION WITHOUT DRUG THERAPY.—

1. *Incubation of Eggs in Small Batches.*—As the rate of transmission via the egg is low (usually less than 1 in 50 eggs laid by a carrier bird is infected), eggs are incubated in batches of 50 or less. Birds of each batch are then blood tested at approximately 10 weeks of age to detect the presence of infection. If any birds in a batch are positive, the batch is discarded. This method is useful if the rate of egg transmission is low but it is not often practicable.

2. *Controlled Infection.*—In this procedure the birds are infected during the growing period with the expectation that the worst effects of the infection will be overcome and that the birds will be immune to some degree during the egg-laying period. A disadvantage of this method is that birds protected in this way remain carriers, and in the presence of stress factors may develop clinical signs and excrete the organisms into the eggs.

3. *Force Moulting.*—If breeders are infected they are force moulted and brought back into a second session of lay. The older birds are less likely to lay infected eggs.

In methods described under (2) and (3), attempts are made to reduce the number of infected eggs laid so that method 1 can be used to establish a *Mycoplasma*-free flock.

ERADICATION WITH USE OF DRUGS.—

1. *Eradication with Use of Drugs and Other Measures.*—As tylosin, spiramycin, erythromycin, tetracyclines, and streptomycin have a static or a lethal action on *Mycoplasma*, attempts are made to reduce the extent of infection by using these drugs. Tylosin is the most effective drug and is widely used for the purpose (Gupta and Nayak, 1967). As a rule, repeated injections of any of these drugs given at intervals of 2–4 weeks, or more frequently, are more effective than a single injection. Breeders are treated at monthly intervals prophylactically, to reduce the chances of existing infection becoming active and thus reduce egg transmission. Eggs before incubation are treated either by dipping warm eggs in a cold antibiotic solution or by dipping eggs at room temperature in an antibiotic solution at the same temperature. The relative merits of the two methods vary but have not been clearly evaluated. The eggs are usually dipped in a solution of erythromycin containing 800 parts per million for 25–30 minutes or in tylosin solution for 15 minutes. The eggs laid by treated birds or those dipped in antibiotic solution are incubated in batches and the chickens that hatch out are tested at 10 weeks, and are taken into a breeding programme if the whole batch is negative for infection.

2. *Eradication with Drug Therapy Alone.*—This may be done either during the first month of life or during the incubation of the eggs.

a. Eradication of *Mycoplasmata* from chickens during the first month of life. In the technique advocated by Peterson (1966) in the U.S.A., attempts are made to eradicate infection by a series of injections of tylosin tartrate in corn oil, starting on the day-old chick and repeating at 5-day intervals. The birds are treated simultaneously by inclusion of chlortetracycline 300 g. per ton and terepthalic acid 0·4 per cent in the food for 25 days. In this procedure, an attempt is made to destroy all embryo-passed *M. gallisepticum* infection within the treated chicks by the medical treatment.

b. Eradication of *Mycoplasma* during the incubation of the eggs. This method is widely practised in Holland and consists of injecting 3 mg. of tylosin tartrate in 0·1 ml. of the vehicle into the yolk-sac or air-space of the egg at about the tenth day of incubation, so that all the organisms in the eggs are destroyed. This procedure may impair hatchability.

Treatment.—Unless *M. gallisepticum* has become resistant to antibiotics, the tetracycline group (Aureomycin and Terramycin) of drugs is useful in treatment, if given continuously for over a week as soon as the disease is seen in the flock. They are ineffective, however, when the lesions are fully established. The dosage recommended is 100–400 g. per ton of feed. They may also be given in drinking-water. Certain nitrofurans especially furazolidone, though useful, are expensive and may not be economical. Dipping of eggs in tylosin solution for 15 minutes appears to control spread of infection through eggs. Tylosin (Tylan, Eli Lilly), and spiramycin (Rovamycin, M & B) are effective in the treatment of

mycoplasmosis at the dosage rate of 100–200 mg. per kg. s/c. (Inglis and Cook, 1964). Tylosin appears to be the drug of choice; the dosage usually used for adult fowls is 25 mg. per kg. s/c. It is claimed that desoxycorticosterone trimethyl acetate, 20 mg. per bird, is also effective if given within 14 days of contacting the infection.

In an experimental study, Olesiuk, van Roekel, and Chandiramani (1965) found that tylosin tartrate at the dosage rate of 5 g. per gallon of drinking-water for 5–10 days or subcutaneously at the rate of 12·5 mg. per lb. body-weight was highly effective in improving weight gains and preventing or reducing the clinical signs and lesions.

Raines and Porter (1963) found that nihydrazone in concentrations of 0·011 per cent in mash appeared to have a good effect in reducing carcase condemnations and increasing weight-gains in cases which were complicated by infection with *E. coli.*

Infectious Synovitis
(*Mycoplasma synoviae Infection*)

This is an infection of young chickens, especially broilers, and turkeys, characterized by low mortality, inflammation of the synovial membranes of the leg joints, and hepatitis in the acute cases.

Aetiology.—The disease is caused by *Mycoplasma* spp. chiefly *Mycoplasma synoviae* and *Mycoplasma gallisepticum* (Olson, Kerr, and Campbell, 1964). These can be isolated by inoculating the suspected material into the yolk-sac of 5–7-day-old chick embryos. The embryos die in 4–10 days. The disease is spread by direct contact and via the egg as in respiratory mycoplasmosis.

According to Olson and Kerr (1966), infectious synovitis of chickens can be caused by any one of three separate agents, namely *Mycoplasma synoviae*, *M. gallisepticum*, and a viral agent closely related to the pox group of viruses.

Clinical Signs.—The incubation period varies from 24 to 80 days. Infection usually occurs at 4–12 weeks of age and is characterized by retarded growth, emaciation, pale comb, lameness, and distended joints of legs and wings. The hock joints and foot pads become swollen. The exudate is at first mucoid and later becomes creamy, caseous, and orange in colour. The faeces may be coloured green and blisters may be seen on the breast. If chicks are infected early in life there is septicaemia. In the early septicaemic stage, the liver is enlarged and the kidneys become swollen. Morbidity may vary from 5 to 100 per cent while the mortality rarely exceeds 5 per cent. This disease is more responsible for the down-grading of carcases than any other, and the texture of the carcase and the flavour of meat are greatly impaired.

Post-mortem Lesions.—In the early stages, there is a mucoid exudate in the affected keel, bursae, joints, and tendon sheaths, especially those of the hock and foot; later this becomes caseous. The surface of the affected joints becomes yellow or orange coloured. There is hepatitis and nephritis.

Diagnosis.—This is made by isolating and identifying the causative organism in 5–7-day-old chick embryos, when mortality occurs in the inoculated embryos

by about the eighth day, producing oedema and numerous haemorrhages on the skin. The disease can be reproduced experimentally in birds by inoculating via the foot pad. The rapid plate test using whole blood is not very useful for diagnosing the disease in the field.

Treatment, Prevention, and Control.—When the disease is localized treatment is of no value. Chlortetracycline (Aureomycin) given at the dosage rate of 50 to 100 g. per ton will induce satisfactory control. When infection has occurred 200 g. per ton or more has to be used. Often this line of treatment is not economical and is not entirely satisfactory. Furaltadone in water at the dosage rate of 1 g. per gallon of water is said to control the infection. Tylosin and spiramycin adipate are also useful as in respiratory mycoplasmosis.

The disease can be controlled and eradicated by using the methods described under respiratory mycoplasmosis. Unlike *M. gallisepticum*, *M. synoviae* may survive for longer periods outside the host. Principles of good hygiene and reduction of factors that cause stress will help to a considerable extent to reduce the incidence of clinical cases.

INFECTIOUS TENOSYNOVITIS

An infectious tenosynovitis has been described recently in broilers (Dalton and Henry, 1967). This is characterized by fibrous thickening of the tendons above the hock joint, and in some the metatarsal bones get twisted. The growth-rate is retarded and some degree of lameness is seen. This entity apparently differs from synovitis and is caused by an organism closely resembling *Corynebacterium xerosis*.

COLI BACILLOSIS
(*E. coli Septicaemia; E. coli Infection*)

Certain types of *Escherichia coli* can cause infection in fowls, particularly broilers, if their resistance is lowered by unfavourable conditions. However, most strains of *E. coli* are not pathogenic. They usually produce a pericarditis, and in co-operation with other agents, chiefly *Mycoplasma gallisepticum* or the virus of infectious bronchitis, they may produce complicated forms of C.R.D. or other respiratory infections.

Cause.—As the name indicates the disease is caused by certain strains of *E. coli* in the presence of certain debilitating factors. In a recent study it was found that serogroup O_2 was responsible for more than half the outbreaks (Harry, 1964). However, the presence of this O group of *E. coli* usually produces septicaemia (Harry and Hemsley, 1965). This disease is responsible for great economic losses in the broiler industry in Great Britain.

Clinical Signs and Course of the Disease.—Usually broilers of about 5 weeks of age are affected. However, birds of any age may be affected provided their resistance is lowered by predisposing factors. Chickens below 10 days are particularly susceptible if their resistance has been lowered. The affected birds are dejected, their food and water intake is lowered, and they show pasty backs. Infection with this organism may also produce salpingitis, salpingoperitonitis, or peritonitis. In

rare cases, a cholera-like disease producing nearly 100 per cent mortality may be seen in growing chicks (Nagi and Khanna, 1967).

Post-mortem Lesions and Diagnosis.—The most constant finding in broilers is pericarditis where the pericardium is thickened and the heart muscle is surrounded by coarse thick fibrinogelatinous material (*Fig.* 11). The air sacs may be thickened. Chicks may show enlarged livers with necrotic foci. The liver and the peritoneum may be covered with gelatinous material. In acute cases, the liver is swollen and congested and shows blotchy patches.

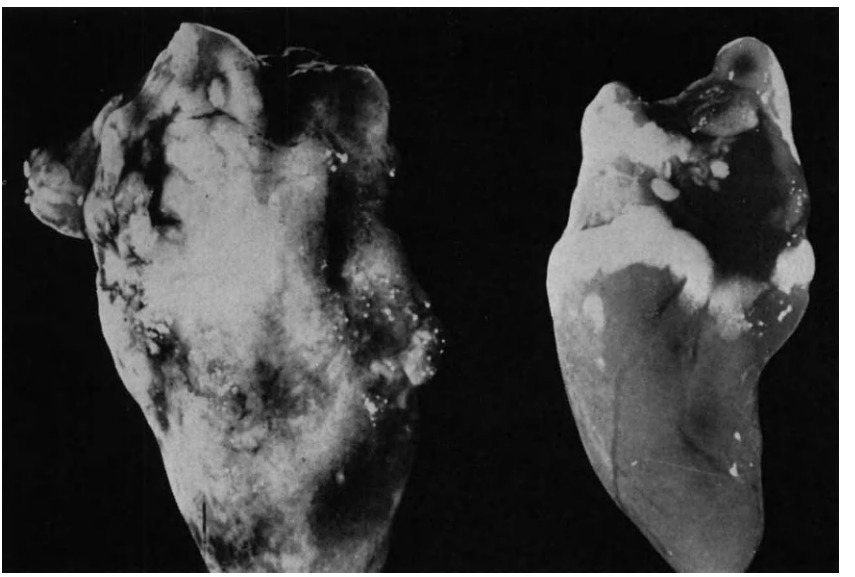

Fig. 11.—The heart of a chicken infected with *E. coli*, showing fibrinous pericarditis. On the right is a normal heart for comparison.

Diagnosis requires bacteriological examination, isolation of *E. coli*, and the demonstration of its ability to reproduce the disease in susceptible birds. The lesions described above, especially fibrinous pericarditis, are highly suggestive of the disease.

Prevention and Control.—This is largely a matter of good hygiene as in salmonelloses and paracolon infection, and the control of predisposing respiratory infections. Fumigation of litter with methyl bromide and formaldehyde is effective in destroying *E. coli* in the litter. Methyl bromide at the rate of 100 g. per c. m. was found to be nearly 100 per cent effective in destroying *E. coli* up to a depth of 20 in. (Samberg and Baroutchieva, 1967). As *E. coli* can survive in dust, particularly if the humidity is low, total elimination of infection is a difficult matter.

Treatment.—The tetracyclines (Aureomycin, Terramycin) and furazolidone are effective against most strains of *E. coli*. These drugs are usually given in food. The tetracyclines are given at levels of 100 g. per ton and furazolidone at 0·04 per cent. Streptomycin may be effective but should not be used as *E. coli* develops

resistance very rapidly to this antibiotic. Nihydrazone at a concentration of 0·011 per cent in food is also said to be effective.

Due to the increasing use of tetracyclines as a food additive, it is possible that the *E. coli* responsible for particular outbreaks are resistant to them. Therefore, it is best when an outbreak of *E. coli* infection is suspected to get this confirmed by a diagnostic laboratory and to obtain their advice as to the susceptibility of the particular strain of *E. coli* to antibiotics.

NECROTIC ENTERITIS IN THE FOWL

This is a disease usually seen in birds between 6 and 7 weeks of age, characterized by loss of appetite, and occasionally blood-stained faeces. This condition appears to be common in broilers in Australia. In the more chronic cases, there is gradual loss of weight and the faeces become watery. Occasionally, some birds make a spontaneous recovery. The disease has been described by Parish (1961) and is caused by a *Clostridium*. The various clostridial infections in fowls have been discussed recently by Saunders and Bickford (1965).

Cause.—The disease is caused by a strain of *Clostridium*, probably *Clostridium perfringens* (*Cl. welchii* type F) which elaborates alpha, beta, and gamma toxins. The spores resist 100° C. for 2 hours. The toxins have a dermonecrotic activity and are responsible for karyolysis of the nuclei. It appears that some dysfunction of the alimentary tract is a necessary predisposing cause of infection. Intestinal stasis, intestinal distention, certain types of feeds, coccidiosis, and other factors may predispose the birds to infection. Further, the capacity of the organism to produce toxins may vary considerably under different conditions. This type has caused haemorrhagic enteritis of man in Germany.

Clinical Signs and Course of the Disease.—The disease may appear in acute form or chronic form. In the acute form, there is haemorrhagic necrosis of the small intestine especially the ileum, congestion and degeneration of the liver, and haemorrhagic necrosis of the terminal digits.

In the chronic form of the disease the body-weight is reduced considerably with degeneration and contraction of the liver and spleen. However, the gall-bladder is markedly distended. There is a thick, dry, yellowish-brown necrotic enteritis involving the lower third of the intestine (Nairn and Banford, 1967).

Histologically the intestines show areas of necrosis, usually sharply demarcated from the normal tissues.

Prevention and Treatment.—No work has been done on these aspects of the disease. However, good sanitation with thorough disinfection and cleaning of the infected premises, removal of all infected litter, and the feeding of antibiotics will probably be helpful. Treatment with zinc bacitracin or penicillin at the dosage rate of 200 g. per ton of feed for 24 hours gives a satisfactory clinical response.

BOTULISM

(Limber Neck; Bulbar Paralysis; Western Duck Sickness)

This is a type of food poisoning caused by the ingestion of spoilt food containing toxins of *Clostridium botulinum*. It affects mammals, man, and a variety of animals.

The disease is caused by the toxins. Wild water-fowl and other birds also die in large numbers due to this disease. This is the most powerful toxin known, being much more powerful than cobra venom. The minimal lethal dose for a guinea pig is 0·00012 mg. per kg. subcutaneously.

There are many sub-types of *Cl. botulinum* but the types that cause disease most frequently in domestic birds are types A and C. Type A predominates. The organisms produce the toxin under anaerobic conditions in an alkaline medium. Hence, canned vegetables are much more dangerous than canned fruit. Maggots, especially the larval forms of *Lucilia caesar*, can ingest the toxins and thus prove fatal to the chickens, which may ingest them.

Clinical Signs and Course of the Disease.—The first signs are seen within a few hours of the ingestion of the toxin and are characterized by progressive flaccid paralysis where the legs, wings, and neck become limp, and the bird is unable to lift its head. Feathers come out easily and eventually complete paralysis and death ensue.

Mild cases may only show loss of appetite, weakness, and difficulty in swallowing. Large numbers of wild ducks used to die of this condition in the United States.

Diagnosis.—This is usually based on the characteristic signs of flaccid paralysis of the neck and the ability to pull out the feathers easily. A definite laboratory diagnosis can be established by inoculating intraperitoneally two groups of mice, one immunized and the other not immunized against the toxin with 1 ml. of serum from each of the dying birds. If the test is positive, mice immunized against the toxin will remain normal while the others will show symptoms or die.

Isolation of *Cl. botulinum* alone is not regarded as sufficient evidence as these organisms are frequently found in the soil and alimentary canal of birds.

Prevention and Control.—This is done by avoiding giving the birds any meat scraps or decomposed or spoilt vegetables likely to contain the toxins. In the case of wild ducks, the most successful method has been to keep them away from the areas likely to contain the toxins.

Treatment.—Severely affected birds will die. Antitoxin can be used but is too expensive to be of any practical value. Mildly affected birds may be treated by giving wet bran mash once a day for 2–3 days with 1 lb. of epsom salts per 75 birds or they may be given epsom salts in water at the rate of 1 lb. per 100 birds.

INFECTED YOLK-SACS
(Mushy Chick Disease; Omphalitis)

This is usually caused by infection of the yolk-sac with a variety of organisms, chiefly those found in the environment. They are not specific disease producers. They invade the yolk-sac either during the last few days of incubation or after hatching, and may cause considerable mortality in the first 10 days of life or be responsible for the dead in shell condition. This is a common disease in turkeys because the nesting habits of this species result in gross bacterial contamination of egg-shells.

The condition is usually one of bad management and is seen only when the eggs and incubator are very highly contaminated and when conditions of high humidity prevail in the incubator. The bacteria penetrate the shell and invade the yolk-sacs. *Escherichia coli* serotype 6103 may cause retention of caseous yolk-sacs and reduced weight-gains.

Most yolk-sac infections result in death from septicaemia, and the invasion of the whole carcase is the cause of rapid post-mortem decomposition, and hence the name mushy chick disease.

Clinical Signs and Course of the Disease.—The disease is usually seen in chickens less than 10 days old. When infection occurs during early incubation it may be responsible for many dead in shell chicks, or more frequently the bacteria may invade the yolk-sacs soon after birth, from the gut. In this case, the chickens will be weak and show retarded growth and high mortality. The signs are not specific. In some cases, infection will occur through the imperfectly healed navel. The highest mortality is seen from the first to the fifth day but it may extend to the tenth day. The mortality does not usually exceed 10 per cent.

Post-mortem Lesions and Diagnosis.—The carcase appears to be smaller than normal and the muscles are redder than usual. The yolk-sac is distended and often contains a foul-smelling yolk. The blood-vessels are either distended or degenerated. There may be, in addition, pericarditis and perihepatitis.

These changes can also be produced by specific pathogens such as *Salmonella pullorum*.

Prevention and Control.—This is largely a matter of good hygiene. All eggs should be dipped in an approved disinfectant and regular fumigation should be practised.

Treatment.—Treatment is not satisfactory because the drugs taken in by the chicks do not reach the yolk-sac in sufficient quantities to destroy or arrest the growth of the bacteria in the yolk-sac. Further, even if treatment is effective it is not economical as the affected chicks remain stunted and do not become productive.

AVIAN LISTERIOSIS

This is usually a chronic or subacute disease of the domestic fowl, especially young birds, associated with bad management. It may produce high mortality in conjunction with concomitant infection. A characteristic lesion of the disease is massive necrosis of the heart muscle. Nervous signs are seldom seen.

Cause.—The disease is caused by *Listeria monocytogenes*, a small motile Gram-positive rod which exhibits a characteristic tumbling movement. The colonies produce β haemolysis after 48 hours in blood-agar. Primary isolation of the organism is sometimes difficult.

Clinical Signs.—These are variable. Nervous signs, frequently seen in this infection in mammals are usually not seen in the domestic fowl though they may be common in geese (Bandaranayake, 1953). The clinical signs are variable and so is

the mortality which may reach 30–50 per cent in chickens below 3 months in the presence of concomitant infection. Recent evidence indicates that certain toxins may enhance the pathogenicity of *Listeria monocytogenes.*

Post-mortem Lesions.—These consist of massive necrosis of heart muscle, pericarditis with pericardial effusion, and foci of necrosis in the liver, which is often enlarged and congested. There is a monocytosis in many cases.

Diagnosis.—This cannot be based on clinical signs and history as they are vague. However, massive necrotic areas in the heart muscle are suggestive of the disease. A specific diagnosis can be established by isolating the organism from the heart blood in blood agar plates. The organisms produce conjunctivitis in guinea pigs and mononucleosis in rabbits.

Treatment and Prevention.—Chlortetracycline (Aureomycin) appears to be the drug of choice. This may be given in food or drinking-water as advised by the manufacturers. The standard dosage is 100 g. per ton of feed. Hygiene should be improved, overcrowding avoided, the litter changed, and the premises disinfected.

AVIAN VIBRIONIC HEPATITIS

This is an acute or subacute disease more commonly seen in mature birds, characterized by hepatitis. Young birds are also susceptible. The disease was first observed in 1955 and later more extensive outbreaks in the United States were reported by Hofstad, McGhee, and Bennet (1958). Bauditz (1967) has reviewed the disease.

Cause.—It is caused by a vibrio which produces yolk-sac congestion and necrosis of the liver with enlargement of the spleen of 10–14-day-old embryos. It is a Gram-negative, pleomorphic, motile organism like other vibrios. The house sparrow appears to be an important carrier of vibrios.

Clinical Signs.—Incubation period varies from 2 to 15 days and mortality from 5 to 15 per cent. A loss of egg production up to 25 per cent is commonly observed. The affected birds are emaciated and listless, and their combs are pale.

Post-mortem Lesions.—The characteristic lesions are in the liver, which may be firm and have irregular diffuse greyish areas on the surface, or it may be swollen and congested with necrotic areas or haemorrhages giving it a mottled appearance. The liver tissue is spongy and easily broken. Histologically, the liver shows lymphocytic and heterophilic infiltration around the veins of the hepatic triad, fatty degeneration of liver cells, and focal necrosis.

Diagnosis.—This is based on isolating the organism from the heart blood or liver of the affected bird in 5–7-day-old embryonating chicken eggs. Bacitracin and polymyxin B may be added to prevent contamination. The organisms may be isolated from the bile of the affected birds on brilliant green blood-agar or blood-agar (Hagan, 1964). A stained caecal smear may also be useful in diagnosing vibrionic hepatitis.

Treatment and Prevention.—Furazolidone appears to be the drug of choice; 200–400 g. per ton of feed for several weeks appears to be satisfactory. Some of the tetracyclines have been found to be useful but do not appear to be as effective as furazolidone. General hygienic measures have to be improved to reduce the incidence of infection. No specific methods are available for the control of this infection.

SPIROCHAETOSIS

This is an acute infectious disease of fowls characterized by fever, locomotor disturbance, and progressive paralysis. It is usually seen in tropical or sub-tropical countries where *Argas persicus*, the fowl tick, is found.

Cause.—The disease is caused by *Borrelia anserina*, a spirochaete, and it is transmitted by all stages of the fowl tick *Argas persicus*. Transovarian transmission also occurs. Transmission may also occur through infected biting flies, insects, or mosquitoes. Birds may also be infected by ingesting infected ticks, their eggs, or contaminated food. An infected tick may remain infective for up to 3 years if the environmental temperature is sufficiently high, i.e., 95° F. However, at temperatures below 69° F. they soon disappear from the tick.

Clinical Signs.—The incubation period is 5–9 days and the average duration of the disease is about 5 days. The chronic form of the disease may last about 21 days.

In the early stages, the temperature is elevated to about 110° F. and there is swelling of the feet and claws, which are turned upward. The comb becomes pale and the bird listless, then greenish diarrhoea sets in. Later, anaemia becomes pronounced and paralysis is usually seen before death. In the chronic cases, the course is prolonged and emaciation, anaemia, and stupor become prominent signs. The temperature drops to subnormal a day or two before death.

Post-mortem Lesions.—The spleen and liver are markedly enlarged. The latter organ is pale and is studded with numerous caseous foci. Fibrinous pericarditis and myocarditis are present. Anaemia and enteritis are very commonly seen. There is marked leucocytosis with absolute lymphocytosis (Soliman, Ahmed, Amrousi, and Moustafa, 1966).

Diagnosis.—At the height of the disease the organisms can be demonstrated in peripheral blood smears stained with Giemsa. In chronic cases, they are not seen in the blood but may be demonstrated in the pericardial fluid or the vascular papillae of the wings. Agglutination and complement fixation tests may also be used in the diagnosis. In sections of the spleen and liver the organisms may be demonstrated by Levaditi's stain.

Prevention.—This is based mainly on tick control measures. Eradication of the vector *Argas persicus* should be aimed at. The methods used to eradicate *A. persicus* are described in the section on this tick. Recent evidence indicates that certain biting insects, chiefly mosquitoes, may transmit this infection. Rao and Gupta (1960) have developed a chick embryo freeze-dried vaccine for immunizing chickens.

The egg vaccine produced by Hart (1963) induced immunity which lasted at least for 66–68 weeks when the recommended dose of 1 ml. was given intramuscularly.

Treatment.—The specific drugs are Atoxyl (sodium arsanilate), the arsenobenzol group of drugs including Soamin and Novarsenobillon, penicillin, and tetracyclines. Oxytetracycline in a single dose of 40 mg. per kg. in oily or watery vehicles subcutaneously has been found to be effective under field conditions (Miklovichne and Kemenes, 1963). A single dose of penicillin at the rate of 4000 units per lb. body-weight given at the height of the disease was found to be fully curative (Rao, Thakral, and Dhanda, 1954). In practice, however, up to 100,000 units of penicillin per bird may be used with excellent results.

STREPTOCOCCOSIS
(*Apoplectiform Septicaemia*)

The domestic fowl may occasionally be infected with *Streptococci.* Newton, Connole, and Ranby (1962) described a rather severe outbreak in fowls in Australia and Peckham (1966) described another in Ithaca, New York, which were caused by *Streptococcus zooepidemicus* (avian strain).

In the early stages of the infection there is fever exceeding 110° F. There may then be diarrhoea with yellowish faeces and the affected birds become markedly somnolent. Mortality may reach 55 per cent but some of the infected birds may recover. Death can occur in a few days in the septicaemic form while in others the disease may last about 2 weeks. Post-mortem examination usually reveals extensive fibrinous peritonitis, perihepatitis, pericarditis, salpingitis, and joint abscesses. The liver is swollen and has rounded borders. The ovary becomes irregular and may often contain solid material. In the septicaemic form, the spleen is swollen, the liver is friable, and there is a sanguineous transudate in the body cavity, trachea, and mouth.

Treatment is not very satisfactory but best results are obtained by giving furazolidone in mash at a concentration of 0·03 per cent for 3–6 days. Sulphonamides are useful.

This disease may be more common than is reported in the literature.

STAPHYLOCOCCOSIS

Staphylococcosis is caused most often by *S. aureus*, usually possessing alpha-haemolysin and delta-lysin. A majority of these appear to be resistant to penicillin and tetracyclines (Harry, 1967). Staphylococcal infection in the domestic fowl may cause septicaemia, purulent arthritis of the tibiometatarsal joint, vesicular dermatitis, sternal bursitis, yolk-sac infection, wing gangrene, and spondylitis resulting in spinal cord compression, paresis, and paralysis in chickens between 7 weeks and 4 months. *Staphylococcus pyogenes* can also cause spondylitis (Carnaghan, 1966).

CHAPTER IV

FUNGAL DISEASES

ASPERGILLOSIS
(*Brooder Pneumonia*)

THIS is caused by *Aspergillus fumigatus* and is often associated with bad management. It is seen when mouldy feed or litter is used or when there is gross overcrowding. It is becoming more common due to the suppression of bacterial infections with antibiotics. It is widely prevalent in the tropical regions where the high humidity and temperature favour growth and sporulation. Thus, in the island of Barbados in the West Indies, fresh or new bagasse which is being used as litter material was found to be a good medium for the growth and sporulation of *A. fumigatus*.

In chicks, the disease usually takes an acute form and in very severe outbreaks may kill about 50 per cent of the birds. It also affects the adults, and usually one or a few birds are affected at a time. The disease is also seen in turkeys, ducks, and captive and wild birds.

Clinical Signs and Course of the Disease.—The symptoms are not very characteristic and may include difficult respirations and gasping movements. Eyes may be inflamed and there may be cheesy deposits in the conjunctival sacs. Usually 5–10 per cent of the chicks will be affected but in severe outbreaks 50 per cent of the flock may die. In adults, it is a chronic disease with no characteristic signs and is usually diagnosed at autopsy. Under conditions of high humidity and moderate temperature there may be serious outbreaks.

Post-mortem Lesions and Diagnosis.—The lesions are fairly characteristic and are usually seen in the lungs and air sacs only. They appear as cheesy white nodules or concave disks, the former resemble those of pullorum disease but may be more extensive. Sometimes the more extensive lesions have a greenish-yellow colour. The air sacs and the syrinx may also be involved. In the early stages, there is congestion of the lungs which may have a sodden appearance. At times, the eyes are affected, producing plaques and cheesy deposits. Often, only one eye is affected. Diagnosis is made from the lesions, which should show the fungal hyphae, and from which the fungus itself can be isolated. The disease may be diagnosed in live birds by the collodion agglutination test (Giessler, 1963).

Prevention and Control.—The fungus thrives in mouldy litter and food and these must be removed and burnt. Overcrowding must be avoided. The premises may be sterilized with a blow lamp using it only on non-inflammable structures. Others may be scrubbed with 0·5 per cent copper sulphate solution. Fumigating with 5 ml. of 40 per cent formalin and 3 g. of potassium permanganate per cu.ft. for at least 30 minutes is considered a satisfactory procedure.

There is no satisfactory treatment for the condition. All affected birds must be destroyed, as the recovered birds show retarded growth and unthriftiness. Trichonat (trichomycin) given at the dosage rate of 40 I.U. per ml. in drinking-water is claimed to be curative (Giessler, 1963).

THRUSH

(Moniliasis of the Upper Digestive Tract; Mycosis of the Upper Digestive Tract; Sour Crop)

This is a chronic mycosis of the upper digestive tract characterized most often by a sour crop and whitish necrotic membranes in the crop and oesophagus.

Cause.—The disease is caused by *Candida albicans* (*Monilia albicans*) though other species of fungi may also be involved, especially in the tropics. Younger birds are more prone to the disease. Its incidence appears to be increasing due to the use of broad-spectrum antibiotics. The affected birds are depressed, stunted, have a poor appetite, and extrude a slimy foul-smelling sour discharge from the mouth.

The disease is usually seen in the presence of other debilitating conditions and is diagnosed at post-mortem examination.

Post-mortem Lesions and Diagnosis.—The crop is often distended and presents a turkish towel appearance with soft whitish raised ulcers which tend to form a false membrane. Necrotic lesions may also be seen in the mouth, oesophagus, and proventriculus.

Diagnosis is made on the basis of these lesions, in which can be seen the fungal hyphae, and by isolating the organisms. The disease has to be distinguished from trichomoniasis and avitaminosis A which produce similar lesions. In trichomoniasis the protozoan parasites can be easily demonstrated and in avitaminosis A there will be coryza-like symptoms and cheesy pustules in the oesophagus.

Treatment.—The addition of one teaspoon of copper sulphate to 2 gallons of water in non-metallic containers and giving only this water for one week is recommended as treatment. Nystatin, an antifungal antibiotic, at a level of 200,000–800,000 units per kg. of diet fed for 10 days appears to lower the incidence and reduce the severity of moniliasis.

Amphotericin B appears to be 4–5 times as effective as nystatin as a preventive when given in mash (Kahn and Weisblatt, 1963). The effective doses used were 14–55 mg. per kg. of the basal ration.

AFLATOXICOSIS

(Mycotoxicosis; Groundnut Meal Toxicity)

History.—The investigation into the death of a large number of turkey poults in England in 1960 and subsequent research led to the discovery of aflatoxin. Initially the disease was designated turkey X disease. However, soon it was recognized that the disease could affect ducklings, chickens, pigs, and calves. Once it was determined that the disease was caused by a toxin produced by *Aspergillus flavus*

and the toxin identified and named aflatoxin after the fungus from which it was derived, the disease was called aflatoxicosis.

Cause.—The disease is caused by aflatoxin produced by certain strains of *Aspergillus flavus*. There are several closely related compounds, namely aflatoxin B_1, B_2, G_1, G_2, M_1, M_2, B_2a and G_2a. Of these, aflatoxin B_1 is the most toxic and widely present toxin (Moir, 1967). The 7-day LD_{50} of aflatoxin B_1 for khaki campbell ducklings weighing an average of 50 g. is 18·2 μg.

More toxin is produced under conditions of high humidity and temperature.

Ducklings and turkey poults are more susceptible than chickens, in which species only young birds are generally affected while the adults are comparatively resistant. It was found that the forms of mycotoxicosis that occurred in Britain in 1959 and 1960 were due to aflatoxin contained in groundnut meal imported from Brazil, certain parts of Africa, and India. The toxicity of groundnut meals from Brazil was considered to be the highest and those from India the lowest.

Chaudhary and Manjrekar (1967) found that 13 per cent of 150 samples of groundnut cake examined at the Bombay docks harboured *A. flavus*, of which probably only two strains were toxigenic. Twenty-six per cent of the damaged and discoloured groundnuts harboured the fungus whereas white nuts were not infected. Further information on mycotoxicosis of poultry is given by Asplin and Carnaghan (1961), Forgacs, Koch, Carll, and White-Stevens (1962), and Forgacs and Carll (1962).

The toxin produces proliferation of the bile-duct epithelium with degeneration of the parenchymal cells of the liver and in low continuous dosage acts as a liver carcinogen in rats.

Contamination of feed or the litter by any of the toxigenic strains of fungi can produce mycotoxicosis, the exact syndrome depending on the nature of the toxin and the species involved. It is possible that this is a common condition in the tropics as fungi multiply more rapidly under tropical conditions because a high temperature and high humidity favour fungal multiplication. In most instances, the toxin may only cause a retardation of growth and once the birds become resistant to the toxin, no ill effects may be noticed.

Clinical Signs and Course of the Disease.—The disease may take an acute or chronic course. At the commencement of an outbreak, the disease runs an acute course, but as it progresses, and the birds become resistant, it takes a chronic course.

The usual clinical signs are depression, diarrhoea, or more often dysentery, anorexia, marked anaemia, and eventually death. In acute cases, death may occur in 2–3 days, after feeding the toxic feed. None of the clinical signs are specific but the characteristic signs for each species occur about 2–4 days before death when the birds become dull, develop a staggering gait, and finally become recumbent. Turkey poults and ducklings frequently develop convulsions and die in opisthotonos.

Post-mortem Lesions and Diagnosis.—Post-mortem examination reveals multiple haemorrhages in the subcutaneous tissues and internal organs similar to those seen in vitamin K deficiency or toxicity due to sulphonamides. Therefore, mycotoxicosis has to be differentiated from these two conditions. However, two or more

of these conditions may exist simultaneously. In the chronic cases, there are discrete white pinpoint lesions in the liver. In ducklings, a marked feature is the proliferation of the bile-duct epithelium followed by nodular hyperplasia. Finally the liver becomes cirrhotic and hard.

Prevention, Treatment, and Control.—Care must be taken to store feed in dry conditions especially in the tropics. Moisture in the litter must be reduced. This is done by adding fresh dry litter or replacing the moist litter with dry litter and providing adequate ventilation. Since avitaminosis K is associated with certain forms of mycotoxicosis it is desirable to give adequate if not surplus quantities of the vitamin K. Agents that suppress fungal growth such as 8-hydroxyquinoline may be useful as feed additives to prevent fungal growth and production of toxins. The amount usually added is 500 parts per million of the mash.

The minimum concentration of aflatoxin permissible in human foodstuffs is 0·03 p.p.m. though man is much more resistant to the effects of aflatoxin. As ducklings and turkey poults are more susceptible to the toxins, any feedstuffs suspected to contain the toxins should not be used for feeding this group of birds up to the age of 12 weeks.

FAVUS

This is a skin mycosis of the domestic fowl seen usually on the comb and caused by *Trichophyton gallinae* (*Achorion gallinae*). The affected areas become white and the comb gives the appearance that it has been sprinkled with flour. The lesions may soon coalesce, become thick, and form a crust. The infection may spread to man.

The applications of preparations containing iodine, mercury, or formaldehyde are very beneficial.

The disease spreads very slowly and is seen more frequently under damp humid conditions. It is not common.

CHAPTER V

PARASITIC DISEASES

THESE may be caused by protozoan parasites, helminths, or arthropods which are mainly ectoparasites. Parasitism is a form of association between two organisms in which the parasite lives at the expense of the host. However, if the parasite is too injurious to the host, the life of the host is endangered and this in turn threatens the existence of the parasite. Thus, we find that in quite a number of parasitic diseases, there is a sort of balanced existence between the parasite and the host in which the injurious effects of the parasite are often not clearly manifested. Under these circumstances, disease becomes evident only when this balance is upset by large numbers of parasites, environmental factors such as poor nutrition, bad hygienic conditions such as overcrowding and intercurrent disease, or lowered resistance of the host.

Unlike bacterial or viral disease, the presence of a few parasites in some cases causes a state of immunity known as premunity. Thus, it is an advantage for a few parasites to remain in the host. Therefore, the demonstration of the parasite *per se* does not necessarily prove that the disease is caused by the parasite. This is particularly true of some protozoan parasites.

1. PROTOZOAN PARASITES

The important protozoan parasites of domesticated animals and birds (*Fig.* 12) have been described and listed by Levine (1961). The various protozoan parasites that may be seen in the blood of poultry are illustrated in *Fig.* 12.

COCCIDIOSIS

Cause.—Coccidiosis is one of the most important and widespread diseases of poultry which is caused by at least 8 species of coccidia of which the most important ones are *Eimeria tenella* and *Eimeria necatrix*. The increased knowledge of coccidiosis at present is largely due to the fundamental research on this problem conducted by Tyzzer (Tyzzer, 1929, 1932; Tyzzer, Theiler, and Jones, 1932). The various aspects of coccidiosis with special reference to immunity have been discussed by Horton-Smith and Long (1963). Edgar and Seibold (1964) have described *Eimeria mivati* from the domestic fowl in the United States and Canada. A new species of *Isospora* has been reported from the fowl in Russia (*Isospora gallinarum*). *E. tenella* causes caecal coccidiosis usually in young chicks and *E. necatrix* causes intestinal coccidiosis in more mature birds. These two are the most pathogenic and most important species of coccidia. The other important species of coccidia in order of pathogenicity are:—

E. brunetti: This species affects the posterior half of the small intestine, rectum, and cloaca. There is catarrhal enteritis and petechiae with blood-tinged exudate in the posterior part of the small intestine, and characteristic yellowish-white caseous material may be found in the lumen of the intestine and rectum. Development of immunity is subimmediate. The oocysts are egg-shaped and measure 23 $\times$ 20 μ.

E. maxima. This species affects chiefly the middle of the small intestines. The intestinal wall is thickened, there are exudations and flakes of blood, and the gametocytes are deep in the epithelium. Development of immunity is immediate but short lived. Oocysts are egg-shaped and measure 31 × 21 μ.

E. hagani. This species affects the anterior half of the small intestines and lesions appear as pin-head sized haemorrhages seen from the serous wall. The mucosae of

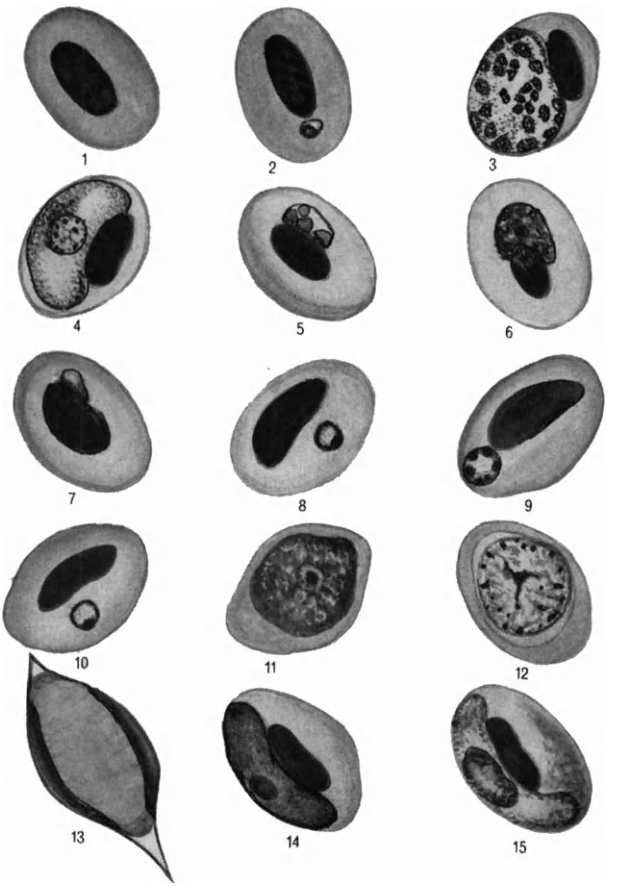

Fig. 12.—The various protozoan parasites that may be seen in the erythrocytes of some birds. 1. An uninfected erythrocyte of a bird. 2. A trophozoite of *Plasmodium gallinaceum.* 3. A schizont of *P. gallinaceum.* 4. A microgametocyte of *P. gallinaceum.* 5. A schizont of *P. juxtanucleare* (after Dhanapala). 6. A macrogametocyte of *P. juxtanucleare.* 7. A trophozoite of *P. juxtanucleare.* 8. A trophozoite of *Aegyptianella pullorum.* 9. A schizont of *A. pullorum.* 10. A young gametocyte of *Leucocytozoon caulleryi.* 11. A macrogametocyte of *L. caulleryi* free in plasma. 12. A microgametocyte of *L. caulleryi* in an erythrocyte where the nucleus has been pushed out. 13. A microgametocyte of *L. smithi* in the erythrocyte of a turkey (note parallel bar appearance). 14. A macrogametocyte of *Haemoproteus columbae* in an erythrocyte of a pigeon. 15. A microgametocyte of *H. columbae* in an erythrocyte of a pigeon (note the sausage-shape of the gametocyte of *H. columbae*). Numbers 2–10, 12 are infections in erythrocytes of the domestic fowl.

the affected portion show catarrhal inflammation. Oocysts are broadly ovoid and measure 19 × 18 μ. There is prompt development of immunity.

E. mivati. This species initially affects the anterior part of the small intestine and then it moves slowly down the gut. The lesions are rounded, the intestinal contents creamy and mucoid, and development of immunity is delayed. The mean measurements of the oocysts are 16 × 13 μ.

E. acervulina. This species generally affects the duodenum of birds which are 6 weeks or older. Hence, the infection is called duodenal coccidiosis and the lesions appear as numerous grey or whitish bands through the serous membrane. In acute cases, the duodenum is acutely inflamed and red in colour. This is a common poultry coccidium and is not very pathogenic. However, massive infections can cause death. This parasite causes a self-limiting infection. There are 4 generations of schizonts within 5 days after infection. No further stages are detectable in the intestinal cells unless there is reinfection (Vetterling and Doran, 1966). Immunity is delayed and the oocysts measure 18 × 15 μ.

At low levels of infection there are characteristic discrete white lesions and at high levels of infection the lesions coalesce and produce mucoid enteritis and anorexia. Pathogenicity appears to be higher in younger and in rapidly growing birds (Hein, 1968).

E. praecox and E. mitis. These species affect the anterior part of the small intestine and they are not very pathogenic. *E. mitis* oocysts measure 16 × 16 μ, are nearly spherical, and are the smallest poultry coccidian oocysts. *E. praecox* oocysts measure 21 × 17 μ. There is rapid development of immunity to *E. praecox* infection.

E. tenella and E. necatrix will be discussed in greater detail.

LIFE CYCLE (*Fig.* 13).—The life cycle of *E. tenella* described below is typical of most of the *Eimeria* of birds. Infection occurs by the ingestion of the sporulated oocysts. For sporulation to take place, oxygen, moderate moisture, and warmth are necessary and under ideal conditions this takes place within 48 hours, the minimum sporulation time being 18 hours. The time taken for sporulation depends on the species. The sporulated oocysts may, under suitable conditions, live up to 1 year or more. Excess of moisture or dryness are both unfavourable for sporulation and survival of the sporulated oocysts. When a non-immune bird ingests the sporulated oocyst the sporozoites come out, reach the epithelial cells of the glands of the caeca, being transported there by the macrophages, and lie below the nuclei to develop into the first generation of trophozoites which have a characteristic eosinophilic globule and are rounded. This eosinophilic globule is maintained up to the stage of the first generation of schizonts, after which it disappears. The first generation of trophozoites grow into the first generation of schizonts, which produce the first generation of sickle-shaped merozoites. Each first generation schizont produces about 900 merozoites. These merozoites come out of the original host cells and invade others to become the second generation of trophozoites and schizonts which do not have the eosinophilic globule. The second generation of schizonts cause the infected epithelial cell to become swollen and assume a migratory habit. The surrounding blood-vessels degenerate resulting in severe caecal haemorrhage. The haemorrhage is also accompanied by massive denudation of the caecal epithelium. These changes usually occur from the fourth to fifth day. This phase of reproduction is asexual.

After two repetitions of schizogony some of the third generation merozoites that enter the epithelial cells produce the sexual phases, the gametocytes which become differentiated into male and female gametocytes. The male gametocytes

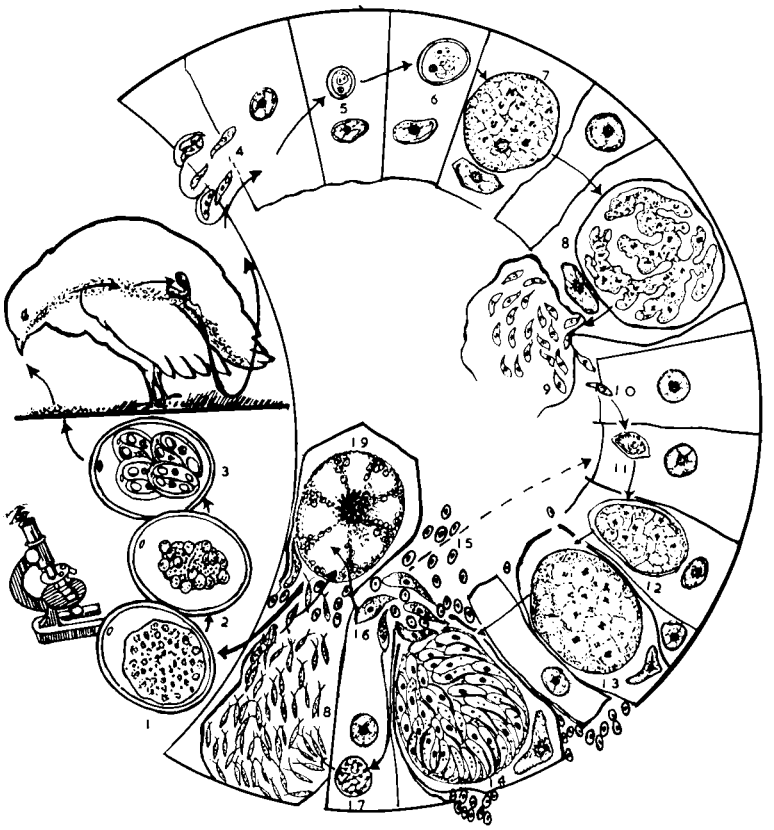

Fig. 13.—Development of *E. tenella* in the chicken (semi-diagrammatic).

1. Freshly passed oocyst. 2. Commencement of sporulation. 3. Sporulated and infective oocyst showing 4 sporocysts and 8 sporozoites. (Stages 1–3 show the microscopic appearance of the oocysts on examination of faeces. These stages in the development occur outside the host. The minimum period for development and for oocysts to become infective is 18 hours. Maximum survival time of oocysts is about 1 year.) 4. Sporozoites liberated from oocysts entering epithelial cell of caecum. 5. Trophozoite (developing stage). 6. Growing first generation schizont. 7. More mature first generation schizont. 8. Nearing schizogony. 9. First generation of merozoites liberated from the first generation schizont. 10. First generation merozoite entering new epithelial cell. 11. Second generation trophozoite. 12. Growing second generation schizont. 13. More mature second generation schizont. 14. The migrating second generation schizont undergoes schizogony, rupturing epithelial cells and causing haemorrhage into caecal lumen. 15. Erythrocytes. 16. Second generation merozoite entering fresh epithelial cell. 17. Third generation trophozoite developing into microgametocyte. 18. Microgametocytes. 19. Third generation of trophozoite which has grown into macrogametocyte. After these stages, syngamy occurs by union of micro- and macrogametocytes resulting in the formation of a zygote, and later the oocyst, which is passed in the faeces. Some third generation trophozoites repeat asexual multiplication by schizogony. This process may be repeated indefinitely until immunity is established. (Stages 4–19 are seen in the cells of the caecal epithelium of the infected chicken.)

undergo division, each to produce many flagellated individuals, the microgametes. The female gametocytes do not divide but undergo maturation after which a microgamete enters each to fertilize them and form the zygotes. The zygote covers itself with a cyst wall to form the oocyst which leaves the epithelial cell and is passed in the faeces. Oocysts are first seen in the droppings on the seventh day after infection. Under optimum environmental conditions they mature in about 48 hours to become infective.

Though some of the third generation merozoites become the gametocytes others perpetuate the asexual cycle by producing successively the third and the fourth generation of schizonts which in turn produce the fourth and fifth generation of merozoites respectively. This may be repeated indefinitely until the bird develops immunity. Simultaneously, some of the merozoites produce gametes and repeat the sexual cycle (*see Fig.* 13).

Pathogenicity, Lesions, and Immunity.—The major outbreaks of coccidiosis in the domestic fowl are due to *E. tenella* in the case of the chick, and *E. necatrix* in the case of the more mature birds.

The pathogenicity is mainly due to the second generation of schizonts which in the case of *E. tenella* and *E. necatrix* cause the infected epithelial cells to enlarge enormously, assume a migratory habit, and reach the deeper layers of the epithelium, damaging blood-vessels and causing haemorrhage on the fifth day of infection. The oocysts appear in the droppings on the seventh day.

In *E. tenella* infections, which cause caecal coccidiosis, the caeca are swollen and filled with a bloody mass (*Fig.* 14) and in chronic cases there are white or pink cores in the caeca which may be passed in the faeces after a long time. Development of immunity is rapid and is probably due to a local cellular mechanism. Usually birds from 3 to 12 weeks old, especially those at 6 weeks, are severely affected. The average measurements of the oocysts are $22 \times 19\,\mu$, and they are egg-shaped. The various developmental stages of *E. tenella* are capable of migration within the tissues of a bird.

When chickens are immunized with multiple doses of *E. tenella*, immunity will last up to at least 105 days (Leathem and Burns, 1968). There also appears to be some degree of cross-immunity between *E. tenella* and *E. necatrix*.

In *E. necatrix* infections there is ballooning and severe swelling of the small intestines. Often, the intestines contain a jelly-like mass consisting of blood and mucus. The haemorrhages are clearly visible from the serous surface of the intestines. What is unusual in this infection is that, while the first two generations of schizonts develop in the small intestines, the third generation develops in the caeca. Therefore, no oocysts are seen in the small intestines. Some schizonts are very large and may attain a size of $66\,\mu$. Development of immunity is delayed. Usually birds from 2 to 12 weeks or more are affected. As the development of immunity is slow there may be the acute form or the chronic form of the disease. The acute form is characterized by the passage of blood in faeces, and the chronic form by wasting, emaciation, and diarrhoea.

A bird that has survived an attack of coccidiosis may continue to excrete oocysts for a long period. Thus, the presence of oocysts in the faeces of a bird does not necessarily indicate that it is suffering from coccidiosis. The life cycle of *Eimeria tenella* is illustrated in *Fig.* 13. Some aspects of immunity of coccidiosis

are discussed by Rose (1963). Immunity is usually species specific and probably the sporozoite is affected by the immune response.

Clinical Signs and Course of the Disease.—Apart from general signs of ill health, droopiness, and tendency to huddle together, which are common signs, acute caecal coccidiosis is characterized by passage of blood in the faeces. Faeces will show a large number of oocysts after the seventh day of infection. In layers affected with intestinal coccidiosis, egg production will go down considerably. Presence of blood in faeces is usually diagnostic of coccidiosis.

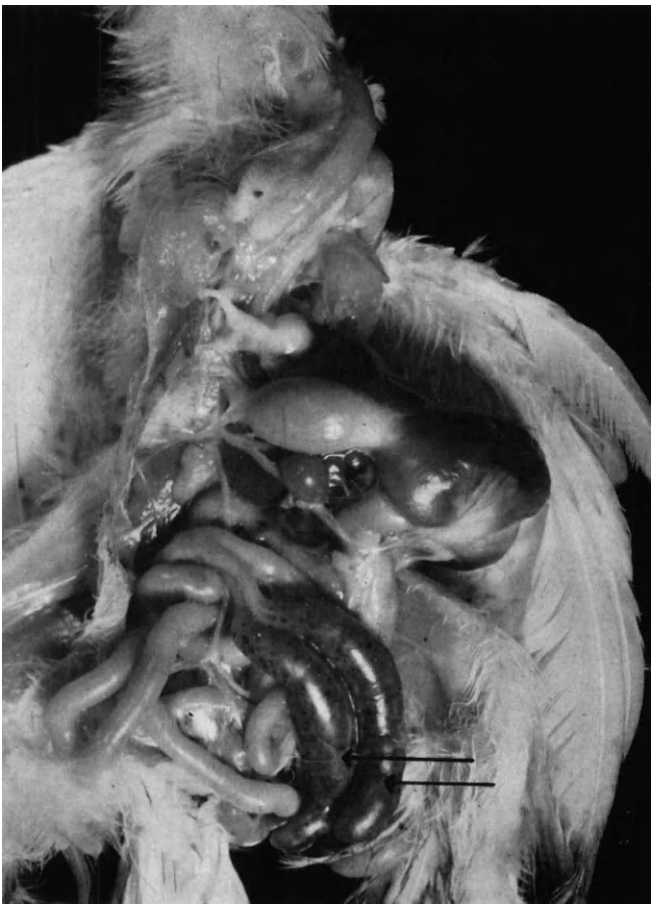

Fig. 14.—The caeca of a chicken infected with *E. tenella*, distended with blood and showing petechial haemorrhages. Arrows indicate the distended caeca.

Transmission.—This occurs by the ingestion of viable infective oocysts in food, drinking-water, and from the litter. Clinically recovered birds may pass oocysts for up to 7 months or more after recovery. Human beings, rodents, and other animals can transmit oocysts mechanically. Sporulated oocysts may live for 1–1½ years.

Prevention and Control.—The prevention and control of coccidiosis is based on two main principles: good hygiene and use of coccidiostats.

GOOD HYGIENE.—The following principles should be followed: avoid overcrowding; rear chicks if practicable on wire netting floors which should be cleaned daily; in the deep litter system, the litter should be dry, and if there is a heavy build-up of oocysts in the litter, it should be removed, and the floor and equipment thoroughly cleaned with 1 lb. of lye per 20 gallons of water, or disinfected by ammonia fumigation and the litter replaced. In general practice, the litter must be gradually built up to effect dilution, and calcium oxide may be added with advantage to keep it dry, at the rate of 10–15 lb. per 100 sq. ft. of floor space. This will help to keep down the number of sporulated oocysts but at the same time help in the development of natural immunity which is the ideal to be desired.

THE COCCIDIOSTATS.—An ideal coccidiostat should be effective against all economically important species of coccidia. While controlling the clinical manifestation of coccidiosis it should enable the bird to develop natural resistance. Most coccidiostats are now fed up to 4 weeks before the commencement of egg production. The common coccidiostats are sulphonamides, nitrofurans, halogenated arsenic acids, nicarbazin, glycarbylamide, nitrophenide, quarternary ammonium compounds, zoalene, amprolium, Unistat, Superstat, metichlorpindol, methyl benzoquate, Pancoxin, ethopabate, buquinolate, and M & B 15,497, a complex carboxylate which controls six of the most common species of coccidia including *E. tenella*, *E. necatrix*, and *E. acervulina* when given at levels of 0·004 per cent in the diet. This appears to act on the early stages of the development of *Eimeria*. It shows very potent activity against the common species of coccidia (Hodgson, 1968). It is well tolerated even at 80 times the therapeutic dose by birds (Lucas, 1968).

Metichlorpindol (Coyden 25, Dow Chemical Co.) is a new compound which has undergone extensive tests as a coccidiostat. One pound of Coyden 25 per ton of poultry feed is said to effectively control coccidiosis caused by seven species of *Eimeria*. It arrests completely the development of coccidia, and so, on the withdrawal of the drug, the birds are completely susceptible to coccidiosis (Reid and Brewer, 1967). However, a concentration of 0·025 per cent was necessary to obtain complete control of *E. acervulina* (Long and Millard, 1967). The drug probably inhibits the early phases in the life cycle. This leads to relapses after withdrawal of medication and therefore, continuous medication is indicated. Because of this property it should be useful mainly in the broiler industry. This drug does not appear to be very effective in controlling outbreaks of coccidiosis due to *E. tenella* (Norton and Joyner, 1968).

Pancoxin (Merck, Sharp and Dhome Ltd.), a mixture of amprolium, ethopabate, and sulphaquinoxaline is said to give overall prophylaxis against the nine species of poultry coccidia. A comparative study of nicarbazin, arsenobenzine, Unistat, zoalene, Whitsyn 10, and Whitsyn 5 on the action of *E. tenella* indicated that arsenobenzene, Unistat and zoalene were superior to the others under laboratory conditions (Gardiner and McLaughlin, 1963). Some of the drugs used in coccidiosis and their dosages are included in *Table* 20, p. 194.

The action of buquinolate is somewhat similar to that of metichlorpindol. It almost completely suppresses the development of coccidia, but on withdrawal of the drug the birds become susceptible again (Brewer and Reid, 1967). The drug

was very effective against *Eimeria tenella*, *E. necatrix*, *E. brunetti*, *E. maxima*, *E. praecox*, and *E. mivati*, when given in the diet at a concentration of 0·0125 per cent. When the action of buquinolate (Butoril, Cooper McDougall and Robertson) against 5 pathogenic species of coccidia was compared with amprolium-ethopabate–sulphaquinoxaline mixture (Pancoxin, Merck, Sharp and Dhome), 3, 5-dinitro-orthotoluamide (Zoamix, Dow Chemical Co.) and metichlorpindol (Coyden 25, Dow Chemical Co.) at the recommended and reduced levels it was shown to be the most efficacious drug (Dunkley, 1968). It also appears to be effective against drug-resistant strains of *E. acervulina*.

Other drugs that have been found useful, either in the treatment or prevention of coccidiosis, are suramin, pamaquin, 6-azauracil, and dithiosemicarbazones. Of these drugs 6-azauracil appears to have given consistently good results. Of the antibiotics, framycetin and chlortetracycline appear to have some effect against coccidia.

The most widely used coccidiostats today are sulphaquinoxaline, amprolium, zoalene, and metichlorpindol. Amprolium is markedly active against *E. tenella* and *E. necatrix* and this activity is enhanced in the presence of synthetic vitamin K. However, its actions against *E. acervulina*, *E. brunetti*, and *E. maxima* are not as marked. Certain sulphonamides such as sulphaquinoxaline are active against the latter group. Therefore, Davies and Joyner (1963) recommend the administration of 0·003125 per cent amprolium, 0·000196 per cent menapthone sodium bisulphate, and 0·008 per cent sulphaquinoxaline in the ration as a general preventive for coccidiosis.

Zoalene shows a wide range of activity and is useful in controlling infections due to *E. tenella*, *E. necatrix*, *E. acervulina*, *E. maxima*, and *E. brunetti* (Hymas and Stevenson, 1962). Today it is one of the most widely used coccidiostats. The levels used are 0·0062–0·015 per cent in feed. However, Light Sussex birds show a peculiar susceptibility to coccidiosis in that zoalene which controls coccidiosis in other birds does not seem to be effective in them.

Amprolium and zoalene are the drugs of choice in the prevention of coccidiosis due to *E. tenella* and *E. necatrix*. However, amprolium, because of its marked activity against *E. tenella*, may cause total inhibition of the infection at the normal dosage level of 0·0125 in food which would result in the suppression of immunity. It could also depress egg production and increase embryonic mortality. The anticoccidial and toxic activity of amprolium appear to be due to reversible thiamine inhibition phenomena. Toxicity causes the manifestation of thiamine deficiency which can be prevented or reversed by administration of thiamine. However, the dietary concentration that produces an unfavourable effect on the coccidia is several times smaller than that causing ill effects in the birds (Ott, Cobb, Cuckler, Polin, and Stoerb, 1962). Therefore, the drug exhibits an adequate margin of safety. The activity of these drugs is generally dependent on adequate levels of vitamin K. When adequate levels of vitamin K are present (2 g. per ton) the levels of these coccidiostats may be reduced.

Recent evidence (Johnson and O'Connor, 1965) indicates that nihydrazone has marked activity against *E. tenella* but a lesser degree of activity against *E. necatrix* infection when used in rations in concentrations of 0·011–0·022 per cent.

Sulphaquinoxaline is more effective as a coccidiostat against *E. maxima*, *E. brunetti*, and *E. acervulina* infections. It has been found that a mixture of 0·001

per cent diaveridine and 0·008 per cent sulphaquinoxaline in the food is very effective in controlling these three species of coccidia which are apparently more widespread in the United Kingdom than *E. tenella* (Clarke, 1964). Recent evidence indicates that 4-amino-2-ethoxy benzoic acid and related compounds have anti-coccidial action.

Methyl benzoquate (Statyl, I.C.I.) at the dosage rate of 10 g. per ton of feed (0·001 per cent in food) is also recommended as a coccidiostat for broilers. It gives excellent protection against the common species of coccidia (Ryley, 1967). It has to be given continuously as it does not permit immunity to develop.

Oral vaccines containing viable oocysts (e.g., Cocci Vac, Dom and Mitchell Products) have been prepared and used experimentally and under field conditions with certain coccidiostats such as trithiodol. These are said to confer protection.

Treatment.—The therapy of coccidiosis is discussed by Joyner (1964). Continuous drug treatment may interfere with development of immunity. Sulphonamides are the drugs of choice. The most popularly employed drugs are sulphaquinoxaline, sulphadimidine, sulphatolamide, and sulphadimethoxine. The first is widely employed because it is readily taken by the birds in drinking-water. These drugs are usually given in drinking-water for 3–5 days or on the 3–2–3 schedule where the drug is given for 3 days, withdrawn for 2 days, and given again for 3 more days. This schedule may be slightly altered to suit one's convenience. The dosages employed are 0·025–0·06 and 0·2 per cent for sulphaquinoxaline and sulphadimidine respectively. A 1 per cent mixture of sulphadimidine in mash inhibits development of sporozoites. Amprolium is also effective as a therapeutic agent against *E. tenella* infection at concentrations of 30 g. per 50 litres of drinking-water for 7 days followed by half this dose for the next 14 days. Sulphadimethoxine at dosages from 0·025 to 0·05 g. per cent in drinking-water had a high degree of therapeutic activity against the common species of coccidia (Mitrovic and Bauerfeind, 1967).

The sulphonamides act on the para-amino-benzoic folic acid metabolic sequence and, therefore, their anticoccidial action is antagonized by para-amino-benzoic acid whereas this action is potentiated by pyrimethamine and chlortetracycline. Pyrimethamine is toxic at a level of 0·0035 per cent or higher.

Ball (1964) found that a ration containing 0·01 per cent of sulphaquinoxaline and 2-amino-4-dimethylamino-5-(-4-chlorphenyl)-6-ethylpyrimidine (M & B 4408) gave good control of *E. tenella* when treatment was delayed until the third day of infection. Horton-Smith and Long (1965) found that a mixture of amprolium and sulphaquinoxaline given in drinking-water for about 7 days at 0·024 per cent and 0·018 per cent respectively appeared to be effective in treating coccidiosis caused by *E. acervulina*, *E. brunetti*, *E. maxima*, *E. necatrix*, and *E. tenella* even if treatment was started 72 hours after infection. As these are the most widely prevalent coccidia the combination of these two drugs used in these proportions should be widely acceptable for the treatment of coccidiosis of the domestic fowl.

Recently, it has been found (Ryley, 1965) that a mixture of a bipyridyl (Paraquat) and sulphadimidine (Paramez, I.C.I.) is highly effective against most forms of coccidiosis in poultry and that the preparation is readily taken by poultry and is less toxic than sulphadimidine. The dosage recommended by the manufacturer is 1 oz. Paramez to 2 gallons of water for 6 continuous days for the treatment of

caecal coccidiosis (*E. tenella* infection) and for 10 continuous days for the treatment of intestinal coccidiosis due to *E. necatrix* infection.

A combined solution containing 0·005 per cent sulphaquinoxaline and 0·0043 per cent diaveridine (Saquadil, M & B; Darvisul, Cooper McDougall and Robertson) had about the same efficacy as a 0·043 per cent sodium sulphaquinoxaline (Ball and Warren, 1965). Though some species of coccidia could be suppressed during a 5-day period of treatment, development could occur when the drug was stopped.

TRICHOMONIASIS

This is caused by *Trichomonas gallinae, Trichomonas gallinarum, Tritrichomonas eberthi,* and *Pentatrichomonas gallinarum.*

T. gallinae is found in the upper digestive tract of the pigeon, dove, domestic fowl, and certain other species of birds and causes disease in the young pigeon, in particular. It does not cause disease in the adult pigeon though a majority of them are infected.

The other three species of trichomonads listed are found in the caecum and sometimes in the liver of the fowl and appear to have a widespread distribution. Though some of them have been associated with outbreaks of disease it has not been fully established that they can *per se* cause disease in the domestic fowl.

Clinical Signs and Course of the Disease.—Chickens get the infection through contaminated water. Pigeons are carriers. Infected birds make frequent attempts to swallow, and a foul-smelling liquid may drool from the mouth. The eyes may show a watery discharge and ultimately become blind. Usually only young chickens are susceptible.

Post-mortem Lesions and Diagnosis.—*T. gallinae* causes trichomoniasis of the upper digestive tract from the mouth as far as the proventriculus in which region the lesions may be found. The lesions are, however, most frequently found in the crop, oesophagus, and pharynx. They appear as necrotic caseous circumscribed masses which may resemble buttons. The virulence of the various strains of *T. gallinae* differs considerably.

Diagnosis is based on the lesions and is made by demonstrating the flagellates. The disease has to be differentiated from avitaminosis A, moniliasis, and fowl pox.

Treatment, Prevention and Control.—Prevent pigeons and doves having access to poultry yards especially watering places.

Copper sulphate in drinking-water has been popularly used but it can prove toxic. The more effective drug is 2-amino-5 nitrothiazole (Enheptin) which is given in drinking-water. The dosage is 0·05–0·1 per cent. Metronidazole (Flagyl, M & B) which has been used successfully in the treatment of human trichomoniasis may be useful in these infections in birds.

Besides the trichomonads listed above, two other flagellates may also be seen in the alimentary canal of the chicken. They are *Monocercomonas gallinarum* and *Chilomastix gallinarum* and have very little or no disease significance.

LEUCOCYTOZOON INFECTIONS

There are at least two species causing *Leucocytozoon* infections in the domestic fowl. They are *Leucocytozoon caulleryi* and *Leucocytozoon sabrazesi.*

L. caulleryi is found in most parts of Southern Asia and it has been reported from India, Ceylon, Burma, Thailand, Indo-China, Malaysia, Sumatra, Japan, and Taiwan. Some consider that *L. andrewesi* is a synonym of *L. caulleryi* in which case North America can also be considered to be within the geographical distribution of the parasite.

The parasite is transmitted in Japan by the biting midge *Culicoides arakawae* (Akiba, 1960) but the vectors in other countries are not known.

Clinical Signs and Course of the Disease.—In Japan, Taiwan, and Thailand it appears to cause heavy mortality in severe infections. Younger birds are more susceptible. In these countries, the acute syndrome is characterized by haemorrhages in the muscles, internal organs, and legs. In India and Ceylon it is milder, the only clinical sign may be anaemia, and unless there are intercurrent infections death usually does not occur. However, in severe infections the disease may resemble that seen in Japan. The haemorrhages are caused by the rupture of the megaloschizonts.

Post-mortem Lesions and Diagnosis.—In acute cases, multiple haemorrhagic or miliary greyish spots in the internal organs, muscles, and even in the legs are characteristic, whereas in chronic cases, anaemia is constantly found. The liver and spleen are enlarged. Histologically, these and the other organs will reveal the characteristic megaloschizonts which are about 0·5 mm. in diameter. These may also be found in the blood-vessels adjoining the lesions. The histopathology of *L. caulleryi* infections has been described by Goto, Fujihara, and Morita (1966).

In *L. caulleryi* infection the fully mature gametocytes are rounded and the infected erythrocytes are enlarged considerably. In some infected cells the nucleus is lacking. In *L. sabrazesi* infection the fully grown gametocyte distorts the cell, making it spindle-shaped with two elongated processes at either end. The nucleus of the infected cell is present and this, together with that of the parasite, produces a parallel bar appearance. Very young gametocytes of both species appear as signet rings and cannot be distinguished from a merozoite of *Plasmodium* sp. or a trophozoite of *Aegyptianella* unless pigment or schizonts are present respectively (Seneviratna, Bandaranayake, and Dhanapala, 1963). Pan (1963) has suggested that *L. caulleryi* gametocytes develop in the erythrocytes as well as in the plasma. Growing evidence in recent years indicates that gametogamy of *Leucocytozoon* spp. does not usually occur in the leucocytes, as was previously believed, but in the erythrocytes.

Treatment and Control.—No effective drug was available until recently though some claimed that certain antimalarials such as atabrine and quinine were useful in the treatment of this condition. It is unlikely that these drugs will have any effect on the tissue phases of the parasite.

The disease can be controlled in the severely endemic areas by the control of the vector. In Japan, *C. arakawae* breeds in paddy fields. D.D.T. and other organic phosphorous insecticides used as sprays will destroy the larvae. Akiba,

Morii, Ebisawa, Nozawa, and Minai, (1963) have found that pyrimethamine, sulphasomezole, and sulphadimethoxine in concentrations of 0·00025, 0·02, and 0·2 per cent respectively prevented development of infection. Furazolidone in concentrations of 0·01 per cent in feed suppressed all clinical signs and mortality. Under field conditions, 0·00005–0·00025 per cent pyrimethamine or 0·005 per cent sulphaquinoxaline or sulphadimethoxine were totally effective as preventives (Akiba, Ebisawa, Nozawa, Komiyama, and Minai, 1964). Furazolidone appears to be the drug of choice.

AEGYPTIANELLOSIS

(Avian Piroplasmosis; Avian Tick Fever)

This disease is caused by *Aegyptianella pullorum*. a protozoan parasite found in the erythrocytes of the chicken in many parts of Africa, India, Southern Europe, Indo-China, and Transcaucasia in U.S.S.R.

The disease is transmitted by the adult fowl tick *Argas persicus*, in which stage-to-stage and transovarian transmission does not take place as in most other tick-borne protozoan diseases. The tick that takes in the infection becomes infective after 26 days or more. Only the same stage of tick transmits the infection.

Clinical Signs.—The incubation period is 12–15 days or more and the disease may take an acute or chronic form. The acute form is usually seen in young birds or in imported stock and the chronic form in adult birds bred in endemic areas. The principal signs are anaemia, fever, and jaundice. In the case of artificially infected birds, peak parasitaemia is reached on the fourth day, anaemia is most pronounced on the fifth day, and after this there is a remarkably rapid recovery and by the eighth day the birds become normal again (von Huchzermeyer, 1967).

In advanced cases, affected birds may show marked depression, inability to move, paralysis of extremities, and a pale yellowish discolouration of the head and mucous membranes. As these symptoms may resemble those sometimes seen in Newcastle disease, aegyptianellosis may be confused with the latter disease. (Ahmed and Elsisi, 1965).

Post-mortem Lesions and Diagnosis.—The principal lesions are enlargement of spleen and degeneration of liver with a characteristic greyish-yellow colouration of the liver.

Diagnosis is made by examination of smears of the peripheral blood and detecting the trophozoites and schizonts. They have to be distinguished from *Plasmodium* spp., from which they differ in the absence of pigment in any stage, and from *Leucocytozoon* spp., from which they differ in that there are no gametocytes and that they do not distort the affected cell at any stage. However, there can be double infections. It is advisable to overstain smears suspected to contain *A. pullorum*, as these parasites do not stain well. In *A. pullorum* infection, though there is marked anaemia, there is no leucocytosis which is pronounced in spirochaetosis, a disease which may often be associated with *A. pullorum* infection (Ahmed and Soliman, 1966).

Treatment, Prevention, and Control.—This is easily done by the control of tick vectors as outlined for *Argas persicus* (p. 103.)

There is no treatment that is worthwhile, though Ichthargan given intravenously has been claimed to be effective.

MALARIA IN THE DOMESTIC FOWL

This is caused by several species, namely, *Plasmodium gallinaceum*, *Plasmodium juxtanucleare*, *Plasmodium lophurae*, *Plasmodium fallax*, and *Plasmodium japonicum*. *P. japonicum* resembles very closely *P. juxtanucleare*. The natural host for the latter parasite appears to be the bambo partridge (*Bambusicola thoracica sonorivox*) (Manwell, 1966). Some authorities consider the former species a synonym of the latter.

P. gallinaceum is present in South East Asia and the area affected extends from India and Ceylon to Indonesia. A variety of mosquitoes can act as vectors under experimental conditions. *Anopheles* spp., *Aedes aegypti*, *A. albopictus*, and *Armageres obturbans* appear to be important vectors of *P. gallinaceum* (Huff, 1965), while the vector under natural conditions in Ceylon appears to be *Mansonia crassipes* (Niles, Fernando, and Dissanaike, 1965). It is not very pathogenic to the local fowls, but it can cause severe disease in recently imported birds, particularly in the presence of coccidiosis. Further information on avian malarias is given by Hewitt (1940) and Bray (1957).

P. juxtanucleare has been reported from Brazil, Uruguay, Mexico, and more recently from Japan, Ceylon, and the Philippines.

In natural infections, usually only a small proportion of the erythrocytes are infected. The vectors of this parasite are not known definitely. The pathogenicity of this species appears to depend on the strain. Ceylonese and Japanese strains do not appear to cause heavy mortality whereas Brazilian and Mexican strains do.

Clinical Signs.—The clinical signs are vague, and consist of anaemia, fluctuations of temperature, and paralysis in some cases. There does not appear to be a temperature elevation in *P. juxtanucleare* infection.

Post-mortem Lesions and Diagnosis.—Post-mortem examination reveals that the spleen is enlarged and the carcase is pale.

Diagnosis is made by identifying the organisms in smears of the peripheral blood, or impression smears of the liver and spleen stained with Leishman's, Giemsa's, or Wright's stains. The plasmodia are differentiated from the other protozoan parasites in the blood by the presence of pigment, schizonts, and the typical signet-ring shaped merozoites in the erythrocytes. *P. juxtanucleare* merozoites usually occur very close to the nucleus and its gametocytes are round and smaller. *P. juxtanucleare* resembles *P. japonicum* very closely and they cannot be distinguished from one another unless a careful study is made. The gametocytes of *P. gallinaceum* are large, irregular, and displace the nucleus.

Treatment and Control.—The best method to control this infection is to use insecticidal sprays with residual effect, such as D.D.T., in and around poultry houses.

Most antimalarial drugs are effective. These include chloroquine, Quinacrine (mepacrine hydrochloride), and pyrimethamine. The dosages are 5 mg., 7·5 mg., and 0·3 mg. per kg. respectively. However, treatment is hardly worth while.

TOXOPLASMOSIS

This is caused by *Toxoplasma gondii*, an organism which causes toxoplasmosis in man and most of the domestic and laboratory animals. It has been reported to occur in the domestic fowl. In the acute form it is characterized by transient botulism-like symptoms and in the chronic form by progressive central nervous disturbance with or without blindess. The main lesions are those of meningo-encephalitis and necrosis with cysts containing *Toxoplasma*-like bodies. Diagnosis can be made on the observation of *Toxoplasma*-like bodies and on the transmission of *Toxoplasma* to chickens and mice. *Toxoplasma* cysts can be demonstrated in the brain, muscle, ovary, and other organs. Occasionally, eggs may be infected. Several serological tests are useful in the diagnosis of this infection. The dye test is one of the most practical under a variety of conditions. Unfortunately, the response of the infected chickens to this test is not as strong as that of mammals or even pigeons. Daraprim (pyrimethamine) appears to be an effective drug under experimental conditions in treating toxoplasmosis of mice. The most effective dose was 1·25 mg. daily per mouse. Higher levels appear to be toxic.

HISTOMONIASIS

This disease is caused by *Histomonas meleagridis* and is not as serious as it is in turkeys. However, in severe infections especially in chickens, they cause typhlitis, caecal cores and necrotic lesions in the liver, which are similar to those seen in the turkeys. Though this protozoan is transmitted primarily by the eggs of the caecal worm *Heterakis gallinarum*, it has been found that the disease can occur in the absence of this worm. It is likely that in these cases certain mites and other arthropods transport the protozoan. However, so far there has been no conclusive evidence to support this view. It is difficult to demonstrate the parasite by usual methods, but the fixation and rapid staining method of Hamilton and Campbell (1961) is useful especially in detecting the early acute stages of the disease. Further information on histomoniasis is given under the section on turkeys.

2. METAZOAN PARASITES

A. HELMINTHS

The helminths may be classified as the round worms or the Nematoda; the tapeworms or the Cestoda; the flukes or the Trematoda; and the thorny headed worms or the Acanthocephala. It is not considered necessary to go into the details of the structure and classification of these parasites. The names of the common helminths of the fowl are listed in *Tables* 6–9, with their location, relative pathogenicity, intermediate host, if any, and treatment whenever this is practicable. The disease conditions caused by the more common and pathogenic worms are described separately.

Helminthiasis in the fowl can be caused by any of the helminths listed in *Tables* 6–9. However, only three of the nematode worms listed are widespread in nature and cause considerable economic losses. These are *Ascaridia galli*, *Heterakis gallinarum*, and *Syngamus trachea*. The most pathogenic tapeworms are *Davinea proglottina*, *Raillietina echinobothridia*, and *Raillietina* spp. These will be dealt with separately.

Table 6.—THE ROUND WORMS (NEMATODA) IN THE ALIMENTARY CANAL

NAME	LOCATION AND DESCRIPTION	PATHOGENICITY AND INTERMEDIATE HOST	SPECIFIC TREATMENT
Ascaridia galli	Small intestines. About 3–12 cms. long and 1 mm. thick depending on stage of development; they resemble noodles	Pathogenic. None; see under *A. galli* infection, p. 96	Piperazine compounds, 300 mg. per kg.
Capillaria annulata	Oesophagus and crop. Filament-like worms 1–6 cm. long and about 0·1 mm. wide	In heavy infection yellowish-white nodules and pseudo-membranes are produced. Earthworms	Methyridine 200 mg. per kg. subcutaneously or 0·3 per cent in drinking-water for 24 hours
Capillaria obsignata and *Capillaria* spp.	Small intestines. Similar to *C. annulata*	Generally not pathogenic. None	Methyridine effective in doses of 100–150 mg. per kg. subcutaneously against all stages. Haloxon in doses of 50–60 mg. per kg. effective against adults only. Thiabendazole at a dosage rate of 1 g. per kg. live weight is also effective but may not be economical for general use
Capillaria quodinflata	Small intestines. Similar to *C. annulata*	Generally not pathogenic. Earthworms	Methyridine may be useful
Cheilospirura hamulosa	Gizzard. Reddish-brown worms about 1–3·5 cm. long and 0·5 mm. wide	The wall of the gizzard may be weakened and eroded in heavy infections. Grasshoppers, beetles, and other insects	None

Dispharynx nasuta	Proventriculus. Creamy-white worms about 1 cm. long and 0·3 mm. wide	Pathogenic if infection is heavy; ulcers are formed and the proventriculus becomes very thickened. Snowbugs and pill bugs	None
Gongylonema ingluvicola	Crop. Thread-like worms 2–7 cm. long; thicker than *Capillaria* spp.	Generally non-pathogenic. Beetles	None
Heterakis gallinarum	Caeca. Small thread-like worms about 0·5–1 cm. long	Not very pathogenic. Transmits *Histomonas meleagridis*, the agent causing blackhead in turkeys. Earthworms act as vectors	Phenothiazine compounds. See under *Heterakis gallinarum* infection, p. 97
Strongyloides avium	Caeca. Minute worms. Only the female is parasitic and measures about 2·2 × 0·04 mm.	In heavy infection the caecal wall becomes thickened. None	None
Subulura brumpti (pin worm)	Caeca. Small worms a little larger than *H. gallinarum*	Not pathogenic. Grasshoppers, beetles, and mealy bugs	Not necessary
Subulura strongylina	Caeca. Similar in size to *S. brumpti*	Not pathogenic. Unknown	None
Subulura sucteria	Intestines	Beetles	None
Tetrameres americana and *Tetrameres* spp.	Proventriculus. Males about 5 mm. long; females same length but with globose dark red bodies	In heavy infection the wall becomes thickened and catarrhal; females can be seen through the wall as blackish-red masses about 0·2 cm. in diameter. Grasshoppers and cockroaches	None
Trichostrongylus tenuis	Caeca. Thin worms about 5–10 mm. long	In heavy infection there is thickening and haemorrhage of the caecal wall. None	None

NEMATODE INFECTIONS

Ascaridia galli Infection.—*Ascaridia galli* (the large round worm) is found in the intestines of the fowl, guinea-fowl, turkey, and a variety of other birds. The male is about 6 cm. and the female about 10 cm. long.

LIFE CYCLE.—The eggs are passed out in the faeces and become infective under suitable conditions in a minimum period of 10 days. Moderately high temperatures, high humidity, and high oxygen content favour the rapid development of the larvae within the egg. The infective egg contains the second stage larva which is fairly resistant and may remain viable for about 3 months.

Infection occurs by ingestion of the infective eggs. Ten days after being ingested, the larva from the egg penetrates the duodenal mucosa where it remains for 7 days and comes out into the lumen. During this phase in the mucosae it produces enteritis.

The larva becomes adult in 5–8 weeks. There is no migratory phase through the lungs. Chickens over 16 weeks old are more resistant and resistance causes arrest in the growth and development of the larvae.

CLINICAL SIGNS AND COURSE OF THE DISEASE.—Young birds suffer more severely from the infection, particularly when vitamins A and B complex are deficient in their diets. The birds have enteritis and diarrhoea, become emaciated and anaemic. There is a drop in egg production and in advanced cases there may be paresis. The fall in egg production may be quite severe in the last few months of the hen's productive life (fifty-sixth to sixty-sixth weeks), when there is a normal reduction in the percentage of egg production. If the infection is not heavy, the birds gradually recover and become more resistant to re-infection. However, in heavy infection there may be many deaths.

POST-MORTEM LESIONS AND DIAGNOSIS.—The mucosal phases cause enteritis. In heavy infections, there may be intestinal obstruction or stasis. Some worms may be seen in the proventriculus, crop, and bile-duct. In early infections, only larval worms about 7 mm. long can be seen in the duodenal mucosa. Diagnosis is made by finding the oval eggs which measure 73–99 × 45–57 μ with smooth shells, and are unsegmented when laid.

PREVENTION AND CONTROL.—Overcrowding and damp litter should be avoided. The litter or the run may be given a top dressing of lime. Nutrition should be adequate, especially in vitamin A and B complex requirements. An effective piperazine compound should be given at regular intervals when the birds are young. If heavy infection is seen in a flock of birds on deep litter, the litter should be replaced. Infected pens should be rested for at least 3 months.

TREATMENT.—Several drugs are available for the treatment of this infection. Some of them are piperazine compounds, butyl-n-phenyl-dithiocarbamate and di-n-butyl tin dilaurate. Piperazine compounds are used widely. Some of the most popular ones are piperazine citrate, piperazine adipate, and piperazine hexahydrate. They may be administered in single doses of 100–500 mg. per kg. per bird. The first two drugs can also be incorporated in feed at the rate of 300 mg. in 100 g. of wet mash or 300 mg. per 200 ml. of drinking-water. Piperazine hexahydrate may be given in drinking-water for 2–3 days at the dosage rate of 8 g. in 1 gallon of drinking-water. Thiabendazole is also effective. This drug may be given in feed for 14–21 days at low levels. Single doses are less effective. Panda (1965) using

piperazine adipate in concentrations of 0·3 per cent in feed obtained optimum results in heavy infections, when routine treatment was given at 15-day intervals.

Tetramisole acetate given in drinking-water at a concentration of 0·04 per cent is 99–100 per cent effective as a therapeutic agent (Bruynooghe, Thienpont, and Vanparigs, 1968).

As a routine measure, worming may be done during the sixth, sixteenth, and twenty-sixth week of a bird's life.

Heterakis gallinarum Infection.—*Heterakis gallinarum* (caecal worm) is a small worm found in the caeca of the fowl, turkey, duck, goose, and many other species of birds. It measures 7–15 mm. long, the male being slightly smaller than the female.

LIFE CYCLE.—The eggs are passed out in the faeces and become infective under optimum conditions in 14 days. Infection occurs by ingesting the infective eggs containing the second stage larvae. The larvae hatch out in the intestines and without migrating through the tissues, reach the caeca, where they remain in the tissues of the anterior two-thirds of the caeca for up to 12 days, then reach the distal third to become adults. The first eggs are passed in the faeces about one month after infection. Earthworms of the genera *Lumbricus*, *Allobophora*, and *Eisenia* act as vectors in the transmission of *H. gallinarum*.

The eggs are swallowed by these earthworms, inside the bodies of which they hatch out into larvae.

CLINICAL SIGNS AND COURSE OF THE DISEASE.—The worms are not very pathogenic but the eggs of the worms transmit the protozoan *Histomonas meleagridis* to fowls and turkeys. It is presumed that the protozoan is carried within the helminth egg. Heavy infections may cause typhlitis, diarrhoea, and nodules in the caeca. Death does not usually occur.

POST-MORTEM LESIONS AND DIAGNOSIS.—Post-mortem examination will reveal typhlitis or caecal cores which are usually suggestive of blackhead in the fowl. Diagnosis is made from the presence of worms in the caeca or eggs in the faeces. The eggs are oval and measure 65–80 × 35–46 μ and are smooth-shelled when laid. They have to be differentiated from those of *A. galli* from which they differ in being smaller in size and having a thinner shell.

PREVENTION AND CONTROL.—This is based on principles similar to those recommended for the *A. galli* infection. Infected chickens and turkeys acquire partial resistance (Lund, 1965).

TREATMENT.—Phenothiazine and di-n-butyl tin dilaurate are effective, but phenothiazine is the superior drug and can be given individually or in mash. The dose for an adult is 0.5 g. A single treatment usually eliminates over 90 per cent of the helminths. In areas where infection is heavy or blackhead is endemic, phenothiazine can be given periodically to the fowls.

Tetramisole acetate given in drinking-water at a concentration of 0·04 per cent is 99–100 per cent effective as a therapeutic agent (Bruynooghe, Thienpont, and Vanparigs, 1968).

Syngamus trachea Infection (Gapeworm infection).—*Syngamus trachea* (the gapeworm) lives in the trachea of the fowl, turkey, and many other species of birds. The sexes are permanently joined in copulation forming a Y shape. The mature

male is about 5 mm. long and the mature female 15–20 mm. They are bright red in colour due to the ingested blood.

LIFE CYCLE.—The eggs are laid in the trachea, coughed up, swallowed, and passed in the faeces. The eggs become infective under optimum conditions in about 3 days. The third stage infective larva will hatch on the ninth day under suitable conditions. These infective larvae may be swallowed by earthworms, snails, slugs, flies, and other arthropods which act as transport or paratenic hosts in which they become encysted and live for a few years. Passage of these larvae through the earthworms make them more highly infective. Infection of the bird may occur, by ingestion of eggs with the infective larvae, free infective larvae, or paratenic hosts with encysted larvae. After ingestion the larvae penetrate the intestinal wall, enter the blood-stream, and are carried to the lungs and the alveoli. In this location two ecdyses occur and the worms migrate to the larger bronchi, where copulation takes place, and then to the trachea. The trachea is reached on the seventh day at the earliest and the first eggs are passed in the faeces 17–20 days after infection.

CLINICAL SIGNS AND COURSE OF THE DISEASE.—Young birds between 1 and 3 months are more susceptible, while adult fowls and turkeys act as carriers. The migrating worms may cause pneumonia and the adult worms in the trachea cause tracheitis in the fowl and nodules in the trachea of the turkey. Where there are large numbers of parasites they obstruct the passage of air causing the characteristic signs to appear. The birds make violent inspiratory efforts with extended neck and a fully or half open beak, a syndrome which is called 'gapes'. They also frequently shake the head and toss it, in attempts to dislodge the worms. In severe infections the birds die of asphyxia. In less acute cases where gaping movements are not shown the birds may recover. If only a few worms are present the characteristic signs are not seen.

POST-MORTEM LESIONS AND DIAGNOSIS.—There is catarrhal tracheitis with blood-tinged exudate. The characteristic red worms will be seen in the trachea. In live birds, the worms can be seen through the larynx when they inspire air, if adequate illumination is available. Faeces will reveal the large eggs measuring 78–110 × 43–46 μ with an operculum at either pole. These eggs are larger than those of *Capillaria* spp. which also have a plug at either pole.

EPIZOOTIOLOGY, PREVENTION, AND CONTROL.—Gapeworm infection is common in birds on free range in moist areas where earthworms, snails, slugs, and other transport hosts are abundant. They are seldom seen in caged or battery-housed birds or those kept on deep litter and where the litter is dry. The development of *S. trachea* eggs requires a high degree of moisture.

To avoid infection, birds must be reared on dry litter and not be permitted to have access to the transport hosts or infected premises. Turkeys should not be reared with fowls. Birds may be vaccinated against syngamosis using irradiated larvae of *S. trachea.*

TREATMENT.—The most effective remedy is barium antimony tartrate which is used as a dust and given as an inhalation. The birds are placed in a box and the powder blown in with a spray gun or other means at the rate of 1 oz. per 8 cu. ft. of space. Tilting the box causes the birds to flap their wings and thus disperse the powder. The birds may be exposed to the powder for 5–10 minutes. The dust is inhaled by the birds and comes into contact with the helminths, which disintegrate.

Unfortunately barium antimony tartrate is not readily available at the chemist, but it can be prepared from sodium or potassium antimony tartrate and barium chloride in the following manner.

Barium antimony tartrate may be prepared by dissolving 244·3 g. of crystallized barium chloride in 800 ml. of warm distilled water and mixing this solution slowly with a warm solution of potassium antimony tartrate made by dissolving 667·8 g. of the crystalline substance in 1700 ml. of distilled water. The mixture is stirred vigorously. A precipitate of barium antimony tartrate is then formed. The liquid is filtered through filter-paper and the precipitate is collected on the filter. The precipitate is first washed with water then with alcohol and dried. Then it is powdered if necessary.

Allyl sulphate and phenothiazine are also said to be effective. Phenothiazine kills the larvae as they hatch and therefore it has to be given continuously in mash at levels of 4 per cent. Individual worms may also be removed with fine forceps or with a wire spiral if they are in the upper third of the trachea. Unfortunately the worms are often found in the lower third when extraction is more difficult.

Methyridine, carbamazine citrate, and thiabendazole appear to have some effect on the worms, but their exact value in clinical cases has not yet been assessed. Thiabendazole may be given in concentrations of 0·05–0·1 per cent in mash for 7 weeks, when it is reported that the majority of worms die. This drug has been used in pheasants with success. Wehr (1964) and Wehr and Hwang (1967) found that thiabendazole at 0·5 per cent concentration in mash given for 20 days continuously after infection, was 98·17 per cent efficient in removal of *S. trachea.* Disophenol (D.N.P., Cyanamid or Akylol, Cyanamid) is also said to be effective but the toxicity of this drug has to be considered.

FILARIAL INFECTIONS

Though filariasis of the domestic fowl is not common, Niles and others (1965) have recently reported 5 different types of microfilariae in the domestic fowl. *Mansonia crassipes* is the vector of at least one of them. According to Crawford, filariasis in fowls can be a clinical problem and is characterized by anaemia and oedema of the comb and wattles.

The adult filariae that have been reported from the domestic fowl are:—

1. *Aprocta caudata* Machado and Medonca, 1961.
2. *Bhalfilaria badami* Bhalerao and Rao, 1944.
3. *Cardiofilaria nilesi* Dissanaike and Fernando, 1965.
4. *Gallifilaria mhowensis* Jain, Alwar, Awadhiya, and Pandit, 1965.

TAPEWORM INFECTIONS

(*Cestodiasis*)

The common tapeworms of the fowl have been listed in *Table* 7. The most pathogenic ones are *Davainea proglottina, Raillietina echinobothridia,* and *Raillietina tetragona.* The last two species have a world-wide distribution.

Clinical Signs and Course of the Disease.—Young birds and those under poor conditions of management are most susceptible. There is anaemia and emaciation, and segments are passed in the morning, especially if the birds are starved.

Table 7.—THE TAPEWORMS (CESTODA)

NAME	LOCATION AND DESCRIPTION	PATHOGENICITY AND INTERMEDIATE HOSTS
Amoebotaenia sphenoides	Duodenum. Armed tapeworm with fusiform body up to 3·5 mm. long. It is one of the smallest poultry tapeworms. The last segment is narrower than that immediately preceding it	Mildly pathogenic. Earthworms
Choanotaenia infundibulum	Jejunum. Armed tapeworm. Rostellum alone has a small number of relatively large hooks. Up to 2·3 cm. long	Not very pathogenic. House fly and dung beetles
Davainea proglottina	Duodenum. Armed tapeworm up to 4 mm. long. It is one of the smallest poultry tapeworms. The last segment is broadest	Very pathogenic. Causes anaemia, emaciation, and even death. It has been associated with paralysis in birds. Slugs and snails
Fimbriaria fasciolaris	Intestine. An armed tapeworm with pseudoscolex	Pathogenicity not known. Water flea
Hymenolepis carioca	Duodenum. Slender thread-like unarmed tapeworm, up to 8 cm. long, segments 3–5 times broader than long	Not pathogenic though it is commonly found in large numbers. Stable fly and dung beetles
Metroliasthes lucida	Ileum. Unarmed tapeworm up to 20 cm. long	Pathogenicity to the fowl unknown. Grasshoppers
Raillietina cesticillus	Jejunum. Armed tapeworm. The rostellum is broad and flat and has hooks. Suckers have no hooks. Up to 12 cm. long	Pathogenic to young birds. May cause inflammation and degeneration of the villi of the intestines. House fly and dung beetles
Raillietina echinobothridia	Intestines. Armed tapeworm up to 25 cm. long	Pathogenic. Causes nodules which resemble tubercles, mucoid diarrhoea, emaciation, and nervous signs
Raillietina peradeniya	Intestines	Non-pathogenic. Unknown
Raillietina tetragona	Ileum. Armed tapeworm up to 25 cm. long	Pathogenic. Will cause death and distension or necrosis of intestines, nodules are not usually formed (*see* 'Tapeworm Infection', p. 99). Ants

D. proglottina penetrates deep into the intestines and produces marked enteritis, while *R. echinobothridia* causes the formation of nodules which resemble those of tuberculosis. In *D. proglottina* infection there is dysentery, paresis, and loss of egg production and in heavy infection death may occur. Paresis may also be seen in *Raillietina* infection. The other tapeworms are not very pathogenic. *Amoebotaenia sphenoides* and *Hymenolepis carioca* are practically harmless.

Post-mortem Lesions and Diagnosis.—In severe infections there is enteritis especially in the duodenum. Nodules are seen in *R. echinobothridia* infections. These are clearly visible from the serous wall. When infections are heavy, the gut wall is thickened and its lumen partially occluded by the worms. Diagnosis is made by seeing the segments of the worms or their eggs in faeces.

Prevention and Control.—Birds on free range are more heavily infected as they are more likely to have access to the intermediate hosts. The best method to reduce these infections is to control the intermediate hosts that have been listed in *Table* 7. Snails and slugs can be controlled by the judicious use of copper sulphate or other molluscicides that are not toxic to poultry, and also by controlling ants with organophosphorous compounds and other insecticides. Control of ants is difficult. They become abundant if food is wasted and thrown around the fowl cages. Proper disposal of faeces by compost-making will reduce the number of eggs available for development as the heat generated during the compost-making destroys the eggs.

Treatment.—Treatment of tapeworm infections is difficult. Kamala, carbon tetrachloride, and tin compounds, especially dibutyl tin dilaurate and dibutyl tin oxide have been used. The last drug is given in single doses of 75–150 mg. per bird in gelatin capsules or at the rate of 500 mg. per kg. (0·5 per cent) feed for 2–3 days. Recently, chlorosalicylamide (Yomesan, Bayer) has been found to be very safe and effective. The dosage recommended is 500 mg. per adult bird. Polystat, a coccidiostat, appears to control *R. cesticillus* infection when given in the manner recommended by the manufacturers. Bunamidine hydroxynaphthoate at a dosage rate of 82 mg. of the base per kg. was effective in removing 50 per cent of the tapeworms *Raillietina tetragona*, *R. cesticillus*, *R. echinobothridia*, and *Amoebotaenia sphenoides*. The efficacy was increased at higher dosage rates and dosages as high as 400 mg. per kg. of the base did not prove toxic (McCulloch and Kasimbala, 1967).

TREMATODE INFECTION

(Fluke Infection)

The common trematodes reported from the fowl are listed in *Table* 8. None of them causes serious losses unless infection is heavy. The only trematode of significance is *Collyriclum faba*, the skin fluke, detected usually in the slaughter-house. The skin may be trimmed off and the carcase passed for slaughter. Trematode infections could be a serious problem in ducks, because these birds are sometimes very heavily infected as they frequently feed on the snail intermediate hosts.

Trematode infections are rarely diagnosed in life and if they become a flock problem, treatment is difficult and often not satisfactory. The only drug that has

Table 8.—THE FLUKES (TREMATODA) AND THORNY-HEADED WORMS (ACANTHOCEPHALA)

NAME	LOCATION AND DESCRIPTION	PATHOGENICITY AND INTERMEDIATE HOST
Eschinoparyphium recurvatum	Intestines. Resembles *E. revolutum* but is much smaller, up to 4·5 mm. long	Severe enteritis and debility. Life cycle similar to that of *E. revolutum*
Eschinostoma revolutum	Intestines, caeca, and cloaca. Body up to 22 mm. long. Oral sucker has adoral disk with spines	Enteritis, diarrhoea, and dysentery in heavy infections. Pathogenic to pigeons. First intermediate host is a fresh water mollusc and the second intermediate hosts are snails or tadpoles
Hypoderaeum conoideum	Intestines. Resembles *E. revolutum* but adoral disk poorly developed	This is essentially a parasite of the duck. Pathogenicity for the fowl not known. Life cycle similar to that of *E. revolutum*
Postharmostomum gallinum	Caeca. Tongue-shaped small fluke up to 7·4 mm. long	Not pathogenic. First and second intermediate hosts are snails
Ribeiroia ondatrae	Proventriculus. Resembles *E. revolutum* but there is no spine-bearing disk. Small flukes, 3 mm. in length	Pathogenic. Causes pitting, inflammation, and oedema of proventriculus with formation of ulcers and fibrinous exudate. First intermediate host is a fresh water snail and the second intermediate hosts are fish or tadpoles
Zygocotyle lunata	Caeca. Oval fluke with a ventral sucker at posterior end. Up to 9 mm. long	Not pathogenic. Only one intermediate host required, which is a snail

shown some promise in the treatment of trematode infections of the alimentary or reproductive tract is carbon tetrachloride. The dosage is 1–3 ml. according to size of the bird. The same amount may be introduced into the cloaca mixed with 2–3 times its volume of a mineral oil such as liquid paraffin.

Control of fluke infections is best effected by destroying the intermediate hosts. In modern systems of poultry husbandry, fluke infections are rarely seen as the intermediate hosts do not usually gain access to modern poultry houses. However, in birds on free ranges, such infections are seen, but rarely in such severe form as to warrant concern or to necessitate control measures.

B. ECTOPARASITES

The ectoparasites of the fowl are arthropods and may be ticks and mites (Acarina), lice (Phthiraptera), fleas (Siphonaptera), mosquitoes, gnats, black flies (Diptera), and other insects. The last group usually feeds on the fowl, other birds, and mammals for short intervals at a time. Therefore, they are not specific and permanent ectoparasites of the fowl, as the others are. They are nonetheless important as some of them can transmit specific agents that cause disease, chiefly protozoan parasites.

TICKS AND MITES
(*Acarina*)

The Ticks.—The most common tick that parasitizes the fowl is *Argas persicus*, the fowl tick. This is a soft tick which is leathery and hides in day-time in the cracks and crevices of poultry houses and under the bark of trees, and feeds on the fowl at night intermittently. It is found in warm or temperate areas only. It is oval in shape and slatey-blue in colour when engorged. The adults vary in size being 4·5–10·4 × 2·5–6·3 mm. according to the degree of engorgement. The nymphs and the larvae are smaller. The larva has only 3 pairs of legs while the nymph and the adult have 4 pairs.

THE LIFE CYCLE.—The eggs are laid in the cracks of poultry houses. The larvae hatch after 3 weeks and they feed on the fowls, remaining permanently attached to them for about 5 days or more until they are engorged, after which they drop off and moult twice, producing two nymphal stages which feed intermittently at night. Unlike the larvae, they are not permanently attached to the fowls. The second nymphal stage produces the adult which also feeds at night, the female laying a batch of eggs after each meal which is taken nearly once a month. The nymphs and the adults can tolerate starvation for several years.

PATHOGENICITY.—The ticks, except in the larval phase, attack the birds at night and suck blood causing anaemia. They also transmit several important pathogens: *Aegyptianella pullorum*, a protozoan parasite causing tick fever, which is not transmitted via the egg, and *Borrelia anserina* which causes spirochaetosis.

ERADICATION.—The parasites, except in the larval phase, are not found on the fowl during the day-time when they hide in cracks and crevices. The aim should be eradication of the parasite from the premises and not merely control. The insecticide which was popularly used in the past was nicotine sulphate, but the modern insecticides such as D.D.T., toxaphene, Gammexane, diazinon, and

Table 9.—HELMINTHS IN SITUATIONS OTHER THAN THE ALIMENTARY CANAL

ORGAN OR SYSTEM	LOCATION AND DESCRIPTION	INTERMEDIATE HOST AND PATHOGENICITY	TREATMENT
Trachea	*Syngamus trachea* (nematode). Red worms, male and female in copula forming a Y-shape	Earthworms act as transport hosts. They occlude the respiratory tract and cause gasping respirations	Inhalation of barium antimony tartarate dust. Methyridine, thiabendazole and disophenol (D.N.P.) are also effective
	Cyclocoelum japonicum (trematode)	Intermediate hosts not known	Not known
Conjunctival sacs	*Oxyspirura mansoni* (nematode). Creamy-white worms 1–2 cm. long	Cockroaches. Not pathogenic. Heavy infections may cause ophthalmia	Not necessary
	Philophthalmus galli and *Philophthalmus* spp. (trematodes)	Intermediate hosts not known. They produce conjunctivitis	Not known
Skin, in small cysts chiefly around the vent	*Collyriclum faba* (trematode). Hemispherical fluke about 7 × 5 mm. in size	Intermediate hosts not known. They produce cysts round cloaca	Not known
Calices and ureters of the kidney	*Tamerlania bragai* (trematode). Elongate body up to 3 mm. long	Land snails are the intermediate hosts. Heavy infection may cause anaemia and occlusion of ureters	Not known
Bursa of Fabricius and oviduct	*Prosthogonimus macrorchis* (trematode). Pear-shaped flukes with spiny cuticle up to 7·56 mm. long	The first intermediate host is a fresh water snail *Amnicola limosa* and the second intermediate host is a dragon fly. Decreased egg production, laying of thin-shelled eggs, oviductitis, and even peritonitis resulting from rupture of the oviduct	Not known

malathion are superior. The following procedure, if followed carefully, can eradicate the parasite from infected premises:—

1. Birds are removed from the infected poultry yards to separate crates, houses, or non-infected premises, and wherever possible they should be kept in these premises for 5–10 days, or at least until the insecticide spray used in the original premises has settled down.

2. The infected poultry houses are sprayed with 2·5 per cent toxaphene, or 3 per cent malathion in water, or 5 per cent D.D.T. in a mixture containing 1 pint used engine oil with 3 pints of paraffin (kerosene oil), using a pressure sprayer, a Shell-tox, or similar sprayer, so that the spray reaches all the cracks and crevices. Usually, 3 bottles (72 oz.) to 1000 sq. ft. of surface area would suffice. It is important to see that wherever possible the spray reaches the cracks and crevices. Instead of these insecticides, 0·5 per cent lindane, chlordane, or Gammexane may be used. The birds are then returned to the fowl house and those larvae that have not fallen off, will drop off to moult and the residual effect of the insecticides then kills them. In no case should the insecticides be used when the birds are in the infected fowl houses.

3. Crates or the premises used to segregate the infected birds are treated as described above after the birds are returned to their original houses.

4. Surrounding areas such as dead shrubs, timber depots, and other infected areas may be treated with a 5 per cent D.D.T. solution in paraffin (kerosene oil).

A second treatment with the insecticide may be necessary in a week if the reintroduced birds harbour larval stages which have not been killed by the residual effect of the insecticide. The larvae appear to be more resistant to insecticides than the other stages.

Other ticks which may be found on fowls are the various species of *Haemaphysalis*, such as *H. intermedia*, especially in India and Ceylon. Other species of *Haemaphysalis* may also be seen in rural areas especially if the fowls are on free range. In Bulgaria, *H. punctata* is said to cause tick paralysis in chickens. None of these ticks transmit any pathogens as far as is known. However, heavy infections can be controlled by using on the infected birds any of the insecticides mentioned before, taking care that more dilute preparations than those recommended for the control of *Argas persicus* are used. In general, these ticks can be controlled by using the methods advocated for the control of lice.

The Mites.—They are usually small or microscopic Acarina which are, in most instances, permanent ectoparasites on their hosts. There are a large number of mites, some of which are very harmful, while some are relatively non-pathogenic. The common mites on the fowl in approximate order of relative importance are:—

1. *Dermanyssus gallinae*, the common red mite.
2. *Knemidocoptes mutans*, the scaly-leg mite.
3. *Knemidocoptes gallinae*, the depluming or body-mange mite.
4. *Ornithonyssus sylviarum*, the northern feather mite.
5. *Ornithonyssus bursa*, the tropical feather mite.
6. *Epidermoptes bilobatus*, the skin mite.
7. *Syringophilus bipectinatus*, the quill mite.
8. *Megninia cubitalis.*
9. *Megninia ginglymura.*

10. *Megninia gallinulae*, a mite infecting the legs and head, causing dermatitis.
11. *Pterolichus obtusus.*
12. *Neoschöngastia americana.*
13. *Revoltasia bifurcata.*
14. *Laminosioptes cysticola*, the sub-cutaneous or flesh mite.
15. *Cytodites nudus*, the air sac mite.

Besides the above, larval stages of the various species of harvest mites, especially those of *Trombicula* spp. will attack fowls and in heavy infections may even cause death. The larvae alone are parasitic and are abundant in spring and summer in certain areas.

LIFE CYCLE.—The eggs are laid on the host in most instances, and hatch out into six-legged larvae which moult to produce eight-legged nymphs. There may be several stages of nymphs, each nymphal stage being preceded by a moult. The final-stage nymphs moult to produce the adults which are sexually differentiated and mature. The life cycle takes 1–4 weeks for completion.

PATHOGENICITY AND CLINICAL SIGNS.—The pathogenicity of each species of mite varies and therefore the clinical signs produced vary with each species.

Dermanyssus gallinae.—Unlike most other mites, it breeds in the fowls' environment and attacks the fowls at night, hiding during the day-time in cracks and crevices of the poultry houses. It is red in colour and about 0·6 × 0·4 mm. in size. It sucks blood causing anaemia and if infection is heavy, young parasitized chickens may die (Kirkwood, 1967). It can also transmit *Pasteurella multocida*, the causative agent of fowl cholera, the virus causing fowl pox, the spirochaete *Borrelia anserina*, and the virus causing equine encephalomyelitis (both Eastern and Western types).

Knemidocoptes mutans.—This is a nearly spherical mite 0·5–0·25 mm. in size causing proliferation of the scales on the legs making them appear thick and erect. The mites are permanently parasitic on the hosts and infection is caused by direct contact.

Knemidocoptes gallinae.—This resembles the scaly-leg mite in morphology but invades the feathered areas of the epidermis causing irritation and inducing the host to pull out feathers. The lesions are seen most frequently on the back and wings, and usually appear as bare or semi-bare patches.

Ornithonyssus sylviarum.—This is found in the temperate and sub-tropical areas. It resembles the red mite but it is found on the body as well as in the environment. It is a vicious blood sucker and causes anaemia and formation of scabs. It lays eggs on the host and can transmit the virus of fowl pox. *O. bursa* is found on birds in the tropics and the eggs are laid in the litter. In pathogenicity and other respects it resembles *O. sylviarum.*

Epidermoptes bilobatus.—This is a skin mite which may occasionally produce dermatitis and scab formation.

The Feather Mites.—Syringophilus bipectinatus, Megninia cubitalis, M. ginglymura, Megninia spp., *Rivoltasia bifurcata*, and *Pterolichus obstusus* are feather mites and are found either inside the quills of the feathers or outside feeding on the epithelial debris, barbs, and barbicules. The presence of these mites is indicated by the appearance of powdery fluff in the quills, loss of feathers, feather picking, or by the presence of cut barbicules giving them the appearance of having been cut by a pair of scissors.

Neoschöngastia americana.—This lives on the skin and causes a dermatitis which may reduce the market value of carcases.

Cytodites nudus.—This lives in the lungs, air sacs and bone cavities of a variety of birds including the fowl, and appears as whitish dots about 0·6 × 0·4 mm. in size. No reliable information is available on its life cycle, mode of dissemination, or pathogenicity.

Laminosioptes cysticola.—This is seen in the subcutaneous tissues of the fowl and its presence is marked by small, flat, oval, yellowish nodules which can be mistaken for bits of fat. They are not pathogenic but their presence will reduce the market value of a carcase.

CONTROL AND TREATMENT.—*Dermanyssus gallinae*, the red mite, is the most troublesome mite and as it lives most of the time in the fowls' environment, control methods must be directed against the parasites on the fowl as well as those in its environment. Nicotine sulphate is effective but this has now been largely replaced by the modern insecticides such as D.D.T., lindane, and malathion. A 1·5 per cent solution of D.D.T. in kerosene may be used on the fowl houses and their environment. Lindane and malathion may be used in the strengths recommended by the manufacturers. Several applications at weekly intervals may be necessary before complete control is obtained. It is not advisable to treat the birds themselves, and the principles used in the control of *Argas persicus* may be used to eliminate *D. gallinae*.

Knemidocoptes mutans can best be controlled by culling the affected birds and treating the environment as described for *D. gallinae*. The individual birds are treated by soaking the scales with a solution of soft soap or kerosene, and applying a 0·1 per cent emulsion of gamma B.H.C. or 0·5 per cent solution of D.D.T. in kerosene.

K. mutans infection may be controlled by treating the affected areas in the bird with sulphur ointment 1:8 or an ointment containing 0·1 per cent gamma B.H.C.

The control of feather mites including *O. sylviarum* and *O. bursa* requires the treatment of the birds as well as their environment. The environment may be treated as for *D. gallinae* infection. Application of 40 per cent nicotine sulphate (Black Leaf 40) to the roosts, perches, and poultry yards is very effective. The birds may be treated by dipping them in a solution of malathion if the weather is warm or using malathion as a spray. Malathion may also be used as a spray on the poultry houses. Dust baths containing sodium fluoride, 1 per cent D.D.T., or 0·1 per cent gamma B.H.C. are also useful. As an alternative, the birds may be treated by sprinkling them with a powder containing D.D.T., B.H.C., or sodium fluoride.

Some of the modern insecticides mentioned before are useful in treatment of the other mite infections, but it is only very seldom that one is called on to treat such infections.

LICE

(*Phthiraptera*)

Lice are the commonest parasites of the fowl in the tropics and it is likely that if one bird in a flock is found to be infected, all the others are infected. They seldom cause any harm *per se*, but if the infection is heavy there is an insidious economic loss due to reduced production, feather picking, retarded growth, and anaemia, causing the condition referred to as lousiness. The common lice in poultry according to Emerson (1956) are:—

1. *Cuclotogaster heterographus*, the chicken head louse.
2. *Goniocotes gallinae*, the fluff louse.
3. *Goniodes dissimilis*, the brown chicken louse.
4. *Goniodes gigas*, the large chicken louse.
5. *Lipeurus caponis*, the wing louse.
6. *Lipeurus lawrensis tropicalis*, the tropical wing louse.
7. *Menacanthus cornutus*.
8. *Menacanthus pallidulus*.
9. *Menacanthus stramineus*, the chicken body louse.
10. *Menopon gallinae*, the shaft louse.
11. *Oxylipeurus dentatus*.

Description of all these lice and a key to their identification is given by Emerson (1956).

Recently, a severe infection of the fowl with *Linognathus setosus*, a louse normally found on dogs, has been reported from the U.K.

Life History.—This is direct and does not involve metamorphosis. Lice are permanent ectoparasites on their hosts and they are more or less host-specific though some are found on closely related hosts, while others are even restricted to certain regions of the body.

Eggs are operculated and when laid are cemented to the barbicules of the feathers of the host. The egg hatches out into a nymph which undergoes three successive ecdyses, the first and the second producing the second and the third stage nymphs respectively, and the third producing the adult which is sexually differentiated. The whole life history takes 2–3 weeks. Infection occurs by close contact, and equipment and personnel may transfer lice mechanically to their hosts.

Effects.—As remarked earlier, they produce a loss in egg production when the infection is heavy, and may cause irritation severe enough to cause death in chicks. This, however, is very rare. The virus of equine encephalomyelitis has been isolated from *Menacanthus stramineus*, the chicken body louse.

Control.—Lice are not very harmful. Treatment is called for when infection is heavy or troublesome, as it is sometimes in winter. The birds may be treated individually or flock treatment may be used.

Flock treatment is performed by painting the perches in the fowl house with strong tobacco extract containing 40 per cent nicotine sulphate, using about 400 ml. for every 50 metres of perch length. The fowl house is then closed on all sides but one, and the birds allowed to remain overnight, when the warmth of their bodies will cause the nicotine sulphate to volatilize and kill the lice. This treatment should be repeated on the tenth day. As an alternative, fumigation may be done, using B.H.C. with 1·5 per cent gamma isomer at the rate of 3 ml. per linear foot.

The University of Nebraska entomologists recommend spraying as follows: Co-Ral 25 per cent wettable powder (cumaphos) using 6 oz. in 1 gallon of water on 125 birds, or application of Co-Ral 0·5 per cent dust at the rate of 1 lb. per 25 sq. ft. over litter floor; Sevin 5 per cent dust at the rate of 1 lb. per 100 birds or per 40 sq. ft. of litter; or malathion 57 per cent concentrate using 10 oz. per gallon, using 1 pint of the solution per 150 sq. feet.

Dust containing 1 per cent D.D.T., 0·2 per cent B.H.C., or 0·5 per cent Sevin may be used individually on birds. Certain organophosphorous compounds, chiefly malathion and dibrom, used as a spray on the birds are very effective (Hoffman, 1961). Dibrom is used at a concentration of 0·025 per cent at the rate of 40 ml. per bird and malathion is used similarly in a concentration of 0·1 per cent in water.

CHAPTER VI

DEFICIENCY OR NUTRITIONAL DISEASES

DEFICIENCY diseases may be caused by a shortage of vitamins, minerals, and other nutritive factors such as proteins and amino-acids. In many cases, gross protein deficiency leads to malnutrition and starvation. A consideration of malnutrition is outside the scope of this section of the book. Therefore, only vitamin and mineral deficiencies will be dealt with under this heading.

VITAMIN DEFICIENCY

The normal vitamin requirements of poultry are given in *Table* 10. With the more intensive methods of poultry keeping, deficiencies of vitamins are likely to be common unless appropriate amounts of vitamins are added to mashes to compensate for the deficiencies in the foodstuffs used. These deficiencies may be due to fat-soluble or water-soluble vitamins.

FAT-SOLUBLE VITAMINS

Vitamin A Deficiency.—A deficiency of vitamin A causes night blindness, keratomalacia, and pityriasis in animals, and also an inability to withstand infections. Vitamin A is necessary for the maintenance of the epithelial cells in good condition. Thus, vitamin A deficient animals and birds become susceptible to respiratory and other infections. Deficiency also causes decreased growth-rate in all animals and birds, and lowered hatchability in birds.

CLINICAL SIGNS.—A deficiency of vitamin A in adult fowls causes nutritional roup characterized by conjunctivitis, oculonasal discharge, sticking together of eyelids, and the accumulation of cheesy material in the eyes and nasal sinuses. In acute cases in chicks there is slow growth, ruffled and harsh plumage, staggering gait, general incoordination, twisting of the tail, swollen eyelids, and high mortality. In advanced cases there is necrosis and keratinization of the mucosa of the alimentary and respiratory tracts. On the other hand, the disease may also be manifest as renal gout, characterized by accumulation of urates in the kidneys, ureters, peritoneum, and visceral surfaces of the abdominal organs.

Vitamin A deficient chicks become more susceptible to coccidiosis and worm infections (Randall, 1964). Egg production in layers goes down. Blood spots in the eggs increase. The yellow pigment in the comb and shanks disappears. There are embryonic malpositions and increased embryonic deaths. A deficiency of vitamin A impairs antibody production.

The diseased chickens, if examined at autopsy, will show white pustules or necrotic cheesy deposits in the oesophagus (*Fig.* 15), pharynx, and eyelids. There may be white deposits in the kidneys, pericardium, and other abdominal organs. This condition is sometimes erroneously referred to as visceral gout. Histological changes in the chicken due to vitamin A deficiency have been described by Aydelotte (1963).

Table 10.—VITAMIN REQUIREMENTS OF POULTRY

VITAMIN	REQUIREMENTS per kg. diet (air dry)								
	Chickens up to 8 weeks	Chickens 8–18 weeks	Broilers	Hens		Poults up to 8 weeks	Poults 8–18 weeks	Breeding Turkeys	Growing Ducks
				Layers	Breeders				
*Vitamin A (I.U.)	3000	3000	3000	5000	5000	5000	5000	5000	5000
Vitamin D_3 (I.U.)	250	250	300	500	500	900	900	900	300
Vitamin E (I.U.) (approx.)	15	10	10	8	10	12	10	15	—
Vitamin K (mg.)	1·0	0·4	1·0	0·4	0·4	1·0	0·8	0·6	—
Vitamin B_1 (mg.)	2·2	2·0	2·5	—	—	—	—	—	—
Vitamin B_2 (mg.)	4·5	3·5	5·0	3·0	4·5	5·0	4·0	4·5	5·0
Nicotinic acid (mg.)	35	30	40	—	—	70	60	50	50
Pantothenic acid (mg.)	12	10	14	8	11	14	12	14	12
Vitamin B_6 (mg.)	3·5	3·0	3·8	3·5	4·0	3·8	3·5	4·0	3·5
Choline (mg.)	1500	1250	1500	1100	1100	2000	1500	1500	1500
Vitamin B_{12} (μg.)	10	8	10	4	6	10	8	8	10
Biotin (mg.)	0·15	0·12	0·15	—	—	—	—	—	—
Folic acid (mg.)	0·8	0·6	0·8	0·3	0·4	1·2	1·0	1·0	—

* Less vitamin A is required when stabilized vitamin A preparations are used, e.g., 1500 I.U. for up to 8 weeks. Higher amounts should be used if oxidation is likely.

N.B. In cases where no figure is given, data is insufficient to allow calculation of exact values.

TREATMENT.—Fish-liver oil and greens are rich in vitamin A. Greens are rich in the precursors of vitamin A. The deficiency is best corrected by the addition of proper quantities of stabilized vitamin A feed supplements, and several are on the market. The vitamin A content of some important foodstuffs is given in *Table* 11.

Vitamin A is easily oxidized. Therefore, poultry rations that have been stored for a few months should not be used as the vitamin A content of such rations will

Fig. 15.—The oesophagus of a chicken with vitamin A deficiency, showing white pustules and cheesy deposits.

have deteriorated. For similar reasons, fish-liver oils that have been stored for some time will contain much less vitamin A than specified. Today, most manufacturers use antioxidants such as butylated hydroxytoluene (BHT), butylated hydroxy anisole (BHA), or Ethoxyquin to prevent oxidation of vitamin A and

Table 11.—THE VITAMIN CONTENT PER KILOGRAM OF SOME IMPORTANT FOODSTUFFS

FOODSTUFF	VITAMIN A (I.U.)	CAROTENE (mg.)	VITAMIN D (I.U.)	VITAMIN E (I.U.)	VITAMIN B_1 (mg.)	VITAMIN B_2 (mg.)	NICOTINIC ACID (mg.)	PANTOTHENIC ACID (mg.)	VITAMIN B_6 (mg.)	CHOLINE (mg.)	VITAMIN B_{12} (μg.)	BIOTIN (mg.)	FOLIC ACID (mg.)
Alfalfa meal, dehydrated	0	50–100	0	±100	3	10	30	20	9	1250	0	0·2	5
Barley	0	0	0	6	4	1·2	30	7	4	1000	0	0·1	0·6
Brewers' yeast, dried	0	0	0	0	25–100	25–75	±400	50–100	25–50	3000	0	1·5	9
Buckwheat	0	0	0	—	—	1·0	40	10	—	450	0	—	—
Carrots, fresh	0	60–100	0	5	0·5	0·4	2	2	1	—	0	—	0·05
Coconut meal	0	0	0	—	1	3	30	6	3	1100	0	—	1·0
Corn gluten feed	0	3	0	—	2	2·0	60	14	12	1300	0	0·2	0·2
Corn gluten meal	0	6	0	—	0·2	1·5	45	9	7	300	0	0·1	0·2
Corn, yellow	0	2	0	5	4	1·0	12	6	4	450	0	0·1	0·2
Cottonseed meal	0	0	0	—	4	4	30	10	5	2750	0	0·6	1
Cow's milk, fresh	200–1000	0·1–0·3	3–25	0·5	0·4	1·5	1	3	1	100	4	0·03	0·1
Fishmeal	—	0	—	0	0·5	6	60	6	3	1500	25–100	0·1	0·1
Grass, fresh	0	15–75	0	±20	1	2	7	2	2	200	0	0·05	1
Grass meal, dehydrated	0	50–100	0	±100	3	10	30	15	9	1250	0	0·2	5
Linseed meal	0	0	0	—	7	3	35	7	6	1400	0	—	3
Millet	0	0	0	—	1·5	1·0	25	10	3	450	0	0·1	0·15
Molasses	0	0	0	0	0	1	40	2	2	600	0	—	—
Oats	0	0	0	5	6	1·1	12	10	3	900	0	0·2	0·2
Peanut meal	0	0	0	—	7	4	150	50	6	1750	0	0·3	0·6
Potatoes, fresh	0	0	0	1	1	0·6	10	2	1	—	0	0·1	0·05
Rice bran	0	0	0	—	22	3	250	22	—	1000	0	0·3	2
Rice polishings	0	0	0	—	20	2	500	12	—	1000	0	0·5	0·4
Rice, rough	0	0	0	6	3	1·0	30	8	4	900	0	0·1	0·4
Rye	0	0	0	10	3	1·2	12	7	3	450	0	0·05	0·6
Sesame meal	0	0	0	—	3	3	30	6	6	1500	0	0·3	1
Skimmed milk, dried	0	0	0	0	4	18	10	30	5	1000	30	0·2	0·6
Soyabean meal	0	0	0	—	4	4	30	14	6	2750	0	0·2	0·6
Wheat	0	0	0	10	4	1·0	50	10	4	900	0	0·1	0·4
Wheat bran	0	0	0	15	8	3	50–100	20	12	1200	0	0·1	1·5
Wheat germ meal	0	0	0	100	18	5	25–50	12	12	3000	0	—	1·5
Whey, dried	0	0	0	0	4	25	10	40	5	1500	15	0·2	0·8

0 indicates that quantities are negligible for practical purposes.
In cases where no figure is given, the data are insufficient to allow calculation of exact values.
± indicates an approximate figure.

other labile vitamins. Vitamin E has a sparing action on vitamin A. Atkinson, Swanson, Couch, and Quisenberry (1963) showed that when an antioxidant is included in the diet the storage of vitamin A in the liver was high.

Vitamin D Deficiency.—A deficiency of vitamin D causes defective absorption and assimilation of calcium, leading to rickets.

CLINICAL SIGNS.—Vitamin D deficiency in adults causes the laying of thin-shelled eggs, reduced hatchability, leg weakness, and 'penguin' sitting posture. The beak, claws, and ribs become very pliable. A characteristic feature is the bending of the keel bone. Costochondral junctions may be beaded. In the tropics, deficiency of vitamin D is not a great problem as birds can synthesize this vitamin in the presence of abundant sunlight. In extreme cases, however, if they are in deep litter or batteries without any access to even diffused sunlight, vitamin D deficiency may result. When the diet contains inadequate levels of calcium and phosphorus, the requirements of vitamin D are raised considerably.

Fish-liver oils are rich sources of vitamin D. However, most of the vitamin-containing feed additives have enough vitamin D to offset any natural shortages. Vitamin D in excess can be harmful.

Vitamin E Deficiency.—The effect of vitamin E deficiency has been studied in greater detail in domestic poultry than in other species of birds or animals. Vitamin E is a powerful antioxidant and has a sparing action on vitamin A. Its physiological antioxidant action in the tissues is simulated by other substances such as selenium, cystine, and some chemical antioxidants. Vitamin E is unstable under tropical conditions and its oxidative destruction is enhanced by minerals and unsaturated fatty acids.

CLINICAL SIGNS AND IMPORTANT LESIONS.—These vary according to the syndrome manifested.

Encephalomalacia.—This is also referred to as crazy chick disease and birds up to 8 weeks old are usually affected. The affected chicks are either sleepy, or highly excitable, and there may be twisting or retraction of the head and neck, and some may fall down and exhibit bicycling movements. If the brains of the affected chickens are examined, haemorrhagic areas of softening, often greenish-yellow or greenish in colour, may be seen, chiefly over the cerebellum (*Fig.* 16). Encephalomalacia can also be produced experimentally by feeding high levels of unsaturated fatty acids, linoleic and arachidonic, in the absence of vitamin E or other antioxidants. Selenium increases the protectiveness of vitamin E against encephalomalacia. Keratinoid pigment may also have some effect in reducing the incidence of encephalomalacia in field cases.

Exudative Diathesis.—This is commonly seen in chickens from 2 to 8 weeks old. There is a sudden loss of condition with fluid accumulations under the wings, abdomen, and other areas. There may also be haemorrhages in the heart, liver, brain, subcutaneous tissues, and fat. This can be prevented by addition of sodium selenite or other organic selenium compounds and 0·1 p.p.m. of selenium prevents the condition (Machlin and Gordon, 1962). This syndrome and muscular dystrophy can also be produced by molecularly distilled unsaturated fatty acid esters or triglycerides from fish oil.

Nutritional Muscular Dystrophy.—This is usually seen in older birds, about 4 months of age. Broilers or rapidly growing birds are more susceptible. The clinical signs are vague and consist of depressed growth and increased mortality rates. The deficiency is usually diagnosed at autopsy and is characterized by the presence of white streaks of degeneration in the breast and other skeletal muscles, as well as on the muscular tissue of the heart and gizzard. This can also be produced by a diet deficient in methionine or cystine, particularly in the presence of high levels of arginine and linoleic acid. This condition can be prevented if adequate levels of vitamin E, an antioxidant, or sulphur-containing amino-acid, are present in the diet.

TREATMENT AND PREVENTION.—Wheat-germ oil is the richest source. Alfalfa meal, greens, germinated pulses, and fish meals are rich sources. Selenium has some sparing action on vitamin E. Treatment of badly affected cases showing

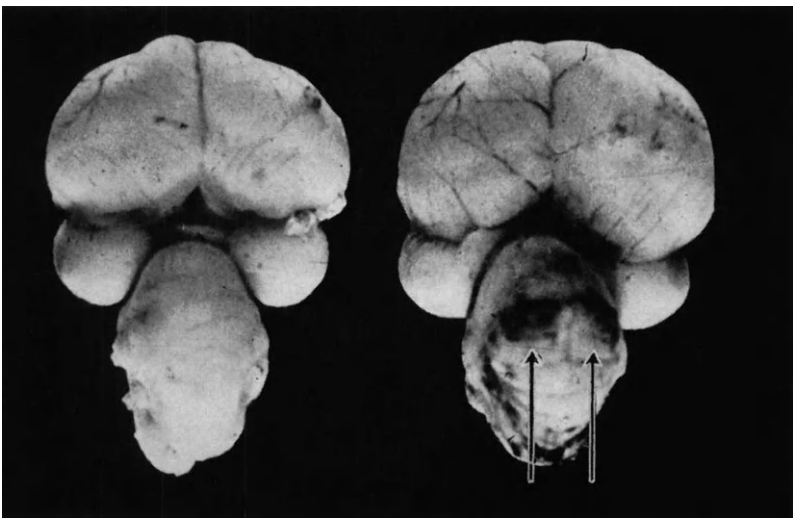

Fig. 16.—The brain of a chicken showing haemorrhagic areas of softening in the cerebellum (arrowed). A normal brain is on the left for comparison.

nervous symptoms is not worthwhile. A single injection of 30 mg. of vitamin E may cure an acute case not having nervous signs. The disease is best prevented by using the foods rich in vitamin E mentioned earlier, or by using a feed additive such as Rovimix E which contains very large quantities of vitamin E. Antioxidants have an action in many respects similar to vitamin E. It has been demonstrated that if sufficient dietary selenium, methionine, or cystine, and an antioxidant are provided the young chick will apparently show no need for dietary vitamin E.

Vitamin K Deficiency.—Vitamin K is required for the synthesis of prothrombin which plays an important part in the clotting mechanism. The richest sources are the green leaves of plants such as grasses. Certain drugs such as sulphaquinoxaline may increase the requirements of vitamin K. This vitamin appears to have a protective effect in coccidiosis. Vitamin K is also required for normal embryonic development and a deficiency may cause an increase of blood spots in eggs.

CLINICAL SIGNS.—The deficiency occurs at 2–3 weeks and is characterized by the appearance of large subcutaneous haemorrhages on the body (blood blisters) and in the abdominal cavity. Blood-clotting time is prolonged. Anaemia may also be seen.

TREATMENT.—Administration of vitamin K or K_3 (menadione sodium bisulphite, which is a synthetic vitamin K) cures the condition.

WATER-SOLUBLE VITAMINS

Vitamin C Deficiency.—A disease termed 'battery sickness', characterized by paralysis and atrophy of the striated muscles has been reported in hens kept 3–4 months in batteries in Germany. This is considered to be due to vitamin C deficiency. Intramuscular injections of vitamin C at the rate of 100 mg. are said to cure nearly 100 per cent of the affected birds (Polster, 1963).

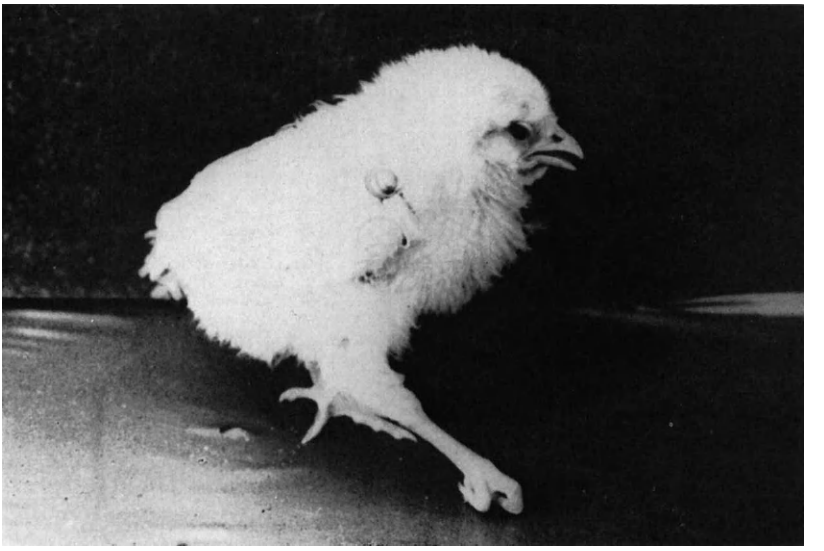

Fig. 17.—A chicken showing curled toe paralysis due to Vitamin B_2 (riboflavin) deficiency.

Thiamine (Vitamin B_1) Deficiency.—Thiamine is necessary for the proper metabolism of carbohydrates. Deficiency of this vitamin causes extreme loss of appetite, polyneuritis, and death. This vitamin is found in abundance in cereal grains and therefore it is not often a problem in poultry nutrition. However, it is easily destroyed by heat in an alkaline or neutral medium, and also by an enzyme, thiaminase, present in fish meal.

CLINICAL SIGNS.—There is usually paralysis of the muscles beginning with the toes. The affected chicken sits on the flexed legs and draws the head back producing a star-gazing attitude which is considered typical of the condition. There may be atrophy of the heart muscles and dilatation of the auricles.

The addition of adequate quantities of rice polish, wheat bran, yeast, or commercially prepared vitamins will cure this deficiency.

Riboflavin (Vitamin B_2) Deficiency.—Vitamin B_2 plays an active part in over a dozen enzyme systems and thus plays a vital role in metabolism. It is known as the yellow oxidation enzyme. This is a water-soluble, heat stable vitamin which may be destroyed by sunlight in an alkaline medium. Milk, grasses, and brewers' yeast are rich sources of this vitamin.

CLINICAL SIGNS.—A deficiency of this vitamin causes diarrhoea and 'curled toe paralysis' (*Fig.* 17). Crusty deposits may be seen over the eyelids and mouth. In the case of adults, there is decreased egg and antibody production, increased embryonic mortality, and dead in shell chicks, with dwarfing and clubbing-down of the feathers because they fail to rupture their sheaths and come out normally. There is degeneration of the myelin sheaths and retardation of growth. The embryo mortality may reach a peak between the eighteenth and twentieth day of incubation. The embryos show a clubbed down condition, which is also associated with dwarfing, curling of the toes, and parrot beak condition.

TREATMENT.—Add 4 per cent brewers' yeast to the mash.

Pantothenic Acid Deficiency.—Pantothenic acid is a vitamin component of coenzyme A, which is concerned in carbohydrate, protein, and fat metabolism. Yeast, liver, meat, and certain cereals are rich sources of this vitamin.

CLINICAL SIGNS AND PATHOLOGY.—A deficiency of this vitamin causes the appearance of scab-like lesions at the angles of the mouth and on the borders of eyelids, which are often stuck together with the accumulation of pus, or the formation of scabs often referred to as exfoliating dermatitis. Deficiency of this vitamin also impairs antibody production. There is retardation of feather growth, the appearance of broken feathers, and dermatitis.

Cracks and fissures appear between the toes, followed by wart-like protuberances on the balls of the feet (*Fig.* 18). Egg production and hatchability are reduced. The oral cavity may present a pus-like substance and the proventriculus a greyish-white exudate.

Embryos that develop from eggs laid by hens on a pantothenic acid deficient diet show a 'stunted chick syndrome'.

Niacin (Nicotinic Acid) Deficiency.—This is a stable member of the B complex group of vitamins which plays a part in carbohydrate, fat, and protein metabolism. Certain cereals, yeast, and fish meal are good sources of this vitamin.

CLINICAL SIGNS.—A deficiency of this vitamin causes enlargement of the hock joint with bowing of the legs, as in perosis caused by manganese and choline deficiencies, but unlike the latter condition, the achilles tendon rarely slips from the condyles. Poor feathering and inflammation of the tongue and mouth (black tongue) may be additional signs of deficiency.

Pyridoxine (Vitamin B_6) Deficiency.—Pyridoxine is necessary for the proper metabolism of amino-acids, the formation of enzymes concerned with deamination, transamination, and decarboxylation of amino acids.

This is a stable member of the B complex group of vitamins, present in cereal grains, yeast, and animal products. A deficiency of this vitamin rarely occurs under normal conditions, and if a deficiency is present it is characterized by spasmodic convulsions in chicks, which are more marked than those encountered in

encephalomalacia. Loss of appetite, loss of weight, reduced egg production, and hatchability are other signs encountered in deficiency. Added choline at levels over 200 mg. per lb. of feed can produce signs of pyridoxine deficiency in broiler chickens, which can be prevented by giving extra pyridoxine.

Biotin Deficiency.—Biotin is a sulphur-containing member of the B complex group present in fresh greens, liver meal, and yeast. Deficiency causes dermatitis and poor hatchability, as in pantothenic acid deficiency, and perosis, as caused by choline and manganese deficiency. There may be dermatitis of the soles of the feet, with bloody or necrotic toes.

Fig. 18.—The feet of a chicken suffering from pantothenic acid deficiency, showing wart-like protuberances on the balls of the feet.

Choline Deficiency.—Choline is present in acetyl choline. Fish meal, liver meal, and yeast are rich sources. A deficiency of this causes perosis, which is initially characterized by puffiness around the hock joint, followed by flattening of the tibiometatarsal joint and twisting and bowing of the metatarsal with slipping of the achilles tendon from the condyles. The addition of 2–4 per cent brewers' yeast will correct deficiencies caused by lack of niacin, pyridoxine, biotin, and choline. A condition similar to perosis may be seen in spondylolisthesis, which is characterized by weakness, paresis, and paralysis. This condition is becoming increasingly common in broilers (Osbaldiston and Wise, 1967a). The aetiology of this condition is not known. It may be due to one of several factors such as heredity, infection, nutritional, or metabolic causes.

Folic Acid Deficiency.—Folic acid is a stable member of the B complex group of vitamins which forms a part of the enzyme systems concerned in single carbon

metabolism. Yeast and greens are rich sources of this vitamin. It can be synthesized by the bird and this synthesis becomes easy when the carbohydrate content of the ration is in the form of starch.

Deficiency is characterized by straight-neck paralysis in turkey poults. There is macrocytic anaemia which is due to megaloblastic arrest of erythrocyte formation in the bone-marrow, and agranulocytosis. Together with lysine, it is required for pigmentation of feathers. There is also an increased embryonic mortality, bending of the tibiotarsus, and deformation of the neck.

Cyanocobalamin (Vitamin B_{12}; Anti-pernicious Anaemia Factor) Deficiency.—This is a cobalt-containing member of the B complex group of vitamins, which is concerned in nucleic acid synthesis, methyl synthesis, carbohydrate metabolism, fat metabolism, and maintenance of blood-glutathione levels.

This can be synthesized by bacteria and is found in all foods of animal origin. (It was earlier referred to as animal protein factor.) Fish meal, meat meal, and milk are rich sources of this vitamin.

CLINICAL SIGNS.—There are no characteristic signs of deficiency of this vitamin. However, there is poor growth and feathering, with high embryonic mortality which reaches a peak on the seventeenth day of incubation, myoatrophy of legs, and haemorrhages in the allantois of the embryo. There is kidney hypertrophy with increase of non-protein nitrogen in the blood.

TREATMENT.—A single injection of 3 μg. of vitamin B_{12} per chick will cure the condition. As an alternative, 4 mg. of vitamin B_{12} may be added per ton of feed.

MINERAL DEFICIENCIES

The common deficiencies of poultry due to minerals are those due to lack of calcium, phosphorus, manganese, and zinc.

Calcium and Phosphorus Deficiencies.—These minerals are necessary for bone formation. The proper ratio of calcium to phosphorus is 2:1 for laying hens and at least 40 per cent of the phosphorus must be in ionizable form. Oyster shell and soluble grit, such as limestone, are very good sources. Gravel and sand are not suitable. A deficiency of these with a deficiency of vitamin D produces rickets characterized by the signs and lesions described under vitamin D deficiency. The claws become soft and the beaks longer. There may be a 'rachitic rosary' type of lesion. Paralysis is a common sign of calcium deficiency.

TREATMENT.—Correct the deficiency. Incorporate 4 per cent shell grit in the ration or keep separate containers with shell grit.

Manganese Deficiency.—Manganese is present in wheat products, oats, and soyabean meal. It is an essential trace element.

A deficiency causes perosis characterized by enlargement of tibiometatarsal joint, with twisting and bending of the distal end of the tibia and the proximal end of the metatarsus, and slipping of the gastrocnemius tendon from the condyles. Perosis can also be caused by deficiencies of choline, biotin, and niacin.

There is chondrodystrophy characterized by shortening of the long bones and overall dwarfing of the long bones. The shell shows poor calcification.

The embryos show thickened legs, short wings, and parrot beak. The normal requirements of manganese are 50–60 parts per million of the ration.

PREVENTION.—Provide 25 mg. of manganese sulphate per lb. of ration. Treatment is not worthwhile.

Zinc Deficiency.—Zinc deficiency occurs fairly widely in poultry on practical rations. The age of the bird, the quantity and availability of zinc in chicken cages, and other factors affect the requirements. Deficiency is more likely to occur in the young, particularly in chicks hatched from eggs laid by deficient hens. Generally zinc deficiency is not severe; however, even moderate deficiencies lower the rate of growth, feed efficiency, and egg production. Poor feathering, brittle feathers and bones, scaly skin, stiff gait, and weakness in legs are the associated symptoms in a deficiency. Hatchability is also affected particularly on a high calcium diet. The normal requirements of zinc are 30–50 parts per million of the ration. Phytic acid markedly reduces the biological availability of zinc.

Selenium Deficiency.—A deficiency of selenium causes a myopathy of the smooth muscle of the gizzard, myocardium, and skeletal muscles, and retardation of growth. The minimum requirement of selenium to prevent these changes appeared to be 0·1 p.p.m. of selenium selenite in the presence of adequate quantities of vitamin E and methionine (Scott, Olson, Krook, and Brown, 1967). However, in the absence of vitamin E the requirement appeared to increase and varied from 0·18–0·28 p.p.m.

CHAPTER VII

MISCELLANEOUS DISEASES

VISCERAL GOUT

THIS condition is due to primary renal damage and can be caused by a variety of factors, such as deficiency of vitamins A or E, high intake of protein, or the impairment of kidneys by some toxic substance, especially artificially degraded animal protein. A similar syndrome may be seen in avian monocytosis and infectious bronchitis virus infection.

The condition is characterized by excess of uric acid in the blood, and therefore the deposition of urates on the tubuli of the kidneys, or the visceral surface of the liver, the pericardium, abdominal air sacs, and the joints, in which case there is arthritis.

Treatment.—Determine the cause and rectify the defect. Magnesium sulphate in drinking-water for 1–3 days may be helpful.

FEATHER PLUCKING AND CANNIBALISM

This may start due to deficiency of protein, particularly the amino-acid arginine, or due to overcrowding or injuries. Later it may become a vice. Some are of the opinion that it may be due to manganese deficiency or to parasitism.

Prevention.—The cause has to be determined and removed; the amount of fish meal may be increased if it is low; debeaking will be useful. It is also desirable to detect the worst offenders and cull or debeak them. Being a vice, it spreads very rapidly and unless appropriate action is taken, may cause alarming losses. Any pecked bird should be removed, for if it is left it is likely that the bird will be pecked to death. Often, reducing the duration of illumination will have a beneficial effect in controlling cannibalism. The optimum is regarded as 8–12 hours. In India and Ceylon, margosa oil is applied to the injured parts to prevent myiasis and also as a repellant.

CHILLS

These are due to mechanical causes such as inadequate heating and draughts. Post-mortem picture reveals unabsorbed yolk-sac, distended gall-bladder, congested lungs, nephritis, empty crop, and fibrous material in the intestines.

Treatment.—Improve management and provide adequate heat.

BUMBLE FOOT

This is characterized by a hard swelling on the sole of the foot and this swelling, if opened, usually reveals a cheesy or hard fibrous mass which can be easily expelled.

This is caused by damage to the sole, continuous friction on the sole, or the entrance of a foreign body with bacteria which set up a focus of infection. Rough perches and bad litter may also cause this condition. It can also be caused experimentally by *Brucella abortus*.

Treatment.—The swelling may be opened, the cheesy or whitish material removed, the area cleaned with tincture of iodine or a suitable antiseptic, the wound can then be plugged with sulphonamide powder or cotton wool soaked in a disinfectant, and the leg bandaged tight.

Care should be taken to avoid excessive bleeding while opening the swelling. The bleeding can usually be controlled by the application of pressure.

EGG EATING

This is a vice which usually starts with one or two birds eating broken or damaged eggs. Soon this is learned by the others and the vice spreads rapidly and becomes problematic unless checked in time.

To control this vice the following actions will be helpful:—

1. Cull out the worst offenders before the vice spreads.
2. Increase the calcium and the protein in the diet to produce strong shells and reduce the birds' craving for protein.
3. Provide sufficient nest space and collect eggs at regular intervals. Collect all eggs that are laid on the floor as soon as possible.

ARTHRITIS

This is usually caused by infection of the joints due to various bacteria, chiefly *Mycoplasma* spp., and is usually characterized by swelling of one or more joints and lameness. Arthritis is also seen in the chronic form of fowl cholera. Arthritis and synovitis in pullets may also be caused by *Staphylococcus aureus*, and is characterized by stiffness, lameness, and loss of condition (*Fig.* 19). There is tibio-tarsal periarthritis, tendonitis, and synovitis.

Treatment.—Not worthwhile.

PARESIS, PARALYSIS, AND LAMENESS

Paresis, paralysis, and lameness are due to a variety of causes, the chief of which are vitamin B_1 or vitamin B complex deficiency, neural Marek's disease, arthritis, chronic cases of Newcastle (Ranikhet) disease, perosis, rickets, gout, rupture of the tendo achillis, and a variety of other factors. Birds may also show signs of leg weakness in bird malaria, spirochaetosis, and in the terminal stages of many acute diseases.

Treatment.—Before any treatment is attempted one should determine the cause, and if several birds are paralysed simultaneously, it is best to conduct a thorough investigation to ascertain the cause.

CURLED TONGUE

This is seen in chicks fed mash deficient in certain amino-acids such as leucine, isoleucine, and phenylalanine. The condition is characterized by upward or

downward curling of the tongue. In the case of turkey pullets, it is said to be caused either by genetic factors or by feeding a dry mash of fine physical consistency during the first few weeks of life.

TRAUMATIC VENTRICULITIS

This has been seen occasionally when sharp objects which penetrate the gizzard are ingested by the chicks. It is diagnosed at autopsy. Often there is secondary peritonitis unless the infection caused by the penetrating object is walled off.

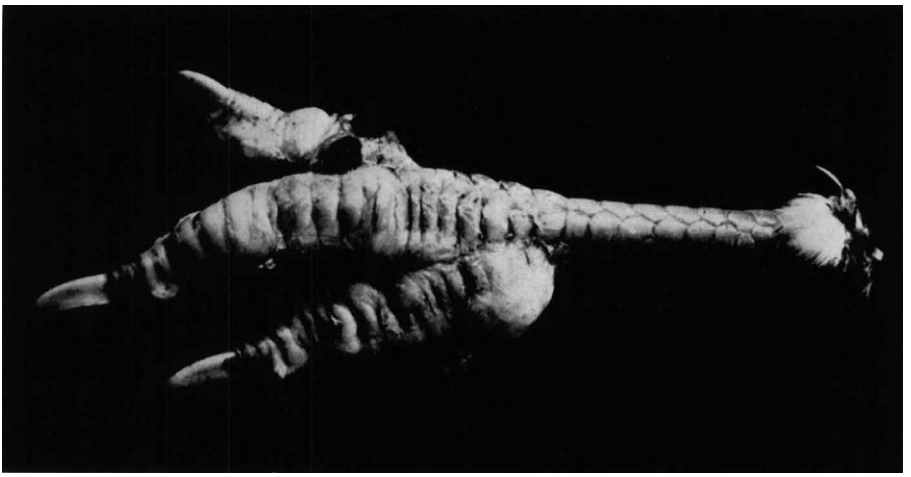

Fig. 19.—The toes of a fowl with gout showing arthritis and distension of the toes due to deposits of urates.

VESICULAR DERMATITIS

This is characterized initially by the appearance of vesicles on the toes, foot, and comb. Soon they coalesce and rupture, and are followed by lesions of necrosis and sloughing. It can be caused by the ingestion of grains contaminated with certain fungi, and photosensitization followed by the ingestion of certain seeds. *Staphylococci* may play a secondary role.

XANTHOMATOSIS

This condition is characterized by swellings on comb, wattles, and skin, which contain a honey-coloured transudate. It is seen most frequently in white leghorns when they reach maturity at 6–7 months of age. Soon the swellings become firm and nodular, containing cholesterol deposits. The cause of the condition is unknown. Carcases of affected birds are not suitable for food.

VENT GLEET

This condition is seen in laying hens and is characterized by the accumulation of pasty whitish or greenish-white material round the cloaca giving rise to an obnoxious smell. Usually only one or two birds are affected at a time.

The exact cause of this condition is not known but it appears to be infectious in nature. The affected birds usually show some degree of nephritis and the

carcases give a highly ammoniacal uraemic odour. This condition may be seen in nephritis or may occur due to any factor causing a high level of blood-urea, such as visceral gout.

Treatment.—Various lines of treatment have been advocated. However, satisfactory results may be obtained by cleaning the vent well with a 1 per cent solution of Cetavlon. Administering a sulphonamide or preferably a tetracycline in drinking-water containing 1 oz. of mag. sulph. per gallon and giving a low protein diet will be useful.

SINUSITIS

This is caused by infection of the sinuses of the head with pathogenic micro-organisms and in acute cases is characterized by swellings around the eyes. Usually this occurs secondarily to C.R.D., infectious coryza, or other diseases which affect the respiratory system.

Treatment.—This depends on the primary cause. Opening of the sinuses, expulsion of the cheesy material, and injection of antibiotics such as tetracyclines (Terramycin, Aureomycin) into the sinuses, or into the muscles, proves useful. However, for economic reasons it is not advisable to treat these chronic conditions.

EMPHYSEMA

This is accumulation of air in the subcutaneous tissues. In birds, this is not an uncommon condition as it is caused by the rupture of one of the several air sacs which give buoyancy to the avian species. The air then enters the subcutaneous spaces of the neck and thorax causing distinct bulges which give a drum-like resonance when percussed. The condition is rather distressing to the affected bird and it may be relieved by making a small incision when the accumulated air immediately escapes. All the air can be expelled by applying gentle pressure. The condition is likely to recur, for often when the incision closes air accumulates again. This may be prevented by applying a pressure bandage over the affected areas. However, this procedure is not practicable in most cases. Birds that show extensive emphysema should be destroyed.

PROLAPSE OF THE OVIDUCT

In this condition the oviduct protrudes partially or completely from the cloaca and is seen as a red mass at the cloacal region. This is seen commonly during the first few months when the birds come into production. If the birds with prolapse are not removed, others are likely to peck them, thus leading to cannibalism and causing the intestines to come out. The exact cause is not known. However, it is likely to be due to some irritation or inflammation of the cloaca or oviduct or too large an egg. Excessive lighting which can overstimulate the pituitary may induce excessive secretion of oestrogenic hormones which in turn may relax the attachments of the oviduct. Hereditary predisposition may play an important role in the aetiology. Certain flocks, especially high egg-producing strains, are more susceptible to it.

Treatment.—The birds with prolapse should be removed, culled and, if found fit, used for table. If the prolapsed part is pushed back it is likely to come out

again unless a purse-string suture is placed round the cloaca. However, a common sequel to such an operation is peritonitis. Thus, treatment is often not economical.

CAGE LAYER FATIGUE
(*C.L.F.*)

As the name implies, the condition is seen in those reared by battery system of management, even if they are on an adequate diet of calcium. The exact cause is not known though it is likely that a genetic predisposition plays a part, whereby the regulatory mechanism that controls the level of bone and blood calcium in relation to the secretion of egg-shell calcium is not efficient. Recent evidence indicates that cage layer fatigue is inversely proportional to the cage size (King, 1965).

Two forms of the disease are recognized—the peracute form where the birds die suddenly without any characteristic signs and the acute form where the bird falls on the side soon after laying and develops flaccid extension of the legs. These birds may recover completely on being fed or may die.

Diagnostic signs in these cases are the great flexibility of the bones which bend like a green stick, the puckering of the ribs at the costochondral junctions (producing the rickety rosary appearance) and the thinning of the bones which become osteoporotic.

Increase of calcium level in the ration may be helpful in preventing the condition.

BREAST BLISTERS

These are false bursae on the surface of the sternal region caused by mechanical pressure or injury to this region. They are commonly seen on broilers, and cause considerable economic losses due to down-grading of carcases. In the American broiler industry they cause a loss of 12 million dollars per annum, and in the U.K., 5 per cent of broiler carcases are down-graded due to this defect (Cherry, 1967). They can be treated surgically by incision and expressing the contents, or by excision. Recent evidence indicates that they are caused by damp shallow litter and that roosts predispose the birds to acquire them (Wisman and Beane, 1965). It is likely that they are caused by repeated localized mechanical pressure. The thickness of the skin over the keel bone and its prominence may have a direct relationship to the condition. They are more common in the male and in heavy birds.

CROOKED TOE DEFORMITY

This is usually seen in chicks and turkey poults when they are 2–4 weeks of age or older. The toes turn inwards or sideways, and it is more common when no perches are provided. There appears to be a hereditary predisposition to the condition. Affected birds in most cases move freely. The condition is not associated with a paralysis.

No treatment is possible. It is desirable to cull the affected birds. The condition must be distinguished from curled toe paralysis where the toes turn downwards and inwards, and which is associated with vitamin B_2 deficiency.

RUPTURE OF THE GASTROCNEMIUS TENDON

This is seen in some domestic fowls as well as turkeys, as they approach sexual maturity, and it causes unilateral or bilateral lameness. The site of rupture is marked by a greenish-blue discolouration or nodular thickening about 1 in. above the hock joint. The exact cause is not known. The affected birds should be culled. However, it is likely to be primarily due to inherited factors, the time of rupture being influenced by gonadal activity.

FATTY LIVER AND KIDNEY SYNDROME

This condition has been described by Hemsley (1965) in the broiler flocks in the U.K. where it is now the most common cause of death in the 2–4-week age-group. It appears to be caused by an interaction of factors carried by the chick and factors in the environment and to be closely related to Gumboro disease.

Usually birds between 2 and 3 weeks are affected, and the mortality may reach 5 per cent. The disease lasts about 5 days in a flock, and the best grown birds are more commonly affected. Some chicks may show trembling and paralysis of the legs. The lesions are characteristic, and consist of a pale or pink carcase, and pale and swollen kidneys and liver, where the cells are degenerated and contain excess of fat droplets.

This has to be distinguished from the following—Gumboro disease, where the bursa of Fabricius is swollen; chick oedema disease, where the carcase is oedematous; and infectious avian nephrosis, where the kidneys are swollen and contain uratic deposits in the kidney tubules.

SIX-DAY CHICK DISEASE

The disease is characterized by mortality in chicks reaching a peak by the sixth to eighth day. Chickens become listless, dull, and droopy. It is said to be caused by certain strains of *Clostridium welchii* (McGaughey, 1959). The disease is essentially seen in the presence of bad management. Overcrowding leads to massive contamination of litter and feed troughs. McGaughey (1959) reported this disease in Ceylon, drawing attention to reports of a similar disease in Japan and to his investigations in Cambridge in England.

Cause.—The exact cause is not known. It is generally believed that certain non-toxigenic strains of *Clostridium welchii*, which produce a heat-stable substance, possibly a histamine, are responsible for the disease. Others state it is due to a deficiency of available energy in all-mash chick starter rations.

Clinical Signs.—The disease is seen between the fourth and fourteenth day of life, causing a peak mortality between the sixth and eighth day. Mortality may reach 30 per cent and deaths occur suddenly. Birds found healthy are seen next morning to stagger and show incoordination of movement or coma. Others may show increased thirst and discharge from the nostrils.

Post-mortem Lesions.—The liver is congested and mottled, the gall-bladder is invariably distended, and uratic deposits may be seen in the kidneys. The caeca and intestines contain bubbly soft faeces.

Prevention and Treatment.—As the exact cause is not known, definite methods cannot be laid down for the prevention of the disease. However, changing the

litter, improving hygiene, and preventing overcrowding of birds might be helpful. Administration of penicillin in the feed and increase of the carbohydrate in the ration are recommended.

BLOOD SPOTS IN EGGS

The exact causes of this condition are not definitely known. However, under different conditions, different factors operating together or individually may cause the appearance of blood spots in eggs. Some of these factors are hereditary causes, deficiency of vitamin K, certain forms of poisoning, such as the ingestion of rodenticides or fungal toxins, or deficiency of vitamin C.

The administration of copper sulphate at the rate of 10 mg. per hen in drinking-water for 35–80 days is said to reduce blood spots in eggs.

INFECTIOUS KERATOCONJUNCTIVITIS

This disease was first reported in chickens by Coles from South Africa (1940) where it was found to be caused by a *Rickettsia*-like organism which is now known as *Ricolesia conjunctivae*. A similar condition has been described from time to time in Russia, Denmark, and Europe. It seems to be common in Russia where it is probably caused by a virus or virus-like agent producing inclusion bodies. Non-infectious keratoconjunctivitis may be caused by ammonia gas irritation (Carnaghan, 1958). This can be prevented by sound management and providing adequate ventilation. Recovery is always slow. Several outbreaks of a similar disease have been seen by the author in Ceylon. The disease was seen in birds aged less than 3 months. The average incubation period appeared to be 4–6 days. The disease was transmissible. Only the eyes were visibly affected. In the clinical cases, one eye was affected first and, in a few days, infection spread to the other eye. Usually, in advanced cases, the eyelids stuck together and opacity of the cornea was noticed in some cases. If there was no bacterial infection, recovery took place in 2 weeks. However, the majority of the infected birds showed retardation of growth. Morbidity was about 20–30 per cent and mortality was usually below one per cent.

Rickettsia-like organisms could not be demonstrated in the scrapings of the conjunctival sac. The infection could be transmitted to chickens by suspending the conjunctival scrapings in saline containing penicillin and streptomycin. Oxytetracycline in feed and drinking-water had no appreciable effect on the course of the disease. These results would indicate that a virus or virus-like agent is the cause of the disease. In Russia, inclusion bodies have been demonstrated in conjunctival epithelial cells.

HAEMORRHAGIC SYNDROME

A syndrome characterized by multiple haemorrhages in the body, the serous membranes, and the liver, has been described from time to time by several workers. The bone-marrow in most instances is pale and fatty, and abnormal thrombocytes are constantly found in blood-smears. They are enlarged, circular, and vacuolated.

This could occur from a variety of causes such as acute fowl cholera, Newcastle disease, and fowl plague, and may be seen occasionally in certain birds for no obvious reason. On post-mortem examination of such birds, oedema of the lungs

is seen frequently. The exact cause is not definitely known. However, it is most likely from the various reports made from time to time in the literature, that it is due to various causes and that some of these outbreaks were due to multiple factors. Some of the well-recognized causes that may lead to a haemorrhagic syndrome are:—

1. Sulphonamide toxicity, particularly caused by continuous feeding of sulphaquinoxaline at levels higher than those recommended.
2. Vitamin K deficiency. Administration of therapeutic doses of sulphonamides on a vitamin K deficient diet can cause multiple haemorrhages.
3. Vitamin E deficiency.
4. Mycotoxicosis caused chiefly by aflatoxin found in *Aspergillus flavus*, which was found to be a common contaminant in Brazilian groundnut cake in the past. Other fungal toxins can also produce a similar syndrome (Forgacs, Koch, Carll, and White-Stevens, 1962).
5. Carr (1962) has reported that Rous I sarcoma virus can produce a haemorrhagic syndrome. Different viruses may produce a haemorrhagic syndrome in birds and in South East Asia it could be caused by *Leucocytozoon caulleryi* infection.
6. Recent evidence indicates that certain coliforms are associated with this syndrome. Selenium and vitamin E may play an important role, at least under certain conditions, in the exudative diathesis (white muscle) syndrome.

The 'Bangkok haemorrhagic disease' of chickens (Campbell, 1954) and a similar disease described recently in Kerala State in India by Sivadas, Nair, Rajan, and Ramachandran (1965) also cause a haemorrhagic syndrome characterized by multiple haemorrhages. These workers were able to demonstrate rod-shaped organisms resembling *Besnoitia jellisoni* in the blood of a few hens before death. According to some authorities (Akiba, 1960) 'Bangkok haemorrhagic disease' may be a form of *Leucocytozoon* infection.

In attempting to control outbreaks of the haemorrhagic syndrome it is necessary to determine the cause and eliminate it.

ROUND HEART DISEASE

This is a disease of unknown aetiology reported from several countries where the prominent post-mortem lesion is an abnormality in the heart where it appears rounder than normal. The heart is often enlarged with a rounded apex which often has an indentation. The myocardium has a parboiled appearance with yellowish linear streaks. This may be associated with fatty degeneration, and necrosis of the cardiac muscle and haemorrhages. Several possible causes have been suggested, such as hereditary factors and sulphonamide toxicity. Wilson (1957) states that this is commonly seen in birds kept on built-up litter and that it appears to be due to an infectious agent. Most outbreaks occur during the first 3 months of production in pullets. It is possible that the disease is caused by a fungal toxin in the litter.

CHICK OEDEMA DISEASE
(Toxic Fat Disease)

This disease, first reported in 1957, is characterized by reduced weight gain, and accumulation of fluid in the pericardium, abdominal cavity, and subcutaneous tissues and is probably caused by one or more toxic factors (Flick, Douglass, and

Gallo, 1963). Recent evidence indicates that 3 toxic factors are responsible for the condition; 2 of these appear to be chlorinated hydrocarbons which are probably contaminants introduced during the processing of fat. The disease is characterized by depression, collapse, gaping, and oedema and may cause up to 100 per cent mortality. Chick oedema factors will also cause a decreased hatch and embryonic deformities. Over 50 parts per billion of the chick oedema concentrate appear to be toxic (Flick, O'Dell, and Childs, 1965). Chlorinated biphenyl (CBP) fed to cockerels at levels of 200 or 400 parts per million in the ration for 3 weeks can also produce this condition. These are hexachlorohexahydrophenanthrenes. Oedema may also be caused by poisoning with common salt.

MANDIBULAR NECROSIS

Mandibular necrosis of cockerels is associated with ulceration of the epidermis and dermis of the lower beak leading to necrosis and sloughing. This is probably caused by an inherent defect in the beak facilitating impaction with food. This usually occurs if wet mash is fed. In severe cases, the mandible sloughs off and the tip of the tongue undergoes necrosis. The condition can be prevented by giving pelleted food (Barr, 1965).

CONGENITAL HEART DISEASE

This appears in certain flocks of broilers and is characterized by the presence of endocardial papillae, mainly in the right ventricle, and especially in association with the right atrioventricular valves (Siller and Hemsley, 1966). Fowls appear to be unique in their ability to develop an apparent compensating mechanism in the form of 'lips' which surround the defects. Clinically, this condition is of no significance.

TRANSIENT PARALYSIS

A transient paralytic condition of 12-week-old pullets has been described from France (Willemast, Montlaur, Verger, Labrousse, and Antony, 1967), characterized by decreased tonus of the extensor muscles of the head, neck, wings, and legs. The condition is sporadic and spontaneous recovery usually follows. The aetiology is unknown. This condition is now relatively common in the U.K. occurring in pullets between 12 and 18 weeks.

It affects growing birds from 6 to 18 weeks old, and is characterized by sudden flaccid paralysis of the neck and limbs and ataxia. The morbidity is usually around 1 per cent and the mortality may reach 10 per cent or higher. The disease usually lasts 1–3 days and the majority of the birds recover completely. The duration of the outbreak may be 2 weeks. The cause of the disease is not known. However, the cerebellum of the affected birds show mild diffuse cellular infiltration, mild interfolial meningitis, swelling and proliferation of the capillary endothelium, and mild perivascular cuffing by mononuclear cells among which are occasional cyst-like spaces (Wight, 1968).

AVIAN MONOCYTOSIS
(Pullet Disease; Blue Comb)

This is an infectious disease usually of young laying birds, characterized by dehydration, relative and absolute monocytosis, and uratic nephritis.

Cause.—Recent evidence indicates that the disease is due to a virus which is cultivable in 8-day chick embryos killing them in 36–72 hours, but this viral aetiology has not been confirmed. Earlier it was thought to be due to a toxic factor such as that present in new wheat. According to Truscott and Morin (1964) blue comb disease of turkeys is apparently caused by a *Vibrio.*

Clinical Signs and Course of the Disease.—Usually the young adult is affected. The average age when the birds are affected is 21 weeks. The disease is generally characterized by whitish watery diarrhoea, dehydration, and a bluish comb in some cases. Laying flocks show a drop in egg production. About 5 per cent of the flock may be affected though this may increase considerably. Mortality ranges from 50 per cent to nil with an average of about 5 per cent. The disease lasts 10–14 days.

Post-mortem Lesions and Diagnosis.—The characteristic lesions are Zenker's degeneration of the skeletal muscles (fish flesh appearance), focal necrosis of liver, haemorrhages on the heart, gizzard, and ovaries, and severe enteritis. The pancreas has a chalky white appearance and uratic deposits are seen in the kidneys. Often there is mucoid enteritis and a sour crop. The intestines are discoloured and contain watery gaseous material; the spleen may be contracted. Necrotic lesions of the liver are characterized by round yellowish areas about 1 mm. in diameter. Histologically the muscles show lack of striation, and the kidneys changes due to uric nephritis.

The blood-picture shows a relative and absolute monocytosis with an average of 8000 monocytes per c.mm. whereas the normal is about 1700. The blood also shows a high uric acid content and non-protein nitrogen.

Diagnosis is based mainly on the clinical signs, the lesions, and histological examination if necessary. It has to be differentiated from fowl cholera, fowl typhoid, and pullorum disease which can be identified by bacteriological examination.

Epizootiology, Treatment, and Control.—The disease appears to be fairly widespread though it was not well recognized earlier due to lack of diagnostic criteria. It is more common in young layers, and it is more prevalent in well-nourished birds and good layers.

Control measures are based on the principles of good management. Avoid excess grain but give plenty of water. Some recommend a mild laxative. However, in view of the dehydration, this line of treatment may not be advisable. Molasses and antibiotics administered in drinking-water or mash are helpful. Use 1 pint of molasses in 5 gallons of water on the first day and give 1–3 g. of a broad-spectrum antibiotic, such as a tetracycline in 5 gallons of water per day for a few days, or 200–500 g. of the antibiotic per ton of feed for a few days.

CHAPTER VIII

SYSTEMIC DISEASES

DISEASES OF THE DIGESTIVE SYSTEM

THESE may be classified into specific and non-specific diseases.

SPECIFIC DISEASES

The specific diseases of the digestive system are classified according to causes which may be:—

1. Viruses.
2. Bacteria.
3. Fungi.
4. Parasites which may be metazoa or protozoa.
5. Avitaminoses.

Several viruses such as those of Newcastle disease and fowl plague can cause enteritis. Fowl pox can cause lesions in the mouth. Several bacteria, especially *Mycobacterium avium*, *Escherichia coli*, *Salmonella*, *Clostridia*, and others, can cause enteritis and they have been referred to in the respective chapters.

The fungi that produce disease of the alimentary tract are *Candida albicans* which causes mycosis of the upper digestive tract and *Aspergillus flavus*, *Alternaria* spp., and some others which produce enteritis due to the toxins they form.

A considerable number of helminths live in the alimentary canal of the fowl and they have been listed under the section on helminths. The parasitic protozoa can cause a variety of lesions depending on the species and the extent of infection. The main lesions caused by protozoa in the digestive system and their locations are listed in *Table* 12. Coccidia are the most common protozoa parasitizing the intestine.

In adult birds, there is usually more than one species of coccidium at a time in which case the lesions are confluent. Diseases caused by protozoa have already been dealt with earlier.

Avitaminosis A will cause characteristic lesions in the mouth and oesophagus, which are diagnostic. These consist of caseous white pustules which vary in size from a pinhead to a pea. Chalky deposits may also be seen in the cloaca, heart, and kidneys. Avitaminosis B_2 will cause intestinal atony. The avitaminoses have been dealt with under deficiency diseases.

NON-SPECIFIC DISEASES

The non-specific diseases will be dealt with according to the organ affected.

Beak.—

SCISSORS BILL.—This is a congenital condition in which the bird finds it difficult to bring the upper and lower beak into apposition. This may also occur when one of the sinuses around the beak is distended.

Table 12.—COMMON PROTOZOAN PARASITES IN THE DIGESTIVE SYSTEM OF THE DOMESTIC FOWL

SPECIES OF PROTOZOAN	USUAL SITE OF INFECTION	CHARACTERISTIC LESIONS	PATHOGENICITY
Eimeria tenella	Caeca	Caeca alone are haemorrhagic in acute cases, but in chronic cases they contain a caseous core	Pathogenic, especially to young birds
E. necatrix	Small intestine and caeca	Red, grey, and white spots visible from the serous wall. There is haemorrhage and catarrhal exudate into the intestines, which are ballooned	Pathogenic to young and old birds alike. Disease takes a long course
E. brunetti	Posterior intestine and cloaca	There is a general catarrhal enteritis with dysentery. White caseous material in lumen	Pathogenic to younger birds
E. hagani	Duodenum, jejunum, and ileum	Petechial haemorrhages in the anterior intestines	Moderately pathogenic
E. praecox	Upper third of intestine	There are mucous casts in the intestines	Generally not pathogenic
E. acervulina	Duodenum	White pinpoint spots and bands visible from the serous wall of the duodenum	Moderately pathogenic
E. maxima	Middle and posterior intestine	Intestinal wall thickened, exudate, and flakes of blood	Moderately pathogenic
E. mitis	Duodenum	None	Generally not pathogenic
E. mivati	Anterior part of intestine	Rounded lesions, petechial haemorrhages, and congestion	Rarely pathogenic
Trichomonas gallinae	In the crop, oesophagus, and pharynx	Yellowish caseous circumscribed areas like buttons	Pathogenic in young chicks
T. gallinarum	Caeca usually	Caseous cores in caeca	Not very pathogenic
T. eberthi	Caeca	None	Non-pathogenic
Histomonas meleagridis	Caeca usually	Caseous cores in caeca	Usually not very pathogenic to chickens

DEFORMED BEAK.—In this condition either the upper or lower part of the beak becomes short. This is usually congenital. In either of these cases feeding is interfered with and the affected birds should be destroyed.

Crop.—

PENDULOUS CROP.—In this condition, the crop hangs down and it is thought that this is partly due to hereditary causes. It can also be seen in moniliasis, *Capillaria* infection, and neural Marek's disease. This condition is gradually progressive but unless it is due to specific infections can be corrected surgically. However, this procedure is not worth while.

CROP BOUND OR IMPACTED CROP.—In this condition, the crop is distended with dry, hard, fibrous food-material or large amounts of grass and similar material. It is usually caused by bad feeding practices. It can be corrected by crop lavage, where the crop is syringed out with normal saline to which a little sodium bicarbonate has been added. This is done by using a syringe with a long nozzle, and massaging the crop. Acute cases can be corrected surgically by opening the crop and removing the material inside it and suturing it with mattress sutures. This procedure is likely to cause a fistula. After syringing the crop or after the operation the bird should be given about $\frac{1}{2}$ oz. of mineral oil.

Proventriculus and Gizzard.—These organs can be impacted, but diagnosis of this condition in the living bird is difficult unless it is associated with impaction of the crop. Treatment is not satisfactory and surgical intervention usually proves to be fatal.

The gizzard may also be penetrated by sharp objects, like nails, due to the firm contractions it undergoes. Such a condition is only diagnosed at autopsy or at the slaughter-house.

Intestines.—Inflammation of the intestines is quite common and is very often due to specific causes discussed earlier. Enteritis is also seen in certain forms of poisoning, especially salt poisoning.

INTESTINAL PERFORATION, OBSTRUCTION, OR INTUSSUSCEPTION.—These conditions are sometimes observed at autopsy but usually cannot be diagnosed in life. Generally, most sharp objects are retained in the gizzard which may be pierced. However, in rare cases they may pass into the intestines and cause perforation leading to peritonitis. As birds are resistant to peritonitis, affected birds may live for some days or even recover if the perforating structure is encapsulated and the perforation walled off.

Intestinal obstruction is usually caused by bad feeding where the defective feed contains excess of fibre, ascarids, or tumours which may partially or fully occlude the intestinal lumen. At times, tumours may cause partial paralysis of the intestines causing atony and stasis. Atony of the intestines may also be caused by avitaminosis B_2.

If intestinal obstruction is suspected, $\frac{1}{2}$ oz. of bland mineral oil orally and B complex vitamins parenterally may be useful.

Intussusception or invagination of one portion of the intestine inside the adjoining portion, is seen rarely in the fowl at post-mortem examination and is caused by some irritation or lesion caused by helminths or other agents. Usually

this leads to arrest of circulation, stasis of blood, and necrosis of the invaginated portion. Some birds with intussusception may also show prolapse of the intestines. The author has seen a case where necrosis of the invaginated portion was followed by spontaneous union of the other portions of the intestines.

Intestinal perforation and intussusception are not usually diagnosed in life, but if detected, the conditions can be corrected surgically. However, it is not worth while and birds having these conditions are so debilitated when seen by the veterinarian that they become very poor surgical risks.

Caeca.—These organs are infected by various parasites, chiefly *Heterakis gallinarum*, *Capillaria* spp., *Eimeria tenella*, *Histomonas meleagridis*, and *Trichomonas gallinae*, some of which may cause disease in young birds. These have been described before.

Vent Gleet.—This is a condition seen usually in layers characterized by inflammation of the vent, formation of yellowish-white diphtheritic membranes and an abnormally obnoxious foul smell. It does not appear to be infectious, and only one or two birds in a flock are affected at a time. Removal of the membrane leaves a raw red surface. Defecation is difficult and the bird strains regularly. The feathers round the vent are soiled with greenish-white excreta and the bird generally looks miserable.

Treatment consists of removal of the membrane or deposits and painting the raw surface with an astringent and antiseptic solution or a tetracycline ointment which is superior. A laxative diet of mash is useful.

Prolapse of the Intestines and Oviduct.—This is a condition which is usually seen in individual birds, generally at the height of production. In rare cases several birds may show prolapse and this may continue to occur in other birds for weeks or months. There may be a genetic susceptibility. The exact cause is not known. Hormones, excessive lighting, strain associated in egg laying, especially large eggs, certain types of feeds containing hormones or hormonogenic feeds, bacteria (chiefly *Salmonella* and *Escherichia*), or parasites such as skin mites may precipitate the condition. There is a tendency for the birds to attack the prolapsed portion. This will lead to cannibalism and cause perforation of the prolapsed portion and peritonitis. As birds are more resistant to peritonitis than mammals, affected birds may live for some days or even recover if the perforated portion is returned to the abdomen and becomes encapsulated. The author has seen cases where the prolapsed portions of the intestines had been pecked at until they were severed.

Birds with prolapses must be isolated and the prolapsed portion washed with an antiseptic lotion such as quarternary ammonium compound or potassium permanganate, and all the debris removed and the intestines pushed back after which a purse-string suture is placed round the cloaca. It is necessary to give a broad-spectrum antibiotic for a few days. Approximately 30 per cent of the cases recur if the purse-string suture is not tight. On the other hand if it is too tight, defecation is interfered with. In any case, the suture has to be removed between the fifth and seventh day. Affected birds should be given a bran mash and kept in isolation for about 1 week, for if they are returned to the pen too early a relapse is likely to occur.

Such treatment is often not worth while when there are thousands of birds in a flock. It is important therefore to determine the cause or causes and eliminate them.

DISEASES OF THE OTHER ABDOMINAL ORGANS

Peritonitis

Peritonitis is inflammation of the peritoneum and occurs owing to a variety of causes which may be specific or non-specific. Some of the specific causes such as *Salmonella* and *Escherichia coli* infections have been dealt with before. Salpingitis and salpingoperitonitis as seen in field cases can be produced experimentally by the inoculation of *E. coli* by the intra-uterine or intraperitoneal routes. The peritoneum can also be inflamed in acute infections, as in fowl cholera or fowl typhoid.

The commonest non-specific causes of peritonitis are rupture of the oviduct and the egg, rupture of the intestines, or rupture of a tumour or a lesion in an organ in the abdominal cavity, causing infection of the peritoneum. A bird with peritonitis will show a depressed appearance with fluid in the abdominal cavity, and if accumulation of fluid is great the bird will adopt the 'penguin' sitting attitude. There will be diarrhoea and the feathers round the vent will be soiled.

Peritonitis is usually diagnosed at autopsy. The usual lesions are serofibrinous adhesions of the peritoneum and other abdominal organs and presence of turbid serosanguineous fluid in the abdominal cavity. It is necessary to determine whether peritonitis is due to specific or non-specific causes. If it is due to specific causes several other birds are likely to be ill simultaneously.

Treatment is not worth while, but preventive measures have to be taken if peritonitis is due to specific causes.

Ascites

Ascites is accumulation of fluid in the abdominal cavity and is sometimes seen in adult as well as young birds. It is usually caused by some obstruction in the portal circulation, a tumour, or a similar condition. It can also be seen in salt poisoning, cardiac disease, impaired kidney function, or anaemia. Unlike in peritonitis, the fluid is clear and copious in volume and contains few cellular elements. In advanced cases, the abdomen is markedly distended, the carcase is emaciated, and the bird will adopt the characteristic 'penguin' sitting attitude.

The fluid can be aspirated by a needle but it is likely to reform. Treatment is not worth while, and in most cases even the carcase is unfit as food.

Salpingitis

Inflammation of the oviduct may be caused by a variety of factors. *Pasteurella multocida* and *Salmonella* spp. have been isolated most frequently in these cases. Usually salpingitis occurs in the presence of predisposing factors.

Hepatitis

Hepatitis is inflammation of the liver and is seen in a variety of specific diseases, such as fowl cholera, salmonelloses, listeriosis, vibrionic hepatitis, and many others.

This organ is affected in a variety of toxic conditions. In ducks there is a virus infection which causes a specific hepatitis and in turkeys the protozoan parasite *Histomonas meleagridis* causes enterohepatitis. All these diseases have been dealt with in the appropriate sections.

DISEASES OF THE RESPIRATORY SYSTEM

These may be specific and non-specific.

Specific Diseases

The specific diseases are caused by a variety of agents and factors, many of which have been dealt with individually before. As many of the agents producing respiratory symptoms cause a common syndrome, the purpose of this section is to outline the main clinical features, the principles of diagnosis, and treatment of each of the diseases, so that the reader, without having to wade through the various sections of the book, will be able to determine the disease in the field, as far as possible, and know quickly the drug recommended for treatment. The respiratory diseases of the fowl may be caused by:—

1. Viruses.
2. Mycoplasma (pleuropneumonia-like organisms).
3. Bacteria.
4. Fungi.
5. Parasites; these may be helminths or insects.
6. Deficiencies.

The respiratory diseases may be primary, those localized to the respiratory tract, and secondary, those generalized diseases that cause secondary respiratory symptoms.

Viral.—The viruses that cause primary symptoms are those causing:—

a. Infectious laryngotracheitis.
b. Infectious bronchitis.

The viruses that may cause secondary respiratory symptoms are those causing:—

a. Newcastle disease.
b. Fowl plague.
c. Fowl pox, primarily the diphtheritic form.

Mycoplasma.—The *Mycoplasma* that causes respiratory symptoms is *Mycoplasma gallisepticum*, which causes a type of chronic coryza of long duration, but which either by itself or in association with other agents, such as viruses of infectious bronchitis and Newcastle disease or bacteria like *Escherichia coli* and Staphylococci or with other viruses, causes chronic respiratory disease.

Bacterial.—The bacterial agents responsible for respiratory symptoms are:—

a. Haemophilus gallinarum which causes acute bacterial coryza of short duration by itself but in co-operation with other agents may cause chronic coryza. The respiratory symptoms are primary.

b. Pasteurella multocida which causes fowl cholera. The respiratory symptoms are secondary.

Fungal.—The fungal agents that cause respiratory diseases are:—

a. Aspergillus fumigatus. This is usually a primary infection.

b. Scopulariopsis spp. These cause a form of coryza in Venezuela and Bavaria.

Parasitic.—The parasites that cause respiratory infections are:—

a. The helminth *Syngamus trachea*, in the trachea.

b. Cytodites nudus, the air sac mite. This mite usually does not produce clinical symptoms.

Deficiency.—The most important deficiency that causes respiratory disease is avitaminosis A which predisposes birds to infection with bacteria and viruses.

Diagnosis.—The common diseases affecting the respiratory system and the methods used for the treatment, prevention, and control of these disorders, are set out in *Table* 13.

Garside (1965) has shown that histopathological examination of the upper respiratory tracts of chickens suffering from respiratory infections is of considerable value in diagnosing the presence or absence of viruses and determining their identities. Thus in infectious laryngotracheitis there is haemorrhagic necrosis of the mucosa and intranuclear inclusion bodies by about 5 days after infection. Later there is vascular congestion, oedema, and separation of the epithelium. There is very little cellular infiltration.

In contrast to this picture, in infectious bronchitis, there is diffuse infiltration on the subepithelial and deeper layers of the mucosa with lymphocytes, plasma cells, and histiocytes, resulting in considerable thickening of the mucous membrane, sometimes up to 8 times the normal thickness. However, these changes last for a short period and the mucosae return to normal in 7–11 days.

In mycoplasmosis there is marked distension of the epithelial glands of the trachea and turbinates, the mucous glands become swollen with vacuolation of the epithelial layer. The mucous glands also become enlarged, elongated but compressed, and extend deeper into the mucosa. The lungs show granulomatous areas, each consisting of an area of necrosis surrounded by giant cells. In mixed infections the histopathological changes are also suggestive of mixed infection.

The histopathological picture in Newcastle disease depends to a considerable extent on whether the bird is initially fully susceptible to infection or partially immune (as would occur as a result of vaccination and reduction of immunity with time). In fully susceptible birds there is oedema and vascular congestion of the tracheal mucosa. The epithelium is tattered, and in many instances no epithelium survives. The subepithelial layers project on to the tracheal mucosa as tufts. The inflammatory exudate is scanty or absent.

However, in those birds that show resistance to this virus, the reaction is different and epithelial hyperplasia is marked. These changes somewhat resemble those seen in infectious bronchitis virus. However, in Newcastle disease the thickening of the epithelium is caused by profuse hyperplasia of the epithelial cells but there is no lymphocytic infiltration and the cellular reaction is least marked. In many instances the nuclei of the hyperplastic epithelial cells are surrounded by a narrow unstained halo of cytoplasmic vacuolation.

Table 13.—COMMON DISEASES AFFECTING THE RESPIRATORY SYSTEM

DISEASE	CHARACTERISTIC SIGNS AND LESIONS	METHODS OF DIAGNOSIS
Aspergillosis	Caseous nodules in the lungs, and less often, greenish masses in the air sacs	The presence of fungal hyphae in the nodules and the isolation of *Aspergillus fumigatus*
Bacterial coryza	This is an acute highly infectious coryza characterized by nasal discharge and swollen eyes and sometimes sinuses. The mortality however is low	Isolation of *H. gallinarum* in the early stages of the infection is diagnostic
Chronic respiratory disease (mycoplasmosis)	Cheesy deposits in air sacs; chronic tracheitis and sinusitis; discharges of mucus from the nostrils and cheesy deposits in the nasal sinuses. The disease spreads slowly and insidiously	The course of the disease and the lesions are very suggestive. Isolation of pathogenic *Mycoplasma* is diagnostic but difficult. H.A.I. test, serum agglutination, and whole blood agglutination test
Fowl cholera	There are no characteristic lesions. The course of the disease varies considerably. In chronic cases the comb, wattles, and joints may be swollen	Presence of bipolar organisms in stained smears of the heart blood and isolation of *Pasteurella multocida* are diagnostic
Fowl plague	Usually very high mortality and haemorrhages in the proventriculus	Virus isolation. Haemagglutination inhibition, cross immunity, and pigeon inoculation tests. The petechial haemorrhages in the proventriculus are suggestive

Fowl pox	The pox lesions on the comb, wattles and lore. These contain thick warty scabs which are characteristic	The lesions are diagnostic. If necessary chick embryos may be inoculated
Gape worms	Severely affected birds show gasping signs	The red worms can often be seen through the larynx when bird gasps for breath
Infectious bronchitis	Caseous plugs in bronchi. Clinical signs vague and may vary from difficult and gasping respirations to rapid drop in egg production or a nephritic syndrome	Virus isolation: serum neutralization test confirms diagnosis. Caseous plugs in the bronchi if present are significant. Need to show rising titre in paired serum samples. Also use gel diffusion precipitin test which is specific for recent infection
Infectious laryngotracheitis	Haemorrhagic tracheitis or laryngitis in acute cases, or diphtheritic deposits in larynx or trachea in chronic cases. Clinically the disease may vary from the acute to the inapparent infection	The post-mortem lesions are diagnostic; virus isolation and serum neutralization tests can also be used to confirm diagnosis. Histopathological examination of lower trachea will show characteristic changes including the pathognomonic inclusion bodies
Newcastle disease	High mortality in Asia, Africa, and Europe. Petechial haemorrhages in the proventriculus; sometimes nervous signs and acute respiratory distress	Virus isolation, haemagglutination inhibition, cross-immunity tests, and pigeon inoculation are used for diagnosis. The petechial haemorrhages in the proventriculus are suggestive of the disease

Treatment, Prevention, and Control.—These are briefly described below for each heading.

INFECTIOUS LARYNGOTRACHEITIS.—No treatment. Isolate birds and use sound principles of hygiene. In endemic areas, live virus vaccines may be used. Vaccination with live virus vaccines should not be performed in areas where the disease does not occur.

INFECTIOUS BRONCHITIS.—No treatment. Isolate birds and use good principles of hygiene. Live and inactivated virus vaccines are used and quite often they are combined with those of Newcastle disease. Live virus vaccines may be given orally or intranasally. In areas where the disease is a problem, chicks should be vaccinated regularly.

NEWCASTLE DISEASE.—No treatment. The control of this disease is governed in most countries by various regulations. In some there is a slaughter policy. Whatever the policy, no treatment is effective and in areas where the disease causes high mortality it is best to kill all the affected birds and vaccinate the healthy birds. Vaccination is done in most countries with live attenuated virus vaccines given in drinking-water, intranasally, or parenterally. In others, vaccination with dead vaccines only, is permitted. All birds should be vaccinated as a routine measure at regular intervals.

FOWL PLAGUE.—No treatment. Destroy the affected birds and vaccinate the survivors. In endemic areas regular vaccination may be done.

FOWL POX.—Treatment is not necessary in mild cases. However, valuable birds if severely affected may be treated by painting the lesions with an antiseptic and removal of the diphtheritic deposits. Sulphonamides or antibiotics should be given in drinking-water to control secondary bacterial invaders.

MYCOPLASMOSIS.—Treatment in advanced cases is not worth while. In milder cases, mass treatment is done by giving tetracyclines or tylosine in mash or drinking-water for several weeks. All badly affected flocks should be culled and the premises rested for one month. The nucleus of the new flock should consist of birds free of pathogenic *Mycoplasma.* Individual treatment is sometimes done by giving tetracyclines parenterally. This is often not worth while.

BACTERIAL CORYZA.—Sulphonamides and many other antibiotics are effective. Sulphonamides, particularly sulphathiazole and sulphaquinoxaline, are widely used. The latter may be given in drinking-water in a concentration of 0·04 per cent for 2–3 days.

FOWL CHOLERA.—Sulphaquinoxaline, if given as in bacterial coryza, is fairly effective. Nitrofurans particularly furazolidone, and tetracyclines are useful in very severe outbreaks. Thorough disinfection of quarters and elimination of chronically affected birds are necessary. Eliminate stress conditions. Vaccines may be used where available.

ASPERGILLOSIS.—Treatment is not effective. Destroy affected birds, burn litter, and disinfect premises thoroughly; use 0·5 per cent copper sulphate to scrub buildings.

SYNGAMUS TRACHEA INFECTION.—Barium antimony tartrate is useful in the treatment. Place birds in a closed space and sprinkle the powdered drug at the rate of 1 oz. per 8 cu. ft. of space, agitate the birds and thus expose them to the dust for 15 minutes. Treat batches of 6–8 birds at a time.

Non-specific Diseases

The domestic fowl is generally not subject to many non-specific diseases of the respiratory tract.

The non-specific factors that may affect the chicken are mainly chilling and ammonia gas irritation.

Chilling *per se* does not cause disease but it reduces the vitality of the bird so that its resistance becomes lowered and it succumbs to disease, mainly to specific respiratory infections. Chilling acts as a stress factor.

The main post-mortem changes of a chicken that has died of chilling are usually sodden and pneumonic lungs.

Ammonia gas irritation also acts as a stress factor and in addition causes irritation of the eyes and the respiratory passages leading to keratoconjunctivitis, lacrimation, and even pneumonic changes. This is usually seen in the ill-ventilated houses where the gas accumulates.

Chilling and ammonia gas irritation can be avoided by proper heating arrangements and correct sanitary practices. Litter should be kept dry so that ammonia gas does not form. Excessive heat and overcrowding may also produce pulmonary changes similar to those produced by chilling.

DISEASES OF THE UROGENITAL SYSTEM

Ovaries and Oviduct

Both these organs can become cystic, and when the oviduct becomes cystic it may distend the abdomen and cause cessation of laying. These conditions are diagnosed at autopsy and no treatment is possible even if they are diagnosed in the live bird. Inflammation of the oviduct occurs in certain infections, chiefly those due to *Salmonella pullorum* and *Salmonella* spp. In these cases, the ovary also shows degenerative changes.

Egg Concretions

These may sometimes be formed in the oviduct as well as the peritoneum, leading to peritonitis. These are concentric masses of accumulated yolk and albumen which when cut give a lamellated appearance. These masses can obstruct or occlude the oviduct. This condition is diagnosed at autopsy.

Egg Bound

This is a condition where the egg is arrested in the oviduct due to the large size of the egg, such as a double-yolked egg, or to the narrowing of the lumen of the oviduct due to disease such as inflammation or egg concretions. Relief may be given to the bird by breaking the egg with the forefinger or a pair of forceps and extracting it in pieces. Insertion of a bland oil will lubricate the oviduct and facilitate the passage of the egg. Care should be taken not to injure the oviduct with pieces of the egg-shell.

If the trouble is due to too large an egg, extraction of the egg *in toto* or in pieces will usually afford relief, but if it is due to some obstruction, the condition is likely to recur and therefore it is necessary to cull the bird.

KIDNEYS

Kidneys are usually affected in a number of specific diseases. Thus nephritis with accumulation of uratic deposits is seen in avian monocytosis, avitaminosis A, and infection with certain strains of infectious bronchitis virus.

It is doubtful whether primary nephritis is common. However, it can be seen under bad conditions of feeding. Spector (1951) concluded that renal disease, variously known as fowl nephritis, visceral gout, visceral gout nephritis, and renal mononucleosis constituted a pathological entity and that the disease was a progressive pyelonephritis which yielded pure cultures of *E. coli.* The kidneys can also be affected with lesions of lymphomatosis and with retention cysts. These are distended vesicles which may be 1–3 cm. in diameter and contain a clear fluid. In ducks, the kidneys are parasitized by a type of coccidium. In the domestic fowl, a nephritis-nephrosis syndrome associated with uraemia has been reported in the United States and Australia (Newton and Simmons, 1963) and the syndrome appears to be caused by a certain strain of the infectious bronchitis virus (Winterfield and Hitchner 1962; Cumming, 1963).

Lathkar and Rajya (1968) in a study of 204 selected cases of nephritis in the fowl in Mathura, India, observed that glomerular nephritis formed 39·2 per cent, pyelonephritis 29·9 per cent, and uric acid nephritis 14·2 per cent of the cases. The other forms of nephritis encountered were those due to tubular nephritis, interstitial nephritis, and nephritis due to the avian leucosis complex.

CHAPTER IX

TUMOURS AND NEOPLASTIC DISEASES

THESE include the leucosis complex, Marek's disease, and the Rous sarcoma which are specific infectious conditions forming the largest group of neoplastic diseases of the chicken. These have been described previously.

Tumours are generally classified according to the origin of the tumour cells, whether they are malignant or benign, and the type of cell present. Generally speaking, 5–10 per cent of a flock may die of tumours. The incidence of tumours increases with age. The exact causes of these tumours are not known but it is likely that a majority of them are caused by virus-like agents which are activated by the presence of predisposing factors, genetic susceptibility, and many others.

CLASSIFICATION

Broadly speaking tumours may arise from the connective tissues, muscles, vascular systems, nervous tissues, epithelial tissues, and serous membranes. They may be benign, malignant, mixed, or pigmented. Various combinations of these groups of tumours can theoretically exist. However, from the disease aspect, only a few of them are important.

Some of the more common ones which may be seen in the field are mentioned below.

Sarcomas.—These are malignant connective tissue tumours and are classified according to the type of cells predominating. Fibrosarcoma is a common type of tumour in the fowl. This tumour is characterized histologically by the presence of irregularly arranged, hyperchromatic fibroblasts with mitotic elements and varying amounts of collagen. A fair proportion of sarcomas are caused by virus-like agents, some of which are not yet sufficiently characterized. These agents are not specific in their action.

The most important avian sarcoma is the Rous sarcoma (chicken tumour No. 1), caused by a ribosenucleic acid virus related to the agents causing avian leucosis complex. Experimentally this virus behaves very differently in different circumstances,though it primarily produces a fibrosarcoma. Various species of birds and even rodents are affected. Infections with the virus cannot be regarded as a clinical entity. Andrewes(1964) states that 'the avian sarcomas are probably an uncommon manifestation of the activity of an ubiquitous virus'. Evidence that they are contagious would therefore be surprising. The lesions appear as soft growths with metastases in the lung, liver, and heart. They are sporadic in appearance and are of greater importance to the virologist and laboratory worker than the poultry pathologist. Avian sarcomas and avian tumour viruses have been reviewed by Rubin (1962). Recent experimental data indicates that the Rous sarcoma virus may be spread by contact (Burmester and Fredrickson, 1968).

The histiocytic sarcoma is common in the ovary. It extends to the surrounding tissues in a rather diffuse manner and is histologically characterized by mixed cells

including fibroblasts, stellate and polygonal cells, macrophages, and foreign body giant cells.

Lymphocytoma.—This is the most common tumour affecting the domestic fowl, and is frequently found in the liver, testes, ovaries, kidneys, spleen, peritoneum, intestines, and other organs rich in lymphoid tissue. The type cell is the neoplastic lymphocyte and this is the type tumour seen in lymphomatosis. Histologically and macroscopically the condition may be diffuse, discrete, or mixed.

The female appears to be more susceptible than the male and sex hormones appear to play a part in determining susceptibility to the tumour. The disease usually lasts 1–2 months, at the end of which it proves fatal to the bird.

Histologically, the tumours consist of extravascular proliferating lymphocytes, usually arranged in foci around blood-vessels. In the lobular type, connective tissue isolates masses of tumour cells into clumps. The disease process spreads directly and metastases are rare. Further information is given in the section on the leucosis complex.

Myelocytoma.—This is a tumour seen in the peritoneum, liver, spleen, gonads, and other tissues. It is soft, diffuse, and dull white in colour. There is anaemia, and myelocytes are found in the circulation. These cells also predominate in the tumour. It is not a common tumour and forms part of the leucosis complex.

Fowl Leucosis.—This is a malignant disease affecting the haemopoietic tissues of the bone-marrow, liver, spleen, and kidneys, characterized by severe anaemia, pale organs, and the presence of immature blood-cells in the circulation. It is caused by uncontrolled and rapid autonomous proliferation of the cells, forming granulocytes and erythrocytes, due to filterable agents. It is generally seen in birds about 1 year old. It forms part of the leucosis complex.

The spleen becomes markedly enlarged and multiple petechial haemorrhages are common. Myeloid tissue fills the bone-marrow space replacing fat cells and giving it a reddish appearance. Leucosis must be differentiated from secondary anaemia and other neoplastic diseases.

Carcinoma.—This is a malignant tumour originating from epithelial tissues. Metastases are common. The incidence of carcinomas in chickens is lower than that of sarcomas, being about 10 per cent of the cases of tumours. Most of the carcinomas are found in the ovary, and are seen in adult birds of 1 year or over. Most carcinomas of the chicken are primary and medullary. Carcinomas can also be seen in other epithelial tissues especially in the skin over the metatarsal region.

Macroscopically, carcinomas vary in appearance but histologically they can be identified by determining the epithelial type of cells from which they originate. They are also characterized by the infiltrative and destructive character of their growth.

Telangiectasis.—This is a benign type of vascular tumour where a group of blood- or lymph-vessels become dilated and distended with blood or lymph and are sometimes referred to as 'bleeding cysts' by farmers.

Besides the above tumours, various types of tumours such as nephromas, teratomas, mesotheliomas, melanomas, papillomas, adenomas, and many others

have been reported from the chicken. Apart from the specific tumours, granulomas may be seen on any superficial region caused by trauma or pathogenic organisms. They are localized and show inflammatory reactions round them with a centre of necrosis. They can be treated surgically. A complete discussion of tumours is outside the scope of this book. Further information has been given by Feldman and Olson (1965).

The other common tumours of the chicken which have not been dealt with before are:—

1. Adenocarcinomas in the ovary, oviduct, serosa of the intestine, mesentery, and thyroid, which are hard, pink or white in colour, nodular or cauliflower-like, or sometimes pearl-like in appearance.
2. Haemangio-endotheliomas and haemangiomas of the liver, spleen, kidneys, lungs, and skin, characterized by their vascularity which at the post-mortem examination may show haemorrhages and ruptured blood-vessels.
3. Gliomas in the brain which appear as soft non-capsulated white foci.
4. Leiomyomas of the smooth muscle of the oviduct (Awadhiya, Bandyopadhyayay, Jain, and Beri, 1968).

EFFECTS

The effects of tumours depend on the nature of the tumour, chiefly its malignancy, the organs involved, the presence of metastasis, the age of the bird, and the duration of the tumour. Some of the tumours are visible externally while those in the ovary will make the bird non-productive and those in the abdominal cavity will usually cause ascites or peritonitis. Ultimately these changes will cause emaciation, anaemia, and death in the case of malignant tumours.

Theoretically it is possible to excise the non-malignant tumours. Though this is a practical possibility in cutaneous tumours, the expense involved in relation to the value of the bird hardly warrants surgical interference in the large benign tumours.

CHAPTER X

LETHAL FACTORS AND INFLUENCE OF HEREDITY ON DISEASE AND PRODUCTION

THERE are a large number of lethal and semilethal factors in farm animals and the domestic fowl and they have been listed by Stormont (1958). According to him 26 factors have been reported in the fowl. These are:—

1. Creeper.
2. Congenital loco.
3. Sticky.
4. Wyandotte lethal.
5. Congenital palsy.
6. Cornish lethal.
7. Sex-linked lethal 1.
8. Sex-linked lethal 2. (occurs during growth from 23–123 days).
9. Amarilla.
10. Flightless.
11. Malformed skeleton.
12. Naked.
13. Short beak.
14. Microphthalmia.
15. Micromelia.
16. Talpid.
17. Chondrodystrophy (extreme type).
18. Chondrodystrophy (less extreme type).
19. Deformed mandible.
20. Wingless.
21. Congenital perosis.
22. Crippled.
23. Jittery.
24. Lethal back.
25. Crooked neck.
26. Shaker.

Most of the phrases used are descriptive of the conditions and a full discussion of these factors is outside the scope of this book. The incidence of these factors is low and they are not frequently seen in poultry practice. Of the various lethals listed the better known ones are:—

1. The wingless lethal expressed as a single autosomal recessive, lethal in the homozygous condition, characterized by complete absence of wings, complete absence of lungs, air sacs, and other anatomical deviations. The embryos continue to develop until the end of the incubation period but fail to hatch.

2. The talpid lethal where extra digits are found, which are frequently webbed. Besides, there is ectopia of organs. Homozygotes die at 8–10 days of incubation.

3. The Cornish lethal where the extremities are shortened in the heterozygotes which survive, while the homozygotes die in the last week of incubation.

Some of the homozygotes carrying the lethal factors die during incubation, as in creeper, Cornish lethal, short beak, and talpid, while others die during the first few weeks after birth. Some continue to live showing the characteristic features. These factors, therefore, contribute to lowered hatchability and lowered survival. These lethal factors are recessives, usually caused by single gene mutations, being lethal or semilethal in the homozygous form and transmitted to the offspring by the heterozygous carriers.

It is now well recognized that innate resistance due to genetic factors exists in the fowl to *S. pullorum*, *S. gallinarum*, and avian leucosis complex infections. In the case of the fowl, unlike in domestic animals, breeding to enhance genetic

resistance appears to be a very fruitful field. Already considerable headway has been made in breeding fowls resistant to some forms of the avian leucosis complex.

Breeding has also made considerable advances in producing birds with increased survival rates, higher rates of egg production, increased growth-rates, lowered feed conversion rates, and earlier sexual maturity, which are the most important and desirable characters in poultry breeding programmes.

CHAPTER XI

DIAGNOSIS OF POULTRY DISEASE

DIAGNOSIS of poultry disease at times involves the use of bacteriological or virological techniques which cannot be used under field conditions. However, often it is possible to diagnose most of the diseases when the history, the age of the affected birds, the clinical signs, and the post-mortem lesions are considered collectively. Therefore, *Tables* 14, 15, 16 list the common diseases of the domestic fowl, arranged according to the age when they occur, the main clinical signs of disease, and the changes seen at post-mortem respectively.

POST-MORTEM EXAMINATION OF POULTRY

Because of the low value of a bird, treatment of individuals is often not undertaken. However, when several birds are ill, it is often advisable to perform a post-mortem examination on a few birds as a diagnostic procedure. Whenever a good history is available it is often possible for an experienced poultry pathologist to make a specific diagnosis after a post-mortem examination without resorting to elaborate, cultural and serological procedures.

The exact details of the procedure of a post-mortem examination will vary from one individual to another. However, the following procedure is recommended:—

Determine the Accurate History.—This involves obtaining data on mortality and morbidity patterns, the age-groups affected, vaccines used, the rations given, and the salient clinical signs shown. Wherever possible the composition of the ration should be obtained, as it is not uncommon to find that diseases are caused by a ration that is deficient or one that has been stored too long.

Superficial Examination of the Carcase.—Before a carcase is opened it should be examined for deformed legs, cysts, growths on the skin, ectoparasites and other abnormalities which may be overlooked at a routine post-mortem examination.

Post-mortem Examination.—The following procedure is recommended as it has been found to be the most convenient.

a. The skin is cut on the inner aspect of the thighs and the hip joints are dislocated and the legs laid flat.

b. The skin over the sternum and the abdomen is removed. At this stage, the breast is examined for breast blisters, white streaks in the flesh, haemorrhages, or deformed keel bone.

c. The abdominal wall and the ribs are cut to expose the viscera and thoracic contents.

d. A blood-sample is taken from the heart for cultural examination if necessary. Smears are made from heart blood, and examined for protozoan parasites such as *Aegyptianella pullorum*, *Leucocytozoon caulleryi*, or spirochaetes if they are

suspected to be the cause of the disease. Material from the liver is taken for cultural examination at this stage. Next the air sacs, pericardium, heart, lungs, trachea, and sinuses of the head are examined, preferably in this order. The peritoneal cavity, mesenteries, ovaries, and kidneys are next examined.

e. The alimentary canal is then removed from the carcase. stretched out and opened; it is examined section by section.

f. The sciatic nerves, the brachial plexi, and the hock joints are then examined.

Table 14.—INCIDENCE OF COMMON DISEASES OF THE DOMESTIC FOWL ACCORDING TO AGE-GROUPS

AGE	COMMON DISEASES
At birth or within the first 2–3 days	Bacillary white diarrhoea, salmonelloses, omphalitis, mushy chick disease, debilities or defects caused by vitamin deficiencies in parent stock or by defective incubation. The diseases like leucosis complex and epidemic tremor are egg transmitted and are usually not manifested at birth
Birth to 6 weeks	All diseases mentioned above, aspergillosis, caecal coccidiosis, curly toe, encephalomalacia, epidemic tremor, exfoliating dermatitis, listeriosis, rickets, six-day disease, infectious bronchitis, fatty liver and kidney syndrome, Gumboro disease, infectious avian nephrosis
6–12 weeks	Aspergillosis, coccidiosis, infectious coryza, infectious bronchitis, listeriosis, Marek's disease, Newcastle disease*, mycoplasmosis, synovitis, helminthiasis, and fowl pox
12–15 weeks (pullets)	Avian monocytosis, leucosis complex, infectious coryza, infectious bronchitis (including nephritis and nephrosis), infectious laryngotracheitis, helminthiasis, Newcastle disease, fowl pox, Marek's disease
Birds over 15 months	Bacterial coryza, fowl cholera, infectious laryngotracheitis, infectious bronchitis, leucosis complex, Newcastle disease, avian monocytosis, fowl typhoid, fowl pox, Marek's disease

* If there is no maternal immunity, Newcastle disease may be seen earlier.

g. The carcase is then turned over, and the rest of the skeletal system, especially the vertebral column, is examined after it has been skinned.

h. Finally, the cranium is opened and the meninges and the brain substance are examined for abnormalities. It is not necessary to open the brain in routine post-mortem examinations, unless the history suggests that the disease is encephalomalacia, epidemic tremor, or a similar condition. A histopathological examination may be necessary in such cases.

i. If necessary, tissues for histological examinations are taken from the liver, brain, and other organs and fixed in formal saline.

The history of the outbreak and the clinical signs often give a clue to the disease that exists. Therefore, during the post-mortem examination, more attention should be given to detecting those conditions one suspects to have caused the disease, while not ignoring others which may appear to be less likely.

Table 15.—SIGNS OF DISEASE IN THE DOMESTIC FOWL*

STATE OF ORGAN	ASSOCIATED DISEASE OR CONDITION	COMMENTS
Abdomen		
Diarrhoea	Non-specific	Many varied causes
Enlarged and firm	Egg peritonitis, internal laying, mushy chick disease	—
Pasted-up vent	Constipation, bacilliary white diarrhoea	—
Protrusion of viscera	Prolapse, cloacitis	Cannibalism frequently present
Scouring	Diarrhoea	Non-specific
Swollen and flabby, with penguin posture	Dropsy, ascites	A variety of causes
Ulceration of vent	Vent gleet	Only seen in laying stock
Beak		
Crossed	Genetic defect of chickens	Noticed soon after hatching
Impacted	Mandibular disease, 'shovel beak'	Often preceded by infection of associated mucous glands
Parrot	Manganese deficiency	—
Pliable	Rickets	Leg weakness also present
Undershot	Not indicative of disease	Normal feature following de-beaking
Breast		
Blistered (cystic)	Breast blisters Infectious synovitis	Common, sometimes infected Affects broilers aged 4–8 weeks
Crooked	Crooked breasts	Common. Genetic + nutrition + management defect. Rickets
Emaciated	Evidence of chronic, non-specific illness, coccidiosis, worm infection	Cull
Comb		
Absent	Normal, following dubbing	—
Bright red	Hyperaemia	Normal sign of good health
Congested	Heart failure	—
Cyanotic	Blackhead, fowl cholera (acute), septicaemia, erysipelas, pullet disease, round heart disease	—
Gangrenous	Frostbite, ergot poisoning, local injury	In turkeys the snood is gangrenous in erysipelas
Pale	Anaemia, bird malaria, aegyptianellosis, heavy parasitism, leucosis complex	Often secondary. Also following internal haemorrhage

* Adapted from Blount (1961).

Table 15.—SIGNS OF DISEASE IN THE DOMESTIC FOWL (*continued*)

STATE OF ORGAN	ASSOCIATED DISEASE OR CONDITION	COMMENTS
Comb		
Scabby	Local injuries	
	Fowl pox	Mouth lesions usually present
Scurfy	Favus (whitecomb)	Parts of face also affected
Yellow	Jaundice, erythroleucosis	Isolated cases, usually in pullets
Crop		
Impacted	Impaction of the crop	Contents doughy and firm
Pendulous (turkey)	Pendulous crop	Contents voluminous
Sour	Sour crop, moniliasis	Contents fluid and very acid
Ears		
Bulging	Mite infested	Head shaking occurs
	Middle ear abscess	Wry-neck
Eyes		
Discharging	Coryza, fowl pox	Lacrimation is otherwise uncommon in poultry
Purulent eyelids	Avitaminosis A, aspergillosis	—
Inflamed	Fowl pox, ammonia blindness, blepharitis	—
Scabby	Deficiency of vitamin B complex	—
Cloudy	Keratitis	Local irritation
Sticky	Vitamin A deficiency	Also occurs when no deficiency of vitamin A is present
Swollen cornea	Ammonia blindness	Old litter in use
Dry	Avitaminosis A	Xerophthalmia
Ulcerated	Keratoconjunctivitis	Ammonia blindness associated with use of old litter
White iris and dilated pupil	Nicotine sulphate poisoning	Rare
Distorted iris (cat's eyes)	Genetic defect	Harmless
Pinpoint pupil	Ocular Marek's disease, iridocyclitis	Blindness present
Opaque lens	Blindness, also encephalomyelitis virus	Cause unknown
Face		
Scurfy	Favus	Comb also involved
Swollen	Cellulitis, chronic fowl cholera	Local infection
Oedematous	Coryza	*Haemophilus* infection of eyes and nose present
Feathers		
Broken off	Genetic defect	Local injury
	Depluming mite infestation	Skin reddened
Curled	Frizzle feather	Genetic/nutritional defect
Dark specks amongst the vent and abdominal feathers	Northern fowl mite infestations	Readily confused with red mites

Table 15.—SIGNS OF DISEASE IN THE DOMESTIC FOWL (*continued*)

STATE OF ORGAN	ASSOCIATED DISEASE OR CONDITION	COMMENTS
Feathers		
Depigmented (white)	Lysine deficiency	Seen in young turkey poults only
Loose	Botulism	Also non-specific feature
Matted with blood	Haemangiomata, local injury	Careful differentiation necessary
Matted with pale mucus	'Colds' Ammonia blindness	Neck and shoulders only
Matted with white urates at the vent	Nephritis *Salmonella* infections, vent gleet	'Pasted-up' vents common
Pecked	Feather pecking (vice)	Boredom an important feature
Ruffled	Coccidiosis, spirochaetosis	—
Vent feathers with white clusters of eggs	Lice infestations	Adult lice often visible
Feet		
'Clutching hand'	Neural Marek's disease	Also of infra-red ray or genetic origin
Crooked toes	Crooked toes (chicks)	Pantothenic acid and biotin deficiency
Dermatitis (poults)	Vitamin B deficiencies	Common local infection
Swollen feet	Bumblefoot, *Pasteurella* abscesses, gout, infectious synovitis, local injury	Uncommon. Urates present in joints. Management faults
Toes 'balled up'	Non-specific Curly toe disease	Vitamin B_2 deficiency symptom in young chicks
Toes sloughed	Local injury, creosote poisoning, biotin deficiency	Very rare
Head		
Anaemia (pale)	Caecal coccidiosis, rupture of liver or aorta, tuberculosis, bird malaria, aegyptianellosis, leucocytozoonosis	A feature of many culls. Indicative of secondary anaemia, or internal haemorrhage
Congested and discoloured blue-green	Heart failure Local injuries (bruising)	Common non-specific feature Associated with presence of sub-cutaneous haemorrhage
Purplish (cyanosis)	Blackhead, erysipelas, round heart disease	—
Shaking	Secondary to coryza or otitis media	Normal feature in many fowls
Sagging	Blindness	—
Swollen	Cellulitis, fowl plague, *Haemophilus influenza* infection	May follow careless implantation of sex hormone tablets
Tremors	Epidemic tremor	—
Twitching or nodding	Newcastle disease	Sometimes sole symptom in turkeys

Table 15.—SIGNS OF DISEASE IN THE DOMESTIC FOWL (*continued*)

STATE OF ORGAN	ASSOCIATED DISEASE OR CONDITION	COMMENTS
Infra-orbital Sinuses		
Swollen	Coryza, *Mycoplasma* infections, infectious sinusitis (turkeys)	Eyes discharging. Very common
	Avitaminosis A, one-eyed roup	Eyes and throat also involved
Legs		
Deformed hocks	Perosis, dislocation	Slipped tendon usually present
Discoloured hocks	Local injuries, ruptured tendons	—
Swollen hocks	Hock disease (turkeys), rickets, tenosynovitis (staphylococcal), atypical pullorum disease, chronic erysipelas, infectious synovitis	Other joints also swollen
Paralysed legs	Neural Marek's disease	One or both legs affected
	Fractures	Only one leg usually affected
	Transient paralysis	One or both legs affected
	Kinky back disease (spinal deformity)	Both legs always affected. Broilers 4–9 weeks
Gross thickening of the shanks	Osteopetrosis, infectious tenosynovitis	Lameness often present
Raised scales (shanks)	Scaly leg disease	Becoming rare
Weakness	Rickets	—
Mouth		
Congested	Nicotinic acid deficiency	'Black tongue'
Gasping	Aspergillosis, infectious laryngotracheitis, gapes, acute fowl plague, Newcastle disease, neural Marek's disease, infectious bronchitis	Air hunger symptoms also arise from other causes, e.g., heatstroke
Pustular and diphtheritic lesions	Fowl pox	Lesions adherent (diphtheritic)
	Avitaminosis A	Pharynx and oesophagus involved
Salivation	Poisoning	Rare
Scabs (angle of mouth)	Deficiency of vitamin B, local injuries	
Neck		
Paralysed	Botulism (ducks), thiamine deficiency, Newcastle disease	Rare
Thrust forward	Folic acid deficiency	Turkey poults (rare)
Tremors	Epidemic tremor	Chicks aged 1–4 weeks
Twisted	Neural Marek's disease, Newcastle disease, genetic defect (poults)	Wry-neck also occurs from other causes

Table 15.—SIGNS OF DISEASE IN THE DOMESTIC FOWL (*continued*)

STATE OF ORGAN	ASSOCIATED DISEASE OR CONDITION	COMMENTS
Nose		
Discharging mucus	Coryza (colds), bronchitis	*H. gallinarum* or *Mycoplasma* infections
Nares impacted with dried mucus	Roup	—
Nervous System		
Bicycling movements	Nutritional encephalomalacia	Also other causes
Coma	Non-specific	A sign of impending death
Convulsions	Nutritional encephalomalacia Poisoning (nitrophenide)	Also pyridoxine deficiency
Dullness	Non-specific	Sleepiness often precedes death Extreme dullness seen in nitrofurazone poisoning
Incoordination	Neural Marek's disease, Newcastle disease, epidemic tremor, duck virus hepatitis, poisoning	—
	Transient paralysis	Pullets 10–16 weeks
	Kinky back disease (spinal deformity)	Broilers 4–9 weeks
Rigor	Fever, chilling, psittacosis	—
Tremors	Epidemic tremor	Affecting chicks up to 5 weeks of age
Walking backwards	Nutritional encephalomalacia, Newcastle disease	—
	Kinky back disease (spinal deformity)	Broilers 4–9 weeks
Respiratory System		
Air hunger (gasping)	Fowl pox, diphtheritic form, aspergillosis, gapes, Marek's disease, infectious laryngotracheitis, gape worms	A sign of discomfort or pain
Cheeping	Salmonellosis, bacillary white diarrhoea	—
Coughing	Infectious bronchitis, infectious laryngotracheitis, Newcastle disease	Blood coughed up in infectious laryngotracheitis
Laboured breathing	Non-specific, obstruction to air flow, *S. trachea* infection	Often related to hydropericardium, dropsy, etc.
Mouth breathing	Roup	'Pip'
Panting	Fever, hot environment	Normal in hot weather
Rattling (throat)	Infectious bronchitis, mycoplasmosis, Newcastle disease	Chicks frequently affected. Nervous symptoms often present
Sneezing	Virus and *Mycoplasma* infections	Adults frequently affected
Squawking	Foreign body in the larynx or trachea	—

Table 15.—SIGNS OF DISEASE IN THE DOMESTIC FOWL (*continued*)

STATE OF ORGAN	ASSOCIATED DISEASE OR CONDITION	COMMENTS
Skin		
Bleeding	Haemangiomas (bloodcysts)	Also local injuries, snood gangrenous
Blotchy (patchy)	Erysipelas (turkeys)	Heart failure with venous stagnation
Congested	Pullet disease, nephritis, egg peritonitis	Non-specific feature seen in hexamitiasis, pullet disease, etc. Also water starvation. Subcutaneous haemorrhages present
Discoloured	Bruising, local injury, haemorrhagic disease, vitamin K deficiency	Rare in turkeys
Eczematous	'Burns'	Chemical irritation chief cause, also smothering
Exfoliating	Dermatitis	Rare diathesis
Oedema	Salt poisoning Vitamin E deficiency	—
Ulceration	Tumours (lymphomas)	—
Yellow	Spirochaetosis, erythroleucosis	Uncommon
Tail		
Bent	Marek's disease or genetic	Rare
Tongue		
Curled	Genetic defect	Common in turkey poults fed on dry mashes
Dehydrated	Secondary to roup	'Pip'
Discoloured	Blacktongue, nicotinic acid deficiency	—
Wattles		
Absent	Normal following 'cropping' operation	—
Swollen	Secondary to coryza	Solid yellow pus present in local *Pasteurella* infection
Wrinkled and thickened	Fowl cholera (chronic)	Oedematous in fowl plague
Wings		
Drooping	Intestinal coccidiosis, Marek's disease, blackhead, bacillary white diarrhoea, normal in hot weather	A debility feature seen also in other diseases
	Fowl cholera (acute)	Often sign of impending death
Flapping	Round heart disease, Marek's disease	One or both wings affected
Paralysed	Local injury, fractures, dislocations, Marek's disease	—

Table 16.—POST-MORTEM DIAGNOSIS OF DISEASES OF THE DOMESTIC FOWL*

CLINICAL AND PATHOLOGICAL FINDINGS	DISEASES TO BE SUSPECTED
Air sac infections. Thickening of thoracic and abdominal air sacs with the presence of caseous exudate or caseated masses but without other respiratory involvement	Secondary complications following Newcastle disease, infectious bronchitis, peritonitis, egg-transmitted *M. gallisepticum* infection, and *Escherichia coli* infection
Blood, watery	Malnutrition, spirochaetosis, bird malaria, aegyptianellosis, leucocytozoonosis, leucosis complex, coccidiosis (*E. tenella* and *necatrix*)
Comb and face, white spots on	Favus
Caecal haemorrhage	Coccidiosis, blackhead (with necrotic areas in liver)
Coryza syndrome: nasal catarrh, sinusitis, conjunctivitis, with or without the presence of caseous masses	Infectious coryza due to *Haemophilus gallinarum*, chronic respiratory disease or coryza due to *Mycoplasma* (Nelson's coccobacilliform bodies), fowl pox (usually with pox lesions in the mouth, larynx, and skin), vitamin A deficiency (usually with white nodules on the oesophagus and crop, and urates in the kidneys)
Gall-bladder distended	Fowl typhoid, chills in chicks, pullorum disease, enteritis, vitamin A deficiency, starvation
Gizzard, erosion of	Nutritional deficiency
Haemorrhages, petechial, on the heart, pericardium, and peritoneum, etc.	Haemorrhagic syndrome, fowl plague, Newcastle disease, pasteurellosis (if haemorrhages occur in other organs), pullet disease (with nephritis), spirochaetosis (with enlarged spleen and liver), erysipelas
Head and wattles swollen	Chronic pasteurellosis, head injuries, coryza, fowl pox, emphysema
Kidneys, nephritic	Vitamin A deficiency, visceral gout, pullet disease, chills, salt poisoning, metabolic disturbances, infectious bronchitis variant virus infection
Legs, weakness of (information usually obtained from history of disease)	Marek's disease, vitamin D deficiency (soft bone), riboflavin deficiency (curly toes), thiamine deficiency, arthritis or synovitis, manganese deficiency, kinky back disease (spinal deformity)
Liver and spleen enlarged	Spirochaetosis (spleen mottled), *Aegyptianella pullorum* infection, fowl typhoid (bronze coloured liver), erythroleucosis (bright red spleen), lymphoid leucosis, visceral Marek's disease (with white nodules myeloid leucosis under the surface of the liver), tuberculosis (caseated nodules raised over the surface of the liver, with nodules in bone-marrow), blackhead (necrotic areas with caseated plugs in caecum), ornithosis, erysipelas

* Adapted from Chu (1960).

Table 16.—POST-MORTEM DIAGNOSIS OF DISEASES OF THE DOMESTIC FOWL (*continued*)

CLINICAL AND PATHOLOGICAL FINDINGS	DISEASES TO BE SUSPECTED
Lungs, nodules in	Aspergillosis, pullorum disease, leucosis complex
Mouth, caseous exudate in	Vitamin A deficiency
Mouth, scab-like lesions in the corners of the mouth and eyes	Fowl pox, pantothenic acid and biotin deficiency, thrush, riboflavin deficiency in turkeys
Nerves enlarged	Neural Marek's disease
Nervous symptoms (incoordination and/or paralysis)	Newcastle disease or vaccination reaction (with respiratory and other symptoms), neural Marek's disease (with enlarged brachial and sciatic plexuses), avian encephalomyelitis (with encephalomyelitis and muscular dystrophy), transient paralysis, kinky back disease, vitamin B deficiency (with polyneuritis in B_1 deficiency and curly toes in B_2 deficiency), overheating (with acute congestion of lungs), vitamin E deficiency (oedema, haemorrhage, and necrosis of brain)
Oesophagus and crop, pseudo-membranous inflammation and ulceration of	Thrush (moniliasis), turkey or pigeon pox, *Trichomonas* infection
Ovaries, degenerated	Fowl typhoid, salmonellosis, egg peritonitis, spirochaetosis, pullet disease
Ovaries, haemorrhagic	Fowl plague, Newcastle disease, spirochaetosis, fowl typhoid, pullorum disease
Pericarditis and perihepatitis	*E. coli* septicaemia, ornithosis
Proventriculus, haemorrhage in	Newcastle disease, fowl plague, spirochaetosis, sulphonamide poisoning (with haemorrhages), Gumboro disease
Small intestines, haemorrhages in, or mucoid enteritis	Spirochaetosis, Newcastle disease, fowl plague, intestinal coccidiosis, pasteurellosis, sulphonamide poisoning (with haemorrhage of subcutaneous tissue), clostridial infections, haemorrhagic syndrome, fowl typhoid, Gumboro disease
Small intestine, nodules in	Tuberculosis, tapeworm infection, leucosis complex, Hjarre's disease (*E. coli* granuloma)
Tracheitis and/or bronchitis with or without air sac involvement or coryza	Infectious laryngotracheitis, infectious bronchitis, Newcastle disease, gape worm infection, aspergillosis, fowl pox, fowl plague, pasteurellosis, chronic respiratory disease due to *Mycoplasma*
Yolk, unabsorbed in chicks	Pullorum disease, chills and other adverse environmental factors, mushy chick disease, salmonelloses

SECTION B

COMMON DISEASES OF TURKEYS

CHAPTER XII

VIRUS DISEASES

TURKEY POX

TURKEY pox is caused by a virus (*Borreliota meleagridis*) which is closely related to the fowl pox virus and is transmitted by certain species of mosquitoes, chiefly those of the genera *Aedes* and *Culex*.

Turkey pox resembles fowl pox but the lesions are more extensive and may be found on the legs, conjunctival sacs, mouth, crop, and oesophagus where they may be cheesy. In severe cases, lesions may be found on the feathered parts of the body. The course of the disease is prolonged and it may end in blindness or death due to starvation. The disease is more prevalent during the insect vector season.

Prevention and Control.—Turkeys may be vaccinated at any age using fowl pox virus, which induces good immunity. Annual vaccinations are recommended about 2 months before the insect vector season. Turkey pox virus causes a stronger reaction and should not be used for purposes of immunization. Vaccination is best done on the inner aspect of the thigh.

There is no specific treatment; severely affected birds may be given antibiotics to control the effects of secondary invaders.

NEWCASTLE DISEASE

The turkey is susceptible to this disease, but in this species it is not as severe as in the domestic fowl. The general principles governing epizootiology, prevention, and control are the same as those outlined in the case of the domestic fowl. Partial or complete motor paralysis of one or both legs may be the only noticeable sign.

VIRUS HEPATITIS

Snoeyenbos, Basch, and Sevoian (1959) and Mongeau, Truscott, Ferguson, and Connell (1959) simultaneously described outbreaks of infectious hepatitis in turkeys in certain parts of the United States and in Ontario, Canada, respectively, which were caused by a filterable agent having the characters of a virus.

The disease is characterized by focal necrotic or degenerative hepatitis and pancreatitis. The mortality and morbidity are low. Usually young turkeys less than 2 weeks old are clinically affected. The chicken is resistant to the organism, which can be grown in the chick embryo by inoculation via the yolk-sac. It is less than 0·3 μ in size.

TURKEY MENINGOENCEPHALITIS

Komorov and Kalmar (1960) described a disease affecting turkeys in the Shomron area in Israel, characterized by progressive paralysis and non-purulent meningoencephalitis.

They determined that the causative agent is a virus, probably a member of the Arbor group which is very pathogenic to mice. It is likely that the virus is transmitted in nature by an arthropod, probably *Aedes aegypti.*

The morbidity is about 50 per cent. In the early stages there is a staggering gait and greenish diarrhoea, finally ending in complete paralysis.

TURKEY RESPIRATORY VIRUS INFECTION

Bankowski and Conrad (1966) have reported the isolation from turkeys of a virus which is capable of producing a respiratory disease characterized by lacrimation, conjunctivitis, severe sinusitis, and drop in egg production. The virus has been designated *Myxovirus meleagrium* (Bankowski and Conrad, 1966).

TURKEY HEPATITIS VIRUS

This virus causes a subclinical disease in turkeys and a moderate degree of hepatitis and pancreatitis. It appears to be different from that causing duckling virus hepatitis though the two viruses are antigenically related to one another.

INFLUENZA A VIRUS OF TURKEYS
(*Wilmot Virus*)

A new virus belonging to the influenza A group has been isolated from the respiratory tract of turkeys. The role of the virus in producing respiratory disease in turkeys has not been determined.

An influenza A virus isolated from turkeys in Canada produces sinusitis in experimental turkey poults, reduces egg production considerably, and causes some mortality in the field. It is related to the tern (Scottish fowl) virus of 1959. Another variant of the turkey influenza A virus (virus 7732) was found to be highly pathogenic to chickens and turkeys (Lang, Narayan, Rouse, Ferguson, and Connell, 1968).

OTHER DISEASES

The other viral diseases to which the turkey is susceptible and which have been described under diseases of the fowl are:—

1. Fowl plague
2. Psittacosis (ornithosis)
3. Equine and avian encephalomyelitis
4. Lymphomatosis

CHAPTER XIII

BACTERIAL DISEASES

FOWL CHOLERA
(Pasteurella multocida Infection)

THIS infection is most common in turkeys between 6 and 8 months of age. The clinical signs and post-mortem changes resemble those seen in the same disease in fowls. The skin becomes pinkish and pneumonia is a constant post-mortem change.

Certain sulphonamides such as sulphamerazine and sulphaquinoxaline are useful in the treatment of this infection. Streptomycin and broad-spectrum antibiotics will also control it. Chloramphenicol can be given at the rate of 1·0 g. per pound of feed or 0·2 g. injected intramuscularly daily. Sulphaquinoxaline is given at the rate of 1·5 g. per 10 pounds of feed.

SALMONELLA INFECTIONS

FOWL TYPHOID
(Salmonella gallinarum Infection)

This disease is similar to that seen in the domestic fowl and is frequently acquired from it. The disease is usually characterized by greenish or greenish-yellow diarrhoea and increased thirst. The initial outbreak usually causes heavy mortality but this decreases as new outbreaks flare up. The post-mortem changes resemble those seen in the domestic fowl. The liver and spleen are enlarged to 2–3 times their normal size and are friable. The heart shows areas of necrosis and the lungs are greyish. In contrast to the lesions in chickens, haemorrhagic enteritis and ulcerations of the duodenum are frequently encountered. The lesions in the ovary resemble those of the domestic fowl. In poults the liver is enlarged and friable, often creamy white in colour, and yolk-sacs are retained.

The disease is controlled by the same principles as used in the case of the domestic fowl. As most of the outbreaks occur as a result of infection transmitted from fowls, turkeys should not be reared in the same premises as fowls. Furazolidone is the drug of choice but it does not eliminate the carriers; the dosages recommended are 50–100 g. per ton of feed for 7 days or more. Chloramphenicol and tetracyclines are useful but not as effective as furazolidone.

PULLORUM DISEASE
(Salmonella pullorum Infection)

The disease usually resembles that in domestic fowl. Adults with the subacute form of the infection will generally show ascites. Prevention and control of the disease is carried out in a manner similar to that used against this infection in fowls. The disease may be treated, but this is usually done as a salvage operation. Sulphonamides have been used for this purpose, but more modern drugs mentioned under

pullorum disease can be used with even better results. However, it is very important to bear in mind that the main aim is the total eradication of the disease and not merely its control. It is generally agreed that of the various tests used to detect the disease, the tube agglutination test is the most efficient.

SALMONELLOSES
(*Paratyphoid*)

As in the case of the domestic fowl, salmonellosis in the present context is regarded as an infection with any pathogenic species of *Salmonella*, other than *S. pullorum* and *S. gallinarum*. Salmonelloses are increasingly becoming an important disease problem in turkey poults. The most common species responsible for outbreaks of salmonelloses in turkeys are *S. typhimurium* and *S. enteritidis*. The former organism appears to be found more frequently in the intestines of the carrier turkeys than in the ovaries. It is likely that the egg-shells are contaminated during laying and that penetration of the egg by the bacterium occurs during incubation. Therefore, ovarian transmission is not as important as in the case of pullorum disease. The disease is most commonly seen between the ages of 3 days and 1 month and in general resembles pullorum disease. Diarrhoea is not a constant finding. The common post-mortem findings are congestion of the liver, kidney, gall-bladder, and heart muscle. Diagnosis is based on the isolation and identification of the causative organism. Agglutination tests used to detect these infections are elaborate and have to be varied according to the individual requirements.

The disease can be controlled by using the principles outlined in the case of salmonelloses of the domestic fowl. Several drugs have been used in the treatment of these infections. Furazolidone appears to be the most useful. However, this drug is likely to interfere with results of the agglutination tests, and will not eliminate carriers in the levels normally given to birds. The drug is used at levels of 0·005 per cent continuously for prevention and 0·01 per cent for treatment.

TURKEY ERYSIPELAS

This disease is caused by *Erysipelothrix insidiosa*, a bacterium that also causes swine erysipelas and disease in man. It is more common in male turkeys nearing marketing age. At the onset of an outbreak sudden deaths occur and swelling of the snood is sometimes seen. The head often becomes cyanotic and nasal catarrh is present in many cases. Erythematous lesions are seen on the face and wattles. (*E. insidiosa* has been found to cause about 5 per cent deaths in pullets.)

Post-mortem examination reveals that the skin of the breast is often coloured purple in irregular patches and the nasal passages filled with mucus. In chronic cases vegetative endocarditis of the bicuspid and tricuspid valves are often seen. There are petechial haemorrhages especially on the heart muscles and liver, and the spleen is often enlarged, and the duodenum often shows a severe catarrhal and sometimes haemorrhagic enteritis.

The lesions are usually characteristic of the disease. However, it has to be differentiated from fowl cholera by isolating the organism or by animal inoculation using pigeons or mice. Pigeons or mice die 24–96 hours after inoculation. Isolation of the organism is best done from liver and heart blood.

The disease can be treated by the use of penicillin (200,000–300,000 units per bird) and prevented by the use of bacitracin. Procaine penicillin can also be given in feed at the rate of 600 g. per ton of feed. Turkeys should not be reared on the same premises as swine or sheep.

An inactivated vaccine given at 8–10 weeks, and repeated prior to point of lay in breeding stock, gives good protection.

STAPHYLOCOCCOSIS

This disease is caused by particular strains of *Staphylococcus aureus* which generally produce an alpha type of haemolysis, ferment mannitol, and are coagulase positive. In turkeys it usually runs a septicaemic course with subsequent localization in certain joints of the leg. The characteristic features are swelling of the feet and hock joints, a sulphur yellow coloured diarrhoea and abscesses in the sternum containing a yellow pus-like or cheesy exudate. Arthritis of the hock joint, foot pads, and other joints may also be caused by *Streptobacillus moniliformis* which is probably transmitted in nature by rats and their bites. Tetracyclines appear to be effective in controlling this infection.

The disease is diagnosed by isolating the particular strain of *S. aureus*. It has also to be differentiated from infectious synovitis.

Treatment is difficult. Some broad-spectrum antibiotics may be useful. However, as a considerable proportion of *Staphylococci* are resistant to several antibiotics, sensitivity tests have to be conducted before treatment is attempted.

STREPTOCOCCOSIS

This is usually a septicaemic condition caused by certain species of *Streptococci*, whereas others may cause a pullorum-like disease. The disease varies considerably but often there are diffuse haemorrhages in the breast muscles and skin, and the disease may be mistaken for fowl cholera infection in the turkey.

MYCOPLASMOSIS

(Infectious Sinusitis; Air Sacculitis; Chronic Respiratory Disease)

Mycoplasmosis in the turkey may be caused by *Mycoplasma gallisepticum* and *M. meleagridis*. The former organism causes both chronic respiratory disease and infectious sinusitis while the latter organism is responsible for a mild respiratory infection of turkey pullets, often referred to as air sacculitis. The latter organism appears to be very widespread in the U.S.A. Infectious sinusitis is usually characterized by swelling of the infra-orbital sinuses which become filled with a gelatinous or caseous exudate.

Symptoms.—The incubation period varies from one to several weeks. Initially there is a discharge from the nostrils but later the disease may take one of the three forms. One form is characterized by distension of the infra-orbital sinuses and is referred to as infectious sinusitis. In the second form the disease process becomes localized in the lower respiratory tract and causes lesions similar to those seen in C.R.D. in chickens. It is characterized by decreased weight-gains and bouts of 'cough'. The disease is chronic in nature and lasts several weeks. Mortality is

generally low. It has to be distinguished from vitamin A deficiency which it closely resembles. The third form is an air sacculitis in turkey pullets caused by *M. meleagridis* which causes considerable retardation of growth.

Sinusitis in the turkey may also be associated with infections due to *Pasteurella multocida*, *Haemophilus gallinarum*, the virus of Newcastle disease, and *Myxovirus yucaipa*, where the trachea also shows severe inflammation. Other organs such as the spleen and air sacs are affected (Bankowski and Mikami, 1965; Bankowski and Conrad, 1966).

Epizootiology.—The disease is transmitted primarily by direct and indirect contact, while egg transmission also plays an important role. The carrier bird is a source of infection.

Treatment.—The gelatinous exudate in the distended sinuses should be removed using a 14–16 gauge needle and syringe and after removal of the exudate, the following drugs may be injected into the sinus, keeping the needle in situ—150 mg. sterile solution of streptomycin, or 50–55 mg. of a tetracycline. The former drug is preferable and may be given by intramuscular injection in cases where there is lower respiratory tract infection. Spiramycin adipate is claimed to be highly effective producing over 90 per cent clinical cures. The dosage employed is 100–250 mg. per kg. body-weight subcutaneously or as a 0·04 per cent solution in drinking-water continuously for 3 days (Inglis and Cook, 1964).

SPIROCHAETOSIS

Spirochaetosis in the turkey is caused by *Borrelia anserina* the organism that also causes fowl spirochaetosis. It has loose spirals, stains readily, and has no hook-like bends at the ends.

Symptoms.—The incubation period is 2–6 days and the disease is transmitted by the fowl tick *Argas persicus*, the common red mite *Dermanyssus gallinae*, and *Culex* spp. of mosquitoes. The disease is characterized by thirst, diarrhoea, cyanosis of the head, and yellowish-green diarrhoea. The temperature rises to about 110° F. and leg weakness and paralysis become pronounced.

Post-mortem Changes.—The spleen is enlarged and mottled due to ecchymotic haemorrhages. The liver is enlarged and shows necrotic spots. Diagnosis is made by seeing the spirochaetes in Giemsa-stained blood-smears or sections of tissues stained by Levaditi's method.

Prevention, Treatment, and Control.—The vectors should be controlled. D.D.T. is effective against most of them and should be used for eradication. Penicillin, tetracyclines, and certain organic arsenicals are effective in the treatment of spirochaetosis. The drug of choice is penicillin; a single injection of 10,000–15,000 units given in the early stages cures the disease. Formalized vaccines have been used to immunize birds in infected areas. However, it is desirable to eradicate or control the vectors, especially *Argas persicus* which is the chief vector.

BOTULISM

This disease resembles that in the fowl and is characterized by flaccid paralysis of the neck. However, the feathers cannot be pulled out easily, and paralysis of the nictitating membrane does not occur usually.

TRANSMISSIBLE ENTERITIS
(*Avian Monocytosis; Blue Comb; Mud Fever*)

This is an infectious disease where the first symptoms are seen usually on the fourth or fifth day after infection, though the incubation period may be as short as 48 hours. It is characterized by watery diarrhoea, loss of weight, and a subnormal temperature. It resembles avian monocytosis of the domestic fowl and hexamitiasis of the turkey. Truscott and Morin (1964) have demonstrated that a vibrio is the cause. There appear to be at least three serologically different strains, and recently Truscott (1968) has been able to reproduce the disease experimentally in turkeys with a *Vibrio* sp.

OTHER DISEASES

The other bacterial diseases to which the turkey is susceptible and which have been described under diseases of the fowl are:—

1. Coli granuloma.
2. Infectious synovitis.
3. Listeriosis.
4. Paracolon infections.
5. Tuberculosis.

CHAPTER XIV

FUNGAL DISEASES

ASPERGILLOSIS
(Brooder Pneumonia; Mycotic Pneumonia)

THIS is common in turkey poults, is caused primarily by *Aspergillus fumigatus*, and resembles the disease in the domestic fowl. Unlike the latter species, more mature turkeys can also be affected. The lesions are more extensive and extend to the liver, kidney, spleen, and abdominal cavity. There are caseated masses in the lungs and other organs, with button-like ulcers and a greenish mould in advanced cases. Diagnosis is usually made by detecting the fungal hyphae. Treatment is not effective and the disease is controlled on the principles outlined under aspergillosis of the domestic fowl (p. 76).

CANDIDIASIS
(Moniliasis; Thrush; Mycosis of the Crop)

This disease is caused primarily by *Candida albicans*. It is usually associated with poor management or excessive use of broad-spectrum antibiotics. The incidence of the disease appears to be increasing. This infection is usually associated with other diseases, and the symptoms are not typical. The lesions are seen most frequently in the crop and have a curd-like appearance in advanced cases. In early cases, the mucous membrane of the crop has a parboiled appearance and may have a slimy exudate. The incidence of the disease can be reduced by adding nystatin (Mycostatin, Squibb) at the rate of 50 g. per ton of feed for 5–7 weeks.

Amphotericin B at levels of 14–55 mg. per kg. of the ration appears to be 4–5 times as active as nystatin.

FAVUS

This is an uncommon disease of the turkey caused by *Achorion gallinae* and resembles that in the domestic fowl.

CHAPTER XV

PARASITIC DISEASES

HISTOMONIASIS
(Enterohepatitis; Blackhead)

THIS is an acute infection of the turkey caused by *Histomonas meleagridis*, characterized by typhlitis, focal hepatitis, and sulphur-coloured diarrhoea.

Cause.—*Histomonas meleagridis* lives in the caeca of the domestic fowl which is the carrier, and the protozoan is passed in the eggs of *Heterakis gallinarum* in which it remains viable for several months, up to 150 weeks. Lund, Wehr, and Ellis (1966) have demonstrated that the earthworms *Lumbricus terrestris*, *Allolobophora caliginosa*, and *Eisinia foetida* are vectors of *H. gallinarum* and *Histomonas. H. meleagridis* is a flagellate with one flagellum but during certain phases of its life cycle it assumes an amoeboid form. In the turkey it causes an acute infection and invades the liver via the blood-stream. Some workers maintain that *Trichomonas gallinarum* or *Candida albicans* can also cause enterohepatitis.

It has been demonstrated recently, using gnotobiotic turkeys, that blackhead is produced by the dual action of *H. meleagridis* and *E. coli* and that *H. meleagridis* alone or with other bacteria cannot produce blackhead (Bradley and Reid, 1966). The aetiology of this syndrome has been reviewed by Reid (1967).

Symptoms.—The incubation period is 15–21 days. The prominent signs are droopiness and sulphur-coloured diarrhoea. The disease is more severe in younger birds, especially those below 12 weeks when mortality is usually over 50 per cent.

Post-mortem Changes.—The caeca are acutely inflamed and in advanced cases show a semi-caseous exudate. The liver shows large, usually circular, areas of necrosis which lie below its surface (*Fig.* 20).

Diagnosis.—The lesions are suggestive of histomoniasis but the disease must be differentiated from tuberculosis, tumours, and fungal infections. As certain fungal infections, notably those caused by *Candida albicans*, may resemble histomoniasis, tissue sections of the liver should be stained by periodic acid Schiff staining technique which will reveal either the fungus or the protozoan.

Treatment, Prevention, and Control.—Several drugs are useful in controlling mortality due to this disease. These are Enheptin or 2-amino-5-nitrothiazole, furazolidone, nithiazide, dimetridazole, Emtryl, certain arsenic acids, quinoline derivatives, metoxidazole and paromomycin. Enheptin is usually fed continuously in the mash at the rate of 0·05 per cent for prevention and suppression. The dosage may be increased to 0·2 per cent if the disease is well advanced. Dimetridazole (0·05 per cent) in drinking-water is effective in treating established infections and

appears to be the drug of choice. This drug does not appear to interfere with establishment of immunity (Joyner, 1963). However, if the drug is given for one generation continuously, it may reduce fertility. Paromomycin sulphate is also effective in controlling histomoniasis. A 0·1 per cent concentration of the drug in feed gave 80 per cent protection. Nidrafur fed at a level of 0·022 per cent acts as a preventive and reduces losses from histomoniasis.

The disease is prevented by administering phenothiazine to remove the caecal worms as described under *Heterakis gallinarum* infection. Domestic fowls must be

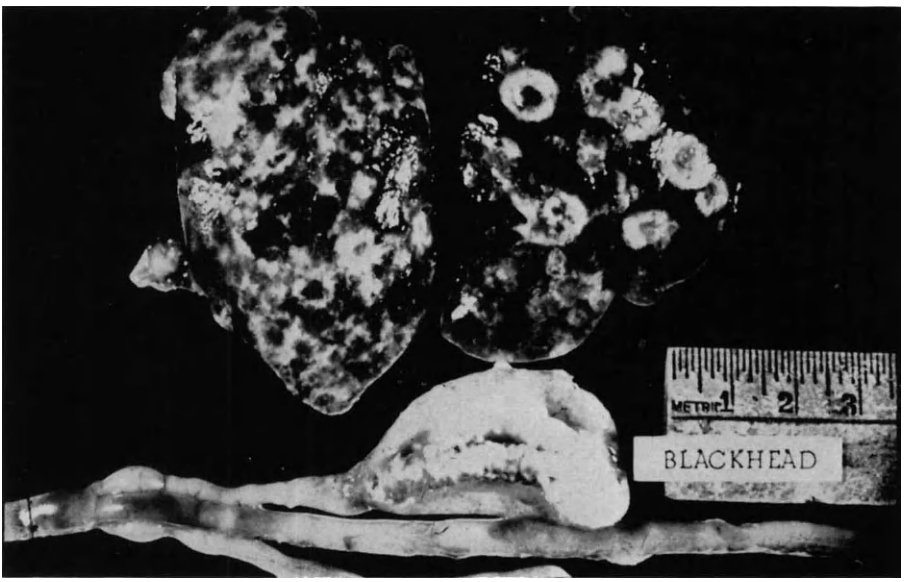

Fig. 20—Two sections of the liver and caecum of a turkey with enterohepatitis (blackhead). Note the large necrotic areas in the liver and the caseous cores in the caecum.

reared separately from turkeys. The dosage of phenothiazine for turkeys is 1 g. and for fowls 0·5 g. The drug may be given individually or in mash.

TRICHOMONIASIS

This is a chronic disease of turkeys reared under unhygienic conditions of management, characterized by chronicity, emaciation, unkempt feathers, and foul odour. It is caused by *Trichomonas gallinae*. The pigeon is the carrier. The characteristic lesions are necrotic ulcerations of the crop. These lesions may also be found in the oesophagus and the proventriculus.

Trichomoniasis may also be seen in the caeca and the liver. This is caused by *T. gallinarum*, but this organism is generally considered to be non-pathogenic.

Copper sulphate 1 : 2000 aqueous solution given instead of drinking-water for 2–3 days and repeated after an interval of a few days is said to be beneficial. Enheptin may also be useful in the treatment.

HEXAMITIASIS
(Infectious Catarrhal Enteritis)

This disease is characterized by foamy diarrhoea, nervousness, and gradual loss of weight.

Cause.—This disease is caused by *Hexamita meleagridis*, a flagellate with four pairs of flagella and two nuclei, each with a large karyosome. In carrier birds they are found in the caecal tonsils as well as in the bursa of Fabricius. In acute infections they are abundant in the anterior part of the intestines. Quail, ducks, and other birds act as carriers of *Hexamita meleagridis*.

Symptoms.—The incubation period is 4–7 days. The temperature usually drops to subnormal and there is foamy diarrhoea, nervousness, a stilted gait, and gradual loss of weight.

Post-mortem Changes.—The duodenum, jejunum, and rectum show catarrhal inflammation with marked lack of tone. The intestinal contents are usually foamy.

Diagnosis.—This can be made only on seeing the parasite in the anterior part of the small intestines. It is best to examine fresh faecal material on a warmed slide. It has to be differentiated from transmissible enteritis.

Treatment and Prevention.—Prevention is largely a matter of good hygiene and no drug has been found to give uniformly good results in this infection. Enheptin fed in the mash, at 0·1 per cent concentration, di-n-butyl tin dilaurate, furazolidone, and nithiazide are said to be useful.

LEUCOCYTOZOON INFECTION

This infection is caused by *Leucocytozoon smithi*, certain stages of which are seen in the erythrocytes. The parasite is transmitted by the black fly *Simulium occidentalis* and other *Simulium* spp.

Usually only birds below 12 weeks of age are affected. Symptoms are vague and after a course of 3–4 days the birds either recover or die. A fairly common symptom is the presence of respiratory râles. The changes that may be seen at autopsy are anaemia, emaciation, catarrhal inflammation of the duodenum, and cirrhotic changes in the liver. The schizonts are seen intracellularly in the liver cells and not in the peripheral circulation. High mortality can occur in severe outbreaks. Drugs used in the treatment of *Leucocytozoon* infections in the domestic fowls may be tried. However, black flies should be kept away from turkeys by screening the turkey houses with fly-proof wire mesh.

COCCIDIOSIS

Cause.—Coccidiosis of the turkey is commonly caused by the following species of coccidia:—

Eimeria meleagrimitis.—The epithelium of the villi of the small intestines is parasitized by this species. Poults 2–3 weeks of age may be severely affected, resulting in very high mortality. This is the most pathogenic species.

Eimeria adenoeides.—This is a pathogenic species and may affect turkey poults from 1 to 5 weeks of age causing high mortality.

Eimeria meleagridis.—The parasite is found in the caeca of the adult and in the posterior half of the intestines of younger birds and is not very pathogenic.

Eimeria gallopavonis.—This is a pathogenic species and can cause high mortality and retarded growth.

Eimeria dispersa.—This is not a very pathogenic species, but it may cause diarrhoea and loss of weight.

Two other species of coccidia of the turkey are *E. subrotunda* and *E. innocua.* The latter species is practically harmless.

Prevention, Treatment, and Control.—This is based practically on the same lines as those adopted in coccidiosis of the domestic fowl. Zoalene is very effective in controlling infections due to *E. adenoeides* and *E. gallopavonis.* The levels of zoalene that are required to obtain effective control vary from 0·012–0·0187 per cent of the ration (Hymas and Stevenson, 1962). Amprolium, sulphaquinoxaline, and sulphadimethoxine at dosage levels of 0·0125 per cent in the ration appear to be more effective than other coccidiostats in controlling coccidiosis in turkeys (Mitrovic, 1968).

OTHER PROTOZOAN INFECTIONS

These include infections by:—

1. *Cochlosoma anatis.*
2. *Haemoproteus columbae.*
3. *Plasmodium durae, P. fallax,* and *P. lophurae.*

None of these cause any serious disease.

CHAPTER XVI

MISCELLANEOUS DISEASES

DISSECTING ANEURYSM
(*Aortic Rupture*)

THIS lesion is usually seen in the posterior aorta caudal to the testes and the renal arteries. Often the aneurysm ruptures, causing fatal haemorrhage and sudden death. Post-mortem examination reveals massive abdominal haemorrhages and blood-clots trapped in mesenteries.

This condition can be produced experimentally by feeding beta-aminopropionitrile (BAPN). The incidence of aortic rupture can be greatly increased by injecting turkeys with diethylstilboestrol. The action of this hormone is enhanced by increased levels of sodium chloride in food. Aortic rupture is associated with hyperlipaemia, hypercholesterolaemia, and hypercalcaemia. It may also be related to *Candida albicans* infection (Tripathy, Mathey, and Kengy, 1965). A deficiency of copper may be one of the causes of aortic rupture. Copper is involved in the synthesis of elastin found in the connective tissues of the aorta.

Reserpine supplementation has been used to prevent aortic rupture in turkeys. However, growth, feed efficiency, and hatchability may be reduced by reserpine feeding.

TURKEY HAEMORRHAGIC ENTERITIS

This disease is characterized by a short incubation period of 5–6 days, massive haemorrhage into the gut, and reduction in water and feed consumption. These signs last only a few days. Haemorrhage lasts only a day or two and all the birds that survive the crisis recover without complications. This is probably caused by a filterable agent which is less than 0·22 μ in size and is probably a bacteriophage, with the co-operative action of a bacterium which may be a species of *Streptococcus* (Gross and Moore, 1967). However, the pathology of the condition is more suggestive of a toxic, rather than an infectious agent. A sensitization reaction might be involved (Gross, 1967).

TURKEY SYNDROME TS 65

A new syndrome in turkey poults from 3–6 weeks old, characterized by retarded growth, poor feathering, and abnormalities in hock joints and shank, was reported in 1965 in the U.K. In some ways it resembled perosis. However, there were also necrotic liver changes in about half of the cases (Gordon, 1965). Diagnosis of the condition should be based on an examination of the whole flock, and wherever possible, after inspection of the premises, rather than on an examination of a few birds.

The exact cause of this syndrome is not known, but there is evidence that stress, overcrowding, and *Mycoplasma meleagridis* may be partly responsible for the

syndrome. According to Wannop and Butler (1968) this is a previously unrecognized manifestation of *M. gallisepticum* infection in the turkey. Pathologically there is focal arrest of ossification resulting in assymmetrical growth of bone which is clinically manifested as bowing.

A NEW TURKEY SYNDROME

A disease causing 5·2 per cent mortality in well grown turkey poults aged 4–20 days, characterized by a swollen liver, distended gall-bladder, parboiled myocardium, and swollen kidneys containing fine uratic deposits on the surface has been described by Laursen-Jones (1968). It was not possible to determine the cause of this, though there was circumstantial evidence to indicate that the syndrome may have been caused by misuse of coal-tar disinfectants.

SECTION C

DISEASES OF DUCKS, GEESE, AND MISCELLANEOUS BIRDS

CHAPTER XVII

DISEASES OF DUCKS

DUCKS are subject to more or less the same infections as the domestic fowl, but the course of the diseases may vary considerably. Due to their habitat and other peculiarities, some diseases may become more virulent or common than others. Thus, ducks are more resistant to Newcastle disease than the domestic fowl. Ducks are more commonly infected with *Salmonella* than the domestic fowl. The common *Salmonellae* infecting ducks are *Salmonella pullorum*, *S. gallinarum*, *S. anatum*, *S. enteritidis*, and *S. typhimurium*. It is not uncommon to find duck eggs infected with *Salmonella*. Similarly, as ducks live in a more humid environment than the domestic fowl, the adults are more subject to infections with *Aspergillus* spp., *Listeria*, *Pasteurella multocida*, and *E. coli* which are common in ducks. They are infected with a variety of acanthocephalids and trematodes, because they feed more on molluscs and fish which are in most instances the intermediate hosts of this group of helminths.

They are also more commonly affected with botulism (especially wild ducks), muscular dystrophy due to vitamin E deficiency (especially ducklings), and sarcosporidiosis.

VIRAL INFECTIONS

DUCK VIRUS HEPATITIS

This is a highly infectious acute fatal disease affecting primarily ducklings and causing a high mortality reaching 90 per cent. In acute cases death occurs within a few hours of the manifestation of symptoms. Usually the dead ducklings have their heads drawn back. The disease is characterized by sudden onset, rapid spread, acute course, and an enlarged liver containing punctiform haemorrhages. The kidneys and spleen may be similarly affected.

The disease is caused by a virus which does not produce disease in the domestic fowl but causes stunting and oedema of chick embryos and a greenish-yellow discolouration of the liver with necrotic foci. The disease has been reviewed by Asplin and McLauchlan (1954). There appear to be several immunological strains of the virus.

The disease may be controlled by immunizing the breeder ducks with the virulent virus type or types that are causing disease on the farm or in the area, and by treating the contacts in an outbreak of disease with 0·5 ml. of duck virus hepatitis antiserum given intramuscularly.

Day-old ducklings can be protected by the foot stab vaccination method, giving adequate immunity for the duration of the susceptible period, i.e., 5–6 weeks. The vaccine is a live virus attenuated by chick embryo passage, and is administered by stabbing a needle dipped in vaccine through the foot web.

Duck Plague

This is a highly fatal peracute viral disease of ducks characterized by petechial haemorrhages in the ovary, mesenteries, liver, oesophagus, and other organs. This disease was first observed in the Netherlands in 1923, but recently it has been reported from West Bengal in India (Mukerji, Das, Ghosh, and Ganguly, 1963a, b; 1965) and in Belgium in 1964; Jansen (1961) has given a full description of the disease. It is perhaps present in China and France as well. Recently several outbreaks have been reported in Long Island, New York State, U.S.A. (Leibovitz and Hwang, 1968).

Cause.—Two strains of a virus, the O (old) strain which is more virulent and the W (Wageningen) strain isolated from recent outbreaks are responsible for the disease. The virus is immunologically and pathologically distinct from the viruses causing fowl plague, Newcastle disease, and duck virus hepatitis. The two serotypes causing the disease have been designated type I and type II.

Clinical Signs.—The incubation period under experimental conditions appears to be 3–4 days. The birds become dull, there is thick discharge from the eyes and nostrils, and the eyelids become swollen and stick together. There is a greenish-white watery diarrhoea. The course of the disease varies from 1 to 3 days. Birds may show an apparent improvement in the condition prior to death. The disease is usually acute or peracute and it appears to be limited in incidence to localities with ponds, moats, and stagnant water. In some cases there are tremors and paralysis, and birds will sit on their breast with drooping wings touching the ground.

Post-mortem Lesions.—The carcase shows sticky eyelids and dirty nostrils. There are multiple petechial haemorrhages on the body especially on the heart, serous membranes, liver, and oesophagus. The ovary is inflamed and shows haemorrhagic patches or is congested. Diphtheritic membranes are seen on the oesophagus and the cloaca in less acute cases.

The disease has to be differentiated from Newcastle disease, fowl plague, and duck virus hepatitis.

Prevention and Control.—Being a peracute virus disease, no satisfactory treatment is possible. Jansen (1964) has reported on the use of a chick embryo vaccine in the Netherlands. This can be given in drinking-water. Two doses of attenuated virus vaccine given 6 weeks apart to parent stock produces a high level of immunity in offspring up to 9 months.

Duckling Virus Infection

An apparently new virus disease of 3–5-week-old ducklings characterized by nephritis and hepatitis has been reported by Mansi, Schofield, and Gonzalez (1964).

The virus differs from that causing duck virus hepatitis. Unlike duck plague virus, the new virus affects ducklings mainly.

Duck Influenza

This is an infectious sinusitis of ducks caused by several strains of *Myxovirus* related to the human influenza virus. The virus has been described from ducks in Britain, Czechoslovakia, and the Ukraine. The British virus has been cultivated in tissue cultures.

Duck Pox

Duck pox has been reported from time to time in various parts of the world. Recently Rao (1965) has reported it from Andhra Pradesh in India. The virus causing duck pox appears to be different from those causing fowl pox and pigeon pox and according to Rao (1965) these two viruses will not infect ducks.

The disease resembles fowl pox in the domestic fowl, there being a cutaneous form and an ocular form. The scabs are large and distinct. No work has been done on the various aspects of immunity of this disease.

Muscovy Duck Disease

A fatal viral disease affecting only Muscovy ducks (*Cairina moschata*) was described in Natal by Kashula (1950). The disease was characterized by an incubation period of 2–4 days, diarrhoea, necrotic spots in liver and spleen, and pulmonary oedema. The main symptoms are complete loss of appetite, pale legs and beak, and high temperature.

Spleen Necrosis of Ducks

A disease fatal to young ducklings in 3–5 days caused by a filter-passing agent has been described by Trager. There was severe anaemia with necrotic lesions in the spleen and hepatitis. The virus was different from that causing duck virus hepatitis. There is a marked drop in temperature by crisis.

BACTERIAL INFECTIONS

Most of the bacterial infections have been referred to earlier and are similar to those seen in the domestic fowl, with the exception of *Moraxella anatipestifer* infection.

Anatipestifer Infection
(*Infectious Serositis; Duck Septicaemia; New Duck Disease*)

This disease is caused by *Moraxella anatipestifer* (*Pasteurella anatipestifer*) and is usually seen in growing ducklings from 4 to 9 weeks old and mortality may reach 75 per cent (Blaxland, 1960). The affected birds show inappetance, greenish diarrhoea, sneezing, a purulent discharge from the eyes and nostrils, muscular incoordination, and continuous nervous movement of the head and neck. The principal lesions are fibrinous pericarditis, peritonitis, and perihepatitis which

characterize the infection. A yellow membrane envelopes the liver which is often enlarged. There is oedema of the tissue surrounding the coronary groove, enlarged mottled liver, and petechiae in the abdominal viscera. The disease can be controlled by giving 100 g. chlortetracycline per ton of mash for 5 days or its equivalent in water. Sulphadimidine in water is also said to be effective at a level of 0·1 per cent for 2 days. This may be repeated after an interval of 3 days. A combination of penicillin and streptomycin is more effective than oxytetracycline.

Western Duck Sickness

This is another name given to botulism affecting wild water fowl in Western America and Canada, which is characterized by flaccid motor paralysis and stretched-out neck.

Sarcosporidiosis

Wild mallard duck are particularly susceptible to infection with Sarcosporidia and the breast muscles may be very severely infected. The species affecting ducks is *Sarcocystis rileyi.*

Leucocytozoon Simondi Infection

This is a common parasitic infection of the duck, especially in Canada and certain parts of U.S.A. The principal transmitter of the infection in Canada is *Simulium anatinum.*

Coccidiosis of Ducks

The most pathogenic species of coccidium appears to be *Tyzzeria perniciosa* which may cause high mortality in baby ducks. Affected birds show the usual signs of illness and may also cry continuously.

The whole of the small intestine is affected. Pathogenicity appears to resemble that of *Eimeria necatrix* in the domestic fowl.

Treatment, Prevention, and Control.—Very little work has been done on this aspect and therefore the same procedures as described under this section in the coccidial infections of the domestic fowl may be followed.

Duck Malaria

Ducks may be infected with *Plasmodium lophurae*, *P. elongatum*, *P. fallax*, and *P. hexamerium.* Many species of mosquitoes transmit these Plasmodia. *P. lophurae* is chiefly transmitted by *Anopheles quadrimaculatus* whereas the other species are chiefly transmitted by *Aedes aegypti.* The disease resembles that seen in the domestic fowl and may be treated in a similar manner.

CHAPTER XVIII

DISEASES OF GEESE AND MISCELLANEOUS BIRDS

GEESE

Goose Septicaemia

This is caused by *Pasteurella septicaemiae* and is common in central Europe. Goslings are primarily affected. The disease is characterized by fibrinous exudation from the serous membranes, liver, and kidney, and congestion, oedema, and dilatation of the heart. The usual mode of transmission is by direct contact.

Goose Plague in the Netherlands

Van Cleef and Mietenburg, in 1965, reported an acute viral infection of geese in Netherlands causing 90–100 per cent morbidity and 10–90 per cent mortality, and characterized by scaling of the skin, beak, and legs, a seromucous discharge from the eyes and nose, and severe diarrhoea. In the subacute cases, there was emaciation and deeply corrosive, yellow areas of necrosis in the oropharynx and on the tongue. Post-mortem examination revealed haemorrhages in the lungs, liver, and gizzard. Chickens and ducklings were not susceptible to infection.

Gosling Virus Infection

Krauss (1965), in Germany, reported an acute, highly fatal, infectious disease in goslings aged 6–8 days, characterized by whitish diarrhoea, nasal discharge, gasping, difficult breathing, pasted eyelids, and coma, followed by death. Though a cytopathogenic agent was isolated it was not possible to determine the exact aetiology.

Gosling Influenza

A disease causing an influenza-like syndrome has been described in goslings in Hungary (Csontos and Castari, 1967). This is probably initiated by a virus which multiplies in the intestines causing enteritis, loss of appetite, and retarded growth. *Haemophilus anseriscepticus* appears to act as a secondary invader to produce the characteristic signs of influenza.

Under natural conditions maternal antibodies appear to be passed in the yolk.

FALCONS AND HAWKS

Trichomoniasis
(*Frounce*)

This is caused by various species of *Trichomonas* and is characterized by necrotic lesions in the mouth, oesophagus, and crop. Falcons usually contract the infection from pigeons, which are often carriers. The trichomonads are easily seen

on post-mortem examination of a fresh carcase. However, if the carcase is old, the trichomonads disappear quickly. The infection may be treated with 2-amino-5-nitrothiazole or nithiazide.

The other common infections of this group of birds are:—

1. Aspergillosis.
2. *Serratospiculum amaculata* infection.
3. Coccidiosis.

OWLS

Rickets

This is one of the most common diseases of owls and is caused by improper feeding. Correction of diet with attention to proper nutrition often prevents the disease. Fractures are a frequent result of rickets.

PHEASANTS AND PARTRIDGES

These birds are susceptible to:—

1. *Salmonella* infection.
2. Omphalitis.
3. Coccidiosis.
4. Cannibalism.

SECTION D

COMMON DISEASES OF CAGE BIRDS

CHAPTER XIX

DISEASES OF CAGE BIRDS

CAGE birds are becoming increasingly popular. The diseases of these birds are described briefly in three parts in tabular form. *Table* 17 deals with diseases of budgerigars, parrots, and parakeets, *Table* 18 with those of the canary, and *Table* 19 with those of the pigeon, the dove, and their relatives.

Canary pox is an important disease in cage birds, and can decimate a flock of birds. Detailed studies of canary pox in Japan have been made by Sato, Sugimori, Ishii, and Matumoto (1962a, b). Pox-like lesions in birds may also be caused by ectromelia virus.

By far the most popular cage bird is the budgerigar and further information on the diseases of the budgerigar and other popular cage birds is given by Jones (1956), Arnall (1960, 1961a–d), Stone (1960), Friedberg (1961a, b), and Frost (1961), while the common surgical conditions are described by Keymer (1960a, b), Michael (1960), Arnall (1961a–d) and Hasholt (1961).

Salmonelloses have become an increasing problem in cage birds. *S. typhimurium* is a very common pathogen. The public health importance of these infections will have to be borne in mind.

Because most cage bird owners treat their own pets, the average veterinarian is more often called upon to treat surgical cases in budgerigars. The most common diseases in cage birds appear to be enteritis, pneumonia, and trichomoniasis.

The Japanese quail is becoming increasingly popular and some of its diseases have been described recently by Bigland, Damassa, and Woodard (1965).

BUDGERIGARS, PARROTS, AND PARAKEETS

Beach (1965) states that the incidence of infectious diseases in the budgerigar is remarkably low, while the incidence of neoplastic diseases and those very probably due to nutritional causes is high.

In pet budgerigars, neoplasia is the most common single cause of death, affecting more than 30 per cent of those examined at post-mortem. The common tumours of budgerigars consist of ovarian and testicular tumours, nephroblastoma, leiomyosarcoma, and lipoma (Blackmore, 1966).

Surgical approach to this problem has been discussed by Graham-Jones (1966) and Arnall (1966). Ether and halothane are the most satisfactory volatile anaesthetics. A 0·5–1·5 per cent concentration of halothane produces anaesthesia of sufficient depth. A Boyle's apparatus may be used for the purpose of obtaining the correct concentration of the anaesthetic. Of the non-volatile anaesthetics, thiopentone and pentobarbitone are useful. The dosage rate of either drug is 30 mg.

per kg. The anaesthetic is given preferably by intraperitoneal injection. The injection is made anterior to the cloaca in the direction of the head, midway between the cloaca and the sternum in the midline. The needle should be nearly parallel to the abdominal wall. With pentobarbitone, anaesthesia will last 45–60 minutes only. Details of the sites where the tumours occur and the operative techniques recommended have been discussed by Arnall (1966).

Surgical treatment of ovarian cysts and tumours, egg binding, and prolapse of the oviduct of cage birds is described by Hasholt (1966). Prolapse of the oviduct and retention of the egg were the most common conditions encountered. The reduction of the prolapse may be done easily with a probe or thermometer.

One of the major problems of the breeders of budgerigars is the entity known as 'French moult' the exact cause of which is not known. However, this condition appears to be due to a deficiency of the proventricular secretion with which the hen birds feed their young during the first few weeks of life. This condition can be prevented by avoiding breeding for 6 months or more. Thyroid disease is another common condition and is caused by thyroid hyperplasia or dysplasia due to a deficiency of iodine in bird-seeds. This condition can also be prevented by giving iodine in traces (*see* Goitre in *Table* 17).

The salient features of the common diseases of these birds are summarized in *Table* 17.

The diseases have not been classified but are listed separately in alphabetical order.

The budgerigar (*Melopsittacus undulatus*) is a native of Australia and is one of the most popular cage birds. It belongs to the family Psittacidae of the order Psittaciformes. The Psittacidae include the parrots and parakeets.

The average body-weight of the budgerigar is 1–2 oz., the normal body temperature averages 108° F. (42·2° C.), the incubation period averages 18 days, and the clutch size is 5–6 eggs, each egg being laid at intervals of 2 days. The hen commences incubation with the laying of the second egg.

Parrots and the parakeets belong to the family Psittacidae and are closely related to the budgerigar. To this family also belong the macaws, the love birds, cockatoos, and the lorikeets.

THE CANARY

The canary (*Serinus canarius*) belongs to the family Fringillidae of the order Passeriformes and belongs to the group of seed-eating passerines. The finches, buntings, sparrows, and cardinals are closely related species.

Before *Plasmodium gallinaceum* was described by the late Professor Emile Brumpt the canary was extensively used in research on malaria. The common diseases of the canary are tabulated in *Table* 18. Besides these, it may suffer from a number of other incompletely studied diseases such as listeriosis, *E. coli* septicaemia, *Leucocytozoon* infections, helminthiasis, and others. A very common and often fatal disease of the canary is canary pox.

The average body-weight of the canary is $\frac{3}{4}$ oz., the normal body temperature averages 110° F. (43·3° C.), the incubation period is 13–14 days, and the clutch size is 4–6 eggs, laid in batches of 2–3 per year.

The canary may also be infected with malaria. This is caused by several species of *Plasmodium*, namely *P. relictum*, *P. cathemerium*, *P. elongatum*, *P. vaughani*, *P. hexamerium*, and other species of *Plasmodium*. Various species of mosquitoes may

Table 17.—COMMON DISEASES OF BUDGERIGARS, PARROTS, AND PARAKEETS*

DISEASE	CAUSE	CLINICAL SIGNS	DIAGNOSIS	POST-MORTEM LESIONS	TREATMENT	REMARKS
Alopecia	Unknown, possibly of dietetic origin. Frequently a sequel to self-plucking of feathers or cannibalism. Psychotic. Experiments with the domestic fowl indicate that this condition may be due to parathyroid hormone deficiency	Bare patches chiefly on head. When not traumatic, unassociated with any lesions	Based on clinical signs	—	Treat cause and give vitamins, etc. Pay attention to diet. Ataractics may be tried, e.g., promazine hydrochloride	—
Arthritis	*Escherichia coli*	Lameness	Based on signs	Polyarticular arthritis of the serofibrinous and seropurulent type	Tetracyclines may be useful	Due either to direct infection or septicaemia
Aspergillosis	*Aspergillus fumigatus*	Sometimes dyspnoea, often vague	Based on lesions and isolation of fungus at autopsy	Typical necrotic nodules, often laminated or showing green hyphae formation involving lungs and air sacs, button ulcers or caseous nodules in lungs are typical	*Prevention:* for parrots 2·5 grains KI dissolved in 2 oz. drinking-water daily or every other day	Nystatin aerosol may be useful. Parrots are commonly affected
Beak, deformity and overgrowth of	Sometimes *Knemidocoptes pilae* (budgerigars). Debility. Dietary deficiencies and imbalances. Congenital	Overshot or undershot beak. Flakiness and brittleness of mandibles. Wry beak and scissor bills	Scrapings will reveal mites	—	Benzyl benzoate for parasitic infestation. Trim mandibles with nail cutters. Attention to diet. Provide branches and hard objects to chew, vitamin supplements, and grit	Sometimes associated with hyperkeratosis of the mandibles or with rickets. Budgerigars and aged parrots primarily affected
Claws, overgrowth of	Lack of sufficient wear	Excessively long claws often becoming curled and twisted. Liable to get caught in bars of cage, etc. resulting in fracture	Based on clinical signs	—	Trim with nail cutters taking care to avoid cutting blood-vessels in claws; provide natural perches varying in diameter	All species, especially caged budgerigars

Crop, impaction of	Probably in most cases due to debility and lack of tone in crop wall	Fluctuating swelling in the region of the crop. In small species, seed often visible through skin and crop. Attempts at regurgitation. Displacement of the crop dorsally	Based on clinical signs	—	Surgical. Attention to diet. Vitamin supplements. In small species evacuation of the crop under anaesthesia. Massage may be useful. Poor prognosis for small species. Administer glucose i.p.	—
Crop necrosis	Unknown	Regurgitation of slimy green fluid, mucoid diarrhoea, and general malaise	Based on lesions found at autopsy	Necrotic areas in crop, covered by yellow diphtheritic membrane resembling Turkish towelling	Penicillin is said to be useful	This condition may be mistaken for salmonellosis and is common in aviaries but is seldom seen in birds kept individually
Cysts, dermal	Unknown	Single or multiple tumour-like growths affecting skin, commonest on wings. Sometimes pedunculated or ulcerated. Contents yellow, granular, and sebaceous	Based on appearance of lesions	—	Complete surgical extirpation	Common in budgerigars
Egg binding	Lack of muscular tone of oviduct. Most frequent at beginning of breeding season. Excessive egg-laying or breeding at unsuitable time of year	Sudden onset; straining; distress; swelling around vent; cloacitis; ruffled feathers; squatting on flat surface	Based on signs in hen bird, especially when young. Palpation of egg through abdomen. Radiography	Egg impacting oviduct. Usually a shelled egg in lower posterior portion of the organ	Raise the temperature of the cage to 70–90° F. Maintain this temperature for 2–3 hr. If the birds show distress at any time, the temperature must be lowered rapidly. Last resort, mechanical removal, preceded by lubrication of cloaca	—
Enteritis	Sometimes associated with specific infectious diseases; otherwise cause unknown. In some cases probably due to ingestion of contaminated or unsuitable food or water. Sometimes associated with Gram-negative flora in gut, e.g., *E. coli* and *Salmonella*	Alopecia, restlessness, plucking feathers with beak, scratching, shivering, and general signs of nervousness, diarrhoea	Based on clinical signs and post-mortem lesions	—	Attention to diet. Chloramphenicol given orally is useful. Coat seeds with drug to facilitate administration	—

*Tables 17–19 are adapted from the *Handbook on the Treatment of Exotic Pets, Part I* by kind permission of the British Veterinary Association.

Table 17.—COMMON DISEASES OF BUDGERIGARS, PARROTS, AND PARAKEETS (*continued*)

DISEASE	CAUSE	CLINICAL SIGNS	DIAGNOSIS	POST-MORTEM LESIONS	TREATMENT	REMARKS
Fits	Various, e.g., sequel to infectious disease, enteritis, of unknown origin, heart failure, neoplasia (e.g., of pituitary), goitre	Bouts of fluttering around floor of cage. Sometimes utter high pitched squeaks. Fluttering wings. Partial paralysis of limbs. Torticollis, etc.	Based on clinical signs and history	—	Depends on cause. Quietness. Subdued light. Sedatives. KBr 1 g. in 1 oz. drinking-water	—
French moult	Probably nutritional. Likely to be caused by excessive breeding and reduced supply of stomach milk or proventricular secretion	Disease of nesting birds characterized by shedding or breaking-off of primary and secondary wing feathers	Based on clinical signs	The clinical signs are the only changes seen at post-mortem examination	Breeding should be stopped for about 6 months	—
Feathers, self-plucking	Variable: vice, boredom, parasitism, dietary deficiency, psychotic, etc.	Alopecia and plucking feathers	Based on clinical signs and history	—	Counteract boredom. Remove cause. Vitamin B_{12} injections and ataractic drugs may be useful	—
Goitre	Hormonal, possibly caused by stress of rearing young. Iodine deficiency	Chronic course; unwell for approximately 2 months before death; fits, appetite normal, obesity. Commonest in hens. Continuous high-pitched squeaking noises with respiratory rhythm	Based on history and post-mortem lesions	Enlargement of thyroids. Thyroids often show yellowish specks on their external surface. Congestion of all internal organs	Supply iodized mineral blocks and prevent overbreeding. 1–2 drops of colloidal iodine in ½ oz. drinking-water	Common in budgerigars and the incidence is particularly high in pet budgerigars. Bird-seeds are deficient in iodine
Haemorrhage	Spontaneous rupture of a blood-vessel, organ, or haemangioma	Sudden death	Based on post-mortem lesions	Liver is the commonest site. Also occurs in lungs, spleen, etc. Blood-clot in body cavity	None	—
Hatchability poor and increased embryonic mortality	Infertility of one or both parents, debility, immaturity, or senility. Failure of hen to incubate properly and subsequent chilling. Nutritional deficiencies, e.g., vitamin B complex, vitamin E, and manganese	Failure of one or more eggs to hatch	Based on clinical signs. Examination of eggs and embryos. Bacteriological examination to eliminate the possibility of *Salmonella* infection	Clear eggs. Deformity or stunting of embryos	Depends on cause	—

Heart disease	Senility	'Fainting', collapse, and complete or partial recovery. Increasing in frequency eventually causing death	Based on clinical signs and post-mortem lesions	Dilated, thin-walled heart. Pale myocardium and auriculoventricular valves. Oedema of lungs. Atheroma or arterio-sclerosis	Quietness. Glucose, nikethamide, 0·25 ml. parenterally	Other species may faint from shock, but will usually recover spontaneously
Liver, fatty degeneration of	Unknown, probably dietetic	Usually the condition is associated with fatty birds	Made at post-mortem examination	The liver is enlarged, fatty, and friable. The subcutaneous fat is abundant	Reduce diet; no specific treatment	—
Mite, red	*Dermanyssus gallinae*	Skin irritation. Restlessness at night; anaemia and death	Identification of mites in feathers at night or in cage	Anaemia. Mites in crop contents	Thorough disinfection and treatment of cage with gamma benzene hexachloride. Isolate birds and transfer to clean quarters during daylight. Care essential if bird dusted with insecticides	—
Neoplasia	Unknown	Unreliable. Usually malaise and usually chronic. Loss of condition. Swellings of integument, abdomen, and abdominal organs. Partial or complete paralysis of limbs. Change of sex. Bony swellings of wings and legs	Clinical signs. Palpation. Radiography. Biopsy. Post-mortem lesions	Vary according to type. Most internal organs can be affected. Kidneys commonest site	Surgery in some cases	Common in budgerigars. They may account for nearly a quarter of all deaths. Lipoma appears to be a common neoplasm
Nephritis	Unknown	Debility, malaise sometimes polydipsia, excessive excretion of white droppings (urates)	Based on clinical signs and post-mortem lesions	Congestion and swelling of kidneys in acute cases. Subacute and chronic cases paleness of kidneys. Excessive amounts of urates. Sometimes lesions of visceral gout, i.e., uratic deposits in viscera, chiefly pericardium, peritoneum, and in joints	Attention to diet; reduction of protein. 1–2 gr. hexamine dissolved in 2 tablespoonfuls of drinking-water. Initial antibiotic therapy	May be associated with debility and sometimes with specific diseases
Obesity	Incorrect diet; possibly hormonal or digestive dysfunction	Excessive fatness, lethargy, weakness in flight	Based on appearance, clinical signs, and palpation of bilateral deposition of fat subcutaneously	Excess deposition of subcutaneous and abdominal fat. Fatty degeneration of liver and myocardium	Attention to diet. Liquid thyroid extract	—

Table 17.—COMMON DISEASES OF BUDGERIGARS, PARROTS, AND PARAKEETS (*continued*)

DISEASE	CAUSE	CLINICAL SIGNS	DIAGNOSIS	POST-MORTEM LESIONS	TREATMENT	REMARKS
Oesophagus, crop, and epithelium, inflammation and necrosis of	Unknown	Acute or subacute condition. Vomition of mucus resulting in matting of feathers around beak with vomit. Often polydipsia and diarrhoea. General malaise	Based on clinical signs	Severe inflammation of oesophageal and crop mucous membrane with longitudinal raised and parallel lines of yellowish necrosis	Oxytetracycline hydrochloride in the drinking water. Or ½ ml. medicated water containing antibiotic every 4 hours or 1 drop chloramphenicol palmitate every 6 hours	—
Psittacosis (ornithosis)	PLV *See* Psittacosis and Ornithosis, p. 40	None in latent infections. Sleepiness, ruffled feathers, greenish diarrhoea, dyspnoea, ocular and nasal discharge, loss of weight. Wings may droop and attacks of shivering occur. Disease may progress and end in convulsions, sudden death, or slow recovery	Isolation of PLV from discharges or carcase. Tissue impression smears. Complement fixation test. History of recent importation. Associated with human illness	Congested and enlarged spleen. Congested liver with rounded edge and sometimes foci of necrosis. Serous or fibrinous pericarditis	Flock treatment for budgerigars in contact: 2 mg. aqueous tetracycline intramuscularly for 15 days	Treatment usually inadvisable owing to danger of human infection. A bird may remain latently affected for months or even years. It is advisable to quarantine recently imported birds for at least a month. Source of infection to humans: airborne transmission. Numerous species of psittacines are susceptible
Respiratory system, disorders of: sinusitis, bronchitis, congestion of lungs, pneumonia, infection of air sacs and 'asthma' of parrots	Sometimes associated with specific infection, e.g., *Mycoplasma*, Staphylococci, *Aspergillus fumigatus*, psittacosis, etc. Chilling and draughts probably predisposing causes	Various: conjunctivitis, nasal discharge, gaping, contracting and extending neck, emitting high pitched squeaks, loss of voice, dyspnoea, holding wings away from side of body, and pumping action of tail	Based on clinical signs and post-mortem lesions	Excess mucus in sinuses; congestion and oedema of lungs, thickening and cloudiness of air sacs, caseous material in air sacs, and necrosis in lungs, especially in cases of aspergillosis	Depends on cause. Inhalation of Tinct. Benz. Co. Administration of oxygen. Injection of antibiotics	—
Salmonelloses	*S. typhimurium* (*S. pullorum* and *S. thompson* also reported)	Malaise, frequently diarrhoea, sudden death preceded by convulsions. Heavy losses. In parrots, thirst, vomiting, dysentery	Based on bacteriological examination of carcase or droppings. Rapid blood or tube agglutination tests	Lesions not diagnostic. Enlargement, congestion and necrosis of liver and spleen. Catarrhal enteritis in parrots	Chloramphenicol by intramuscular injection or orally. Tabs. nitrofurazone or furazolidone. Dose ¼ tablet or 25 mg. dissolved in 8 oz. drinking-water	Source of infection: eggs, other birds, and rodents. Owners should be warned of possible transmission to humans

Scaly face and scaly leg	*Knemidocoptes pilae*	Typical raised yellow encrustation on cere and beak and around eyes. Less commonly on legs, digits, and skin of vent. Sometimes beak deformity	Based on lesions plus identification of mites from scrapings	—	After removal of scabs, benzyl benzoate 10 per cent emulsion applied daily for 3 days. Single application 7 days later; or 0·02 per cent gamma B.H.C. in soft paraffin or arachis oil	See also Beak deformity, p. 180
Scaly leg	*Knemidocoptes jamaicensis*	The legs are scaly	Based on lesions and identification of mites	—	—	Seen in the goldfinch
Thyroid disease	Iodine deficiency	Enlarged thyroids, loss of feathers, fits	Enlarged thyroids	Enlarged thyroids and thyroid hyperplasia or dysplasia	Iodine supplement or mineral mixture containing iodine	The incidence is particularly high in pet birds as all bird-seeds are deficient in iodine

act as intermediate hosts. *Culex pipiens* appears to be an important vector of *P. relictum*.

Nephritis is a common condition in canaries and finches. This is often characterized by deposits of urates in the kidney tubules and the visceral organs. *Listeria monocytogenes* is another common organism which causes abscesses in the liver and spleen.

PIGEONS AND DOVES

The common domestic pigeon, *Columba livia*, and its relatives, the other pigeons and doves, belong to the family Columbidae in the order Columbiformes.

The common diseases of the domestic pigeon and its near relatives are given in *Table* 19. Besides those listed in this table, pigeons and doves are frequently infected with the protozoan parasite *Haemoproteus columbae* which is normally seen in the blood. Though it is not considered a very harmful parasite, heavy infections in young birds may cause some losses.

The principal transmitter of the parasite is the degenerate fly *Pseudolynchia canariensis*, which can cause considerable harm to fledglings if the infection is heavy, because it sucks large amounts of blood. These flies can be controlled by the judicious use of D.D.T., malathion, and pyrethrins. The pupae laid by these flies should be picked up and destroyed. Capillariasis is a widespread infection in pigeons.

The average weight of an adult pigeon or dove is about 1·5 lb. and the normal temperature varies from 105–109° F. (40·5–42·2° C.). The clutch size is 2, the second egg is laid 48 hours after the first. The incubation period varies from 15–19 days depending on the species. On the fifteenth or sixteenth day of incubation both parents secrete crop milk. The squabs are fed entirely on this secretion on the first 4 days after which they are trained to feed on small grain.

Table 18.—COMMON DISEASES OF THE CANARY

DISEASE	CAUSE	CLINICAL SIGNS	DIAGNOSIS	POST-MORTEM LESIONS	TREATMENT	REMARKS
Alopecia	Unknown: probably nutritional, hereditary, or related to debility	Baldness, especially common on head and neck usually no inflammation when not traumatic in origin	Based on clinical signs	—	Attention to diet, with addition of greenfood. Vitamin supplements. Advise remanagement. Bathing facilities	Sequel to feather picking
Feather picking (cannibalism type)	Overcrowding, unhygienic conditions, occasionally ectoparasitism	Alopecia with inflammation of skin. Parent birds may peck nestlings. Immature canaries of about 1 month of age when kept together sometimes peck one another	Based on clinical signs and circumstances under which birds are kept	—	Separation of offending birds, treatment of wounds and removal of evidence of blood, debeaking	May become a vice
Lice	*Docophorus communis*, *Ballicola ortygometrae*, *Bruelia delicata*, and *Menacanthus* spp.	Irritation, scratching, frequent ruffling up of feathers, restlessness	By identification of parasites on plumage	—	Dust feathers with 0·5 per cent pyrethrum powder or spray with an aerosol containing pyrethrum synergized with piperonyl butoxide	—
Mite (Red) infestation	*Dermanyssus gallinae*	As for lice. Mites attack at night. Anaemia at times leading to death	By identification of parasites on plumage or in living quarters especially in nest boxes	Anaemia. Mites sometimes found in crop	See Lice above	—
Moult, abnormal (stuck-in-moult)	Believed to be due to sudden changes of temperature, shock while moulting, or incorrect diet	Shedding of feathers checked during normal moulting, resulting in prolongation of normal period of moult (i.e., 8–10 weeks)	Based on clinical signs	—	Correct management and feeding	—
Pox (canary)	Virus	Pustules and scabs or wart-like growths on head, margins of eyes, body, and legs. In acute cases there is respiratory distress, involvement of the upper respiratory tract, and septicaemic signs	Based on lesions and virus isolation	The pox-like lesions and high mortality and morbidity are characteristic	No specific treatment. Local antiseptics and antibiotics given orally in drinking-water may be used to control secondary invaders	This disease is becoming increasingly important and widespread

Table 18.—COMMON DISEASES OF THE CANARY (*continued*)

Salmonelloses, paratyphoid, septic fever	*S. typhimurium* (*S. cholera suis*, *S. suipestifer*, *S. pullorum*, and *S. anatis*). Common source of infection in egg food	Incubation period 4–5 days. Death after 2–4 days of illness. Ruffled plumage, greenish diarrhoea. Convulsions frequently precede death. Very contagious. May cause heavy losses in aviary. Often capricious appetite	Based on bacteriological examination	Marked congestion of internal organs, enteritis, enlarged liver and spleen sometimes showing petechiae, often extensive pneumonia	*See* under Salmonelloses, *Table* 19	—
Toe, slipped (slipped claw or stiff claw)	Unknown. Possibly dietetic, especially vitamin B complex deficiency	The hind claw or digit (usually of one foot), becomes partially paralysed and curls forward under the foot	Based on clinical signs. Occurs at 6–10 weeks of age, seldom seen in adults	—	Provision of padded perches of small diameter or removal of perches. Soft flooring in cage. Vitamin B complex	—

Table 19.—COMMON DISEASES OF THE DOMESTIC PIGEON AND ITS RELATIVES

DISEASE	CAUSE	CLINICAL SIGNS	DIAGNOSIS	POST-MORTEM LESIONS	TREATMENT	REMARKS
Aspergillosis	*Aspergillus fumigatus*	Sometimes dyspnoea but frequently no obvious clinical signs. Usually a chronic condition but sometimes relatively acute	Based on post-mortem lesions and isolation of fungus	Lesions usually confined to the respiratory system. Small necrotic nodules in lungs may coalesce and form large areas of consolidated necrosis. Varying degree of congestion of lungs. Necrotic plaques or plugs in air sacs. Sometimes mycelia seen	Nystatin aerosol may be used. As a control measure fumigate with formaldehyde using 5 ml. formalin and 3 g. of potassium permangenate per cu. ft. of air space for 30 minutes	For control pay strict attention to hygiene and removal of all damp and mouldy litter, droppings, food, and other organic matter. Loft must be kept dry
Bumblefoot	Usually localized staphylococcal infection	Swelling of ball or soft pad of foot, which in some cases may not be obvious unless the bird is handled and the foot closely examined. Lameness	Based on clinical signs and bacteriological examination of swab of opened lesions	Incision of lesions may show synovitis with production of yellowish synovia. In chronic cases formation of inspissated pus, caseous, and necrotic material	Surgical, with local antibiotic or antiseptic treatment. Fomentation, application of iodine, antibiotic or sulphathiazole therapy	—
Conjunctivitis, coryza, roup, eye cold, one-eyed roup, ophthalmia	Various: one authority considers that some cases may be due to a vitamin A deficiency. *Mycoplasma* infection may be a contributory cause in some cases	Watery ocular discharge. Swelling of periocular region. Chiefly affects birds under one year of age. Apparently contagious. When recovery takes place birds are seldom affected again. Usually only one eye is affected. Bird often rubs eye on shoulder. A similar condition has been seen in the domestic fowl	Based on clinical signs	—	Depends on cause. Palliative treatment of eyes with antibiotic eye ointments, hygiene, and attention to feeding	The condition, which is a well recognized entity with pigeon fanciers, may not be a specific disease
Lice infection or pediculosis	Several species of lice	Heavy infestations normally present only on debilitated and diseased birds	By identification of the dorsoventrally flattened brownish-yellow insects in plumage. Feather lice may be readily seen by holding the wing and tail against the light	—	General management, correct feeding. Add 1 oz. of sodium fluoride to 1 gal. water. Withhold bath for 2–3 days prior to treatment. Proprietary aerovac in loft, or dusting with 10 per cent D.D.T. powder or spraying with 10 per cent D.D.T. sol.	Entire life cycle spent on host and completed in 2–3 weeks

Table 19.—COMMON DISEASES OF THE DOMESTIC PIGEON AND ITS RELATIVES (*continued*)

DISEASE	CAUSE	CLINICAL SIGNS	DIAGNOSIS	POST-MORTEM LESIONS	TREATMENT	REMARKS
Poisoning by chlorinated hydrocarbons	Seed dressed with insecticides, e.g., Dieldrin	Become ill about 48 hr. after eating dressed corn. Listless, loss of appetite, incoordination, erratic flight and may actually fall dead when flying, nervous signs most marked 2 hr. before death, side to side movements of head and slight tremor, blinking of eyelids	From clinical signs, history, general post-mortem picture, together with absence of specific disease. Diagnosis based on circumstantial evidence. Detection of poison in carcase fat	Pectoral muscles rather wasted. Internal organs rather congested. Horny lining of gizzard usually easily detached from underlying muscle. Digestive tract usually empty except for some grit in gizzard. Histological examination: deposits of haemosiderin in liver and kidneys	Emergency: immediate evacuation of crop	—
Pox	Virus	Vesicles or pustules develop on skin of almost any part of body, although chiefly confined to eyelid, near beak, and on feet and lower part of leg, less frequently seen on wings and around cloaca. Lesions eventually become hard brownish granulomatous growths causing lameness when on feet. Sinusitis, conjunctivitis, keratitis, and blepharitis also described but this appears to be less common. Diphtheritic lesions in mouth	Clinical signs, lesions, age of bird, and isolation of virus from lesions. Histopathology of lesions	No lesions in internal organs	Dress lesions daily with liquor iodi fortis. Remove scabs with care to prevent haemorrhage. Large lesions may take 6 weeks to heal. Pigeon pox vaccine is unsuitable for vaccinating pigeons	Mortality low, usually under 5 per cent
Salmonelloses	*Salmonella typhimurium*. Other species of *Salmonella* occasionally encountered are: *S. cholerae suis, S. abortus equi, S. anatum, S. california, S. oranienburg, S. montevideo, S. dublin,* and *S. canoga*. Poor management and hygiene are contributory factors	Poor hatchability (dead in shell). Young: usually acute. Squabs less than 15 days chiefly affected. Death within 6–8 hr. after sudden prostration, dyspnoea, polydipsia. Mortality up to 75 per cent. Subacute: duration 2–6 days. Dejection, greenish diarrhoea, dyspnoea, emaciation, death, glassy appearance	Clinical signs, bacteriological examination, and blood testing using rapid slide or tube agglutination test	Young: lesions of septicaemia in acute form. Subacute: congestion of spleen, kidneys, and liver. Small greyish foci of necrosis, the size of a pin-head in the liver. Desquamation of intestinal mucosa. Haemorrhagic enteritis. Adults: arthritis, at first exudative. Synovial fluid clear and thin, later	No satisfactory treatment. Detection of carriers by agglutination test unreliable. Hygiene, disinfection, heavy culling, isolation, and treatment with furazolidone may be worth trying, especially when valuable birds involved. Suggested dose: 10 mg. furazolidone capsule twice daily for 3 days. Do	Egg-transmitted, spread by apparently healthy carriers. Fairly common, females chiefly affected

		of eyes. Joint swellings may develop. Adults: occasionally acute septicaemia, but usually chronic, characterized by arthritis of wing joints especially humero-ulna-radial joint, less frequently leg joints, swelling of wing joints, drooping or trembling of wing, lameness, loss of weight, occasionally twisting of head and neck, megrims		becoming thick greyish and purulent. Arthritis commonly seen in wing joints. Necrotic liver lesions are sometimes seen	not exceed this dose because drug toxic to pigeons. Tetracycline or chloramphenicol therapy worth trying	
Trichomoniasis or canker	*Trichomonas gallinae*. Poor hygiene and overcrowding contributory causes	Clinical signs vague and unreliable. Healthy adults may be carriers and infect squabs. Disease and death usually confined to squabs. Acute, wet canker: occurs usually at 6th to 15th day of age; death within 10 days. Inappetence and sneezing, diphtheritic membranes in mouth and throat. Chronic (dry): small caseous lesions in the throat and in the commissures of the beak	Lesions and demonstration of live organisms in fresh lesions	Lesions vary. Some birds which die show practically no lesions, in others lesions in mouth, oesophagus and crop may be extensive. Acute (wet canker): small yellowish adherent masses on oral or upper oesophageal mucous membranes. Lesions multiply, coalesce, and cause extensive areas of caseation, sometimes obstructing oesophagus. Caseation may be present on crop epithelium, in liver, and occasionally other organs. Chronic (dry): usually no obvious lesions in either upper digestive tract or liver. Organisms readily isolated, from sinuses, mouth, pharynx, oesophagus, and crop	Aqueous solution of 2-amino-5 nitrothiazole 1 ml. containing 10 mg. of drug pipetted into oesophagus every day for 7 days, or 30 mg. per kg. b.w. daily in 1 gal. drinking-water for 7 days, or 6·3 gm. Enheptin in 1 gal. (i.e., 1 level tablespoon of powder) for 7–14 days, or 1 dessertspoon of 16 per cent solution aminonitrothiazole in 2 gal. drinking-water for 4 days. Metronidazole may be used in treatment as well as prevention of trichomoniasis. Daily dose of 60 mg. per kg. given for 5 days to parent pigeons will protect progeny for 20 days. Metronidazole or dimetridazole at 50 mg. per kg. b.w. daily for 5 days given orally is effective in eliminating *T. gallinae* from pigeons (McLaughlin, 1966)	Doves and pigeons can be source of infection to other species. Sometimes erroneously called thrush. Lesions may disappear and birds recover. Losses may reach 75 per cent. Birds remain healthy carriers for years, and pass infection to others, especially squabs. Drinking-water may also be a source of infection
Virus infection	Herpes group virus	Serous conjunctivitis, rhinitis, and general malaise	The virus produces whitish cream pocks on chorio-allantoic membrane of 10-day embryonated eggs	Focal hepatic necrosis	Symptomatic	Maintained in nature by healthy adult carriers

Table 19.—COMMON DISEASES OF THE DOMESTIC PIGEON AND ITS RELATIVES (*continued*)

DISEASE	CAUSE	CLINICAL SIGNS	DIAGNOSIS	POST-MORTEM LESIONS	TREATMENT	REMARKS
Worms (capillariasis or hairworms)	*Capillaria columbae* and *C. obsignata*	Dejection, birds huddle on floor away from flock, anaemia. vent feathers soiled with greenish droppings, diarrhoea, emaciation leading to death in chronic cases. Usually adults only affected. May be acute. Sudden death without any signs of illness	Based on clinical signs and presence of eggs with bipolar plugs in droppings or parasites and eggs in intestinal tract at autopsy	Marked enteritis especially involving duodenum. Scraping of epithelium reveals hair-like worms approximately 1 cm. long fixed to intestinal mucosa	None satisfactory. Repeated treatment with piperazine citrate together with attention to hygiene. Single dose 200 mg. tablet of 1 diethyl-carbamyl-4-methyl piperazine, or a double dose orally in drinking-water. Dithiazanine iodide is effective against *C. obsignata*. Six doses totalling 350 mg. should be given to each bird. Thiabendazole may also be effective. Best drug for treament appears to be methyridine, given as 5 per cent aqueous solution s.c. at the rate of 100 mg. per kg. or approximately 35–45 mg. per bird (Wehr, Colglazier, Burtner, and Wiest, 1967)	Life cycle direct
Worms (round worm or ascariasis)	*Ascaridia columbae*	Heavy infestations only cause clinical signs. Loss of weight, alternate constipation and diarrhoea for several weeks, anaemia, droopiness	Based on clinical signs and presence of worms or eggs in droppings or intestinal tract at autopsy, associated with lesions	Flaccidity and dilatation of intestinal tract, congestion of mucous membranes, presence of large numbers of round-worms over 1 in. in length, occasionally gut ruptures and causes peritonitis or becomes impacted with worms	8 g. piperazine citrate per gal. drinking-water. Give medicated water for 60 hours. Deprive birds of water for 24 hr. before treatment. After a week further treatment can be given if necessary. Attention to hygiene and management. Thiabend-azole at dosage-rate of 0·5 per cent in mash for 1 week also eliminates mature and immature worms	Life cycle of worm is direct

SECTION E

MISCELLANEOUS

CHAPTER XX

SOME COMMON DRUGS USED IN AVIAN MEDICATION

BECAUSE of the low individual value of the various birds except the exotic species, individual treatment is not usually recommended. Instead, medication is done on a mass scale and drugs are administered on a flock basis, usually given in either water or feed.

The common drugs used for this purpose are the sulphonamides, antibiotics, nitrofurans, vermifuges, and growth promoters. Other drugs are used less frequently. These drugs are listed in *Table* 20.

Table 20.—COMMON DRUGS USED IN AVIAN MEDICATION

OFFICIAL NAME Vet C., U.S.P., or B.P.	TRADE NAME	DOSAGE for *a*, domestic fowl and duck *b*, turkey and goose *c*, budgerigar and parakeet, etc.	USES AND REMARKS
Acetarsol sodium	Turk-E-San, Boots	*b*, 1 tablet per 2½ gal. water	Continuously as an antihistomonad but discontinue 7 days before killing
Acinitrazole	Entramin, A, M & B (22½ per cent premix)	*b*, 0·025 per cent fed continuously in mash for prophylaxis. Double strength for therapy for 7–14 days followed by lower level	For the treatment and prophylaxis of enterohepatitis (blackhead) and hexamitiasis in turkeys
Aminonitrothiazole	Entramin, M & B (16 per cent solution)	*b*, ½ oz. per gal. for therapy for 14 days	As above. May affect fertility
Amprolium	Ampromix, Merck, Sharp & Dohme	*a*, 0·0125 per cent in food, may be reduced to 0·006 per cent	Continuously in food as coccidiostat
Arsanilic acid	—	*a*, *b*, 0·01 per cent in mash or 90 g. per ton	In feed continuously as growth promoter
Buquinolate	—	*a*, 0·003–0·008 per cent in food	As coccidiostat
Chloramphenicol	Chloromycetin, Parke, Davis & Co.	*a*, *b*, 25–50 mg. per lb. per day *c*, 1 mg. per oz. body-weight i.m. once daily or 2 mg. per oz. body-weight orally every 6 hours *a*, *b*, *c*, 250 mg. capsule in 1 gal. drinking-water for 3–5 days. This antibiotic could be toxic.	To treat infections caused by susceptible bacteria mainly *Escherichia coli*, *Salmonella*, or paracolons
Chlortetracycline hydrochloride (CTC)	Aureomycin, Cyanamid	*a*, *b*, 25–50 mg. per lb. per day or 100–200 g. per ton used in feed *c*, 1 mg. per oz. body-weight i.m. once daily, or 10 mg. per oz. body-weight s.c. of oily suspension every 5 days or 25–50 mg. in 1 oz. drinking-water	Special preparations may be used in drinking-water or feed, as advised by manufacturer, to treat acute infections due to susceptible organisms. Absorption is reduced in the presence of calcium but is increased in the presence of terepthalic acid. Used as premix as a growth promoter

Diethylstilboestrol	—	*a*, 30 mg.	Tablet used as implant in cockerel for fattening at 4–6 weeks
Erythromycin	Illotycin, Lilly	*c*, 0·1 mg. per oz. body-weight s.c. once daily	Erythromycin susceptible organisms. This antibiotic may also be used for dipping eggs for the control of mycoplasmosis. A 800 p.p.m. solution is used for the purpose
Furaldatone (a soluble nitrofuran)	—	*a*, *b*, 0·04 per cent in drinking-water for 1–2 weeks	For the treatment and prevention of coli septicaemia and infections with enterobacteriacae, other susceptible organisms and infectious synovitis. Actions and uses similar to furazolidone
Furazolidone	Neftin, Smith, Kline and French	*a*, 0·04 per cent for 10 days for therapy and 0·01–0·015 for prophylaxis *b*, 0·015 per cent continuously for prophylaxis 0·04 per cent for 2 weeks for therapy	For the treatment and prevention of *Salmonella* infections and control of mycoplasmosis (C.R.D.) Used as premix in feed for the therapy and prophylaxis of histomoniasis
Magnesium sulphate	—	*a*, 0·5 g., *b*, 1 g., *c*, 0·1 g.	As a laxative given in drinking-water or individually
Methyl benzoquate	Statyl, I.C.I.	*a*, 0·001 per cent in food or 10 g. per ton	As a coccidiostat for broilers. It does not permit immunity to develop
Methyridine	Promintic, I.C.I.	*a*, *b*, 0·05–0·1 ml. per lb. body-weight of a 90 per cent sol. s.c. Dilute 1:10 with water before use	For the treatment of capillariasis and *Heterakis* infections. Side effects are common when the higher dosage is used
Metichlorpindol	Coyden 25, Dow Chemical Co.	*a*, 0·0125–0·025 per cent in food or 1 lb. per ton	As a coccidiostat for broilers. It does not allow immunity to develop
Nicarbazin	Nicoxin, M & B; Nicrazine, Merck, Sharp & Dohme	*a*, 0·0125 per cent	Continuously in the feed for the prophylaxis of coccidiosis up to 16 weeks
Nihydrazone	Nidrafur, Norwich Phar. Co.; Vio Bin, Hess & Clark; Canada	*a*, 0·011 per cent in mash continuously	Useful in the prophylaxis of coccidiosis, C.R.D., air sac complex, and salmonelloses

Table 20.—COMMON DRUGS USED IN AVIAN MEDICATION (*continued*)

OFFICIAL NAME Vet C., U.S.P., or B.P.	TRADE NAME	DOSAGE for *a*, domestic fowl and duck *b*, turkey and goose *c*, budgerigar and parakeet, etc.	USES AND REMARKS
Nithiazide	Hepzide, Merck, Sharp & Dohme	*b*, 0·025 per cent in water; i.e., 1 oz. Hepzide per gal. of water	For the prevention of histomoniasis
Nitrofurazone	Nefco, Smith Kline & French; Chixun, I.C.I.; N.F.Z., Cooper McDougall & Robertson	*a*, 0·011 per cent continually for prophylaxis; 0·02 per cent for therapy for 7 days followed by 0·011 per cent	In feed for the therapy and prophylaxis of coccidiosis
Nitrophenide	Megasul, Cyanamid	*a*, 0·05 per cent	Continuously in feed for the prophylaxis of coccidiosis. Not effective when infection is heavy. Higher doses are toxic
Nystatin	Mycostatin, Squibb	*a*, *b*, 100–200 g. per ton for 7–10 days; i.e., Mycostatin 20, feed supplement for 7–10 days	For the treatment of moniliasis in the domestic fowl and turkey
Oxytetracycline hydrochloride (OTC)	Terramycin, Pfizer	*a*, 25–50 mg. per lb. per day	In two or three divided doses orally for 3–7 days
		c, 1 mg. per oz. body-weight i.m. once daily	Special preparations may be used in drinking-water or feed as advised by manufacturer for the control of infections by susceptible organisms as under chlortetracycline hydrochloride
		a, *b*, 100–200 g. per ton of ration	This antibiotic is not as readily absorbed from the gut as is tetracycline hydrochloride. An oily suspension may be used subcutaneously for treatment of C.R.D. and infections by susceptible organisms. Used as premix in feed as growth promoter

Penicillin G	Various preparations	*a*, *b*, 5000–100,000 units per lb. per day	Intramuscular in aqueous suspension daily for 2–3 days in infections caused by susceptible organisms; specific for spirochaetosis
		c, Procaine penicillin not used. Sodium penicillin given at dosage rate of 10,000 units per 2 oz. body-weight every 6 hours	As premix in feed as growth promoter
		a, *b*, 4–10 g. per ton in feed or more	
Phenothiazine	Phenovis, I.C.I.	*a*, 0·5 g. per adult. *b*, 1·0 g.per adult	Individually or in mash for the treatment of *Heterakis gallinarum* infections. For mass treatment follow manufacturers' instructions
	Phenosan, Boots	*a*, *b*, 1 lb. for 900 fowls or 450 turkeys	
Piperazine adipate, hydrate, or citrate	Coopane, Cooper McDougall & Robertson	*a*, *b*, 8 g. per gal. for 3 days of Coopane	In drinking-water for the treatment of *Ascaridia galli* and other *Ascaridia* infections
	Hetrazan, B. & W.; Safersan, Boots; Verban, Cyanamid; Dowzene, Dow	*a*, *b*, *c*, 8 g. per gal. per 2–3 days. Repeat after 3 weeks if necessary	Always follow manufacturer's instructions while using proprietary preparations
Saline 5 per cent Dextrose	—	*c*, 0·25–0·3 ml. s.c. in wing web, repeat 2–3 times daily as required	In dehydration, diarrhoea, and other conditions of fluid loss
Sodium pentobarbitone	Nembutal, Abbot	*a*, 0·5–0·75 ml. per bird *b*, 1·1 ml. per 5 lb. *c*, 0·00083 ml. per kg. body-weight or 30–40 mg. of drug per kg. body-weight s.c. or i.p.	*a*, *b*, Given by intravenous injection. Anaesthesia may last up to 2 hr. *c*, Intramuscular, for induction of anaesthesia which is smooth, lasts about 30 minutes. Recovery takes place in about 90 minutes. If microlitre syringe is not available the standard solution may be diluted 10 fold or more

Table 20.—COMMON DRUGS USED IN AVIAN MEDICATION (*continued*)

OFFICIAL NAME Vet C., U.S.P., or B.P.	TRADE NAME	DOSAGE for *a*, domestic fowl and duck *b*, turkey and goose *c*, budgerigar and parakeet, etc.	USES AND REMARKS
Stilboestrol	—	*c*, 0·0001 ml. per oz. i.m. of a solution containing 25 mg. per ml. daily	More than 10 times the recommended dose may be used without ill effects. If microlitre syringe is not available the standard solution may be diluted 100 fold
Streptomycin or dihydrostreptomycin sulphate	—	*a*, 0·1 g. per adult *b*, 0·5 g. per adult *c*, toxic	This antibiotic is fairly toxic for poultry; the standard dose is 25–50 mg. per kg. as aqueous solution by intramuscular injection twice daily for the treatment of infections caused by susceptible organisms such as those due to *Haemophilus gallinarum*, *Mycoplasma gallisepticum* or *Escherichia coli*
Sulphadimidine sodium B.P. or Sulphamethazine sodium U.S.P.	Sulphamezathine, I.C.I.	*a*, 0·44 per cent in mash or 0·2 per cent in drinking-water for therapy for 3–5 days. *c*, 2 oz. of 16 per cent solution to 1 gal. water for 5 days	In mash or drinking-water for the therapy of coccidiosis. Or as an alternative follow manufacturer's instructions as to interrupted schedules
Sulphathiazole	Avizol, M & B	*a*, *b*, *c*, 1:100 in drinking-water, i.e., 2 oz. per gal. for 2–3 days	For the treatment of infection with *H. gallinarum* and susceptible organisms
Sulphaquinoxaline sodium	Embazin, M & B	*a*, 0·0125 per cent in mash continuously for prophylaxis, or 0·04 per cent in drinking-water for 3 or more days for therapy *b*, 0·05 per cent in water for prophylaxis continuously or 0·06 per cent in drinking-water for therapy	For the therapy and prophylaxis of coccidiosis, for the treatment of fowl cholera and fowl typhoid

Sulphaquinoxaline	Sulquin, Salisbury's	*a*, Follow manufacturer's instructions	For the therapy and prophylaxis of coccidiosis, for the treatment of fowl cholera and fowl typhoid
Testosterone	—	*c*, As for Stilboestrol	
Tetracycline	Achromycin, Cyanamid	*c*, 1 mg. per oz. body-weight i.m. once daily	For the treatment of infections with susceptible organisms
Tetramysole	Nilverm, I.C.I.; Appercol, Jansen Pharmaceutica, Beerse	*a*, As acetate in drinking-water at a conc. of 0·04 per cent for one day	99–100 per cent effective against *Ascaridia galli*, *H. gallinarum*, and *Capillaria obsignata*
Trichomycin	Trichonat	40 I.U. per ml. drinking-water	Probably would be useful in early fungal infections only. Its usefulness not yet fully evaluated
Zoalene	Zoamix, Dow	*a*, *b*, 0·0125 per cent in food	Continuously for the prevention of coccidiosis

N.B. Further information on the actions and uses of these drugs in the domestic fowl, duck, and turkeys is given by Jones, M. L. (1966); in budgerigars, in *J. small Anim. Pract.*, **4**, 27; in cage birds in the B. V. A. handbook (1964) on the treatment of exotic pets; and the choice of the drugs for variety of conditions in the domestic fowl and in turkey by Blaxland (1961a).

CHAPTER XXI

DEFECTS OF INCUBATION

IT is not uncommon to encounter diseases of chicks which are directly associated with defects of incubation or diseases which may be transmitted via the egg and are manifested during incubation by increased infertility, embryonic mortality, or neonatal deaths. Good examples of these conditions are pullorum disease and mushy chick disease.

Storage of eggs at a high environmental temperature in the tropics is an important cause of embryonic mortality and malposition. Hatching eggs should be stored at 55–70° F. (12·75–21° C.) at a relative humidity of 70 per cent or more. The period of storage under these conditions should not be more than 10 days.

The wastage due to embryo mortality in England and Wales in 1963 has been estimated to be 23 per cent (Beer, 1967). Incubation environment probably constitutes the major hindrance to securing 100 per cent hatches; apart from this, diseases due to viral and bacterial agents, feeding faults, genetic factors, and defective transport and storage of eggs contribute to embryo mortality. Contamination of the eggs with *Aspergillus fumigatus* may cause peak mortality on the sixteenth day of incubation (Beer, 1967).

Defects in nutrition, such as deficiencies of certain vitamins such as A, B_1, B_2, B_6, B_{12}, D, and E may cause high embryonic mortality and infertility. The various diseased conditions associated with incubation are given in *Table* 21.

Deficiencies of niacin, folic acid, biotin, pantothenic acid, calcium, phosphorus, manganese, and zinc may also increase embryonic mortality.

To obtain optimum results eggs for incubation should be stored at 55° F. (12·75° C.) at a relative humidity of 75–80 per cent.

Table 21.—DEFECTS OF INCUBATION*

DEFECT	PROBABLE CAUSES	NECESSARY ACTION
1. Many clear or infertile eggs or lowered hatchability	*a*, Wrong proportion of males to females	*a*, Use 1 male to 15–20 hens of light breeds, and 1 male to 10–15 hens of heavy breeds
	b, Defective males	*b*, See that the cockerels are well nourished, fertile, and of good stock
	c, Ration of adults deficient in vitamins A, D, E, or B_{12}	*c*, Correct diet of adults
	d, Salmonelloses, especially *S. pullorum* infection	*d*, Disinfect premises, add furazolidone to mash as recommended by manufacturer or give furadantin in drinking-water
	e, Eggs kept too long or under the wrong conditions before setting	*e*, Do not keep hatching eggs longer than 10 days; store them in the cool at 55–70° F. (12·75–21° C.) and a relative humidity around 70 per cent or more
2. Blood rings, indicating very early embryonic death	*a*, Incubator temperature too high or too low	*a*, Check thermometers, thermostats, and current supply. Follow the maker's instructions
	b, Incorrect fumigation procedure	*b*, See that the correct amount of fumigant is used. Do not fumigate between the 24th and 84th hour after setting
	c, As in 1 *c*	*c*, As in 1 *c*
3. Many dead in shell	*a*, As in 2 *a*	*a*, As in 2 *a*
	b, Eggs not properly turned	*b*, Turn the eggs regularly twice a day, always turn the eggs in the reverse direction each time, never in the same direction
	c, Faulty nutrition of breeding stock, if deaths are heavy in the 10th to 14th day period	*c*, Pay particular attention to the supplying farms from which the eggs come; check that the feeding is sound

* Modified from *Incubation and Hatchery Practice* by W. M. Allcroft (1961), Bulletin No. 148 of Ministry of Agriculture and Fisheries. London: H.M. Stationery Office.

Table 21.—DEFECTS OF INCUBATION (*continued*)

DEFECT	PROBABLE CAUSES	NECESSARY ACTION
3. Many dead in shell (*continued*)	*d*, Faulty ventilation of the incubator	*d*, Increase the ventilation by normal means; additional oxygen is not needed at altitudes less than 5000 ft. above sea level
	e, Pullorum disease, other forms of salmonelloses, avian encephalomyelitis, yolk-sac infection, or *M. gallisepticum* infection	*e*, Use only eggs from sound, disease-free stock. Check that the hatchery hygiene is sound and is carried out; practise fumigation
	f, Avitaminosis B_2 causes high mortality from 14th to 21st day	*f*, Correct diet
4. Pipped eggs failing to hatch	*a*, Insufficient moisture in the incubator	*a*, Increase the evaporating surface of water or increase the sprays. Check the wet-bulb readings
	b, *Salmonella* and other bacterial infection of eggs	*b*, *c*, As in 9 *c*
	c, Avian encephalomyelitis infection	
5. Hatching too soon, too late, or sticky chicks	Incorrect temperature	Make quite sure that the temperature-regulating gear is efficient, and in particular that it is at the correct temperature when the current cuts off. If the temperature rises after this during a hatch, reduce temperature at which current cuts off
6. Malformed chicks	*a*, Incubator temperature too high *b*, Incubator temperature too low *c*, Eggs set incorrectly or not properly turned after setting	*a*, As in 2 *a* *b*, As in 2 *a* *c*, As in 3 *b*; also take care to set the eggs broad-end up in cabinet-type incubators
7. Spraddling chicks	Hatching trays too smooth	Use wire-mesh tray floors with non-slippery material

8. Weak chicks, small chicks	*a*, Overheating of the incubator or hatching unit	*a*, As in 5
	b, Setting small eggs	*b*, Only set eggs of the recommended sizes
	c, Too little moisture in incubator	*c*, As in 4 *a*
9. Heavy-breathing chicks	*a*, Too much fumigant left in hatcher	*d*, As in 2 *b*
	b, Too much moisture in hatcher	*b*, Decrease evaporating surface or decrease sprays
	c, Possibly infectious disease especially brooder pneumonia	*c*, Send chicks to a veterinary laboratory for diagnosis and carefully clean out and fumigate the incubator, using formaldehyde at higher strength. Pay particular attention to the disinfection of all equipment
10. Mushy chicks	Low average temperature during period of incubation	As in 2 *a*
11. Hatch not coming off evenly	Setting eggs too diverse in age	As in 1 *e*
12. Parrot beaks	Manganese deficiency	Remedy deficiency in breeding stock
13. Stunted chick disease	Pantothenic acid deficiency	Remedy deficiency by adding 4 per cent brewer's yeast to layers' ration
14. Clubbed down with dwarfing, curled toes, and parrot beak	Riboflavin deficiency	As in 13

CHAPTER XXII

POISONS AND TOXINS

Two types of specific intoxication, namely those due to the toxins of *Clostridium botulinum* (botulism) and the toxins of certain fungi (mycotoxicosis) have been dealt with before.

Several drugs and chemicals can be toxic if used carelessly. Similarly, insecticides if carelessly used may cause alarming symptoms. Treatment in acute cases is often not worth while because of the low individual value of the birds. Even if they recover, the damage to internal organs in acute cases would be so severe as to retard growth and productivity after recovery.

Therefore, once signs of toxicity are detected it is best to identify the toxin or the poison and remove it rather than attempt to treat the sick birds. In most cases an accurate history of the outbreak of the disease will be useful. *Table* 22 gives the common poisons or toxins to which poultry may have access, the minimum lethal doses, characteristic symptoms, and lesions if any, together with the method of treatment or the antidote.

Besides the chemical toxins listed in the table, several species of plants contain certain alkaloids or glucosides which cause toxicity if ingested in large amounts. The most common of these are found in:—

1. The potato plant (*Solanum tuberosum*). The green tubers produced by exposure to light and young potato shoots contain the alkaloid solanin which is highly toxic. Therefore, such potatoes or young shoots should not be used either for human consumption or animal feeds.

2. Cottonseed meal. This contains gossypol which is toxic to animals and birds. Therefore, excessive quantities of this should not be used for feeding birds. It has been found that feeding one ounce of cottonseed meal daily produces toxic effects. Gossypol toxicity can be reduced by addition of ferrous iron, up to 500–1600 parts per million of the ration.

3. *Crotalaria spectabilis* (the crotalaria plant) which contains the alkaloid monocrotalin in its seed. Other species of *Crotalaria* may also contain this toxin. The toxin causes cirrhosis of the liver and muscular haemorrhages, ascites, oedema, and hydrothorax in turkeys.

4. *Nerium oleander* (the oleander plant), all parts of which are toxic.

5. *Lathyrus* spp., the seeds of which are toxic.

6. *Nicotiana tobacum* (the tobacco plant), which contains the alkaloi dnicotine in its leaves.

7. *Solanum nigrum* (the black nightshade); the immature fruit contains solanin and solanidin which are toxic to man and animals.

8. *Asclepias* spp. (the milkweed plants), which contain the bitter glucoside asclepidin which is toxic to animals and birds.

These and many other species of plants contain toxins which may be poisonous to birds. However, birds are usually selective in their feeding habits and will not consume the poisonous plants unless they are compelled to do so due to starvation. Therefore, the losses of poultry resulting from poisonous plants are extremely small. Positive diagnosis of poisoning must rest on an accurate history and the identification of the poison in the bird's tissues or its feed. The clinical symptoms and lesions are rarely of diagnostic value. Further information on toxicology is given by Garner (1961) and Radeleff (1964).

Table 22.—Some Toxic Agents

Poison or Toxin	Minimum Lethal Dose for Fowls or Toxicity	Common Source	Characteristic Symptoms or Lesions	Specific Antidotes
Arsenic compounds	0·05–0·3 g. per single dose of arsenic trioxide	Rodenticide and insecticide	Gastro-enteritis and jerking of wing, twisting of head to one side	British anti-Lewisite (B.A.L., dimercapol), sodium thiosulphate
Copper sulphate	0·9 g. per kg. body-weight	Medicinal agent and orchard spray	Gastro-enteritis	B.A.L. (dimercapol), ammonium molybdate, and sodium thiosulphate
Lead compounds	0·16–0·6 g. per kg. of absorbed lead	Paints, orchard and garden sprays, and lead shot	Stippling of erythrocytes in characteristic cases	Ca EDTA (Calcium disodium versenate)
Nicotine sulphate	0·5–1 ml. of Black Leaf 40	Black Leaf 40	Dilatation of pupil, convulsions, dark red blood, and ecchymoses in lungs and heart	Nil. Bicyclic amines and stimulants may be useful
Phosphorus	0·02 g.	Rodenticide and in crackers and fireworks	Phosphorescence of intestinal contents and smell of garlic in abdominal organs	Nil
Sodium chloride	10 g. or over 6·0 per cent in mash is definitely toxic	Fish meal, ice cream, salt, rock salt	Enteritis, oedema, and degenerative changes in heart, liver, and kidneys	Nil
Strychnine	2·0 mg. per kg.	Rodenticide and certain plants. As medicinal agent in drinking-water	Muscular convulsions	Nil. Barbiturates and potassium permanganate may be useful, the first to sedate and the second to oxidize the unabsorbed alkaloid
Potassium permanganate	2 g.	In drinking-water	Mucous membrane of the crop and gizzard are blackened	Nil

Table 22.—SOME TOXIC AGENTS (*continued*)

POISON OR TOXIN	MINIMUM LETHAL DOSE FOR FOWLS OR TOXICITY	COMMON SOURCE	CHARACTERISTIC SYMPTOMS OR LESIONS	SPECIFIC ANTIDOTES
Chlorinated hydrocarbons; D.D.T., B.H.C., etc.	Birds are comparatively resistant to D.D.T. and B.H.C.	Insecticide	Muscular incoordination	Nil. Barbiturates and sedatives may be employed. Calcium gluconate is said to be useful
Organic phosphorus compounds; malathion diazinon, dimethoate, etc.	Higher than 14 mg. per kg. for diazinon and 200–400 mg. per kg. for malathion	Insecticide	Muscular tremors and acute congestion of lungs	Atropine 0·1–0·2 mg. per kg. 2-PAM, P2S and TMB-4
Sodium fluoracetate	14 mg. per kg.	Rodenticide	None	Magnesium sulphate may be given in dilute solution
Sulphonamide (sulphanilamide, sulphaquinoxaline, etc.)	Toxicity depends on concentration in mash and period of medication. Not safe to exceed recommended dose	Coccidiostat	Haemorrhages in the internal organs	Nil
Arasan	Levels about 7·5 p.p.m. are toxic	Fungicide used to treat grain	Misshapen and soft shelled eggs	Nil
Cyanides	2 mg. per kg. body-weight of HCN	Bird bait used to kill starlings and sparrows	Blood oily, odour of bitter almonds	Nil
Carbon monoxide	0·4–0·5 per cent in air	Defective heating units and bad ventilation	Bright cherry-red colour of lungs and blood	Nil

CHAPTER XXIII

ELEMENTARY POULTRY SURGERY

AVIAN surgery is often not economically worth while, due to the low value of the individual bird. However, one may be called upon to resort to surgical procedures in the case of valuable show birds or exotic pets.

Generally speaking, avian surgery is comparatively simple due to two basic differences in the birds from the mammal, namely the high threshold to sensitivity, and the comparative resistance of the avian species to the common pyogenic organisms thus making asepsis not essential.

If anaesthetics are required, a general anaesthetic is indicated and for this purpose pentobarbital sodium (Nembutal) 0·5–0·75 ml. intravenously will produce anaesthesia of up to two hours duration in the case of the domestic fowl.

Restraint and anaesthesia in cage birds is discussed in detail by Graham-Jones (1965). He considers that halothane is the best volatile anaesthetic and that this could be best administered by using a spiral gas bag designed by him (Graham-Jones, 1962). A dose of $\frac{1}{2}$–$1\frac{1}{2}$ per cent halothane produces sufficient concentration of the anaesthetic to induce anaesthesia. The gas-flow for maintenance should be at a minimum of 200 ml. per min. A mixture of chloral hydrate and magnesium sulphate can be used as a narcotic and light anaesthetic. Ethyl chloride can be used as a local anaesthetic for pinioning operations. Barbiturates may be used for induction or maintenance of anaesthesia but the dosage of the drugs has to be computed carefully. The dosage rate is 30–40 mg. per kg. given intramuscularly, intravenously, or intraperitoneally. The last route is the most convenient. Further information on anaesthesia and tumours of cage birds is given in Chapter XIX.

Nembutal diluted with 5 parts of physiological saline is recommended for the narcotization of small cage birds. The dosage recommended is 0·005 ml. per g. body-weight of the diluted solution given intraperitoneally or intramuscularly. The diluted solution should be freshly prepared.

A mixture of halothane and oxygen using a semi-closed circuit (McGill's system) was found to be an efficient anaesthetic in turkeys (Jones, R. S., 1966). This may prove equally effective in other birds. Another method for inducing anaesthesia of the fowl has been described by Anderson (1967) where anaesthesia is obtained by passing oxygen over halothane at a flow-rate of 0·5 l. per min. Surgical anaesthesia can be induced in 3 minutes and maintained for 2 hours.

A convenient method of anaesthetizing the adult domestic fowl with diethyl ether has been described by Donovan and Boone (1965).

Caponizing used to be done by surgical techniques in the past. However, due to the use of stilboestrol for this purpose, surgical caponization is now rarely done.

The common surgical procedures are the following:—

1. Opening of crop to remove an impacted mass.
2. Amputation of the comb (dubbing) and wattles (cropping).
3. Trimming of claws and spurs to prevent spur development.

4. Flight control.
5. Debeaking.
6. Draining of sinuses.
7. Removal of tumours.
8. Treatment of fractures of the legs.
9. Abdominal surgery involving laparotomy (rare).

Opening of Crop to remove Impacted Mass.—The feathers on the mid-ventral line over the crop are removed, the area painted with a disinfectant and a sufficiently large incision made through the skin and crop; the contents or foreign body, if any, are removed, the area cleaned and sponged dry, and the wall of the crop and skin are closed separately by independent line of sutures. The crop is closed with cat-gut or nylon. Silk thread may be used to close the skin incision. The line of sutures on the crop should be close to one another to prevent seepage of crop fluid. Fluid and food should be withheld for 12–24 hours.

Amputation of Comb (Dubbing) and of Wattles (Cropping).—This may be done in the day-old chicken by electrocautery without any haemorrhage by using the appropriate machine or by just trimming off the trace of the comb and wattles seen at this age with a pair of scissors. In the adult, however, unless proper surgical technique is followed, amputation of one or more of these appendages will cause fatal haemorrhage. A special clamp or a pair of intestinal clamps are applied tightly over the structure to be amputated so as to arrest circulation. If necessary, a local anaesthetic such as 2 per cent Planocaine (procaine hydrochloride) with 1:10,000 adrenaline may be infiltrated over the area to be sectioned. However, to get effective local anaesthesia the drug has to be injected at several sites. The comb is amputated with a pair of scissors $\frac{1}{8}$ in. distal to the clamps. Then the cut surface is seared with a hot iron to arrest haemorrhage and the clamp removed. It is desirable to observe the bird for 15–30 minutes. If haemorrhage is seen the clamp has to be reapplied and the cut surface of the comb seared as before. Haemorrhage can recur if the bird starts to shake the head due to the irritation caused by the trauma.

Trimming Claws and Spurs to prevent Spur Development.—Trimming of the claws and spurs can be done effectively without an anaesthetic by using a pair of shears or nail clippers used on dogs. In male turkeys it is desirable to clip the nails up to the corium to prevent damage to the back of the turkey hen during mating. If haemorrhage occurs this can be arrested by applying a local haemostatic such as tannic acid.

The spur buds of developing male birds may be clipped off at 10–16 weeks and the remaining tissue cauterized with a stick of potassium hydroxide.

Flight Control.—This can be done permanently by removing the last segment of the wing at the joint with a pair of shears or scalpel; a tourniquet has to be applied before the operation is done and after the operation the wound has to be cauterized to prevent haemorrhage and infection. An alternative method is tenotomy: a small section, about $\frac{1}{4}$ in., of the tendon, which extends on the underside of the wing parallel to the blood-vessels, is removed at any convenient site. This is suitable for all birds.

Functional ankylosis of the arista may also be used in flight control. Details of the various methods used have been described by Sedgwick (1967) and Startup (1967).

Debeaking.—This operation is performed to prevent cannibalism and is done usually with an electrical debeaker which removes one-third of the upper and lower beaks when the chicks are a day or a few days old. Debeaking, if performed on mature birds or growers, without proper precautions, will cause fatal haemorrhage.

Draining of the Sinuses.—This may be done by using an 18 gauge needle. The needle is inserted into the softest area of the swelling, the contents aspirated, and 1 ml. of a 4 per cent silver nitrate solution introduced.

If the contents of the sinuses are cheesy, the distended sinuses will have to be incised with a scalpel. An incision about $\frac{1}{4}$ in. long is usually made along the anterior part of the inferior border of the distended sinus. As this area is highly vascular, bleeding is likely to occur. This is arrested by irrigating with 4 per cent silver nitrate and, if necessary, by plugging the sinuses with cotton wool soaked in silver nitrate. Excess of this solution can be harmful.

Tumours and Fractures.—These are attended to surgically as in the case of domestic mammals. However, in most cases surgical interference is not worth while.

Abdominal Surgery involving Laparotomy.—Laparotomy may have to be done to remove eggs in the abdomen or tumours.

General anaesthesia may be used for this operation. The feathers along the mid-ventral lines are plucked and the area disinfected. An incision about 3 in. long is made between the tip of the sternum and the cloaca, the eggs removed and the wound closed with continuous silk or nylon thread.

In cases of ascites, the fluid may be withdrawn by using an 18 gauge needle.

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British Poultry Science, published quarterly by Oliver and Boyd Ltd., Tweeddale Court, 14, High St., Edinburgh 1, Scotland.

Poultry Science, published quarterly by the Poultry Science Association, A. & M. University College Station, Texas, U.S.A.

The Veterinary Record, published weekly by the British Veterinary Association, 7, Mansfield St., London, W.1.

The Veterinary Bulletin, published monthly by the Commonwealth Agricultural Bureaux, Surrey, England.

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INDEX