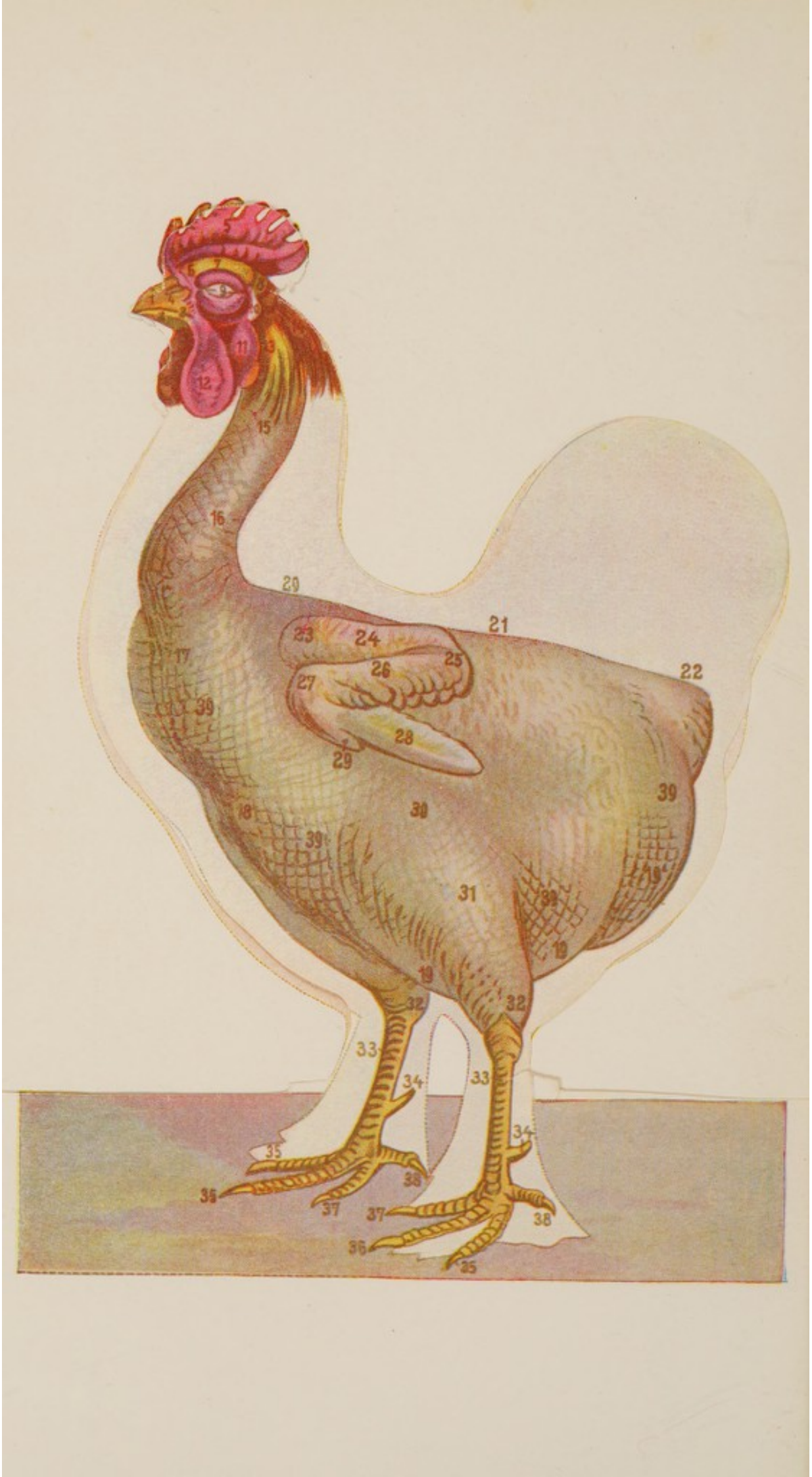
Diseases of poultry in New South Wales / T.G. Hungerford.

Diseases of Poultry in New South Wales

T. G. HUNGERFORD.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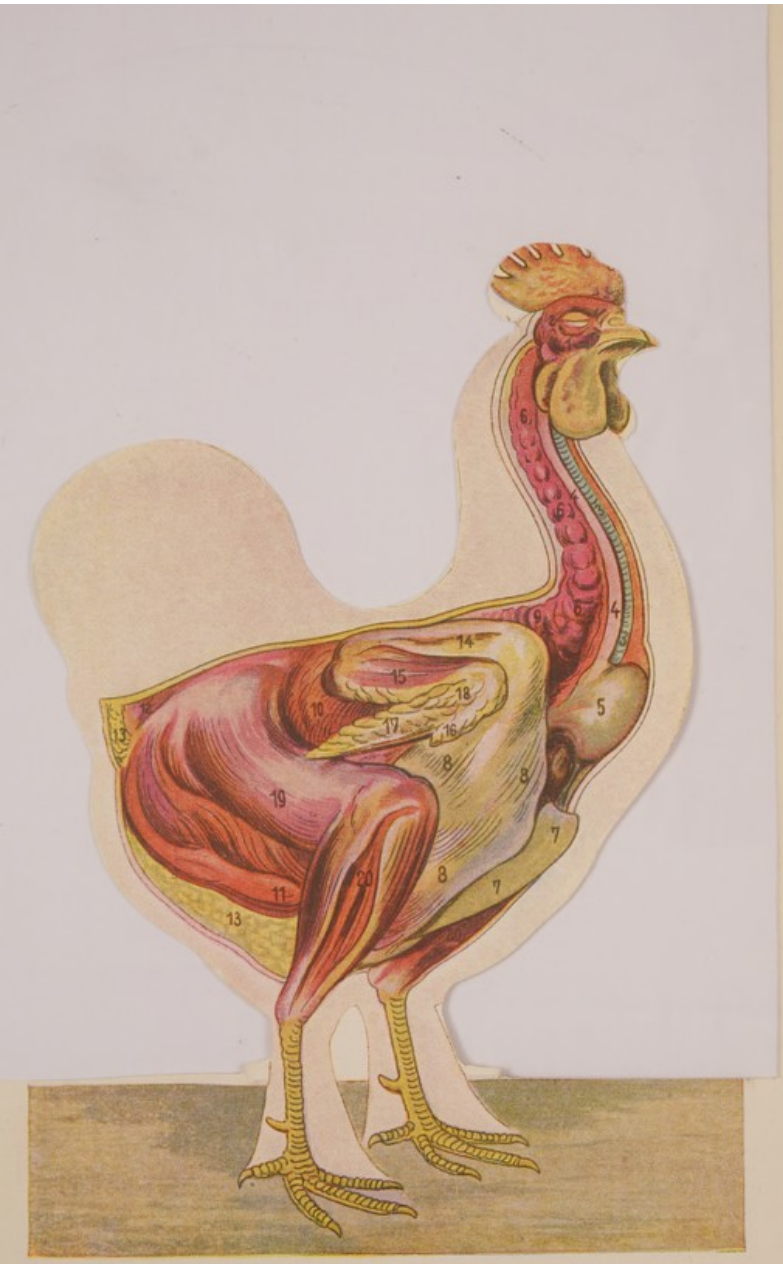
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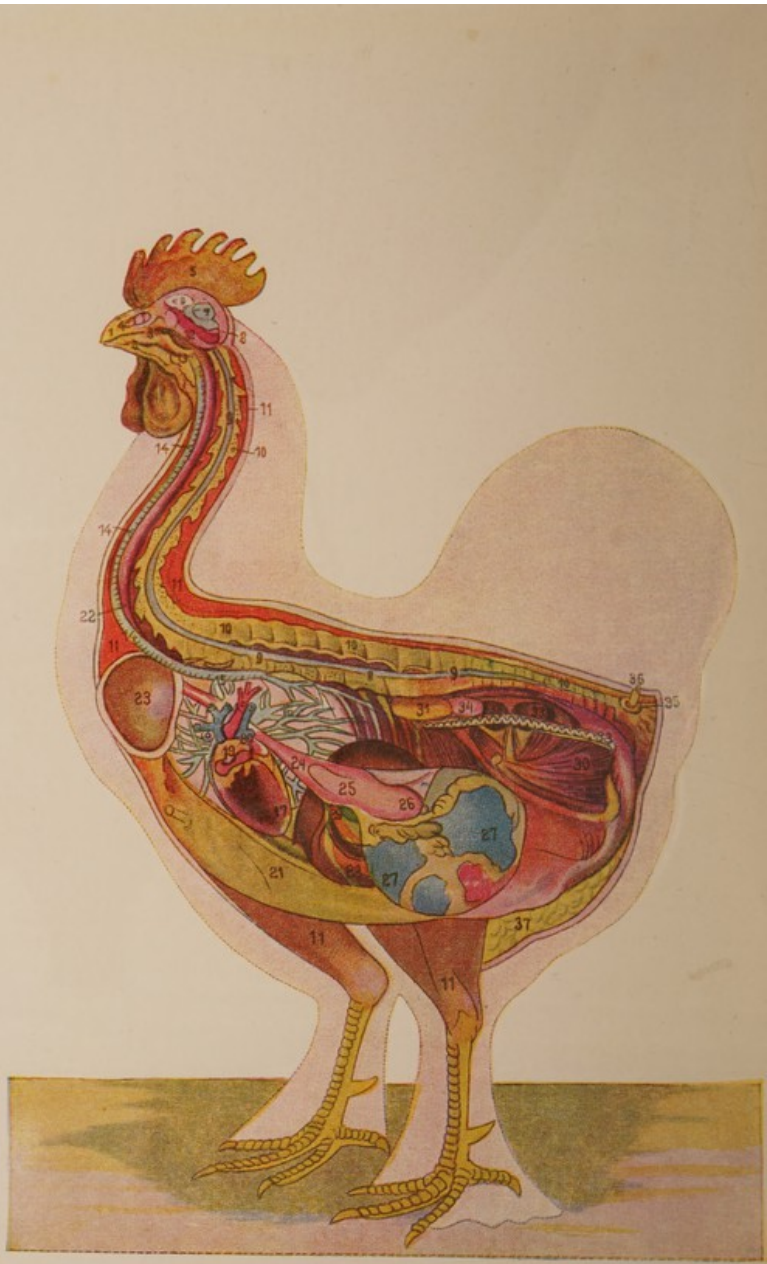
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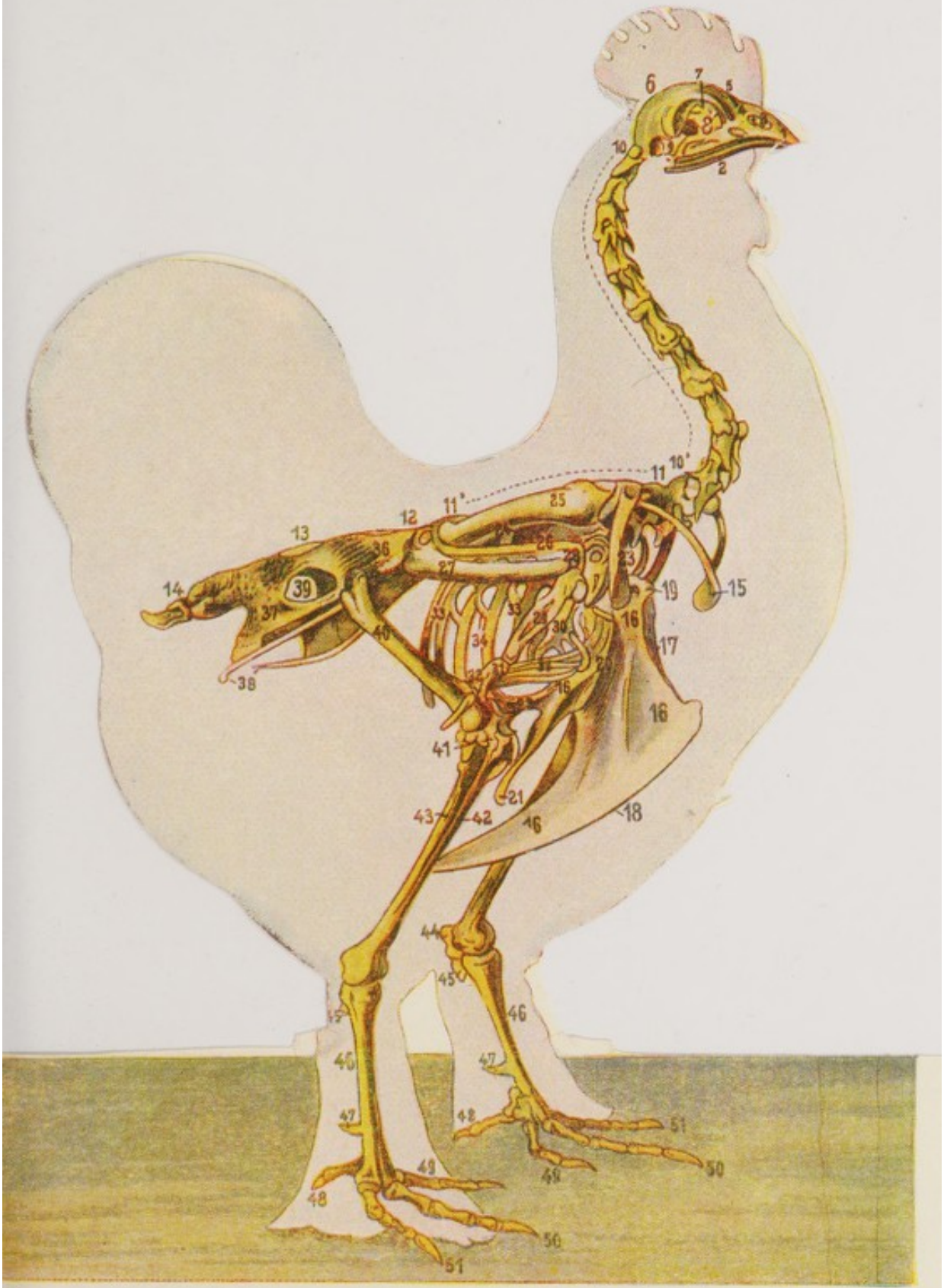
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EXPLANATION OF THE COLOURED PLATE.

The Exterior.

1 Upper bill.
2 Lower bill.
3 Cleft of bill.
4 Nostrils.
5 Comb.
6 Forehead.
7 Crown of head.
8 Occiput.
9 Eye.
10 Ear-lobes.
11, 12 Wattles.
13 Throat.
14 Nape of neck.
15 Upper neck.
16 Lower neck.
17 Neck covers.
18 Fore breast.
19 Lower breast.
20 Belly.
21 Rump and anus.
22 Upper back.
23 Lower back.
24 Saddle.
25 Angle of wing.
26 Wing coverts.
27 Shoulder coverts.
28 Primaries of wing.
29 Small wing coverts.
30 Saddle feathers.
31, 32 Tail feathers.
33 Knee.
34 Lower part of thigh.
35 Hock.
36 Tarsus.
37 Spur.
38–41 Toes.
42 Boots.

Skeleton.

1 Intermaxillary.
2 Lower Mandible.
3 External nares.
4 Nasal.
5 Frontal.
6 Cranium.
7 Eye socket.
8 Interorbital foramen.
9 Quadrate.
10 Cervical vertebræ.
11 Dorsal ,,
12 Lumbar ,,
13 Sacral ,,
14 Caudal ,,
15 Clavicle.
16 Sternum.
17 Body of same.
18 Spine of same.
19 Anterior process of same.
20 Costal process of same.
21 Styloid process of same.
22 External process of same.
23 Coracoid.

Skeleton (*continued*).

24 Scapula.
25 Humerus.
26 Radius.
27 Ulna.
28 Carpals.
29 Metacarpals.
30 Thumb.
31 Middle finger.
32 Third finger.
33 Ribs.
34 Uncinate processes of same.
35 Sternal ribs.
36 Ilium.
37 Ischium.
38 Pubis.
39 Acetabulum.
40 Femur.
41 Patella.
42 Tibia.
43 Fibula.
44 Calcaneum.
45 Tarsals.
46 Metatarsal.
47 Spur core.
48–51 Toes.

Exterior without Feathers.

1 Upper bill.
2 Lower bill.
3 Cleft of bill.
4 External nares.
5 Comb.
6 Forehead.
7 Crown of head.
8 Occiput.
9 Eyelids.
10 Ear-lobes.
11, 12 Wattles.
13 Throat.
14 Nape of neck.
15 Upper neck.
16 Lower neck.
17 Fore breast.
18 Lower breast.
19 Belly.
20 Upper back.
21 Lower back.
22 Rump.
23 Shoulder-joint.
24 Arm.
25 Elbow.
26 Forearm.
27 Carpus.
28 Middle finger.
29 Thumb.
30 Knee.
31 Lower thigh.
32 Hock.
33 Tarsus.
34 Spur.
35–38 Toes.
39 Skin, with feather areas.

Muscles.

1 Masseter.
2 Temporal.
3 Trachea.
4 Oesophagus.
5 Crop.
6 Neck muscles.
7 Keel of sternum.
8 Pectorals.
9 Latissimus dorsi.
10 Intercostals.
11 Abdominal muscles.
12 Tail muscles.
13 Fat tissue.
14 Wing muscles.
15 Lower wing muscles.
16 Thumb.
17 Middle finger.
18 Third finger.
19 Thigh muscles.
20 Leg muscles.

Internal Organs.

1 Upper bill.
2 Lower bill.
3 Cleft of bill.
4 External nares.
5 Comb.
6 Cerebrum.
7 Cerebellum.
8 Medulla oblongata.
9 Spinal cord.
10 Cross-section spinal cord.
11 Cross-section muscleature.
12 Pharyngeal cavity.
13 Larynx.
14 Trachea.
15 Bronchi.
16 Lung.
17 Heart.
18 Aorta.
19 Pulmonary artery.
20 Posterior vena cava.
21 Sternum.
22 Oesophagus.
23 Crop.
24 Thoracic part of oesophagus.
25 Entrance into stomach.
26 Fore stomach.
27 Gizzard.
28 Pancreas.
29 Jejunum.
30 Cloaca with anus.
31 Kidney.
32 Vas deferens.
33 Testicle.
34 Suprenal bodies.
35, 36 Oil-gland.
37 Fat tissue.

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NEW SOUTH WALES
DEPARTMENT OF AGRICULTURE.

Diseases of Poultry
in
New South Wales

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Compilation Completed
March, 1939.

PRICE
10s. 6d

Author's Preface

THIS book is not written as a technical treatise on poultry diseases, but comprises a statement of the diseases of poultry which cause most economic loss in this State, together with a popular account of symptoms, control measures, as well as an indication of what is still unknown concerning some of the disease conditions. Neither is the book written as a veterinary text-book, but is designed for those connected with the industry, who, by virtue of interest or necessity, desire to make a careful study of poultry diseases. Those who have only a passing interest in disease may find that the amount of detail given is somewhat irksome. This has been done intentionally, as a passing interest which leads one to make an ill-founded diagnosis and to carry out incorrect treatment will usually demonstrate that "a little knowledge is a dangerous thing."

Whilst this book does not go into scientific detail, it is pointed out that the diagnosis of poultry disease has, by modern scientific methods, been brought to a degree of accuracy comparable with that of other branches of veterinary science and of human medicine, and, with the advent of qualified veterinarians into poultry practice, the poultry farmer now has available the advice of specialists to assist him in dealing with his disease problems.

Above all, it is desired to point out that the information in this book must be considered conjointly with information concerning housing, management and feeding, as the subjects of disease and management are inseparably interrelated. For such information, the reader is referred to "Poultry Farming in New South Wales," issued by the Department of Agriculture.

Scientific information concerning diseases of poultry is making such vast advances that, without doubt, by the time this book is published it will already be partly out of date. The most recent information on new advances might be sought from a practising veterinary surgeon or from the Chief Veterinary Surgeon of the Department of Agriculture.

Diseases are grouped not as to cause, which would perhaps be the most logical arrangement, but according to the symptoms present. Thus all conditions which produce "roup" are grouped. This method has been adopted, because a farmer whose birds are showing such well-known symptoms will want to know of all the diseases which may cause such symptoms. The convenience of this grouping, it is felt, over-rules the annoyance which must be experienced by a veterinarian who finds, for instance, in the "roup" diseases, a disease such as avitaminosis A (a food deficiency disease) grouped with laryngo-tracheitis (a virus disease).

Home remedies have been listed for most diseases, not because they are of value (usually they have no value), but because farmers *will* use something, and it is perhaps best to mention remedies which for the most part are harmless, so that they may be employed by the poultry keeper to satisfy his urge to "do something."

It is desired to draw attention to the fact that various home remedies for different diseases sometimes gain unmerited reputations. In some cases a disease will run its course, killing a certain number of birds; then mortality eases off, and if at this time medicinal treatment is adopted, undue credit is given to the medicament as a curative agent, whereas the mortality would have eased in any case. Where the disease is running its course, the only reliable check on the value of a treatment employed is to take a number of infected birds, divide them into two even groups, treating one group, leaving the other untreated, and comparing the results.

Differential diagnosis has been dealt with prior to dealing individually with each disease in the group. This again is convenient rather than logical, and it is hoped that listing the characteristic symptoms of different diseases will serve as a guide to those whose birds are suffering from one of the diseases mentioned, and thus save the tiresome reading of irrelevant matter.

With a view to making the book of some use to veterinarians, comprehensive technical articles on particular diseases are mentioned in appropriate places.

Acknowledgments.

Current literature has been freely drawn upon. Actually, material from over a thousand separate books, bulletins, articles, or abstracts of articles has been used, and as it is not possible to mention all, none of the authors has been acknowledged individually.

It is with pleasure and gratitude that the opportunity is taken to acknowledge the encouragement and co-operation of Mr. Max Henry, B.V.Sc., M.R.C.V.S., Chief Veterinary Surgeon, and the valuable technical criticism of Mr. W. L. Hindmarsh, B.V.Sc., M.R.C.V.S., Director of Veterinary Research, and of Mr. L. Hart, B.V.Sc., H.D.A., Veterinary Research Officer.

It is desired to acknowledge the helpful literary criticism and proof reading of my wife, H. Roslyn Hungerford, B.A. (hons.).

It is also desired to acknowledge the helpful co-operation of the editorial staff of the Department of Agriculture.

T G Hungerford

Department of Agriculture,
Sydney.

Scope of the Information.

The diseases dealt with do not comprise a complete list of diseases of commercial poultry. Many diseases which are of common occurrence and great economic importance in other countries receive no mention here (*e.g.*, fowl typhoid, infectious bronchitis, air sac mite infestation, fowl plague or pest, and many others), as they either do no occur in Australia or are not of economic importance.

Diseases have been dealt with in order of importance from the practical standpoint. Thus a disease such as fowl pox, which occurs on all farms, is dealt with at length, and a disease such as botulism, which is seen only occasionally, receives but brief mention.

Turkey and duck diseases, for the main part, receive only passing attention, as (with the exception of the diseases mentioned at length) heavy economic losses from disease in these species do not come under notice.

Contents.

CONTENTS—*continued.*

DISEASES OF POULTRY in New South Wales.

GENERAL CONSIDERATIONS.

In the control of poultry diseases one must remember that prevention is the aim. Flock treatment, though of great value in some particular instances, is, generally, only of secondary importance. Individual treatment of birds is rarely an economical undertaking for the commercial poultry farmer. Other reasons which contra-indicate treatment of diseased birds are:—

(1) Birds which have recovered from disease (*e.g.*, pullorum disease) may remain carriers of the condition.

(2) Birds which are temporarily affected may have a hereditary tendency to contract the particular disease (*e.g.*, leucosis) and should not be retained for breeding purposes.

(3) Birds which have been seriously diseased and do recover are sometimes stunted and unprofitable.

Such birds may be more susceptible to other diseases and parasitic infestation, and consequently may act as a source of infection to the rest of the flock.

Notifiable Diseases—

When diseases which are scheduled under the Stock Diseases Act, 1923-1934, affect a flock, the poultry farmer should remember that he is under legal obligation to report the matter to an Inspector under the Act. (A report to the Chief Veterinary Surgeon is, of course, taken as such.) The result of this action is twofold: (a) it enables the Department to assist the individual in his problem, and (b) it enables control measures to be applied, in order to limit the disease outbreak and prevent, if possible, its spread to neighbouring farms.

The diseases scheduled are: coccidiosis, fowl tick or fowl tick fever (*spirochætosis*), infectious catarrh, laryngo-tracheitis, psittacosis, pullorum disease, gape-worm infestation, fowl cholera, fowl plague, fowl pox (concerning vaccination only).

Predisposing Factors.

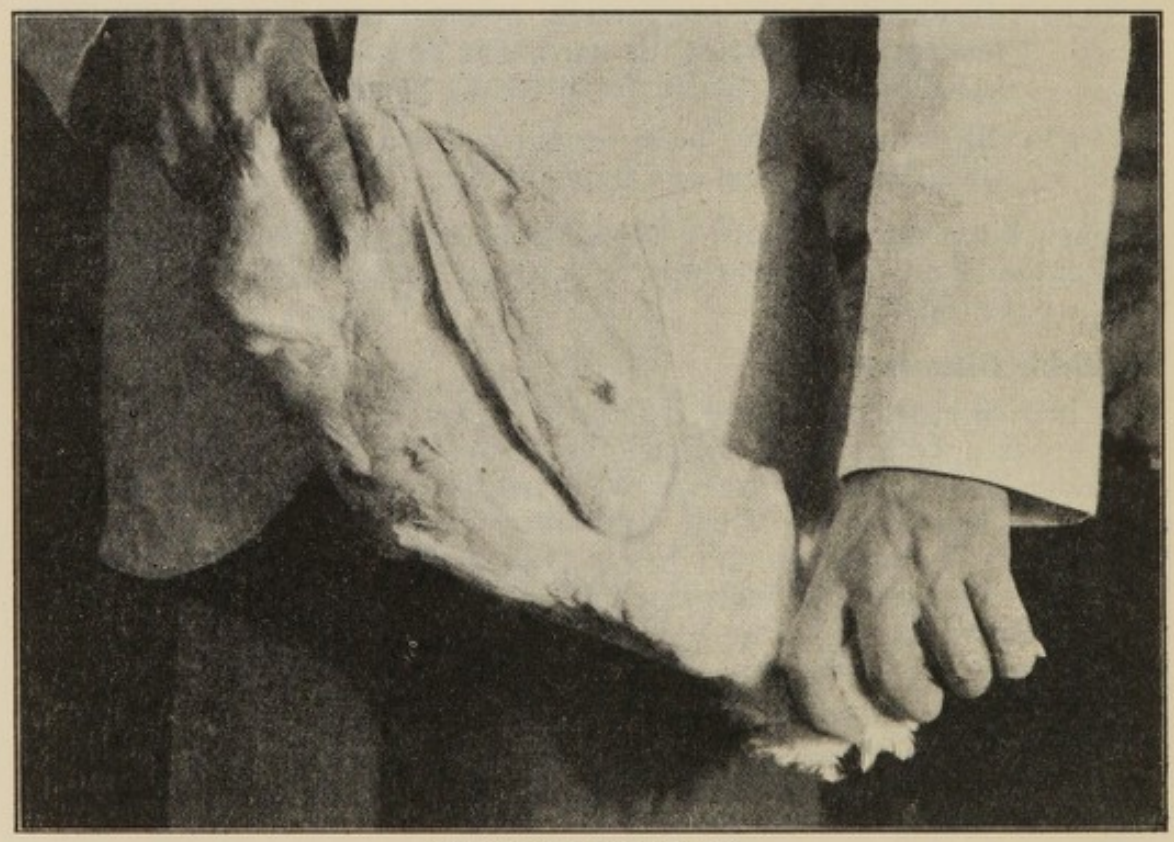
The cause of many diseases can be considered as twofold: (a) the actual cause (*e.g.*, the micro-organism concerned), and (b) the predisposing cause, which makes the birds more liable to contract a particular disease.

Of these predisposing factors, the following may be mentioned as most important:—

(1) *Deficiences in the Diet.*—Various dietetic deficiencies will rapidly manifest themselves in poultry, because of the large intake and output of food constituents by a laying hen, *e.g.,* if a hen receives insufficient vitamen A (supplied by green feed) a definite disease may be produced. Even without producing symptoms of this specific disease, the resistance to various catarrhal diseases and worm infestation may be lowered.

(2) *Bad Feeding.*—An incorrectly balanced ration lacking in one or more constituents, or having an undue excess of any constituent may actually cause disease (*e.g.,* rickets, cannibalism, visceral gout). Less severe deficiencies may lower the bird's vitality. Faulty feeding methods may predispose to disease.

(3) *Parasitic Infestation.*—Internal parasites, such as roundworms and tape-worms, may cause ill-health and lowered disease resistance. External parasites may cause irritation, lack of rest, and lowered disease resistance, and may also transmit disease micro-organisms.



"Wringing a Bird's Neck."

Notice that the fowl's head is held in the left hand with the little (fourth) finger elevating the beak as the head is pressed down rapidly to dislocate it from the neck.

(4) *Faulty Ventilation.*—The provision of correct ventilation is of utmost importance, as stuffy houses may predispose the birds to an outbreak of such a disease as coryza.

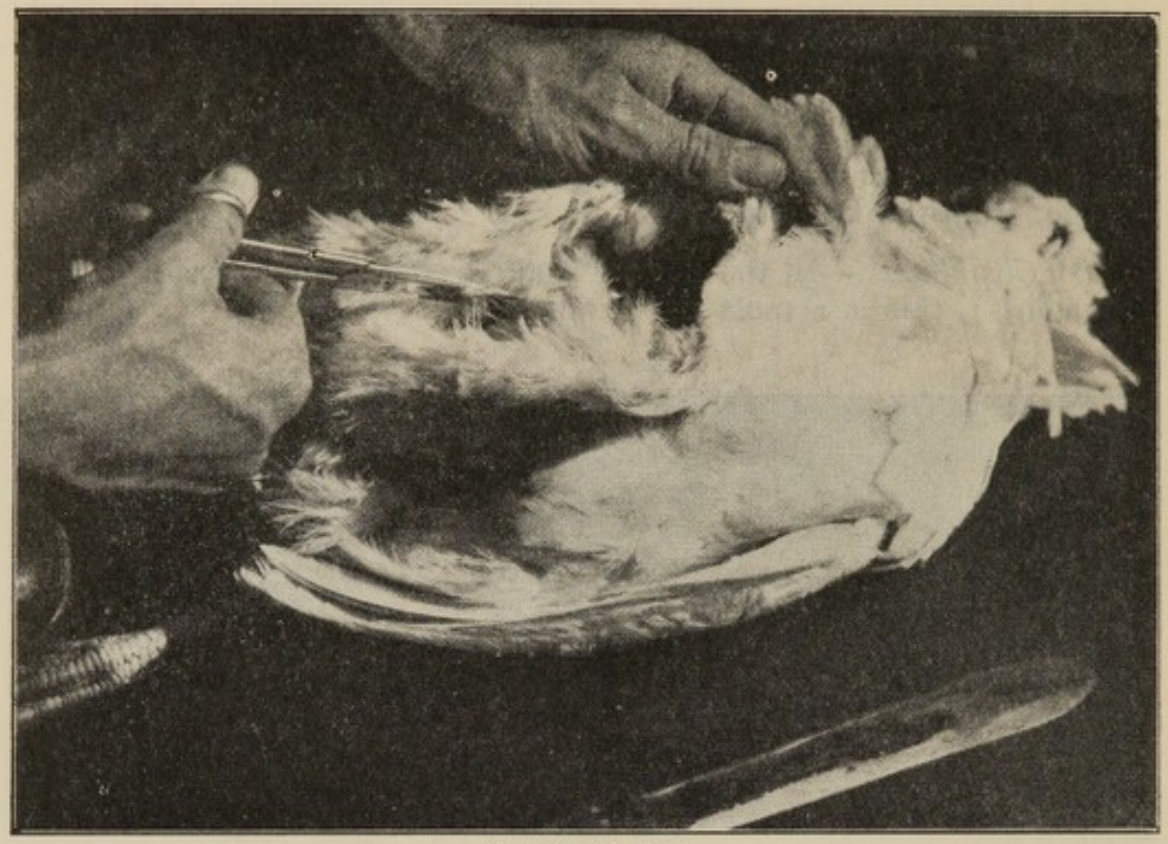
(5) *Over-crowding.*—This is of particular importance, and standard recommendations should be carefully followed. Excessive over-crowding predisposes to heavy worm infestation and assists in the spread of most disease conditions. (A pamphlet giving standard recommendations concerning housing of poultry is available, free of charge, from the Department.)

(6) *Cleanliness.*—In the case of some diseases (*e.g.*, coccidiosis and omphalitis), this is a most important factor.

An Alternative Method of Killing.

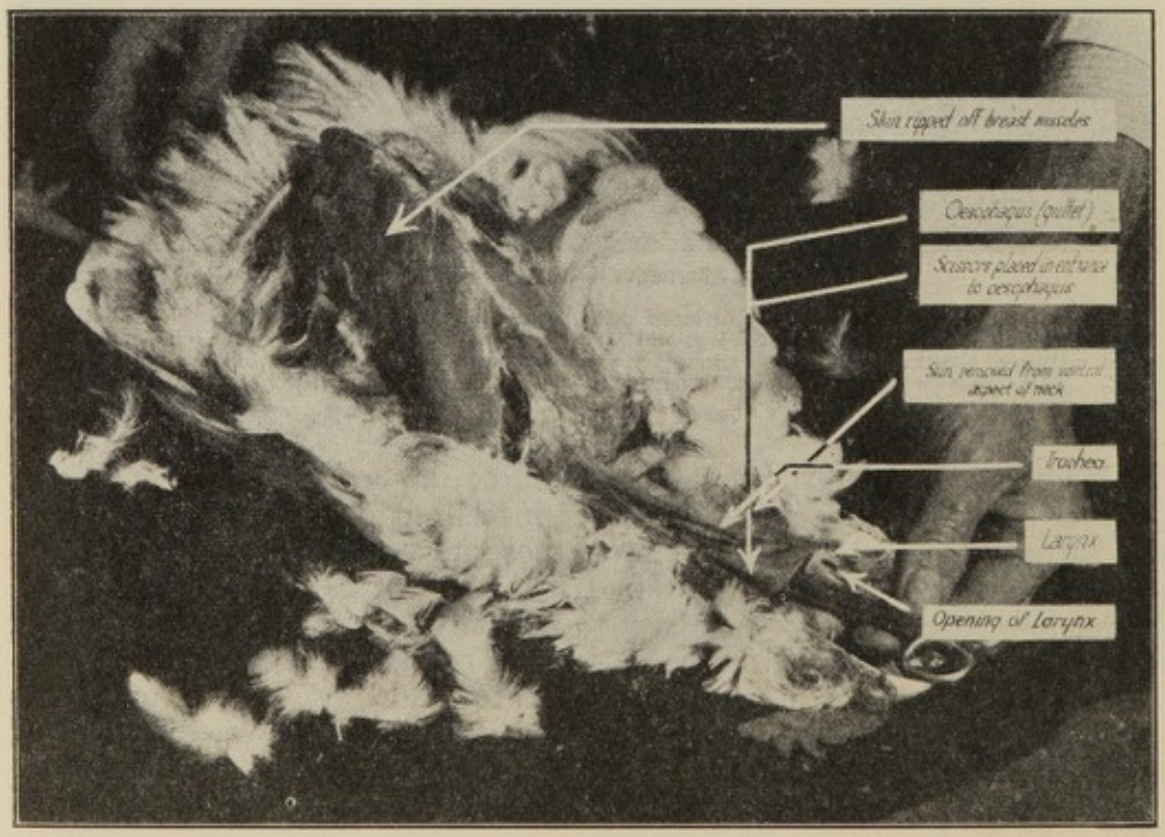
This method prevents the entrance of blood into the mouth, throat, and windpipe. Note that the operator's right foot is placed on the legs and wing tips of the bird, the fingers of the left hand grasp the tissues of the throat and upper windpipe, which structures are held clear, while the point of the knife (edge away from operator) is driven through the bony junction of the head and neck.

Whilst these predisposing factors are of importance, one should not forget that the fundamental cause of infectious disease is the presence of certain micro-organisms. Unless these micro-organisms are present, no amount of bad feeding or bad management can possibly produce infectious disease. It is incorrect to assume that these micro-organisms are *always* present awaiting a favourable opportunity to develop. This is not the case. If it were so, quarantine and eradication measures would be useless. The history of Newcastle disease in Victoria shows quite clearly that eradication measures, where practicable, can be most successful. The disease occurred on a wide scale, but was stamped out by radical slaughter and quarantine measures. The vigorous action of the Victorian veterinary services in the case of that disease saved the Australian poultry industry from enormous losses.



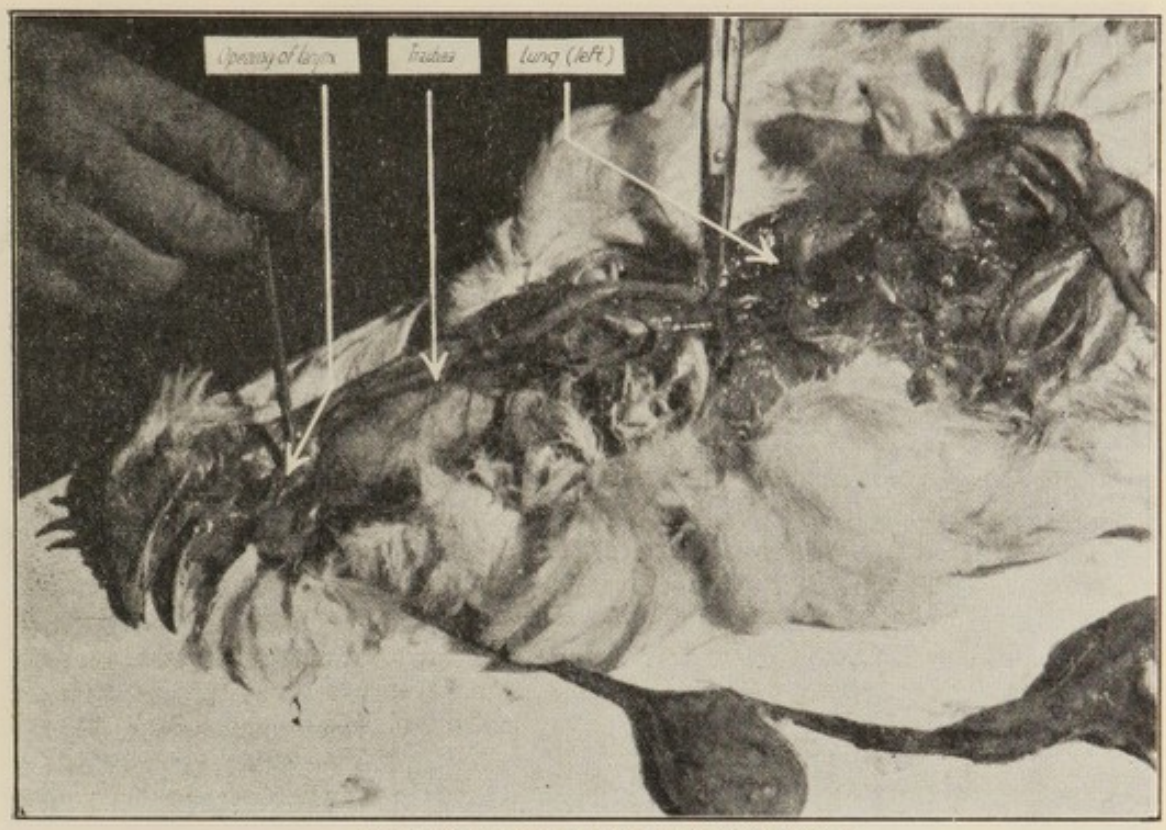
Skinning the Bird.

The skin is first incised with scissors, then ripped off the entire ventral aspect of the bird from cloaca to beak.



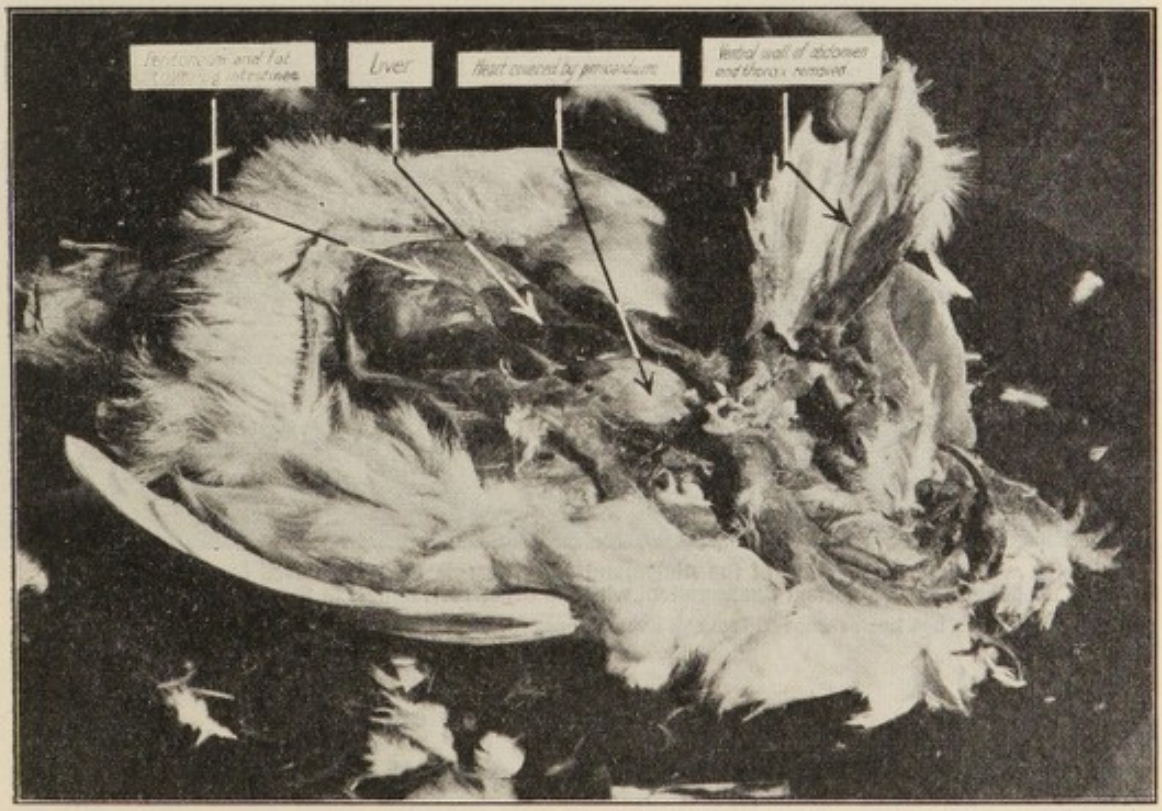
Next Stage.

The skin has been ripped off the entire ventral aspect of the bird, and the junction of the lower and upper beak has been cut through. The scissors are held in the opening of the oesophagus (gullet) just above the opening of the gullet—the opening of the larynx can be seen.

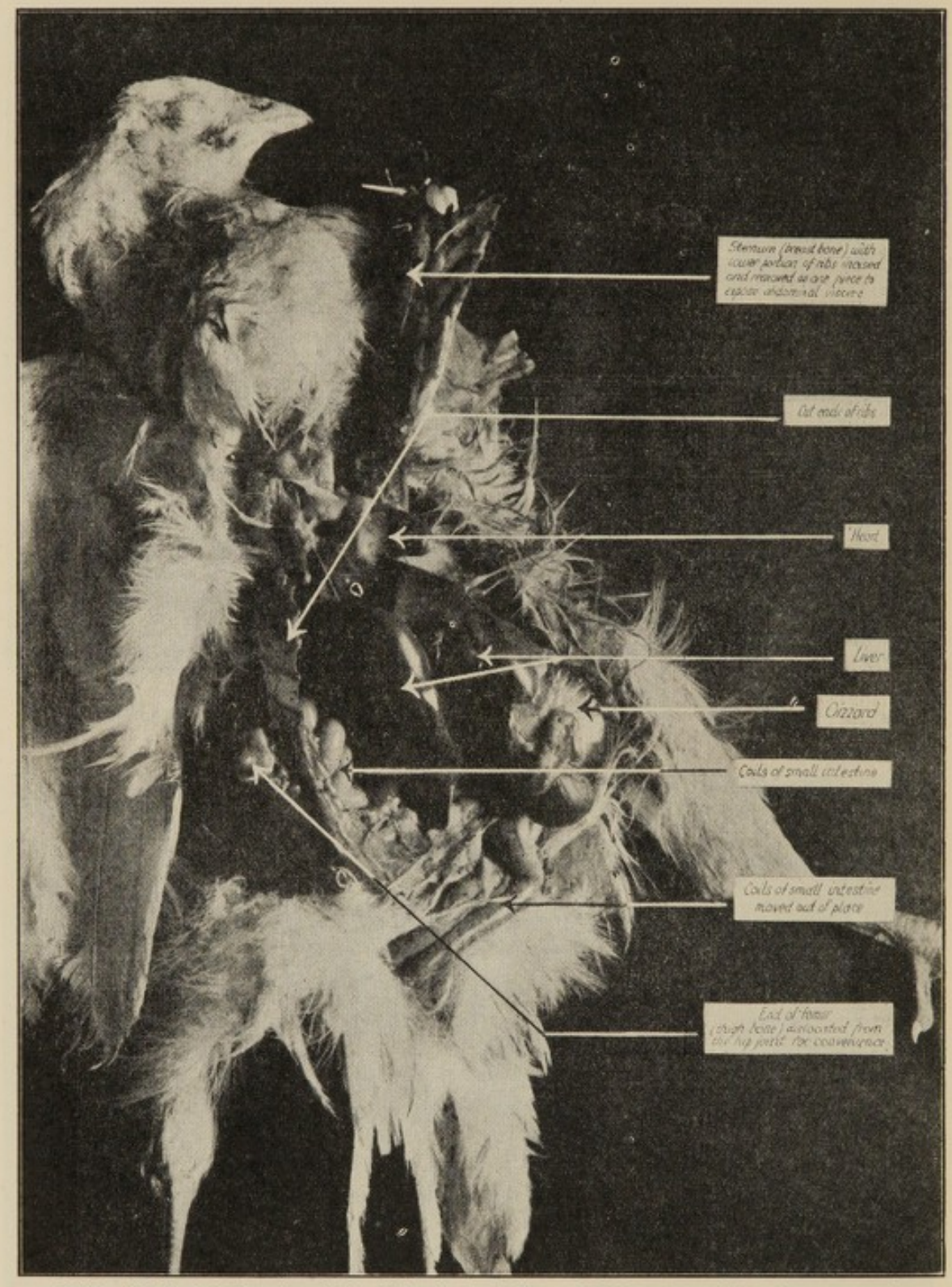


View of a portion of the Respiratory System.

Note the end of the probe placed at the opening of the larynx and the scissors point placed on the lower end of the trachea (windpipe) where it divides into bronchi.



Another view of the Bird with the Ventral Wall of the Abdomen and Thoracic Cavity removed to Expose the Viscera.



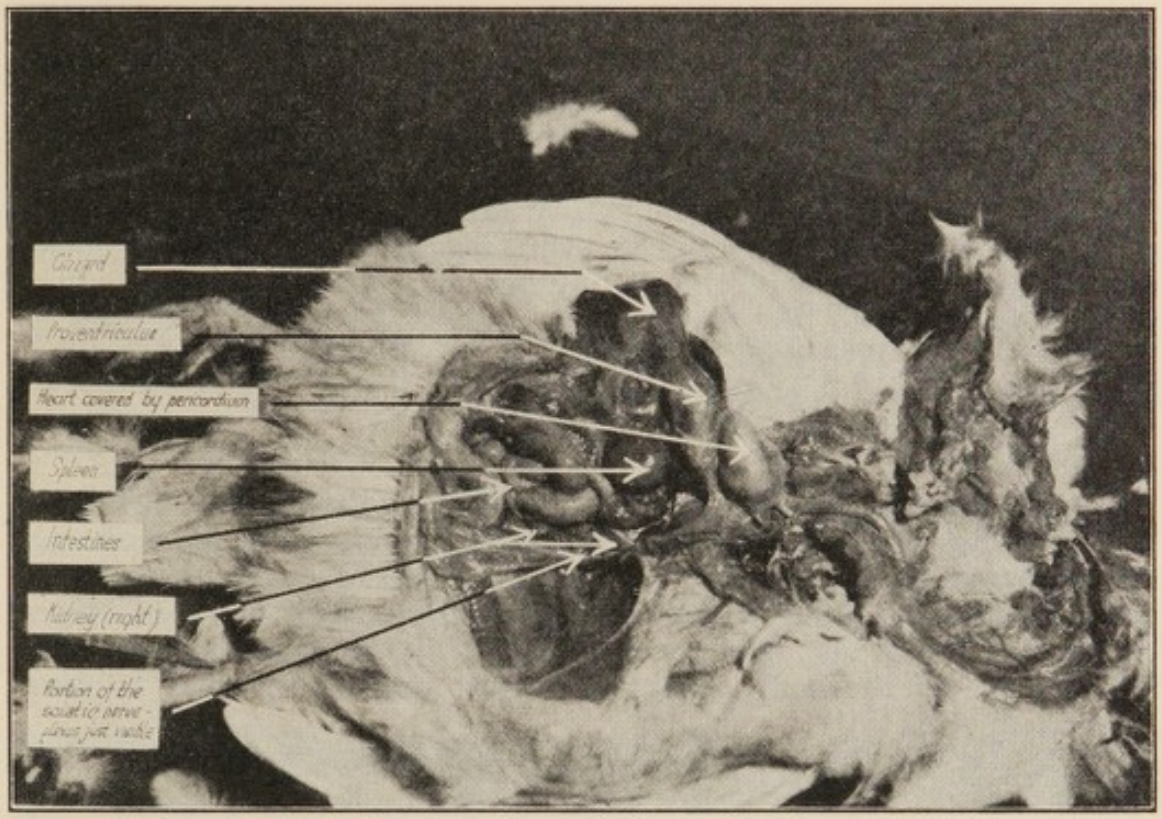
Pullet with Ventral Covering of the Abdominal and Thoracic Cavity removed for the purpose of conducting a Post Mortem Examination, with the Organs left almost in their natural position.

Skin has not been removed from the ventral aspect of the crop and neck in this case.

Post-mortem Examination.

In arriving at a diagnosis, the history of the disease outbreak, the clinical symptoms and the post-mortem lesions need very close investigation.

Concerning the latter, *i.e.*, post-mortem examination, where the identity of the disease is not quite definite, a veterinarian may take sterile Pasteur pipettes from affected organs for bacterial culture, as well as from the heart blood, spleen, and other sites whether they show abnormality or not. Portions of tissue, or organ contents may also be collected from affected organs, and blood and other smears may be



The Proventriculus and Gizzard moved to one side to expose the Spleen and Right Kidney.

made if this is indicated. If poisoning is suspected, gut contents and affected organs may be collected for analysis. These measures can be undertaken *only* by a veterinarian, as specialised knowledge is necessary to know just what is required. It is, however, important for a farmer to be able to conduct a post-mortem examination, and to make intelligent observations of the obvious abnormalities present.

A detailed knowledge of anatomy and physiology is not necessary, and even a summary of such knowledge is not included in this volume, as such summaries are of little, if any, practical value. Most poultrymen can recognise the identity of the internal organs, and the illustrations show their relative positions. A farmer would be well advised to kill and examine one healthy bird in the manner described below, and after the identity of each organ has been recognised, he should examine the organs of normal birds killed for table purposes, to get a good idea of normal appearances.

(a) Killing—

In killing a bird that has not shown any abnormalities involving the head or neck region (*e.g.*, coughing, "roup," etc.) the neck may be "wrung" in the usual way. The bird's legs are taken firmly in the

right hand, the head is taken in the left hand, with the bird's beak resting on the fourth or little finger, and the skull against the base of the index finger; the head is then twisted backwards, and downwards, so that the bony junction of the skull and neck is broken.

Where a careful examination of the head cavities and trachea is to be made, the above method should not be adopted, as occasionally blood may gain entrance to the trachea.

In such cases, a better method is to place the tip of both wings under the right foot and both the fowl's legs under the operator's left foot, with the fowl prone on its right side. Just sufficient pressure is placed on the legs and wing tips to hold them in position. With the thumb and index finger of the left hand the loose skin of the throat is grasped so that the throat and windpipe are clearly separated away from the bony skeleton of the head and neck. A sharp butcher's knife is then taken in the right hand, and with the edge turned away from the operator the point is driven down through the bony junction of the skull and neck. The bird is then held until struggling ceases. By this means access of blood to the throat is completely prevented.

(b) Skinning—

The bird should now be placed down, back to the ground, and both hip joints dislocated by merely rotating the leg out and down. This can be done most conveniently as the skin is pulled off. A snick with a pair of scissors is made in the skin near the vent and the skin is then ripped up the mid line, over the breast, crop and right to the head, and pulled back off the body, using the fingers only—this leaves a clear featherless field.

(c) Opening the Bird—

The scissors are inserted in the mid line near the vent, and, cutting round the side of the body, the abdominal wall is incised. The line of incision is continued through the ribs until the stout bones of the shoulder girdle ("wish-bone" and "collar" bones) are encountered. This line of incision is completed round both sides.

If the operator's left thumb is now placed under the point of the breast bone, the rest of the fingers along the keel or crest of the breast bone (face of hand down), and the carcase held firmly with the right hand, the whole ventral covering of the body can be neatly wrenched upwards, snapping the bones of the shoulder girdle in the act. After snipping the remaining muscles of the shoulder girdle, the whole covering can be lifted off, leaving all the internal organs undisturbed and in their natural positions.

This is a great advantage, and care should be taken to examine each organ in its natural position, and then to remove it neatly (by snipping out), leaving the other organs in the natural location until examined.

(d) Examining—

On opening the bird as indicated above, the liver, gizzard and some of the intestines are apparent. These should be examined for any abnormality. The glandular stomach (proventriculus) which is attached on the head side of the large muscular gizzard should now be located.

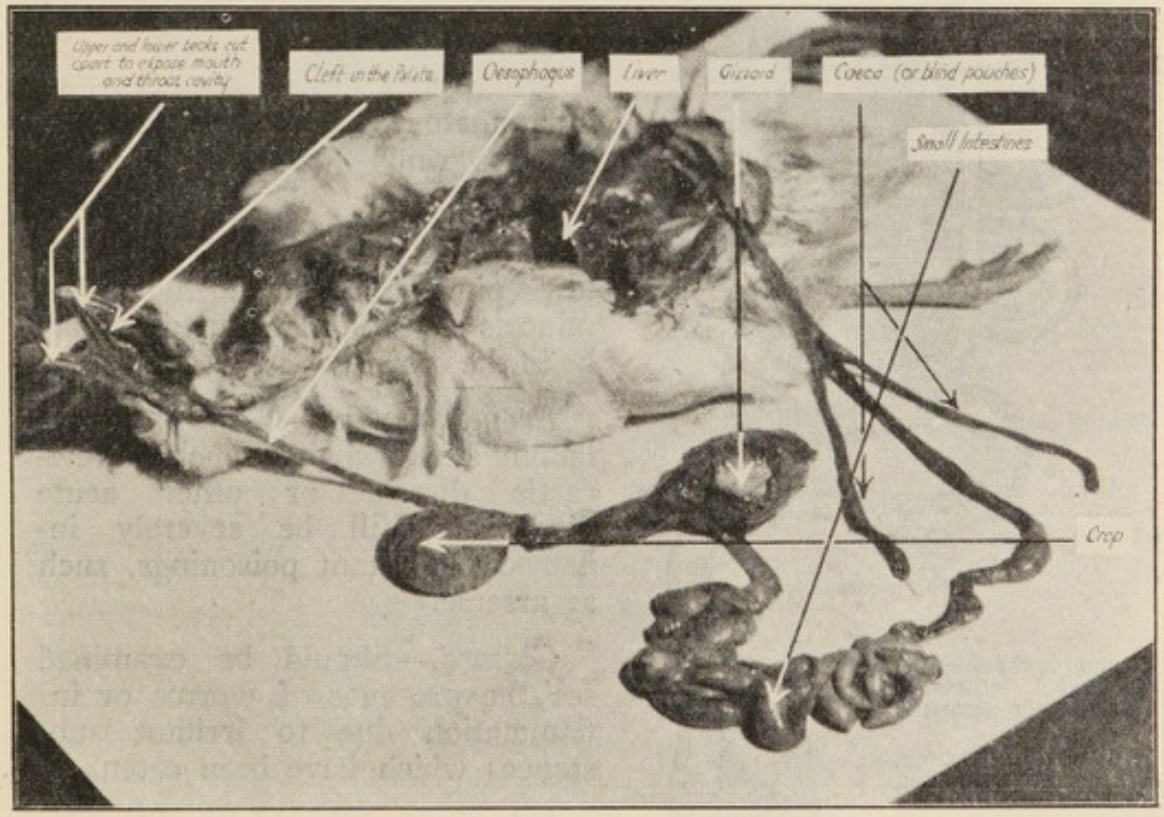
The junction of the gullet with the proventriculus should be snipped through, and after snipping away the attachments of the stomach and gizzard, the intestines should be drawn out. It will be necessary to snip these organs clear from the liver and spleen. Both these organs should be carefully observed during the process.

The proventriculus and gizzard should now be slit open and the horny lining of the latter peeled off. The intestines should then be slit down from one end to the other. Care should be taken to examine thoroughly all structures, not overlooking the pancreas, which is situated between the looped portion of the first part of the intestine. Both caeca or blind guts should be slit up with the scissors.

The rest of the organs should now be carefully examined and removed one by one—liver, lungs, spleen, ovary (or testicles, in the case of male birds) and kidneys.

Turning towards the front of the bird, the mouth is slit open from the beak down the gullet, laying the latter open to the crop. The nasal cavity and infraorbital cavities should next be slit open. The windpipe (trachea) should then be cut down and opened out right from the top (larynx) down to the lungs.

The above comprises a very crude examination, but will allow most necessary observations to be made. Such an examination can be carried out in two or three minutes when one becomes thoroughly proficient and, apart from killing the bird, a pair of sharp scissors is the only equipment required.



The Digestive Tract Separated Out.

Note the two long blind pouches or caeca. In suspected outbreaks of coccidiosis, or blackhead, particular care should be taken to locate, open, and examine these.

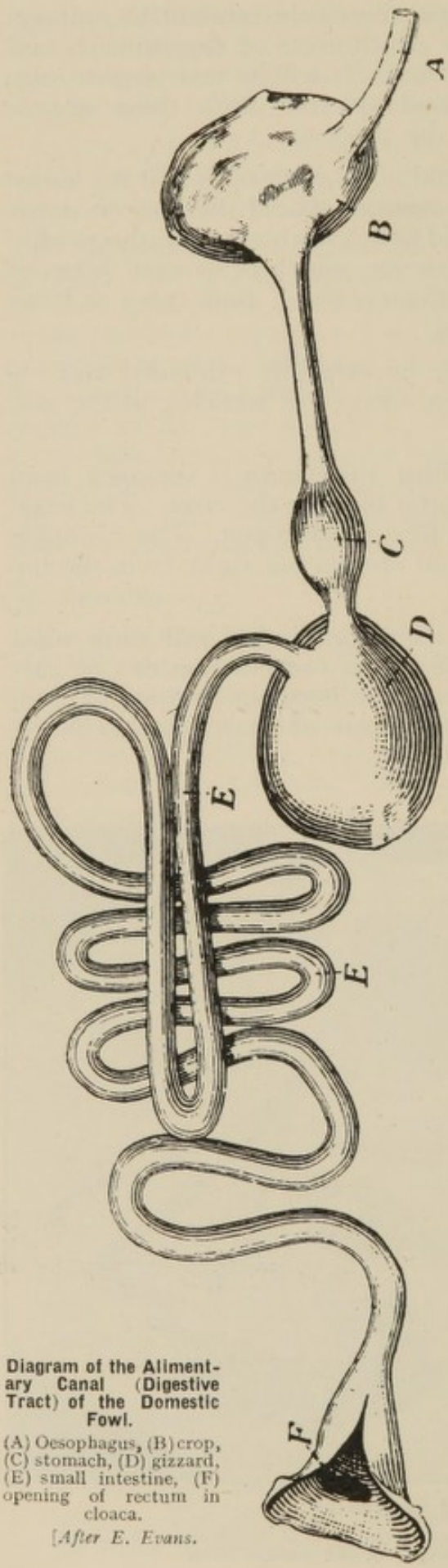


Diagram of the Alimentary Canal (Digestive Tract) of the Domestic Fowl.

(A) Oesophagus, (B) crop, (C) stomach, (D) gizzard, (E) small intestine, (F) opening of rectum in cloaca.

[After E. Evans.

Care might be taken to observe the abdominal and thoracic air sacs, which are cavities lined by a thin membrane. The Bursa cloacæ (of Fabricius) on the dorsal aspect of the cloaca in young birds should also be opened and examined.

The likely changes in each organ are as in the section headed "Post-mortem Lesions" under each disease, but the following rough resumé of the diseases indicated by a particular abnormality may be helpful:—

Nasal Cavity.—Mucoid discharges, cheesy deposits, or inflammation of the nasal cavity indicates a catarrhal (or roup) disease, as do similar changes in the sinuses and turbinate bones connected with the nasal cavity.

Larynx and Windpipe.—Inflammation and free bleeding of their lining membranes may indicate laryngo-tracheitis or other changes may indicate various catarrhal (roup) diseases.

Mouth and Throat show various inflammatory changes when infected with various catarrhal (roup) diseases.

Gullet (or Oesophagus).—Yellow pimples indicate hypo-vitaminosis A. Similar pustules may be in the crop.

Proventriculus.—May be inflammed in laryngo-tracheitis, Newcastle disease or other acute diseases. Will be severely inflamed in irritant poisonings, such as arsenic.

Gizzard.—Should be examined for the presence of worms or inflammation due to irritant substances which have been eaten.

Intestines.—Should be slit from end to end and should be examined for inflammatory changes which

may indicate coccidiosis, entero-hepatitis, worm infestation, leucosis, bacterial infection, such as infection with *Bacillus coli*, enteric pullorum disease, cholera, and irritant poisons such as salt or arsenic.

There may be casts in the intestines, for instance, in the caeca, which indicate coccidiosis or blackhead (*Enterohepatitis*). The small caecal worm is usually present in the blind end of the caeca, but unless present in very large numbers may be disregarded.

Structural abnormalities should be noted, such as tumour formations (*e.g.*, in leucosis), ulcers of the lining membrane, or shedding of the lining membrane. Small spots visible from the outside of the intestine may indicate one type of coccidiosis.

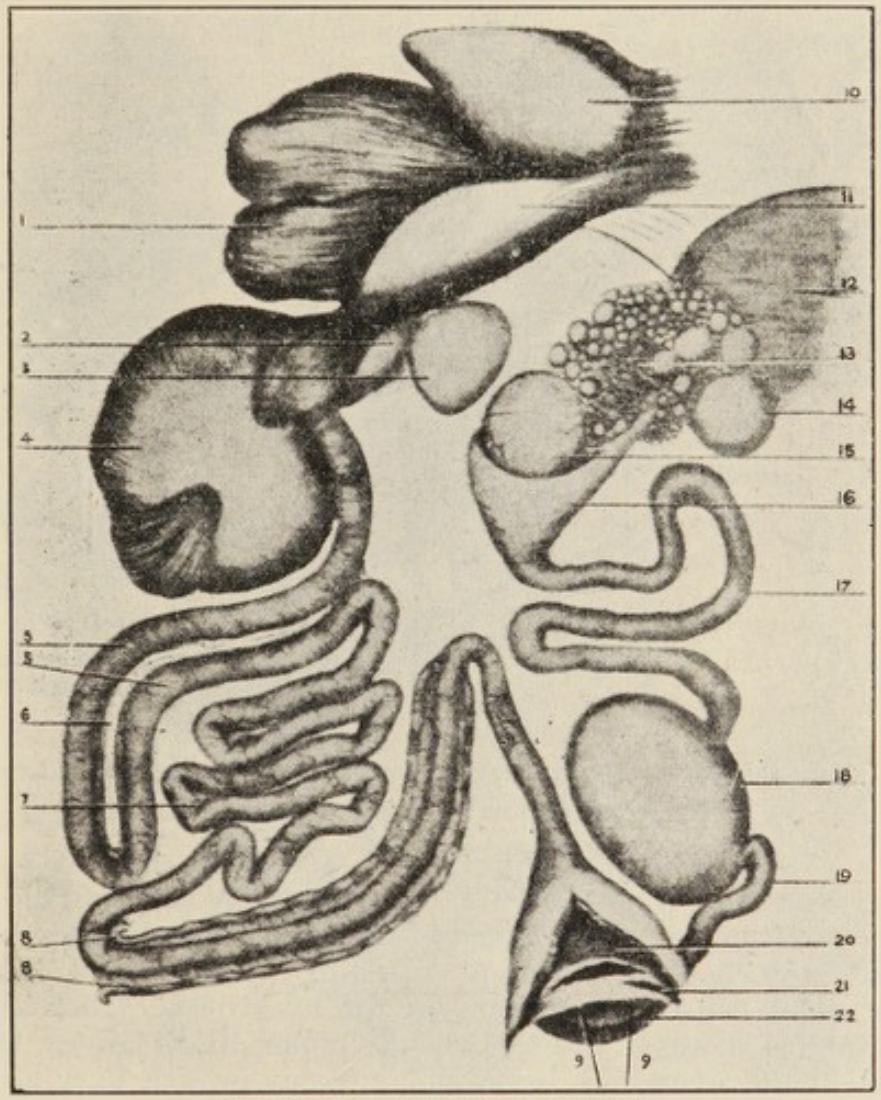


Diagram of Organs and Ovary of Hen.

(1) Liver, (2) gall bladder, (3) spleen, (4) gizzard, (5, 5) duodenum, (6) pancreas, (7) intestine, (8, 8) free extremities of caeca, (9, 9) opening of ureters, (10) heart, (11) stomach, (12) lung, (13) ovary, (14) egg, (15) egg entering oviduct, (16) infundibulum, (17) oviduct, (18) egg passing through oviduct, (19) oviduct, (20) cloaca, (21) opening of the oviduct, (22) margin of anus.

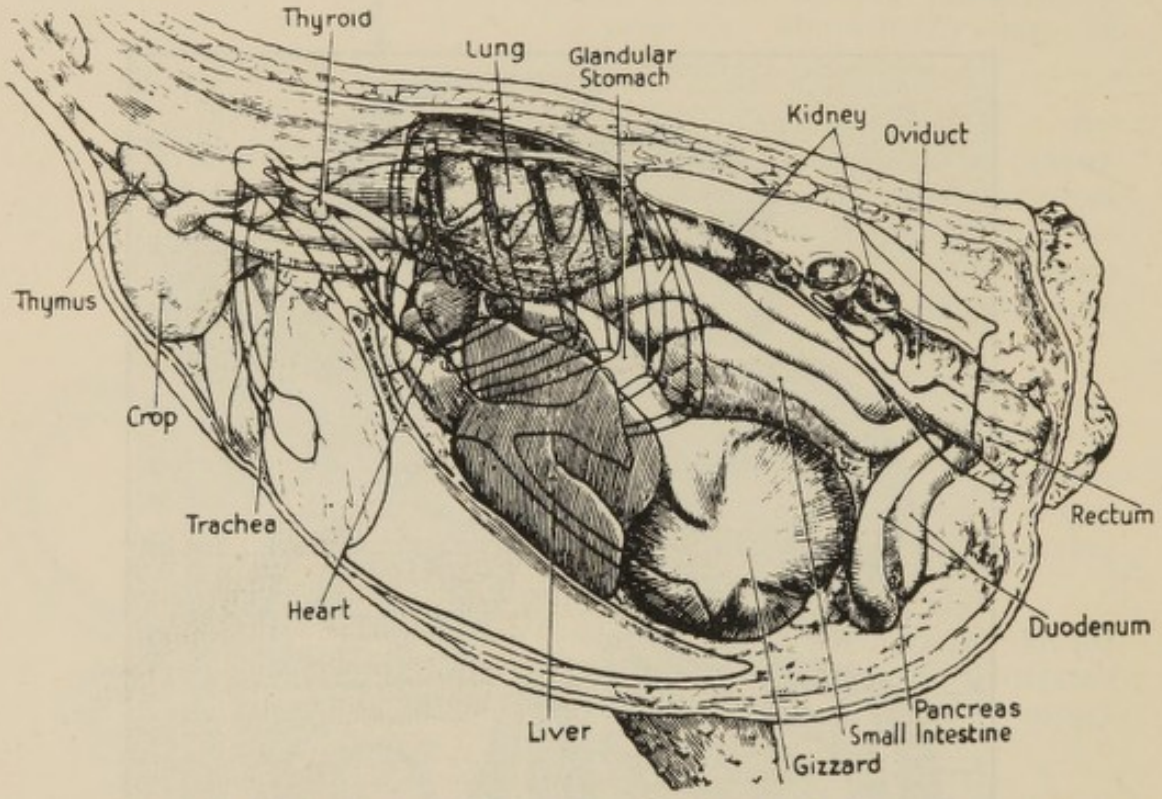
[*After Bradshaw.*

Cloaca.—Inflammation may be due to vent gleet or other specific infections of the oviduct or intestines.

Oviduct.—May be inflamed showing diffuse reddening or may be affected with abscessed conditions. Egg masses, or other foreign bodies, may be present. Ovarian diseases are extremely common in this State, and precise investigation in this group of diseases is needed, as it would appear that some of them are highly infectious. In some cases inflammation of the organ is due to pullorum disease infection.

Ovaries.—Are sometimes inflamed. The presence of dark, hardened, discoloured ova is typical in chronic carriers of pullorum disease.

Liver.—Abnormalities are very common, as this organ plays an important part in digestion and assimilation. It also has detoxifying and excretory functions, thus changes are common in a large number of

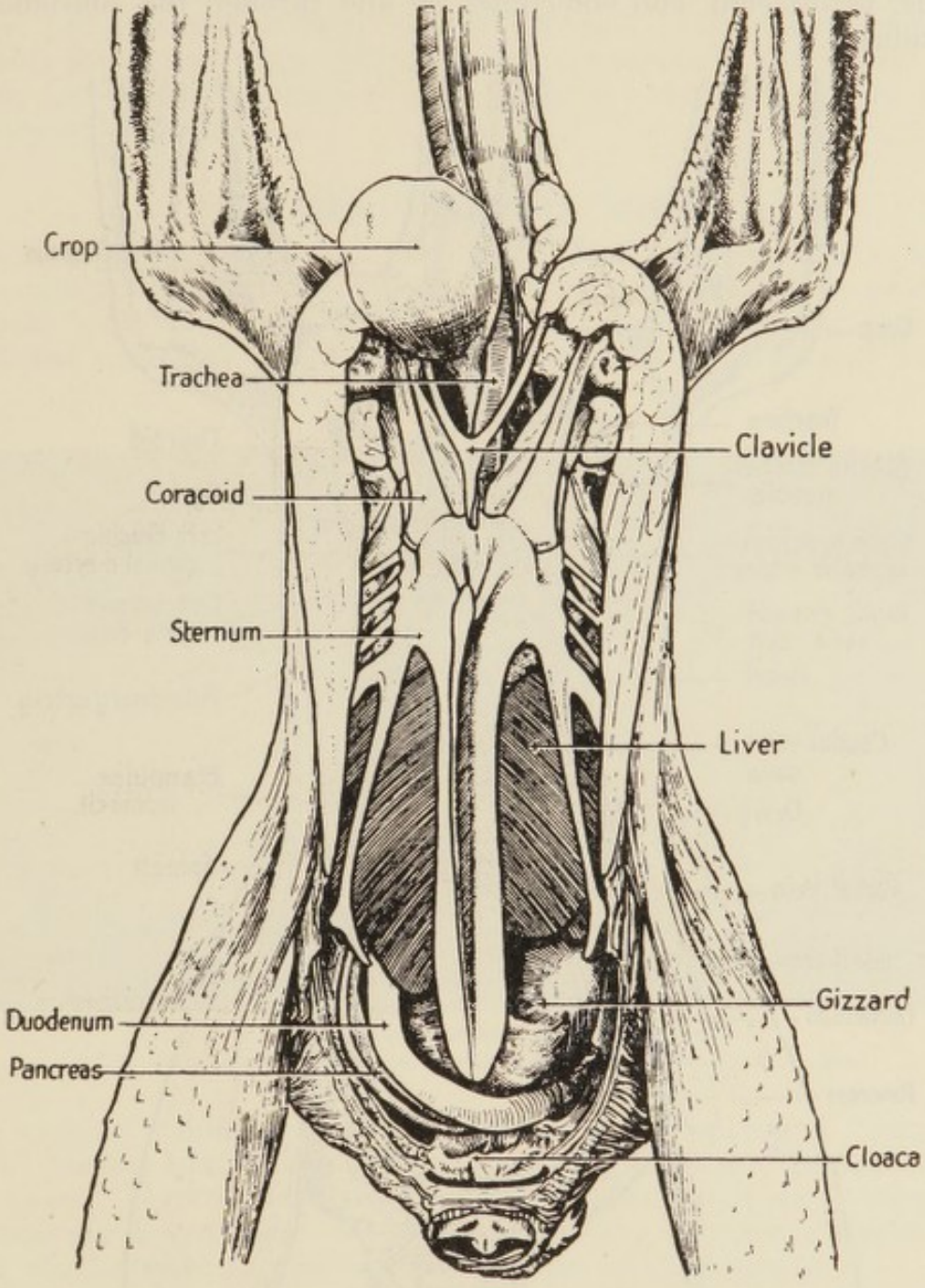


Thoracic and Abdominal Viscera Organs (Hen), viewed from the Left Lateral Aspect.
The skeleton, which has been removed, is outlined.
[*After Bradley.*

diseases. Normally, it should be a dark mahogany colour. Pale coloured abscesses or tumours are frequently noted in such diseases as enterohepatitis, spirochaetosis, various types of leucosis, and in systemic diseases which cause degeneration of the liver tissue (such as cholera, some catarrhal diseases and septicaemic pullorum disease). Discoloured areas in the liver may be from pin-point in size to a large mass, which involves the complete liver lobe. Because liver lesions are so common, many symptoms have obtained the name of "liver disease," because on post-mortem the liver is found to be affected. Tuberculosis, mycosis and trichomoniasis may show typical liver lesions (see text). Cancerous tumours may also be present.

Spleen.—This organ is usually about the size of a small grape. In septicaemic diseases, such as septicaemic pullorum disease, cholera and spirochaetosis, it will be greatly enlarged. In the latter disease there

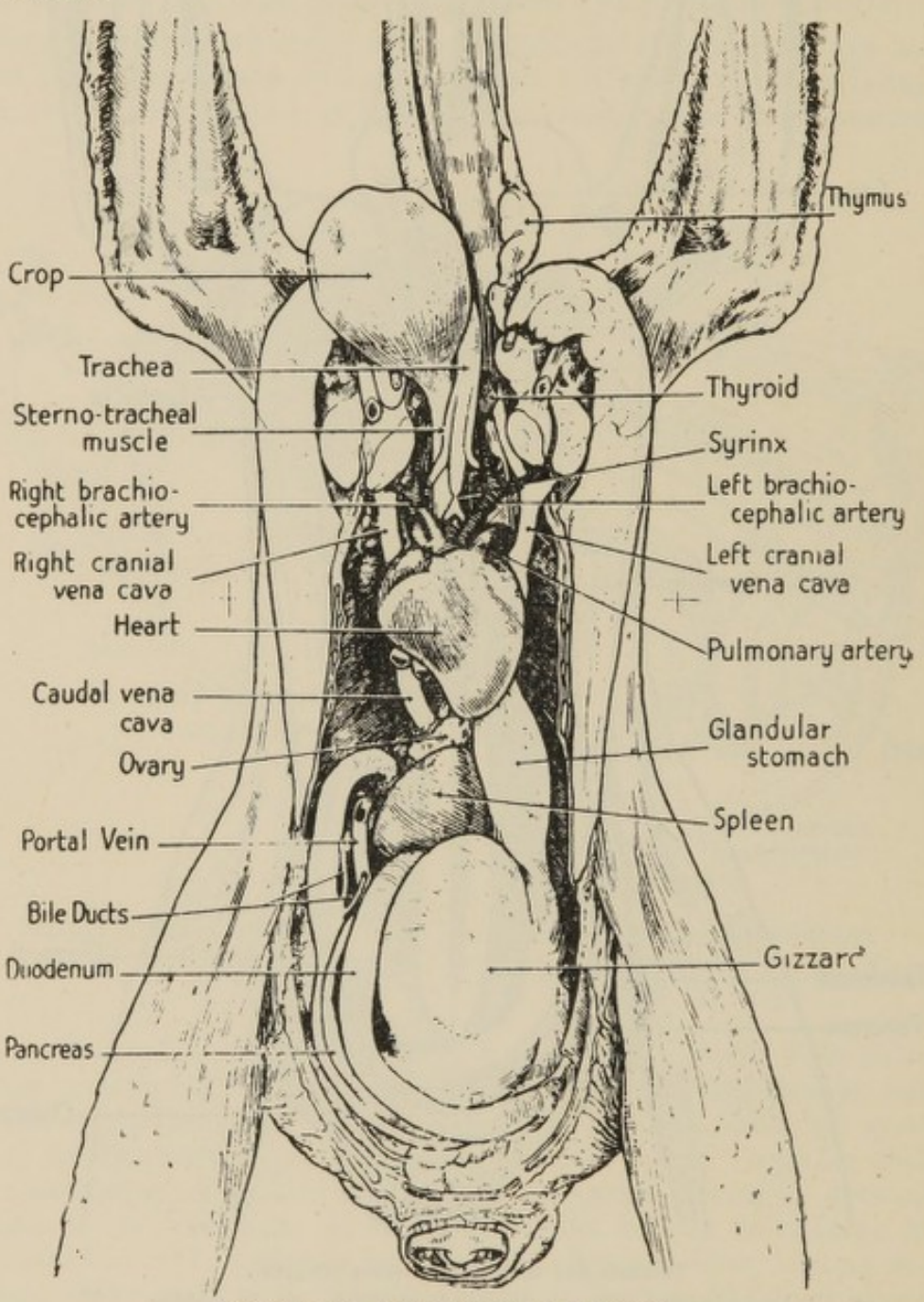
are yellow sago-like patches throughout its substance. In the leucosis diseases large tumorous masses may be formed in it. Apart altogether from the tumorous masses, in the leucosis group of diseases it is usually much enlarged. Characteristic abscesses are seen in a number of diseases (see text).



Thoracic and Abdominal Viscera (Organs).
Ventral aspect, with the sternum (breastbone), etc., in position.
[*After Bradley.*

Pancreas.—This is an elongated organ situated between the loop of the first portion of the small intestine, and is sometimes inflamed. This inflammation is usually associated with inflammation of the intestines or the peritoneal cavity in generalised disease conditions.

Heart.—Normally is red in colour, and closely enveloped by the pericardial sac. In septicaemic diseases, such as cholera, there may be haemorrhages in the heart muscles. The presence of excess fluid in the pericardium and also chalky deposits may indicate either septicaemic pullorum disease or visceral gout. In the latter condition, chalky deposits are present on the outside of the pericardium, on the abdominal organs, peritoneum, and sometimes on and through the substance of the kidneys.



Thoracic and Abdominal Viscera (Organs).

Ventral aspect after removal of the liver (non-laying hen).

[*After Bradley*

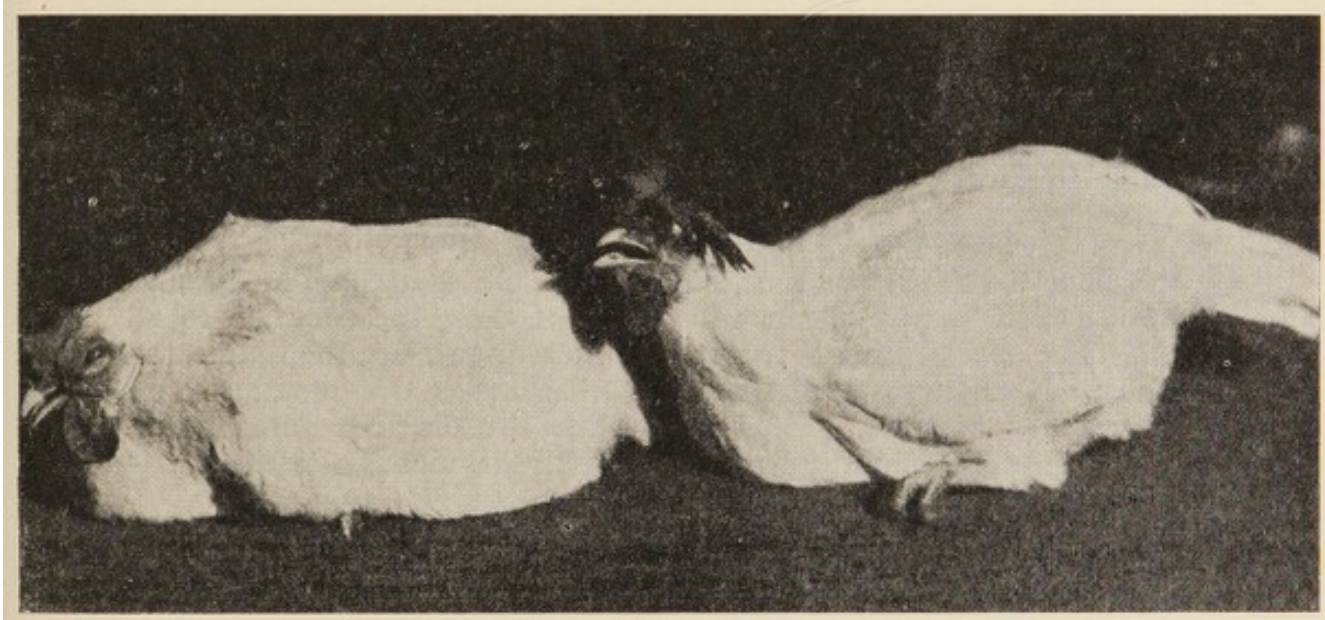
Lungs.—Are not frequently noted to be affected. The presence of yellow, greyish or black patches or concentric rings is indicative of mycosis (fungal infection). Very rarely the lungs are oedematous

(soggy) in laryngo-tracheitis. Inflammation of air sacs may be due to infestation with air sac mites or infection with fungi, in which case there may be yellow deposits in them, or masses of fungal, fluffy material, or the whole lining may be covered with dark or black slimy material.

Kidneys.—Normally, are red in colour and embedded in the cavities of the pelvic bones. Chalky deposits on their surface and crystalline deposits throughout their substance with distension of the ureters with urates indicate either visceral gout or gout conditions, due to a deficiency of Vitamin A.

DISEASES CLASSED AS "ROUP."

In New South Wales the term "roup" would appear to be as old as the poultry industry. It is a term applied to quite a large group of diseases with somewhat similar symptoms, and, depending upon the particular variety of symptoms present, a particular bird may be labelled



Two Cases of Infectious Laryngo-tracheitis.
Left—Chronic case, with cheesy deposit in the eye. *Right*—Peracute case.

as a case of "diphtheric roup," "eye roup," "nose roup," "catarrhal roup," "canker roup," "throat roup," "nutritional roup," "internal roup," and so on. Correct differential diagnosis of these conditions is important as some can be prevented or cured. For others, no cure is known, but steps can be taken to minimise the severity of their attack.

When "roup" affects a flock a correct diagnosis must be made before rational measures of control or treatment can be recommended. Failure to obtain a correct diagnosis has often proved a costly matter. One such case may be cited as an example.

A farmer's pullets started to die from "roup." They were coughing and had cheesy masses in the mouth and eyes. He spent some money on proprietary remedies without result. After about 200 pullets had died, competent advice was sought, the condition diagnosed as green

feed deficiency disease, and he was advised to feed cod liver oil in appropriate amounts. Under this treatment the disease disappeared with the usual suddenness. About a month later another outbreak of "roup" occurred. The owner felt he knew all about "roup" now, and spent money feeding copious quantities of cod liver oil (though by this time he had plenty of green feed). The cod liver oil had no effect in this case, and so he concluded this cure for "roup" was all a hoax. Advice was sought again, and the condition diagnosed as acute infectious catarrh. (The possibility that the disease was laryngo-tracheitis was eliminated by laboratory examination.) It was pointed out that there was no definite cure for the condition, but that certain measures should be taken to maintain the fowls' natural resistance. The owner would not believe there was no cure, but spent much more money on certain proprietary drugs. The infectious catarrh ran its typical course and then cleared up. The particular preparation in use at the time when the symptoms abated received the credit for curing the condition. Had advice been sought early, and followed, the first 200 deaths would not have occurred. Further, the birds' natural resistance would not have been lowered, so that the second disease outbreak, if it had occurred, would not have taken such a heavy toll. Furthermore, the money spent on cod liver oil, unnecessarily used in the second outbreak of disease, and on drugs, would have been saved.

The above case should illustrate that veterinary advice is frequently a valuable investment.

Differential Diagnosis of the "Roup" Diseases.

It will be noted in the attached diagram that the symptoms of diseases commonly called "roup" are listed. Some of these are common to all diseases. The special clinical features of each disease are then indicated separately. It is these special features that make any separation on clinical grounds possible. Thus, to explain the diagram further, the following points are important as distinguishing symptoms of the particular disease which is affecting the birds:—

(1) Fowl Pox—

There are three forms of this disease (see later). The "canker" form shows all the symptoms of roup. In the cutaneous form, warts are present on the comb and wattles. The oculo-nasal form may strongly resemble coryza (or colds), in which the symptoms comprise mucous discharges from the nose and eyes, and sometimes inflammation and swelling of the structures surrounding the eye.

(2) Green Feed Deficiency Disease—

Green feed (or its substitute) will have been absent in the ration for some little time. In addition to the usual symptoms of roup, yellow pustules or pimples are present on the lining membrane of the gullet, and may extend even to the crop.

(3) Infectious Laryngo-tracheitis—

In addition to the "roup" symptoms, there is severe inflammation of the wind-pipe, and in the most acute form of the disease there may be bleeding from its lining membrane, which results in the coughing of blood.

[To face page 22

DIFFERENTIAL DIAGNOSIS OF THE "ROUP" DISEASES.

Fowl Pox.—The oculo-nasal form resembles coryza. The cutaneous form does not show "roup" symptoms but only pox lesions on head and at times on body.

The canker form shows "roup" symptoms.

Coryza (a "roup" disease) does not show all these symptoms:—Lachrymation (tears), later frothy mucous discharge from nose and eyes, congestion and inflammation of nose, throat and larynx, but no cheesy material. Affects chiefly pullets in autumn—about 90 per cent. of flocks affected.

General Symptoms of "Roup."

Roup in its various forms shows the following:—Cheesy (diphtheritic) deposits in mouth, on tongue, palatine cleft pharynx (throat), larynx (top of windpipe), in nasal cavity and infraorbital sinuses. "Bunged up eyes," i.e., panophthalmia with cheesy material distending the orbital cavity. Cheesy material may be present on the top of the larynx and cause suffocation. Coughing, gasping and gaping may be present. Poor condition, wasting diarrhoea, and at times acute emaciation, exhaustion, and death.

In addition to the above general symptoms of "roup," the particular roup diseases have the following characteristic symptoms:—

1. Peracute Laryngo-tracheitis.—Haemorrhages occur in the larynx and trachea (windpipe). Congestion and inflammation of trachea. The bird coughs out bloody mucus or pure blood, shows great distress, weakness, and dies in a convulsion.

If it recovers it passes into a case of chronic laryngo-tracheitis.

2. Subacute and Chronic Laryngo-tracheitis, in which some congestion (reddening) or inflammation of larynx and trachea is present.

3. Infecticus Catarrh shows similar symptoms to subacute laryngo-tracheitis (differentiate by cross immunity tests).

Green-feed Deficiency Disease (Avitaminosis A).—Yellow pimples are seen in throat, gullet, and even as far down as the crop. Whitish flakes in the eyes and cheesy material right down the windpipe at times.

In these three (1 to 3) types of "roup" the birds cough and may cry out in a shrill manner due to the mucous obstruction in the lower portion of the windpipe.

(4) Infectious Catarrh—

This resembles a mild attack of laryngo-tracheitis and cannot be differentiated on clinical grounds with any certainty, but cross-immunity and other laboratory tests must be depended upon.

(5) Coryza (or Colds)—

In this condition "canker" formation is the exception rather than the rule. Otherwise the "roup" symptoms are in evidence.

(6) Sinusitis—

This occurs as a specific disease in turkeys only.

(7) Localised Forms of Cholera—

Affecting the mucous membranes of the nose, pharynx and mouth may produce symptoms of roup. Such cases have not been seen in New South Wales, however.

The above differential diagnosis is, of course, based on clinical grounds. It is stressed, however, that in many cases laboratory confirmation of the diagnosis is of critical importance. Thus, in the case of suspected infectious catarrh, a recovered bird can be tested for immunity to laryngo-tracheitis. If the disease has been infectious catarrh, a recovered bird can be tested for immunity to laryngo-tracheitis. If the disease has been infectious catarrh, then the bird will succumb to experimental infection by laryngo-tracheitis, but if the disease was laryngo-tracheitis the bird resists further infection. Similarly, if laryngo-tracheitis is suspected, immune bodies in the blood serum can be demonstrated with the aid of chorio-allantoic egg membrane culture technique to prove the suspicion correct. Whilst these laboratory measures are of critical importance, they are beyond the scope of this present work, and no further mention will be made except to point out that in all cases of doubt, birds in the early stages of the disease under investigation should be submitted to a laboratory.

Fowl Pox.

This infectious disease is also known as "roup," chicken pox, "warts," and by various other names.

Occurrence—

It is extremely common throughout the poultry farming areas of New South Wales. Outbreaks are more common and severe in certain localised areas where it may constitute the worst disease affecting the poultry.

Birds may be infected at any time of the year, but serious outbreaks of the disease are seen affecting flocks, only in late summer and in autumn, that is, from February to May. The seasonal incidence varies in different localities. In some centres, the first cases are regularly seen in February, while in others the disease seldom occurs before April. When autumn hatching is carried out (for the table poultry market), the farmer soon recognises the seasonal incidence of the disease amongst chickens. For example, one farmer who is in an area particularly prone to severe outbreaks of the disease, has the following experience year after year. Chick hatched out in February often escape, those hatched out early in March suffer heavily from the disease, but those hatched out in April are very seldom affected.

Birds Susceptible—

Fowls and turkeys are particularly susceptible. Pigeons are highly susceptible to the special strain of pigeon pox virus, but not, under normal field conditions, to the fowl pox virus. Pigeons have been artificially infected with fowl pox, but even this infection is very difficult to bring about. Geese and guinea fowls are stated to be susceptible, but natural outbreaks are rare. Pheasants, parrots, pea-fowls, quail and various wild birds are susceptible. Some authors state that ducks are susceptible, but others have found them refractory to experimental inoculation.

The disease chiefly affects young stock, that is, the last season's hatch, and if the outbreak occurs in March the ages of the birds affected will vary from six to nine months. Autumn-hatched chicks are not usually affected before the third week. Older birds (for example, first- and second-year hens) that have been subjected to an attack previously are resistant to further infection by the same strain of virus. If it should so happen that old birds have never been in contact with infection, they may contract the disease when first exposed.

Cause—

(*a*) *Predisposing Cause.*—The large combed breeds seem to be more liable to the disease. Overcrowding of birds, or the running together of cockerels which fight will assist in rapid spread of an outbreak through a flock. Though there is little precise information on the subject, it is to be anticipated that anything which lowers the general health and condition of the bird (for example, faulty feeding, worm infestation, green feed deficiency, overcrowding, poor ventilation) will render them more susceptible to an attack.

(*b*) *Actual Cause.*—Fowl pox is caused by an ultramicroscopic virus, that is, a micro-organism too small to be seen under the microscope. This virus will pass through special filters which retain bacteria.

Without the causal virus fowl pox does not occur. Though there are several different forms of the disease, they are all caused by the same virus. This can be clearly demonstrated, for if "canker" material is taken from the mouths of the affected birds and rubbed on the wounded comb, typical "warts" will be produced. Similarly, when the crusts are taken from the comb and rubbed on the wounded mucous membrane of the mouth, "canker" is produced.

It has been shown recently that there is more than one fowl pox virus strain in New South Wales*. This is important, as fowls may suffer from the disease caused by one strain of the virus, then become infected later by another strain of the virus, and again show symptoms of the disease. This has happened on a number of farms.

The virus of fowl pox is readily killed by disinfectants, particularly by oxidising agents such as Condy's crystals, or the chlorine group of disinfectants (chlorize, chloride of lime, etc.).

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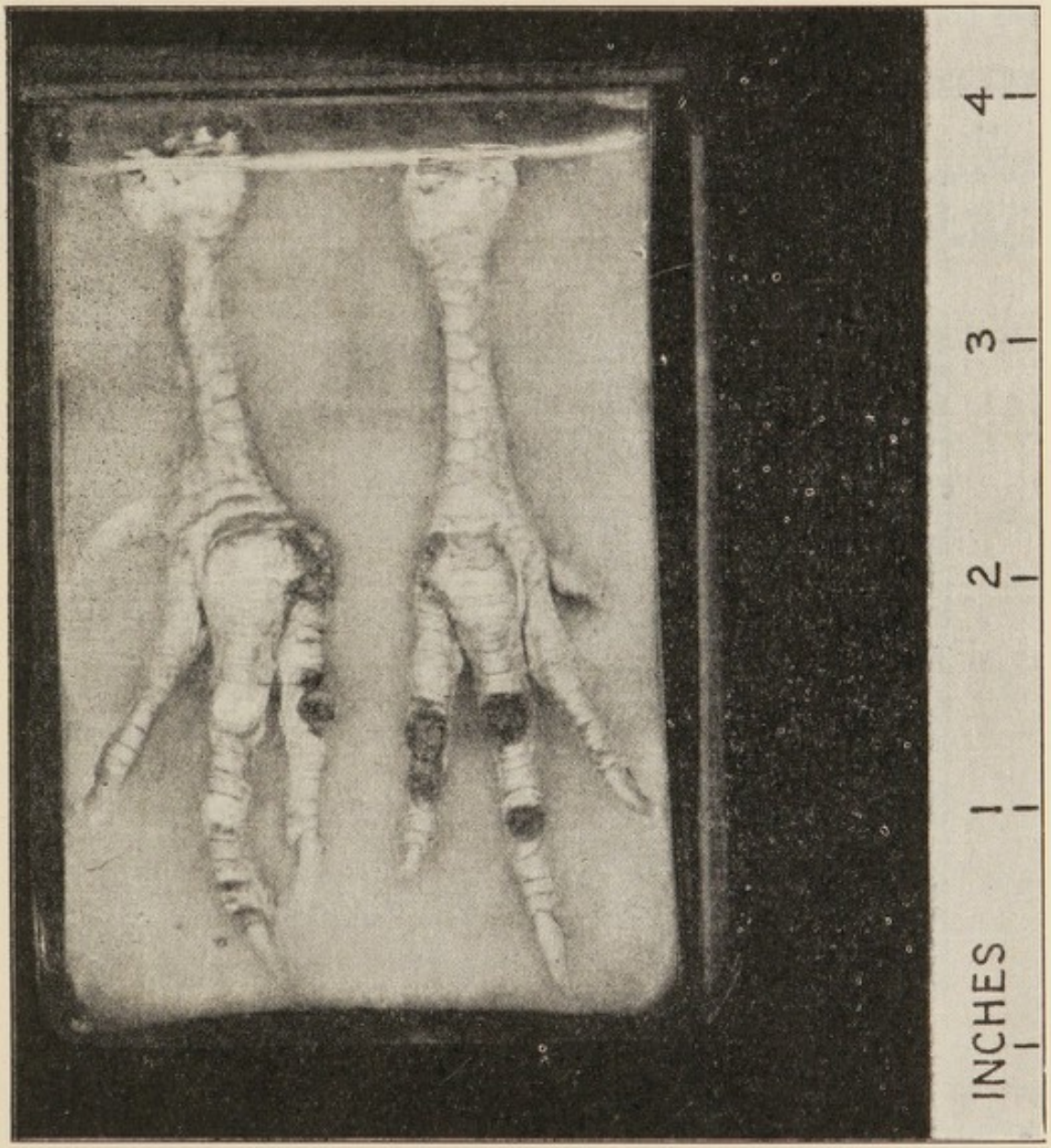
*The critical confirmation of this carried out by L. Hart, B.V.Sc., is as yet unpublished.

Mode of Infection—

It is popularly believed by poultry farmers that the infection is spread by mosquitoes only, and that without these insects there would be no spread of the infection through the flock. This view is incorrect. Probably the chief way in which the disease is spread from bird to bird is by mosquitoes, but direct contact will also enable the disease to spread rapidly. Birds are continually fighting and pecking at one another, and by this means they wound and infect the skin and mucous membranes of the head.



Fowl Pox Lesions on the Feet of a Chicken.

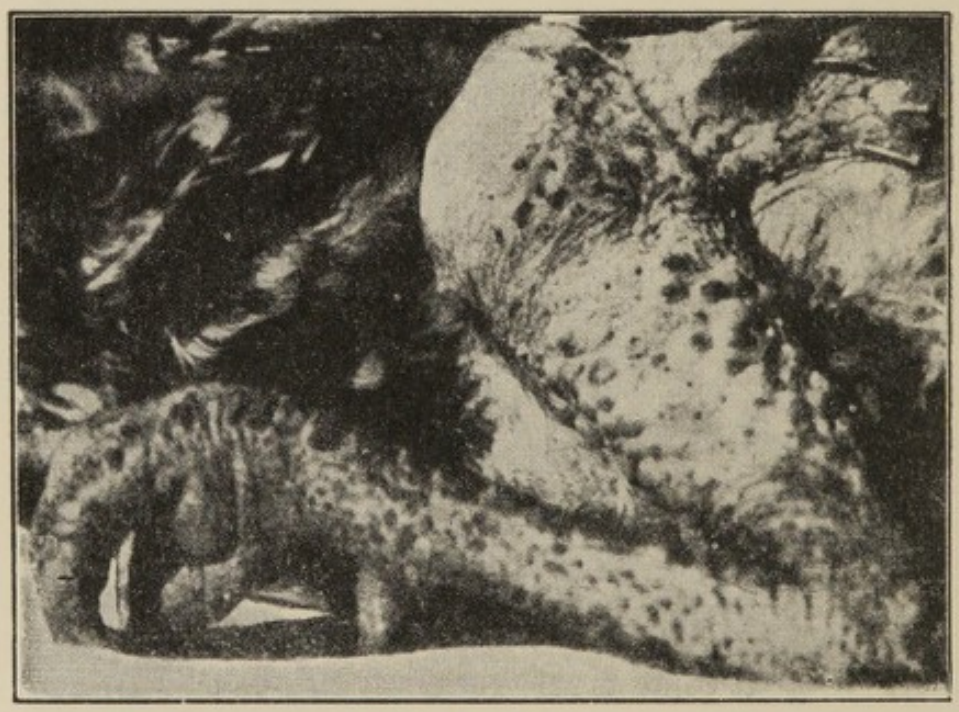
A bird, the lining membrane of whose mouth has been wounded by sharp grit, will, upon drinking infected water, readily develop infection. It has been shown that when an infected bird and a non-infected bird are placed in separate cages in an insect-free room, with the cages 3 feet apart, no transmission of the disease takes place. When the cages

are placed together, but with a very fine-mesh gauze between to prevent any possibility of pecking or direct contact, no spread of the disease takes place. When, however, the cages are placed together with only a large mesh wire netting between, then the healthy fowl contracts the disease.

Further, it has been found with pigeons that when water infected with pigeon pox was given to healthy birds kept separately and fed no shell grit, the disease did not develop. Other birds supplied with sharp shell grit and infected water developed the disease. It will thus be obvious that once fowl pox breaks out it readily spreads right through the flock by direct contact, as well as by insect transmission.

The Role of Insects—

Mosquitoes have been shown to spread the disease. Authorities still differ as to whether they act as mechanical carriers only, or whether the virus lives and grows in the body of the mosquito. The living virus has been found in the body of mosquitoes, and this virus has been used



Cutaneous Form, showing "Pox" Lesions on Fowl's Leg.

[After Kanra and Injer.

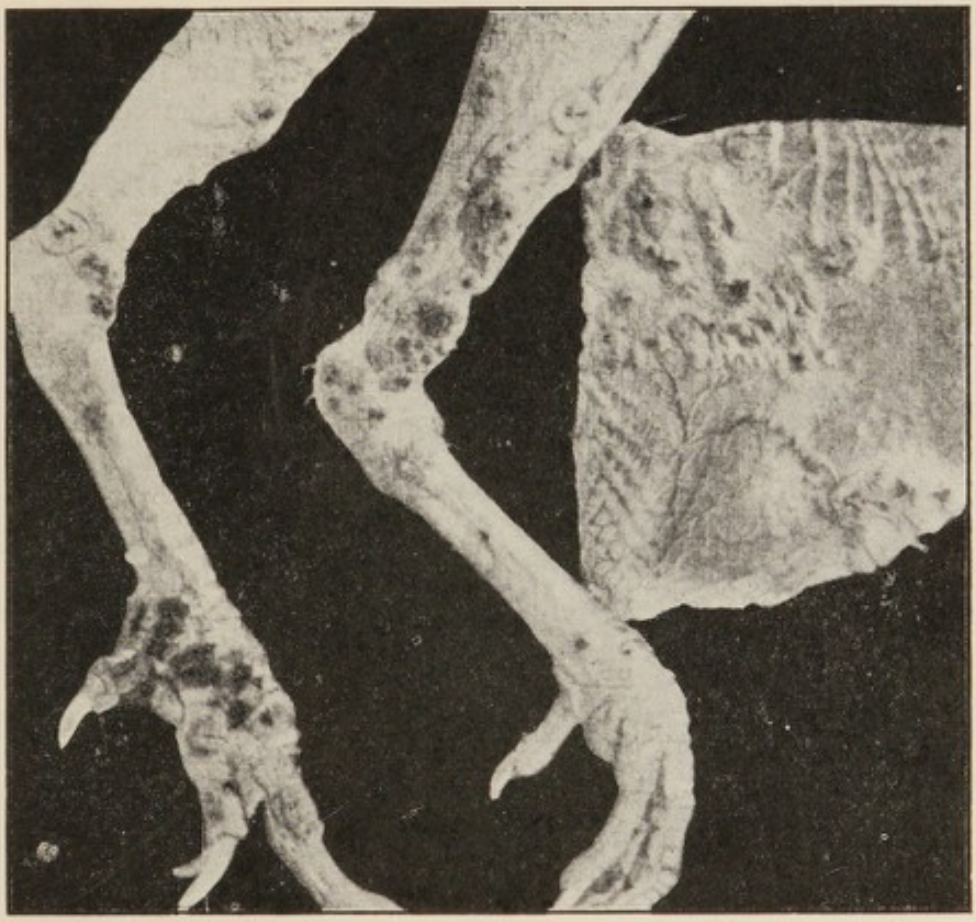
to infect other birds. Further, mosquitoes have been found to transmit the disease seventy-five days after last feeding on an infected bird. It is therefore possible that a mosquito may remain infected right through its hibernation period. A mosquito that has fed on an infected bird may feed on other animals, then come back and infect a healthy bird. Apart from mosquitoes, it is probable that any blood sucking parasite may transmit the disease. Fowl tick and biting lice have been found to do so, as also have biting flies.

Immunity—

Once a bird has been affected with the disease, that bird has an immunity against further infections from *that strain* of virus. As mentioned above, there are at least two strains of the virus in this State, and therefore (unless vaccinated) birds may suffer from two attacks of the disease.

Incubation Period—

Following the introduction of the virus into some slight abrasion of the skin or mucous membrane, or per medium of a mosquito bite, local symptoms develop in a period of from three to fourteen days, usually being noticeable on the sixth to eighth day. The potency of the virus, the amount introduced, and the individual resistance of the bird, all modify the length of the incubation period.



Fowl Pox Lesions on Legs and Skin.

[Eng Ministry Agric. photo.

Symptoms—

Quite a variety of clinical symptoms are seen in fowl pox, and it is usual to group these into three different forms.

(1) *Cutaneous Form.*—At the places where the virus is introduced a tiny blister (vesicle) develops. Large numbers of such blisters (about one-sixteenth of an inch across) may be seen by a careful observer on the comb, wattles and unfeathered portions of the body, but usually this stage passes quite unnoticed. These blisters rapidly become small

fleshy growths covered by a crust of yellowish material, which quickly increases in size. This "wart" or "pox" is the well-known symptom and is usually the first stage to be observed. The yellow crusts become darker in colour and may be almost black before they fall off. These "warts" often tend to coalesce, forming yellowish or black scabs covering the comb and wattles. The bird shows a general systemic upset for several days, but this is not as marked as in the other forms of the disease. In severe cases these typical warts may appear on all the unfeathered portions of the body, and in very severe cases on the feathered portions as well.

The Comb Form of Fowl Pox
The most common yet least harmful type of infection.
[*Eng. Ministry Agric. photo.*

(2) *Canker or Diphtheritic Form.*—In the early stages the bird is dull, mopey and huddles in a disconsolate heap on the perch, with no appetite for food. A cough may then develop. On examination of the mouth, yellowish cheesy material (canker) will be found on the inflamed mucous membrane of the tongue, palate, mouth or throat. This yellowish material may also be present on the opening of the windpipe (larynx), causing gasping and gaping, and when the closure of the windpipe is complete, suffocation will result. Should these cheesy masses be removed forcibly they leave a raw, bleeding, inflamed surface.

The development of this canker material is due to the entry of the virus into the mucous membrane. When the virus begins to grow in the mucous membrane it causes necrosis and liquefaction of cells. Various other bacteria then play a prominent part in causing further inflammation of the already damaged tissues. It has been found that when birds are suffering from green feed deficiency, there is a marked tendency for this diphtheritic form of the disease to develop, due apparently to the lowered resistance of the mucous membranes. It must be remembered, of course, that quite apart from fowl pox infection, similar cheesy masses will develop on the mucous membranes in green feed deficiency disease.

In a fowl affected with the diphtheritic form of fowl pox, absorption of the toxic products from the lesions causes severe systemic upset as shown by diarrhoea, emaciation, and in severe cases this is followed by death. A foetid sickly smell is characteristic of this form of the disease.

(3) *Oculo-Nasal Form.*—The fowl is dull, then shows a watery discharge from the eyes and nose, and sneezes frequently. The tissues surrounding the eye and eyelids become inflamed, swollen, and may be glued together by a tenacious exudate. If both eyes are affected in this way, the bird will rapidly starve to death. The nasal discharge thickens, becoming mucoid and muco-purulent. There may be a swelling of one or both sides of the face due to inflammatory material collecting in the infraorbital sinus. This material in the sinus rapidly becomes cheesy in consistency. Frequently cheesy (cankerous) material develops on the eyelids, on the tissues of the eye, and over the cleft of the palate. The latter being closed, will lead to mouth-breathing. The eye lesions may be seen without the nasal lesions and vice versa.

The Canker Form of Fowl Pox.
[Eng. Ministry Agric. photo.

Birds may be found showing varying combinations of the above forms, so that the disease affecting a particular bird may not fit into any of the above descriptions, but may show features characteristic of all of them.

Symptoms in Young Chickens—

In young chickens the disease is usually observed first in the form of a fleshy wart developing at the base of the chick's comb. More growths of a similar nature develop, covering the face and eyes. In severe outbreaks, in autumn-hatched chicks, the disease may become generalised, in which case the typical warty growths may appear all over the body and thighs. In such outbreaks the mortality rate may be very high (over 50 per cent.).

Economic Importance—

Some diseases (*e.g.*, Newcastle disease) may wipe out an entire flock. Such is not the case with fowl pox. Other diseases (*e.g.*, the peracute form of laryngo-tracheitis) may cause considerable mortality (20 per cent. or more). Such losses do not usually occur as the result of a fowl pox infection, except in autumn-hatched chicks, where mortality may be severe.

Though the death rate is slight, heavy monetary loss often occurs, the cause of which is threefold:—

(1) Pullets coming on to lay may be thrown into a heavy moult and put off the lay for two months. This disastrous check to laying usually occurs when egg prices are highest, and also at a time when the farmer can least afford it, as much money has been expended on brooding and rearing just prior to this period.

Fowl Pox.

Eruption of the skin and mucous membrane of the mouth.

[*After Hutyra and Marek.*

(2) A very severe economic loss may be sustained due to the birds being checked in development. Birds badly affected often fail to put on weight for many weeks. Some of the most severely affected may never develop into well-shaped, robust birds. This check to development renders the pullets less resistant to various other diseases and parasitic infestation. It is not uncommon for such pullets to suffer from an acute outbreak of infectious catarrh or to become heavily worm infested. While the fowl pox is not directly responsible for these conditions and for the further loss sustained, it is indirectly responsible. In any case, the check to development necessitates a heavy culling of the pullets.

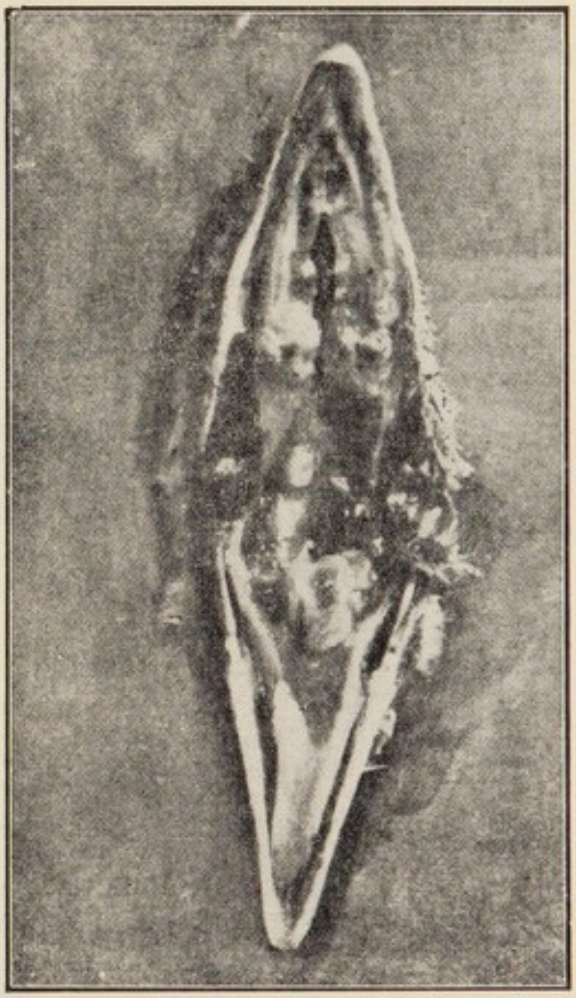
(3) Some deaths do occur, particularly when birds are affected with the mouth and eye forms of the disease. When the eyes are severely affected the bird is blind and may starve to death. In some very severe outbreaks in autumn-hatched young stock, more than 50 per cent. of the birds have died. This is exceptional, and with stock hatched in season (June to September) no deaths occur in birds affected with only the comb form of the disease, and seldom more than 5 per cent. in those affected with the mouth or eye form.

Control by Vaccination—

Vaccination is the only effective method of preventing outbreaks of fowl pox. Speaking in general terms, vaccination comprises taking the virus that is responsible for the disease and introducing it into the skin, usually of the bird's thigh. It was found that the virus which causes pox symptoms in pigeons did not produce the typical disease in fowls. In England use has been made of this fact and vaccination with pigeon pox virus has been carried out for many years. Pigeons are used to produce material with which a suitable glycerine suspension is made ready for vaccination. The advantage of vaccinating with pigeon pox is that neither serious checking of the birds nor cases of generalization of the disease occur as a result of the vaccination. The disadvantage is that the immunity to fowl pox, developed as a result of vaccination with pigeon pox, is not very solid. If, following pigeon pox vaccination, a fowl is heavily infected artificially with fowl pox, most workers record that quite a serious attack of the disease may result.

Efficacy of Pigeon Pox Vaccination—

Usually, following vaccination with pigeon pox, the fowl has sufficient resistance to withstand a natural infection with the disease for some months. However, this varies, and quite a large number of authors point out that following successful pigeon pox vaccination (*i.e.*, where a "take" results), the birds may still contract fowl pox naturally, but that the disease runs a much milder course than usual. It is claimed by the advocates of pigeon pox vaccination that it gives a temporary resistance to natural infection for about six months. During this period, in localities where fowl pox is prevalent, the birds will be infected with the naturally occurring disease without showing symptoms. When this does occur the partial immunity already conferred by vaccination will be greatly increased.

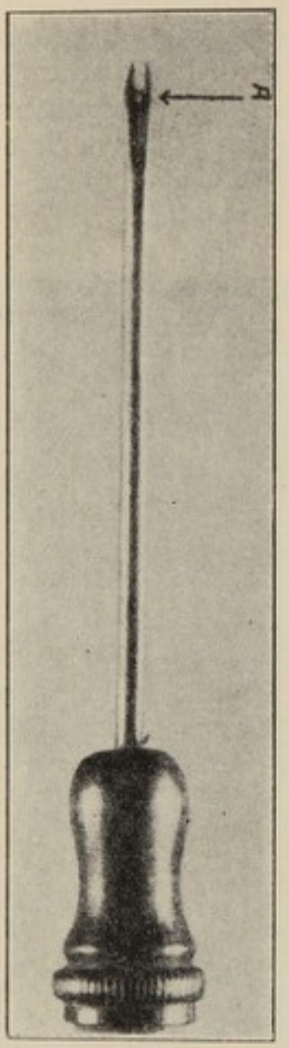


Another Illustration of the Canker Form of Fowl Pox.
[After Kanra and Injer.

Though this type of vaccination (pigeon pox) is regularly used in Great Britain, it is not carried out to any extent in America. There, vaccination with the living and fully potent fowl pox virus has been adopted, and the "stab" method of vaccination is almost exclusively used.

Fowl Pox Vaccine—

With the live fowl pox virus it is sometimes found that birds may be seriously checked in development, and their resistance to other diseases lowered. The advocates of this vaccine (fowl pox) claim that in spite of the possibility of checking and other harmful effects, fowl pox vaccination is much better in practice than pigeon pox vaccination. They claim that the loss sometimes sustained due to birds being checked by the fowl pox vaccine is not so great as when a bird is vaccinated with pigeon pox, develops only a mild resistance, and a little later becomes affected by a natural attack of the disease. Furthermore, if pullets are checked in December many poultry farmers claim that this is desirable, as it stops the pullets coming on to lay prematurely. This results in the production of a greater number of eggs during the period of high egg prices.



Needle enlarged) used for Vaccinating Day-old Chicks.

[After Lubbehusen, Beach and Busic.

In New South Wales the matter was taken up by the Department of Agriculture, and a weakened fowl pox vaccine was produced at the Glenfield Research Station by storing the virus for some months in its dry form. This vaccine was, therefore, "stronger" than pigeon pox vaccine, but weaker than the American fowl pox vaccine. It had the advantage of producing a solid immunity to the disease, so that birds which once reacted to the vaccination would not subsequently contract the disease. At the same time, this weakened vaccine was less likely to produce the harmful results, sometimes recorded after the use of fully potent virus. The method of producing the virus vaccine was less likely to produce the harmful results, sometimes recorded after the use of fully potent virus. The method of producing the virus vaccine involved standardising it to a given strength, the strength of each particular batch of vaccine being tested out on experimental birds before issue for general flock use.

Commercial vaccines now sold in this State are (or should be) made according to this standard method, and should, therefore, conform to a standard potency. Batches of vaccine which are made up with vaccine not sufficiently weakened by storage, or put up insufficiently diluted may induce a very severe reaction. Vaccines that are put up in too strong a dilution or made from virus that has been weakened too much will fail to produce a local or systemic reaction in vaccinated birds. Such birds will still be susceptible to a natural attack of the disease.

Method of Vaccination—

The original method recommended when the weakened fowl pox vaccine was first experimented with in this country was the intrafollicular method, and recommendations were as follows:—

"Only birds in perfect health should be vaccinated; pullets suffering from colds, infectious catarrh, worm infestation or green feed deficiency disease should not be treated. External parasites (lice, red mite, etc.) should also be eliminated before vaccination. Vaccination should be carried out at least a month before there is danger of a natural outbreak of the disease. Only birds between the ages of twelve and sixteen weeks should be vaccinated. Such birds are usually crated for convenience. The vaccine is obtained in the dry form, 1 milligram being ground up in a small pestle and mortar with 1 c.c. of sterile normal saline, and any visible particles strained off through sterile cotton wool. The amount of dry vaccine taken will vary with its potency. One milligram per c.c. is an average quantity. The exact amount will be decided by the laboratory making the vaccine, after the potency has been tested."

Vaccine is sometimes supplied mixed ready for vaccination in fluid form. In this case it is important that the vaccine be freshly prepared, as the potency of fluid preparations tend to deteriorate rapidly.

The Intrafollicular Method.—The bird is held by an attendant. The operator plucks out from two (where the feathers are large) to four (if small) feathers from the outer part of the left thigh. A small tightly wound cotton wool swab is then dipped in the vaccine and rubbed on the follicles from where the feathers have been removed. The swab is made by winding a small piece of cotton wool on a thin wooden stick (a little thicker than a match). These swabs, together with all other equipment, are sterilised by heat, and only those needed are opened for use on the farm. Proportional amounts of sterile saline and virus are supplied in separate containers ready for mixing. When mixed and filtered, small quantities at a time are placed in a small conical flask so that when this becomes foul it may be thrown out (into a bucket of disinfectant).

Great care should be taken not to spill drops of virus, or accidentally to infect the birds with virus around the head, as in this case the actual disease may be produced.

After completing vaccination, all spare virus, swabs, flasks, mortar, etc., should be disinfected to destroy the virus.

Many poultry farmers who have had their flocks vaccinated each year since the operation was first introduced, still have this method carried out with entire satisfaction. Its main disadvantage would seem to be that it is a little wasteful of vaccine, and with equally deft operators it is perhaps a trifle slower than the "stab" method.

The "Stab" Method.—This method is the most popular in America, and it is also the method used by most licensed vaccinators in this State. It comprises introducing the virus into the skin by means of dipping some sharp-pointed instrument in the virus and then stabbing it through the skin. Various devices are used. Those that ensure an even amount of tissue damage (even depth of penetration) and an even dose of virus are suitable. A small pointed blade with cotton wool firmly wound round so as to prevent penetration of the blade into the deeper

tissues of the leg is often used, and in most cases two light stabs are given. This method cannot be recommended, as, with uneven stabbing, different amounts of tissue damage are inflicted, and different doses of virus are squeezed out from the cotton wool.

Fowl Pox.
Thick caseous deposits in the angle of the mouth.
[*After Hutyra and Marek.*

Probably the best instrument is the two-pointed needle used and recommended in America (see illustration). The instrument has two sharp points one-twenty-fifth of an inch apart. A drop of vaccine adheres between these points when they are dipped into it. On stabbing the bird's thigh this drop of vaccine is introduced evenly into both punctures. With a needle it is an easy matter to regulate the depth of penetration. At present the vaccination "outfits" as supplied commercially comprise the dried virus put up in a sealed capsule, and also a small bottle of glycerine saline. If the instructions issued with these outfits are followed carefully, the procedure roughly complies with that outlined above and is satisfactory in practice.

Whatever method of vaccination is used, the one constant site (most conveniently on the lateral aspect of the left thigh for a right-handed operator) should be chosen for the operation, so that reactions may be checked up more easily.

Reactions to Vaccination—

When a vaccination "takes," the reaction is twofold: (a)local; (b) systemic.

Local Reaction.—If birds are examined on the seventh day, a small swelling about the size of a large pinhead will be found at the site of inoculation. This local reaction may occur as early as the fifth day, or may be delayed till the eighth or ninth day, depending largely on the potency of the virus, the amount delivered into the skin, and the amount of tissue damage. Soon after the swelling appears it becomes encrusted with a yellowish scab. This scab is a typical fowl pox "wart" as seen on other portions of the body when a natural outbreak occurs. The scab remains in place for some time, usually about three to four weeks. It must be realised that this scab contains the infective virus. It will, therefore, be seen that when reacting pullets are run with those that are not

inoculated there is a small added possibility of infection being transferred to them. The longer a fowl pox scab remains, the less infective it becomes.

Examination for local reactions to check the effectiveness of vaccination should be carried out on the twelfth to fourteenth day. Where the local reaction is severe a slight lameness may be in evidence.

Systemic Reaction.—This typically occurs about the sixteenth to twenty-first day, though it has been observed as early as the eighth day, and in severe cases, where the vaccination is unsatisfactory, the general systemic disturbance may continue on for a month or more, the bird then usually dying or being killed.

Where the reaction is satisfactory, the birds appear a little dull and mopey on the seventeenth or eighteenth day, and eat only a small quantity of food for two or three feeds. Within twenty-four hours of showing symptoms they start to brighten, and within forty-eight hours are back to normal again. Without this reaction it is probable that the birds will not have a solid enough immunity to withstand all natural infection.

Fowl Pox Lesions, showing Skin and Mucous Membrane Involvement.

[Eng. Ministry Agri. photo.

Factors Influencing Severity of Systemic Reaction—

(1) *Condition of the Bird.*—This is by far the most important. If any disease condition is present it will probably "light up" after vaccination and cause severe loss. More will be said of this later.

(2) *Age.*—If birds over sixteen weeks' old are vaccinated, a severe systemic shock may be sustained. Further, if late in the season, there is danger of a natural infection occurring before they have developed an immunity. At this age a systemic shock checks egg-laying.

When the original experiments were carried out, it was found that from twelve to sixteen weeks appeared to be the ideal age at which to carry out vaccinations. American investigators have found recently

that birds, even quite healthy ones, vaccinated when over thirteen weeks of age usually suffer a mild check, which, though scarcely appreciable, does exist. These particular investigators found that in America the best age at which to vaccinate healthy birds with fowl pox vaccine was when they are from thirty to ninety days' old. A large scale field experiment locally has indicated that ten to twelve weeks is the best age at which to vaccinate. When vaccinations are carried out earlier, there is danger of an outbreak of such diseases as blackheads or coccidiosis occurring at the time of the systemic reactions to the vaccination.

(3) *Severity of Local Reaction.*—Where there has been a very severe local reaction, the systemic reaction occurs sooner than usual and is usually severe.

Dangers of Vaccination—

Vaccination to prevent outbreaks of fowl pox is now a well established custom. In perhaps five out of every hundred farms where vaccination has been undertaken, serious trouble may follow. The others, perhaps, vaccinated by the same operator and with the same batch of vaccine, experience no harmful effects. There are districts in which numbers of farmers who vaccinate year after year record no trouble whatsoever, and moreover avoid the heavy economic toll that fowl pox would otherwise have taken.

Fowl Pox.
Nodular formation on toes of a pigeon.
[After Hutyra and Marek and Manninger.

In several cases, following vaccination, a farmer has had serious trouble with his poultry due to the occurrence of catarrhal diseases and has discontinued the practice after two or three years. The next year he has still had a recurrence of the catarrhal disease trouble, e.g., colds or infectious catarrh, in an even worse form in the stock that were left unvaccinated. In such cases, fowl pox vaccination has been unfairly blamed as a cause of the trouble. Other farmers who have experienced trouble have vaccinated some pullets and left others unvaccinated. Following vaccination, the ones treated have been checked, and never developed nor looked as well as the ones left unvaccinated. In this case, fowl pox vaccination is undesirable, even though it has given immunity against the disease. In other cases farmers who have trouble record that the birds are checked at the time of the reaction and never seem to make up this check, though no other birds were left unvaccinated to see if some other factor were operating.

Now all this sounds very confusing, but actually it is what one would expect. For example, a careful check on the results of vaccination was made in one district. Most farmers spoke in glowing terms of the vaccination, pointing out that there had been no checking of the birds following vaccination and there had been no fowl pox. In times before vaccination was carried out, eggs were lost for a period of six weeks to two months from over half the pullets, these being affected with fowl pox. These eggs were lost at the time of peak prices (fowl pox occurring late in the district referred to). Two of the farmers in the district recorded disaster as a result of vaccination, and in these cases vaccination was done by the same operator, who did most other farms in the district. One lost over 1,000 pullets and also the egg production for about two months from the surviving pullets, the estimated direct financial loss being about £1,000. On careful investigation it was found that some of the birds had been coughing at the time of vaccination. A transmission experiment showed the diseased birds to be affected with infectious laryngo-tracheitis. Apparently this disease was present in a mild form, and when the birds' health and disease resistance was lowered during the systemic reaction to fowl pox vaccination the mild form of laryngo-tracheitis that had been present "flared up" in the acute form and caused serious mortality. The harmful results, though due to laryngo-tracheitis, were indirectly due to fowl pox vaccination.

On another farm about 1,700 pullets were vaccinated and at least half of these were very seriously checked. They went steadily downhill for four weeks, and then a mild outbreak of laryngo-tracheitis occurred amongst them. Over 100 birds were lost. Some 200 left unvaccinated were much brighter in appearance. All the birds on this farm were suffering from a heavy worm infestation. A number that had been most seriously checked were killed and post-mortem examinations made. In these, the small intestine was filled with worms, over 100 mature round worms (Ascaridia) being present in some cases. It will thus be seen that in such cases vaccination may be an extremely dangerous procedure. This should be apparent, as even where the vaccination is successful the birds' health will be upset somewhere about the seventeenth day. Any other disease condition affecting the birds at that time, however mild it may be, will probably become very serious during this period. Particular care should be taken to note whether the flock is suffering from even a mild form of cold (coryza), laryngo-tracheitis, catarrh, greenfeed deficiency, or worm infestation (round worm or tape worm.)

Risk versus Gains—

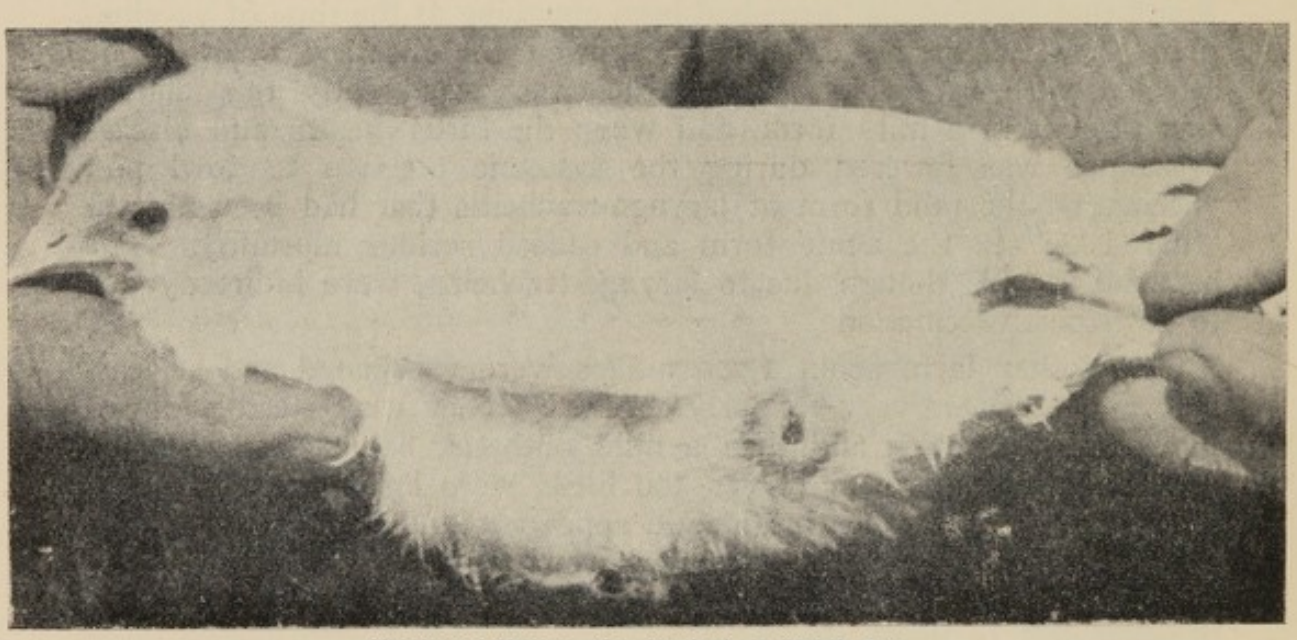
Even where all care is taken there is still an element of risk, as the birds may be in the incubation stage of one of these diseases at the time of vaccination, or may contract such a disease between the times of vaccination and reaction for fowl pox. This risk must be weighed, and where other catarrhal diseases are much more prevalent than fowl pox it may be a bigger risk to vaccinate for fowl pox than to refrain from doing so.

Apart from other diseased conditions becoming super-imposed during the time of reaction, vaccination with a vaccine as originally prepared is a success, consistently saves large economic loss to the industry and **is definitely recommended.**

Vaccinating Part of a Flock—

As the birds are vaccinated with living virus there is a slight possibility—and it has occurred on rare occasions—that when one yard is vaccinated, mosquitoes may carry the infection from a vaccinated bird, infect the head of an unvaccinated bird, and thereby set up a case of the disease.

Another possibility is that mosquitoes may pick up the weakened virus from the site of the local reaction on vaccinated birds, later biting unvaccinated birds and so infecting them with weakened virus.



Vaccinated Chicken, showing Scab on Eighth Day.

[After De Volt, Malthorp and Davies.

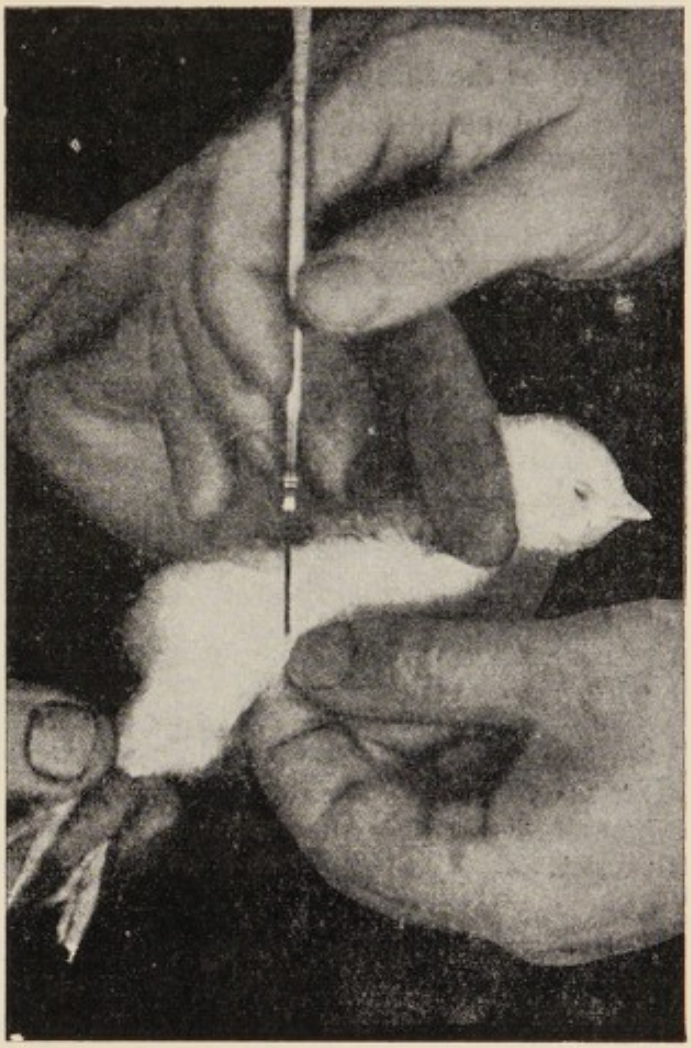
Perhaps this does occur, for it is a common observation that the vaccination of a proportion of the pullets in flocks which normally suffer heavily from the disease confers a protection (more or less inadequate) on the remainder. This feature is utilised by some poultry farmers who have a hundred or so pullets vaccinated out of each batch, and hope for the mosquitoes to further the vaccination. Such a procedure is not recommended as it is far too risky to be seriously considered.

Precise scientific work has not as yet been carried out to confirm the above field observations. It may yet be shown that some other factor is operating which explains why in certain cases the vaccination of a proportion of the flock appears to safeguard the whole flock to some degree.

Chicken Vaccination—

Large-scale experiments have been undertaken in various parts of the world to test the value of this procedure. The advantages of immunising birds as day-old chicks are apparent, but unfortunately these are more than offset by the serious checking in development which results.

Vaccination at this stage is much more likely to produce harmful results than when the birds are twelve weeks old. In one experiment carried out in America with 36,000 chickens it was found that there was considerable danger from two standpoints. Many chicks contracted a generalised form of the disease as a result of vaccination, and a considerable proportion of those that did not develop the disease were seriously checked in development. In such chickens general disease resistance was greatly lowered, and they were, therefore, likely to succumb to any infectious disease with which they came in contact. It will thus be seen that it is not advisable to attempt vaccination of young chickens hatched in season (July to September).



Method of Vaccinating Day-old Chicks.

With autumn-hatched chicks, the position is different. In some localities, autumn-hatched chicks cannot be reared owing to fowl pox. It may be found that this has a strictly seasonal incidence, and that in a certain locality February - March hatched chicks contract the disease most severely. In such cases, vaccination, though a dangerous procedure, may be justified, in that the ill effects (if any) are certainly not as bad as an attack of the disease. Those farmers who hatch chickens for the table poultry trade, and who have adopted this method of vaccination have, in some instances, found it a profitable undertaking.

To Vaccinate Day-old Chicks—

The "stab" method is mainly employed. The site chosen varies somewhat, but the loose fold of skin connecting the front of the thigh to the body is usually found to be the most suitable. Some operators vaccinate on the lateral aspect of the thigh, but there is a danger of introducing the virus into the deeper structures (muscles and blood vessels), and this may aid in generalisation following the vaccination. The most suitable instrument for vaccination is that recommended for the "stab" method in older fowls, *i.e.*, a two-pointed needle with the points one-twenty-fifth of an inch apart, so that a standard quantity of fowl pox vaccine is held between the two points. In America the

same strength vaccine is used for day-old chicks as for older birds. No precise experimental work has been done here to demonstrate the ideal strength.

If it were possible to do so, the chickens should be protected from mosquitoes, which so readily infect them with the disease. This, of course, is impracticable. It will readily be seen that if a chick is vaccinated and then becomes infected naturally the same day, the vaccination will merely comprise an added dose of the infective agent and will but add to the severity of the disease symptoms in that chicken.

Control of the Disease—

It will be seen that the chief means of control is by vaccination correctly carried out.

Fowl Pox.
Swelling of the infra-orbital sinus.
[*After Hutyra, Mareh and Manninger.*

In New South Wales, for over eight years vaccination has been employed on a large scale as a control measure against the disease, and each season the practice seems to increase in popularity. A few individuals have had bad results and discarded the procedure, but after a season or two some of these have returned to vaccination. In most cases bad results are due to the presence of other disease conditions, which light up in a disastrous manner as soon as the birds are checked by the vaccination.

In some localities where catarrhal diseases occur every year in nearly all flocks, vaccination, as carried out in the past, is definitely a dangerous procedure. In such cases competent veterinary advice should be sought as to the correct time to vaccinate, the method of vaccination, the strength of vaccine to be used, and other procedures to be carried out. Without this advice, and where mere routine vaccination is practised, trouble is to be anticipated.

Whilst vaccination is the principal item considered by most poultry farmers, the natural factors previously mentioned should not be overlooked. Thus, where birds are overcrowded in filthy, dusty conditions, coryza will probably develop, the health of the flock will be lowered, and when fowl pox does occur its harmful effects will be much greater than usual. Such conditions should, therefore, be avoided.

Green-feed deficiency, worm infestation, faulty feeding, and bad management will all upset the health and disease resistance of the birds. Such birds will be more susceptible to any disease (including fowl pox) with which they come in contact. Care should, therefore, be taken to eliminate such factors.

The birds' ration should be adjusted, and if green feed is scarce, 1 per cent. of a reliable cod liver oil should be fed in the mash (freshly mixed). The poultry houses should be kept clean and free from vermin.

Having regard to the part played by mosquitoes in the spread of the disease, it is advisable for poultry farmers to co-operate in eliminating wet or marshy areas which will allow mosquitoes to breed. Water surfaces should be covered by a thin film of kerosene to kill the developing mosquitoes. Some owners who have a few valuable birds go to the trouble of spraying the houses with mosquito repellent preparations, or burning dry cow dung just outside the house during the fowl pox season. If pieces of dry cow dung are placed in a row and one end lighted, they will smoulder away all night, producing an aromatic smoke highly repellent to mosquitoes. This simple expedient is well-known and used by bushmen, but is hopelessly impracticable on a commercial poultry farm.

Fowl Pox affecting a Turkey Poult.
The eyes are closed with the pox lesions.
[After Bice.

"Medicinal Tonics"—

Hitherto much reliance has been placed on the use of various medicines, such as epsom salts, sulphur, and various tonic preparations to prevent an occurrence of the disease. As yet, controlled experiments have failed to demonstrate the value of such practices. It must be realised that so long as a fowl is in perfect health there is no need for "medicinal tonics," and some preparations thus designated may even be harmful.

Finally, as fowl pox is an infectious disease, any plan of control must include separation of diseased from healthy birds, and thorough cleansing of houses, yards, food and water vessels that have been in contact with diseased fowls.

Home Remedies and Treatment—

With this, as with most other diseases, there is a strong desire on the part of the farmer to treat the symptoms shown by the birds. Treatment of these external symptoms does very little good as the disease is systemic.

Where canker material is present on the entrance to the windpipe, suffocation may result if the material is not removed. The obstruction may be removed by a piece of looped wire or a small piece of soft flat wood, care being taken to prevent the material falling down the windpipe. The raw area may then be lightly touched with friar's balsam, or a weak solution of bluestone (¼oz. to 1 pint of water), tincture of iodine, or a weak solution (1 per cent.) of silver nitrate.

The pox lesions on the head are often treated by swabbing with tincture of iodine or some astringent (e.g., ordinary washing blue). A saturated alcholic solution or picric acid is quite suitable. These treatments hasten the drying up and shedding of the scabs. The eyes may be treated by instilling a drop of 2 per cent. zinc sulphate or 10 per cent. Argyrol several times a day. This will assist in reducing inflammation and prevent glueing together of the eyelids. In the nasal form volatile preparations such as eucalyptus or small quantities of turpentine and camphor may be mixed with liquid paraffin and smeared around the nostrils. All these local treatments are merely palliative, seldom merit the trouble taken to apply them, and, in most cases, the good done does not counterbalance the harm done by catching and handling the bird. It must be emphasised that the above suggestions are treatments of the outward symptoms only. The actual disease condition runs its course practically unaltered by treatment, however thoroughly this may be carried out.

Green Feed Deficiency Disease.

This disease is also known as *Hypovitaminosis A, Avitaminosis A,* nutritional "roup" and is also referred to as roup of varying kinds (eye roup, canker roup, etc.) The term "roup" is badly used in connection with this disease, as by the term one usually implies an infectious disease, whereas this condition is not contagious or infectious.

Importance of Green Feed—

It has long been recognised that green feed is a necessary portion of the ration, and that the provision of an abundance of first-class succulent green feed has a most beneficial effect on the general appearance of the birds, and on their egg production.

In recent years more precise observations have been carried out, and it has been shown that the question of an adequate supply of suitable green feed is one of greatest economic importance, for the following main reasons:—

Green feed supplies Vitamin A* to the bird, and this vitamin is essential to life. (Actually the green feed supplies Provitamin A which carotenoid material is converted to Vitamin A during assimilation). Practically speaking, little, if any, of the vitamin is present in pollard and bran. As will be seen later, a certain amount is supplied by the feeding of dried lucerne dust or meal, and yellow corn.

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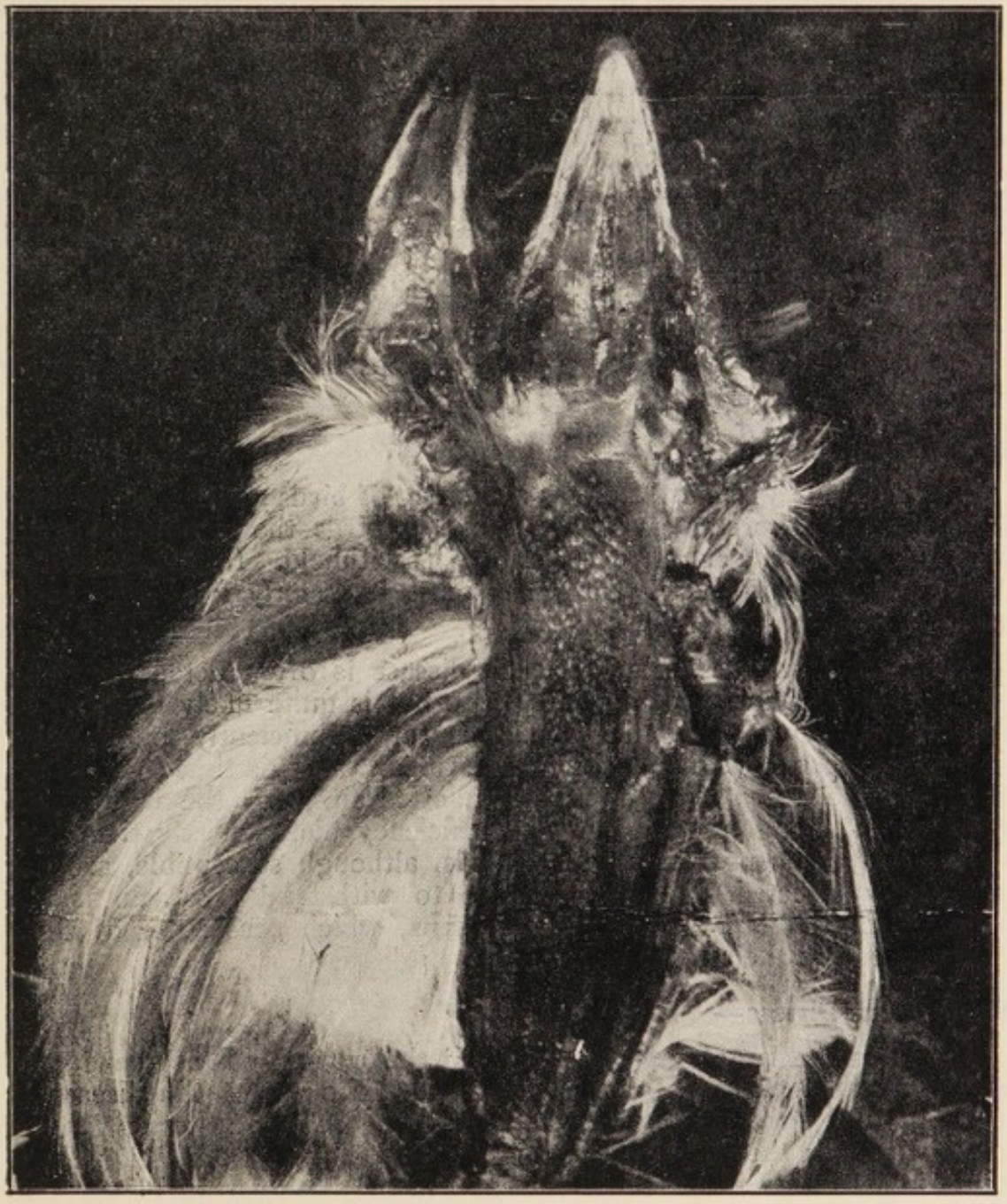
*Recently a new vitamin, A2, has been described; no reference is made to this. For further facts, see Scorgie, N. J., *Veterinary Record* 50: 775 (1938), also Morton, R. A., *Veterinary Record* 50: 1169 (1938).

Relatively large amounts of Vitamin A are present in the egg where this vitamin is supplied in sufficient amounts to the hens by feeding greenstuff, cod liver oil, or some other food containing it.

When insufficient Vitamin A is fed in the ration, the egg-laying is decreased, and the Vitamin A content of the egg is markedly lowered. Further, in the case of breeding stock, the percentage hatchability



Mouth and Throat of Fowl Opened Up to show Pustules in Wall of Gullet.

of the eggs is decreased. Where the deficiency continues, the birds become dejected, unthrifty, and, in the case of young stock, their development is poor. Finally, where the deficiency is not relieved, a definite train of disease symptoms sets in which may culminate in death.

Vitamins are essential to life. They are complex substances, and the most important fact known about them is the serious effect of their absence from the ration. Vitamin A serves to promote growth and prevent this deficiency disease.

While most poultry-farmers grow crops to provide a suitable suppiy of green feed all the year round, it frequently happens that, at times, through drought or lack of water supply, or through the ravages of plant pests and diseases, no green feed is available. It is a common belief of poultry-farmers that green feed is an additional foodstuff producing better results, and that its temporary absence can be suffered without detriment to health; it is not realised that it contains a substance rarely supplied by any other foodstuff. Many poultry-farmers include cod liver oil in the diet, but without realising that it can act as a substitute for greenstuff. When neither cod liver oil nor greenstuff is available for a period of a few weeks, the complaint under consideration is liable to result.

The condition occurs most frequently during summer months, through failure of crops owing to lack of water. It is, of course, likeiy to occur at any time of the year when crops have been destroyed by plant disease or pests.

Birds Susceptible—

Deficiency of Vitamin A affects all classes of birds no less than it does animals (pigs, horses, cattle, etc.), but in birds the symptoms occur more quickly if the supply of vitamin A is withheld. In this State it is fowls, turkeys and ducks that suffer most frequently from the condition.

With fowls, green feed deficiency disease is often noted in young stock, such as pullets coming on to lay, but is quite likely to occur in older fowls if the deficiency continues for a sufficiently long period. It is rarely seen in young chicks for the reason that in a time of shortage the available green feed is usually allotted first for the use of the chickens. Laying pullets show the deficiency more quickly than first or second-year hens. Adult male birds, although susceptible, are not frequently affected, as they are able to withstand the lack of the vitamin much more than the laying hens, which pass some of their Vitamin A into their eggs.

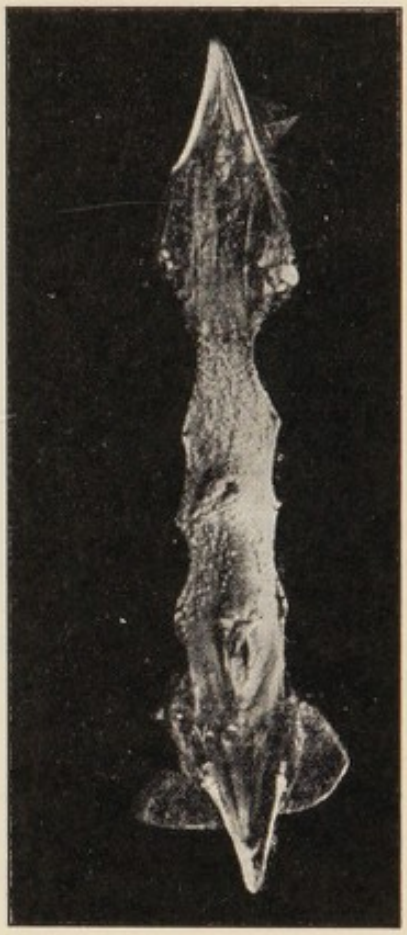
Onset of the Disease—

In fowls the length of time between the cutting off of the greenstuff or other Vitamin A supplement and the onset of the disease is variable. A good deal depends upon the health and condition of the birds when they first receive the deficient diet, and of equal importance is the ration of the fowls for some months prior to this time. The vitamin is stored within the body and the fowl is able to withstand its absence for some little time. The disease may be expected in from three or four weeks in pullets (even less in young pullets), but may be delayed for four months in grown cocks. If only a slight deficiency is present in the diet, the onset of symptoms may either be delayed longer or not be apparent. Slight deficiency may produce only a lowered egg production and decrease in general vigour.

Symptoms—

In fowls the first symptom in green feed deficiency disease is a general falling off in condition; the feathers lose ther lustre, growth and development are at a standstill, or retarded, and egg production is diminished, though the appetite is still unaffected. As the condition progresses some birds are noticed to waste away.

If the throat is examined at this stage small white pimples or pustules may be seen either in the mouth, on the back (at base) of the tongue, in the throat, and in the gullet. Later these pustules extend down the gullet and some are found even in the crop. If examination of the throat is undertaken at this time and the presence of these pustules observed, considerable losses can be avoided, as the disease may be stopped immediately by the addition of green feed, cod liver oil or other Vitamin A supplement to the ration.



Head, Throat, and Gullet Split Down to Show the Nodules on the Gullet Wall, due to Green Feed Deficiency.

[Photo. by courtesy of Glenfield Veterinary Research Station.

As the condition progresses some affected birds show a slight discharge from one or both nostrils, but this is variable. In the later stages, however, the nasal cavities are frequently plugged up with cheesy material which closes up the cleft in the palate, and usually causes a lowering of the palate itself into the mouth cavity.

Towards the late stages of the disease there is profuse watering from the eyes, usually from both, with reddening and swelling of the eye structures. Not infrequently the tear duct running from the eyes to the nostrils is distended with a discharge which cannot escape, and the tissues below the eyes become puffed out. A white material, which may readily be squeezed out by slight pressure, accumulates within the eyelids, and when this is present there is an absence of watery discharge. This feature is very characteristic of green feed deficiency disease, and, although not exclusively so, should warrant immediate inspection of the mouth and throat for pustules, which, if found, indicate the need for a corrective diet. Naturally, the affected eyes do not respond to local treatment, and a few hours after the removal of the white material from the eyelids more may be found.

At the same time as the eyes become involved, or even a little earlier, the breathing is rendered difficult and frequent attempts are made by an affected bird to "clear its throat." Inspection shows that the glottis (which is the entrance to the windpipe) is more or less closed up with cheesy material. This again is distinguished from that seen in other diseases of the throat in that the material is easily removed. Some-

times a complete tubular mass accumulates along the lining of the windpipe. These cheesy deposits may cause the death of the bird by asphyxiation. In one overseas report of the disease in fowls, the most prominent symptoms were a staggering gait, lying or sitting down, rotating in semicircles, or falling sideways or backwards. Chickens in all groups of this experiment showed the paralytic condition as the most prominent symptom of Vitamin A deficiency.

Not infrequently in young birds a staggering inco-ordinated (drunken) gait may be seen. If chickens hatched from normal eggs are fed a diet quite devoid of Vitamin A they develop this staggering gait at the approximate average age of twenty-five days.

In fowls death may occur one to five days after the eyes become affected, but the total period of sickness may vary from a few to fourteen days or even longer, the time depending largely on the amount of Vitamin A in the deficient diet.

In turkeys the absence of green feed or some effective substitute also causes a train of symptoms which may culminate in death.

The symptoms resemble an outbreak of acute infectious disease, except that temperature is not raised. When turkey chicks hatched from normal turkey eggs are fed a completely deficient diet from the time of hatching, symptoms develop on about the twenty-fifth or twenty-sixth day and death may occur about five or six days later, and all such poults will probably have died by the forty-fourth day after hatching. Actually, compared with chickens, turkey poults show symptoms a few days earlier and also die some days earlier. The first symptoms are listlessness, unsteady gait and sagging wings. These symptoms are nearly always noticed first. The bird may stand in a somnolent attitude with closed eyes and perhaps a foamy eye discharge. The third eyelid or nictitating membrane may be dry, rough and swollen, so that it covers half the eyeball. The surface of this membrane may be covered by a whitish, powdery material. Where the deficiency has been severe in turkeys, death may occur before any yellowish white pustules or cheesy deposits have occurred in the mouth, throat and crop. Where the deficiency is not so acute, these yellowish white pustules develop on the tongue, roof of the mouth and down the gullet, as seen in the case of fowls. In such cases sinusitis is a very common symptom, the infra-orbital sinuses becoming filled with a mucoid opalescent material. This condition is difficult to differentiate from the infectious condition of sinusitis described elsewhere. In a few cases there may be slight inflammation of the windpipe in turkeys suffering from this deficiency.

Apart from the above, the only other difference in symptoms and lesions shown in turkeys compared with fowls is that on post-mortem examination the kidneys in turkeys are not usually infiltrated with urates and the ureters are not packed with such material. It will be noted that this is a characteristic symptom in fowls. Further, urates which commonly occur in the abdominal cavity in fowls on the liver,

heart, and other abdominal organs are seldom, if ever, seen in turkeys. The Bursa cloacæ (of Fabricius) is commonly swollen with whitish, flaky material in both fowls and turkeys.

It should be particularly noted that where both types of birds are kept on the same ration, turkeys are more prone to show symptoms of Vitamin A deficiency than are fowls. Thus growing chickens may show no symptoms of Vitamin A deficiency on a particular ration, whilst growing poults on the same ration may show mild symptoms of the deficiency. With adult stock, such may not be the case, as laying pullets, for instance, will require much more Vitamin A in the diet than mature non-laying turkey hens.

In ducks, deficiency frequently produces cheesy material in the Bursa cloacæ (of Fabricius) as in the case of fowls and turkeys. In addition a mucoid eye discharge is often present and is referred to as "white eye."

Sick Bird, showing Swollen Eyes with Accumulation of Greyish white Material between the Lids.

Diagnosis—

Diagnosis is based on the characteristic appearance (see above) particularly (in fowls) by the pustules in the gullet, the changes in the Bursa cloacæ (of Fabricius) and also by noting that no Vitamin A rich food is present in the ration.

Differential Diagnosis—

Perhaps no more disastrous error could be made than to mistake a case of green feed deficiency disease for laryngo-tracheitis. The error may cost the owner a large proportion of his flock. The clinical differential diagnosis of roup diseases on page 22 should be referred to.

Pathogenicity and Economic Losses—

It has been shown that Vitamin A deficiency in the diet causes:—

(a) decrease in number of eggs laid;
(b) decrease in vitamin content of eggs;
(c) decrease in hatchability of eggs;
(d) decrease in weight of fowls;
(e) the actual death of birds;
(f) lowered resistance to other diseases.

Thus unless the condition is diagnosed and remedied losses may be enormous. In some experimental groups fed an otherwise adequate diet (lacking Vitamin A) *all* the birds died.

Post-mortem Appearances—

In fowls the appearance of affected eyes has already been described. On opening the mouth, pustules are usually seen on the tongue, particularly between the little fringe at the back and the glottis. The tongue may be "woody" in parts. There is mucous or whitish material in the glottis and this may extend for some distance down the windpipe. If the gullet is opened numerous small pin-head-size pustules are almost invariably seen in affected fowls and from them white material may be squeezed with the fingers. These pustules may extend even to the crop.

Apart from the poorness of flesh, other features seen on post-mortem examinations are as follows:—The kidneys are usually speckled with small, whitish, crystalline spots which are deposits of crystalline urates, and the tubes which lead from the kidneys to the vent (the ureters) may be dilated with deposits of urates. If the Bursa cloacæ (of Fabricius) is examined it will be found that in slightly more than half of those which die it contains whitish material which varies in quantity from a few flakes to a mass which distends the organ. The wall of this Bursa may also be thickened.

Treatment—

Treatment consists in the immediate supply of the vitamin lacking, all fowls on the deficient diet being given either green feed, carrots, some fish liver oil, or other substance rich in Vitamin A. Not only the obviously affected birds, but others which have not as yet shown the complaint should be so treated. The supply of the vitamin arrests the disease almost dramatically. Apart from those so severely affected that their death may be expected within thirty-six hours, there are good prospects of saving all sick fowls.

The obviously sick birds should be treated individually, each being given one-quarter of a teaspoonful of reliable cod liver oil daily by means of a dropper. Fowls less seriously affected and those not already showing symptoms should be given cod liver oil at the rate of 4 per cent. in the mash for the first two days, and thereafter at the rate of 1 or even 2 per cent. The oil should be added daily to the mash, as oil-containing mashes lose in efficiency on keeping. Poultry farmers should take care that pure and reliable cod liver oil is procured.

Green feed should be provided liberally as soon as this is available. It may be given at the same time as the cod liver oil, but when the disease has once occurred it is advisable to make certain that the fowls receive the necessary vitamin by means of cod liver oil, rather than to rely on all of them eating the green feed offered.

Little or no treatment of the affected eyes is necessary. The white material within the eyelids may be removed and the eyes bathed with boracic acid solution.

As the disease is of nutritional origin, it will be realised that disinfection of houses and liming the runs is unnecessary.

Prevention—

This conditions may be prevented by supplying some Vitamin A rich food such as green feed. It is to be noted that succulent dark-coloured green feeds such as lucerne, spinach, Berseem clover, Wong

Bok and half-grown cereal crops are richer in Vitamin A than light-coloured crops such as lettuce and cabbage. Maize that is nearly mature is a very poor class of green feed.

An adequate supply of green feed all the year round is of vital economic importance to the poultry farmer, and a variety of green feed is of great benefit to the birds. If the greenstuff is young and succulent the birds will eat ample when it is chaffed and placed before them as a separate feed. It is better to feed it separately, placing it out in troughs in a shady position during the middle of the day.

If the green feed is mixed with the mash either the increased bulk may prevent the bird consuming sufficient food or else sufficient greenstuff will not be in the mixture. If the green feed is not of first quality, a little appetising mash should be mixed with it, say, three parts of green feed to one part of mash, and this mixture should be fed in the middle of the day.

In very hot weather, it may be consumed better if fed late in the evening.

Vitamin A Supplements—

Where no green feed is available, some supplement such as cod liver oil, pilchard oil, sardine oil, or mutton bird oil should be fed. These animal products contain both Vitamins A and D. When pure cod liver oil is added to the mash, less than 1 per cent. fed continuously will supply all the vitamin necessary when no green feed is available. Mutton bird oil seems to vary widely in potency, and nothing definite can be stated, except that in some cases 1 per cent. has proved quite adequate.

Chickens require about 125 to 200 (U.S.P. or international) units of Vitamin A per 100 gms. of feed, while actively laying hens do best on about 400 to 500 units per 100 gms. A good sample of cod liver oil (pure) contains about 1,000 units per gm.* Thus 1 per cent. should supply twice the normal requirements at least. **Unfortunately many oils are far below this standard.**

In mixing these oils great care should be taken to incorporate them evenly in the bran first, then to mix the bran evenly with the mash.

A note of warning is issued concerning made-up mashes which are guaranteed to contain given quantities of cod liver oil. It is known that when cod liver oil is mixed with pollard or other powders it rapidly loses its Vitamin A content. Thus in one experiment in America it was shown that when cod liver oil was mixed in the ration it lost 85 per cent. of its potency in ten days.

Lucerne Dust and Yellow Maize—

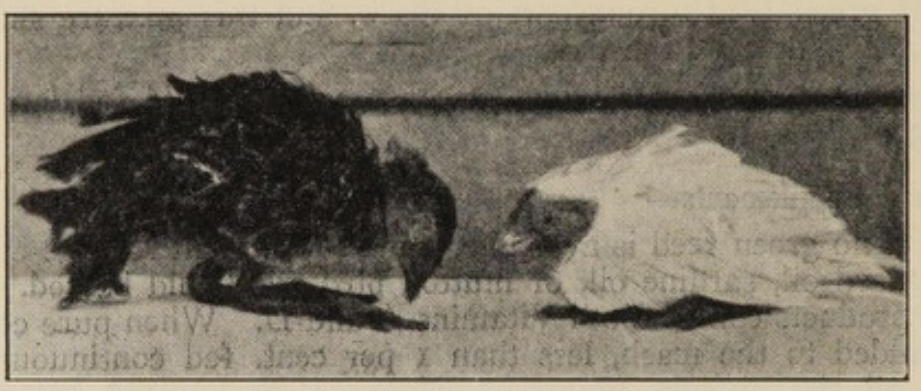
When green feed is not available most farmers include 8 to 10 per cent. of lucerne dust in the ration. Lucerne dust, if of good quality, does supply Vitamin A, and in nearly all cases the above procedure will prevent the development of marked symptoms. But with the average sample of lucerne dust, leaf or hay on the market, the birds

*For complete list of Vitamin A content of all foods, see Fraser M. A. B. and Roscoe M. H. 1938—Nut. Abs. and Reviews 7: 823 (257 references).

reflect the mild deficiency present, in a lowered egg production, and increased susceptibility to disease, and should be fed some cod liver oil.

The amount of Vitamin A in lucerne meal varies greatly. Good green leafy samples may be relatively rich when first cured, but one investigator found that, if kept, it lost about 7 per cent. of its total content each month.

Yellow maize supplies some Vitamin A, but one pound of yellow maize supplies only about one-eighth as much Vitamin A as one pound of good quality lucerne dust. As the amount of vitamin supplied by lucerne dust is usually inadequate, yellow maize cannot be seriously considered as a sole source of Vitamin A.



A Five-weeks-old Poult and a Six-weeks-old Chick.

Both received the basal ration from time of hatching, and were showing typical symptoms of Avitaminosis for the respective specie.

[*After Hinshaw and Lloyd.*

Green feed is the only economical way of supplying this vitamin, and the poultry farmer should attach a great deal more importance to growing adequate quantities of it.

Provision of Green Feed—

As already stated vigorous growing young green feed with a rich deep-green colour contains most Vitamin A. Apart from supplying Vitamin A many other important constituents are supplied, and green feed is essential to ideal health. Green feed is best fed separately either at midday or, in very hot weather, after the evening feed. If added to the evening mash more than 20 per cent. of the bulk should not be used.

Crops Suitable.—Lucerne, Berseem clover, rape, silver beet, young cereal crops, such as barley, oats, Hungarian millet, Wong Bok (Chinese cabbage) are all suitable. Lucerne is the crop of outstanding value. Some farmers grow crops of kikuyu grass—where beds of this grass are kept heavily manured and constantly wet an enormous bulk of green fodder may be produced. The value of the latter, however, is less than lucerne; and the grass, if it escapes, may over-run cultivation land.

Continuous Supply.—Care should be taken to maintain a constant supply of green feed. Full information regarding the growing of crops may be obtained on application to the Field Branch of the Department of Agriculture.

A Cropping Programme*—

The following table, supplied by the Director of Agriculture, shows the crops which may be planted each month for the maintenance of a supply of green feed throughout the year :—

Crops to Sow.		Crops to Sow.	
Month.	Crop.	Month.	Crop.
January ...	Maize, cowpeas.	June ...	.....
February...	Maize, barley, rape, field peas, kale, silver beet, Berseem clover.	July ...	Rye.
March ...	Lucerne, wheat, oats, rape, barley, field peas, kale, silver beet, Berseem clover, Chinese cabbage (Wong Bok).	August ...	Silver beet.
		September	Hungarian millet, maize, lucerne.
April ...	Lucerne, oats, barley, field peas, kale, red clover, Bokhara or sweet clover, silver beet, Chinese cabbage (Wong Bok).	October ...	Cowpeas, maize, Hungarian millet, lucerne.
		November	Cowpeas, maize, Hungarian millet.
May ...	Lucerne, clovers, oats, barley, silver beet.	December	Cowpeas, maize.

Crops sown according to the above table would provide fodder for poultry in the following months :—

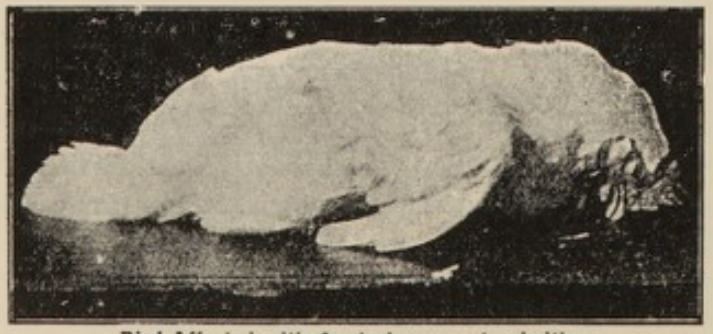
Month.	Feed available.	Month.	Feed available.
January ...	Lucerne, cowpeas, maize, silver beet.	July ...	Wheat, oats, barley, rape, field peas, kale, Berseem clover, Chinese cabbage.
February...	Lucerne, cowpeas, maize, silver beet.	August ...	Wheat, oats, barley, rape, kale, field peas, rye, Berseem clover, Chinese cabbage.
March ...	Lucerne, cowpeas, maize, silver beet.		
April ...	Lucerne and clovers, wheat, oats, cowpeas, silver beet, barley, rape, maize.	September	Clovers, oats, barley, field peas, rye, Berseem clover.
		October ...	Lucerne and clovers, field peas, silver beet.
May ...	Lucerne and clovers, cowpeas, silver beet, barley, rape, kale, Berseem clover.	November	Lucerne and clovers, maize, Hungarian millet.
June ...	Wheat, oats, barley, rape, field peas, kale, Berseem clover, Chinese cabbage.	December	Lucerne, maize, Hungarian millet, and clovers.

*Extract from "Green Fodder for Poultry," by E. Hadlington. This pamphlet is available free of charge on application to the Department of Agriculture.

Infectious Laryngo-tracheitis.

Bird Affected with Peracute Laryngo-tracheitis.
Gasping for breath and emitting shrill cries.

Chronic Laryngo-tracheitis.
Bird wasted away and unable to stand.



Bird Affected with Acute Laryngo-tracheitis.
Lying exhausted after a convulsion.

This is perhaps the most important of all the roup diseases. It is also known as "roup" of various forms, tracheo-laryngitis, tracheitis, bronchitis (incorrectly, as this refers to another disease), Canadian 'flu, or 'flu.

The disease was first reported (as far as is known) by Beaudette in Kansas in 1920. In 1921 it was described in Canada. In 1930 it was shown to be due to a virus. In 1931 it was reported in almost every state of the United States of America. In this country a report was received in 1934, from a veterinary practitioner on a mortality suspected by him of being an outbreak of infectious laryngo-tracheitis. Investigations at the Glenfield Veterinary Research Station and at the Walter and Eliza Hall Research Institute in Melbourne confirmed this suspicion.

The disease has continued widespread in this State since that time.

In 1935 it was diagnosed in Great Britain.

Cause of the Disease—

(*a*) *Actual Cause.*—Laryngo-tracheitis is caused by a tiny micro-organism known as a filtrable virus. This organism is too small to be seen under the microscope, but it can be grown on egg membranes under laboratory conditions. Without the

presence of this virus, the disease cannot occur. It would appear that the severity of the symptoms in a particular outbreak depends mainly on the potency of the particular strain of virus with which the flock becomes affected.

(*b*) *Predisposing Causes.*—Various other factors may influence the severity of the symptoms shown when once the birds become infected. The chief of these are green feed deficiency disease, parasitic infestation and incorrect management.

(1) *Green Feed Deficiency Disease.*—Where birds are fed insufficient green feed and no other supplements such as fish liver oils are provided, the birds' resistance to laryngo-tracheitis and other catarrhal disease is lowered.

(2) *Parasitic Infestation.*—Where a flock is heavily infested with large round worms (*Ascaridia* sp.) such infestation will lower the general health of the flock and it has been noted that when laryngo-tracheitis is introduced on to such a farm, mortality is severe. Infestation with tape worms will lower the bird's disease resistance, and appears to have definite effect in increasing the mortality rate in some outbreaks. In very young birds, coccidiosis, acting concurrently with laryngo-tracheitis, may cause a disastrous mortality. It is well known that coccidiosis on its own may cause severe mortalities, even up to 80 per cent. of the affected chickens dying.

(3) *Over-crowding.*—The over-crowding of birds is at all times an unfortunate proceeding, favouring heavy lice infestation, outbreaks of coryza, cannibalism, and poor health. When laryngo-tracheitis breaks out amongst birds which are kept under over-crowded conditions, mortality is much augmented.

(4) *Fowl Pox.*—Fowl pox may occur at the same time as laryngo-tracheitis and will contribute to the death rate. There is, unfortunately, a common correlation between fowl pox vaccination and outbreaks of laryngo-tracheitis in districts where both diseases occur. When pullets are vaccinated for fowl pox, they undergo a systemic reaction about fourteen to eighteen days later. During this period the pullets are particularly susceptible to laryngo-tracheitis infection, which may be contracted from old carrier birds on the farm, these birds having "recovered" from an outbreak of laryngo-tracheitis the previous season.

(5) *Other Factors.*—Faulty ventilation, dusty yards, uncleanliness and incorrect feeding are all factors which predispose the birds to this disease and also aid in its spread when once introduced.

Characteristics of the Causal Virus.—The virus which causes the disease is less than 0.082 μ in size (a μ is approximately 1/25,000 of an inch) will pass through filters which hold back bacteria, and this bacteria-free filtrate will produce the disease when susceptible birds are infected with it.

The virus can be grown on the egg membranes of the developing chicken embryo, and on these membranes the growth has a characteristic appearance. The New South Wales strain of virus is identical with the United States strain, though it is doubtful whether this is the

case with a Victorian strain of low virulence.* The blood serum from a bird recovered from laryngo-tracheitis contains substances which neutralise virus and destroy its infectivity.

Infectivity—

(*a*) *Mode of Infection.*—With laryngo-tracheitis, the natural means of spread is by direct contact. An infected bird coughs, making a fine vapour spray of the infectious material, which is then inhaled by other birds in the vicinity.

Carriers.—The disease may be introduced on to a farm in many ways. Quite frequently an outbreak occurred following the introduction of birds which were affected with the disease many months previously, and had apparently recovered. Such birds have been shown to remain carriers of the virus for very long periods—in some cases, over a year. Thus, it will be seen that the introduction of cockerels for breeding purposes is a potential source of infection.

The new season's pullets should be kept strictly separate from older stock previously affected, unless the new season's stock is first vaccinated.

Crates.—Infected houses, coops, etc., may cause an outbreak, for, when birds infected with the disease were placed in metal coops for a time, then removed, and shortly afterwards susceptible fowls were placed in these coops, such birds contracted the disease. From this, it is clear that when carting agents bring market crates on to the poultry farm and place them in yards where poultry are to be loaded, considerable risk from this disease is involved. There is evidence that the disease has been spread in this way on many occasions in the county of Cumberland.

Birds.—It would seem probable that sparrows, which frequent drinking troughs (for bathing, drinking) may readily carry the infection from one farm to another.

Parasites.—It has been shown that mites may carry the virus.

The danger of transmitting the virus through the egg is remote, and no cases have come to notice which arouse suspicion. In the case of young chickens being affected, it has been noted on at least one occasion that the virus was introduced by sparrows, hundreds of which frequented the battery brooder in which the chickens became affected.

(b) *Animals Susceptible.*—Fowls, of all ages and breeds, pheasants, and hybrids of these, would appear to be the only susceptible species of birds so far recognised.

(c) *Viability of the Virus.*—Fortunately, the virus is killed very easily by disinfection, and it does not remain alive for more than short perods when dried and kept at ordinary room temperature. In one series of experiments where the virus was kept moist at room temperature it did not remain infective longer than 100 days. When dried this period is much reduced. In this respect, the disease is in marked

*For further information see Burnet, F. M. (1936), Jour Exp. Med. 63: 685.

contrast to such conditions as pullorum disease or coccidiosis, as in these latter diseases, the causal micro-organism may remain alive in protected positions for more than a year.

Immunity—

Birds which have been once affected with the disease are resistant to any further infection, and it is this fact which makes possible the control of the disease by vaccination. Once a good reaction to vaccination is obtained the bird, in most cases, remains immune during the normal commercial hen life.

No particular breed appears to be more susceptible than others.

Incubation Period—

Investigations at the Glenfield Veterinary Research Station have shown that in the more acute infections the incubation period is shorter than in mild outbreaks. Thus, where highly infective material is blown down the windpipe of a fowl, the disease may develop in less than forty-eight hours. Symptoms will rapidly become violent and the bird may die after a short period. When the disease is spread by natural contact, the period between contact and the first appearance of symptoms usually varies from three to ten days.

Symptoms—

Symptoms in outbreaks of laryngo-tracheitis show a very great variation. The bird affected with the peracute form may gasp a little, cough out some blood-stained mucus, and then rapidly succumb to the disease. Alternatively, such a case may linger on and pass into the acute or chronic phase of the disease, in which all the typical symptoms of "roup" are apparent.

When the disease first makes its appearance in a flock, it is usual for many of the birds first affected to show the peracute form. Birds which become affected in the next few weeks may exhibit only chronic or mild symptoms. On some farms the disease starts as a mild outbreak only, in which no peracute cases are noted, and in such instances the mortality is correspondingly lower. Symptoms observed in a typical severe outbreak would appear as follows:—

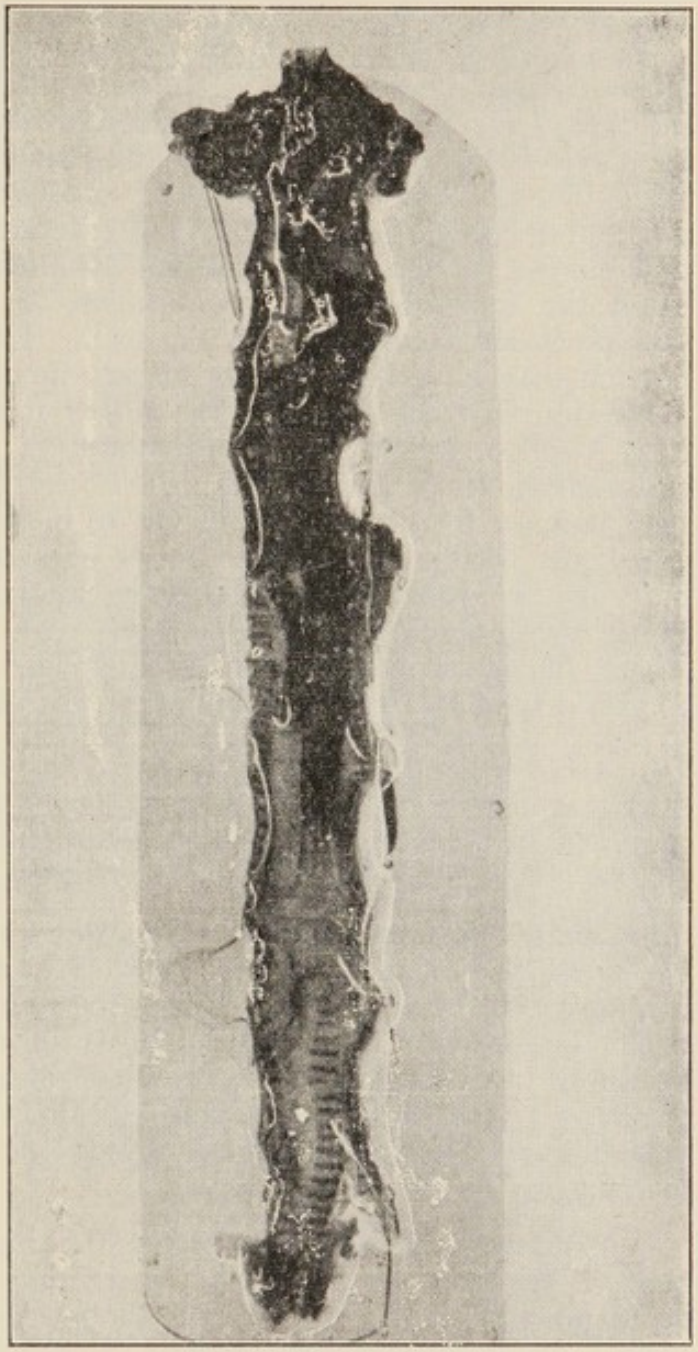
Some of the birds will be noticed to swallow frequently, and soon after a frothy discharge will appear from the beak, nostrils and eyes. If such a bird is watched for a time it will open the beak during respiration. Soft, gurgling sounds may then be heard as the bird holds the beak slightly open during inspiration and extends the neck. After several gurgling respirations, the bird will give a moist cough, flinging its head as it does so and spraying mucus into the air. The bird appears to be a little depressed and is manifestly "feeling sick." After several hours the depression deepens, and if the bird is caught and examined, blood-stained mucus may be noticed in the windpipe, or small haemorrhages may be seen on its lining membrane. After coughing, the fowl frequently wipes the discharges from its nostrils and eyes on the feathers of its back. These discharges dry and mat the feathers, giving a characteristic appearance. In some cases the bird may cough out blood. If this happens at night-time, a torchlight examination will enable such symptoms to be detected, but during the day-time the other

birds peck up the blood-stained discharges. After some hours, the affected bird may exhibit a darkened, somewhat shrivelled comb and wattles (due to faulty aeration of the blood and failing heart action) and a little later go into a convulsion and die.

The disease rapidly spreads from bird to bird, and within two or three days perhaps 80 per cent. of the birds in the yard affected will be showing symptoms. After two or three days some birds which have been affected with the peracute form will show less violent symptoms. Such cases will exhibit cheesy deposits in the mucous membrane of the mouth, throat, tongue, palate, in the windpipe and in the eyes. In all cases the formation of these cheesy membranes is preceded by a thin mucoid discharge, which gradually thickens during a period of one to two days, to form the cheesy deposits. Such birds cough frequently. If the windpipe is obstructed at its upper end with a cheesy plug, the bird may die of suffocation. If the obstruction occurs at the lower end of the windpipe, the bird may utter shrill cries as it endeavours to expel the obstruction from the respiratory passage. The eyes and the infra-orbital sinuses (cavities between the eyes and the nostril) may become enormously distended with cheesy material so that the orbital cavity stands out perhaps half an inch further than normal. Such cases result in complete blindness, and the bird may die of starvation. Poultrymen, coming on the farm at this stage, would say that such chronic cases were typical of "diphtheric" and "canker roup," and, of course, these descriptive terms are quite true. By mere clinical examination, such cases cannot be differentiated from infectious catarrh. The disease continues to spread. New cases occurring in that particular fowl-house after the disease has been present for a week or so seldom show the peracute symptoms; that is to say, no blood is coughed out from the windpipe and, as a rule, the bird merely shows a nasal and eye discharge, followed by the development of cheesy deposits on all the mucous membranes of the head. Throughout the course of the disease a particularly foul smell is in evidence. In some cases many of the birds show a greenish-yellow diarrhoea. The birds show little inclination for food, but seem very thirsty, as they drink copiously. Careful examinations by day and night reveal the fact that discharges are more profuse at night. Cases which occur in a yard after the first week seldom cough out blood, though at times mucus which is slighly blood-tinged may be expectorated. Birds so affected may present a horrible spectacle. They cough, exhibit a slimy, stinking discharge from the nose, protruding orbital cavities, and make strangled, gasping cries as they endeavour to breathe through the obstructed windpipe. All forms of roup symptoms may be in evidence.

As a result of the small food intake and the feverish condition present during the disease (the temperature may run over 110 degrees Fahr.) affected birds rapidly waste away and become emaciated. Some birds may show a slimy discharge from the ear, and in some cases the head may be held on the side or upside down as a result of the inflammation of the balancing mechanism located in the ear. In severe outbreaks, nearly all the birds in the yard may show these severe and spectacular roup symptoms, and the farmer who has had no previous experience with the disease is seized with despair. Such

a position offers wide scope for the activities of proprietary medicine vendors, as many of the birds affected with the chronic form, which shows such spectacular symptoms, will recover. Unless the treatment is markedly harmful, such birds, if treated, will probably recover in spite of the disease and the "remedy!" The "remedy" used then enjoys a reputation as a cure for laryngo-tracheitis. Actually, in some cases, farmers have treated birds affected with this chronic form, and left another yard in which a few coughers were just appearing, as a comparative group. As would be expected, while the birds in the treated yards mostly recovered, this comparative group developed the whole range of the disease symptoms, many birds dying. As they entered the chronic stage and started to appear hopeless (but actually on the point of recovery) these birds in turn were treated, and any doubts the farmer had entertained as to the efficiency of the drug were swept away.



Trachea of a Peracute Case of Laryngo-tracheitis.
Opened up to show free blood in the upper two-thirds of its length.

Asymptomatic (symptomless) Form of the Disease.—Some birds in an affected yard may fail to show symptoms of the disease, and when infected artificially are shown to have a resistance to the infection. These birds have obviously suffered from a very mild form of the disease without actually showing clinical symptoms.

Post-mortem Appearances—

Peracute Form.—In the peracute form the disease runs its course so rapidly that the bird has not had time to become emaciated. Thus, well-conditioned birds may be found dead with discharges from the

mouth, nose and eyes. In most cases, on conducting a careful post-mortem examination, the only striking changes seen are in the trachea (windpipe). Care should be taken to remove this organ intact and to clean all the blood off the outside before opening.

The characteristic symptom of this type of the disease is an acute haemorrhagic inflammation of the windpipe with free haemorrhage into its cavity, together with a copious mucous secretion. In some cases, the cavity of the lower part of the trachea may be completely filled with blood. Haemorrhages are present at various parts or all along the lining membrane of the trachea and larynx. The trachea and larynx are acutely inflamed, sometimes throughout, but very often the inflammation is more acute on the lower portion of the windpipe down towards the bronchi. The lining membrane in this portion may have been coughed away in the worst cases, leaving only a bleeding surface. Where the lining mucous membrane is present, it is swollen and watery (oedematous). The nasal cavity, nostrils, turbinate bones, palatine cleft, infra-orbital sinuses, mouth and throat may contain blood-stained mucus or blood. In some peracute cases which die within twenty-four hours of showing symptoms, free blood may not be present. In such cases, numerous sub-mucus (under the lining membrane) haemorrhages are seen in the larynx and trachea. These haemorrhages appear as little red spots or patches varying from pinhead size to a quarter of an inch in diameter.

If the bird dies in a convulsion, very often it will be found that greyish-yellow crop contents have been vomited up from the crop and will be present in the mouth and nasal cavities. This material should not be confused with the discharges due to the disease.

In many outbreaks where peracute and acute cases occur there have been small haemorrhages (blood spots) in and under the lining mucous membrane of the proventriculus (first stomach). These are by no means constant, only some dead birds showing them. This particular symptom has been put forward as characteristic of Newcastle disease, which is a completely different condition. Some birds have been seen with an acute inflammation involving more than half of the lining membrance of the proventriculus.

Apart from the above, no very striking post-mortem changes are seen. The heart may be dilated and flabby. The lungs usually appear quite normal to the naked eye, though on rare occasions they may appear oedematous or congested.

Acute Form.—No free blood is seen in the trachea, but haemorrhages (red spots) under the lining membrane of the larynx and trachea are usual. The typical symptom is inflammation of the trachea with copious mucoid discharges. Yellow, cheesy material is usually present in the mouth around the opening of the larynx, sometimes down the trachea, and very frequently in the infra-orbital sinuses. The lining membrane of these two cavities situated under the eyes if often inflamed, and at times small pin-point haemorrhages are present. Small patches of degeneration are seen in the wall of these sinuses from time to time.

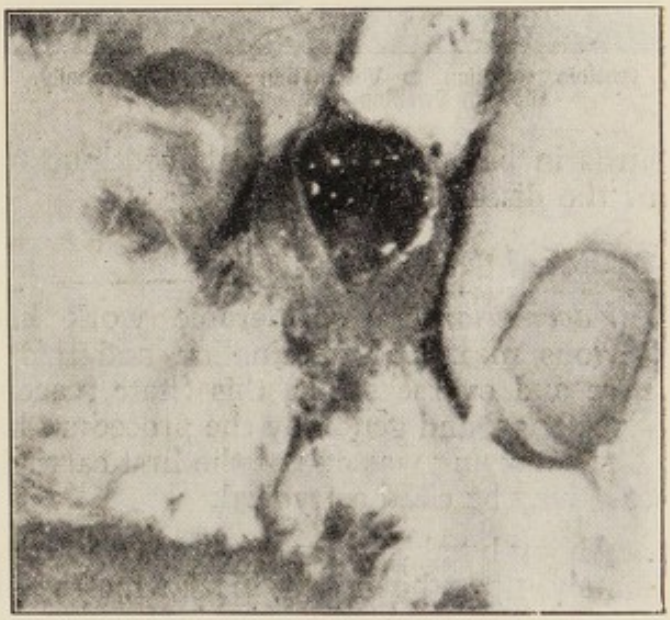
Large quantities of slimy discharge are present in the mouth, throat, larynx, trachea, nasal cavity, palatine cleft and infra-orbital sinuses. As mentioned before, haemorrhages under the mucous membrane of the proventriculus are sometimes present. Enteritis with patchy congestion (reddening) of the upper third of the small intestines is not uncommon. No other changes are regularly seen.

Chronic Form.—In this type, yellow, cheesy deposits are present in the eyes and infra-orbital cavities, nasal cavity, mouth, throat, tongue, and top of the larynx. Congestion (reddening) or mild inflammation of the larynx and trachea is present, and cheesy cankerous material may be seen at times right down the cavity of the windpipe. No constant changes are present in other organs.

Diagnosis—

(*a*) *Clinical.*—(See symptoms and post mortem above). When a severe outbreak of a "roup" disease occurs with a heavy mortality and all birds affected show severe inflammation of the trachea, the diagnosis of laryngo-tracheitis is a strong probability.

(*b*) *Technical.*—In the laboratory positive transmission of the disease from bird to bird (by placing infective material in trachea) with the production of characteristic symptoms of the peracute disease, will confirm the probability. Growth of the virus on chicken embryo membranes will clinch the matter.



Positive Reaction to Vaccination for Infectious Laryngo-tracheitis, showing swelling and inflammation of Cloacal structures.

[*After Beach et al.*

(*c*) *Serological.*—If a bird recovered from the disease is taken to a laboratory, blood serum from such a bird will neutralise the virus of laryngo-tracheitis, and further, such a bird cannot be reinfected experimentally. These facts can be used to determine if a particular disease that occurred in a flock, say a month ago, was actually laryngo-tracheitis.

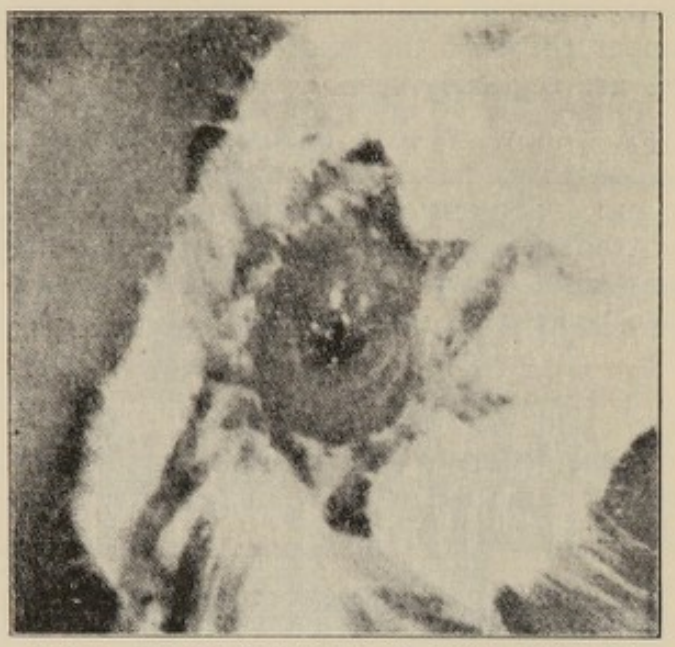
Differential Diagnosis.—(See page 22).

Pathogenicity—

Mortality varies very greatly in outbreaks of laryngo-tracheitis. In two outbreaks in July, 1938, a typical contrast is noted:

On one farm where 1,200 birds were involved, a very mild form of the disease occurred and only one bird died. Within a few days another outbreak of the disease occurred on a farm of a little over 1,400 birds.

Of these, 500 died within ten days of the commencement of disease symptoms. In both cases there was a severe loss in egg production during the course of the disease. The variation in the mortality is attributed both to the difference in potency of the strains of virus, and to other predisposing causes limiting the virulence already mentioned.



Positive Reaction to Vaccination viewed Externally, showing Swelling of the Cloacal Lips.

In very severe outbreaks the mortality may exceed 80 per cent. The usual mortality rate varies from 10 to 35 per cent. unless some other complicating factor (e.g., deficiency of Vitamin A) augments the severity of the disease. Economic loss is often fairly heavy from decrease in egg production. It is, however, a noticeable fact in some outbreaks that birds in badly affected yards continue to lay right through the course of the disease.

Control of the Disease—

*Vaccination.** Considerable work has been done on testing out various means of vaccination and this work is still continuing both here and overseas. In this State, vaccinations have been carried out since 1935, and generally the procedure has been a very definite success. The following was one of the first carried out and the experience in this case may be cited as typical.

An outbreak of laryngo-tracheitis occurred on a farm of approximately 1,000 birds within the metropolitan area in December, 1936. It was then decided to vaccinate one yard of pullets at a considerable distance away from the yards in which the infection was known to be present. One hundred and forty White Leghorn pullets about sixteen weeks old were vaccinated and on examination on the fifth day, 95 per cent. were shown to have taken positively to the vaccination. The disease spread from the first three yards affected, progressing from yard to yard. In January, 1937, only one yard of pullets apart from the vaccinated yard remained unaffected and these birds were then vaccinated, 105 birds, seventeen weeks old, being so treated; 97 per cent. of these showed a positive take to the vaccination. It is interesting to note that the disease progressed right to the yard first vaccinated, missed this yard and continued through the farm, missing only the

*Technique of vaccination which is carried out in the *Bursa cloacæ* (of Fabricius) is not given here as being entirely a matter for veterinarians. For details see article Hungerford, T. G., and Hart, L., 1939, at present (March, 1939) in course of publication in *Australian Veterinary Journal*.

second yard vaccinated, *i.e.*, the two vaccinated yards were the only ones to escape the outbreak of the disease. Since that time, the vaccinated birds have been freely intermingled with the carrier stock and only one case of laryngo-tratcheitis has occurred in the vaccinated birds and this may have been one of the birds which failed to take to the vaccination. Mortality on this farm, yard by yard in the unvaccinated birds, was as follows:—

Pullets, yard 1—27 out of 102; yard 2—31 out of 280; yard 3—27 out of 250.

First-year hens—33 out of 150.

First-year hens—12 out of 100.

Second-year hens—8 out of 100.

Three months later, a further check up on the results of vaccination was made and it was found that no harmful results in any way had occurred as an effect of vaccination.

Practical Aspects of Vaccination—

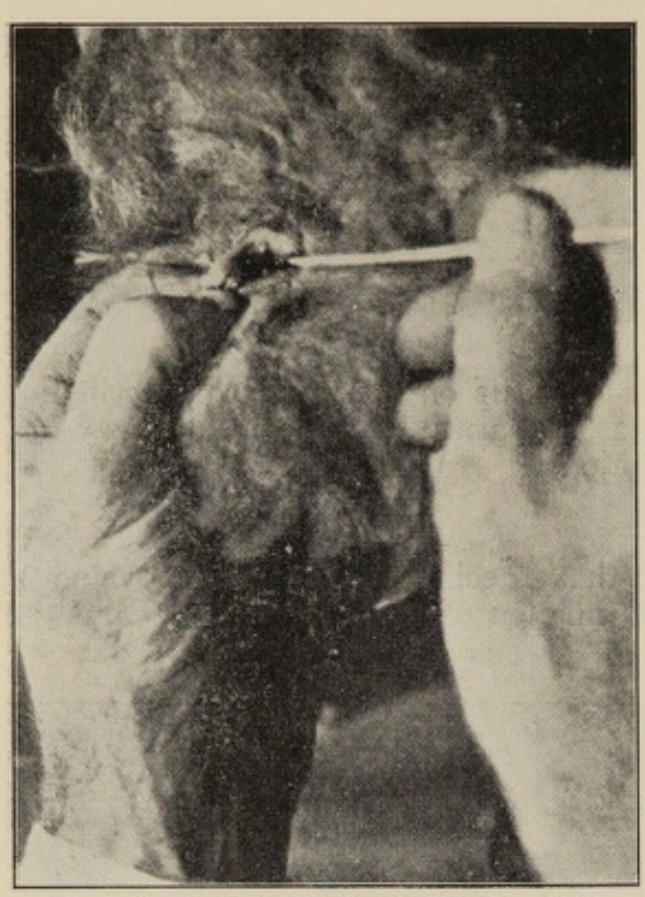
It must be clearly realised that vaccination is only of value in *preventing* the disease. The vaccination of birds affected is valueless. Vaccination of yards of birds where a large number are already affected has also been found valueless in most cases. Thus when the disease breaks out in one yard prompt action should be taken to have all other birds on the farm vaccinated.

In vaccinating for laryngo-tracheitis, the live virus is used. It will be realised, therefore, that there is danger in the procedure, and in view of this fact, it is highly undesirable that any flock should be vaccinated unless the disease has already occurred in the birds. Following vaccination, it is possible that a few cases of the disease (sometimes up to 5 per cent.) may develop.

A Bird Infected with Laryngo-tracheitis.

When once the disease does occur on a farm, prompt action should be taken. As soon as a few birds are noticed to be affected in one yard, veterinary advice should be sought, and if vaccination is to be carried

out, it should be done at once (within a few hours). In many cases, a delay of even a few days has allowed the disease to spread throughout the flock. In view of the work involved, costs of vaccination are necessarily high. Under the present method of vaccination, the veterinary surgeon concerned has to make a visit to the farm, collect the affected birds, and then take them to the laboratory. He has the vaccine prepared, and within a few hours returns to the farm to carry out the vaccination. A further visit is made on the fifth day to examine reactions, and those birds which have failed to react are re-vaccinated from positive "takes." These three visits necessitate a fairly heavy charge being made. But it is a noteworthy fact, that each farmer who has experienced a severe outbreak of the disease considers that the cost of vaccination is negligible compared with the cost of the losses which occur when it is not carried out.



Vaccination against Infectious Laryngo-tracheitis.
The Bursa groove and the aperture leading into the Bursa of Fabricius, exposed for vaccination.
[After Gibbs.

Further information concerning vaccination may be obtained from the Department. Those interested should note that only registered veterinary surgeons are allowed to carry out vaccination, that the disease must first be diagnosed on the farm, that the vaccine must be prepared at an approved laboratory, and that vaccination must be reported to the Chief Veterinary Surgeon within five days. These legal requirements are, of course, framed in the interests of the poultry farmer and others concerned.

Vaccination of Laryngo-tracheitis.
The correct way to hold the bird for vaccination.
[After Gibbs.

The Dangers of Vaccination—

The more one sees of vaccination the more confident one becomes in recommending it. Observation of over 30,000 birds vaccinated and a check up on nearly 100,000 confirms the impression that once a severe outbreak of laryngo-tracheitis occurs in one yard on a farm, only prompt vaccination of the rest of the birds as yet unaffected can, and will prevent mortality.

Against this may be mentioned that where vaccination results are poor (*e.g.*, where only 10 per cent. of the birds "take" to vaccination) results may be very distressing as the birds which fail to react to the vaccination may then catch the disease from those which have reacted, or from other infected birds on the farm.

In the past in this State, faulty reactions (when fresh vaccine has been used on susceptible birds) have invariably been due to the failure to use sufficient friction when applying the vaccine, and in vaccinating 20,000 birds experimentally using adequate friction not one such failure was experienced.

Where fowl pox and laryngo-tracheitis both occur on the farm each year, simultaneous vaccination may well be carried out. In such a case failure of the birds to react to the laryngo-tracheitis vaccination is very unfortunate for the reason that the birds normally react to laryngo-tracheitis vaccination at four to eight days and show the systemic reaction to fowl pox about ten days later. Hence a bird which fails to react to the laryngo-tracheitis vaccination is particularly liable to contract it when the health is upset ten days later due to the systemic reaction to fowl pox.

Vaccinated birds carry the living virus in the cloaca for six or seven days. They never remain carriers of the virus for more than eleven days unless actually affected with the clinical form of the disease after vaccination.

Control Apart from Vaccination—

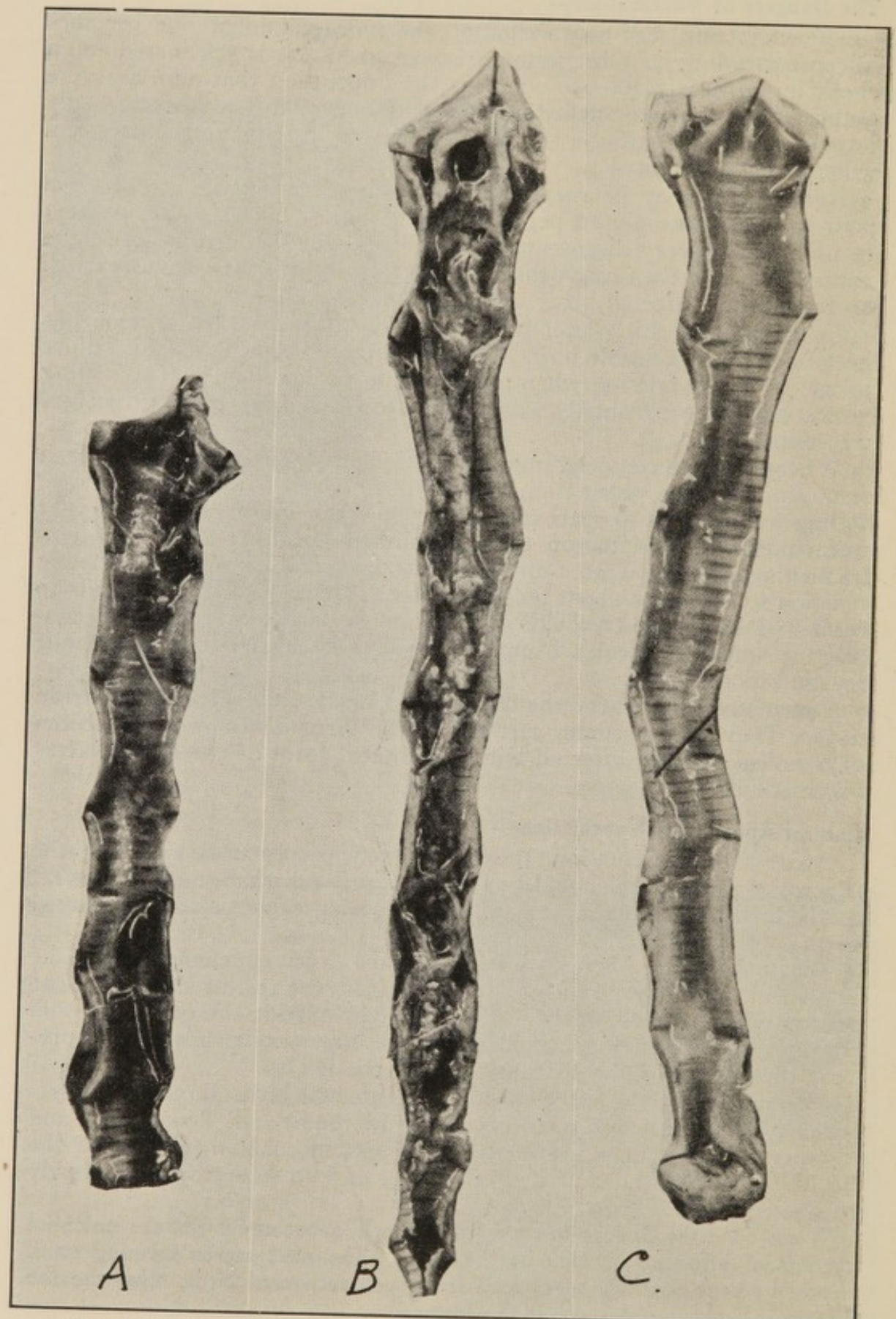
Apart from vaccination, the only practical measures aimed at the control of the disease should be to maintain the disease-resisting powers of the bird by eliminating all the pre-disposing causes already referred to.

When the disease breaks out in one yard every endeavour should be made to keep this yard of birds isolated from the rest of the flock. One precaution is to place a tray of disinfectant outside the gate and walk through this on leaving. Such measures, however, usually fail to prevent the spread of the disease through the flock.

As far as possible, infected and non-infected birds should be segregated. Make a nightly inspection and remove all "coughers" and "gaspers." This may prevent to some extent mass infections of the healthy birds by those actively infected, and so minimise the severity of the epidemic.

When once the disease has affected a flock, recovered birds are potential carriers of infection as long as they live. The next season's young stock should be kept carefully segregated from such recovered birds, otherwise the disease is likely to break out in the former.

Such an occurrence has been commonly seen where mingling of the new season's pullets with the hens (previously affected) has occurred



(A) Trachea Slit Open. Free blood present on the lower portion of the lining membrane—a peracute case.
(B) Trachea Slit Open. To show cheesy masses down the entire length of the organ—a chronic case.
(C) Trachea Slit Open. To show slimy mucus covering its surface—a subacute case.

Treatment of Laryngo-tracheitis—

Many claims are made that certain medicines are cures for laryngo-tracheitis. Unfortunately, such claims are ill-founded. At present there is no known cure for the disease and there is little reliable evidence that any medicinal preparation is of much value in its treatment. Certain home remedies such as kerosene and solutions of such drugs as Condy's crystals, bluestone, eucalyptus, borax and salt, melasol, eusol, lysol, and scores of proprietary preparations are in common use. Most owners claim to have at least one cure for laryngo-tracheitis, but as already explained, most of the alleged cures are based on the treatment of birds affected with the sub-acute or chronic forms of the disease, and many of such birds recover with or without treatment. Some preparations which aim at lowering excessive temperature of the bird and loosening up the tracheal discharges have a logical claim to value as remedial measures. Such measures can only assist the course of the disease, and their practical usefulness must be assessed with careful regard to the expense involved. Where a poultry farmer desires to test the usefulness of any drug, it is suggested that one yard of birds which are affected should be divided into two exactly even groups. For every bird affected with the chronic form in one yard, another bird so affected should be placed in the other yard. For every bird beginning to show symptoms that is placed in one yard, a similar bird should be placed in the other yard, for comparison. If one of these yards is then treated and the other yard left untreated, an accurate assessment of the value of the home remedy under trial will be possible. So far, in each case that has come under the writer's notice, where a poultry farmer has carried out such a trial, the farmer has discontinued the treatment of his flock after the completion of the trial.

In furtherance of the above, it is pointed out that treating the eyes with an astringent solution such as a colloidal-silver preparation or zinc-sulphate (1 oz. zinc-sulphate to two pints of water) may assist in preventing blindness. Bathe the eyes clean, and then flood in a few drops of the solution several times a day. Removal of the cheesy deposits from the top of the windpipe may temporarily prevent birds from suffocating, and these two forms of treatment may save the lives of a small number of birds which would otherwise succumb to the disease. After removal of the cankerous material the raw area may be painted with a solution of 1 in 50 of copper sulphate in water, or 1 in 20 of picric acid in alcohol. The smearing of a mixture of eucalyptus oil 1 part and liquid paraffin 2 parts around the nostrils is thought by some to loosen up the secretions and a mixture of eucalyptus 1 part, friar's balsam 1 part, and liquid paraffin 2 parts is also used.

The value of these treatments is so little that the expense of the labour involved in application is not justified.

Owners frequently hold the head of affected birds in various disinfectant solutions, until they gasp. Should the disinfectant fluid be gasped down into the lungs, the bird frequently dies as a result of the treatment.

Infectious Catarrh.

This disease has probably been present in poultry in New South Wales since the beginning of the poultry industry. When careful investigations were made into laryngo-tracheitis outbreaks, it was found that the chronic form of laryngo-tracheitis and also the milder types of the acute form closely resembled a form of "roup" which had been present for many years, and which, apparently, was not due to green feed deficiency, fowl pox, or coryza.

Investigation into the exact etiology of outbreaks of infectious catarrh has been extremely limited in New South Wales, and it may be that this disease entity will be shown in the future to be a group of several closely allied diseases, the relationship of which is not at present certain, but which may have some connection with the group known as coryza.

Cause—

(*a*) *Actual Cause.*—This is uncertain, but would appear to be a virus. With reference to the cause and identity of the disease, information has been accumulated along the following lines. In 1935, twenty-four outbreaks occurred of a catarrhal disease that simulated mild laryngo-tracheitis, and in each of these outbreaks it was impossible to transmit the condition by injecting infectious material from birds in the outbreaks concerned into the wind-pipe of birds susceptible to laryngo-tracheitis.* In one outbreak a severe mortality (over 20 per cent.) occurred with typical symptoms of infectious catarrh. It was impossible to transmit this disease by taking the nasal and tracheal discharges and placing them in the trachea of experimental birds. (Laryngo-tracheitis transmission readily takes place by this means.) In a number of outbreaks birds recovered from symptoms of infectious catarrh have been infested with laryngo-tracheitis, showing that they could not have been previously affected with the latter. In a number of cases in the field, birds which have recovered from an outbreak of laryngo-tracheitis have been later affected with a clinical outbreak of infectious catarrh. In one case it has been shown that birds affected with infectious catarrh, are, on recovery, still susceptible to laryngo-tracheitis, as demonstrated by developing egg membrane technique.† Frequently it has been found that this condition can be transmitted by intra-nasal instillation into susceptible birds, but that after several passages the disease tends to die out, and cannot be kept going by any means of transmission (such as intra-tracheal). Thus, it will be seen that the evidence, which is not final proof at present, points to the presence of a separate cause for this disease which so closely resembles a mild attack of laryngo-tracheitis.

(*b*) *Predisposing Causes.*—The same causes as predispose to coryza are operative in the case of infectious catarrh. For this, see page 69.

*This laboratory work was carried out by L. Hart, B.V.Sc., of the Glenfield Research Station.

†Unpublished data of work done by L. Hart, B.V.Sc., Glenfield Research Station.

Infectivity—

The mode of infection would appear to be by direct contact, *e.g.*, affected birds coughing over their neighbours on the perches at night, or by means of feed and water trough transmission. Fowls would appear to be susceptible at any age, but outbreaks are perhaps most common when birds are from three months to one year old.

Immunity—

In some cases one outbreak appears to confer an immunity for the rest of life, as in the case of laryngo-tracheitis, but in other cases birds seem to suffer from recurrent attacks, as is the case in coryza.

Course of the Disease—

The course is a fairly protracted one and varies from about three weeks to two months.

Symptoms—

The symptoms as seen in mild or sub-acute and chronic cases of laryngo-tracheitis are in evidence. Affected birds show discharge of thin mucus from the nose and eyes. This thickens and forms cheesy membranes on the mucous membranes of the mouth, nose, throat and larynx, as well as in the infra-orbital sinuses. Coughing, and gurgling respiration frequently occur.

Post-mortem Appearances—

On post-mortem the same lesions as in chronic laryngo-tracheitis are seen.

Differential Diagnosis—

In an outbreak of disease which resembles both mild laryngo-tracheitis and infectious catarrh, differential diagnosis depends on laboratory examination. It should be particularly noted that, on clinical grounds the disease is clearly marked off from the peracute form of laryngo-tracheitis because the birds do not cough out blood or blood-stained mucus. Similarly, infectious catarrh may be differentiated from coryza in that cheesy membranes (often referred to as "canker" or "diphtheritic roup") occur in the former but not in the latter.

Pathogenicity—

The mortality rate is not usually heavy, although it may be serious when the disease follows on an outbreak of green feed deficiency disease. In one such case more than 20 per cent. of the affected birds died. This was in addition to the losses some weeks previously, due to green feed deficiency disease. Usually losses vary from 1 to 5 per cent. as compared with laryngo-tracheitis where normal losses vary from 10 to 35 per cent. The main loss from the disease is the diminution in egg production which is fairly marked in most cases.

Control—

Up to date no satisfactory means of vaccination or other technical control has been worked out, and control measures should therefore be directed towards the elimination of all predisposing factors of

disease, mentioned on page 7, particularly with reference to providing ample ventilation, green feed and the elimination of a concurrent worm infestation.

Home Remedies—

Home Remedies.—Perhaps there is no disease in which home remedies are more employed by the average farmer than in the treatment of infectious catarrh, but it is regretted that to date there is no evidence that any of these medicaments are of marked value in controlling the severity or limiting the mortality from the disease. On the other hand, many of the medicaments used may be definitely harmful.

Coryza.

Under this heading is included the types of "roup" often referred to as "colds."

The term implies a catarrhal disease in which the prominent symptom is a "running" of tears or thicker mucus from the eyes and nose, coughing and sneezing, without the formation of any cankerous (cheesy) deposits and without any marked inflammation of the windpipe.

Identity of the Condition—

From overseas reports it would appear that several separate causal micro-organisms may operate to produce disease symptoms which bear a very close similarity to one another and thus it may yet be found here, as overseas, that the disease coryza is a disease group.

Common overseas names for diseases grouped under this heading are:—Rhinosinusitis, coryza of slow onset, coryza of rapid onset, Nelson's disease, infectious rhinitis.

Seasonal Incidence—

Outbreaks of the conditions are seen at any time of the year, but are extremely common in the late summer and in autumn—about the end of January to March.

It is suspected on clinical grounds that one type has a strict seasonal incidence to this period and that the outbreaks in other parts of the year are due to other types of infection, as mentioned above.

Cause—

(*a*) *Actual Cause.*—Little precise work on the cause of coryza has been carried out in this State. As regards the group of "roup" diseases, the first three—fowl pox, green feed deficiency disease and laryngotracheitis—are the only ones of which the cause has been satisfactorily investigated and finalised. In America it is only in about the last ten years that careful work has been done as to the cause of coryza, and at the moment there is no finality in scientific literature. Thus, in the United States four separate causes have been mentioned as being incriminated in particular outbreaks, as follows:—

(1) *Haemophilus gallinarum*, a gram-negative bacillus, has been shown to cause symptoms of coryza which occur after a relatively short incubation period of up to a week, and this disease, in common with

the others, can be transmitted from bird to bird by intranasal injection. *Haemophilus gallinarum* has certain features in common with the human "influenza bacillus."

(2) *Coccobacilliform bodies,* which are larger than virus particles but smaller than most common bacteria (they are held back by bacterial filters, however), are the cause of a coryza of relatively slow onset (a fortnight or longer incubation period).

(3) *A virus* with particles so small that they pass through bacterial filters. It is not clear whether the disease caused by this virus is a separate entity from the disease referred to here as infectious catarrh.

(4) *Various other bacteria* have been incriminated as the cause of some outbreaks of coryza; among these may be mentioned *Bacillus coli, Shigella nasalis* and *Streptococci.* It is probable that in most cases these organisms are secondary invaders which invade tissues already weakened by the organisms mentioned above.

(b) Predisposing Causes—

These are of the utmost importance in determining the onset of an attack of coryza. In fact, where birds, particularly late hatched pullets are over-crowded an attack of coryza is *almost certain,* whereas, when affected birds are placed out and forced to roost in trees, the attack may clear up. Reference should be made to the predisposing causes listed on page 7, and particular stress should be laid on the provision of ample air circulation, green feed supply and the elimination of worm infestation.

Bacteriology—

Much confusion has resulted overseas in the investigation of the cause of this disease, because *Haemophilus gallinarum,* (perhaps the commonest and most active cause in this State) will grow only in media which contain blood and must be freshly transplanted each fourteen days.

The coccobacilliform bodies, referred to above, can be grown on developing egg membranes, and actually one strain was transplanted 100 times on such developing membranes at one to three day intervals.

None of these organisms forms spores and they are therefore quite susceptible to the usual disinfection measures.

Infectivity—

In most cases, infection can be readily transmitted from bird to bird by taking nasal discharges and injecting them into the noses of other birds. Clinical observations would seem to indicate that the disease spreads rapidly by direct contact, affected birds coughing over those uninfected. Fowls of all ages appear to be susceptible, but birds from four to nine months seem to show the greatest susceptibility. Ducks, turkeys, pigeons, sparrows and crows are not apparently affected with the condition.

The question has been raised by one American observer as to whether *Haemophilus gallinarum*, the common cause of coryza, is not also the cause of sinusitis in turkeys (see later).

Immunity—

When birds have been affected with the disease they do not develop immunity, as is the case in laryngo-tracheitis, but appear to be susceptible after a very short period of time. Thus it is frequently noted where particular birds which are affected are ringed, they may recover, remain normal for perhaps two weeks, then contract another infection from birds still affected in that yard, and suffer from another outbreak of the disease. In this way a bad outbreak of coryza may continually recur in a flock for many months, and it is frequently seen continuing on from December or January till about the end of May, when the incidence of cold weather sometimes has the effect of checking the epidemic.

Symptoms—

The first symptom noted is the discharge of mucus from the eyes and nose. If the lower corner of the eye is pressed with the finger foamy mucus may be pressed up into the eye. This mucoid material may gradually thicken until it attains the consistency of fluid glue, but seldom, if ever, does it develop into cheesy deposits. At this stage the bird frequently blows bubbles at the nose, this feature being more particularly observed at night when the discharges are more copious. Sneezing is a prominent feature, more particularly at night. Apart from these symptoms, little abnormality is noticed. Inappetence is frequently noticeable at first, followed by emaciation. In some severe cases the infra-orbital sinuses (the cavity below the eye and connecting with the nose) may become distended with fluid, and there may be an oedematous (doughy) swelling of the structures of the head. On rare occasions it would appear that infection with various secondary invading organisms causes quite a range of other symptoms. Thus, for example, in the bird whose resistance has been lowered by coryza, other organisms may infect the air sacs of the body and produce inflammation there.

Post-mortem Appearances—

A mild inflammation of the eyes and nose, the sinuses, and on rare occasions congestion of the trachea, are in evidence. Usually, apart from this, nothing of note is seen.

Differential Diagnosis–

It will be observed that the absence of cheesy deposits on the mucous membranes of the mouth and nose mark the condition off from infectious catarrh and laryngo-tracheitis. The absence of fowl pox warts would differentiate coryza from that form of fowl pox (unless, of course, fowl pox occurs concurrently). Critical differential diagnosis by laboratory methods is always advisable in cases of doubt. It is pointed out, however, that this condition comprises a well-defined and easily recognised disease which is widespread in most of the poultry flocks in the commercial poultry farming areas in this State.

Pathogenicity—

In most outbreaks of this disease no deaths occur at all. The loss due to decreased egg production is the most serious feature, as frequently an outbreak will cause a reduction of from 20 to 30 per cent.

Control—

Unfortunately, there is no successful vaccination which can be recommended for this disease. As in the case of infectious catarrh, the only means of control is to avoid all factors which predispose to disease. Recently it has been suggested that a deficiency of calcium salts will markedly lower the resistance of birds to this particular disease. A deficiency of calcium salts is always to be avoided. Calcium in this State is mainly supplied by making adequate supplies of sea-shell grit available at all times to the birds.

Home Remedies—

The use of Argyrol, 15 per cent. or Protargol, 5 per cent. (colloidal silver preparations), or zinc sulphate, 2½ per cent. (*i.e.*, ½ oz. zinc sulphate to one pint of water), is of some use in checking the eye discharges and may prevent eyelids being glued together. These materials should be dropped into the eye at frequent intervals.

Such materials as eucalyptus or even kerosene when smeared around the nostrils may act as expectorants, *i.e.*, they aid the mucous secretions. It is held by some that they assist in the course of the disease, but there is some doubt about this.

Many owners claim great success from giving Douglas Mixture in the drinking water, others from the use of a very weak solution of copper sulphate in the drinking water, while others claim that the feeding of chopped-up onions in the mash is a cure. There is no reliable evidence that these practices are of material benefit when a correct ration balanced in all respects is used. No harm can arise from their use as generally employed, *e.g.*, when copper sulphate is used, usually 8 oz. are dissolved in 1 gallon of water, and one tablespoonful of this solution is placed in each gallon of drinking water.

Various proprietary remedies are on the market, for some of which extravagant claims are made. There is no basis for such claims, but some drugs which act as general diffusible stimulants and expectorants may hasten the course of the disease, and their use, therefore, may be of some small help but not sufficient to justify paying exorbitant prices for them.

Conclusion—

To review the disease from the practical poultry farming standpoint, serious mortality never occurs, but the disease is a most annoying and costly menace to a large number of poultry farmers. The only practical method of dealing with the disease is to avoid errors in feeding and management, and where the disease occurs, careful nightly examination should be made during the first fortnight, and every bird coughing should be removed to a separate pen. Ample green feed should be fed, and, if a worm infestation is present, this should be rectified at once by dosing with a reliable worm medicine (such as carbon tetrachloride—see later).

Sinusitis.

General—

This disease is extremely common in the turkey raising districts of New South Wales and appears to have been present for many years, causing serious mortality. It is also known as "swelled head," and as turkey "roup." With the possible exception of blackhead, it is probably the most important disease affecting the turkey industry in this State. Unfortunately, there appear to be very few and scanty references in the literature relating to the condition, and if conclusions can be drawn from overseas literature it would appear that this is one of the very few diseases of birds which occurs widespread in this country and not in other countries to any marked extent.

Cause—

The cause of the disease is unknown, and as yet, only limited investigational work has been carried out locally. Probably the cause is a virus, but so far this has not been conclusively demonstrated.

Two forms of sinusitis are described in America:—

(*a*) *Nutritional* due to deficiency of Vitamin A (which is supplied by green feed).

(*b*) *Infectious.*

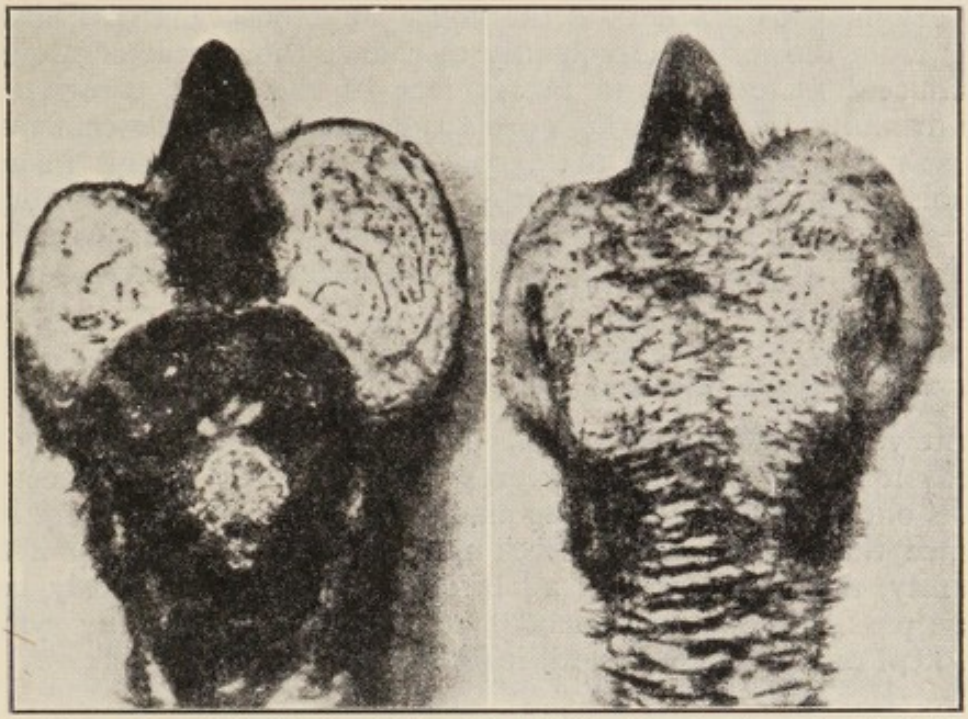
It is claimed that whilst these are quite separate, the symptoms are the same. However, from descriptions given, apparently the greater proportion of cases in the nutritional type develop cheesy flakes, in the sinuses, rather than the mucoid material.

It has been noted that the nutritional type occurs where turkeys are subjected to moderate deficiency rather than a complete absence of Vitamin A, as in the latter case the birds die before swelling of the sinuses occurs.

It would appear that, quite apart from the nutritional deficiency disease, deficiency of Vitamin A may be a predisposing factor to the infectious type of sinusitis. In one investigation overseas, approximately half of the outbreaks were in flocks in which there was a slight to moderate Vitamin A deficiency.

In America, it was noted by one investigator who was working on coryza in fowls that infection of turkeys with the germ *Haemophilus gallinarum*, which normally causes coryza in fowls, also produced a swelling of the sinuses in turkeys, and the symptoms described closely resemble the common condition seen here. It has been stated in America that the nutritional type and the contagious type of the disease are indistinguishable.

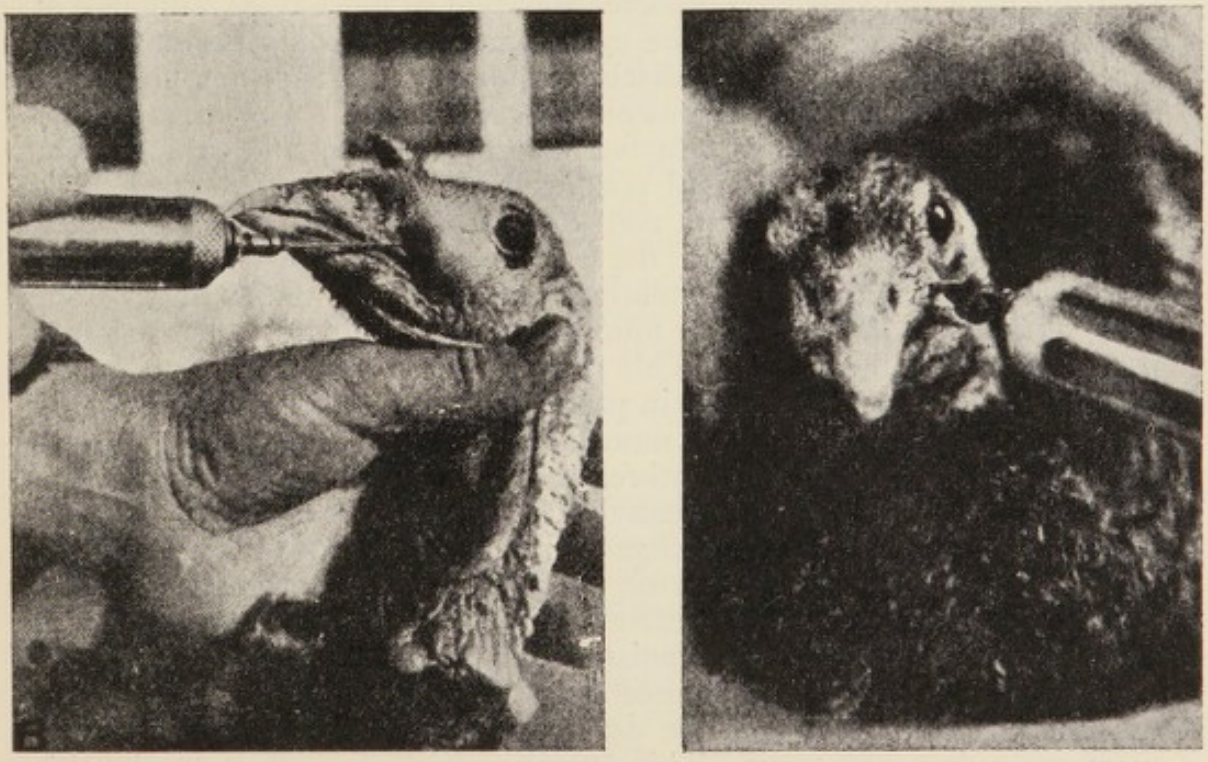
In New South Wales, the disease has occurred in a severe epidemic form amongst the turkeys on numerous holdings when there has been abundance of high quality green feed, and, further, fowls running in conjunction with the turkeys showed no symptoms of coryza. The field evidence suggests that the disease is one peculiar to turkeys and is of a highly infectious nature. In Utah (United States of America), it has been stated that the disease is contagious but only slightly so.



An Extreme Case of Sinusitis in a Turkey Hen Suffering from A-avitaminosis after being Fed for Eight Months on a Ration Containing a Low Level of Vitamin A.

The picture on the left is a sagitta section of the head shown on the right. Note the massive accumulation of the whitish-yellow caseous exudate typical of sinusitis as associated with A-avitaminosis in turkeys.

After Hinshaw and Lloyd.



Left.—**Inserting Needle into Sinus Preparatory to Removal of Exudate.**

Right.—**Sinus after Exudate has been Drawn into Syringe.**

After Mardsen.

Infectivity—

The mode of infection would appear to be by direct contact, affected birds coughing over others in the flock. It is observed that birds suffering from the disease frequently discharge the characteristic opalescent mucoid material on to the surface of the water trough or dam when drinking. Other turkeys are attracted by this opalescent material, at which they proceed to peck, and this appear to result in a rapid transmission of the disease from bird to bird. It has been found by overseas workers that, if infectious material is taken and injected into the sinus of a healthy bird, the disease develops in from one to ten days.

Age Incidence of the Disease—

Quite young poults (three weeks old) have been seen with the disease. Usually it is observed in its severest form in birds from three to five months old. From observations made by various owners, there is some evidence that birds once affected with the disease develop a lasting immunity, as it appears that all birds which have previously had the disease pass through subsequent epidemics without showing symptoms when kept in close contact with infected young stock.

Symptoms and Course of the Disease—

Typically turkeys about three to three and a half months old are affected and first show listlessness and mope about, obviously feeling sick. These birds shake the head frequently and may be noted to swallow from time to time. Shortly after this they are observed sneezing, and some scratch at the nostrils occasionally. A mucoid discharge from the nose and eyes then becomes apparent in most cases, although at times the lachrymation from the eyes is absent and only the nasal discharge is in evidence. These discharges are at first thin and clear, but thicken slightly and become mucoid in consistency. The infra-orbital sinus, which is the cavity situated under the orbit and which communicates with the mouth and nose, then becomes puffed out. This enlargement is quite obvious, and comprises a puffy swelling which involves the entire area below the eye, and as far down as the beak. The distension is soft and fluctuating, moving freely under finger pressure. Often on manipulation, mucoid material will be expressed into the nose or into the mouth. This material is, at this stage, opalescent and about the consistency of thin paste. In many outbreaks the material does not thicken but remains mucoid until the bird wastes away and dies, or recovers. In other outbreaks (which are the exception rather than the rule) it is noted in many birds affected that after a week or two, the mucoid material thickens and develops into a cheesy mass which forms a cast of the sinus concerned. It is probable that the development of this cheesy material depends upon the presence of secondary invading micro-organisms which affect the mucous membrane of the sinus, the resistance of which has already been lowered by the sinusitis infection. The development of the cheesy material in most cases does not occur. This forms a marked contrast to sinusitis in fowls, where inflammatory material produced by such diseases as laryngo-tracheitis always becomes cheesy.

The bird wastes away apparently from the combined effect of an indifferent appetite and absorption of toxic materials from the inflamed sinuses, though head pains, bossing by other birds, and defective eyesight, may play a part. Gurgling and snuffling sounds are sometimes made by the affected bird, and coughing is frequently in evidence. Usually coughing is a more frequent symptom during the early part of the disease. The symptoms which are exhibited at the time when the bird dies are not such as would be thought sufficient to cause death. If the bird does not die in from twelve to eighteen days after the onset of symptoms, recovery is gradual and is often complete by the fourth or fifth week. Nothing definite can be laid down as to this, however, as in some cases birds have continued to show symptoms for months. In most cases the affected birds are stunted and do not catch up again in general constitutional vigour to the unaffected birds. Recovery of birds in this State seems to be much more common than in the United States of America, where it is reported that losses are usually 80 to 100 per cent. of affected birds which are left untreated.

Diagnosis—

The diagnosis of the disease is based upon the clinical appearance, as there is no other disease condition which it closely resembles. Fowl pox in turkeys produces warts on the unfeathered portions of the head as well as cheesy membranes on the mucous membrane of the mouth, but these symptoms do not resemble sinusitis at all.

Differential Diagnosis—

As already stated, it has not been conclusively shown that this disease is not due to deficiency of Vitamin A complicated by secondary invading micro-organisms, or on the other hand that it is not caused by the *Haemophilus gallinarum* (the cause of "colds").

However, in the case of green feed deficiency, the *Bursa Cloacae* (of Fabricius), which in young birds is a pouch opening into the cloaca, will be thickened and distended with flaky whitish deposits. In this condition (deficiency of Vitamin A) there may also be cheesy material in the larynx and windpipe, and in some cases yellow pimples in the mouth, top portion of the gullet and sometimes even down as far as the crop. Enlargement of the proventriculus (first stomach), with a milky exudate of its glands, is also sometimes present in green feed deficiency disease.

Post-mortem Appearances—

Inflammation of the mucous membranes of the infra-orbital sinuses (also known as the para-nasal sinuses) is present, but is not as marked as would be anticipated. A large excess of mucoid material is present in these cavities. In a small proportion of cases (say, about 10 per cent.), this material may be cheesy. Usually, no other abnormalities are noted. In some cases the eyes and nasal cavity are also inflamed. On several occasions inflammation of the larynx, which inflammation extends for a small distance down the trachea (windpipe) has been seen, but this is not a constant or typical lesion. In cases of birds which have died from the disease, it has been noted that the heart muscle is flabby and in some cases the liver has been pale. The exact

significance of these changes is not certain, but it would appear possible that they are due to absorption of toxic products from the inflamed sinuses, or from the inflammatory exudates that are swallowed.

Pathogenicity—

The death rate varies enormously. In some outbreaks all the affected birds die. This is particularly so when very young poults are affected by the disease. In one case where over 500 poults were affected all died, and in another case of over 800, more than 90 per cent. died. Other outbreaks have been seen in which there was no mortality, but the birds remained chronically affected for months. In such cases the disease prevents the birds from developing and maturing with a suitable bloom for market requirements. Affected birds may gain less than half as much as unaffected birds from the same batch.

It would appear probable that the death rate may be largely influenced by other factors such as the green feed supply or mineral deficiency.

Control—

Great care should be taken to avoid the introduction of this disease by bringing carrier birds into a flock which has been free of the disease. Inquiry should be made as to the freedom from disease of the flock, from which the introduction is intended, and, if possible, all new introductions should be kept isolated for a month.

At the moment, there is no effective means known of immunising the birds against infection (though bacterial vaccines are in common use in America). Nor is it known whether any particular strains of turkeys are more resistant than others, but, from field evidence, it is strongly suspected that this may be the case.

When an outbreak occurs ample green feed and minerals should be provided. Three per cent. of bone-meal in the mash and ample shell-grit will supply the necessary minerals. If green feed is not available, 1½ pints of cod liver oil per 100 lb. (dry weight) of the mash should be fed each morning.

Treatment—

Various treatments have been tried, but, although control groups were arranged, the local experimental and field evidence is not by any means conclusive, though it bears out oversea observations. In one case, it was found that the injection of 1 per cent. silver nitrate into the swollen sinuses gave much better results than injection of 1 per cent. copper sulphate or zinc sulphate. Further trials* with silver nitrate indicated that the efficacy of the drug increased if 2 per cent. were used, and best results were gained with 5 per cent. (5 per cent. is 1 oz. to the pint of water; similarly, 1 per cent. is 1 oz. to 5 pints, or equivalent proportions). Favourable reports have been received of the use of injections of a 15 per cent. solution of Argyrol, but this is expensive. If injections of 5 per cent. silver nitrate are used they sometimes result in the temporary acute inflammation of the sinus and possibly sloughing of its walls. If this treatment is used the inflammatory material should first be gently squeezed out of the sinus.

*This trial was supervised by E. G. Macdonald, B.V.Sc., District Veterinary Officer (West), N.S.W.

When the material comes through the nose no special care is necessary, but if, as is usually the case, it squeezes into the mouth, the bird's head should be held depressed and the index finger of the operator's hand held far back in the mouth to prevent the inflammatory products being swallowed or breathed into the wind-pipe. The solution should then be injected into the sinus by means of a hypodermic syringe at the position that represented the centre of the swollen area before the inflammatory products were expressed. The treatment may need to be repeated three or four times at intervals of from five to seven days. An alternative method is to open the sinus by means of a sharp scalpel at the lowest point and syringe frequently with some antiseptic and astringent solution such as 5 per cent. silver nitrate or a strong solution of permanganate of potash.

It would appear that the treatments are most effective if carried out in the early stages of the disease. Whilst the treament may only be palliative, it is, however, remarkable that in some cases early treatment by injection appears to prevent a heavy mortality. But before a definite statement can be made, further carefully controlled observations will need to be made, and it is hoped that more precise information both as to the cause, prevention and treatment of this disease will be available in the near future.

In the United States, very favourable results from the use of silver nitrate have been recently reported. The method used is as follows:— The bird is held and a hypodermic syringe fitted with a No. 16 needle is inserted into the swollen sinus at the lowest point. All the mucoid material is then withdrawn and emptied into a bucket of disinfectant. The syringe is then fitted with a No. 18 needle, which is re-inserted through the same hole and 1 ml. of 4 per cent. solution of silver nitrate in distilled water (1 oz. to 1 pint=5 per cent.) is injected into the sinus, which is then gently massaged. This solution causes the death of the mucous membrane of the sinus. It is reported that only occasionally is a repeat treatment necessary.

Care must be taken to insert the needle into the sinus. If a blunt needle is used it may not penetrate the lining membrane of the sinus and the silver nitrate, if injected subcutaneously, merely causes a painful sloughing wound. This error, which has been observed locally, should be particularly avoided.

Other "Roup" Diseases.

Eye-worm (*Oxyspirura parvorum*)—

This is a nematode parasite which occurs in the eyes of poultry in the northern portions of Australia, although a few cases have been observed in this State (one as far south as Braidwood). The condition causes a discharge and inflammation of the eyes which is classed as "eye roup." So far, the disease is of no importance economically.

Gape-worm (*Syngamus trachea*)—

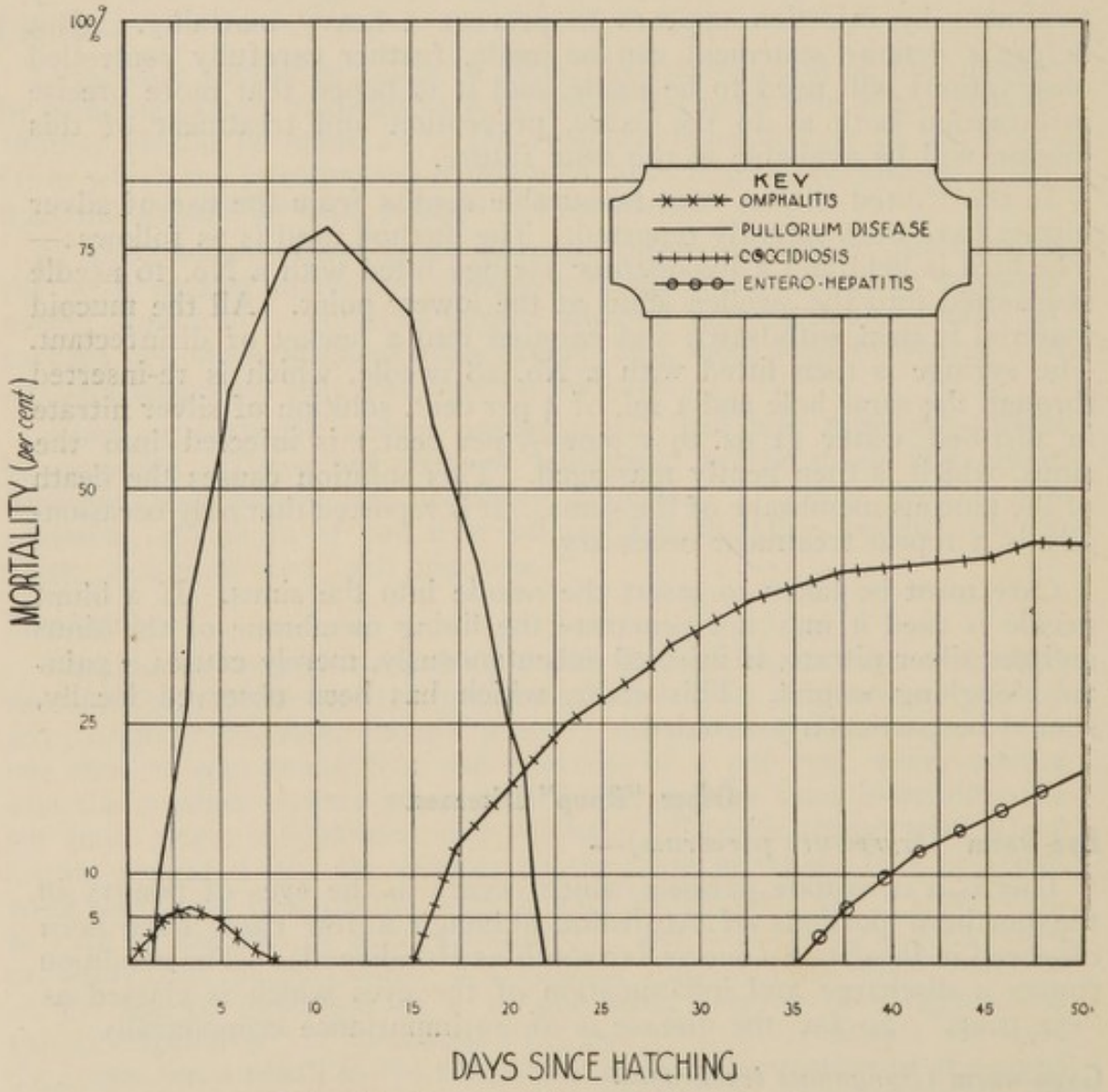
This is a worm parasite which occurs in the upper portion of the trachea and causes inflammation there which might be confused with other symptoms of "roup." As far as is known, it does not occur in this State.

DISEASES OF CHICKENS AND YOUNG STOCK.

Under this heading are included dieases which, for the most part, affect both young and adult stock, but in which the most severe, most usual, or most characteristic occurrence is seen in the former.

The diseases dealt with here are:—

1. Diseases due to faulty management.
2. Pullorum.
3. Coccidiosis.
4. Entero-hepatitis.
5. Omphalitis.
6. Mycosis.



Differential Diagnosis of Diseases of Young Stock.

It will be noticed in the above chart that diseases of chickens partly group themselves according to the age at which they occur. Thus navel infection is a disease of the first week of life, pullorum disease commences to cause mortality in the first week, in the second week losses are greatest and by the end of the third week mortality is at an end. Coccidiosis causes trouble mainly from the third week onwards, and entero-hepatitis mainly from the fifth week onwards.

Features of Clinical Differential Diagnosis—

The post-mortem appearances, and also the symptoms, characterise each disease fairly clearly. These lesions and symptoms are dealt with separately under each disease heading. However, apart from these, mortalities in chickens can partly be classified according to the age at which they occur. Thus, losses from pullorum disease usually commence in the first week of the chickens' lives and seldom continue beyond the third week. The heaviest mortality usually occurs about the tenth or twelfth day. In contrast to this, losses from coccidiosis do not usually commence before the end of the second week.

If a heavy mortality in chickens is experienced in the first ten days following hatching, and one does not suspect injuries sustained during sexing, or diseased navels, then the two most common causes of death are faulty brooding and infection with pullorum disease.

In the latter condition, if the chickens have been infected whilst in the incubator, the mortality typically commences on the second day. The death rate increases up to about the ninth or tenth day, and then decreases till about the twentieth day, after which few chickens die as a result of this disease.

In coccidiosis the mortality seldom commences before the twelfth day. (Mortalities have been seen in New South Wales much younger than this, but are very rare.) Usually it commences not earlier than the third or fourth week, and then continues for an indefinite period.

Blackhead (entero-hepatitis) has a somewhat similar age incidence, though most frequently outbreaks occur between six to ten weeks of age. It must be differentiated by post-mortem examination.

Losses from navel infection, or from sexing troubles, occur almost entirely in the first week (second to seventh day).

Diseases Due to Faulty Management.

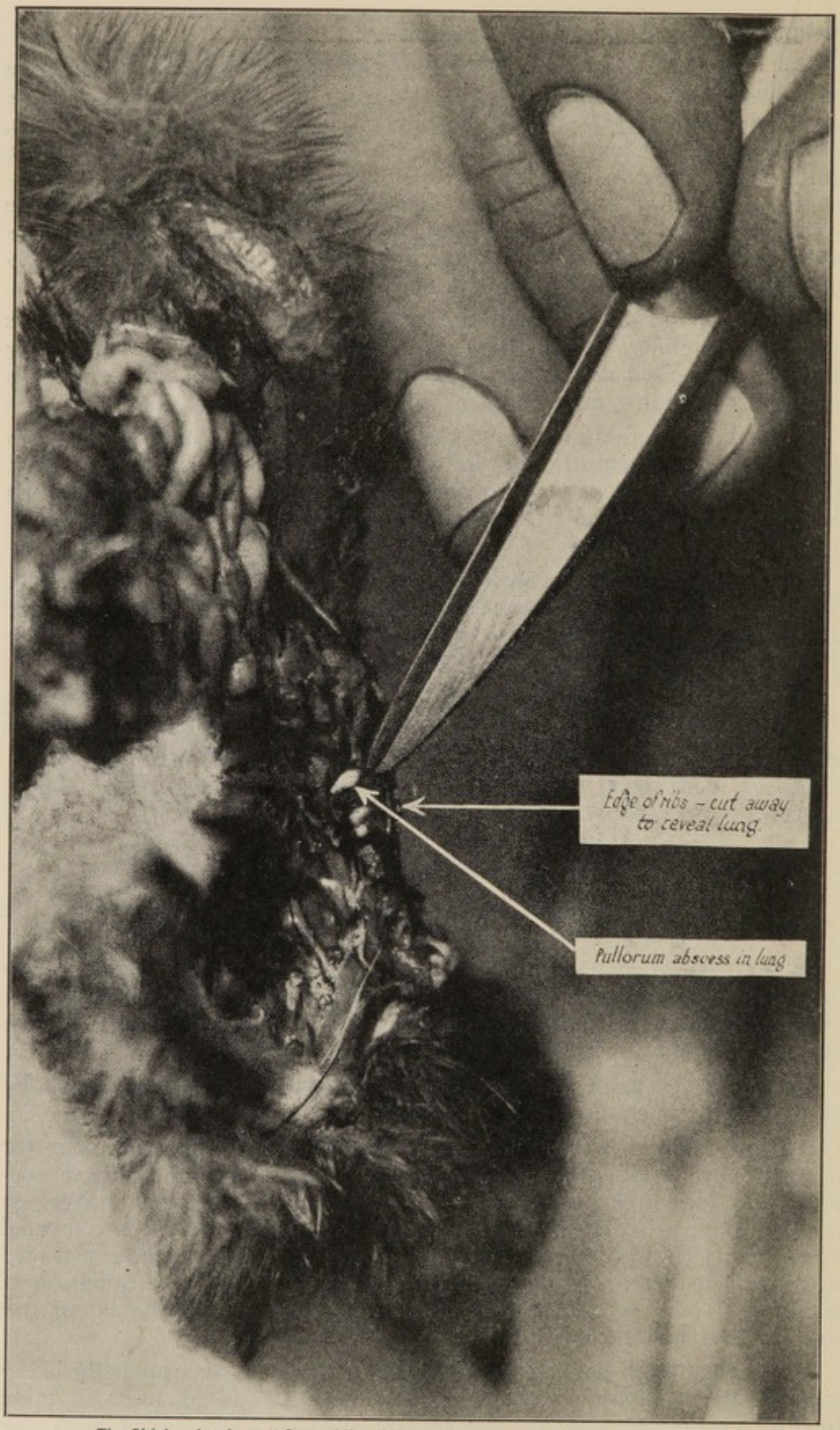
When mortality commences the owner sometimes jumps to the conclusion that they are suffering from some infectious disease.

In the case of an experienced poultry farmer who is careful to use brooding plant that he knows to be satisfactory, and who exercises every possible care with the feeding and general management of the chickens, deaths are usually the result of some disease.

On the other hand very many heavy mortalities are caused wholly or partly by faulty brooding methods.

In the artificial brooding of chickens heat must be maintained for the first few weeks after hatching. This is done by one of three systems: (i) Hot brooding, *e.g.,* waterpipe systems covered by hovers or hover boxes, or coke, coal, sawdust, kerosene, gas or electric heated hovers; (ii) cold brooding, where small numbers of chickens are placed in a semi-insulated compartment so that their body heat radiation is conserved; (iii) semi-heated systems, *e.g.,* where kerosene lamps are used to heat hovers under which the chickens nestle.

Large mammoth battery brooders are of the first type, usually being water-heated. Any one of the three systems can be used to raise healthy chicks, *so long as the correct measures are taken* with the



The Chicken has been " Opened " and the Viscera pulled aside to Expose the Lung.

One large abscess (actual size is that of a small wheat grain) can be seen just below the lower point of the scissors. Two other abscesses, close together, can be seen in the lung a little further forward. These abscesses are greenish-yellow, and are characteristic of pullorum disease.

particular system employed. (Full information concerning the correct brooding of chickens may be obtained from the Poultry Expert of the Department of Agriculture.)

Badly brooded chickens are weakly and are more susceptible to any infection with which they may come in contact. Apart from infectious disease, the chicks under very bad conditions of brooding may die from congestion of the lungs, indigestion with diarrhoea, suffocation and exhaustion.

Congestion of the lungs may result from severe chilling of the chickens, especially if combined with conditions of dampness. Dampness and cold usually go together, as for example where a brooder boiler fire has gone out the temperature of the brooders drops perhaps to 60 deg. Fahr. or less in a night in June. Droppings voided by the chicken are semi-fluid, and in a few hours the brooder floor is thoroughly damp.

Because of the cold, the chickens huddle together and where there are, say, a hundred in one compartment, they will crush into a space which normally would be occupied by ten or fifteen. The chickens in the centre and bottom of this huddled mass, sweat, gasp, become exhausted and finally die from suffocation. The farmer may come out, stoke up the fire, restore the heat, and so cause chickens to spread out again. Perhaps hours later he finds 10 per cent. of the chickens dead and may think that pullorum disease is responsible. It is here that veterinary advice should be sought to save unnecessary panic and to indicate the origin of the mortality.

Incorrect feeding methods have also caused disastrous mortalities. Here again is a case where competent advice should be sought before assuming that mortality is due to some specific infectious disease.

The subjects of feeding and brooding receive attention in the book "Poultry Farming in New South Wales," which is available from the Department of Agriculture, N.S.W.

When deaths occur in chicks, the farmer should satisfy himself that a standard ration that has given widespread satisfaction is being fed by the standard method. He should also make sure that the chicks are not overcrowded (due to too many at first, or to failure to thin down the numbers as they grow), underheated, suffering from faulty ventilation and stuffiness on the one hand, direct draughts on the other, or from fumes (*e.g.*, from heating lamp) or excessive heat from which they cannot withdraw, without obstruction.

Pullorum Disease.

(Once Known as Bacillary White Diarrhoea.)

Historical—

In the latter part of the last century this disease was found to cause serious mortalities in hen-hatched chickens and also in chickens hatched in the primitive type of incubator then in use. About 1900 to 1905 it was erroneously held that the disease was due either to faulty absorption of the yolk or to deficiency of carbon-dioxide in the atmosphere of incubator and brooder. Faulty brooding and filthy conditions

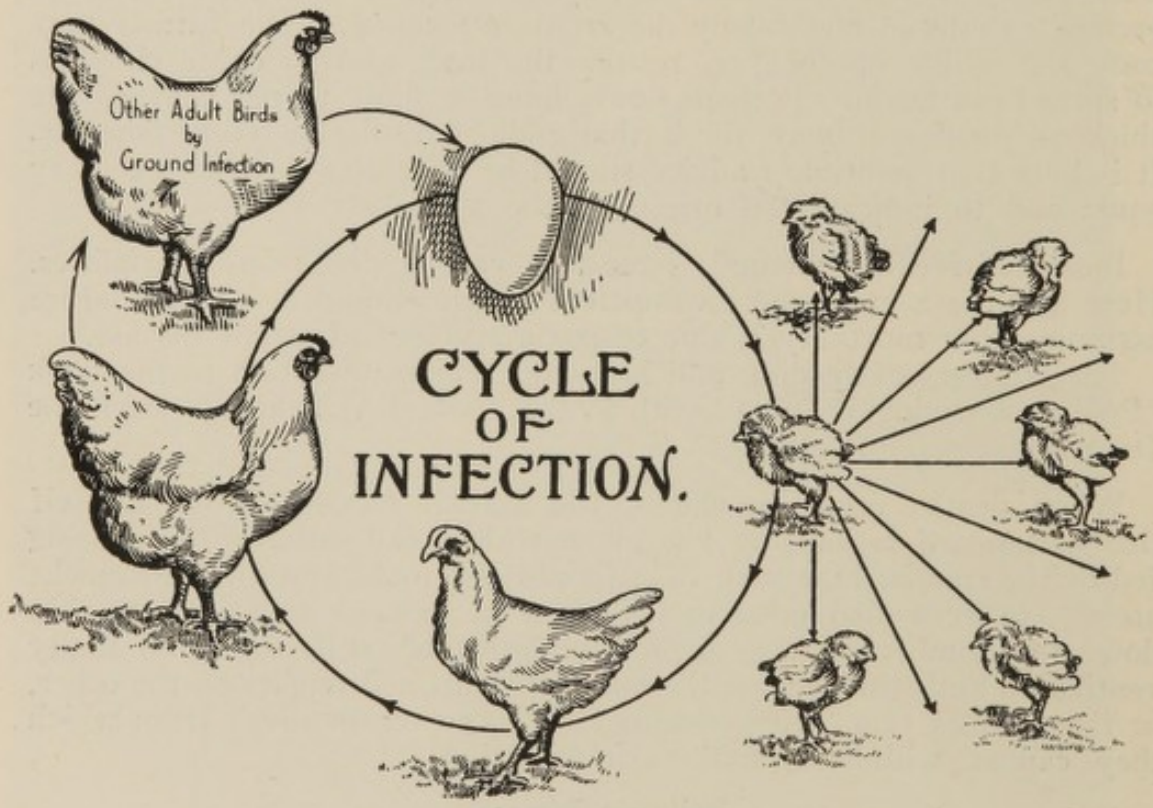
were also blamed. Many mortalities of up to 80 per cent. in large batches of hen-hatched chickens were reported, as well as in incubator chickens. *Bacterium* (now *Salmonella*) *pullorum* was described in 1900*. During the year 1900 to 1911 this germ was conclusively proved to be the cause, and in 1914 the first effort to eliminate the disease from a commercial flock was undertaken.

In this State, popular appreciation of the facts has been slow, and actually less than ten years ago doubts were expressed as to *Salmonella pullorum* being the sole cause of pullorum disease.

These doubts were shown to be without any foundation in fact, by carefully planned laboratory experiments conducted by the Department at Glenfield Veterinary Research Station. These experiments fully confirmed previous overseas findings. Quite recently the disease has been found to cause mortality in adult fowls. It has also been found to affect many other species of young birds.

Synonyms—

The disease is also referred to as bacillary white diarrhoea (B.W.D.) and pasted vent. It is also wrongly referred to as "brooder pneu-



Cycle of Infection in Pullorum Disease.

[After U.S.A. Dept. of Agri.

monia," chills, congested lungs, pneumonia, liver disease, cramps, and staggers. These last seven terms refer to other definite and distinct diseases which should not be confused with pullorum disease.

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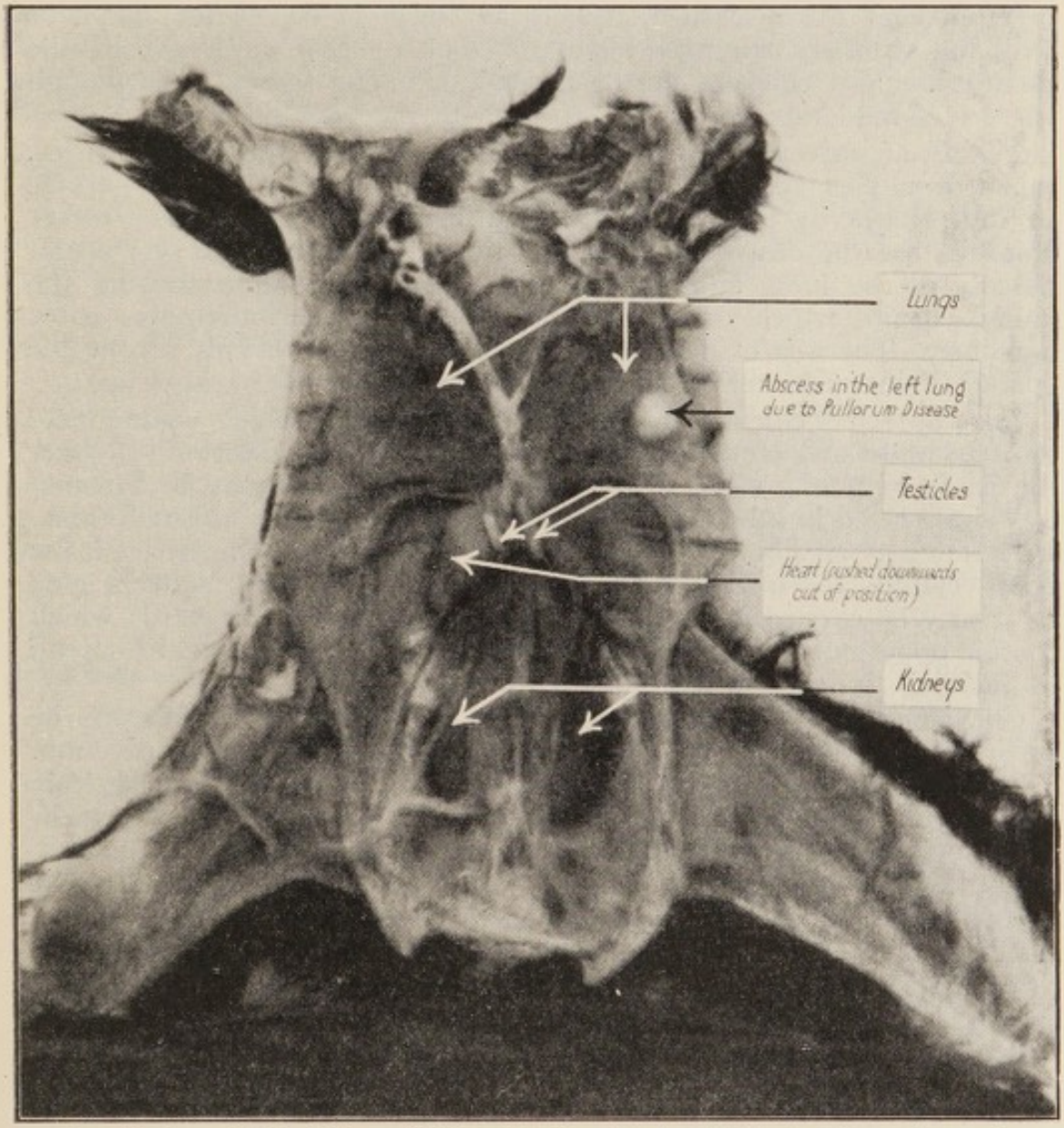
*For complete and full account of all matters dealing with pullorum disease up to 1932 (over 200 ref.), see Rettger, Leo F. & W. N. Plastridge, Bull. 178, Storrs Agr. Exp. Stat., Connecticut.

Cause—

The cause of the condition is a germ known as *Salmonella pullorum*. Without this germ the disease cannot occur. If the chickens are once affected with the micro-organism the mortality may be very heavy, quite apart from any fault in brooding methods.

Filthy methods, chills, over-crowding, fuming brooder lamps and draughts cannot cause the disease, though, if the germ of pullorum disease is present, these factors will increase the mortality. It must



An Eight-days-old Male Chicken with Ventral Portion of Body and Abdominal Organs Removed.
Note the yellowish abscess in the left lung. This abscess is characteristic.

be added, however, that under ideal conditions, mortalities of more than 95 per cent. have been seen. Of course, the other conditions, of themselves, may cause very heavy losses indeed, but they *cannot produce pullorum disease.*

Bacteriology—

The causal germ is readily isolated by laboratory culture from chickens which have died from the disease. The micro-organism does not form spores and thus is easily killed by any reliable disinfectant.

Infectivity—

How the Infection is Spread.—If the adult fowls in the flock are carriers of the germ (*Salmonella pullorum*) such breeding hens, *whilst quite healthy in appearance,* may lay a proportion—for example, 10 to 15 per cent.—of eggs which are infected with the germ. Fortunately, in most cases, these infected eggs do not hatch out. The fact that infected eggs are discarded, usually as "dead in the shells," explains why the hatcheryman may have only one or two outbreaks of the disease amongst chickens during a whole hatching season, even though there is a moderate degree of infection in the breeding stock.

Where an infected egg hatches out, the fluff on the chicken dries, much of it flies about in the incubator atmosphere, and each particle of fluff is heavily infected with the disease micro-organisms. Other chickens breathe down these fluff particles. Each particle sets up an abscess in the lung, and this abscess is instrumental in causing the chick's death, which occurs on the second to the fourth day after hatching. There is evidence that forced draught machines favour the spread of infection more than the still air machines.

Such infected chickens pass infected droppings, which contaminate the feed and water troughs and so rapidly spread the disease throughout the batch of young stock. A new batch of chickens may be infected by being placed in a brooder contaminated by an earlier affected hatch. Infection may also be conveyed from one brooder run to another on the boots, clothes and hands of the attendant, or by brooms, shovels, etc., or it may be carried in by the attendant's boots from the yards which contain apparently healthy adult carrier birds. Infection may be spread to ducklings by contact with infected chicks.

Chickens which "recover" from the disease remain carriers of the infection, may lay infected eggs, and so carry on the cycle of infection.

These features indicate the importance of destroying (where it is economically possible) all survivors of an outbreak of the disease in chickens and testing and removing all adult carrier birds.

As regards the adult bird, it has been shown, both overseas and locally, that if carrier birds are allowed to mingle with healthy adults the disease spreads slowly through the latter.

The infection may be introduced by mouth, into the eye, under the skin (e.g., of the foot) or into the oviduct. A cock bird infected in the testicles may play an important part in the spread of the disease.

Animals Susceptible—

Chickens, turkey poults, young pheasants and young quail are highly susceptible. Ducklings are also susceptible (though some authors have found certain breeds resistant). The germ has been recovered from sparrows and also from a bullfinch. Infected eggs when fed to 128 rabbits have caused the death of 125. Canaries and guinea fowl are susceptible, but pigeons appear to be resistant unless the germ is injected into their abdominal cavity.

Viability—

The causal germ has been shown to remain virulent in soil for fourteen (14) months.

Immunity—

It is well known that heavy breeds, such as the Australorp, Rhode Island Red, and Light Sussex, suffer more severely and are more commonly affected than light breeds, such as the White Leghorn. The disease occurs in all breeds, but it is unusual for mortalities of up to 90 per cent. to occur in batches of White Leghorn chickens, whereas such mortalities are by no means unusual in the heavy breeds once the chickens become infected. It is well recognised that the Mediterranean breeds possess a partial immunity or at least an increased resistance to the fatal effect of the disease in chickens.

Heredity—

There is good genetic evidence that increased resistance to pullorum disease may be an inherited factor, such factor being known (genetically) as "dominant."

Course of the Disease—

In chickens the course of the disease varies according to the mode of infection. Thus, where the egg is infected, the embryo may die during incubation. Actually the greatest mortality occurs on about the nineteenth day of incubation. If such embryos hatch, deaths may occur at any time after hatching, but in practice, only an occasional infected chick hatches out. If infection is contracted after hatching, mortality commences on about the fifth day and reaches its peak about the eleventh day.

Symptoms—

Frequently no symptoms are noted except that chickens die in large numbers. If careful observations are made, chickens may be seen to mope about, chirp, stand in a huddled position, show a certain degree of leg weakness, and in some cases a white diarrhoea, which pastes up the vent, may be in evidence. This "pasting" prevents the passage of further droppings. As a rule, the only prominent feature to the casual observer is that the chickens die. Close observation will detect affected chickens in the early stages chirping disconsolately, taking no food, and holding themselves in such a position that the back appears short, the legs longer than normal and the belly distended.

Post-mortem Appearances—

In many cases dozens of affected chickens may be examined post-mortem and no abnormalities may be detected by the farmer. Frequent lesions noted are small abscesses about the size of a pin's head in the lungs, and less frequently in the liver and heart wall. These abscesses are yellowish and sometimes greenish in colour. Their incidence varies. Thus some authors regard their presence as being typical of the disease. In one investigation where 270 chickens died, lung abscesses were present in forty-three and heart abscesses in four. Sometimes liver abscesses are most common.

It is frequently stated that the liver is yellow or cedar coloured. Actually this colour of the liver is quite normal for day-old chickens and the normal liver colour does not occur in young stock until the yolk is absorbed (after the sixth or seventh day). The absorption of the yolk in affected chickens may be delayed. The caeca or two "blind guts" are frequently filled with greyish soft to semi-cheesy material, which is not so hard as in the case of coccidiosis or blackhead. The liver may be mottled or of streaky appearance in some cases, and in some outbreaks is congested throughout.

Differential Diagnosis—

The main condition with which pullorum disease is liable to be confused is faulty brooding. When brooders are run at temperatures which fluctuate greatly, or which are too low, or when too many chickens are crowded into a small brooder, the young birds may huddle together and many of them will suffer from congestion of the lungs. The only certain method of differential diagnosis, in some cases, is to forward freshly dead chickens to a laboratory where bacteriological culture methods will detect the causal germ of pullorum disease if this condition has actually caused the death.

Pathogenicity—

Mortality in chickens varies from 5 to 100 per cent. In heavy breeds it would average about 60 to 95 per cent. In Mediterranean breeds 10 to 20 per cent. is a typical mortality when the disease occurs. "Recovered" birds are frequently poor-doers, and remain unprofitable. In the adult variable loss occurs, due to infection of the ovaries and consequent diminution in egg production. If the ovary is only slightly affected, there may be no appreciable decrease in egg production. In one comprehensive investigation the following figures were given:—

	Percentage		
	Production.	Hatchability.	Viable Chicks.
Infected hens	37·37	35·9	22.4
Non-infected hens	45·46	43·1	94.02

Many authors give similar figures. Local experience has demonstrated that old chronic carriers which have been affected with the disease for years show very markedly lower production than their uninfected fellows of the same hatch. In first-season pullets the difference in production between carrier and healthy stock is not nearly so striking.

Cure—

There is no cure for the disease and poultry farmers should be careful to note this, so that money is not wasted on various proprietary "cure-alls."

Control—

The only satisfactory way to control the disease is to test all the adult stock and remove the carrier birds. This must be repeated at intervals, so that the birds which are re-infected from micro-organisms picked up from the soil can be eliminated.

Various Agglutination Tests—

There are quite a number of different techniques employed in applying the agglutination test for the detection of carriers of pullorum disease.

Testing Outside a Colony Shed Under Field Conditions.

The two methods in most common use are the "tube" agglutination test (carried out in the laboratory) and the "rapid whole-blood" test as described below. Of the two, the "tube" test is the more accurate as the exact proportions of antigen and serum are precisely measured, placed up in a range of different dilutions, and kept at an even temperature for twenty-four hours. Details of this test are not given here

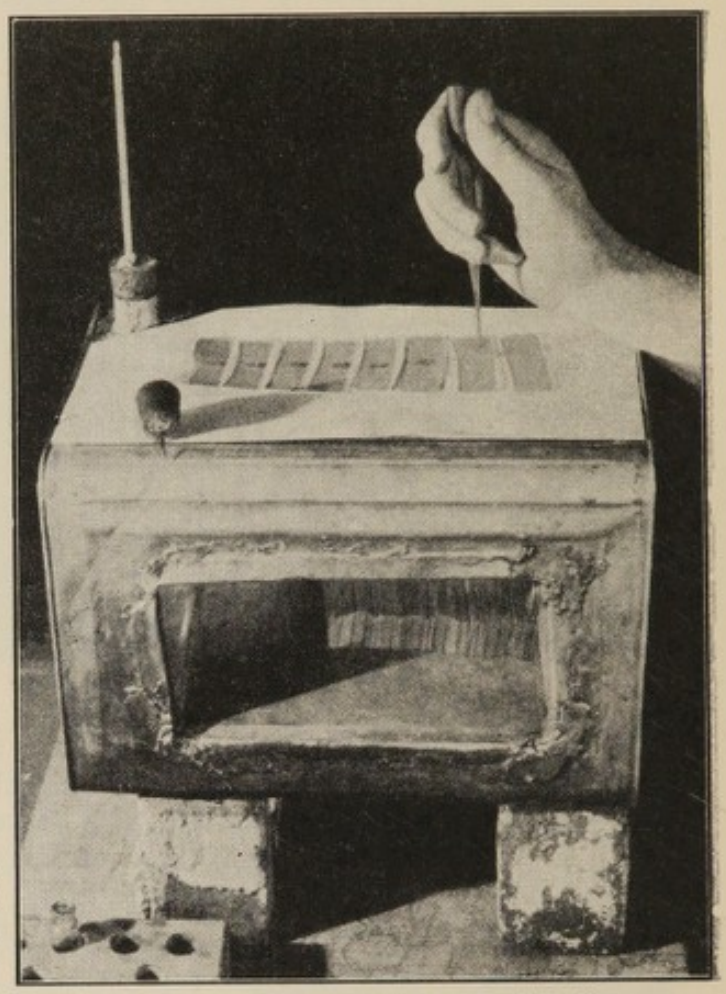
Reading the Reactions.

as it is a laboratory matter. For the test, blood from the birds is collected into small bottles, and the bottles are labelled to correspond with the number on the bird's leg- or wing-band.

Technique of Collecting Blood—

The birds are best crated or otherwise confined. The operator, for convenience, is seated. The attendant holds the fowl upside down with its breast facing the operator, who takes its left wing over his

right knee (for a right-handed person). About an inch below the elbow joint, a few feathers are plucked away, thus disclosing the blood vessels running along the underside of the wing. A very sharp scalpel (with a smooth shaving edge) should be used to stroke lightly across these blood vessels at right angles. If any pressure is exerted, usually both the vein and the artery are incised. This is undesirable. By carefully and lightly stroking the scalpel across the blood vessels, it is possible to incise the vein only. The blood drips freely from this and is collected by placing a two drachm ($\frac{1}{4}$ oz.) bottle at the lower end of the incision. About half to two-thirds of the bottle of blood is collected and the bottle placed on the slant. Later the bottle of clotted blood may be transported direct to the laboratory, or it may be allowed to stand until the serum separates off, in which case the latter is poured into another bottle, labelled and forwarded to the laboratory.



Apparatus Used for the Whole Blood Agglutination Test.

Antigen is being dropped on to the glass slides. Other slides are in the oven, and the cork, into which the needle and the wire loop used for collecting the blood are inserted, is shown on the top of the oven.

The Rapid Whole-blood Test—

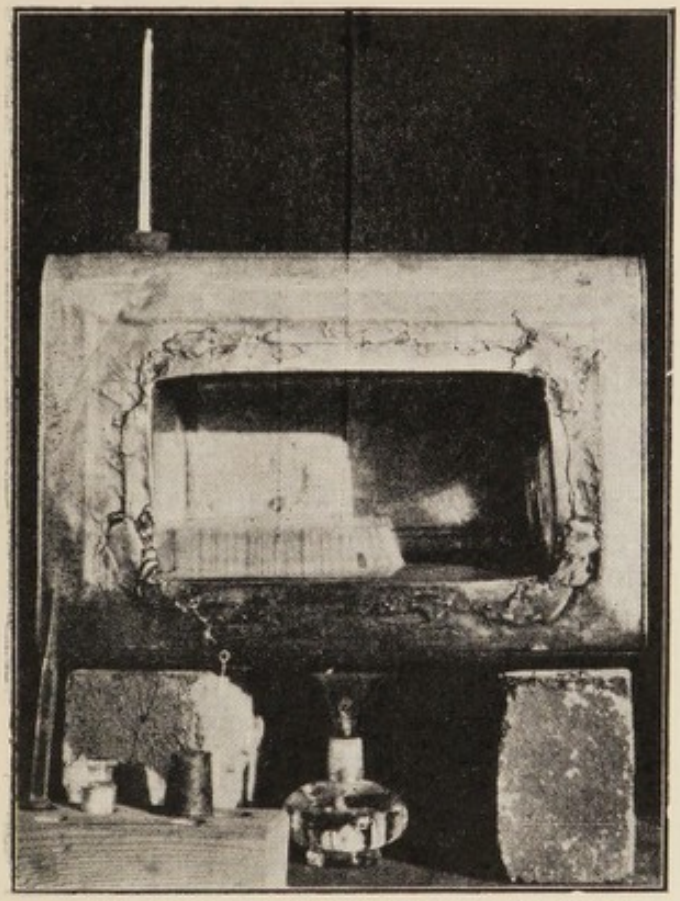
Undoubtedly the "tube" agglutination test is the more precise, as the exact quantities of antigen and serum and the temperature can be controlled. However, the "tube" test is necessarily expensive and the collection of blood checks egg-laying to some extent. On the other hand, the "rapid whole-blood" test is reasonably efficient when carried out by a proficient operator. Most poultry farmers who have been shown the method can carry it out. Some farmers, however, can never become proficient, because of faulty eyesight or other disabilities. One case was noted where a very careful poultry farmer had eliminated less than 20 per cent. of birds as positive reactors from his flock. Retest by a departmental officer of the "negative" birds, showed that more than 50 per cent. of these were positive reactors which had been missed by the farmer. It is particularly stressed that a farmer should attend a demonstration of this test before he attempts to test his own birds.

Further, it is stressed that any apparatus which does not ensure an even temperature and satisfactory mixture of the antigen and blood is sure to give very unsatisfactory results—and it is regretted that often such equipment has been purchased by poultry farmers.

If testing is to be carried out, the poultry farmer should commence in time to test all the breeding stock, so that eggs may be collected from negative reactors only.

How the Test Acts—

In many cases, when a bird or animal is affected with a particular disease-producing micro-organism, the blood of that animal or bird develops substances, known as "antibodies." These "antibodies" act upon the disease-producing micro-organism so as to bring about its destruction. One method of doing this is to cause the disease germs to agglutinate or clump together after which they are destroyed by the action of the white bloood corpuscles (leucocytes).



Glass Slides being Heated in the Improvised Water Bath Oven.

Note the spirit lamp and the thermometer through the cork.

In the case of pullorum disease, after the blood of an affected bird is taken and mixed with the antigen, which is a suspension of *Salmonella pullorum* (the micro-organism which causes pullorum disease), these micro-organisms will be clumped together. Should the bird not be affected the germs will not be clumped together or agglutinated.

To enable one to read this reaction more clearly, the micro-organisms are stained purple.* It is, therefore, apparent that the clumping together of small purple masses after mixing the blood and the suspension of the micro-organism, indicates that the bird is a carrier of the disease.

Practicability—

As mentioned earlier, the "rapid whole-blood" test can be carried out by anyone who has been shown how to do it. Difficulty may be experienced, however, in the interpretation of a proportion of the reactions. Such reactions should be classified as "positive," as it is

* It has recently been shown that reactions can be read more easily when the micro-organisms are stained red.

better to throw out some birds unnecessarily, than to retain even a few positive reactors in the flock. Once proficiency has been acquired, five hundred birds can be tested in an eight-hour day by an operator with two assistants to catch and hold the birds. After much practice the writer has tested 1,000 birds in one such day. Care should be taken, however, to avoid unduly speeding up operations.

The Apparatus Required—

Quite a variety of methods may be used; the simplest only is described here. The following apparatus will be required:—

Several hundred microscope slides.

Thermometer.

A loop of nichrome wire, No. 24 gauge (obtainable from any electrician). The loop should be 3/16th inch in external diameter.

A glass dropper with an external size such that it will just pass through a No. 13 wire gauge. (This dropper will deliver 0.04 ml. of antigen).

Needle.

Antigen.

Bucket of water and cloth.

Retaining boxes, numbered from 1 to 8.

Some heating apparatus.

The simplest form of heating apparatus is a water bath oven, which can be made by soldering a one-gallon oil tin, from which one side has been removed, inside a four-gallon petrol tin, in one side of which an opening the size of the oil tin has been made. This leaves an oven with an open front which may be surrounded on all sides with water, by filling through an opening made in the top of the petrol tin. After filling with water, a thermometer to record the temperature should be inserted through the cork, which should be placed in this opening. (See illustration.) This water bath oven is heated by means of a primus or spirit lamp. It is preferable to fill it with water at the requisite temperature, and then maintain that temperature by means of a spirit lamp or candle.

Preparation Prior to the Test—

On commercial poultry farms it is quite convenient to carry out testing in a semi-intensive house. All birds can be confined to this house and testing carried out on the spot. All litter and dirt should be cleaned out of the house prior to commencement of testing, and the floor should be watered to eliminate dustiness. This is most important, as free dust in the atmosphere will seriously hamper the reading of reactions.

Prior to the commencement of testing, the water bath should be brought to a temperature of 40 to 42 deg. C. (104 to 107 deg. Fahr.), and the microscope slides placed in the oven and left there for an hour or more, prior to the commencement of testing. Alternatively, the slides may be placed in a kitchen oven prior to the commencement

of testing, heated to a temperature much in excess of 40 deg. C., and then placed in the water bath oven and allowed to cool down for five or ten minutes to the requisite temperature.

It is desirable to set up the apparatus at a convenient height, that is, about 2 feet 6 inches from the ground, and to have a seat at a convenient height immediately in front of this. A bucket of water should be placed on the right-hand side of the operator, and a clean cloth kept handy. It is most convenient to have the needle used for pricking the wing-vein and also the wire loop, both inserted into one large cork, so that the vein can be pierced, and the blood collected without having to place down or pick up any further appliances.

The Technique of Testing—

Eight of the clean, warmed slides are placed on top of the oven, and one drop of antigen deposited upon each, from the standard-size dropper which is held absolutely vertically. This is important, because the dropper held at varying angles will deliver varying sized drops of antigen.

The attendant catches a bird by means of a wire, or a bird net, and hands it to the holder. This assistant holds the bird upside down with the head towards the operator, who then plucks the feathers away from the elbow joint, so as to expose the large vein which runs across this joint. He then pierces this vein with the needle, and as a drop of blood wells up, he collects this drop of blood in the wire loop, takes the first slide with the drop of antigen upon it, and mixes the blood on the loop with this drop of antigen, using a rotary motion so as to spread the mixture over an area almost the size of a shilling. The slide is then rotated backwards and forwards rapidly about six times to ensure a very thorough mixing of the antigen and blood, and placed back in the oven. The bird is placed in retaining coop No. 1, and the wire loop is dipped in water and dried on a cloth.

Bird No. 2 is then held ready by the attendant and the procedure is repeated; this is continued until bird No. 8 is reached. When a routine is developed, the slide from bird No. 8 will be placed in the oven a minute and a quarter or a minute and a half after bird No. 1 has been dealt with. Immediately bird No. 8 is dealt with, slide No. 1 is read. If a negative reaction is recorded, the bird is thrown out into the yard. If a positive reaction is recorded the bird is placed in the reactor coop.

Particular care should be taken to expedite the routine technique. The antigen should be placed out on the slides only two or three seconds before blood is taken from bird No. 1, and there should be no delay from bird No. 1 to No. 8. This is particularly important in view of the fact that slides should not be read after two minutes, as misleading results may be recorded, due to the coagulation of the blood and antigen mixture. Furthermore, if the antigen is left on the slide without being mixed with the blood for more than one and a half minutes, it will tend to dry up and precipitate the stained material on the glass slide, and this may later confuse the reading of the reaction.

What Constitutes a Reaction?—

It is pointed out that some people can never exercise sufficient manual dexterity, nor is their sight good enough to detect "fine" reactors, and with such people testing will always be unsatisfactory.

With very definite, positive reactors, as soon as the antigen and blood are mixed, granular purple formations will be noticed. This granulation may be almost as coarse as sago grains and is made up of many thousands of the germ *Salmonella pullorum,* which have clumped together by the action of the blood from the bird which is a positive reactor. In the less strongly positive case, the reaction may show up only after about forty to sixty seconds. In mild reactions, the reaction may not be noted unless very thorough mixing has been carried out, and even then the reaction may be seen only when the slide is held up against a white background or held up against the sky. As the slide is rotated from side to side very fine granulations, about the size of coarse grains of salt, can be seen throughout the mixture. Many such cases may be doubtful, as it is difficult to differentiate between the fine coagulations seen in the blood of some negative reactors and the agglutination shown by a suspicious reactor. Such doubtful cases should be discarded as "positive."

Reliability of the Test—

It is a well-known fact that a positive reactor, if tested every month for a year, may, on odd occasions, give a suspicion or even negative reaction. Even apart from this, it is not claimed that the "rapid whole-blood test" is 100 per cent. accurate, for the reason that the mixture of blood and antigen and the temperature cannot be kept constant, and this test is only a crude approximation of what should be a precisely controlled laboratory procedure. It will, therefore, be apparent that a farmer should under no circumstances depend upon one or even a few tests to rid his farm-stock of this disease.

Furthermore, one must bear in mind that if all positive reactors were eliminated at one test, the ground would still be infected with the causal micro-organism. This micro-organism may remain alive in shady positions for at least a year, and negative birds may become infected during this period. An owner should not, however, feel despondent about this, because spread of infection in this way is not great. That is to say, a few fowls may become so infected, but the spread of infection through the flock is by no means rapid. The fact of ground infection does, nevertheless, necessitate a series of tests, *e.g.*, at two-monthly intervals, being carried out for a year after the last batch of positive reactions has been obtained.

From the practical standpoint, it is not economical to push the testing to this point. It should be remembered that if 100 birds were infected, probably only thirteen of these infected birds would lay infected eggs. Further, these hens would not lay as many eggs, in most cases, as non-affected birds. The few eggs which they would lay, if placed in an incubator or under a hen, would, in most cases, not hatch out, so that even where a large proportion of the flock is affected, one may have quite a number of hatchings without experiencing mortality from pullorum disease.

If pullorum infection is reduced down to the vicinity of 1 or 2 per cent., then the possibility of trouble from the disease is reduced to a very small dimension. It is recommended that when the number of carriers has been brought down to the vicinity of 1 per cent., blood samples should be sent to a veterinary laboratory, where a precise tube agglutination test may be carried out in the endeavour to eradicate the disease from the flock.

Disposal of Reacting Hens—

Where a heavy infection is found to exist at the primary testing, *e.g.*, 40 or 50 per cent., it may not be economical to dispose of all these birds to a table poultry market at once, and in such circumstances it is suggested that these birds may be kept in rigid isolation until such time as it is practicable to dispose of them. It must be remembered, however, that the presence of these birds on the farm is a constant menace to the health of the non-reacting birds, and it is desirable to have them located a considerable distance from the healthy stock. If possible, a separate attendant should attend to them, or failing that, one should wear separate boots and use separate utensils, such as brooms, shovels, etc., as the disease may readily be carried from diseased to healthy birds, and so the beneficial effect of testing would be nullified in some degree, or entirely.

Finally, it should be emphasised that to get a satisfactory understanding of this test, it is necessary to have a positive, a negative and a suspicious recation carried out in the fresh state. Without seeing this it is difficult for any would-be operator to assess his results. Disappointment and doubt of the test almost inevitably follow when one endeavours to test without first attending a demonstration.

Control Apart from Testing—

The disease is of a highly infectious nature and when mortality commences as a result of the infection, chicks in contact should be isolated. Any brooms, buckets, or other utensils used in tending such chickens should not be used in other brooder runs. The attendant who walks into the infected yard should disinfect his boots prior to leaving.

As infection can be spread via the droppings, feed and water troughs should be so arranged that the feed or water cannot be contaminated with droppings. The battery type of feed and water troughs, in which the chickens have access to the feed or water through circular portholes, is to be commended.

When any chickens in a particular batch have died from the disease, the survivors are a potential menace, as many of them will be carriers of the disease. Such carriers, when mature, may lay infected eggs and so complete the disease cycle. Whether it is better to destroy all survivors of an outbreak immediately, and then test the rest of the stock on the farm is a matter which the farmer's economic position must decide.

Correct feeding and brooding methods and the maintenance of strict hygiene (by cleaning brooders and brooder runs, feed and water troughs) is of importance in minimising the spread of the disease. This

would appear to be of most significance with light breeds, for with heavy breeds the disease causes a very severe mortality in any case. In White Leghorns, such is not the case, and in the author's experience mortalities exceeding 60 per cent. have been noted only where the chickens have been confined indoors in moist unclean brooding quarters. This has also been observed by overseas authorities.

Pullorum Disease in the Adult Fowl—

As has been indicated already, apparently normal adult fowls may carry the infection and appear to be perfectly healthy. Thus it is not at all unusual, after testing for the disease, to have several yards of carrier birds, which appear equally as healthy as the negative birds.

Occasionally, the germ, instead of remaining localised in the ovary or some other organ, gains access to the blood stream and sets up the septicaemic form of the disease. Isolated birds frequently show such symptoms. On rare occasions, outbreaks of this septicaemic form, which cause serious mortalities in adults, have been seen.*

As the condition is of comparatively rare occurrence, it will not be dealt with in detail here. Affected birds appear dull, mopey, have a yellowish diarrhoea and may suffer from leg weakness. Death is fairly rapid, occurring in a day or two.

On post-mortem, marked inflammation and discolouration of the liver is frequently a prominent feature. Flakey deposits on, or abscesses in the heart muscle, and fibrinous material in the pericardium are sometimes seen, whilst discoloured misshapen ova, and cystic ova, which at times becomes detached and float freely in the abdominal cavity, are usual.

Carrier Birds—

Apart from the comparatively rare outbreaks of septicaemic pullorum disease as above, the vast majority of adult birds, which harbour the infection, show no abnormality when alive. If killed for post-mortem examination, it is usual to find misshapen ova. Instead of the ova being perfectly round, smooth and filled with material of egg yolk consistency, they become compressed, angular, cheesy in consistency and markedly discoloured. Instead of the normal cream to yellow colour, the affected ova are darker, varying from yellow to brown, green or blackish.

The diagnosis of the carrier bird is by means of the agglutination test as already indicated.

The Role of the Carrier Bird in Spreading Disease—

It has already been mentioned that infected adult birds lay a small proportion of infected eggs, and in the event of one such infected egg hatching, a severe outbreak of the disease occurs in that hatch of chickens.

Apart from this, it has been conclusively demonstrated that running affected with non-affected birds results in the spread of infection to the latter. In the course of eradicating the disease from a flock this is

*For further information see *Aus. Vet. Journal*, 1936, vol. 12, p. 17.

a factor of great importance. In one case infected faeces from a carrier bird were shown to contain the live germ of pullorum disease after lying in a shady position for 100 days. It is found when all carrier birds are removed from a flock that ground infection, left behind by the carrier birds, leads to a further infection of a small proportion of birds, and in an eradication campaign it is, therefore, necessary to continue a series of tests until all ground infection has ceased. Occasional negative birds in contact with carriers may, if tested frequently, give a positive reaction for a few weeks and then fail to react—this causes the reliability of the test to be questioned when actually the test is not at fault. Such birds are infected, develop a resistance and recover. All positive reactions, however, should be discarded as it is better to reject perhaps a dozen birds like this, than to leave one carrier in the flock.

Adult carrier birds may also transmit the disease by contact (direct or indirect) to young growing stock.

Coccidiosis.

This is a serious disease of chickens, which occurs widespread in all poultry farming areas of the State.

Cause—

(*a*) *Actual.*—The disease is due to infestation with a microscopic animal (protozoan) parasite. There are a number of different types of coccidia which infect poultry and several different species have been reported in New South Wales. The commonest organism is *Eimeria tenella* which produces haemorrhagic inflammation of the caeca in chickens.

(*b*) *Predisposing Causes and Source of Infection.*—Moist and filthy conditions, overcrowding, running chickens on "stale" ground, allowing the feed and water troughs to become contaminated with droppings, and allowing direct contact (*e.g.*, attendants' boots) between adult fowls and young stock, are prominent factors which pave the way for an outbreak of this disease.

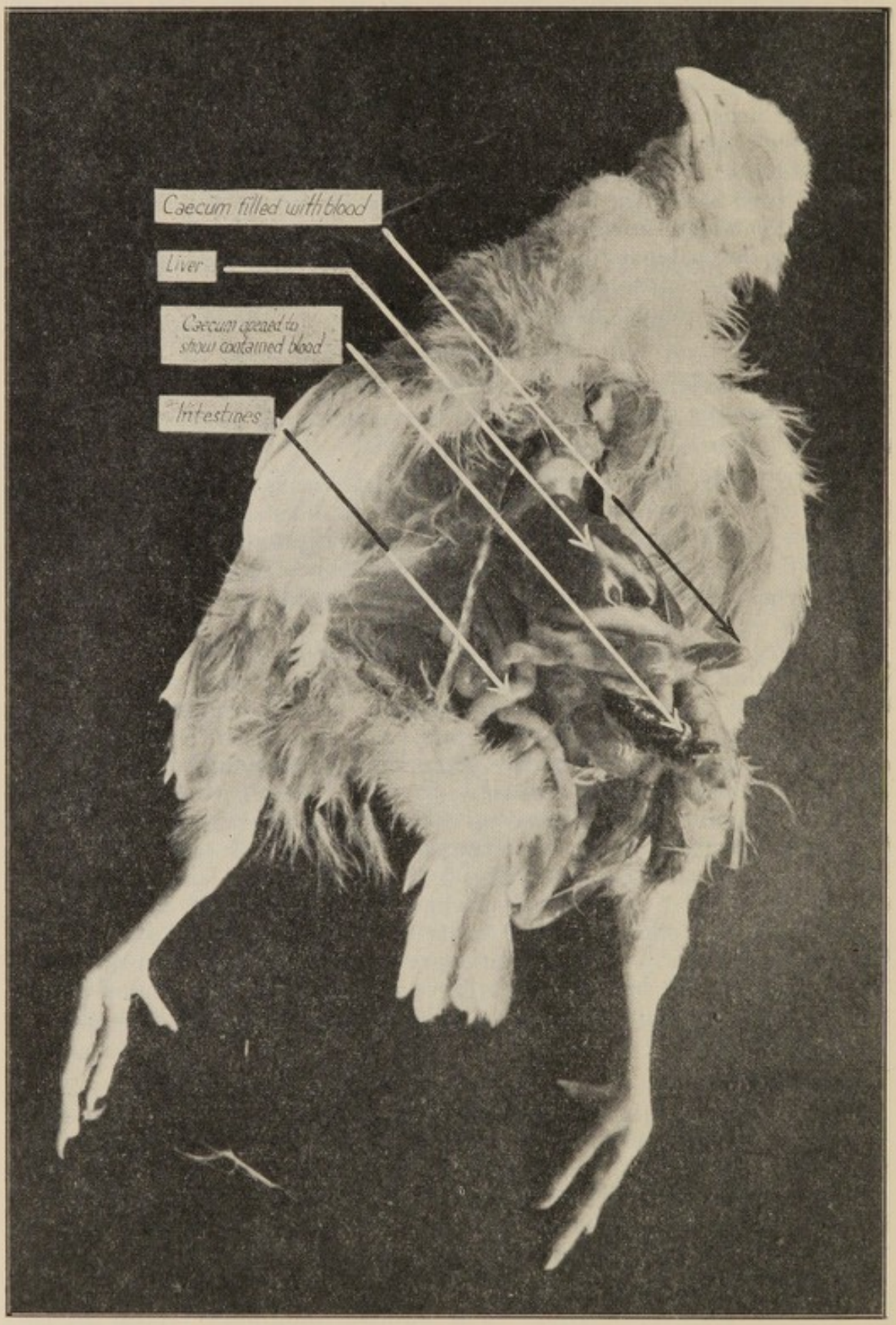
If all droppings were cleared up each day and the runs, brooders, etc., were kept dry and clean, the chances of an outbreak occurring would be reduced to a minimum. This is because of the life cycle of the causal parasite. (See later.) It has been shown that deficiency of Vitamin A will greatly augment the mortality in coccidiosis. It has been recorded that White Leghorns are more susceptible than the heavy breeds, and whilst bad outbreaks have been seen in all common breeds in this State it would appear that White Leghorns are perhaps slightly more susceptible.

Life History—

The birds are infected with coccidia by means of droppings from other birds which, when a day or so old, carry the infective form. The infective form of the parasite is encapsulated and is very resistant unless exposed to sunlight drying or heat. When ingested, this infective stage of the coccidia develops in the intestinal wall to produce a number of cells. These cells continue reproducing in either one or two ways, as follows:—

(*a*) *Asexual reproduction.*—The parasite multiplies rapidly in the intestine and each generation re-infects the chicken. After this process has been going on for some time, most of the cells cease reproducing in

this manner and reproduce according to the sexual cycle. Both forms of reproduction occur to some extent all the time.



An Eight-week-old White Leghorn that has Succumbed to the Peracute Form of Coccidiosis.
Notice the two caeca or blind pouches) filled with bloody exudate. One has been opened.

(*b*) *Sexual Cycles.*—Cells become differentiated into male and female cells, which produce a micro-organism known as an oocyst. The oocyst is passed out in the droppings. It is encapsulated and may resist weather conditions for some time. Within the capsule the oocyst divides up into a number of cells known as sporozooites, which await a favourable opportunity to infect some other chicken.

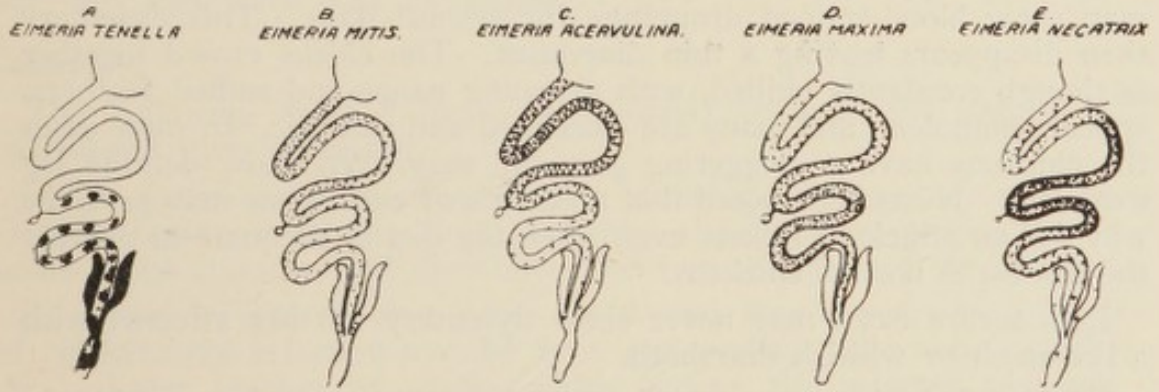
It will thus be seen that to interrupt a complete life cycle of the coccidia absolute cleanliness, or some means of preventing contact of chickens with infected droppings, is indicated.

Infectivity—

The disease spreads from bird to bird by means of infected droppings. An affected bird may void the parasite in the dung, and this infective dung may contaminate the feed, or water, or may be picked up along with other material by another chicken. To be infective, segmentation must have taken place, and the time for this varies from thirty hours to several days, according to environmental conditions, and also according to the species of coccidia concerned. The infective bodies will remain alive in moist conditions for more than a year. It appears that putrefaction destroys the oocysts and that they are destroyed in decomposing heaps of fowl manure. Birds which have recovered from an attack of the disease have been shown, for the most part, to remain carriers of the infection for long periods.

Fowls of ages from two weeks to fifteen months (if kept apart from infection) have been shown to be liable to outbreaks of coccidiosis from *Eimeria tenella* infection.

There is some variance of opinion as to the susceptibility to infection of chickens younger than two weeks. It is probable that predisposing causes such as deficiencies may have a bearing on this.



The Typical Distribution of different Coccidia and the Lesions they Cause in the Intestinal Tract of Fowls.

The blood-filled caecal pouches in Eimeria tenella should be noted.

[*After Bayon.*

Locally an outbreak has been seen in chickens less than a week old, and a small number of outbreaks in chicks seven to eleven days old. It would, however, appear that after two weeks of age the chickens are more susceptible, the most severe outbreaks occurring in chickens three to seven weeks old.

All classes of birds appear to be susceptible to their own particular species of coccidial parasites. Thus in chickens—

Eimeria tenella causes severe inflammation of the caeca.

Eimeria acervulina causes inflammation of the duodenum.

Eimeria maxima } cause inflammation of the middle portion
Eimeria necatrix } of the small intestine.

Eimeria praecox } are apparently of little importance.
Eimeria mitis }

Isospora spp. infest sparrows.

Eimeria truncata affects geese.

There is some evidence that *Eimeria tenella* affects pigeons as well as fowls.

Immunity—

Following an outbreak of the disease either mild or acute, an absolute immunity to further infection is not developed, though after one attack the birds' resistance is very much increased and serious symptoms, as a result of later infections, are not usually seen.

Symptoms—

Symptoms naturally vary with the age of the bird when it is first infected, the species of coccidia present, and the massiveness of the infection.

In very severe cases of coccidial infection in young birds (e.g., one month old), the bird passes blood-stained droppings, which are fluid or semi-fluid in consistency. The affected birds appear very depressed, ill, and stand about in a huddled or crouching position. The feathers round the vent are usually soiled with faeces. In the most severe cases prostration and death occur very soon, sometimes within a few hours of the first appearance of symptoms. Cases which do not succumb, may show blood-stained droppings for several days. This dysentery then disappears leaving a thin diarrhoea. The chicks crowd together as though constantly chilled, with drooping wings and ruffled feathers, appear somnolent, and many are emaciated and anaemic. In some cases the chickens have a staggering gait and may even show definite leg weakness. It has been noted that an attack of coccidiosis may pave the way for an attack of rickets even when the diet is adequate to prevent the disease in normal chickens.

Less severe cases may never show dysentery but are affected with a brownish or whitish diarrhoea.

Carrier birds, i.e. "recovered" cases that still pass a few coccidia in the droppings, may be stunted in development, poor in condition with a capricious appetite, increased thirst and sometimes anaemia, emaciation and leg weakness are in evidence.

Lesions—

The main changes are to be seen in the two blind guts or caeca. In the very acute type these will be greatly distended with blood and semi-solid exudate. On removing this nearly all the lining membrane is seen to be haemorrhagic and inflamed.

In less acute cases the caeca will be distended with solid, or semi-solid caseo-purulent material; this may be tinged or spotted with blood. The walls of the caeca are thickened and show patches of reddening.

The membrane lining the first part of the intestine is often greatly inflamed, and the contents are dirty-grey and thick instead of the normal yellow soft consistency.

The post-mortem appearances will vary with the type of causal coccidia. Thus, in some types (particularly those affecting adult birds), inflammatory changes may be confined to the small intestine.

Differential Diagnosis—

The disease, when in very young chicks, might be confused with pullorum disease. However, the inflammation of the caeca seen on post-mortem should distinguish the condition as coccidiosis. No lung or liver abscesses as sometimes seen in pullorum cases are present. Though symptoms are characteristic, absolutely positive diagnosis can only be made by examining the bowel wall or the droppings under the microscope and finding coccidia.

Entero-hepatitis (blackhead) may also be confused with this disease. For differentiation see page 79.

Pathogenicity—

Death from the disease may be very high under conditions which favor the propagation of the coccidia, and mortalities exceeding 90 per cent. have been seen. Apart from actual mortality severe loss may be sustained due to checking in development and egg production. In one experiment where 100 birds were housed in two similar pens, the birds in one being affected chronically with coccidiosis, the following observations were made:—In the affected pen there were 23 per cent. more deaths, and the birds in the unaffected pen laid 15.2 eggs more per bird and were 0.44 lb. heavier.

Control—

The following facts (already mentioned) indicate the nature of control measures necessary.

Adult fowls carry coccidia without showing any symptoms. The farmer coming from the fowl yard into the brooder carries on his boots a few coccidia, and distributes them on the floor of the brooder.

It is noted regularly that when the disease first appears in a batch of chickens the infection may be light, and perhaps only a few chicks die. Later batches of chicks which occupy the infected premises experience increasingly severe attacks.

Chicks can contract the disease only by picking up coccidia passed by other birds. No amount of faulty management can produce the disease, though such faulty management will seriously lower the chicks' resistance and increase the severity of an attack once infection has been introduced.

Prevention should, therefore, be based on methods of improved cleanliness and care should be taken to avoid introducing the disease. If possible, the person who attends the brooder should not go into fowl

yards. If this is not possible, a special pair of boots should be kept just inside the door of the brooder house, and put on before going amongst the chicks. This may also prevent pullorum disease being introduced. Wearing rubber boots and disinfecting them before entering the brooder is not completely effective against coccidiosis, as the coccidia are not killed by ordinary disinfectants.

When a mild outbreak of the disease occurs it may be sufficient to institute a thorough method of frequent cleaning of the runs. Chickens can only be infected further by coccidia from droppings of other affected chickens. The droppings should, therefore, be cleaned up once or, if possible, twice daily. After cleaning out, lightly dust the floor of the brooder run with sand to prevent the droppings sticking to the floor. Usually this frequent cleaning will make it necessary to engage more labour, and in view of this it is often cheaper and certainly more effective to proceed as in the case of a severe outbreak.

When a severe outbreak occurs, kill those chicks showing symptoms. Place the rest on wire-netting floors and make feed and water available in such a way that the droppings cannot contaminate them, preferably using the method adopted in battery brooders. The wire-netting floors may be provided by making light frames which exactly fit the brooder and brooder run. Great care must be taken to make these frames so that the netting does not rest directly on the wooden frame-work, as in this case the droppings are caught on the woodwork and the chickens are, as a result, infected almost as effectively as if on a solid floor; wire or ¼ inch steel may be used to support the netting. The infected droppings fall straight through the netting and should be collected fairly frequently (at least once a week) from below. Any fault in the construction of the netting flooring which leaves droppings available to the chick will obviously nullify the benefit of the outlay. The netting arrangement should, therefore, be examined critically, with this in mind.

Where wire-netting floors are used to control the disease, a note of warning must be given. If chickens are kept on netting, quite apart from any infection, until they are most highly susceptible (three or four weeks old) and then placed down on infected runs, a very heavy mortality will be experienced.

Coccidiosis is most emphatically one of the diseases in which good sanitation and cleanliness are of high value.

Infected droppings should not be used as manure on the areas on which green feed for the poultry is growing, as this is one of the methods by which the disease can be spread. If used it should first be allowed to decompose thoroughly in a tightly-packed heap.

It is pointed out that the raising of chickens on wide open range conditions (the range being kept for chickens only) is ideal. Under such conditions chickens normally pick up a very mild infection at some time in their early life and develop a satisfactory resistance to further infections.

Treatment—

Many drugs have been experimented with in the treatment of coccidiosis in chickens, but the only one widely used at present is colloidal iodine, and even here experimental work has not regularly supported field observations. The best that can be said is that it may aid in the control of the disease, but cannot be regarded as a complete cure. When administered in drinking water, care must be taken that no other water supply is available to the chickens, even for a brief period. Otherwise, they will not touch the medicated water.

One method of supplying colloidal iodine is as follows:—Dissolve 1 oz. iodine crystals and 2 oz. potassium iodide in 1 pint water, add 1 part of this to 5 parts of milk (skim milk or dissolved powdered milk will do), and heat at about 70 to 80 deg. C. (158 to 176 deg. Fahr., well below boiling-point) until the milk goes white. Add one pint of this iodised milk to each gallon of drinking water.

Treatment with one part of 5 per cent. acetic acid in 50 parts of drinking water, and another treatment involving the use of 10 c.c. of formalin (40 per cent. formaldehyde) per gallon of drinking water were found to be of some value (in critically "controlled" experiments) to control outbreaks of the disease.

It has been shown by several workers that sulphur fed in the ration prior to infection confers some protection on the chickens, but unfortunately checks their growth and development. Thus, where 10 per cent. of sulphur was fed in the ration for seven days before and after infection, the chickens were completely protected from moderate infections, but not from very heavy infections.

The use of molasses in the drinking water (½ pint to the gallon) is also claimed to reduce the harmful effects of an infection.

The use of milk and milk products has also been widely advocated; here again experimental evidence is not altogether favourable so far as direct control of the disease is concerned, but there is evidence that the growth rate of the chickens is improved by its use. (The same has been recorded with the use of iodine).

When an outbreak of coccidiosis occurs, the dried milk in the ration, usually fed at the rate of 5 per cent., may be increased up to about 40 per cent. It is hoped by this to produce an unusually acid condition (lactic acid) in the caeca, which checks the growth of coccidia in this site. It is doubtful whether such occurs. The beneficial effect (where existent) is perhaps due to the supply of extra vitamins (particularly D) and minerals as, in an outbreak of coccidiosis, there is an increased demand for these substances. Provision of artificial heat has been claimed to assist in reducing mortality.

To summarise, when an outbreak of coccidiosis occurs no medicinal treatment should be relied upon to cure or limit the disease, but every endeavour should be made (hygiene, cleaning, wire-netting floors, etc.) to keep the chickens rigidly out of contact with their droppings, and to maintain a dry, well-lighted (sunlight) environment.

Attention to the ration to ensure adequate supplies of all necessary constituents, and perhaps the addition of extra milk products, is of value.

Immunisation—

Though much work has been done, some of which has been promising, no satisfactory vaccine has yet been produced, as it has not been possible to so weaken coccidia that they do not set up the clinical disease, and yet provoke a satisfactory immunity in treated birds.

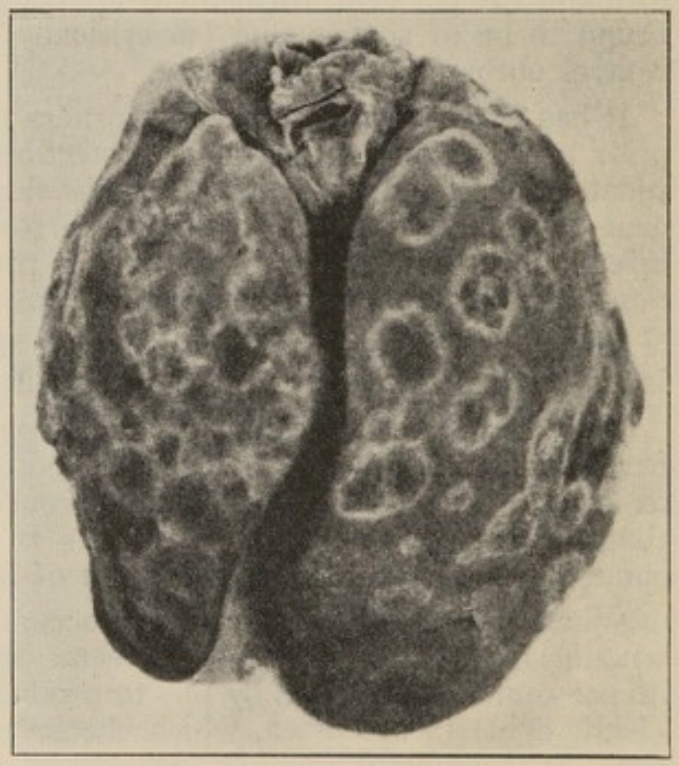
Entero-hepatitis.

This disease is also known as blackhead and is an acute contagious disease affecting mainly turkeys and fowls. Until recent years the disease was thought to be of economic importance in turkeys only.

Cause—

(*a*) *Actual.*—The actual cause of the disease is a protozoan parasite *Histomonas meleagridis*. Overseas workers have also recorded that a disease clinically indistinguishable from "blackhead" has been caused by fungal infection. This has not been noted here.

One school of thought (particularly German authors) holds that trichomonads are the cause of entero-hepatitis. Whereas these organisms are sometimes present in livers affected with blackhead, it is not possible to produce the disease regularly with trichomonads. Trichomoniasis is a separate disease entity.



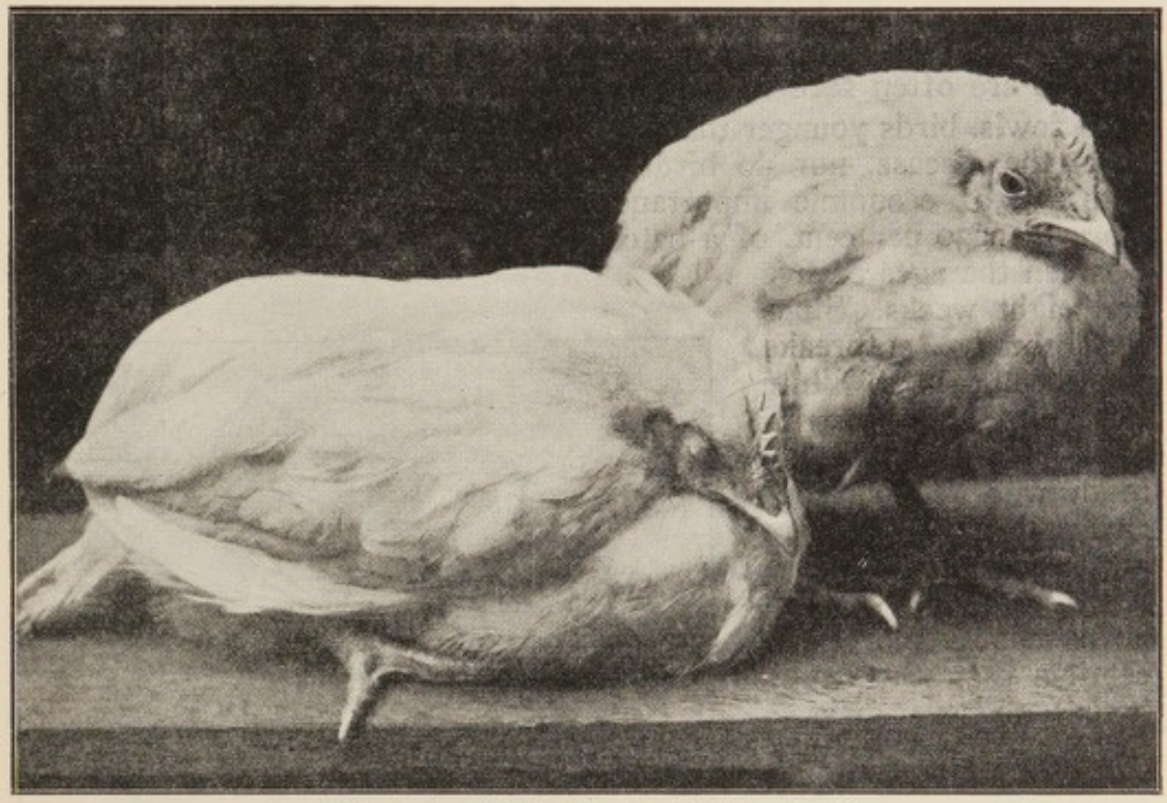
Blackhead—Liver of a Chicken.
[*After Craig.*

(*b*) Predisposing Cause.—Overcrowding, moist dirty conditions, and anything which allows contamination of food and water by droppings are predisposing causes as in the case of coccidiosis.

It has also been shown that the infective protozoa may be carried in the eggs of *Heterakis gallinae* (the small round caecal worm), and the presence of this worm parasite is a predisposing cause to entero-hepatitis. As adult fowls frequently carry the infection (and this worm) without showing any symptoms of disease the running of turkeys and fowls together is considered to be a predisposing cause of an outbreak of the disease in the turkeys.

It would appear that normal fowls and chickens may harbour this parasite without showing any symptoms of disease and that seemingly the predisposing causes, rather than the presence or the absence of the organism are the critical factors in the occurrence of symptoms. One common predisposing cause is vaccination against fowl pox. In the

third week following the vaccination the birds are off their feed and obviously suffering from a systemic disturbance for about forty-eight hours. This may occur at any time from fourteen to twenty-one days after vaccination. In some cases young fowls have contracted blackhead at this stage and mortalities in excess of 30 per cent. have been noted.



White Leghorn Pullets affected with "Blackhead."

In this outbreak in the Metropolitan Area over 20 per cent. of a large batch of such pullets died as a result of infection with the disease.

Characteristics of the Causal Micro-organisms (*Histomonas meleagridis*) —

This protozoan organism has three distinct phases in its life cycle—invasive, vegative and resistant. In the invasive phase it is amoebiform, *i.e.*, a microscopic jelly-like organism. In the resistant phase it is surrounded with a dense surface membrane. The mirco-organism is easily killed by drying or by disinfectants. Even under ideal conditions it is difficult to keep the micro-organism alive for more than eighty days in the laboratory. Although it is so susceptible to harmful influences and normally does not live long outside the animal body, it may remain alive for quite long periods when encased within the eggs of *Heterakis gallinae*.

Infectivity—

Infection apparently takes place directly from bird to bird by means of infected droppings. Experimentally it can be produced by feeding infected droppings or organs to susceptible birds. The worm *Heterakis gallinae* also appears to play a very large part in the transmission of the disease and some authorities go so far as to state that without this worm parasite the disease does not occur. The causal micro-organism is

protected in the eggs of *Heterakis gallinae* and further it appears that the wounding of the mucous membrane of the caeca by this parasite allows the entry of the protozoan "germ." Flies are regarded with suspicion as mechanical transmitting agents of the infection.

Turkeys and fowls appear to be the main species susceptible. Turkeys on badly infected ground may be affected when three weeks old but are particularly susceptible at about the stage when they "shoot the red" (when the caruncles begin to develop and redden up) that is when about six to eight weeks of age. Older birds may be affected, and adult turkeys are often seen with the disease.

In fowls, birds younger than four weeks of age seldom appear to die from the disease, nor do birds older than six months. In the first outbreak of economic importance seen by the author in this State more than 30 per cent. of a batch of Rhode Island Red chickens died between the ages of five and eight weeks. Since then many outbreaks have been seen in White Leghorns and Australorps. It is not possible to draw a reliable conclusion on such matters but, so far, it has been noted that White Leghorns appear more susceptible than the heavy breeds to this disease. It would appear probable that in both fowls and turkeys resistance increases with age. One attack of the disease does not usually confer immunity against further attacks for more than about fifty to sixty days. In this State turkeys have been noted to suffer from more than one attack, but each attack appeared relatively mild.

Liver of a Turkey affected with Blackhead.

Course of the Disease—

The causal protozoa invades the blind end of the caeca, and a few days later spreads via the blood stream to the liver where it produces abscesses. After artificial feeding of infectious material symptoms develop in four to nine days, and death occurs usually about the ninth to fifteenth day.

Symptoms—

Symptoms are similar in both fowls and turkeys. In young birds they are noted to "droop" and stand about in a huddled position, with the tail depressed, and the head retracted—an attitude typical of sick birds.

In young fowls the normal pinkish colouration of the skin of the face frequently assumes a faint leaden colour, and the comb and wattles (if developed) appear shrunken. In turkeys the skin of the head and caruncles particularly may be distinctly blackish or bluish (hence the name "blackhead").

In acute cases the droppings may be blood stained. In less acute, and in protracted cases diarrhoea is present, the droppings varying in colour from light yellowish to dark orange.

Paleness of the comb, shanks (in chickens) and dullness of the plumage are noticeable features in many cases.

Post-mortem Appearances—

Post-morten appearances are much the same in both fowls and turkeys. The caeca (or blind guts) are seen to have dirty yellow ulcers which vary in size. These ulcers may be deeply embedded in the intestinal wall and at times lead to perforation, peritonitis and death. The wall of the affected intestine is usually much thickened. In chickens the caeca are first filled with blood-stained mucus and this hardens to form a cheesy core, which also is frequently blood-stained. This cheesy core seems to be laid down in successive layers, as when cut across it has annular markings, which on cross-section appear like the markings on the cross-section of a tree trunk.

Liver lesions are extremely common in turkeys but not so common in fowls. In the latter many dead chickens may be examined before liver abscesses are seen. The characteristic liver lesions are numerous, yellowish abscesses from pinhead size to half an inch in diameter, scattered right throughout the substance of the liver. These abscesses are circular, and frequently have concentric markings.

In fowls the liver may sometimes be congested and at other times enlarged and pale with no visible abscesses.

Differential Diagnosis—

The liver abscesses are quite characteristic when present. If intestinal material is examined microscopically at a laboratory, the protozoan parasite will be seen in large numbers and this will confirm the diagnosis. When the only lesion is blood-stained mucus in the caeca the condition could easily be confused with coccidiosis, and in such a case microscopic examination will be necessary to differentiate the two diseases. Usually, however, some birds are found to have the typical caecal casts laid down in concentric layers with the appearance of annular rings on cross section.

Pathogenicity—

Over 50 per cent. of deaths in chickens and over 90 per cent. in turkey poults have been seen.

As has already been stated, fowls and chickens for the most part tolerate an infection without showing symptoms.

Control—

As the disease is spread in the same manner as coccidiosis control measures (apart from treatment) are the same as recommended for that disease. Running birds on wire-netting, as recommended for

coccidiosis, or on pens with smooth concrete floors which are cleaned and washed down daily, and all other measures aimed at improved hygiene and sanitation are the main lines of attack.

Turkeys should not be run on the same range with fowls, in view of the much greater susceptibility of turkeys to the harmful effects of the infection. Similarly young birds should be kept quite apart from old birds.

Where infection is heavy, one effective way of rearing turkeys is to keep them on wire-netting until about 10 weeks old and then to place them out in clean lucerne paddocks.

Surgical removal of the caeca prevents the disease but this procedure is hopelessly impractical.

Treatment—

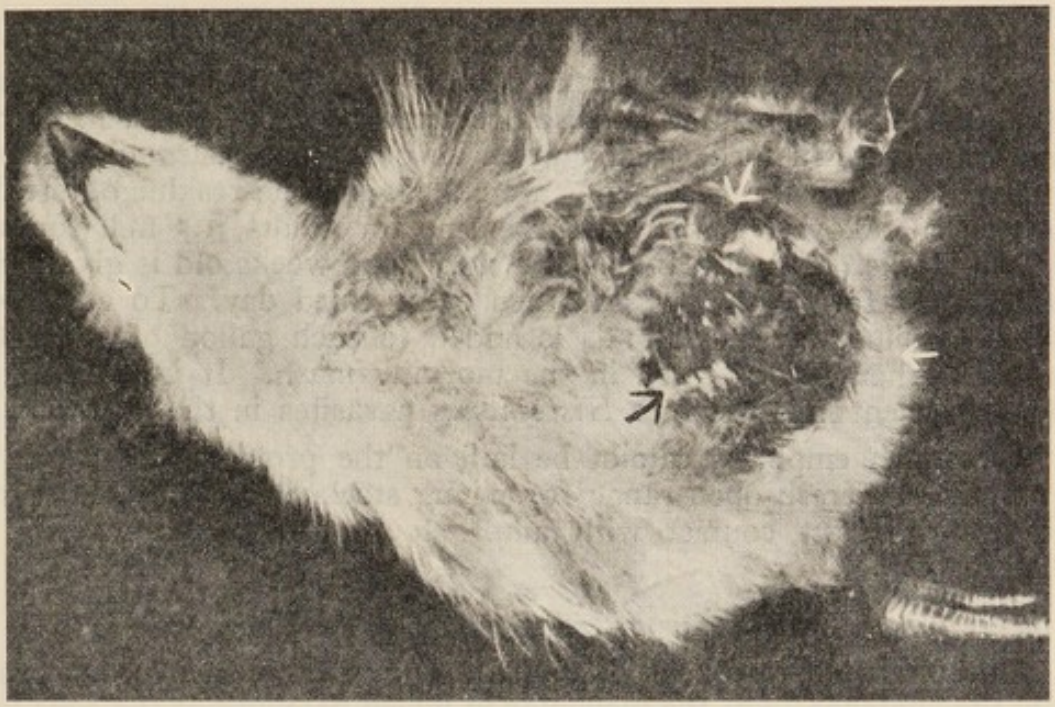
Many treatments have been recommended for this disease but most appear to be valueless. For turkeys Neosalvarsan injected intravenously or Myosalvarsan injected intramuscularly, both at the rate of 0.005 grams per lb. of body weight in 0.1 m.l. of water appear to be fairly effective if the bird can be detected in the incubation stage of the disease, but unfortunately the treatment is fairly expensive. A second dose may be necessary in twenty-four hours. Neokharsivan is a similar preparation for intravenous use.

Tryparsamide has been favourably reported upon for turkeys. With birds three to five weeks old a course of three intravenous injections (into the large wing viens) of 0.25 grams of Tryparsamide in 1 m.l. of water at ten-day intervals is given. Double this dose should be given in the acute stage of the disease.

Recently in Great Britain two preparations have been reported upon favourably, as to their curative effect on turkeys affected with entero-hepatitis. Both these drugs are available locally. Mapharside (also known as Mapharsen) is given by intravenous or intramuscular injection in the following approximate doses:—Turkeys three months old, 0.004 grain; five months old, 0.006 grain. The other drug, Spirocid, is put up in tablet form for administration by the mouth. The dose for turkeys three to six weeks old is half a tablet at weekly intervals, and for six weeks and over, one tablet every ten days. It is pointed out that a bird may not show symptoms until a large amount of liver tissue has been destroyed, and treatment may then be too late. Thus where some birds are affected the incontacts should be treated. This unfortunately is expensive.

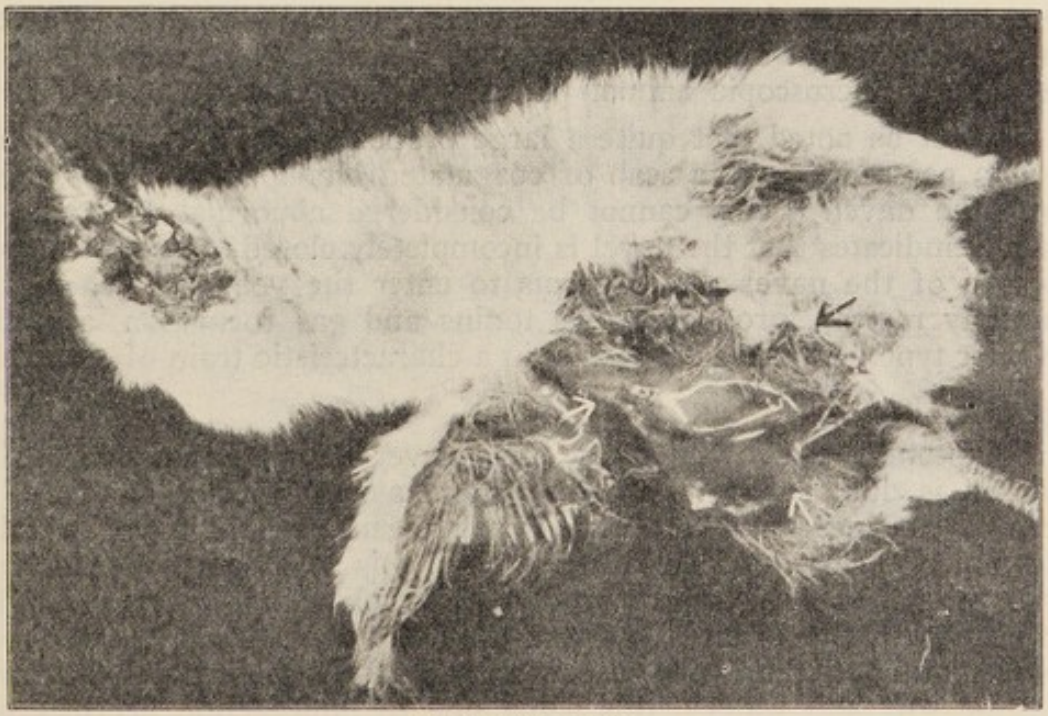
Treatment of fowls and chickens is only in the experimental stages locally, and definite advice cannot be given as yet. It is emphasized that where entero-hepatitis is the cause of heavy loss in fowls or turkeys veterinary advice should at once be sought as to the preventive treatment to be adopted.

Some authors have claimed slight value from the administration of tartar emetic and ipecacuanha. No reliable evidence of the value of these drugs is to hand. As regards ipecacuanha, one drachm of the powdered drug is sometimes placed in each gallon of drinking water. Using the ipecacuanha wine one dessertspoonful is sometimes



Omphalitis, showing a Scab over the Navel, and Surrounding Inflammation.

This and the other illustration are of chickens from a farm where an outbreak resulted in the loss of 10 per cent. of the chicks in the first three days.



Purulent Omphalitis.

The abdominal wall has become involved, infection having spread from the navel.

placed in each quart of drinking water and this given to the birds one day each fortnight—in the case of poults, from a week before they start to "shoot the red." Affected poults are dosed with ten drops of the wine. In any treatment emphasis should be laid on the fact that for success administration must take place before extensive liver damage has occurred. Turpentine has been recommended as of value for treating the disease in turkeys, and when an outbreak occurs all sick birds and those with yellow droppings are culled, and the residue of the flock treated as follows: 2 m.l. of turpentine for adults, 1.5 m.l. for birds fifteen weeks old, and 0.25 m.l. for birds four weeks old is given every second day for eight days and then every third day. To administer, 1 oz. of oil of sweet almonds is added to each gallon of turpentine, which is then incorporated in the morning mash. It is claimed that this treatment kills the free *Histomonas* parasites in the intestine.

Too much emphasis cannot be laid on the provision of clean, dry quarters and fresh open range for young stock, with complete absence of even indirect contact with adult stock, for the control of this disease.

Omphalitis or Inflammation of the Navel.

This disease of chickens is typically due to dirty incubators, especially the "hatcher," i.e., the incubator in which hatching of the eggs takes place. However, as it is due to an infection, it may sometimes occur in the cleanest and best managed hatcheries, causing considerable loss. It is very seldom noted in hen-hatched chickens.

No one particular germ is responsible for all cases, and on culturing from the inflamed area around the navel and yolk sac, quite a variety of organisms may be recovered. Coliform types of germs are the most common, while *Clostridium sporogenes* and *Clostridium tertius* have been recovered as the cause in some cases. Recently an oversea investigator recorded an outbreak of omphalitis caused by Trichomonad infection (a microscopic animal parasite.)

It must be noted that quite a large proportion of chickens, perhaps 2 or 3 per cent., have a scab of coagulated blood and serous material over the navel. This cannot be considered abnormal in itself, but usually indicates that the navel is incompletely closed. This incomplete closure of the navel allows germs to enter the yolk sac, where they multiply rapidly, producing pus, toxins and gas formation, according to their type. This in turn results in a characteristic train of symptoms.

Symptoms—

Symptoms may show up at the time, or very soon after hatching. The chicken is dull, lethargic and may stand alone, chirping uneasily. A scab of varying type and size is usually seen over the navel, which is incompletely closed. The tissues around this scab are reddened, or in later stages may be discoloured, the discolouration varying in hue according to the progress of the disease. This area is often very swollen and puffed up with gaseous material. At other times the tissues may be soft and watery, this being due to liquefaction necrosis produced by the bacteria causing the condition. The symptoms vary greatly, depending mainly on the different types of causal bacteria. A foul smell of putrefaction is a very common feature.

Deaths usually occur a few hours after the chickens first show symptoms.

Post-mortem Appearance—

On opening the chicken, the yolk sac is seen to be abnormal. Usually it is enlarged in size and the yolk contained is abnormal in consistency, sometimes being consolidated into cheesy masses, at other times being liquified and containing gas bubbles. The wall of the yolk sac is inflamed.

The changes seen in other parts of the body vary enormously, depending upon the type of infection. Sometimes the whole chicken at the time of death is "soggy" and falls to pieces with very little traction, all the tissues having undergone liquefaction necrosis. The liver is usually paler than normal, but may be congested, showing dilated blood vessels. It is at times easy to confuse the disease with pullorum disease (bacillary white diarrhoea).

Differential Diagnosis—

The disease may be distinguished from pullorum disease, coccidiosis, and salt poisoning by the following features:—

Pullorum Disease.—The pale greyish-green or yellow coloured abscesses in the lung and liver are absent in omphalitis.

Deaths from pullorum disease begin about the second day, typically increase in number up to the tenth or twelfth day, and do not stop until about the twentith day. In omphalitis the deaths occur mainly in the first four days, and very few after the eighth day.

Coccidiosis usually occurs in chicks over two weeks of age. The intestinal symptoms and blood in the droppings, characteristic of coccidiosis, are not seen in omphalitis.

Salt Poisoning.—Excess fluid in the tissues is often noted in this, but the characteristic lesions of the navel are absent.

When any doubt exists, the farmer should consult a veterinarian. If in doubt he may take dying chicks, or chicks just dead, to a bacteriological laboratory which deals with such matters, in order that the causal germ may be ascertained.

Although this disease seldom causes such a heavy mortality as does pullorum disease, losses may at times be serious.

Prevention—

Prevention of omphalitis is to be directed along the lines of good hygiene. Incubators should be scrubbed out with disinfectant between hatches, and during each hatch two fumigations should be given. (See fumigation, page 215.)

It is to be noted that after "sexing," a chicken with omphalitis will often show symptoms of distress, gasping and perhaps not being able to stand. In some cases the abdominal wall may rupture during the sexing operation. It is thought that in these cases there is an increased abdominal pressure, and when the person "sexing" squeezes the abdomen it embarrasses the heart and lung action, producing temporary breathlessness, or even asphyxia.

Providing the person "sexing" is reasonably dexterous and careful in handling the chickens, those that show symptoms of distress as a result of the operation should be looked upon as probable cases of omphalitis.

Treatment of omphalitis will meet with varying success, depending upon the type of germ responsible. Iodine in collodion used locally is satisfactory in some cases; however, in an outbreak of this disease a qualified veterinarian should be called in to make a correct diagnosis.

In most cases it is preferable to kill the affected chicks and burn them promptly. All places where they have been should be disinfected, incubators fumigated, and egg shells from the incubator burned.

Mycosis.

This group of disease conditions, due to infection by various fungal parasites is rather uncommon, but occasionally causes mortality in fowls, turkeys, geese, ducks and pigeons in this State. The condition is also referred to as "brooder pneumonia" and as "black mould pneumonia." Unfortunately, the term "brooder pneumonia" has also been used erroneously to designate pullorum disease. The disease usually affects young birds, but older birds may be affected and in one case in New South Wales nearly 20 per cent. of adult turkeys were lost due to mycosis.

Cause—

(*a*) *Actual.*—Various different fungal parasites have been incriminated. In New South Wales *Aspergillus fumigatus* appears to be most common. In Queenslands, *Monilia albicans* is more commonly reported. In other countries, *Oidium* spp. sometimes cause quite heavy mortalities, as also do other species of *Aspergillus.*

(*b*) *Predisposing.*—Damp brooding conditions, dirty quarters, mouldy food, e.g., mouldy maize or wheat, or mouldy haystacks available to the birds, are all predisposing causes.

Symptoms—

Affected birds may be seen at any age, but birds from ten days to four months are most commonly affected. They appear mopey, listles, and in some cases gasp for breath. Sometimes diarrhoea is present.

Post-mortem—

On post-mortem examination quite a range of different appearances may be seen. Sometimes fluffy ball-like growths may be seen on the air passages and on the membranes of the air sacs. More frequently the lung substance and air sacs may contain nodules or masses of caseous necrotic (dead) material. Sometimes greyish or greenish to black membranes are seen lining the windpipe, air sacs and in the lungs. In adults the post-mortem appearance may resemble tuberculosis.

Differential Diagnosis—

The lesions do not resemble other common diseases of birds, but for certain diagnosis, microscopic examination is necessary to detect the presence of the fungal bodies.

Control is to remove the damp conditions, or the source of contamination with the moulds.

Attention should be given to correct diet and management. No treatment is known to be successful for mycosis (or "brooder pneumonia").

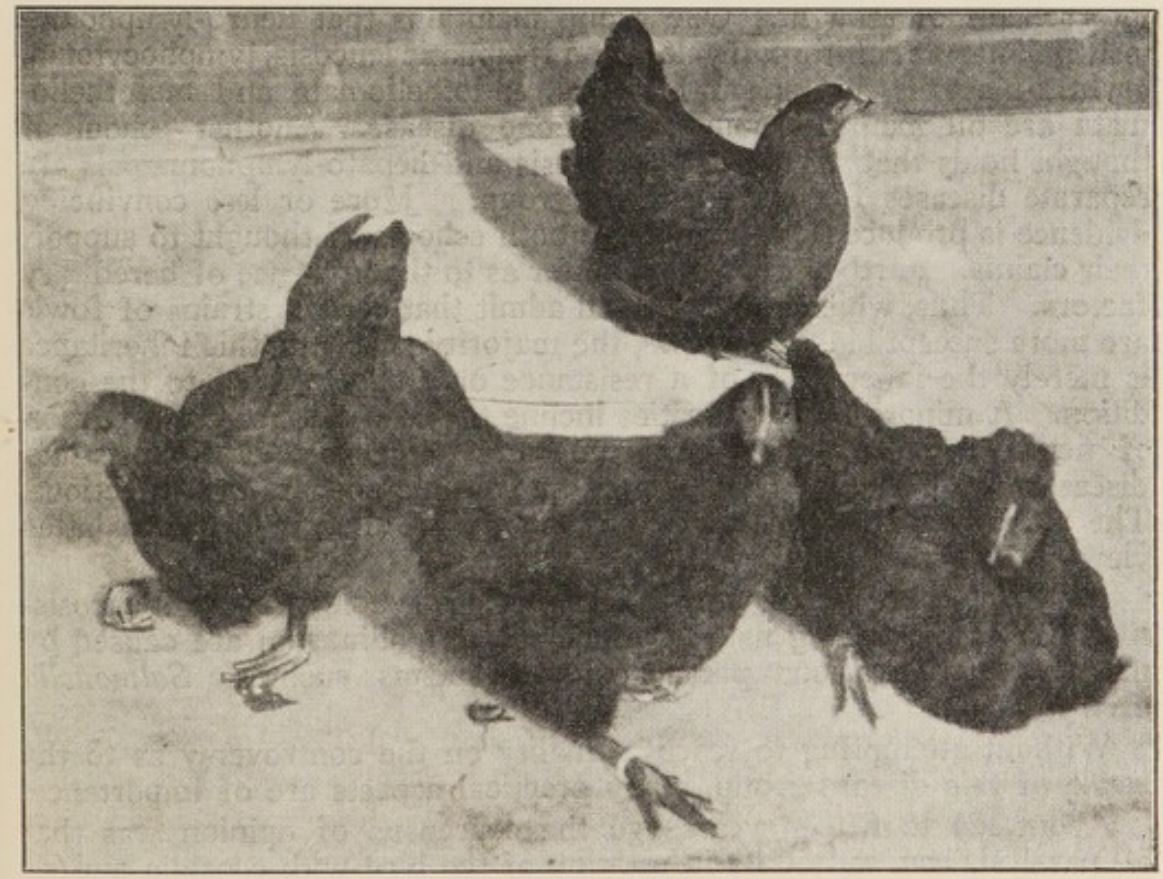
LEG WEAKNESS AND WASTING DISEASES.

Under this symptom group is classed a large range of diseases. Some only of these, indicated below, are dealt with:—

(1) Leucosis and fowl-paralysis (Neuro-lymphomatosis gallinarum).
(2) Tuberculosis.
(3) Rickets (see Deficiency Diseases, page 151).
(4) Perosis (see Deficiency Diseases, page 155).
(5) Tape worm infestation (see Parasitic conditions, page 172).
(6) Spirochaetosis (see Diarrhoea Diseases, page 130).
(7) Waterbag.
(8) Gout.
(9) Anaemia.

Differential Diagnosis of Diseases in this Group—

In dealing with poultry diseases in the field one frequently encounters epidemics of diseases in which a prominent feature is leg weakness varying from a staggering gait to complete paralysis. Outbreaks of



Australorp Pullets (5 months old).

All these birds are quite unable to stand, and sprawl along the ground when attempting to move. In such cases of paralysis the sciatic nerves are usually enlarged (seen on post-mortem examination). These cases were from a fairly severe local outbreak of the disease.

[Photo. taken by Mr. Freeman, Veterinary Research Station, Glenfield.

disease in which the prominent symptom is wasting or going light, are also common. Differentiation of these diseases is not usually a difficult matter.

A careful post-mortem examination of the gut of some of the affected birds should be made. The presence of round worms and heavy tape worms infestations will produce wasting and partial paralysis. Care should be taken to avoid overlooking infestation with minute tapeworms located in the slimy material covering the mucous membrane of the gut. Paralysis may be caused by spirochaetosis. Septicaemic pullorum disease may cause weakness simulating paralysis and diarrhoea. The presence of febrile diseases such as cholera, injuries, or other acute organic disorders, e.g., nephritis, abdominal tumours and cysts, liver abscesses, should be eliminated.

Heavy infestations with head lice may cause paralysis.

The diet should be investigated to see that no deficiences are present.

If all the above have been eliminated from the diagnosis, and paralysis is present and the bird appears otherwise more or less healthy, the probability is strong that the condition is neuro-lymphomatosis gallinarum or one of the leucosis diseases.

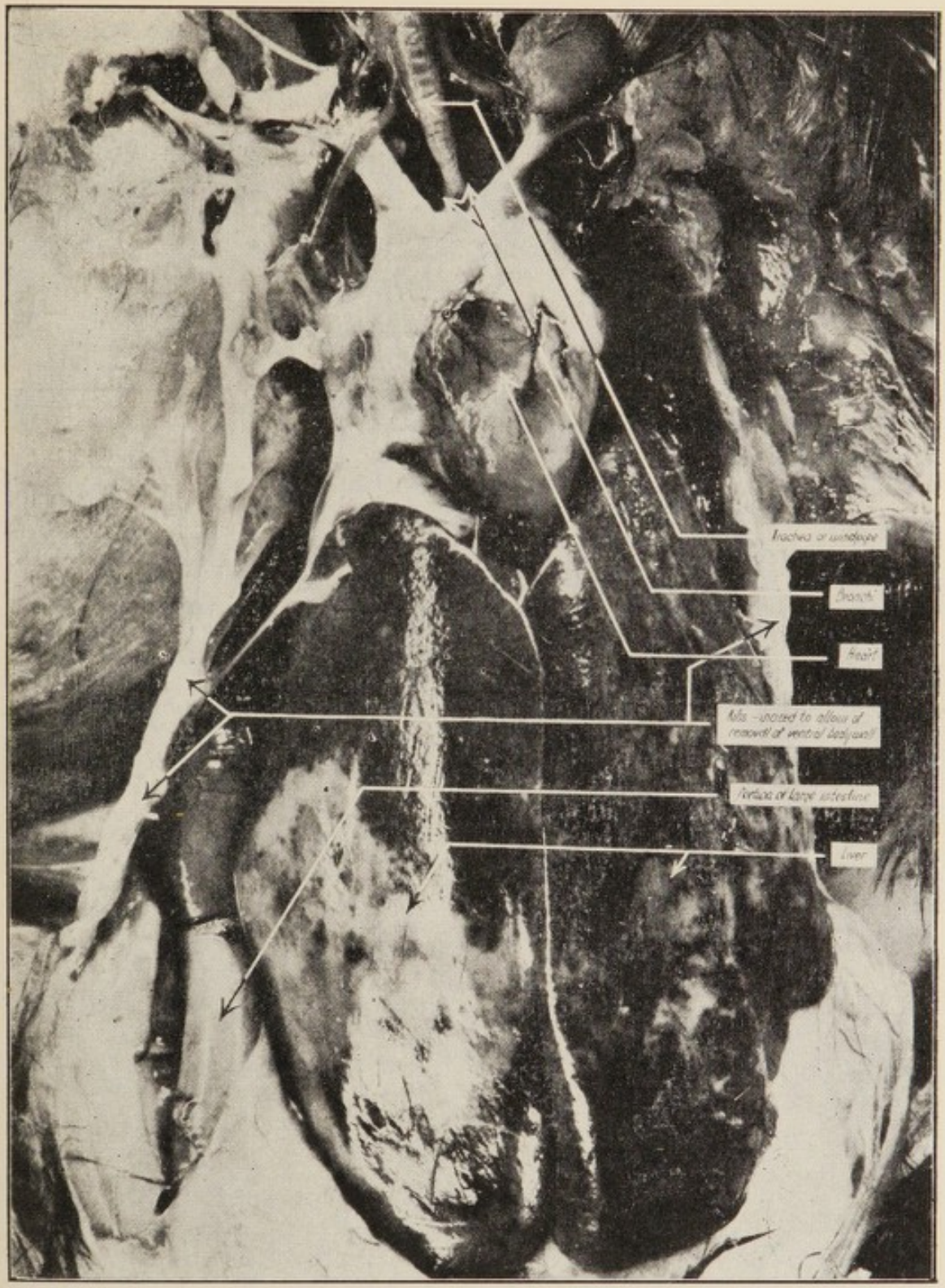
Leucosis, Leucosis-like Diseases, and Neuro-Lymphomatosis Gallinarum (Fowl Paralysis).

The veterinary literature of the world abounds in comprehensive articles on this group of diseases, and leading authorities can be divided into schools of thought. One group maintains that neuro-lymphomatosis gallinarum, erythro, myeloid and lymphatic leucosis, lymphocytoma, eryhtro-blastosis, haemocyto-blastosis, endotheliomata and mesotheliomata are all manifestations of the one disease. Another school of thought holds that neuro-lymphomatosis and hepato-lymphomatosis are separate diseases from the leucosis group. More or less convincing evidence is produced by scientists in both schools of thought to support their claims. Further, authorities differ as to the influence of hereditary factors. Thus, whilst practically all admit that certain strains of fowls are more susceptible than others, the majority hold that this inheritance is merely the inheritance of a resistance or a susceptibilty to the condition. A minority of authorities incline to the view that the condition of neuro-lymphomatosis gallinarum, and some include leucosis-like diseases, are heritable diseases pure and simple and are not infectious. The weight of better informed opinion would indicate that this latter view is incorrect.

Another school of thought, claim that the whole range of leucosis-like diseases, including neuro-lymphomatosis gallinarum are caused by infection with various paratyphoid organisms such as *Salmonella aertrycka.*

Without attempting to reach a finality on the controversy as to the cause of this disease group, many practical aspects are of importance.

About ten to fifteen years ago the consensus of opinion was that all paralysis was caused by infestation of the bird with coccidia and/or other internal parasites. It has been shown that this is quite incorrect. Since then various authorities have produced symptoms of leucosis by injecting infective material from cases of neuro-lymphomatosis gallinarum and vice versa. The criticism of this has always been that it cannot be absolutely disproved that the birds used were absolutely free from any taint of infection or inherited susceptibility to the disease under investigation.



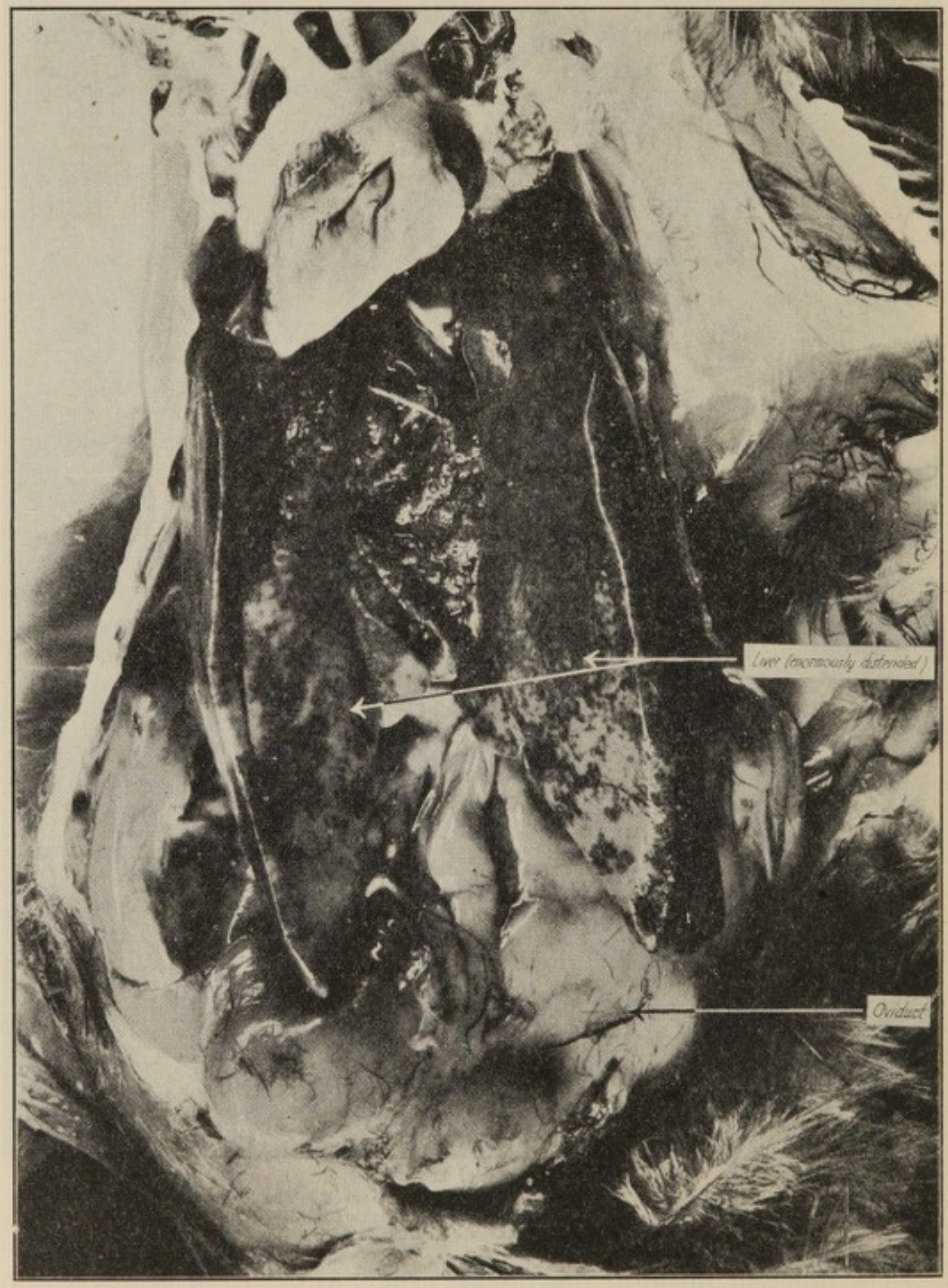
A Case of Leucosis.

Note the enormously distended liver, which is patchily discoloured. The lower portion of the right side of the liver has been ruptured, causing internal haemorrhage and death in this case. Many cases of leucosis terminate in this way.

The different forms of this disease group, as described overseas, are becoming increasingly common locally.

In the space of this small volume it is not possible to do justice to the information contained in the publications of many hundreds of authors who have come to differing conclusions. Those statements and conclusions which seem to fit in with the facts observed here are

freely drawn upon. In view of the progress of knowledge regarding this disease group, it is inevitable that the information published here will be out of date by the time it has left the printer's hands.



Another View of a case of Leucosis, showing Affected Liver.

Identity of the Disease—

It is not possible to define here the various symptoms which are grouped together under numerous names by different reliable authorities. For the purpose of convenience, all these associated diseases will

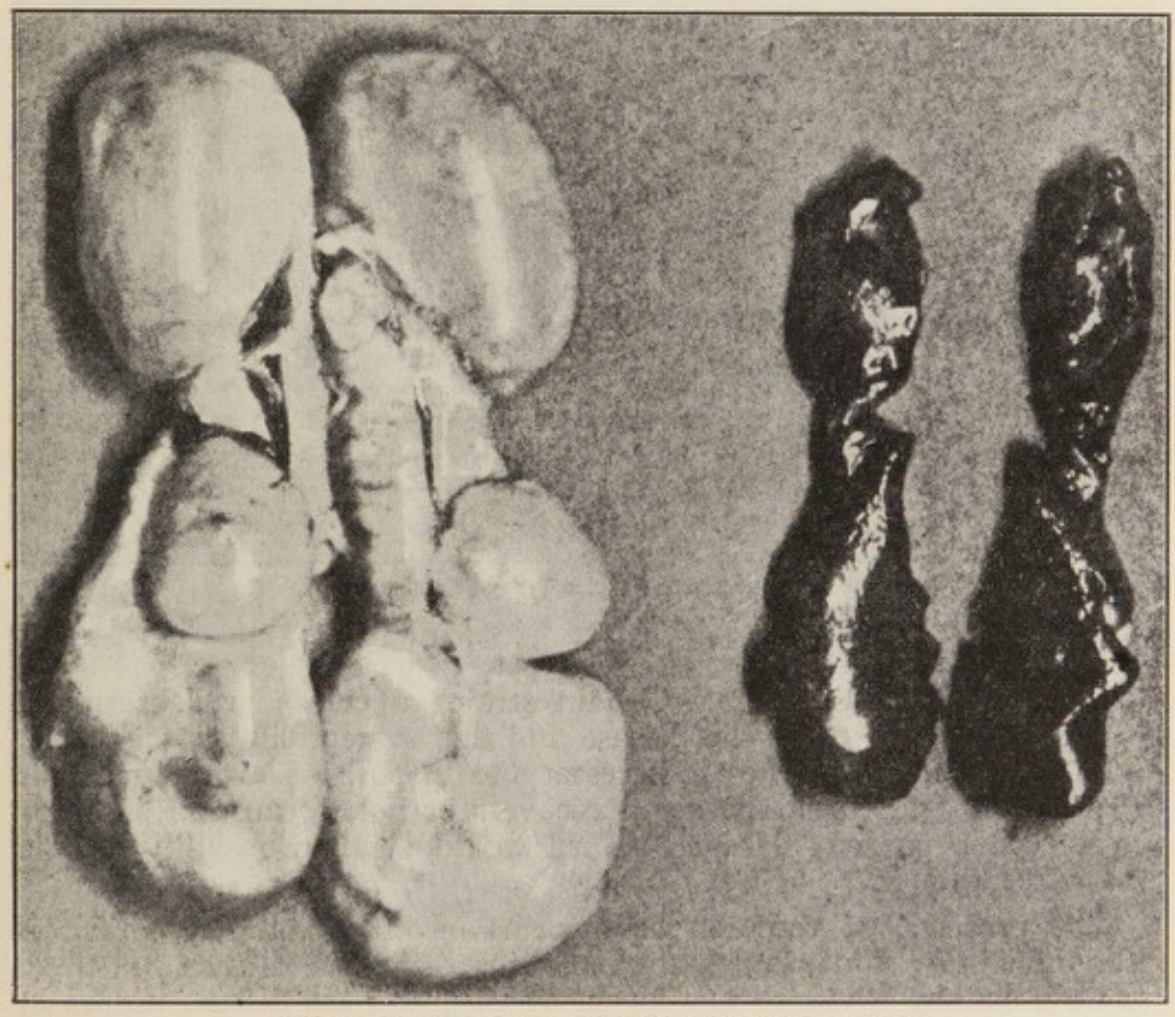
be dealt with as leucosis and leucosis-like diseases and in this designation erythro-, myeloid-, lymphatic-leucosis and neuro-lymphomatosis gallinarum are specifically included.

Synonyms—

Wasting disease, fowl paralysis, "going light," leucaemia, lymphocytoma, lymphomatosis, neuro-lymphomatosis gallinarum, hepato-lymphomatosis, erythro, myeloid and lympatic leucosis, myelocytomatosis, endothelioma, range paralysis, haemocytoblastosis, haemocytoblastoma, mesoblastoma, sarcomatosis, fowl sarcoma, erythroblastosis granuloblastosis.

Cause—

(*a*) *Actual.*—A virus or virus-like micro-organism would appear to be the cause of all the varying forms of the disease. Against this statement it is only fair to add that of works consulted by authors who



Kidney of Leucotic Fowl and Normal Kidney for Comparison.

Note enlargement and paleness of diseased organ.

[*After Fitch.*

have reviewed the literature and also done critical experimental work, twenty-seven different publications have been noted which incline to the view that fowl paralysis (neuro-lymphomatosis gallinarum) is a separate disease condition from leucosis.

Other authorities hold that the disease is cancerous in nature. One investigator (with his co-workers) claims to have shown that the different forms of the disease are due to a certain group of bacteria known as the Salmonella group.

Leucotic Liver—Nodular Type of Swelling.

[After Fitch.

(*b*) *Predisposing Cause.*—Certain strains of birds are susceptible to the various forms of the disease and this susceptibility is heritable. It is this fact that makes it necessary to dispose of every bird that ever shows symptoms whether it recovers or not, because if used for breeding the progeny of such a bird would in all probability inherit the susceptibility.

The presence of coccidia, tape-worms or other internal parasites, it is thought by some, favour the establishment of the disease, but there is little definite evidence in support of this, and the disease occurs in parasite-free birds.

Infectivity—

It has been shown repeatedly that by injecting ground-up suspensions of organs affected with one form of the disease into susceptible birds any or all of the other forms of the disease may be set up. The disease is also readily transmitted by running susceptible birds on

ground previously inhabited by infected birds. There is much reliable evidence to indicate that the disease is transmitted through the egg. When a needle or swab used for vaccination for fowl pox is infected when vaccinating an affected bird, it has been shown that this needle or swab may then infect a large number of birds subsequently vaccinated.

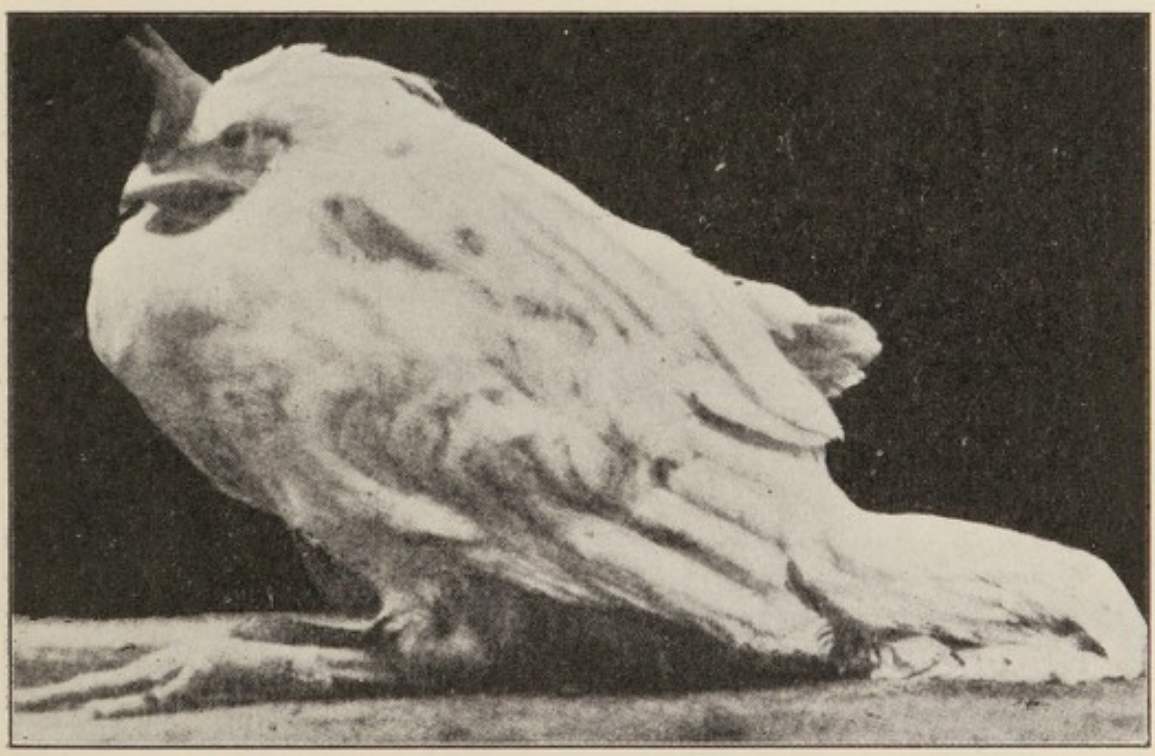
The disease has been transmitted by the bites of red mite, and by the feeding of infected faeces. It has been demonstrated that mosquitoes may transmit the disease from bird to bird, and it is probable that mosquitoes play a large part in mechanical transmission in this State.

Birds Susceptible—

It would appear that each different variety of bird life has its own leucosis, though little critical work has been done on this point. Some birds which recover from one attack of the disease resist further infection, but some succumb to it.

Course of the Disease—

The incubation period varies enormously. It has been observed to be as short as a week and in other cases longer than six months. This



Bird Experimentally Infected and showing Advanced Symptoms of Leucosis

variable incubation period has often produced much confusion in experimental work. Usually the incubation period is from two to four months.

Symptoms and Post-mortem Appearances—

A wide range of varying symptoms is exhibited. Thus in the type commonly referred to as fowl paralysis, the causal virus affects the nervous tissue, and the symptoms shown will vary according to the

different nerves affected. Where the nerves which supply the muscles of the wings or legs are affected, the wing or leg will become paralysed. At first there may be loss of control, and then perhaps a rigid condition of the muscles, which is followed by a flaccid limp condition. Sometimes in milder cases the birds walk with a peculiar hesitant, stilted gait. In other cases the walk is uncontrolled and staggering. If the nerves supplying the neck muscles are involved, the neck may be held in a twisted position, or the bird may "weave," and bury the head in the feathers.

Where the sympathetic or parasympathetic nerves which supply the gut are affected, constipation and then diarrhoea result. If the virus affects the brain and spinal cord, quite a wide range of symptoms may be shown. In such cases the iris (the coloured portion) of the eye may be pale and milky in appearance and the pupil may be distorted.

In one of the worst four outbreaks of this form of disease in New South Wales, the pullets showed only paralysis of the legs, and death; in another twisting of the neck and paralysis of the legs, with rapid deaths (within two or three days) were noted; in the third, the commonest symptom was paralysis and drooping of the wing; and in the fourth outbreak all the above symptoms were shown. Usually, affected birds show an increasing degree of paralysis, become emaciated, and die. Some may gradually recover from the symptoms.

White Leghorn Hen, from a Suburban Flock, showing Lymphomatosis of the Eye.

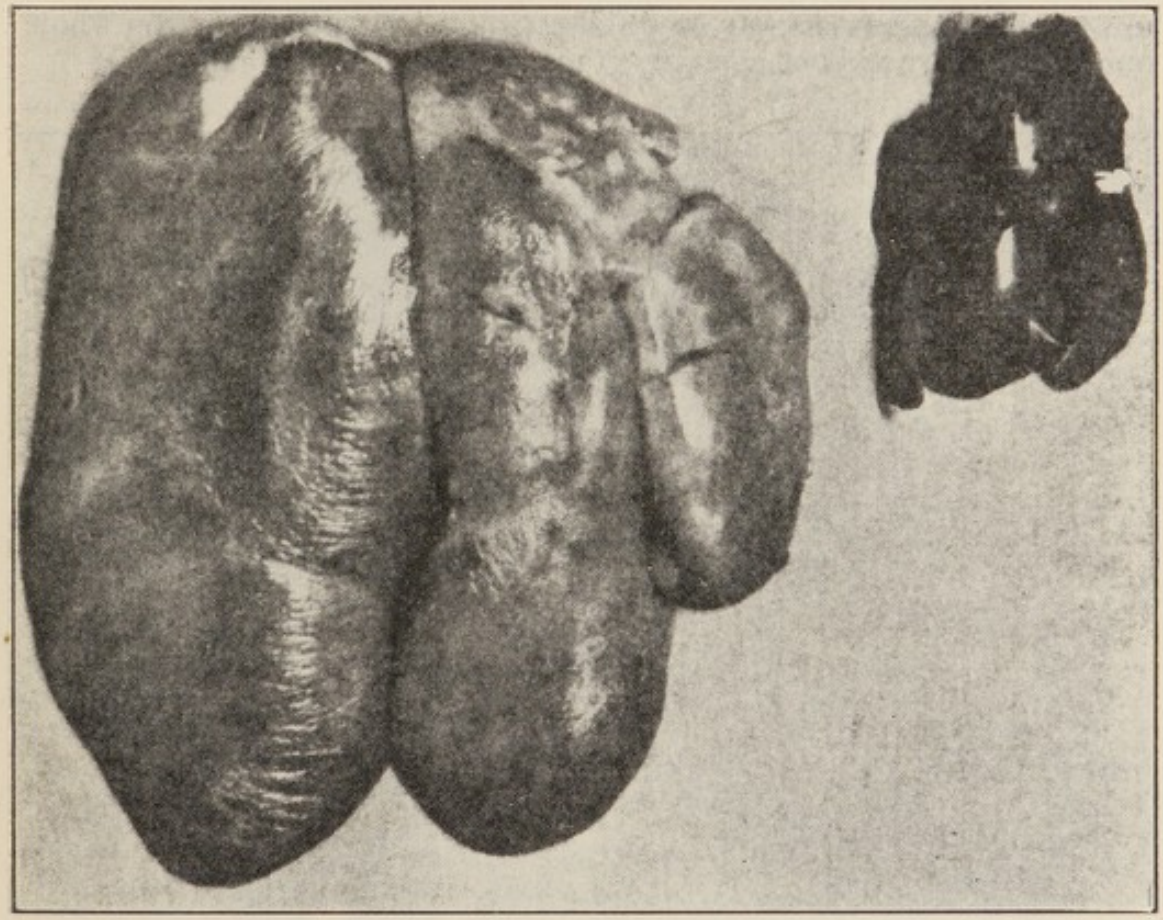
The iris (coloured portion surrounding pupil) is greyish-milky coloured (instead of orange). The pupil is smaller than normal, and does not vary normally with changing intensities of light.

On post-mortem examination no marked abnormalities may be noticeable. A critical examinaiton may reveal that the affected nerves are swollen, *e.g.*, where the legs are paralysed the sciatic nerve may be much thicker than normal and where the wing is paralysed there may be a marked thickening of the brachial nerve plexus.

Types of Leucosis—

Odd cases of fowl paralysis were seen commonly in numerous flocks in 1935 and 1936, but since then there has been a large increase in the incidence of the disease and also of the other forms of it referred to as "wasting" disease," technically known as erythro-, myeloid-, and lymphatic-leucosis.

The technical details of these conditions are of no value to the poultry farmer except in one instance, viz., where he submits a bird affected with one of these conditions to a laboratory, and although the correct diagnosis may be returned to him, he will not be able to understand the diagnosis unless he has some knowledge of the disease. A brief summary is, therefore, given of the types of leucosis.



Enlarged Liver from Leucotic Fowl of Similar Weight for Comparison.
Note paleness of affected organ.
[*After Fitch.*

1. Erythro-Leucosis—

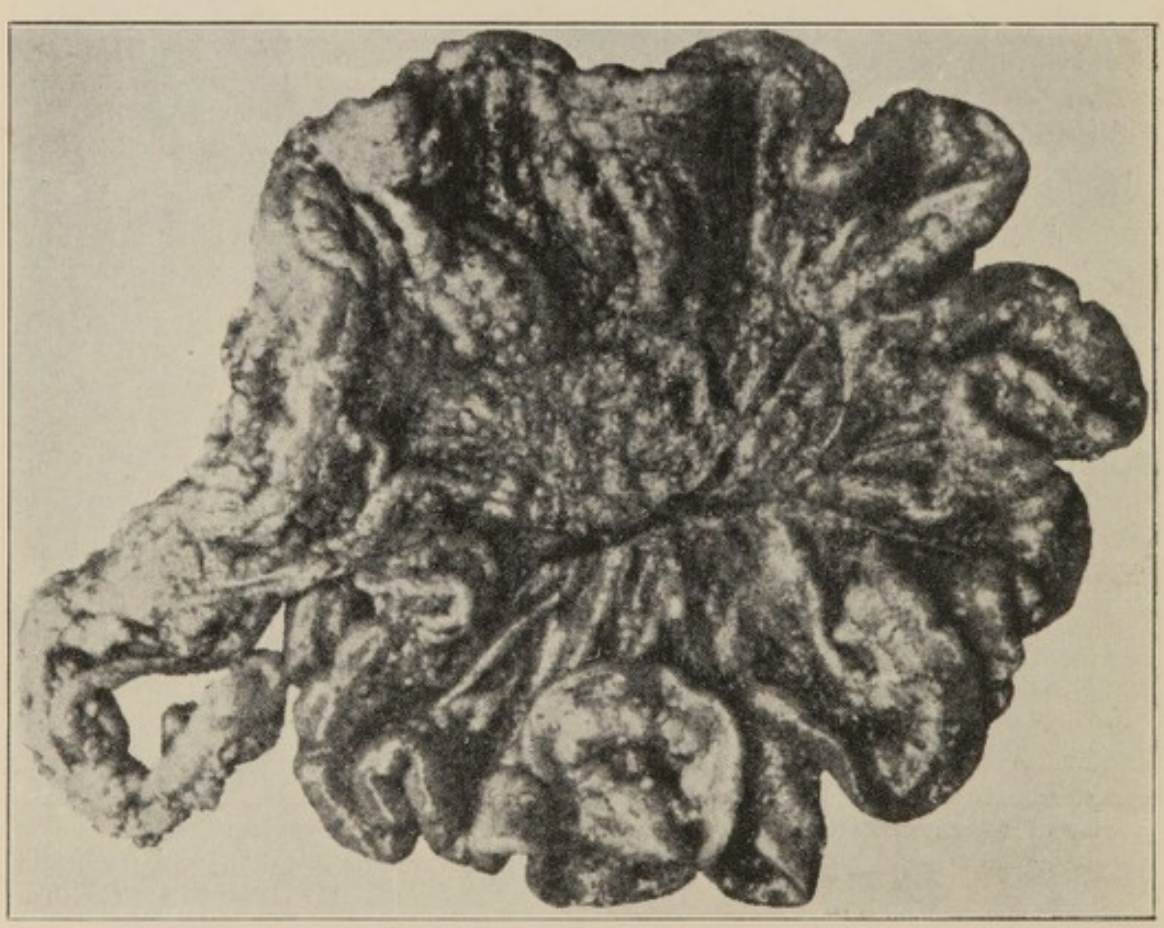
In this condition there is a marked anaemia. When the blood is examined microscopically an enormous number of immature red blood cells are seen, particularly in the tiny blood vessels(capillaries) of the bone marrow, liver and spleen. Haemorrhages are often present in the small intestines. The bone marrow occupies more of the cavity of the bones than usual and has a greyish discolouration.

2. Myeloid (Myelogenous) Leucosis—

In this type of disease, the blood-forming tissues of the bone marrow undergo a cancerous-like growth, and this results in the characteristic type of cells appearing in the blood stream in great numbers, so that the proportion of white to red blood cells becomes altered; there is often as many as one white to every two red blood cells, instead of the approximately normal proportion of one white blood cell to every hundred red blood cells. The myeloid cells may also accumulate in various organs of the body giving rise to tumour formation.

3. Lymphoid- or Lymphatic-Leucosis (sometimes known as big liver disease)—

In this type, when the diseased internal organs, particularly the liver and spleen, are examined under a microscope, enormous numbers of immature blood cells with a characteristic appearance (lymphocyte type of cell) are seen. These cells may be grouped together to form tumour-like masses, or may be diffuse, producing swelling of the whole organ. This type is not as easily transmitted as types 1 and 2.



Intestines and Peritoneum covered with Flattened Nodules.
This condition is often mistaken for tuberculosis.
[*After Fitch.*

A marked enlargement of the liver is a prominent symptom, the organ being flecked with pearly coloured patches of pin-head size. Frequently, the spleen also is enlarged. Marked enlargement of the liver and spleen and diffuse whitish yellow discolouration may be seen in any of the three above types.

In the three forms the bird becomes wasted and pale, and sometimes jaundiced. All the obvious symptoms of ill-health develop, the fowl standing about listlessly with shrunken comb, and being indifferent to feed or disturbances of any kind. Diarrhoea is not infrequent. In some cases, the skin bleeds readily following any slight mechanical injury. The symptoms shown are obviously indefinite, and may be confused with such conditons as tapeworm infestation, until a post mortem examination is made.

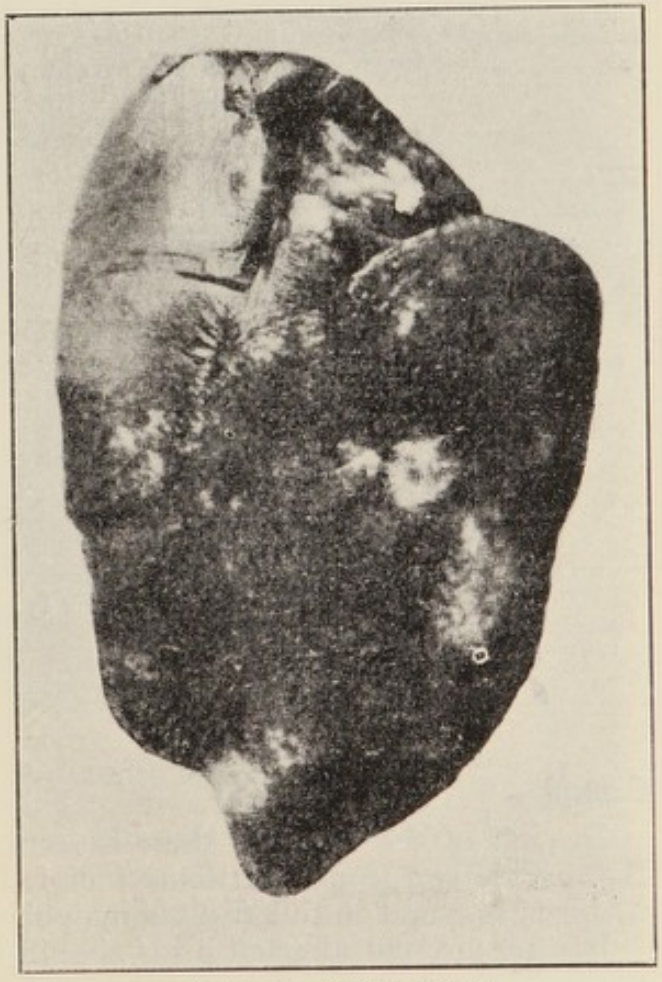
Large tumorous masses may be observed in any of the organs, and are seen quite frequently on the liver, spleen, ovary, kidney, and intestines.

Multiple tumours about the size of peas may occur and are almost invariably confused (by the poultry farmer) with tuberculosis.

It is stated by one authority that bony enlargement of the shank is one symptom of leucosis. In this condition (odd cases of which are seen on most farms from time to time in maturing pullets and cockerels) the shank bones become thickened to two or three times their normal size.

Differential Diagnosis—

A careful search (post mortem) must be made for fine tape-worm infestations, other worm infestations, tuberculosis, spirochaetosis or any obvious causes of wasting or paralysis (*e.g.* injuries, faulty rations causing rickets). Septicaemic pullorum disease in the adult fowl produces a liver enlargement which may be confused with leucosis, and the same is sometimes true of cholera. One cannot over-emphasise the importance of laboratory (microscopic) examination of sections of affected tissue and the examination of blood smears for the positive diagnosis of this condition in doubtful cases. In most leucosis cases, immature forms of blood cells are present and an enormous proportionate increase in diseased or immature blood cells is usually in evidence.



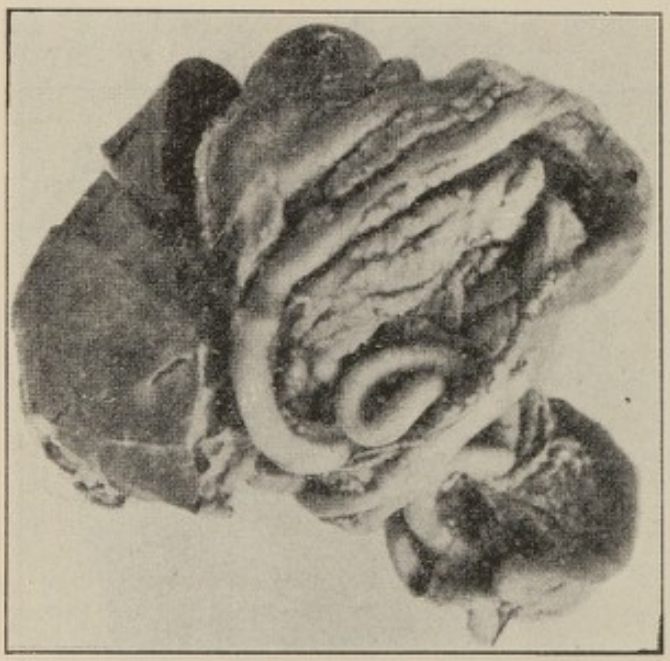
A Case of Ungeloid Leucosis.
Portion of liver, showing pale infiltrated areas.
[*After Fitch*, 1938.

Pathogenicity and Losses Incurred—

Until about 1936 serious mortalities from this disease had not been observed. Several outbreaks have been seen lately in which about 30 per cent. of pullets were affected, most of them succumbing. Contrary to overseas observations about two-thirds of the birds affected with paralysis recover. Birds affected with one of the other forms seldom recover.

In an alarmingly large proportion of commercial flocks there is an intermittent loss of birds from this disease, such birds being regarded as part of the mortality described as "just the normal loss." This "normal loss" is apparently rated in the industry as about 20 per cent. of the flock (above chicken age) annually.

The condition commonly referred to as haemocytoblastosis would appear to occur here. This condition may affect a large percentage of the whole flock of young pullets, causing systemic upset, marked depression of egg laying, inappetence, and being followed by the advanced forms of leucosis as already described, in a small proportion of the flock, the rest apparently recovering. Such an epidemic may cause very heavy loss. For example, in one case suspected to be such an outbreak, a flock of pullets that were costing £6 per week to feed were put right off the lay, the production falling from 60 per cent. to less than 5 per cent. for nearly four weeks, after which it slowly returned to normal, over the next six weeks. A large proportion of the pullets died as a result of the disease.



Leucosis.

Nodular lesions over peritoneum. Such leucotic tumours are often mistaken for tuberculosis by the farmer.

[*After Fitch*, 1938.

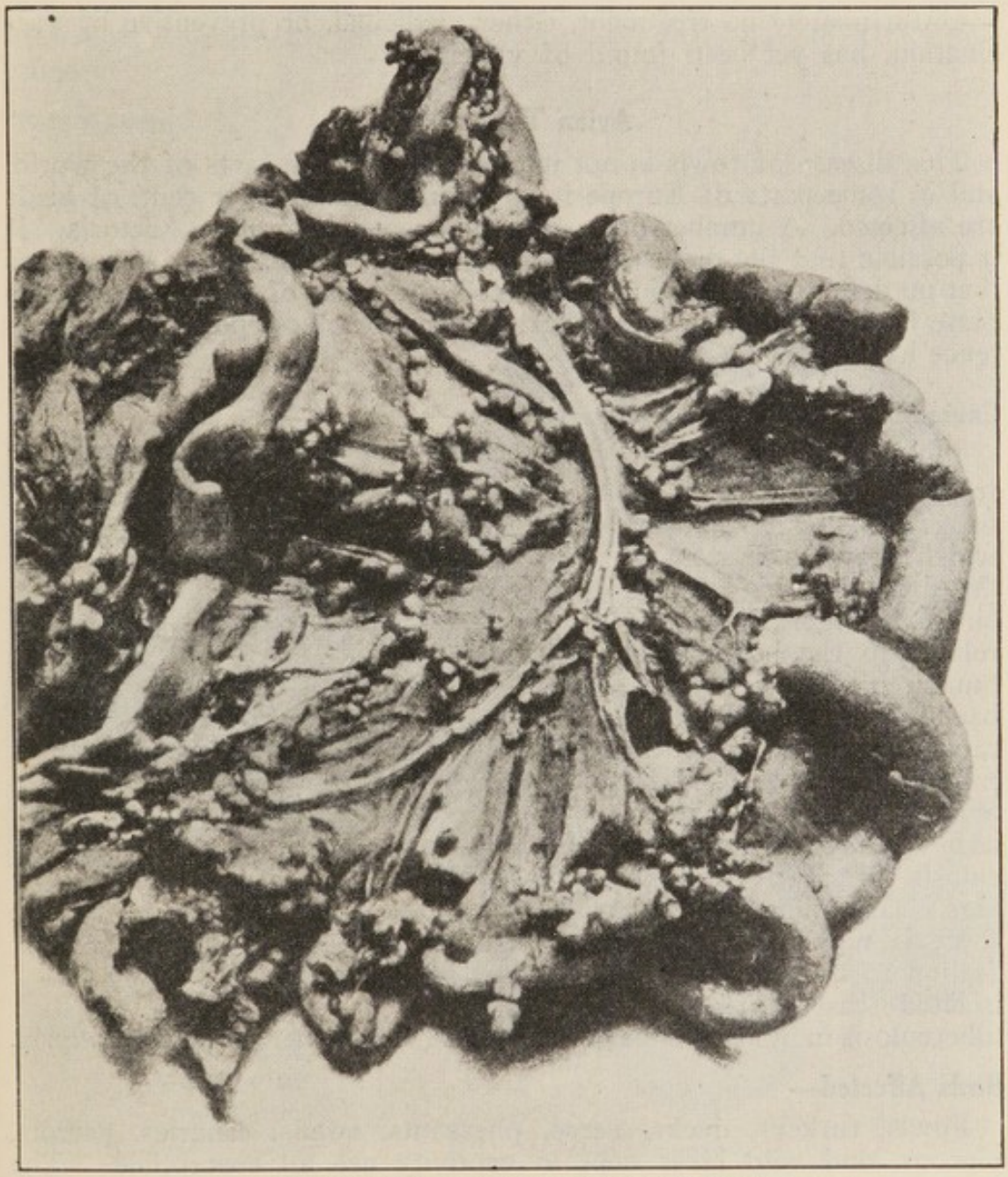
Apart from the above losses, cases of the disease occurring in stock purchased from a stud breeder or large hatchery will naturally cause clients to avoid such stock in future, and so may result in very heavy economic loss to the supplier.

Control—

In view of the fact that there is some evidence that susceptibility to the disease is a simple heritable factor, there is some hope that strains of birds resistant to this disease may be developed in the future.

For the present affected birds should be destroyed or used for table purposes the moment they are detected. Affected birds pass on the infection, and should they recover, they are susceptible to the disease and should not be used as breeding stock.

When the disease is not present in a flock every care should be taken to avoid its introduction. When eggs or day-old chicks are purchased, very careful enquiries should be made as to the freedom from disease of the parent stock. Market crates supplied by a carter to many suppliers may carry infection serially and infected droppings, and should not be brought on to the farm.



Intestines and Mesentery of a Fowl affected with Tuberculosis.

[After U.S.A. Dept. of Agric.

Licensed vaccinators (fowl pox) should be careful to sterilize the instruments used between farms and preferably between say every fifty birds on a farm where the condition does occur.

As the disease can be transmitted through the egg, the danger of its dissemination by the day-old chick trade should be apparent, and great care should be taken by hatcherymen to breed from unaffected flocks only.

Young stock should, as far as possible, be reared on clean ground or ground reserved for them only, and sanitation should be as ideal as possible.

Treatment—

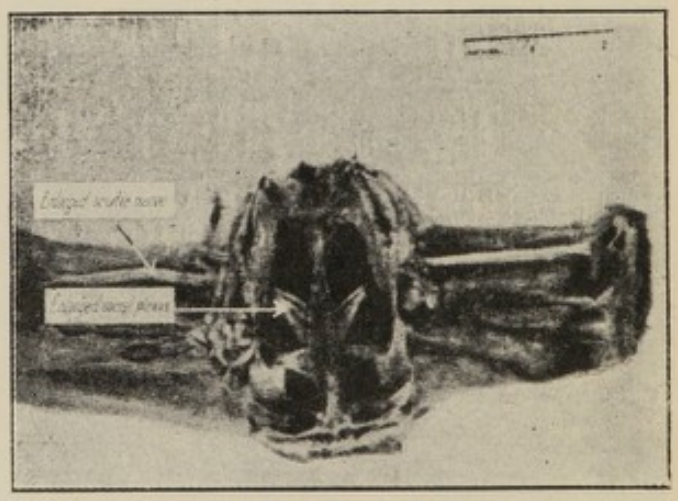
Unfortunately no treatment, either medicinal, or preventive by vaccination, has yet been found of value.

Avian Tuberculosis.

This disease of fowls is not uncommon in other parts of the world, and in some parts of Europe it is reported that 10 per cent. of birds are affected. A number of cases have been diagnosed in Victoria. It it possible that the disease is not rare in this State, but it so happens that no definite information is available. No case of the disease in this State has been seen by the author, nor any reliable report of its occurrence here.

Cause of the Disease—

The cause is the avian tubercle bacillus (*Mycobacterium tuberculosis*) which may be passed in the droppings by infected birds. This germ is closely related to the bovine and human type of tubercle bacilli, but bacteriologically can be quite easily differentiated from them. The avian type of tubercle bacillus may set up tuberculosis in fowls and in pigs. Generally speaking, there is no danger of infection to humans, though isolated cases of avian tuberculosis in humans have been reported from other parts of the world.



Fowl Paralysis.

Kidneys removed and dissection carried out to reveal sacral plexus and sciatic nerves. Plexus and sciatic nerve on left side enlarged.

[*After Craig.*

Birds Affected—

Fowls, turkeys, ducks, geese, pheasants, swans, canaries, parrots, pigeons, and wild birds kept in captivity are all susceptible. The disease is seldom seen in birds under six months of age, and even then the disease does not usually develop sufficiently in the fowl for it to be a spreader of infection until it is twelve months old or over.

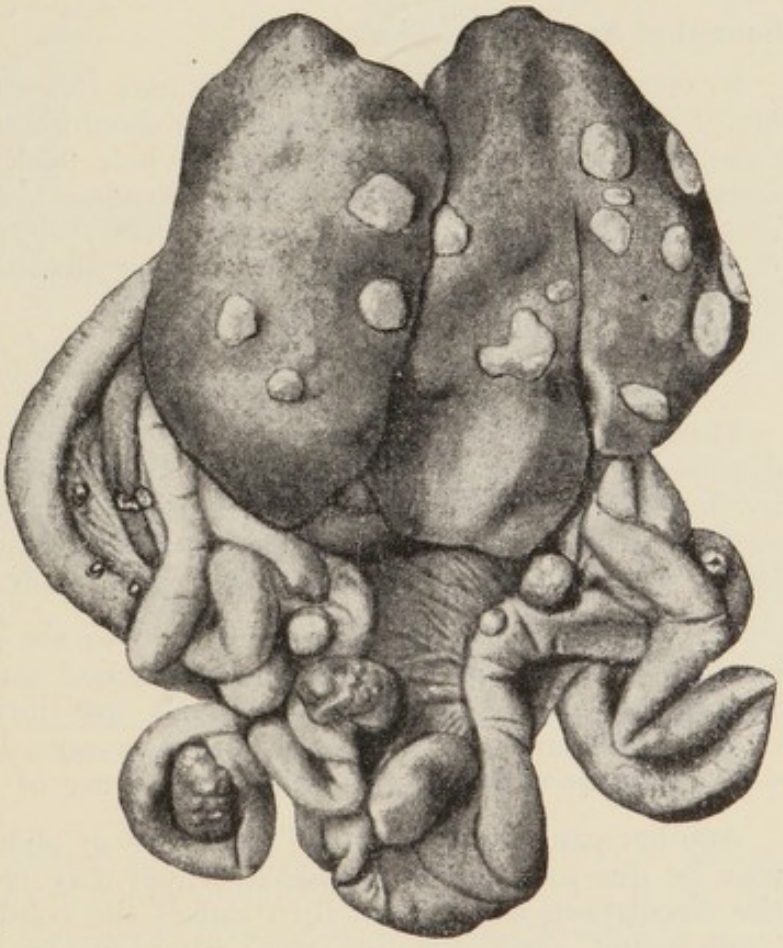
Birds are chiefly infected by eating the tubercle bacilli passed in the droppings by affected birds, and infection is set up in the intestines, liver, peritoneum, or elsewhere. (Infection by inhalation is rare in poultry.)

The disease progresses slowly and the bird gradually wastes away, becoming emaciated and anaemic, or, in the language of the poultryman, "goes light." The comb and wattles are pale and the breast muscles waste away, leaving the breast bone prominent. The bird is listless, weak, and dejected and may have a tuberculous inflammation of one or more of the joints which makes it disinclined to move. The wasting away and general loss of health is due primarily to the production of toxic substances by the bacteria. At first the appetite remains good, but later becomes capricious. Towards the late stages of the disease diarrhoea is a usual symptom, and causes exhaustion, thus hastening death.

Post-mortem—

If the bird is killed and opened typical lesions will be found. Most commonly the liver, intestines and spleen are involved, and show round yellow nodules or tubercles which vary from pinhead to marble size. The very small early pinhead nodule will be white or pearly, but rapidly changes into yellow material, which after a time becomes cheesy in consistence and the whole nodule may then be shelled out quite easily. In other animals lime salts are often laid down in the tubercles, which become hard (calcified). This is very rare in birds.

When there are several tuberculosis nodules in the liver or spleen, the size of the organ will be greatly increased. Nodules in the intestinal wall are easily seen protruding. Some of these may open into the intestine, in which case the droppings are highly infective.



Avian Tuberculosis.
Showing tubercles in the liver, intestines and mesentry.
[After Hutyra and Marek.

The peritoneum (glistening membrane lining the abdominal cavity and supporting the intestines) may be affected, showing numerous nodules. The kidneys, ovaries, lungs, heart and, less frequently, other organs may become infected.

The bones and joints quite commonly show lesions, the yellow colour of the tuberculous material being more easily seen in the form of yellowish patches when the bone is split open. The joints may show yellow cheesy masses and the ends of the bones forming the joint may be eroded.

Differential Diagnosis—

It is not to be thought that every fowl "going light" is a case of tuberculosis. Far from it. The most common causes of a bird going light are indicated in the description of the other diseases dealt with in this section. When in doubt veterinary advice should be sought. Many cases of alleged "tuberculosis" have been brought under notice, but have been small multiple tumours which closely resembled tubercules. Laboratory examination is necessary to confirm the diagnosis of tuberculosis in many cases. A veterinarian may, of course, make use of the double intradermal tuberculin test to diagnose the disease in the living bird.

Control of Avian Tuberculosis—

In view of the fact that the disease may cause most serious economic loss if it once becomes established, care should be taken to have doubtful cases diagnosed. Following this the whole flock may be tested by the intradermal tuberculin test. The avian tubercle bacilli is found to live long periods (as long as two years) and still remain infectious. An early diagnosis and eradication of the disease is, therefore, very desirable.

"Waterbag."

This a well-known condition in which the fowl's abdomen is markedly distended. As a result of this, and sometimes as a result of injury to the sacro-sciatic ligaments, the abdomen is carried in an almost vertical position, with the posterior portion either in contact with, or barely clearing the ground, as the bird progresses.

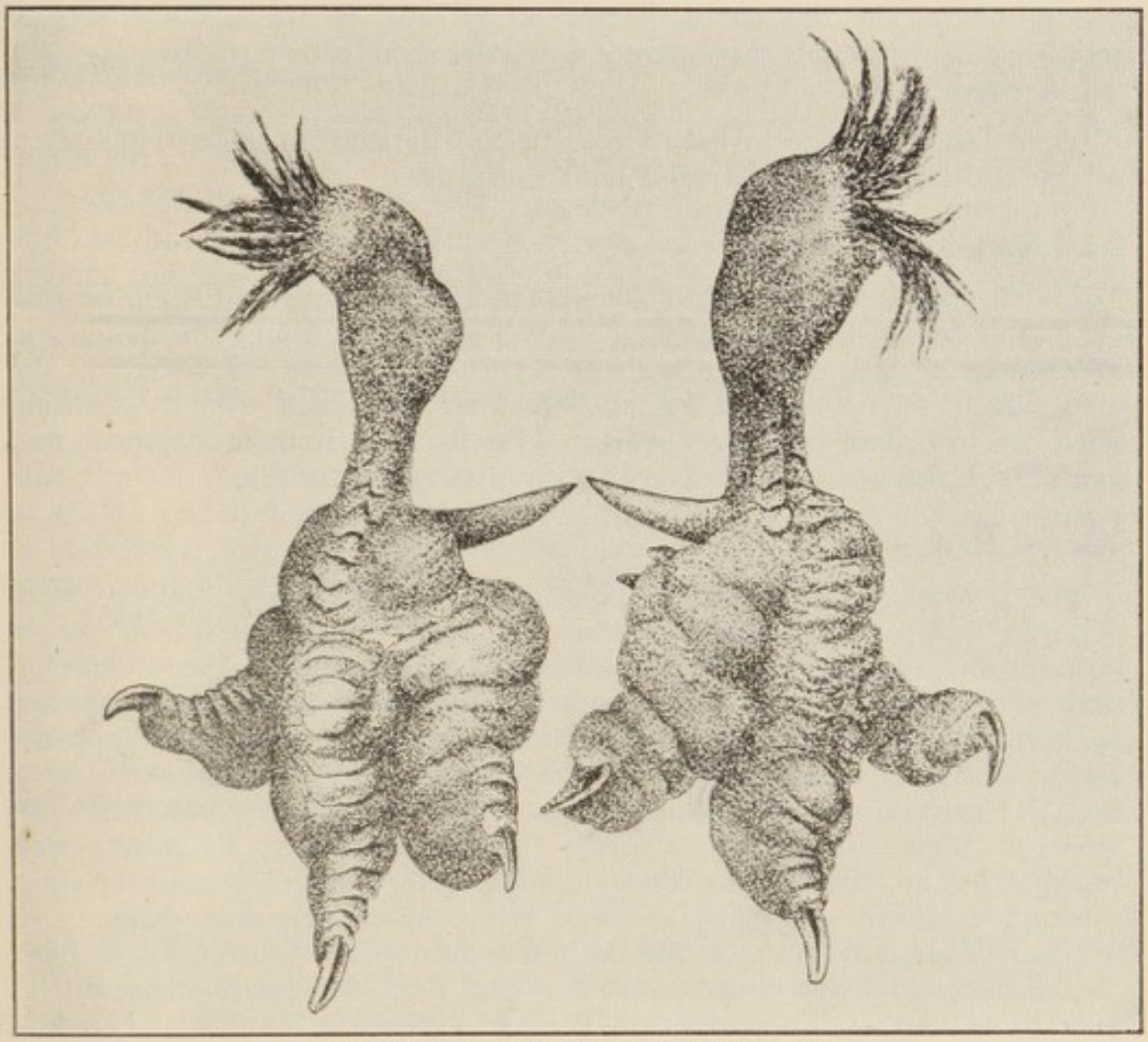
The condition is most commonly due to the development of large ovarium cysts which may become detached and float freely in the abdominal cavity. When these cysts are incised on post-mortem examination large quantities of fluid flow out. Infection of the ovary with pullorum disease is sometimes the cause of cyst formation.

Another cause of "waterbag" is ascites or abdominal "dropsy." This may be due to some liver disease which may result in obstruction of the circulation, and this in turn causes the condition of ascites. The "yolk bound" condition (see later) where masses of yolks accumulate in the abdominal cavity may also bring on similar symptoms.

Gout in Poultry.

This disease is seen to affect odd birds from time to time. It may be confused with other important disease conditions which are infectious, and it is, therefore, necessary to be able to differentiate between this condition and others of more importance.

Gout is due to the deposition of urates in various portions of the body. These urates are derived from uric acid, which comes from the breakdown of nucleo-proteins. These nucleo-proteins may come from the food which is eaten or from the tissues of the bird's own body. Why these normal food constituents should meet this abnormal fate is a little difficult to understand. It may be due to some defect in the normal function of the kidney, to excessive destruction of the tissues of the bird for some particular reason (other concurrent disease), or to



Legs of Cock, showing Lesions of Articular Gout.

[After Ward and Gallagher.

the over-feeding of animal protein material which is rich in nucleo-proteins. Thus the cause is not completely understood, but it has been observed that several conditions predispose birds to this disease.

Predisposing Conditions—

In one instance recorded overseas, hens were fed exclusively on a diet of minced horse meat which had been freed from fat, and within the space of three to fifteen months every birds become affected with gout in some form.

It has been found that if birds are kept in damp, cold conditions and given little exercise, the disease is likely to occur.

Experimental ligation of the ureters (the tubes which connect the kidneys with the cloaca) will sometimes produce gout, demonstrating the relation between this disease and abnormality of kidney function.

Often the only time gout is observed is when fowls are slaughtered for table purposes, or when a post-mortem is conducted in the course of other disease investigations, and unless it is recognised as a clearly separate disease confusion may result.

Where birds are run under conditions closely approximating their natural state, the disease does not occur, and it must, therefore, be regarded as primarily associated with bad conditions of feeding and management.

There are different forms of the disease depending upon which part of the body becomes infiltrated with the urates.

Joint Gout—

There may be a deposit of urates in the cartilages of the toe and leg joints and in the surrounding tissues of the bird, and this results in inflammation with swelling and tenderness. This is evidenced by the bird standing first on one leg and then on the other, and by walking with an irregular or hoppy gait. The pain and inconvenience, and presumably loss of sleep, may result in emaciation.

Visceral Gout—

The pleura, peritoneum and pericardium may become covered with deposits of urates. The surface of the liver, intestines, spleen, lungs, kidneys and the heart may similarly be covered, so that the abdominal cavity (on post-mortem examination) has the appearance of being dusted with chalk; or, at other times, the urates have a more cheesy appearance. The pericardium may be covered with this material on the outside and the inside. In the latter case it becomes attached to the heart muscle. The kidneys show fairly marked changes, in most cases being paler in colour than normal, and having white crystalline spots throughout their substance. These white spots are also deposits of urates. This condition of the kidneys cannot be differentiated from the similar condition due to acute green feed deficiency disease. In both these diseases the ureters will also be abnormal and are noted as hard round tubes filled with urates.

All of these symptoms may not be seen in any one case of visceral gout, but will vary with the severity of the disease. In very severe cases deposits of urates may be seen in the liver and spleen. Mild inflammation of the intestines may also be present.

Symptoms—

The cases of joint gout will be easily noted as described above, but cases of visceral gout may show no symptoms whatsoever, the first indication of the disease being found after killing the bird. At times visceral cases may show all the typical symptoms of general metabolic upset—darkness of the comb, thirst, ruffled plumage, inappetence and depression. Such cases as this may die within a week of showing symptoms.

Preventive Measures—

The methods of feeding and management should be critically examined and all errors rectified. It is important not to feed excess of animal proteins, and if meat meal is the source of protein, 6 per cent. is sufficient, as a protein supplement to the rations normally used in this state for laying stock. Ample green feed of a good succulent quality should be provided, and the birds should be encouraged to take exercise by being placed on open range, or, in intensive and semi-intensive systems, by placing their grain in litter so that they have to scratch for it.

Differential Diagnosis—

Bumble Foot.—See later. Joint gout may be confused with this condition.

Tuberculosis.—This disease has been dealt with (see page 124). and, as there described, cheesy tubercles may be seen in the abdominal organs and mesentery. Cases of visceral gout may be easily confused with abdominal tuberculosis, and when in any doubt whatsoever veterinary advice should be sought.

Tuberculosis of the joints may also be confused with joint gout.

Green Feed Deficiency Disease.—This is described in full on page 42. The typical symptom is the presence of yellow pimples down the back of the throat and gullet. One lesion of advanced cases of this disease is, however, whitish, crystalline spots through the substance of the kidney and filling of the ureters with urates. This lesion is also seen in visceral gout.

Septicaemic Pullorum Disease in the Adult.—In this condition white necrotic areas may be seen on the pericardium, and cheesy necrotic material may occur on the mesentery, giving the appearance of visceral gout. However, in septicaemic pullorum disease, the other characteristic symptoms will clearly differentiate the two conditions. (See page 94.)

Anaemia.

From time to time cases of anaemia are seen exhibiting paleness and wasting which apparently are not due to any of the diseases already dealt with, nor are they due to parasitic infestation. Such cases are of little economic importance.

DIARRHOEA DISEASES.

[Or Diseases in which Diarrhoea is a Prominent Symptom.]

It will be noted that in most diseases diarrhoea may be a symptom. However, in many diseases this is not the outstanding, pathognomonic or only symptom. Thus the following diseases, in which diarrhoea occurs, are not dealt with here for the reason indicated:—

Worm Infestation.—The worms can be seen on post-mortem examination.

Coccidiosis and pullorum disease (in chickens) are chicken diseases with other characteristic lesions—*q.v.*

Blackhead.—Occurs in turkeys and young fowl with other distinguishing features as already listed.

Septicaemic pullorum disease.—Has characteristic lesions, see page 94.

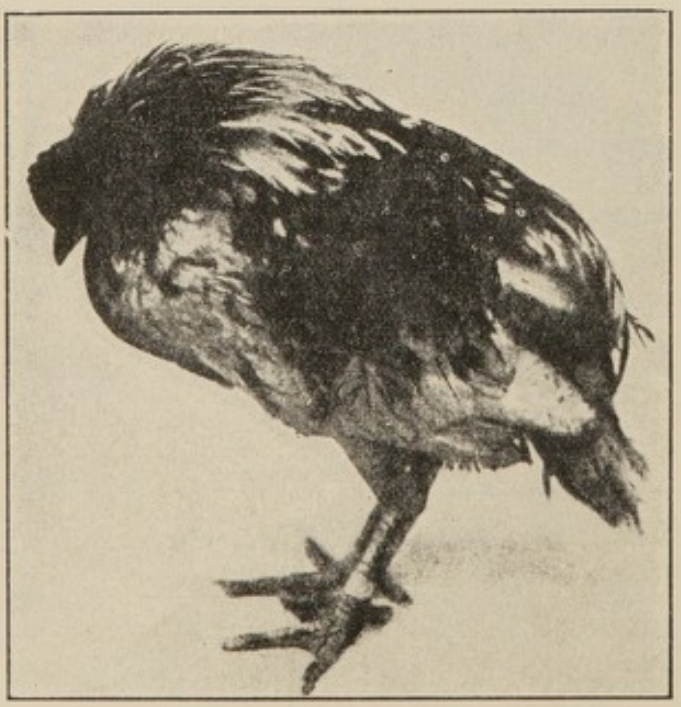
Leucosis is characterised by wasting, paralysis, or liver and spleen changes.

Incorrect feeding is a matter best ascertained after an investigation of the diet.

In the following diseases the outstanding symptom is diarrhoea and this is frequently the only abnormality recorded by the farmer in the event of his writing for information on one of these diseases:—

(1) Spirochaetosis (fowl tick fever).
(2) Fowl Cholera.
(3) Cloacitis or Vent Gleet.
(4) Trichomoniasis.

Fowl Tick Fever (Spirochaetosis).



A Fowl Affected with Spirochaetosis in the Early Stages.
[*After Schwanner.*

Although this disease is most common in the western districts of the State, where the warm, dry climatic conditions favour the development of the fowl tick (*Argas persicus*), quite a number of cases have occurred on the coast. The disease in coastal areas usually follows the introduction of fowls from western districts, though this was not the case in two serious outbreaks which came under notice.

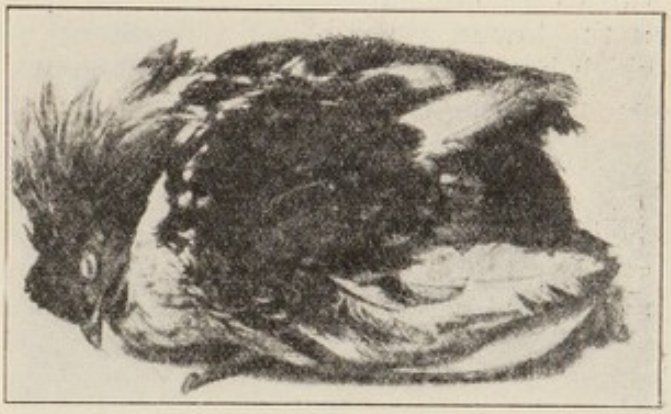
Fowl tick fever seldom occurs without the presence of fowl tick or red

mites to transmit it. At the same time it must be noted that not all tick and mite are affected with the causal spirochaete, *Treponema anserinum.*

Fowls, ducks, geese are all definitely susceptible. Most authorities state that pigeons are so, but some have found them refractory to inoculation. Turkeys are reported (by some) to be susceptible.

The Cause of the Disease—

The cause of this disease is a spirochaete or small animal parasite or protozoan known as *Treponema anserinum.* When blood smears are made frome affected fowls at the height of the disease and examined under the microscope, these parasites can be seen. The spirochaetes can spread from bird to bird only by the agency of external parasites. Both the fowl tick (*Argas persicus*) and the red mite (*Dermanyssus avium*) may act as intermediate hosts. The spirochaetes are able to multiply in the fowl tick, and will be found in the salivary glands of an infected tick. Such ticks remain infective for at least five months after feeding on a "fevered" bird. Ticks can survive long periods—over two years—without feeding. Red mites do not live quite so long without feeding, but will survive three to five months. It appears that red mites act only as mechanical carriers and probably the infection cannot be carried by them longer than forty-eight hours after feeding on an infected bird. It has been shown overseas that mosquitoes act as carriers of the disease. There is some slight evidence to suspect that at times other parasites in addition to those already mentioned may spread the infection in this State.



Spirochaetosis.
Final stage, with paralysis of legs and wings.
[*After Schwanner.*

Should a fowl eat an infected tick or tick eggs, or ingest an infected fowl's blood, infection will take place quite readily.

The Incubation Period—

When a bird is inocluated with infected blood, symptoms will develop in forty-eight hours. In natural infection where the dose of spirochaetes introduced is not as heavy, usually seven to nine days elapse from the time of infection until symptoms are shown.

Symptoms—

Both acute and mild forms of the disease occur.

Acute Form.—In very acute cases the bird may be found dead with no premonitory symptoms; this, however, is unusual. Briefly the picture usually seen in the acute form is that the bird becomes sick (*i.e.*,

mopey, hangs the head, closes the eyes, huddles up with ruffled feathers), shows green diarrhoea, has great thirst, becomes paralysed, then dark in the comb, and usually dies in a convulsion.

When careful observations are made, an affected bird is noticed to be drowsy, and when the rest of the flock comes running over for feed it hangs back.

When a bird dies soon after infection there will be a general congestion of blood vessels and darkening of the comb, but if death is delayed an extensive breaking down of red blood cells takes place, resulting in anaemia, one symptom of which is a pale comb.

In many cases as the head is lowered to pick up feed, the comb and wattles become darkened. This is due to the fact that blood accumulates in the lowest portions as a result of insufficient action of the heart, which has been "weakened" by the acute fever and toxins produced by the infection.

The rapidity with which further symptoms develop varies very much. If the dose of spirochaetes was heavy and the incubation period short, then the train of the symptoms proceeds rapidly, particularly in young pullets. Usually about the same time as the drowsiness is noticed, a profuse green diarrhoea, which aggravates the exhausted condition of the bird, is present. Some five or six hours after first symptoms are noted the bird may stagger in a "drunken" manner or limp on one leg. This partial paralysis develops further so that the fowl may be completely paralysed in both legs and partially paralysed in the wings. Death usually occurs within twelve hours of this.

One of the most constant and reliable clinical signs of tick fever is jaundice with the characteristic yellow tint of the comb, wattles and skin of the body. Moreover if the temperature is taken during an acute attack, it will often be found to exceed 110 degrees Fahr.

Where a heavy infection (due to a large number of infected mites or ticks feeding on the birds) is set up in pullets never previously in contact with this disease, over 80 per cent. may die. In one outbreak in a yard of about seventy pullets, out of which all obviously sick and dead ones had been removed the day before, eight were lying dead, three were lying completely paralysed, and about twenty were drowsy, showing greenish diarrhoea and a variable amount of weakness and paralysis. This will give some idea of the spectacular manner in which the disease may wipe out a flock.

Mild Form.—In the mild or chronic form of the disease birds show depression and general "sickliness." Diarrhoea may be present. In less severe cases the paralysis may not develop until twelve to twenty-four hours after the first symptoms, and then does not usually progress into complete paralysis. Birds which eventually recover seldom go beyond the stage of partial paralysis. They remain partially paralysed for one or two days, showing little appetite, but marked thirst. Appetite returns and recovery is gradual and uneventful except for the fact that anaemia and jaundice may be present. This is noted by a paleness and yellow tint of the comb, face and wattles, which gives them a "waxy" appearance especially in the case of White Leghorns.

In the mild form of the disease, emaciation is often a marked feature.

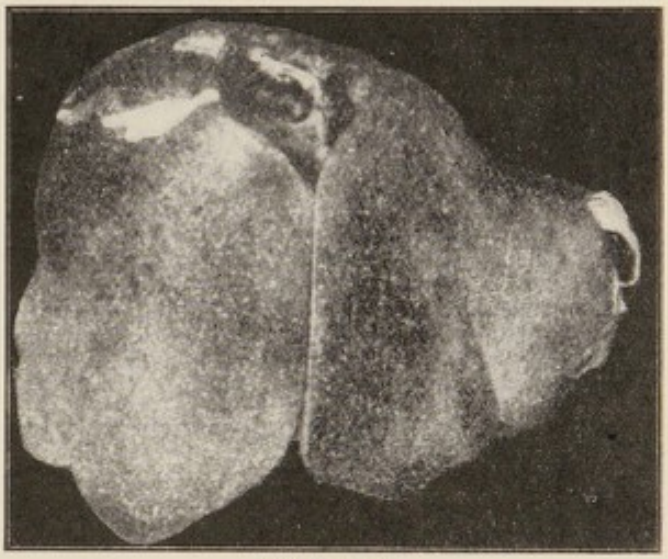
A fortnight may elapse from the first showing of symptoms to the complete recovery of severely affected birds (cessation of diarrhoea, paralysis gone, and bright appearance returned). Such birds will probably go into a moult and cease egg production for some considerable time.

In some cases of the disease, particularly the mild cases, the diarrhoea may be whitish instead of the typical grass-green or yellowish-green colour.

Post-mortem Appearance—

If the bird has been sick for some days, the carcase will be emaciated and the flesh will have a yellowish jaundiced tint.

On opening a bird that has died from the acute disease, most marked changes are noted in the spleen and liver. The spleen is enlarged, sometimes as much as six times its normal size. White patches (of necrotic tissue) from pinhead to millet-grain size are scattered through its substance, and may be seen even before the spleen is cut open. This change in the spleen is quite a constant and characteristic feature.



Spirochaetosis.

Small necrotic foci in the liver of a fowl.

[After Hutyra and Marek and Manninger.

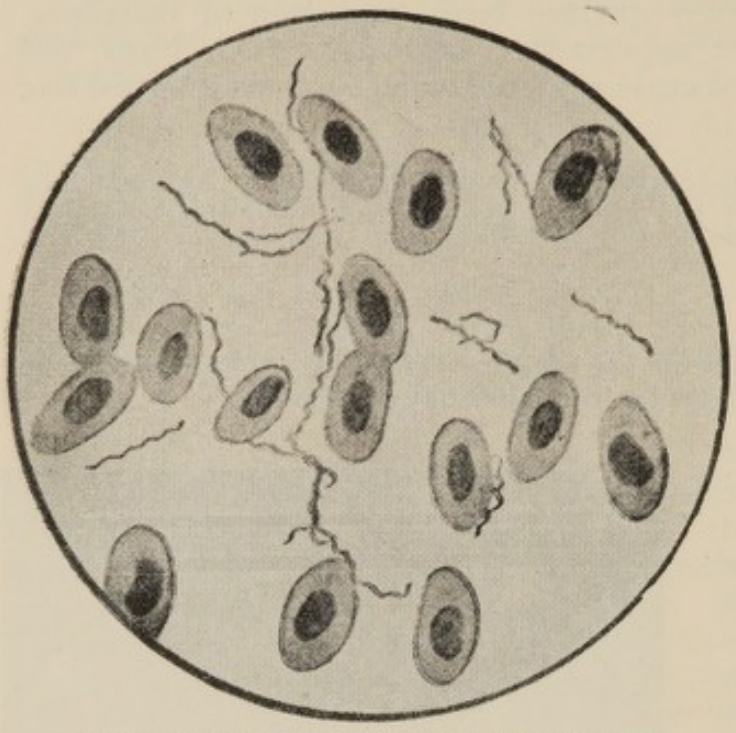
The liver is enlarged (sometimes up to twice its normal size) and is flabby. Frequently—in every case in some outbreaks—there are patches of grey, dead tissue right throughout its substance from the size of a wheat grain to the size of a pea. Several cases have been seen where the liver was a bright green colour, being about the shade of a light-coloured lettuce leaf.

The small intestines are inflamed in some cases and contain green-coloured faecal material. Sometimes subcutaneous haemorrhages may be seen, the result of blood sucking by the ticks.

Age Incidence—

Fowls of any age are susceptible, but when chickens become affected, the mortality is very high, up to 100 per cent. dying in some cases. Fowls that are affected and recover from an attack are immune, and this explains the fact that in districts where infected ticks exist, hens seldom, if ever, become affected, as they have already developed an immunity as pullets.

It appears that there is a large variation in the susceptibility of birds to this disease, as some fowls that have not been previously exposed to infection show quite a solid resistance. It has been shown that eggs from hens which have an immunity to the disease carry immune bodies, especially in the yolk. This results in a variable amount of resistance to the disease in chickens hatched from such eggs. This immunity lasts only a few weeks, eight weeks being about the limit of its effect. Such resistance may persist long enough, however, to allow chickens to be re-infected by ticks or red mites, and so develop a solid immunity. This explains why in bad tick areas stock bred and reared locally may not be affected with tick fever.



A Micro-photograph of a Blood Smear from a Case of Spirochaetosis, showing the Causal Spirochaetes between the Red Blood Cells.

[After Hulyra, Marek and Manninger.

Differential Diagnosis—

There are a number of conditions with which tick fever may be confused, and where it is suspected a careful search should be made for the presence of fowl tick or red mite. These parasites, if present on the farm, will be found under the bark of trees (particularly pepper trees), in crevices of houses or nest boxes, or between perches and their supports. It is very easy to miss these parasites, and a casual examiner is almost sure to do so.

The main diseases which show symptoms somewhat similar to tick fever are briefly as follows:—

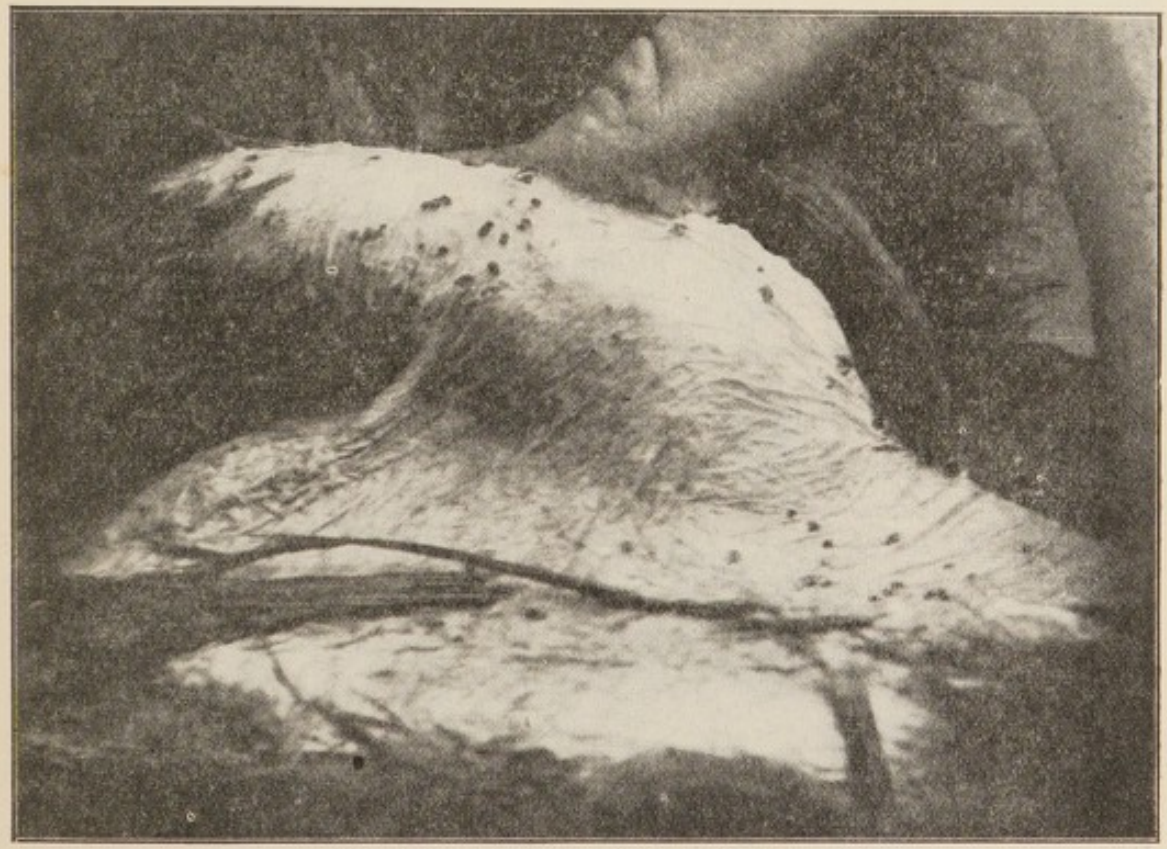
Pullorum Disease (Bacillary White Diarrhoea)—When this disease occurs amongst adult stock they show drowsiness, weakness, but not paralysis, and a whitish or greenish diarrhoea, which may be followed by death. On post-mortem examination, the liver is usually enormously enlarged with quite a range of different colours (black, yellow and greenish) in the patches of dead tissue in it. Some ova are nodular and discoloured, and the spleen shows no marked changes. Pronounced green diarrhoea is not present. Thus the two conditions are fairly clear clinically. On submitting a bird to a laboratory the germ (*Salmonella pullorum*) can be recovered in the case of pullorum disease, and in the case of tick fever the spirochaetes can be seen on microscopic examination.

Fowl Cholera has occurred in this country in the septicaemic form and clinically is very difficult to differentiate from tick fever, as in both diseases a greenish diarrhoea is present. There is usually marked enlargement and discolouration of the liver in fowl cholera. All cases should be sent to a laboratory for accurate diagnosis.

Worm Infestation causes diarrhoea, wasting and anaemia. If an affected bird is killed, the worms will be seen readily, except in the case of the very small tapeworms. In any case the typical liver and spleen changes are absent.

Chronic Coccidiosis.—This will cause diarrhoea, anaemia and emaciation, but the typical liver and spleen changes are not present.

There are many other disease conditions which cause drowsiness, listlessness, diarrhoea, wasting and anaemia, but in none of them will spirochaetes be found when blood smears are taken for examination. If the typical changes of the spleen and liver are present with a very high temperature, large numbers of fowls are affected, and ticks or mite can be found in the sheds, then one can suspect spirochaetosis. In any doubtful cases sick birds in the early stages of the disease should be forwarded to a laboratory.



Engorged Larvae on Breast of Black Orpington Hen.

This photograph was taken during a recent outbreak in County Cumberland.

Prevention—

Recently a method of successful preventive vaccination has been described in Palestine. At the moment this has not been tried in the field here.

Control—

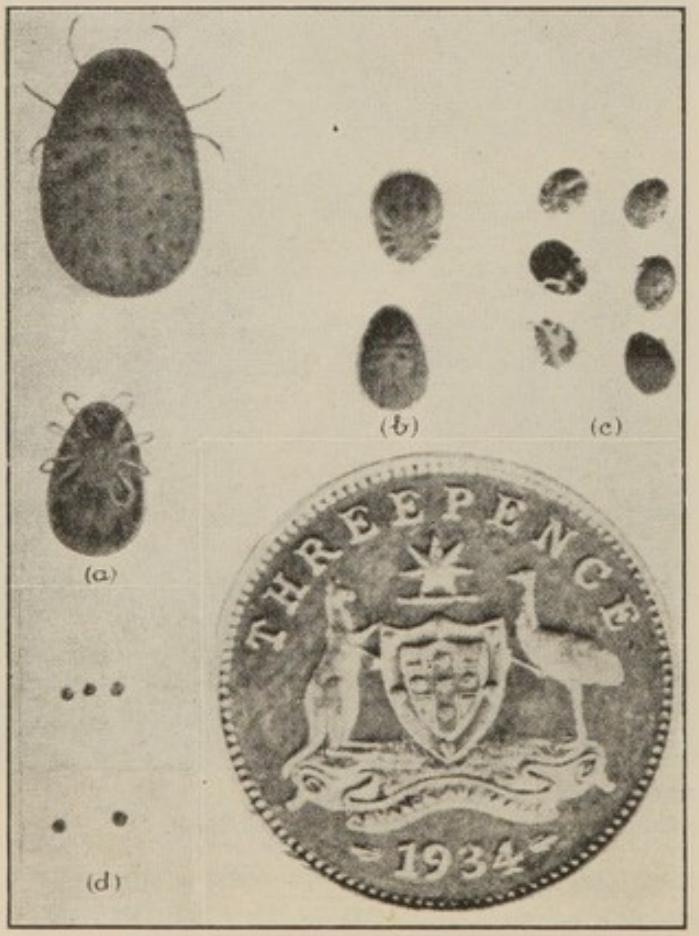
Apart from preventive vaccination (as yet untried here), control should be by eradication of the ticks or mites, without which the disease cannot spread. This is by far the most practical method of control.

Treatment—

Intravenous injections of neokharsivan, neosalvarsan, atoxyl, soamin and other arsenic compounds have been used with success to treat the disease, but these treatments are expensive and, therefore, only warranted in the case of stud birds, and they should be carried out by veterinarians. Neosalvarsan is injected intravenously into the large wing vein in doses varying from 0.005 grams for each kilogram (2.2 lb.) of the bird's weight to 0.005 grams for each pound of weight. Kharsulphan and myosalvarsan are used in the same dosages for intramuscular injection. Mapharsen is used in much smaller dosages.

The Carriers of Fowl Tick Fever.

The parasites fowl tick and red mite are both carriers of the spirochaete (*Treponema anserinum*) which causes fowl tick fever (spirochaetosis).



Showing Size of (*a*) Adults, (*b*) Engorged Nymphs, (*c*) Engorged Larvae, (*d*) Eggs as compared with a "Three-penny Bit."

Specimens from a recent outbreak in County Cumberland

The adult tick is pale yellow in colour, oval in shape, and flat when unengorged. After engorgement the tick becomes thickened dorso-ventrally and greyish-blue or leaden-coloured. The adult tick may be up to one-third of an inch long. The adults and nymphs have eight legs and the larvae six legs. In the adult form the mouth parts do not project beyond the anterior border of the body. The nymphal stage resembles the adult but is smaller. The larval stage is smaller still, and the mouth parts do project beyond the anterior border of the body.

The red mite is a very small parasite which is pale yellowish-white in colour before engorgement, and bright red when engorged. It is

A Piece of Wood from a Fowlhouse, showing how the Ticks Shelter under the Splinters and Crevices.

just visible with the naked eye, being up to one-thirtieth of an inch long. When heavy infestation is present, a tablespoonful or more of these parasites may be congregated under the junction of perches. Differentiation of the different forms of red mite cannot be made with the naked eye.

Whereas the fowl tick is extremely common in New South Wales in the inland districts where warm, dry conditions prevail, such as the Riverina and central-western areas, but not common on the coastal area, red mite, on the other hand, is wide-spread.

Red mite may be seen at one time or another on practically every poultry farm and will appear as soon as carefull preventive measures are relaxed. Under conditions prevailing in this State, red mite is very seldom a carrier of the spirochaete which causes tick fever, but it has acted as a carrier in the coastal district. Fowl ticks do not always carry the spirochaete, but they very frequently do, and it has been shown conclusively that the spirochaetes can multiply in their bodies, whereas this point is in some doubt as regards red mite. Thus where red mite occurs on a commercial poultry farm the farmer should not feel apprehensive of an outbreak of fowl tick fever, though if the spirochaete is in some way introduced, *e.g.*, by a carrier fowl, the red mite will readily transmit it to other birds. It will be noted that the term fowl tick fever is misleading, as the causal spirochaete is at times transmitted by the red mite.

At one time it was thought that fowl ticks (*Argas persicus*) did not occur on the coast, but on numerous occasions they have been found to infest the poultry in this area, sometimes following the introduction of fowls from western districts, and at other times when there has been no such history.

Losses Due to Fowl Tick and Red Mite—

(1) The adult forms of these two parasites cause irritation to the birds at night time by feeding upon them. Further, ticks suck a considerable quantity of blood from the birds, and if in large numbers may cause a severe anaemia. Turkeys are particularly susceptible to this. Fowls, geese, ducks, pigeons and canaries also may be attacked.

Red mite may occur in enormous numbers and will cause a similar condition of anaemia. The larval stages of these parasites remain attached to the bird. The larval ticks may remain attached from five

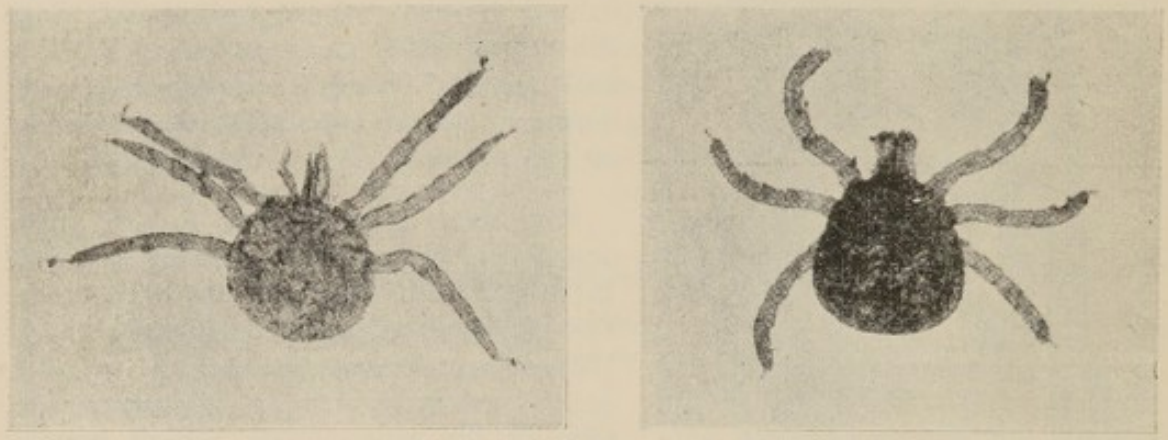


Fig. 5.—Larval Ticks (Enlarged).

Left—Newly-hatched, semi-transparent larval tick before feeding.
Right—Larval tick after having attacked a bird and engorged with blood.

[*From Bul. 74, S. Aust. Dept. of Agric*

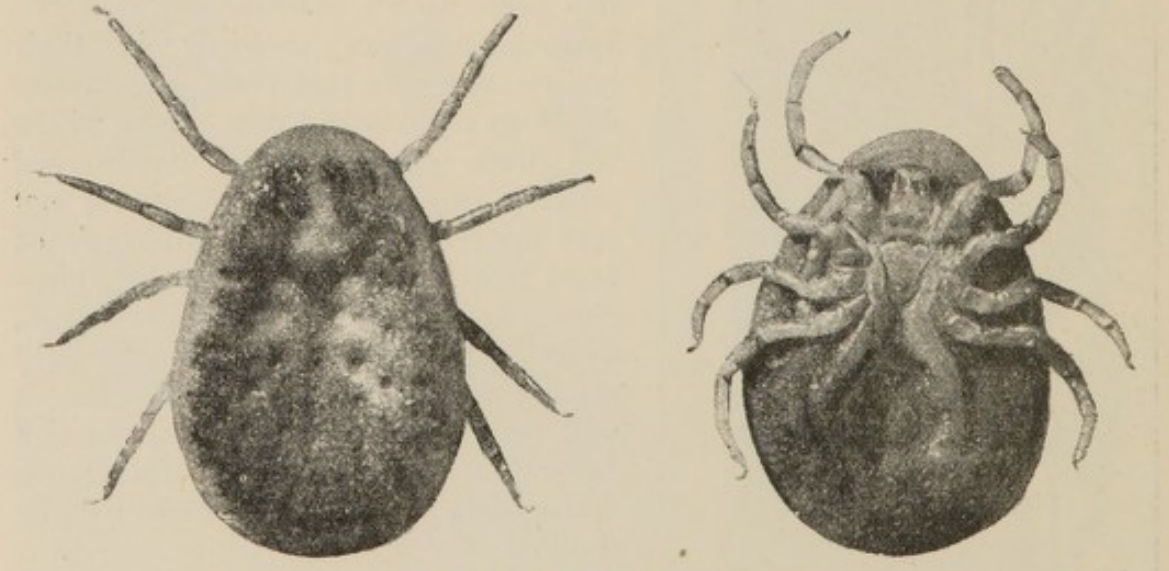


Fig. 6.—Adult Tick (Enlarged).

Left—Viewed from above. *Right*—Viewed from below.

to ten days, and if in large numbers cause very severe irritation. This irritation causes restlessness, lack of sleep, emaciation and even death of the birds, particularly in growing stock. Even a relatively mild infestation of either of these parasites may cause unthriftiness, poor egg-laying and general deterioration of the appearance of the flock.

"Broodies" (fowls, turkeys, ducks) may be put off the brood. Resistance to other disease conditions will be lowered as a result of the general constitutional disturbance.

(2) Both these parasites may transmit *Treponema anserinum* and thus bring about tick fever as already described.

Habits of the Parasites—

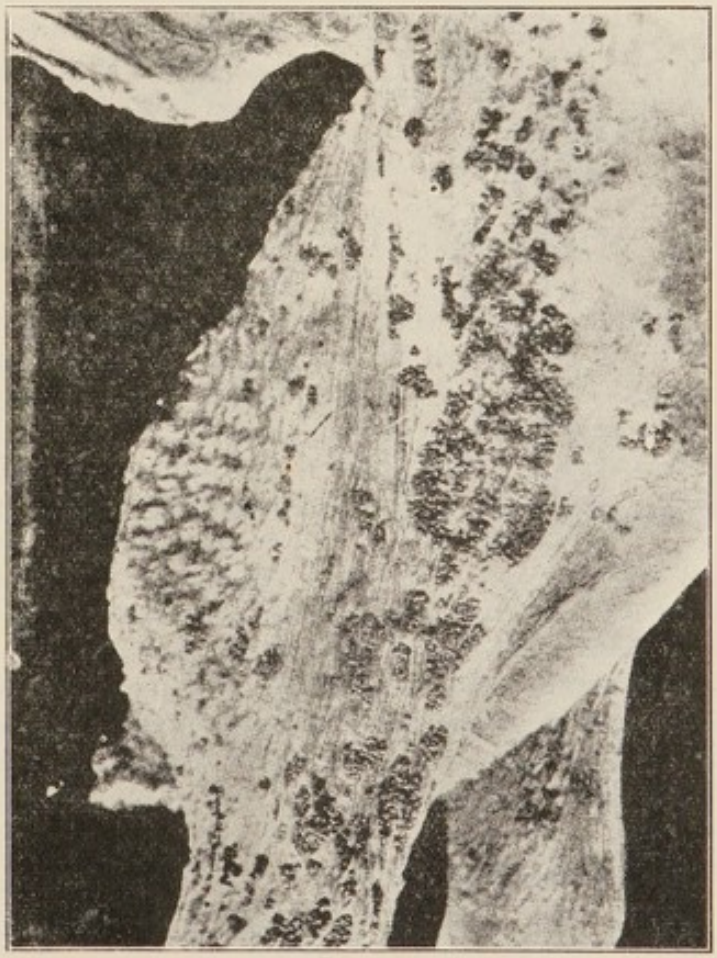
During the daytime these parasites shelter in dark protected places such as cracks and crevices of nest boxes, under perches, crevices in rotten timber in the houses, under the bark of trees nearby (particularly pepper trees) and other concealed places.

At night time the adults emerge at intermittent periods and feed upon the fowl for a short time until fully engorged with blood, and then return to their hiding place. The only form which stays upon the fowl during the daytime is the larval form. In the case of the fowl tick, this form remains attached to the fowl, usually for about five days, but in extreme cases up to ten days. In the case of the red mite the larval form remains attached for about two days.

Life Cycle—

Fowl Tick.—The eggs are laid in concealed places in batches of from twenty to one hundred about a week after the female tick is engorged. After four or five days (in warm weather) to three weeks, the larvae hatch out and actively migrate to the nearest bird, attach themselves forthwith, and remain with the bird up to ten days, after which they abandon the fowl, seek shelter again and moult in about eight days. During this stage they have six legs. After this moult the eight-legged nymph emerges, seeks the fowl again, and engorges with blood, seeks shelter again, and after four to nine days moults. This process is repeated and then the adult tick emerges.

These periods may vary very gently with climatic conditions, and four weeks or more may elapse between moults in cool weather.



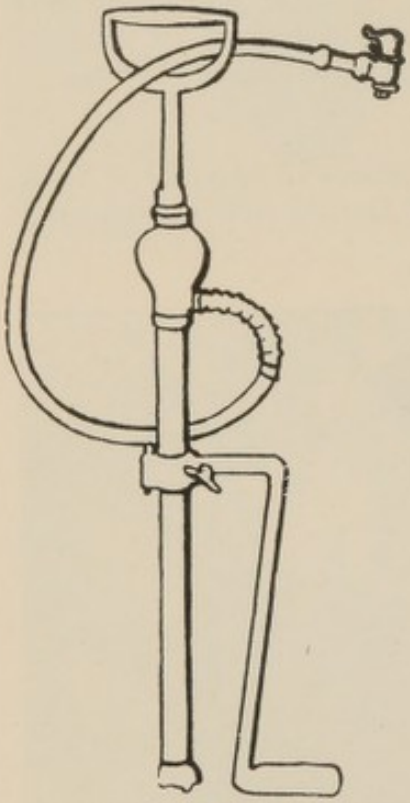
Clusters of Tick Larvae Attached to the Body of a Fowl.
[*From Bul. 74, S. Aust. Dept. of Agric.*

The adult tick usually engorges about once a month. Where fowls are not present, ticks will readily feed on turkeys, geese, pigeons or even on other birds. This should be borne in mind in any scheme of eradication.

Red Mite.—The life cycle of the red mite is similar to that of the fowl tick, only that each stage is undergone more rapidly, so that in summer the whole life cycle may be completed in one week.

Locating the Parasites—

When an outbreak occurs of a disease that appears to be spirochaetosis, it is of critical importance to determine the presence or absence of these parasites, as this is one of the factors which aids diagnosis. They are not easy to locate, and a casual seeker will be almost sure to miss the parasites. A painstaking search should, therefore, be made under perches, and in the crevices between the boards of the houses and in nest boxes. Any white-ant eaten timber should be pulled off and split up so that a careful examination may be made. The bark of trees near fowl houses should also be lifted layer by layer. In western districts where fowls often sleep in trees with loose bark, such as pepper trees, thousands of ticks may be found on the trunk of one tree. If the parasites cannot be located, some bagging material may be placed over the perches and left there overnight. Very often when ticks and red mite come out to engorge they crawl back under these bags and can be found there in the morning.



A Common Type of Bucket Spray Pump

Examination of the birds for adult parasites will, under normal circumstances, only be of value at night time. Sometimes the parasites may be found on broody fowls during the day. The larval form of the tick after engorgement may be seen as reddish-purple globules attached to the breast and thighs of the bird. These may easily be confused with certain lice which infest fowls. The use of a fine-bladed knife for cleaning out crevices may unearth some of the hiding ticks which cannot otherwise be located. Dark, brownish spots may be noted around the edges of cracks or crevices, or upon the eggs. These marks are caused by the ticks' excreta and will indicate the necessity for a careful search.

Control of the Parasites—

Once the presence of either of these parasites is noted, vigorous action should be taken. When they are not present on the farm, care should be taken to avoid introduction, remembering that the most

common means of introduction is in coops which harbour the adult forms, to avoid which the coops or crates should be saturated with kerosene emulsion. Another common means is by introducing birds affected with the larval forms of the tick or mite. To guard against this, all newly introduced birds may be placed in separate coops with a wire netting floor, under which is placed a tray of water covered with kerosene or a tray of shavings. In the former case the larvae drop off into the water, and in the latter case they drop off into the shavings in which they moult. This tray of shavings should then be burned.

Theoretically, eradication is easy, but offers great practical difficulties on some farms. Thus, if there are tick and mite harbours which cannot be effectively dealt with, it will not be possible to eradicate the parasites. Where the timber of fowl houses has been riddled with borers or white ants, or where the wood is old and rotten with many crevices and cracks, it will usually be found impossible to exterminate the last tick, and such harbours should, if finances permit, be burned down and rebuilt with suitable material.

For the eradication of this parasite, the ideal house is one in which the framework is made of metal; *e.g.*, a piping framework, covered with galvanised iron. The front should be left open and adequate space provided for ventilation at the back. Failing this, only sawn timber should be used in the construction of houses, and they should be so built that crevices and cracks are reduced to a minimum. The perches should be suspended from the roof by No. 6 wire or steel on to which is soldered a metal cup which should be kept filled with kerosene. The use of bagging, split timber, bark roofs, shingles or other similar improvised materials will make the control of these parasites impossible.

The application of whitewash to houses to prevent infestation often proves more harmful than good, as in many cases the flakes of whitewash lift slightly and provide an effective harbour for the tick and mite.

Where the houses can be treated, all crevices and cracks should be filled in with boiling tar or wood preserving oil. The junctions of all timber used in the construction of the house and the perches should also be painted with wood preserving oil. Creosote or sump oil from motors or tractors, and other mineral oils such as red oils and miscible oils as used for orchard spraying, are quite suitable for the destruction of the parasites.

Great care should be taken in the use of these materials, which are highly irritating. If possible, the fowls should not be allowed to roost in the houses for several days after treatment. Failing this, the treatment should be carried out early in the morning.

By careful treatment as above, all cracks and crevices can be effectively closed, and then the whole house may be sprayed with some insecticidal solution. There are many such preparations that are satisfactory; one every commonly used is kerosene emulsion. This is made by chopping up 1 lb. of ordinary washing soap and stirring it into a gallon of boiling water until dissolved. Take this solution, allow it to cool a little, and add 1 gallon of kerosene while stirring constantly. The stirring should be continued until an even creamy

emulsion results. This is the stock solution, and when it is desired to use it, 8 gallons of water should be added and the whole thoroughly agitated. If small quantities are required for spraying, add half a gallon of water to each pint of stock solution.

If the water is "hard," there may be some difficulty in making the emulsion.

Another suitable solution is 8 oz. chloride of lime to the gallon of water. A strong solution of sheep dip is also satisfactory. Five per cent. of a miscible red oil in water is suitable, but is more costly than the kerosene emulsion solution.

When spraying, any ordinary orchard pump which will exert a good force and which has adjustable nozzle is suitable. The more force that can be used the better, in order to drive the spray into all cracks, crevices and joints of the houses.

When a tick infestation exists, most owners spray once and then again when convenient, usually in about three weeks or so. This procedure is merely waste of money; should one succeed in killing all the adult ticks and mites, there will be some larval forms on the birds which will drop off and mature. Spraying must, therefore, be repeated at least three times at five-day intervals, and then a most exacting examination made to detect the presence of any parasites. It may be that as many as ten sprayings will be necessary before a complete eradication is effected. When this has been accomplished, great care should be taken as previously indicated to guard against further introductions of the parasites.

Fowl Cholera.

This disease has been recognised in Europe since the eighteenth century. In this State several outbreaks of the septicaemic form of the disease have occurred in the past few years, and many cases which appear suspicious. Unfortunately there has for many years been a popular impression that the disease does not occur in this country, and cases which showed symptoms and lesions indicating cholera have, in most cases, been diagnosed as cases of fowl tick fever by the poultry farmer.

Cause—

The actual cause is a germ known as *Pasteurella avicida* (*avisepticus*). It is to be noted that other *Pasteurella* organisms (germs of the same family) may cause deaths of birds in isolated cases.

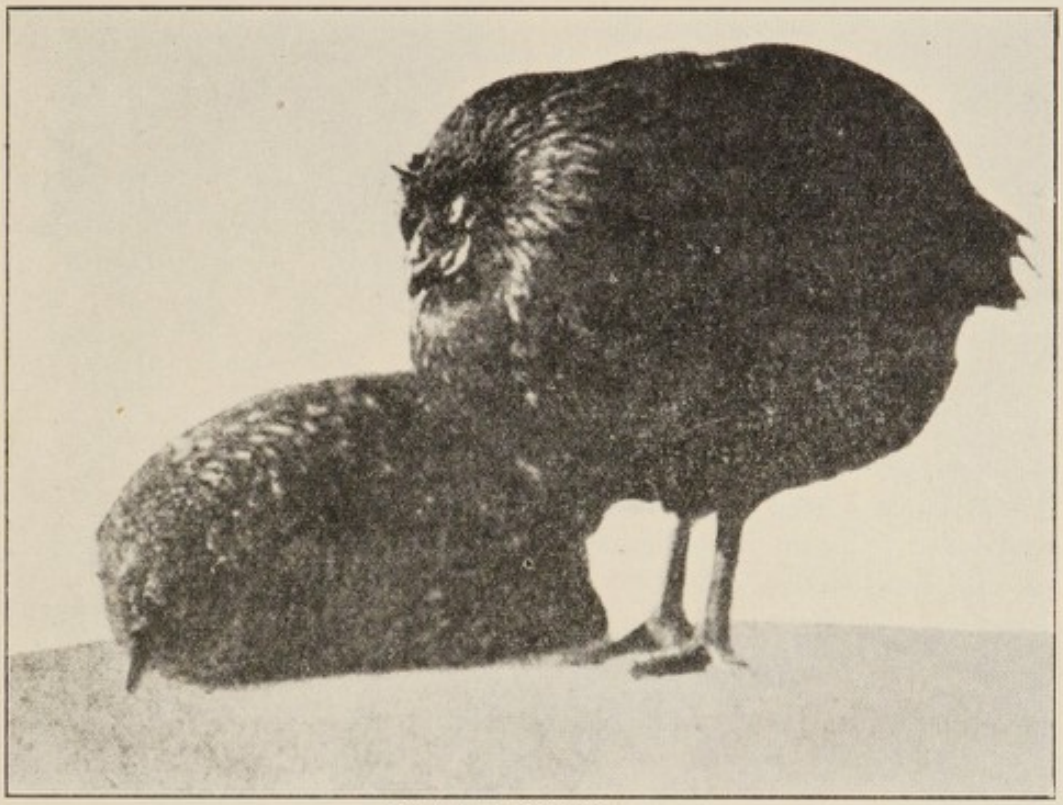
Predisposing Cause—

Controversy still exists as to the effect of predisposing causes on the incidence of disease outbreaks. Thus in Europe one investigator found that in over 20,000 hens 35 per cent. were carriers of the infection (as shown by agglutination test) and none showed any effects of the disease. This author inclines to the view that when the causal germ is present in a large proportion of the birds, predisposing causes, such as wet cold conditions, ill health, etc., determine the onset of cholera symptoms. Others strongly contest this.

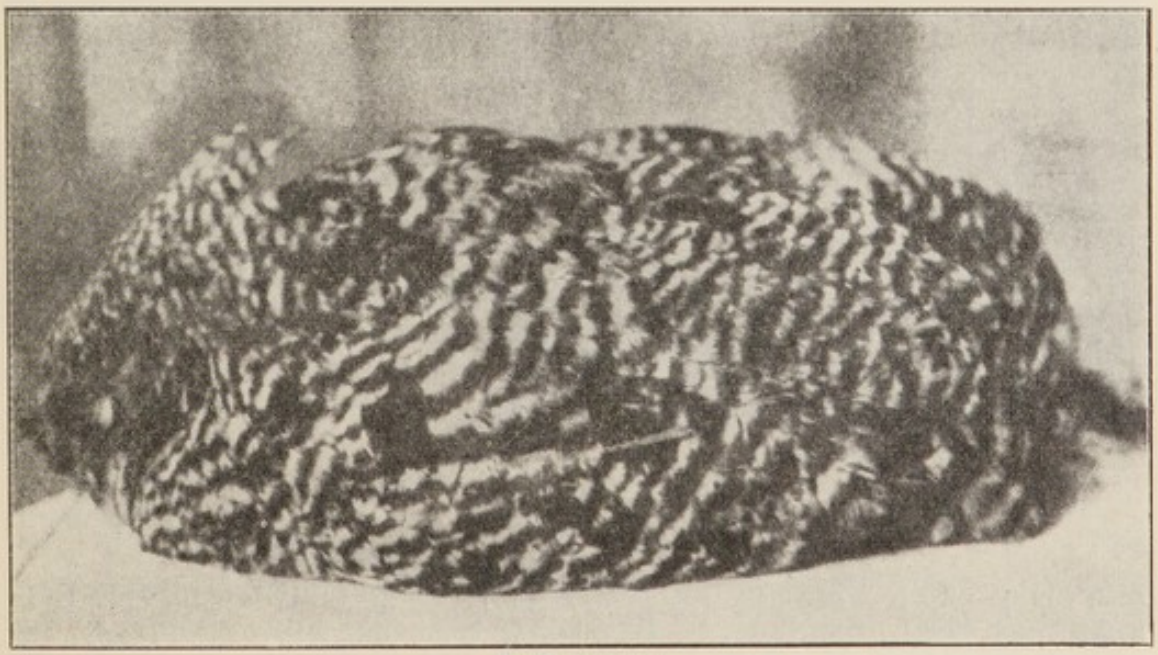
It would appear that cold, parasitic infestations, dirty and wet conditions, and faulty feeding (*e.g.*, feeding excess protein) favour outbreaks of the disease. In New South Wales it has been a notable feature that odd birds in yards widely separated on a farm may die

from the disease at intervals of weeks or months apart, showing that the infection is widespread on the farm, and yet the disease has not assumed epidemic proportions in most cases.

In America, the disease occurs most frequently in the winter months. In the case of oedema of the wattles, when the infection is present, fighting among cockerels is a cause predisposing to outbreaks.



Fowls with Cholera. [*After Patton.*



Acute Cholera.
Rapid death. [*After Patton.*

Infectivity—

(*a*) *Mode of Infection.*—Birds may carry the infection and show no symptoms. Such carriers readily transmit the disease. It has been shown that the germ may remain alive in mites for as long as four

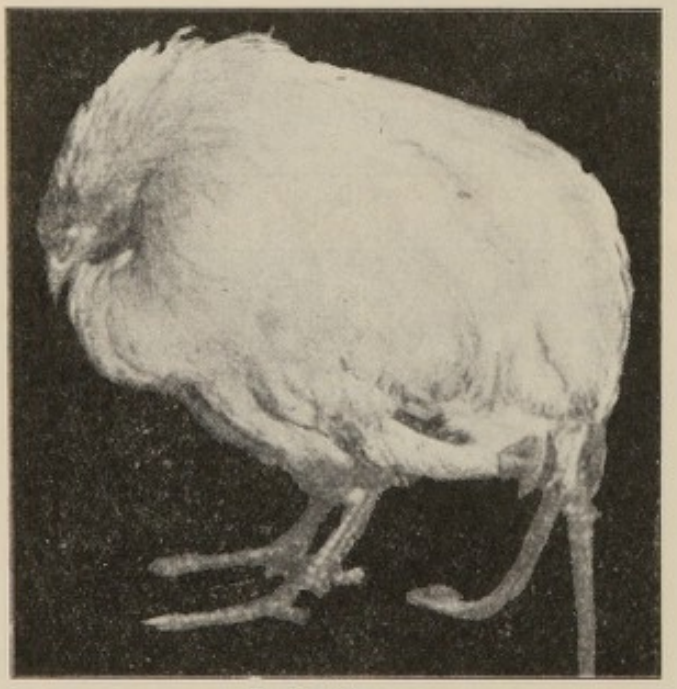
months, and if a fowl eats such mites infection may occur. External parasites—flies, fleas, ticks, mosquitoes and mites—may all play their part in spreading infection. Infection may take place by the birds eating the infective germs, especially if the digestive tract has been injured by parasites. Infection also readily occurs by inhalation, particularly when the atmosphere is damp. Eggs may carry the infection and its transmission to chickens has been demonstrated. It has been shown that birds may carry the infection in the nasal passages and also in swollen wattles. Sparrows and market crates may introduce the infection mechanically on to the farm.

(*b*) *Birds Susceptible.*—Fowls, turkeys, ducks, geese, pheasants, various aviary and wild birds, pigeons and sparrows are all susceptible to the disease. Geese seem to be very susceptible, ducks are stated by many to be unusually susceptible, and this seems to be the case here, though in America the disease is rarely reported in ducks.

(*c*) *Viability of the Germ.*—The causal bacteria will remain alive in droppings or in garden soil for at least three months, but exposure to sunlight destroys it in two days or less.

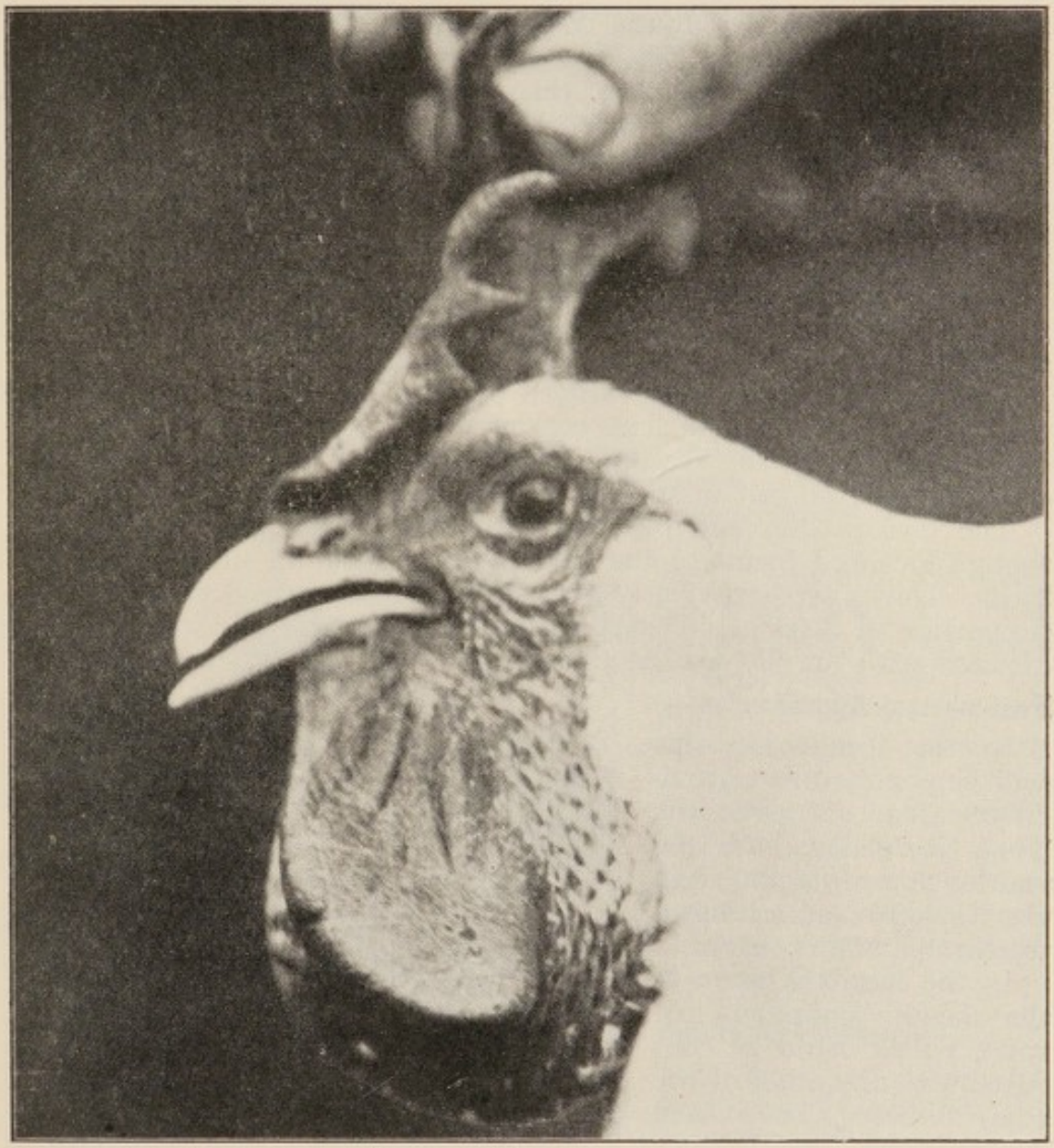
Epidemiology—

As already stated wide controversy exists as to the infectivity of this disease. Some investigators find that the causal micro-organism is one of the utmost virulence, and when once introduced a disastrous epidemic occurs. Others have found that the presence of the infection is of little importance, that only odd cases of the disease occur, and that most cases take on only a localised form when conditions of management, feeding and sanitation are ideal. From local experience it would appear that the latter view is perhaps the more correct. Cases of "oedema of the wattles" or "swollen wattles" have been quite common, and odd deaths from the disease frequently occur, yet severe epidemics of the disease are most unusual. It is felt that the presence of the subacute type of the disease with numerous carrier birds in the flock is much more common in this State than has been suspected. In such cases the only feature is the occasional death of birds which are found under the perch in the morning. Infection of chickens in which the resistance is low will result in heavy mortality, and unless competent advice is sought the farmer could readily confuse such an outbreak with pullorum disease.



Fowl Cholera.
Lethargy, with Diarrhoea.
[*After Hutyra and Marek and Manninger.*

It has been found that certain strains of the germ can live indefinitely in the tissues of the host and produce only occasional deaths. Other strains do not live long in tissues of the host (poor vegetative capacity), but cause a heavy mortality (high virulence).



A Case of Oedema of the Wattles on a Local Poultry Farm where Generalised Cholera Cases had never been Recorded.

Symptoms—

The Localised Type.—Those strains of the germ which have high vegetative capacity and low virulence set up the localised form of the disease. Where conditions of feed and environment are unsuitable, there is a greater tendency for systemic symptoms to occur. The most common form of localisation is the condition known as "oedema of the wattles" or "swollen wattles." In this condition the wattles and sometimes the tissues of the adjoining areas of the face become distended with an oedematous (doughy) swelling. This gradually becomes harder and firmer. It is at first red, then becomes darker and purplish. The affected bird appears sick and depressed, and in some cases dies. In cases which recover (and this is the general rule) the swollen area hardens, becomes black, shrivels and falls off, leaving only a scar.

Other localised forms are swelling of joints and lameness. In some cases infection of the mucous membrane of the nose and palate may produce symptoms which resemble colds and "roup."

Septicaemic Form.—In the most severe type (*e.g.*, where bird is infected with a virulent strain of the germ) no symptoms may be noticed, but the bird is found dead under the perch.

In less sudden cases the bird exhibits the following symptoms:—Ruffled plumage, no appetite, thirst, disinclination to move, and a greenish diarrhoea is frequently present. The respiration may tend to be gasping, slime is often dribbled from the nose and mouth. The head, comb and wattles are dark in colour, and trembling may be observed. After lingering for perhaps twenty-four hours since first showing symptoms, the bird goes into a convulsion and dies.

In cases which linger longer than about eighteen hours, a yellowish-greenish diarrhoea is always a prominent symptom. This symptom may cause the farmer to think that the disease is spirochaetosis.

In protracted cases the diarrhoea persists, the yellowish or greenish material adheres to, and dries on the feathers of the part, emaciation becomes a prominent feature, inflammation of the joints sometimes develops and the gait in such cases becomes staggering.

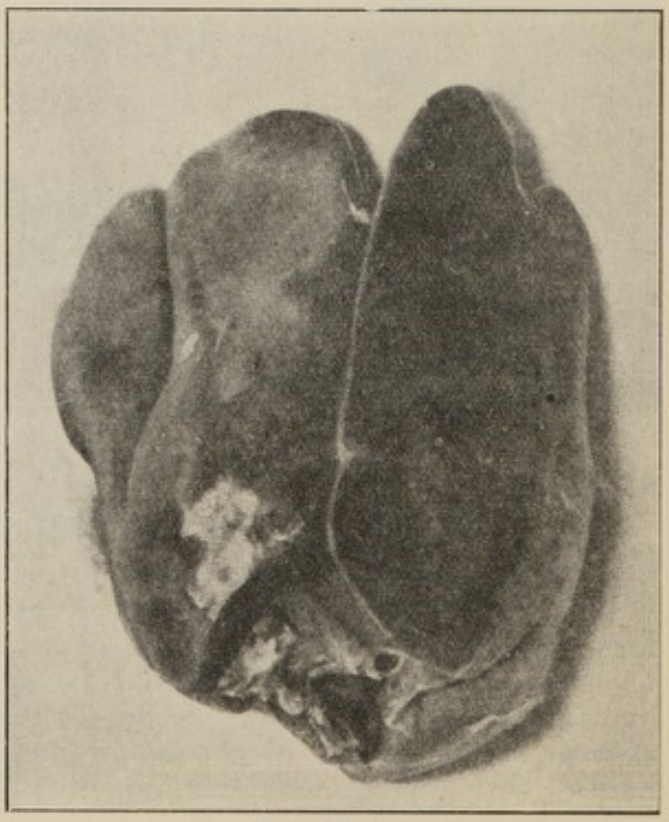
The bird is thin and pale for some days, and may then recover, though localised forms of the disease often occur in such "recovered" birds. Ducks are particularly susceptible to joint infection, and a large proportion of cases of lameness is common in such birds which become affected with the disease but do not die.

Post-mortem Appearances—

In the localised forms will be confined to a particular area or structure. Thus in oedema of the wattles nothing apart from the swollen discoloured wattles may be observed.

In the localised form in the head, symptoms of roup, which could be confused with any other of the roup diseases, have been recorded. Thus cheesy exudates may be present in the nose, infraobital sinuses in the ear cavities, and base of the brain, and slimy material may be present in the nasal cavity. This "roup" form of the disease has not, as yet, been detected in New South Wales.

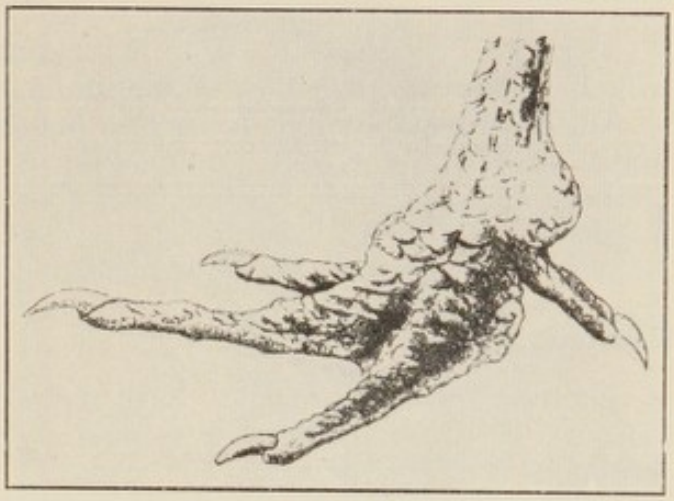
In the localised form in joints cheesy exudates frequently distend the joint capsule.



Fowl Cholera.
Extensive Necrosis in the Liver.
[After Hutyra and Marek and Manninger.

Septicaemic Form.—In the most acute cases in which birds die without showing symptoms few or no changes are in evidence on post-mortem examination.

In cases which linger the liver is usually enlarged, congested, and there may be necrotic (dead tissue) areas which are discoloured. The spleen is almost invariably enlarged as are the kidneys, and pin-point haemorrhages are often seen under the capsule of these organs. The lungs are congested, and small haemorrhages in the heart muscle are common. The pericardium may contain a turbid fluid with fibrinous flakes in it. The intestines are usually bright pink, inflamed on their inner surface and have greenish-yellow contents. Haemorrhages are often present in the first part of the intestine. Reddening of the skin of the breast and abdomen is frequently observed. In some cases the abdominal cavity may contain a mass of cheesy exudate similar to light-coloured egg yolk.



Fowl Cholera.
Inflammation of the Joints.
[*After Hutyra and Marek and Manninger.*

Oedema of the Wattles.
[*After Gwatkin.*

Differential Diagnosis—

In cases of doubt, affected birds should be submitted to a laboratory for culture of the causal micro-organism from the blood stream and affected organs.

Control—

This is one of the diseases in which correct management and strict cleanliness plays a large part in control.

In suspected outbreaks of the disease, affected birds should be strictly isolated, if not destroyed at once. Crates and the lorry used for transport of birds suspected to be affected should be thoroughly cleansed

and disinfected, and premises occupied by the birds should be cleansed, disinfected and then left for six months at least before further use.

Immunisation—

In 1880 Pasteur claimed to have produced a satisfactory vaccine which conferred an adequate immunity on birds vaccinated.

An enormous amount of work has been done since then, but in most cases failure to immunise birds has been recorded. Many authors have claimed success, but their good results have not been regularly confirmed. At present it does not appear that any method of vaccination gives thoroughly reliable results.

Treatment—

Though some treatments have been favourably reported on, the successful treatment of birds is strongly contra-indicated as such birds probably remain carriers.

Cases of oedema of the wattles can be treated surgically by completely removing the wattles.

Vent Gleet or Infectious Cloacitis (Infectious Venereal Disease of Poultry).

This is an infectious condition on which very little scientific work has been carried out, and at present the nature of the infective agent is in some doubt. It is mainly transmitted from hen to hen by the cock during copulation. It may occur in some hens in a flock, but until a rooster is introduced very little spread from bird to bird takes place. It would appear that the cock bird may be a carrier harbouring infection for some time. One way of introducing the disease is by the introduction of an infected cock bird, and therefore care should be exercised in examination of the cock for the presence of this disease before introducing him into a clean flock.

Symptoms—

The first change which can be noted is a reddening of the cloaca and vent. If the cloaca is opened the reddening and inflammation may be noted to extend up into the rectum and oviduct for some little distance. The skin around the vent then becomes swollen and doughy in consistency, and ulcers usually develop. At this stage an extremely offensive odour is present. A severe degree of irritation is present, droppings are passed at frequent intervals, and the bird strains. The droppings are liquid, contain mucus, and are peculiarly foul smelling. The cloaca may be protruded as a result of the straining, and in these cases it is common for other fowls to peck at the exposed mucous membrane, and in this way an outbreak of cannibalism may be started.

Of itself (apart from cannibalism) vent gleet seldom causes the loss of many birds, but if it is at all prevalent, will seriously lower the egg production.

Two or three weeks may elapse from the time the bird is first infected until definite symptoms appear.

Treatment—

Treatment is often unsatisfactory, and the condition proves to be very resistant to medicinal intervention. It is, therefore, usually far more satisfactory in a commercial flock to kill off those birds showing symptoms, before the disease spreads.

If treatment is to be attempted, affected birds should be isolated, the roosters removed, and a 20 per cent. solution of Argyrol injected into the vent. Cresol in 2 per cent. solution or sulphate of iron, 1 per cent. injected into the vent twice a day sometimes effects a cure.

For the external ulceration carbolic ointment 2 per cent. or a solution of one in a thousand perchloride of mercury may be used to bathe the part.

Control Measures—

There is no specific means of preventing the occurence of this disease except by keeping a watchful eye for the first case which appears.

Trichomoniasis of Poultry.

This is a condition which has occurred in this State, though not frequently. It has been recorded that both pigeons and turkeys are frequently affected in other countries. The cause is a microscopic protozoa (animal) parasite.

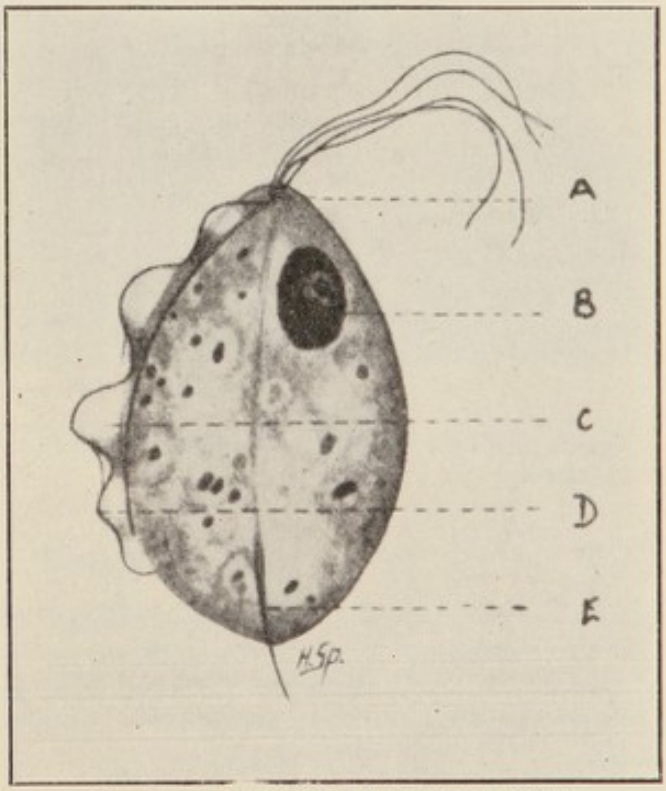
In turkey poults the symptoms are foamy semi-liquid droppings, which may be blood-stained, together with listlessness followed by death.

In pigeons the disease, which is also known by the name of "yellow button,"

Trichomoniasis of Pigeons.

Shows the greatly increased size of the liver and the concentric necrotic patches.

[After Sporri.



The Causal Organism Trichomonas hepatica.

(A) Flagellum, (B) basal nucleus, (C) nucleus. (D) undulating membrane, (E) edge flagellum,

[After Sporri.

produces thickening under the mucous membrane of the gullet and skin of the head. These swellings are yellowish to brownish in colour, and may develop to the size of an almond. General systemic symptoms such as sickness, diarrhoea, together with yellowish discharge from the nasal openings, may be in evidence. An outbreak of omphalitis (navel disease) in chickens has been found (overseas) to be caused by trichomonad infection. "Canker" in a dove's mouth or throat was shown to be due to trichomonads.*

On post-mortem examination, enlargement of the gall bladder, inflammation of the intestines, diphtheritic lesions of the oesophagus and crop (common lesions in turkeys) and occasionally (more particularly in pigeons) cheesy, greyish-yellow areas may be noticed in the liver, which somewhat resemble blackhead. The mortality may be very high (up to 75 per cent.). The disease is readily transmitted in feed and drinking water. In pigeons the disease is transmitted from carrier adult birds (which appear healthy) to the squabs during feeding.

Control—

Absolute cleanliness and sanitation is of the greatest value in limiting spread of the infection from bird to bird. 0.5 per cent. of copper sulphate solution would destroy the causal micro-organism. This amount of copper sulphate (one-tenth oz. to the pint) may be placed in the drinking water during the first few days of an outbreak.

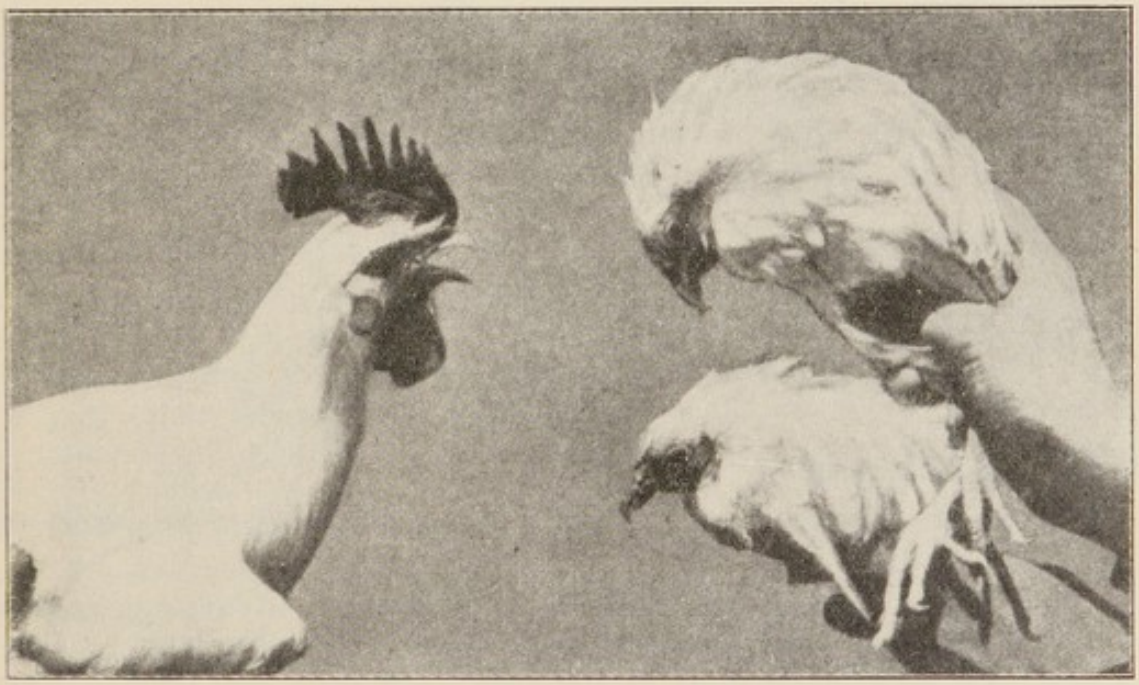
*Unpublished data by L. Hart, B.V.Sc., Veterinary Research Station, Glenfield.

DEFICIENCY DISEASES.

The most important diseases occurring in poultry which are the result of some deficiency in the diet are:

1. *Avitaminosis A.* (Green Feed Deficiency Disease) see also Infectious Laryngo-tracheitis and White Eye in Ducks).
2. Rickets.
3. Perosis or Slipped Tendon.
4. Crooked Breast.
5. Cannibalism.
6. Nutritional Paralysis.

In any serious disease resulting from a deficiency, correct diagnosis is of great importance in view of the fact that once a few deaths have occurred, a major and dramatic mortality may be imminent, due to the operation of the common cause.



Three Cockerels of the same Age.

The one on the left received ordinary diet and sunlight. The others ate the same food, but were not exposed on the run. Note the vulture-like appearance and crossed beak of the deficient cockerels.

[*After Bayon.*

Rickets.

This is a condition of faulty calcification of the bones, and is associated with deformity, lack of growth and various symptoms of ill-health. The disease is due to a deficiency of Vitamin D or a deficiency, disproportion, or faulty assimilation of calcium and phosphorus, and is recognised most frequently in young animals and birds. In birds symptoms occur particularly rapidly.

General Considerations—

Rations fed to growing stock vary enormously. The basis of rations in this country is wheat products. In the ration recommended by the Department, 3 per cent. of bonemeal is included, and shell grit is made freely available. These substances form the main supply of calcium. Bonemeal also supplies phosphorus, and wheat products are relatively rich in phosphorus. The milk products included also supply small quantities of calcium, phosphorus and Vitamin D. The ratio of calcium to phosphorus should be approximately 3 : 1. The ration recommended by the Department (a free pamphlet is available on the feeding of poultry, on application) approximates these requirements closely enough for practical purposes and will prevent rickets if sufficient Vitamin D is available to the bird. Vitamin D is necessary for the assimilation and utilisation of calcium and phosphorus. If chickens have access to direct sunlight (sunlight that has *not* passed through window glass) sufficient Vitamin D is produced by the bird (due to irradiation by sunlight of ergosterol in the skin). If birds are reared indoors, *e.g.*, in battery brooders, or in the open in very cloudy wet weather, Vitamin D supplements are necessary in the ration.

It will thus be seen that supply of calcium, phosphorus and Vitamin D are interrelated, and deficiency of any of these will cause rickets. Further disproportion of one may make added amounts of another necessary—thus in one experiment when 0.45 per cent. phosphorus only was present in the ration more cod liver oil (Vitamin D) was required to prevent rickets than when 1.0 per cent. phosphorus was fed (Ca : P ration was 3 : 1). Below 0.45 per cent. phosphorus no amount of cod liver oil would prevent the incidence of rickets.

The practical significance of the above facts is that if the Departmental recommendations are followed as to feeding and the chickens have access to direct sunlight or alternatively are fed 1 per cent. of a reliable commercial cod liver oil, rickets will not develop.

Period of Development—

The age at which chickens show symptoms of rickets will depend on two main factors:

(*a*) The amount of Vitamin D and minerals in the eggs—this varies (within limits) with the ration fed to the layers. The greater the amount in the egg the longer the chicken can withstand deficiency.

(*b*) The amount of deficiency (vitamin or mineral) in the ration.

Where the deficiency is acute, symptoms may occur on the eleventh day or even earlier. Usually they are not apparent before about four weeks (as the deficiency is usually only slight).

Symptoms—

Cessation of growth or much retarded growth is apparent. The gait of the chickens becomes proppy, unsteady and ultimately the chickens are unable to stand. Prior to this stage the beaks become soft and deformed. Frequently the first beak abnormality is an increased

curvature of both the upper and lower portions of the beak, so that when the point of the beak is closed, there is a gap between the posterior portions. The beak may be so distorted and crossed that the chick can scarcely eat. Softening and swelling of various bones may be seen. This is particularly in evidence at the junction of the ribs with the rib cartilages. At each such junction there is a marked enlargement which constitutes a rachitic "rosary." The breast bone (also the pelvic bones) may be markedly deformed.

Although the chickens may be suffering from diarrhoea, they are quite bright, and have a good appetite. Their muscleature, however, is usually flabby and the abdomen relaxed.

On post-mortem examination the long bones of the leg and wing are observed to be fully pliable, bending between the fingers instead of snapping as in a normal bird. On analysis, such bones are found to have less mineral matter or ash than bones from normal chicks from the same hatch.

Effects of Deficiencies in Adult Hens—

Symptoms of rickets, as above, do not occur in adult birds. Deformed bones will be noted in adults which were affected with rickets as chickens. When laying fowls are on a diet markedly deficient in calcium, soft-shell eggs may be laid, and hatchability seriously affected. When there is a deficiency of Vitamin D (no sunlight and insufficient in ration) hatchability is seriously affected, there being a serious embryo mortality about the eighteenth or nineteenth day. Chicks which do hatch are predisposed to rickets.

Cod Liver Oils and Other Fish Liver Oil Vitamin Supplements*—

It has already been pointed out (see Green Feed Deficiency Disease) that if fresh green feed (which supplies Vitamin A) and sunlight (which produces Vitamin D) are available to birds, then no fish liver oils or other vitamin supplements are necessary. The natural supply (ample fresh green feed and sunlight) is superior to supplemental supply. However, when the latter is necessary, it is important to know something of relative values to be expected in various oils.

Vitamin A Content†—Most fish liver oils contain large quantities of Vitamin A. Medicinal cod liver oil is guaranteed to contain 1,000 international units of Vitamin A per gram. Oils which contain this amount will provide ample Vitamin A when freshly fed at the rate of 1 per cent. in the mash, because 3 units per gram of feed are sufficient for laying and growing stock. Where 1 per cent. of an oil with 1,000 units 1 gm. are fed, this equals 10 units per gram of the mash. If the mash is half the ration, this equals 5 units per gram in total ration. It has already been noted that if a fish liver oil is mixed with a mash and left, the Vitamin A content is rapidly destroyed, so much so that in ten days only a small proportion of potent Vitamin A is left.

*For article on cod liver oil quality see Branion, H. D., A. F. Dawson, J. R. Cavers and I. Motgale, 1938. Poultry Science 17: 213.

†For complete statement of Vitamin A and Vitamin D content of foodstuffs see Fixsen, M. A. B., Roseal, N. H., in *Nut. Abs. and Rev.* 7: 823, 1938.

*Vitamin D content.**—Only a relatively few natural fish oils have a high Vitamin D content. Medicinal cod liver oil is guaranteed to contain 100 international units of Vitamin D per gram.

Feeding 1 per cent. of such an oil will provide ample Vitamin D for laying hens as 0.78 units per gram of feed is sufficient for laying pullets, and half this is sufficient for growing chicks.

Some oils (such as shark, dogfish, etc.) may contain less than a quarter as much Vitamin D as pure cod liver oil, and if such materials are depended on to prevent rickets heavy losses may be sustained.

It is to be noted that sometimes the unit content of Vitamin D in oils is ascertained by feeding it to laboratory animals other than chickens. In such cases the guaranteed unit content may be misleading.

The only guide a farmer has is the guaranteed statement of the unit content of the oil in vitamins tested by chicken-feeding tests. Thus an oil with 100 units Vitamin D per gram at 6s. per gallon, will be cheaper than an oil with 60 units Vitamin D per gram at 4s. per gallon, as regards its value in preventing rickets.

Colour varies from lemon-yellow to dark red or nearly black. Oils rendered by direct steam method are light in colour. Those extracted from livers kept for some time are dark and oil extracted by the sun-rotting process are dark red to black in colour. Generally speaking (though there is no direct relationship) the best oils are light in colour.

Acid Content.—F.F.A. (or free fatty acid) varies in samples sold for poultry feeding from under 1 per cent., to as high as 30 per cent. It has been found that oils with a high F.F.A. are not necessarily unsuitable for poultry (they cause nausea in humans) when fed at 1 per cent. only in mash. On the other hand, some samples with a high F.F.A. contain substances which are deleterious to chickens. Further, the acid content hastens deterioration in the Vitamin A content and therefore an oil with a high F.F.A. may be regarded as potentially of less value than an oil with a low content.

Nitrogenous material in fish liver oils is sometimes harmless, but such materials may on occasion be toxic.

Amounts of Cod Liver Oil Required—

As a guide to the information already given, an American recommendation is quoted here:—

Vitamin D potency of oil, U.S.P. units per gram.	Units per lb. of Oil.	Number of units required in 100 lb. of mash.	Approximate per cent. of oil to be added to mash.	Approximate lb. of oil to one ton of mash.
50	22,700	17,700	0·78	15
85	38,590	17,700	0·46	10
310	140,740	17,700	0·13	2½
400	181,600	17,700	0·1	2

*It is now known that there are at least four (probably five as known at present) antirachitogenic substances classed under the term Vitamin D. Details of these are outside the scope of this work.

It will thus be seen that the recommendation to include 1 per cent. of a reliable cod liver oil allows a generous margin. This margin has been necessary unfortunately in the past in some instances.

The oils mainly available on the market are cod liver oils, pilchard oils, sardine oil, mutton bird oil. Statements that a certain oil is so many times more potent than some other product may mislead, as this comparison may refer to Vitamin A content only.

Perosis or Slipped Tendon.

Historical—

Historical.—It appears that this disease was first recognised as a separate condition from rickets in 1928. Much confusion as to the identity of the condition has occurred in the past.

Cause—

Cause.—It was thought that excess minerals and/or disproportion of the amounts of calcium and phosphorus present in the ration were the cause. It was found that in some cases bran, wheat germ, wheat pollard, oats and several other ingredients would prevent the occurrence of the condition.

Quite recently it has been shown that the condition is due to deficiency of manganese in the diet, and it has been found that substances which have a preventive action are rich in manganese. It is probable that excess of minerals and perhaps other factors aggravate the condition brought on by the deficiency of manganese.

Birds Affected—

In this State serious outbreaks occur frequently in the western districts in turkey flocks, young growing poults from four weeks to six months showing symptoms of the disease.

Occasional outbreaks in chickens have occurred. Most serious outbreaks in turkeys have been noted in dry seasons where no green feed is available and, following rains, further cases have not occurred. This may be due both to green feed supply and increased supply of insect life, as nearly all turkeys are run on unlimited open range conditions.

Symptoms—

A slight enlargement of the tibia-metatarsal joints occurs; this is then followed by the outward deviation of the ends of the long bones (tibia and metatarsus) which make the hock joint. The gastrocnemius tendon or hamstring then slips laterally, and the hock joint appears to be flattened. The bird is unable to support its weight and if both legs are affected it may starve. The bones are quite hard and may be slightly shortened and thickened.

Differential Diagnosis—

The condition is really quite characteristic. Confusion with rickets should not occur, as in rickets the bones are soft and will not snap on being bent, whereas in perosis they are normally brittle.

Control—

When balanced rations are fed as recommended by the Department, the condition should not occur. However, it is probable that the problem is one of soil deficiency and when wheat and wheat products are used from a wheat crop grown on a manganese deficient soil, the disease will develop.

To prevent its development, small quantities of manganese should be added to the ration. One-quarter lb. of manganese sulphate per ton of foodstuff is usually adequate so long as it is evenly incorporated. For small amounts of feed, a solution of 1 oz. of manganese sulphate could be added to 1 pint of water and ¼ pint of this added to each 100 lb. of feed. To add this, mix the ¼ pint thoroughly with several pounds of bran. Add another 10 lb. of bran, mix thoroughly, then mix this "mineralised" bran through the rest of the ration. Excess of manganese will cause stunting of growth and only trace amounts should be added. Similarly 0.1 per cent. of manganese dioxide (which provides 0.06 per cent. manganese added to a ration) will prevent the disease.

In addition, poults (or other susceptible young birds) should be fed 3 per cent. of bonemeal in the morning mash and ample green-feed (or 1 per cent. cod liver oil) should be supplied.

Crooked Breast.

This is a condition affecting birds, particularly turkeys, in which the crest (keel) of the sternum (or breast bone) becomes uneven in depth, twisted, tortuous or deviated.

The condition is important in fowls from the show standpoint. It has an added importance in turkeys for table purposes, as the deformity of the bony skeleton markedly decreases their market value.

Critical investigations have as yet not clearly finalised the causes but the following features are relevant.

When a deficiency or disproportion of calcium phosphorus and Vitamin D exists in the ration, mild symptoms of rickets with deformity of the long bones of the skeleton and the sternum occur.

Certain strains of birds seem more liable to rickets than others. Certain strains of birds inherit a tendency to develop crooked breasts and inbreeding will fix and intensify this factor.

Early roosting of young birds, particularly on narrow roosts, may accentuate any tendency towards development of breast deformity.

Prevention—

(1) No birds with crooked breasts should be used as breeding stock.

(2) Inbreeding should be avoided.

(3) Ample supplies of good quality shell grit should always be before the birds.

(4) Green feed (or cod liver oil, 1 per cent. in ration) should always be fed.

(5) Bone meal (3 per cent.) should be fed in the morning mash to growing birds.

Cannibalism.

This is a condition in which birds young or old start to peck at and eat one another. Commonly the pecking commences at the vent, and an attacked fowl may have the abdomen penetrated and the intestines pulled out, resulting in immediate death. Chickens frequently peck one anothers' toes or feathers, and adult fowls may, in bad attacks of feather eating, almost denude one another of feathers.

Very much has been written as to the cause of these various conditions of cannibalism, and while it is clear that faulty management plays a part, it may well be shown that the cravings which provoke cannibalism are due to deficiencies. Recently it has been shown that oat hulls in America appear to provide some substance which prevents cannibalism. Many factors have been shown to have some bearing on outbreaks. Amongst the most common are lack of minerals (*e.g.*, common salt), animal protein (meat meal) in the diet, overcrowding, having the nest boxes too exposed, the occurence of vent gleet in the flock, the accidental wounding or injury of a bird producing bleeding, or one or more of these causes acting together. Whatever the cause which acts as a starting point, it would appear that the habit is rapidly acquired and spreads throughout the flock.

On numerous occasions it has been noted that where no animal protein food is fed, an outbreak of cannibalism occurs, and it would seem logical to regard these as cause and effect. In some such cases an epidemic has ceased when meat meal has been added to the diet. Under natural conditions a bird will correct any protein deficiency in the diet by catching insects and worms, but under concentrated methods of rearing, the only avenue for increasing the protein intake in their diet is by feeding upon one another.

Epidemics also occur when ample or excess meat meal is fed, but in almost every such case some other cause can be demonstrated. Thus on one farm the birds were grossly overcrowded. All endeavours to check the habit of cannibalism were quite useless until the survivors were placed out in a large run overgrown with blackberries. Victimised fowls sheltered in the dense undergrowth and after about ten days the whole trouble had ceased.

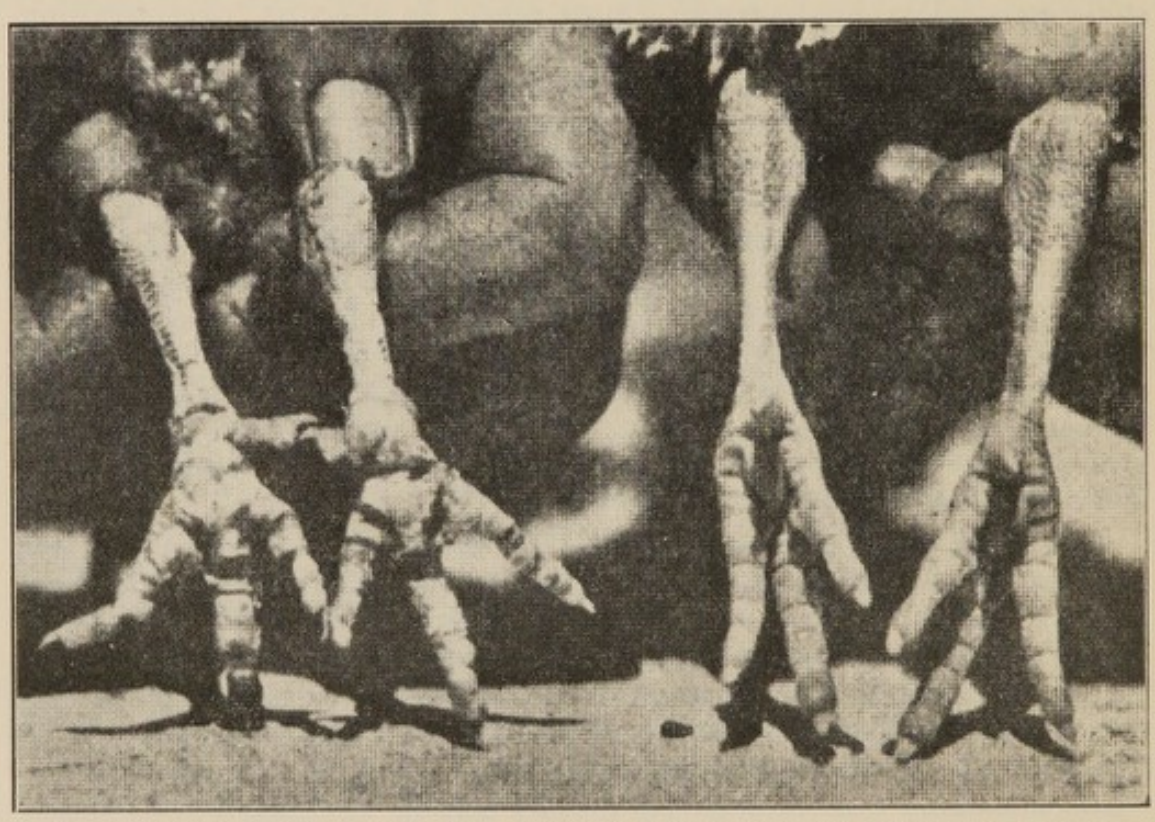
Once pecking has commenced, a fowl may be completely eviscerated, and the thirst for blood is progressive, causing the surviving birds to continue the slaughter.

Feather picking and toe pecking are very common, particularly in battery reared chickens. Deficiency of salt is often set down as a cause, but the habit occurs where ample salt is fed. Feed adhering to the toes of chickens will cause pecking and bleeding of the toes, which immediately arouses the attention of other chickens. In other countries this has been so serious at times that red lighting of the battery brooder has been adopted. The chickens do not then notice the blood, and the habit ceases.

To summarise: Where any cannibalistic habit develops, offenders should be promptly removed before they teach other fowls. Overcrowding should be rigidly avoided, and 5 or 6 per cent. of meat meal should be fed in the mash. A well-balanced ration with the requisite amount of salt (about 1 per cent.) and green feed should be fed.

Suitable shell-grit should always be before the birds. Further, they should be placed in an environment as far as possible approximating their natural habitat. Birds which are run on large open ranges seldom develop these troubles, even where the ration is grossly deficient and unbalanced. Thus, where an outbreak of cannibalism in any of its various forms occurs in an intensive or semi-intensive house, it may often be checked by turning the birds out into such an area, if available.

Even after everything possible has been done, outbreaks of cannibalism do occur in some battery brooding plants, apparently as a sequel to boredom, and unless care is taken the outbreak may prove costly. Some owners use blacking, stockholm tar, or other dark-staining material, which obscures the colour of the blood and so prevents vicious curiosity and attack.



Swelling and Fissuring of the Feet of a Poult Fed a Diet Deficient in Vitamin B-G Complex (riboflavine).

[After Jukes.

Nutritional Paralysis.*

This name refers to a disease of poults in which the toes and shanks become swollen and oedematous (doughy). Following the swelling, the skin becomes cracked and fissued and the toes may become crumpled and powerless. In some cases, pronounced "corny" swellings occur. The condition is not uncommon in poults run in this State in the western areas.

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*For summary of the present status of information concerning the vitamins of the B-G complex readers should consult an article by D. Miller in *Poultry Science* 17: 523, Nov. 1938.

It appears to be due to a deficiency of one of the components (riboflavine) of the Vitamin B-G complex, and can be prevented by feeding milk, whey or whole or skim milk products, or milk products containing the "whey" components.

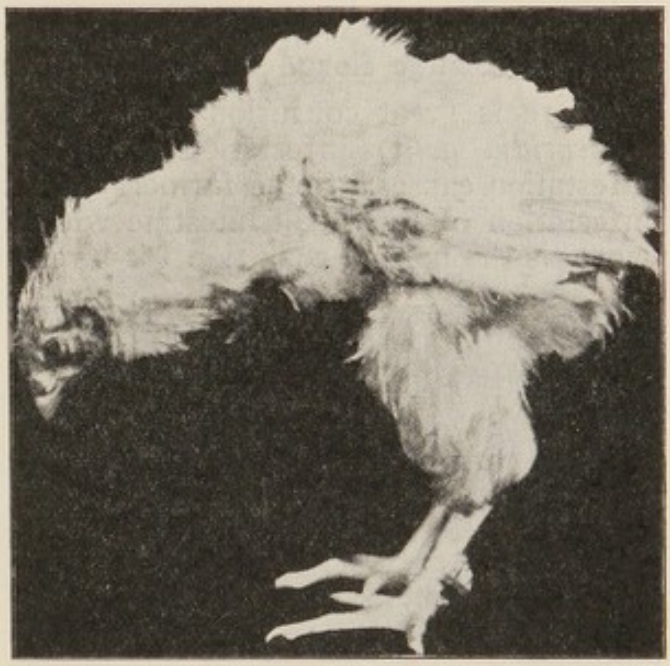
Attention to feeding and correct ration otherwise is also indicated. (Free departmental pamphlet is available on feeding). Particular care should be taken to provide fresh green feed, if available, as well as milk products.

Other Vitamin Deficiencies.

There are a number of other diseases of poultry due to deficiency of various vitamins in the diet. Thus deficiency of any of the several vitamins in the Vitamin B-G complex will produce symptoms of disease, such as beri-beri (a nervous affection), cessation of growth, paralysis and dermatitis.

Deficiency of Vitamin K will lead to persistant bleeding from any portion of the body on the slightest injury.

With the rations normally fed in this State such symptoms do not occur, and for this reason the conditions are not dealt with here.



A Case of Polyneuritis.

Due to acute deficiency of Vitamin B. This does not occur when birds are fed the usual commercial rations.

[After Graham.

PARASITIC DISEASES.

When birds were in their wild state the eggs of parasites were normally distributed over wide areas of forest and the chances of the host making contact again with the parasite were decreased. Under such conditions a normal host-parasite balance was maintained.

Under conditions of domestication where birds are gathered in enormous concentrations on small land areas contact between host and parasite is such that heavy infestation with the parasite frequently occurs. It is for this reason that special methods of prevention and treatment have to be adopted to prevent economic loss.

Large Round Worm (*Ascaridia galli*) Infestation.*

There is great confusion of ideas on the subject of round worm (*Ascaridia galli*) infestation of poultry and the harm which this infestation causes. Some farmers have the impression that a moderate infestation of the small intestine with the common large white round worm is the normal condition for birds. Other farmers are persuaded by medicine vendors that the presence of up to a dozen round worms per bird will cause very serious economic loss. Both of these views are incorrect.

Worms Protruding from a Section of Intestines of Fowl.
Taken from a six-weeks-old chicken.

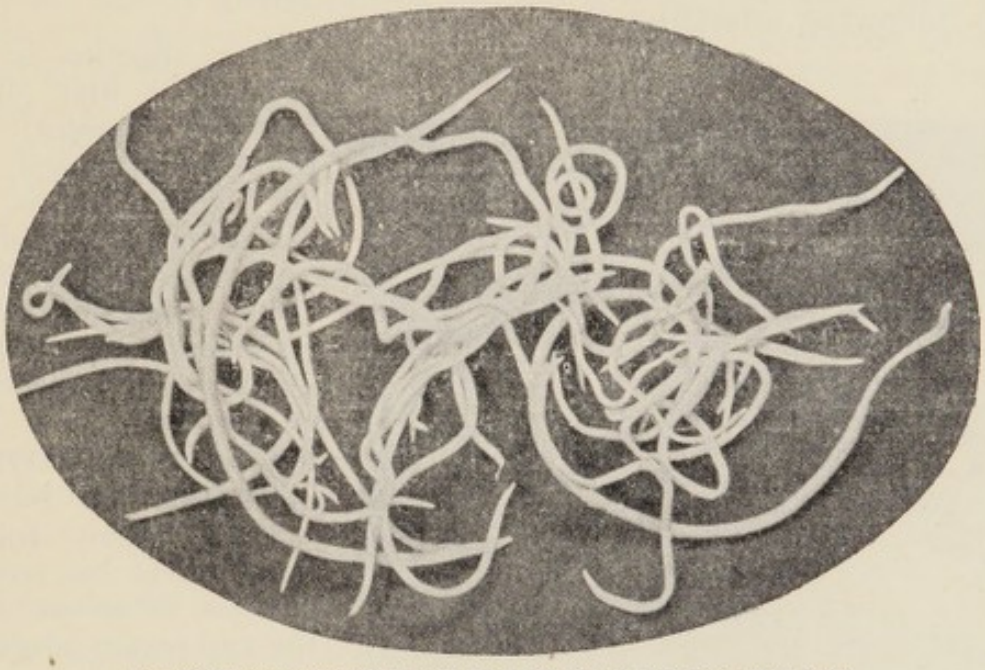
The question is often asked: "How many worms can be present before a bird shows harmful effects of such an infestation?" It is impossible to state a set number in view of the fact that it will vary with the age of the bird, the individual resistance of the bird, the conditions of management and feeding, and the inherent stamina of the particular strain of poultry. However, as a rough guide, it may be stated that where pullets twelve weeks of age are infested with twenty or more worms, harmful effects may be in evidence, and treatment should be carried out.

*For fuller information on this subject readers might consult Bulletin No. 2 of the Department of Agriculture Animal Health Station, Yeerongpilly, Queensland, by F. H. S. Roberts, D.Sc.

Economic Importance—

The parasite infests fowls and may also infest ducks, turkeys, geese and pigeons. Heavy economic loss may be sustained due to:

1. Poor development of birds for market.
2. Retardation by many months in the commencement of egg laying.
3. Loss of egg production from adult fowls.
4. General poor condition of stock (with its business effects on stud stock sales, etc.)



Round Worms (*Ascarida galli*) from the Small Intestines of a Fowl.

Survival of Infective Eggs—

It has been shown that in sunlight worm eggs survive only a few hours. In shaded, moist areas they may survive up to a year, but in shaded dry areas they seldom survive as long as a month.

It will thus be seen that the elimination of damp, shady areas is of utmost importance in the control of this parasite.

The eggs are somewhat resistant to ordinary disinfection, but the use of a boiling solution of 1 oz. to the pint of a disinfectant such as cyllin or similar coal tar preparation is effective.

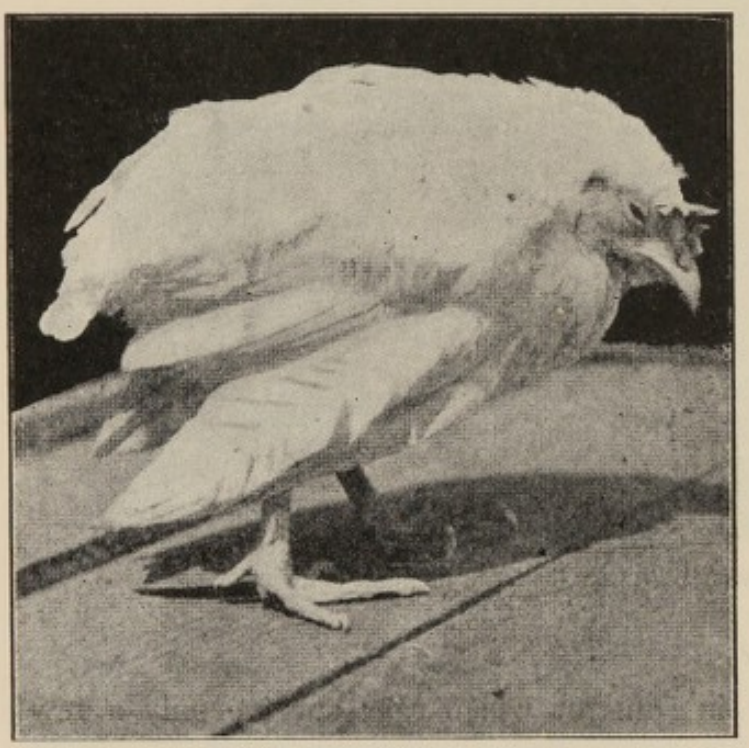
Life History—

The adult worm is usually located in the first portion of the small intestine, though in odd cases it has been seen in the crop, caeca, gizzard and in the oviduct (chance infestation). In the intestine the female worm deposits eggs which pass out in the droppings. Under ideal conditions of warmth and moisture this egg may hatch out to produce a larva which later moults to produce a second stage larva. It is this form which is infective and this may develop as early as the eighth day. If this form is then eaten by a bird it hatches out fully, and lives for about nine days freely in the lumen of the small

intestine. After this the minute worm attacks the living membrane of the intestine where it remains for about ten days. Some of the larval forms may actually penetrate the intestinal wall and become lodged in the liver or lungs. After this burrowing stage the development proceeds in the lumen of the intestine where the adult worm develops and remains.

Symptoms of Infestation—

In light infestation few symptoms may be noted. Where heavy infestations ocur, very marked symptoms may be noticed during the burrowing stage of the life cycle, that is, during the second and third weeks following infestation.



Heavily Worm-infested Pullet.

[After Roberts.

The appetite is decreased at first, diarrhoea is present, and thirst is increased. In very severe cases, the diarrhoea may be blood-tinged. Paleness of the comb, shanks and a general indefinable "withered" appearance is present. The bird huddles up in the typical "sick-fowl" attitude.

Death may follow. Some birds that recover have a voracious appetite and regain normality gradually. Others remain emaciated and sickly, never developing fully, and sometimes become paralysed or extremely weak so that death occurs from inanition.

Adult birds seldom die from the disease, but the symptoms usually comprise general unthriftiness, paleness and poor egg production, together with varying degrees of diarrhoea.

The presence of yellowish, fluid, frothy droppings is frequently an indication of fairly heavy worm infestation.

Diagnosis—

Whilst the above symptoms of infestation may be a fairly reliable guide, the intestines should be slit open (with a pair of scissors) in the case of all birds which die, or are killed for table purposes, and a careful examination for the presence of worms carried out.

Resistance to Infestation—

Older birds (over four months of age) have an increased resistance to infestation. Infestation also results in the infested bird gaining a

certain amount of resistance. When infestation has occurred the value of dosing the pullets at four to five months of age should, therefore, be apparent.

Treatment—

The method of control which appeals to the farmer is flock treatment, through the feed, as it is labour-saving and inexpensive. Unfortunately, it is probable that flock treatment will not be satisfactory in all cases. When a suitable drug is used it will undoubtedly get rid of a large proportion of the infestation in a number of birds, but the most heavily affected birds will not be efficiently dealt with, owing to their depressed appetite, which is often one symptom of heavy worm infestation. On the other hand, the most vigorous and healthy pullets will eat a large quantity of the medicated food, with the inevitable result that they ingest an overdose, and this may upset their health and check their development.

In most cases when the dose rate is cut down so that it is well within the safety limit, and does not seriously decrease the palatability of the ration, the efficiency of the drug is usually very much diminished.

In view of these facts, little mention is made here of mass treatment. It would appear that out of a large range of drugs which have been tried by this means, nicotine sulphate put up in various forms gives the most promising results. One author has found that 0.5 ml. of nicotine sulphate* in 150 ml. of water per lb. of mash (dry weight) kept constantly before the birds gives a reasonably high degree of efficiency. The nicotine sulphate is diluted with the water to ensure even mixing throughout the dry mash.

Tobacco dust fed at the rate of 2 to 3 per cent. in the mash has been advocated for many years in this State, and many authorities claim that this treatment is highly satisfactory. In this State, under field conditions, it has been found that satisfaction is given in many instances, but in others the feeding of this material seems to have little effect on the worm infestation. It has frequently been noted that, although pullets have been fed a mash containing 2 per cent. of tobacco dust since the brooder stage, they still suffer from a very heavy worm infestation.

These varying results would seem to be referable to the fact that some samples of tobacco dust contain more nicotine than others. Samples with a high nicotine content give highly satisfactory results, but samples with a poor nicotine content have little or no effect on the worms. If more than 3 per cent. of low nicotine content tobacco dust is included, the food is unpalatable.

Individual Treatment—

The objections raised against individual treatment have been that it is too costly, that too much labour is involved and, finally, that it is impracticable under poultry farming conditions.

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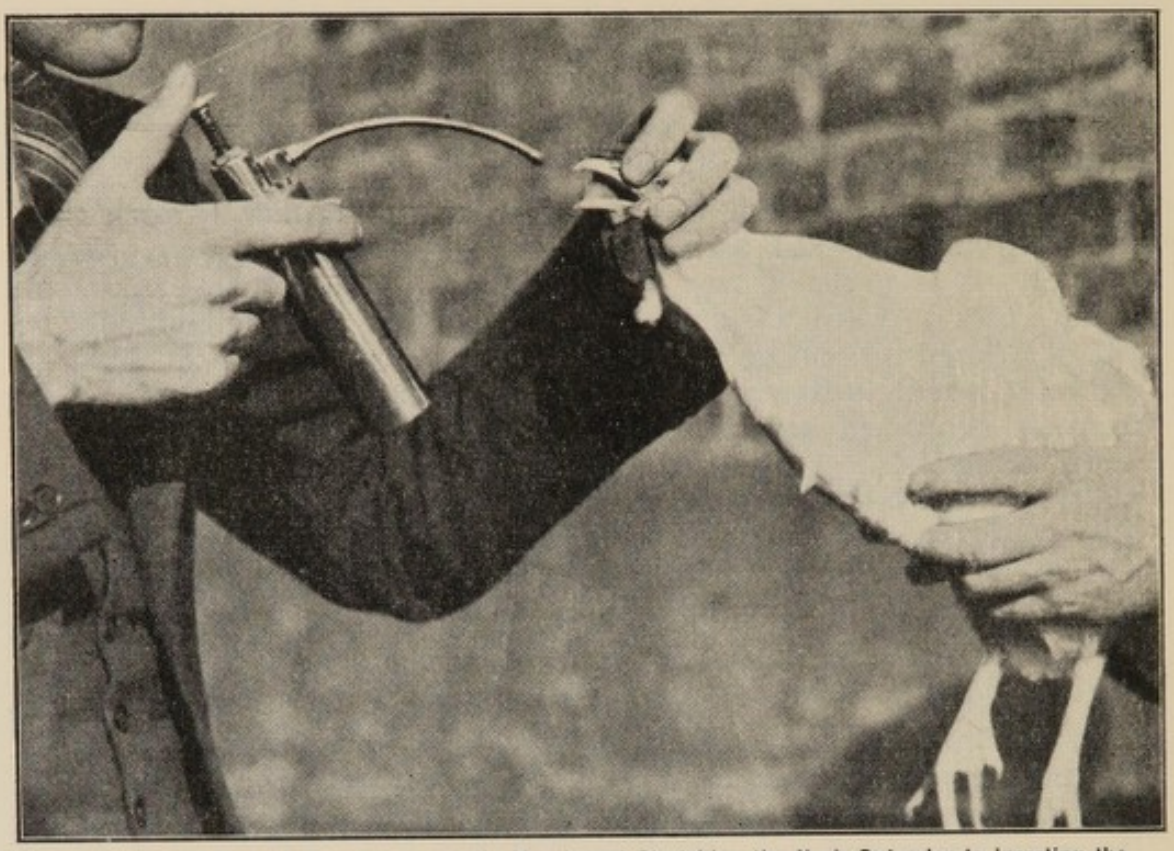
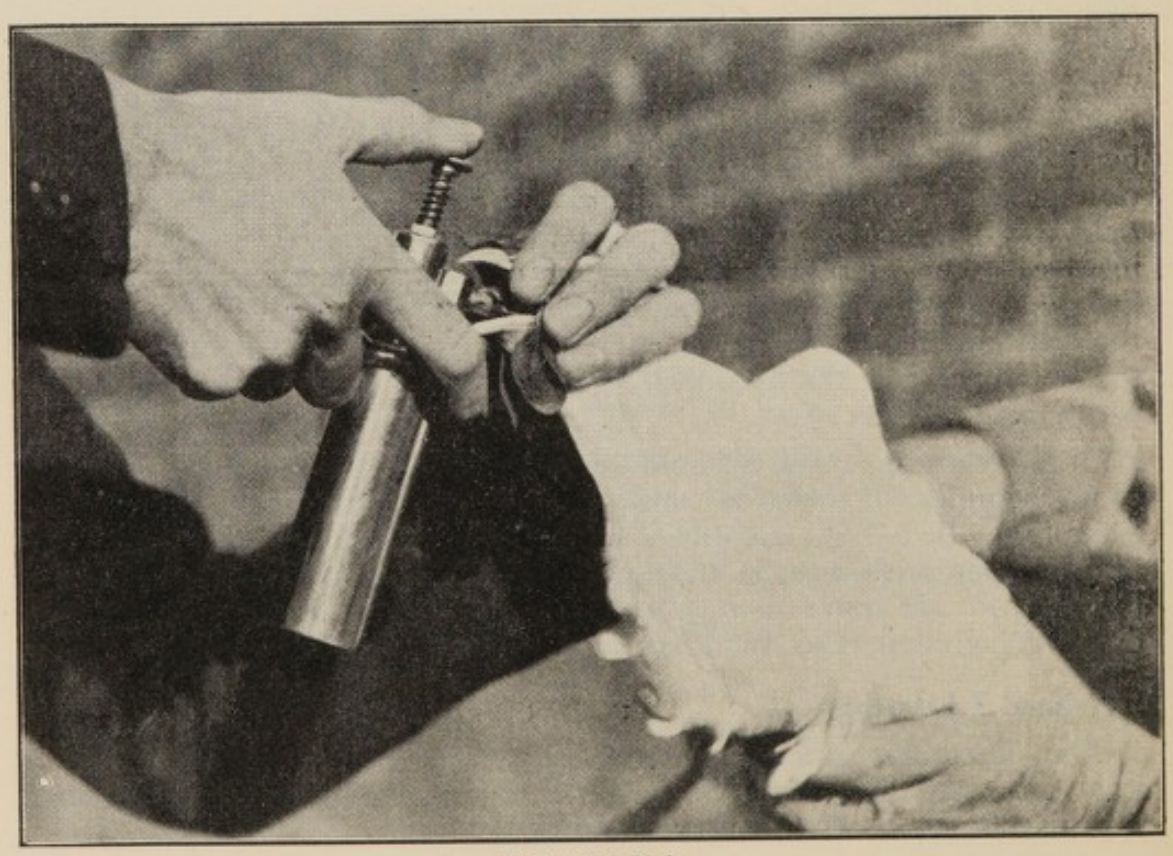
*The dosages are given throughout in millilitres (cubic centimetres). To reconcile these measurements with apparatus available on the farm the following should be borne in mind:—30 ml., 1 fluid oz.; 2 normal tablespoonfuls, 1 fluid oz.; 20 fluid oz., 1 pint; 30 grams, 1 oz.



Showing the Method of Opening the Bird's Mouth and Stretching the Neck Out prior to inserting the "Gun" to Administer the Dose.



Dosing the Bird

Note that the nozzle of the drenching "gun" has been placed right down into the crop. This avoids any of the carbon tetrachloride welling back and gaining access into the trachea. With the particular drenching instrument illustrated, the dose is administered in four rapid injections, each of 0·5 ml., and this delivery of the dose in four sections is also designed to prevent the welling back of the material into the trachea (which would result in almost immediate death). With a dexterous operator and capable assistants 500 to 700 birds per hour can be dosed in the manner illustrated.

It is suggested that these views are incorrect. If dosing is carried out with carbon tetrachloride, using a suitable gun, or syringe and container for the drug, one operator, with two capable assistants, can dose from 300 to 700 fowls per hour, depending upon his dexterity and organisation. It is pointed out that 4,800 pullets, given 1 ml. of carbon tetrachloride, can be dosed with one gallon of this material, which costs approximately £1 per gallon at any of the bulk chemical suppliers in the city. The cost is, therefore, very small both for labour and material used.

The advantage of individual dosing is that every bird gets a uniform dose.

A very wide variety of drugs and medicinal mixtures has been tried out and recommended to expel round worms from fowls. Many of these are highly efficient, but too dangerous, and many are inefficient. A brief notation is made of most of these later, so that poultry farmers may be in a position to avoid being misled.

Carbon tetrachloride has been found to be highly effective. It may be administered in the pure state or may be mixed with liquid paraffin. Mixing with liquid paraffin would appear in some cases to diminish efficiency and add to the cost. The chief advantages of paraffin is that it facilitates the administration of the dose with most syringes. Pure carbon tetrachloride rapidly dissolves all oily material from the washers and syringe fittings, and almost inevitably leads to the instrument leaking. However, it is considered better to administer the carbon tetrachloride in the pure form.

Method of Dosing—

The method and instrument used to administer the dose will determine the practicability of individual dosing.

A special "gun," which delivers ½ ml. per injection is available. For a full adult dose four rapid injections are given. With this gun 500 to 700 birds per hour can be dosed if the attendants can handle the birds quickly enough. Speeding should, however, be avoided, as if a drop or two of carbon tetrachloride is placed in the trachea immediate death of the bird occurs.

Apart from the special gun and also a special syringe on the market, the drug can be administered by any suitable syringe. One convenient method is to carry the carbon tetrachloride in a suitable container attached to the operator's belt. The syringe is dipped into this container and a quantity of the drug is drawn into it. The latter should be graduated in half millilitres, and should have a gently curved nozzle 4½ inches long. This nozzle should not be more than three-sixteenths of an inch in external diameter, and it is an advantage if the point is rounded so that it will not lacerate the birds mouth or throat. Three or four birds can be dosed in rapid succession and then the syringe refilled from the container.

The speed of the operation is usually limited by the rapidity with which the assistant can make the fowls available; with practice the operator can dose fowls as quickly as they can be held in position for

him. Immediately after administration of the dose, the bird should be held in position for about half a second, so that the drug passes right into the crop and is not discharged by the bird flinging its head about.

Birds should be starved for seventeen to twenty-four hours prior to dosing; that is to say, the evening meal should be omitted and the birds should be dosed the next morning. Water should be available to the birds during this fasting period. Feed may be supplied five to ten minutes after dosing. The bird is held by an attendant. The operator takes the bird's head in his left hand, pushes the beak open with his left index finger, and holding the syringe or gun in his right hand, gently but rapidly inserts the nozzle of the syringe down the oesophagus for 4½ inches. In doing this, care should be taken to keep the nozzle of the syringe pressed against the dorsal (top) wall of the gullet to ensure that it passes down the gullet and not down the wind-pipe. While the nozzle is being passed down, the bird's neck should be be held extended. If the nozzle is not placed down the gullet at least 4 inches (that is, almost into the crop), the dose of carbon tetrachloride may be regurgitated, and some of it may pass down the trachea, thus killing the bird. Practice and dexterity are required to avoid such deaths.

Dose Rate—

The desirable dosage of carbon tetrachloride is 0.75 ml. per lb. of body weight, with a maximum dose of 2 ml. That is to say, a chicken ¾ lb. in weight would receive approximately ½ ml., a pullet 2 lb. in weight would receive 1.5 ml., and an adult bird will receive 2 ml.

Birds may be dosed at practically any age, though it will seldom be necessary to treat them before they are five weeks of age.

After Treatment—

Immediately after dosing, the birds should be placed on wire netting so that worms voided may be collected on the floor of the house. Placing birds on wire netting may be arranged in several ways: Large crates may be made into which the birds are placed, or wire netting frames should be constructed and placed on the floor of the house. These frames should be made in sections to fit all houses in which it is desired to treat birds. Birds should be left on the wire netting for at least six hours, by which time most of the worms will have been voided.

The practice of treating birds for worms and then allowing them their usual range cannot be too strongly condemned. If the drug is effective (no matter what particular drug), the worms will be voided in the droppings, and this will result in a heavy infestation of the ground surface with worm eggs, which may later reinfest the poultry.

Local Field Experience—

On one farm, nineteen pullets, mainly White Leghorns of an average weight of 3 lb., were dosed with carbon tetrachloride, 6 mls. being administered directly into the crop, following twenty-four hours starvation. Within three hours some thousands of worms were passed in the droppings of these birds. The pullets were off their feed and

generally showed symptoms of depression for the next twenty-four hours. After this period they were brighter, grew more rapidly and in every way were superior to pullets in an undosed control group.

In the next season on the same farm, three colony yards containing in all approximately 700 birds seven to twelve weeks old were selected. After twenty-four hours starvation the birds in one of these yards were dosed with 1.5 mls. of carbon tetrachloride and confined on wire netting in their houses for five hours. Epsom salts at the rate of ¾ oz. per gallon of drinking water was supplied and two and a half hours after dosing many thousands of worms were found on the concrete floor underneath the wire netting.

Several birds were examined post-mortem within the next fortnight and in no case were any worms found to be present in the intestine. No inappetence or depression of any kind was noticed as a result of the dosing with carbon tetrachloride. A fortnight after dosing, these pullets were so markedly ahead of the other two yards in development that the owner declined to leave any birds untreated as a control group. These two yards were then dosed with carbon tetrachloride as before, the dose varying from 1 to 2 mls. of the drug, depending on the size of the bird.

In these three yards, approximately 700 birds were treated, and in no case were any harmful effects noted. Very large numbers of worms were voided and there was a marked improvement in vigour, growth and general appearance immediately subsequent to dosing.

On the same farm in July of the same year, two even groups of birds were made, each comprising twenty White Leghorns and twenty Black Orpingtons. One group was dosed with 1.5 mls. carbon tetrachloride following twenty-four hours starvation, and drinking water with ¾ oz. Epsom salts per gallon was available to them following dosing. The other group was left undosed.

	Weights.	
	Undosed Controls.	Experimental Group.
	lb.	lb.
20th July, 1937	70	73
8th October, 1937	113	120
Gains	43	56

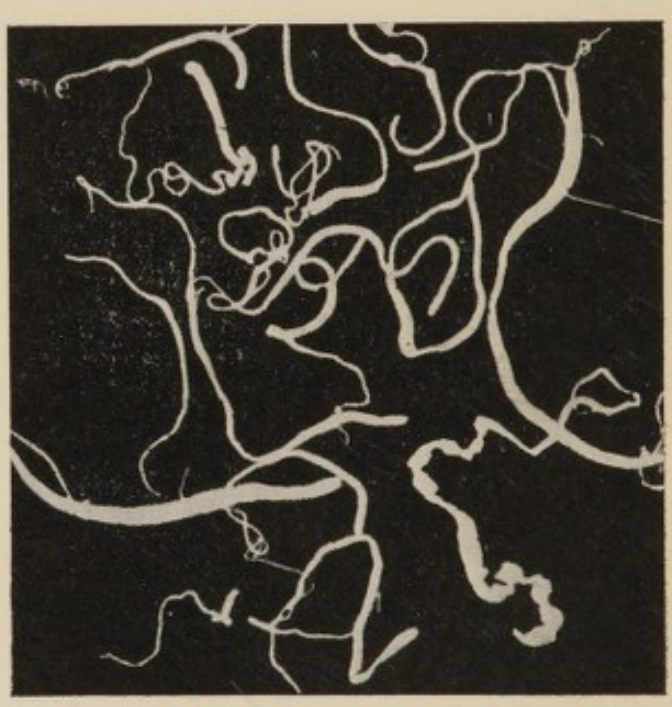
The weights do not wholly reflect the general improvement shown by the birds. The experiment was not continued beyond October, but at the time of disbanding the birds, most of the dosed group were "reddening up" prior to laying, whereas the undosed controls were many weeks from this stage.

The birds were run in small colony yards under exactly the same conditions as the rest of the young stock reared on the property. The control group was typical of the rest of the untreated pullets on the farm. The improvement shown by the treated birds was constant throughout and would immediately be noted by the most casual observer.

A month after dosing, two treated birds that were affected with fowl paralysis were killed, and only one small round worm was found in one of these birds. Every undosed bird killed on the farm showed a moderate to very heavy worm infestation.

In order to test the toxic effect of carbon tetrachloride, thirty-two birds which had been culled on another farm because of their sickly appearance were divided into two even groups. One of these groups was dosed with 3 mls. of carbon tetrachloride. No birds in either group died within the next three days and the treated birds were in no wise badly affected by the drug.

On a third farm, eighty-four Leghorn pullets (fourteen weeks old) that were very heavily worm infested were divided into two equal groups, and forty-two of these were dosed with 1.5 mls. of carbon tetrachloride. Large numbers of worms were passed within a few hours. The birds had been starved for twenty-four hours prior to dosing. No systemic upset was noted as a result of the administration of the drug. From a week after dosing the treated birds were markedly brighter that the untreated controls. A fortnight after dosing their growth rate was obviously greater than the control pullets. Unfortunately the groups were accidentally disbanded prior to weighings being made. Many more similar cases could be cited.



Larger Tapeworms of Poultry
(*Raillietina tetragona*).

Efficiency of the Drug—

It would appear that in most cases the drug is nearly 100 per cent. effective where treatment is carried out in the correct manner as above indicated.

Doses of up to 6 mls. have been administered without causing fatalities, though this dose rate causes depression of the birds. With a dose rate varying up to 2 mls. the drug would appear to be perfectly safe and highly effective.

Treatment can be repeated at fortnightly intervals on two or three occasions if so desired. It is, however, usually found sufficient to dose the birds once, or at most to repeat dosage in six weeks.

Other Drugs and Medicinal Mixtures—

Tetrachlorethylene.—This drug has been recommended by many authorities and has been found safe and effective in doses from ½ to 5 mls., the latter dose being given to birds 3 lb. or more in weight.

It has, however, been found more toxic than carbon tethrachloride, and it would appear to be somewhat irregular in its efficiency, though, generally speaking, it removes a large proportion of the worms.

Nicotine Sulphate.—Individual dosing with this drug has not always been satisfactory, many authorities recording toxic and lethal effects. When administered with a substance known as Lloyd's alkaloid reagent, its toxity is reduced, but it is then found by some authorities to be inefficient.

Oil of Chenopodium.—This drug has been very favourable reported on in doses varying from 0.15 ml. to 0.5 ml. per kilo of the bird's live weight. However, it is dangerous when enteritis is present, and, as the latter condition is so common in chickens and pullets (as a symptom of coccidiosis, entero-hepatitis and other epizootics), it is considered that the drug is not suitable for general use.

Copper Sulphate.—This drug is widly used in this State. A dose of 15 grains or 1 gram (1 oz. to thirty birds) has been found effective in removing worms from adult birds, but this dose rate is highly dangerous for pullets. When fed in safe dosages it has only a low degree of efficiency.

A proprietary preparation of copper sulphate and mustard added to the ration in small quantities failed to remove worms.

Nicotine Sulphate and Copper Sulphate.—Both in a preliminary field trial in this State and in other trials, when adequate amounts of these materials were fed in the mash, the birds refused to eat the mixture owing to its unpalatability.

Oil of Turpentine.—This substance is widely used for the de-worming of poultry in this State. A dose of 1 ml. per bird, given in twice the volume of castor oil, is not completely effective in removing worms from pullets. Double this dose is effective, but has a toxic effect upon the birds. It will, therefore, be seen that the effective dose is too near to the harmful dose of this drug to be recommended.

Flock treatment with oil of turpentine is frequently carried out, but in view of the toxicity of turpentine this cannot be recommended.

Pyrethrum.—One author claims 95 per cent. efficiency from dosing with 200 mg. of this drug. Other workers have not gained this success and it would not appear that the drug is a reliable anthelmintic.

Nicotine and Arsenic.—In a preliminary field trial carried out by the Department, there was no positive evidence that this treatment was of value.

Powdered Pomegranate Root.—In one preliminary field trial no positive evidence was obtained of the value of this preparation. However, the material was not analysed for rotenone content, and it is understood that the efficacy of the drug varies in direct proportion to the rotenone content.

Normal Butilydene Chloride.—Favourable reports have been made following the use of this drug in doses from 1 to 6 ml.

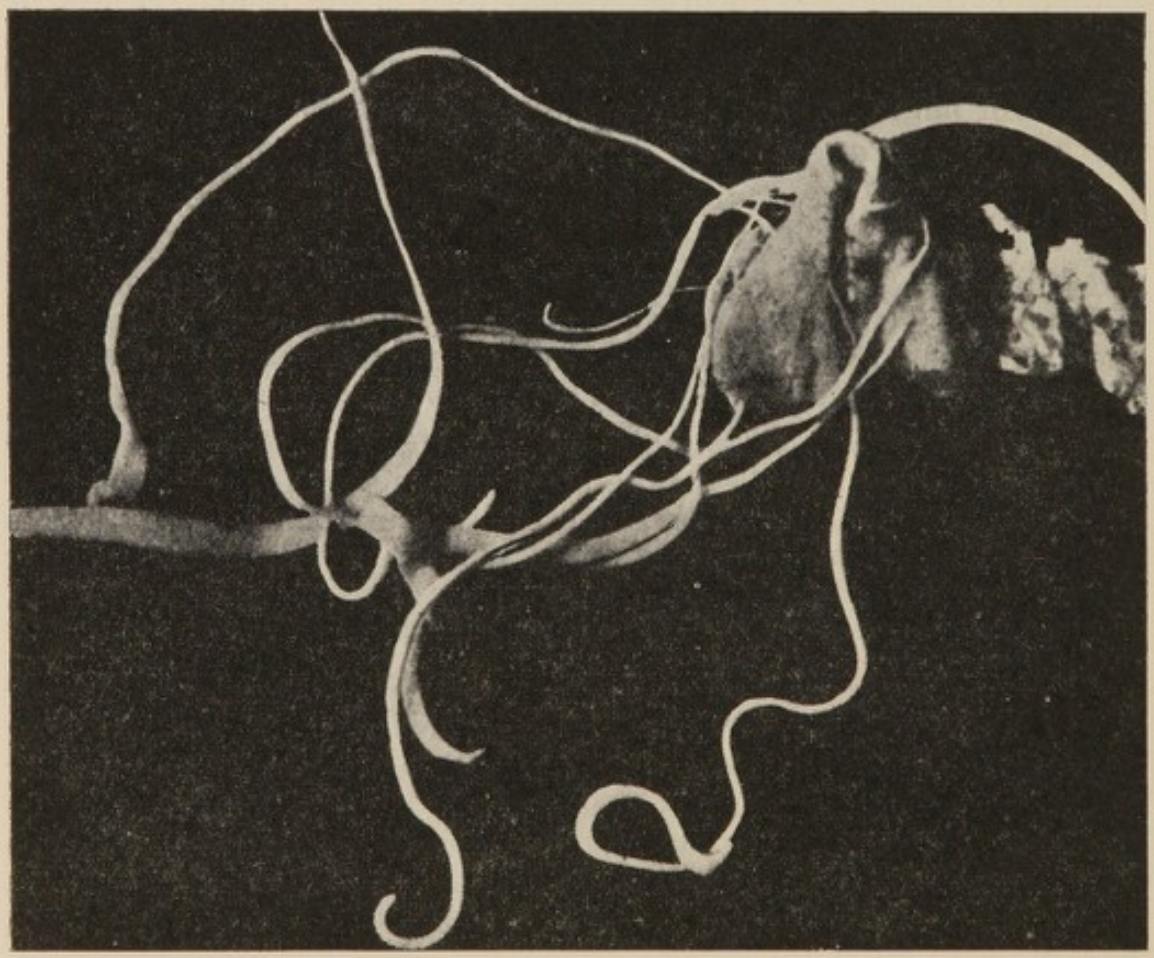
Carbon Bisulphide.—One authority in Queensland reports that this drug would appear to be too toxic for general recommendation, though fairly effective.

Iodine Vermicide.—This is largely used in America, and is claimed to be reasonably effective in the removal of round worms.

Ascaridol and Nemural.—These preparations are largely used on the Continent for the treatment of round and tapeworms.

Naphthalene.—It has been claimed that this drug is very effective.

Miscellaneous.—The following materials have been tried, but their efficacy in removing round worms would appear to be poor:—Benzine, arecoline, oil of eucalyptus, kamala, calomel, bismuth carbonate, santonin, kousso, potassium bichromate, ferri perchloride, pelletrine, thymol, oil of philmaron, ipecacuanha, tansy, beta-naphthol, bismuth subnitrate, terebene.



Another Type of Tapeworm Infestation.

Prevention of Infestation—

In the control of worms in poultry it is of the utmost importance, wherever possible, to prevent infestation. Yards should be spelled for a portion of each year if this can be arranged, and chickens should be

placed on new ground, or at least on ground to which adult fowls have not had access for many months. Where insufficient space precludes this, it is highly desirable to remove the top inch of soil from the chicken runs and replace it with fresh soil.

Green stuff fed to chickens should not be from a patch which has been treated with fresh fowl manure as such feed may be heavily contaminated with worm eggs.

Damp, shady areas in the chicken runs should be avoided, as they may retain worm eggs and larvae in a viable condition for long periods.

Food and water troughs should be so arranged that their contents will not be contaminated by droppings.

Rigid attention to hygiene, feeding a suitable ration, the avoidance of over-crowding, the provision of large open runs, and the maintenance of the general health of birds are all important factors in any scheme for the control of worm infestation.

Other Round Worm Infestations.

The small round worm (*Heterakis gallinae*) is a slender white worm about half an inch in length. This worm is located at the blind end of the caeca (or blind guts) and is present in the vast majority of birds examined in New South Wales. It infests fowls, turkeys, ducks, geese, guinea fowls, pheasants and pea fowls. It appears in most cases to be rather a harmless parasite. In very severe infestations inflammation of the caeca (typhilitis) may be produced.

One feature of importance is the association of this worm with the incidence of enterohepatitis (blackhead) already mentioned. The protozoan organism causing enterohepatitis is readily killed by drying and exposure when alone, but may remain alive when protected in the eggs of this worm for considerable periods.

It also appears that slight damage caused by the worm favours the establishment of infection, and without the presence of this worm it is stated that blackhead seldom, if ever, occurs.

Whilst a number of treatments are partially effective, none of them can, as yet, be recommended to be economical and effective.

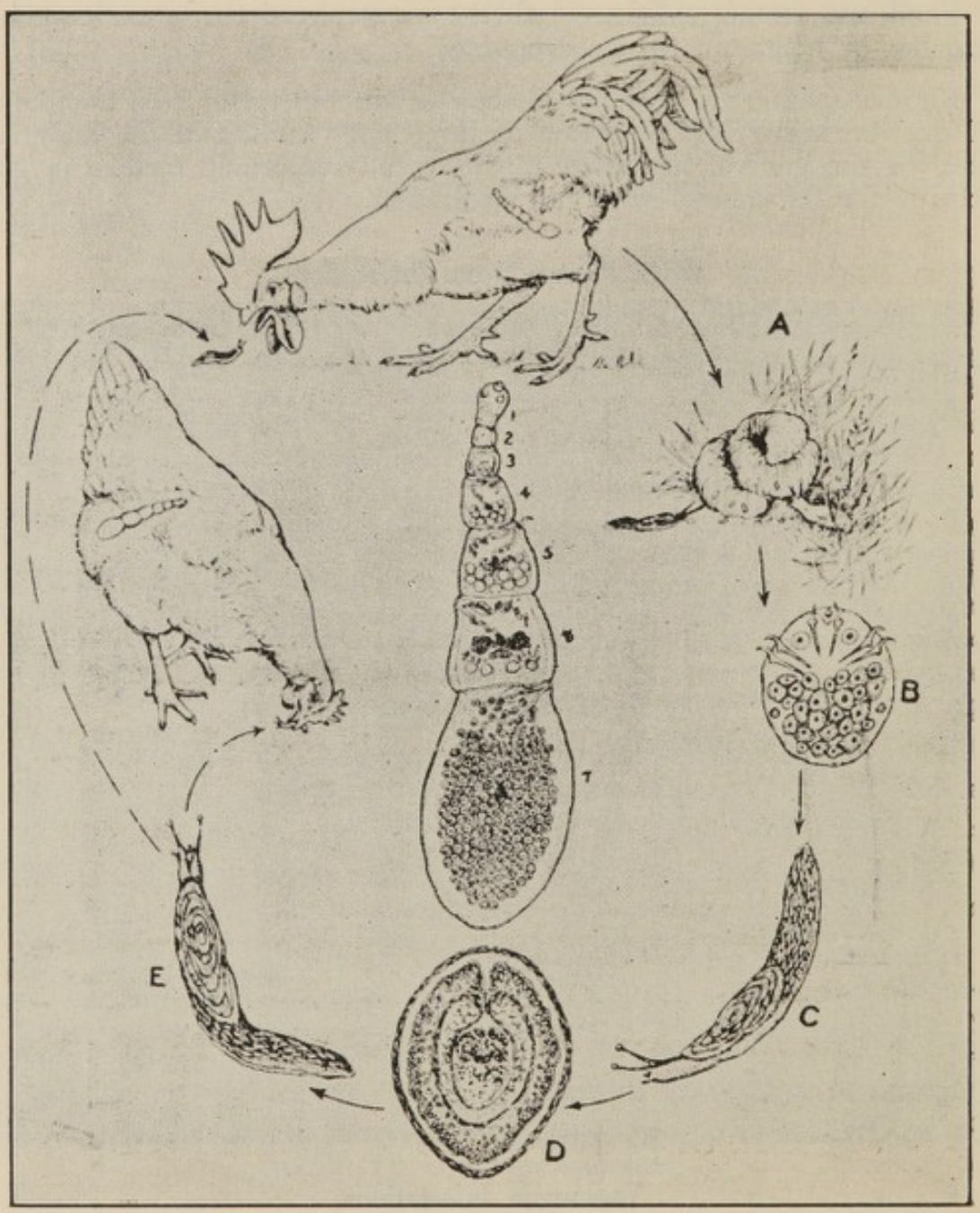
It has been noted following extensive field drenchings with carbon tetrachloride for large round worms (*Ascardia galli*) that the incidence of this caeca worm has been greatly reduced. Critical tests have not as yet been carried out. Other general control measures as used for the control of the large round worm will also aid in the control of this parasite.

Tapeworm Infestations.

Tapeworms are flat, segmented worms which vary in size from very tiny thread-like worms only about one-eighth of an inch long to quite stout worms over a foot in length. These parasites have an indirect life cycle, that is, the worm eggs are passed in the droppings,

are picked up by some secondary host which varies with the individual worm, but may be forms of animal life such as snails, slugs, butterflies, moths, grasshoppers, beetles, flies, and so on. In the body of these secondary hosts the tapeworm develops into an infective resting stage. If the secondary host is then eaten by a fowl, the infective tapeworm parasite develops to maturity.

Unfortunately, it often happens that where fowls are run on wide open range conditions (which are otherwise ideal) the opportunity is present for the extensive development of tapeworms.



(1-7) Tapeworm *Davainea proglottina*, (1) scolex or head with suckers and hooks, (2-3) undifferentiated segments, (4-5-6) segments with male and female generative organs, (7) mature segments or proglottids with eggs; these drop off at intervals and carry on the infestation. (A) Droppings with proglottids, which crawl out and are being eaten by slugs, (B) small embryo (oncosphere) armed with six hooks, (C) slug containing oncosphere, which develops into— (D) encysted shape, which forms in the slug and is eventually eaten by a fowl, and develops into a fully grown tapeworm (*Davainea proglottina*).

Types of Tapeworms in New South Wales—

The following types of tapeworm parasites occur commonly in the flocks of this State:—

Raillietina spp.
Choanotaenia spp.
Davainea spp.
Amoebotaenia spp.

Effects of Infestation—

The birds become emaciated, pale and appear as "poor doers," with a characteristic dull plumage. Where the infestation is heavy the birds are much lighter than normal. Diarrhoea is present and the fluid droppings may contain segments of the tapeworm.

The appetite is capricious, the bird is depressed and easily caught. In heavy infestations weakness of the legs or even flaccid paralysis of one or both legs is not uncommon.

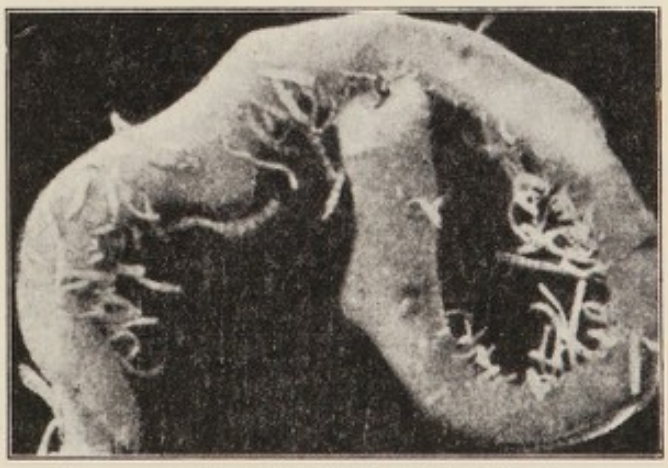
A Common Tapeworm of Poultry in Illinois (*Raillietina cesticillus*).

Measuring about 1 to 6 inches in length, this parasite is usually found in the forepart of the small intestine. One stage in the life cycle is spent in either the housefly or in some species of beetle. At the left is the immature stage magnified x 1½. At the right is the mature stage magnified about x 2

A flock of pullets which becomes badly infested at six weeks of age may have the development checked, and many birds at eight and nine months of age will not have come into lay or may not have even "combed up" prior to laying, but still have the appearance of birds about five months old.

Treatment—

Whilst many drugs (*e.g.*, Kamala, malefern, turpentine, oil of chenopodium, pomegranate root or flowers, arecoline), will remove some or most of the strobilae or bodies of the tapeworms, none of these drugs appears to be effective in removing the tapeworm heads, which are firmly embedded into the intestinal wall under the covering of slime. It has been shown that such heads rapidly build up another body.

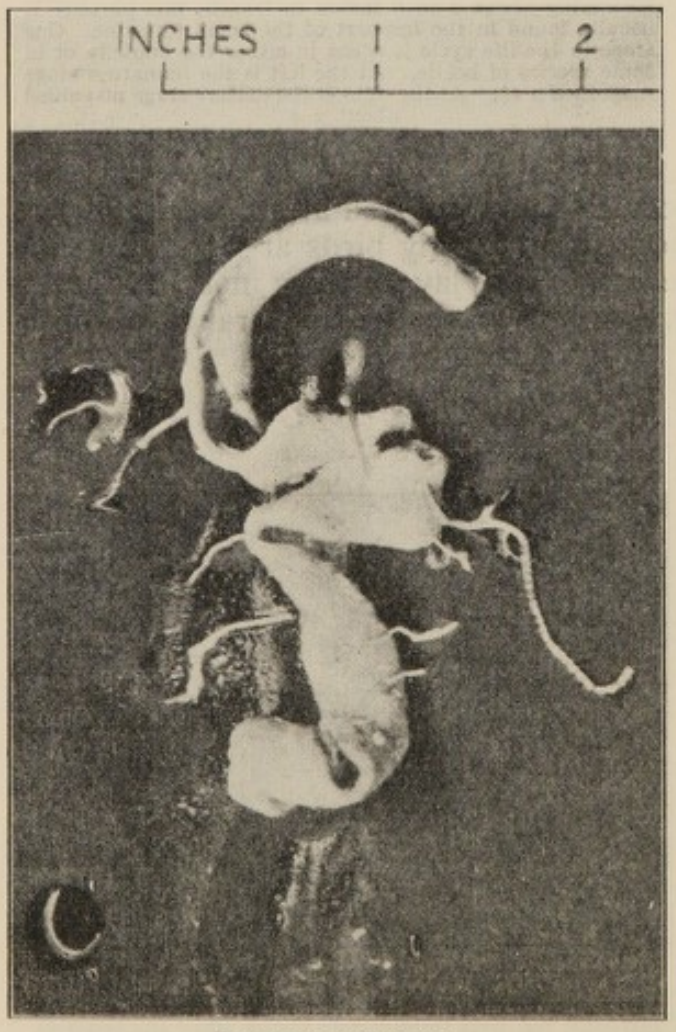


Tapeworms Attached to Inside Wall of Intestine.

These worms could not be seen from the outside of the intestine, but were immediately visible when the intestine was turned inside out. Natural size.

Immature and Mature Stages of a Smaller Tapeworm.

The small intestine of chickens and turkeys is the site of this threadlike, very fragile, and delicate worm, which is about 1½ to 3½ inches long. Stable flies and dung beetles are the intermediate hosts. The upper illustration shows the immature stage magnified about x 2½. The lower picture, the mature stage magnified about x 2.



Tapeworm Infestation.

At present there is urgent need for research work to ascertain some drug which will remove the worm head and will be practicable as regards cost and mode of administration.

It has been noted that when doses of from 2 to 6 mls. of carbon tetrachloride have been administered to White Leghorn hens that the bodies of the tapeworms come away.

Control—

No satisfactory means can be recommended. That is to say, when all precautions possible have been taken with pullets on large colony ranges infested with the parasite, infestation still occurs.

So far as possible, all moist areas should be avoided and where possible new land should be used for young stock or land kept only for this purpose. Frequent cleansing, collection, and conservation of droppings will limit the infestation of the intermediate hosts.

Destruction of the intermediate hosts may in some cases be practicable, or exclusion of fowls from the areas where they are located may be possible in other cases.

When a farmer's flock is troubled with tapeworm infestation, veterinary advice should be sought, so that the whole matter may be investigated on the farm and appropriate advice given.

Eye Worm (*Oxyspirura parvorum*).

This parasite is reported to occur in Queensland. Odd cases have occurred in this State, but these are probably introduced from warmer climes and infestation with this parasite is of no economic importance here.

Gape Worm (*Syngamus trachea*).

This parasite has been reported in other States but not in this State. The parasite infests the upper portion of the windpipe and larynx, causing irritation and inflammation, which may cause confusion with the roup diseases except for the fact that so far it has not been noted locally.

A number of other worms, such as *Capillaria* and the gizzard worm, are known to infest the crop, glandular stomach and gizzard, but none of them seems to be at all common or to cause marked economic loss in this State and further mention will not be made of them.

Pigeon Worm (*Ornithostronglus quadriradiatus*).

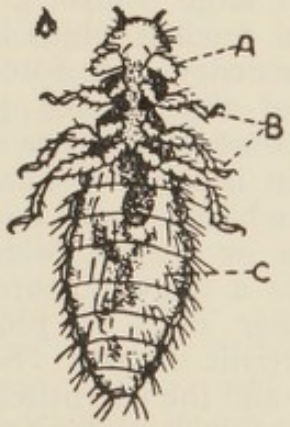
This is a worm parasite which causes severe mortality in pigeons in other countries. Little investigation of pigeon mortality has been made here, and it is not known whether the worm often causes trouble in local pigeon lofts. Symptoms are diarrhoea, emaciation and death in a very short period of time, and control is by rigid sanitary methods.

External Parasites.

A Photomicrograph of the Goniocotes Gigas.

Head, showing antennae or feelers at 1, (2) the mouth parts, (3) thorax with legs provided at extremity with small hooks at 5. (6) abdomen.

[*After Kaupp.*

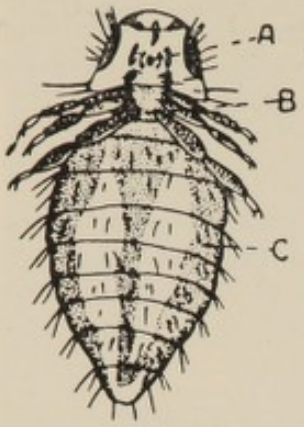


Menopon Biseriatum.

(A) Head provided with mouth parts for biting, feelers or antennae, and eyes, (B) legs attached to the thorax, (C) abdomen.

[*After Kaupp*

Quite a large variety of external parasites may trouble poultry. Some of the more common and most important are: —



Menopon Pallidum.
(A) Head, (B) thorax provided with three pairs of legs, (C) abdomen bearing hairs.
[*After Kaupp.*

(1) Mites.—
  (*a*) Red Mite—*Dermanyssus gallinae* (see Fowl Tick Fever, *spirochaetosis*).
  (*b*) Scaly leg (*Cnemidocoptes mutans*).
  (*c*) Depluming mite (*Cnemidocoptes gallinae*).
(2) Lice.—
  (*a*) Body lice, including shaft and feather lice.
  (*b*) Head lice.
(3) Ticks.—The fowl tick, *Argas persicus* (see Fowl Tick Fever, *spirochaetosis*).
(4) Fungi forms (see also Mycosis).
(5) Fleas.
(6) Mosquitoes.

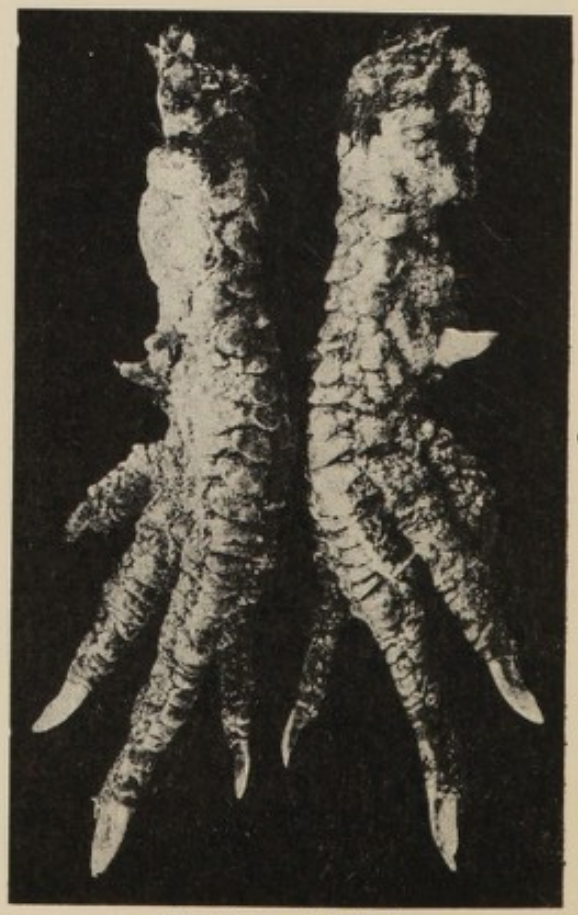
Scaly Leg in Poultry.

This disease is also erroneously called "Elephantiasis." It is due to the activities of a mite *Cnemidocoptes mutans*. The mite is microscopic, burrowing under the scales of the leg, producing irritation.

Birds affected.—Fowls, turkeys, cage birds. Unsanitary conditions favour its spread. Heavy breeds seem to be more prone to infection than light breeds. Chickens brooded by an affected hen may show symptoms of the condition when a fortnight old.

Symptoms.—In adult fowls it appears as a marked thickening of the shanks and toes, with the presence of large whitish grey, dirty-coloured deposits which are chalky in consistency and force up the scales of the affected parts. If careful observations are made, the condition is seen to commence as a minute blister under the scales of the toes or shanks, which bursts to exude a straw coloured serum. The latter dries to produce the characteristic deposits. Scales may be shed and the encrusted legs may develop to twice or three times their normal thickness. Irritation, most severe at night, is produced; soreness is unusual but may occur in most severe cases. The irritation may produce loss of sleep, illhealth, and depression of egg laying.

Infectivity.—Scales from the legs spread the infection for as long as thirty days. Dirty conditions facilitate spread from bird to bird.



A Bad Case of Scaly Leg.

Treatment.—In bad cases must be persisted with. Scrubbing with soap and warm water as a preliminary measure softens the scales and the affected area may then be treated with any of the following:—

(1) Kerosene and lard or oil, equal parts.

(2) Sump oil.

(3) Phenol—1 oz. (two tablespoons) to the pint of water.

(4) Kerosene emulsion, made strongly, *e.g.*, 1 gallon kerosene, 2 lb. soap, 4 gallons of water.

(5) Lime-sulphur solutions.

It is important to treat the first few cases seen and so save much labour and expense.

Depluming Mite (*Cnemidocoptes gallinae*).

Infestations with this parasite occur now and then in this State. The mite becomes located at the base of feather quills, particularly of the head, neck and rump. The affected quill may fall out or be plucked out by the bird, due to the irritation.

Treatment.—Where individual birds only are affected, these should be disposed of promptly. Individual treatment may be carried out by dusting the birds with a mixture of sodium fluoride one part and flour four parts, or by treating the affected areas with a mixture of 1 part creosote, 20 parts vaseline, sulphur ointment or kerosene emulsion (as used for fowl tick spraying—which see). Birds may be dipped in a soapy emulsion of:—Sulphur, 2 ozs.; sodium fluoride, 1 oz.; soap, 2 oz.; water, 1 gallon.

Dipping is always undesirable in laying stock.

Lice Infestation of Poultry.

Many different types of lice affect fowls and other birds. A detailed account of these cannot be given for reasons of space, but the remarks applying to the lice infestation of fowls apply in a less degree to other domesticated birds, each domesticated species having its own groups of lice. Some species of lice occur on more than one bird. In poultry the biting lice are the only ones of importance in this State. Some of the commonest of these affecting fowls are as follows:—

Head Lice of Chickens (*Lipeureus heterographus*).—This is a greyish louse, which infests the bird on the top or back of the head, underneath the beak and around the ears. The louse remains close to the chicken's skin and eggs are laid on the small feathers in the vicinity of the head. The life cycle of this species may take only three weeks, and when an infestation occurs in a brooding plant the parasite may rapidly spread from chicken to chicken. Chickens which are hen-reared may contract the infestation from the broody hen.

This species does considerable harm, producing irritation, wasting, and even death of a chicken infested. On occasions symptoms of paralysis may be noticed in the infested chickens, but cases of paralysis occurring in chickens are for the most part caused by diseases other than infestation with head louse.

Treatment for this louse is by smearing the infested area with olive oil or a mixture of kerosene and lard, one part of kerosene to two parts of lard. The infected area may also be smeared with a dilute solution of Blackleaf 40 (40 per cent. nicotine sulphate) in water.

The Body Louse (*Menopon biseriatum*).—This is a large yellow coloured skin louse. It infests chickens and fowls, and in heavy infestations results in skin irritation, which is evidenced by reddening and at times the formation of yellowish crusts. The eggs of this species are laid in large numbers in matted clumps around the base of feathers. The life cycle may be completed in three weeks.

The Shaft Louse (*Menopon pallidum*).—This is a very common, pale-coloured louse, which normally exists on the shaft of the feather. When the feathers are opened this louse may be seen scurrying towards the body.

Other species of lice are common, such as the wing louse and the fluff louse. All species produce severe irritation when the infestation is heavy, and this may result in malaise, capricious appetite, diarrhoea, and the general indefinable appearance of "sickness."

It is not generally realised that lice infestation may be a very serious drain on egg production.

Treatment.—As the parasites remain on the fowl during their life history, treatment must be aimed at killing them on the bird. The most satisfactory method is to select a still night when there are no air currents. Just before the birds roost a fine trickle of Blackleaf 40 (40 per cent. nicotine sulphate), the material commonly used for spraying in orchards, should be placed along the perches. This may be applied with a fine camel hair brush, or better still, a small hole may be punched in a small tin, such as a tobacco tin, and a very fine trickle run along the centre of the perch. Too much should not be placed on, both from the standpoint of economy and from the fact that excessive application, leading to heavy fuming, may result in the anaesthetisation or even death of birds.

When the bird rests on the trickle of Blackleaf 40, the heat of the body causes this material to fume. The fumes rise through the plumage, causing the death of the lice. Treatment will need to be repeated two or three times at intervals of seven to ten days, to kill all the lice which hatch out from the eggs, which are not affected by the Blackleaf 40 fumes. If a windy night is chosen for treatment, the fumes do not rise evenly through the plumage and treatment will be of little value. The provision of loose soil and ashes as baths for birds is desirable and will keep infestations in check, particularly if some flowers of sulphur is added.

Other methods of treatment for lice are the dusting of birds, while held upside down with a mixture of sodium fluoride and some vehicle such as flour or dust, one part of sodium fluoride being mixed with four parts of flour. Dipping may also be carried out, the birds being dipped in a solution of 1 oz. of sodium fluoride in each gallon of water. This latter method is not recommended, as dipping is a serious upset to a fowl's health and will probably put a bird off the lay. If it is desired to dip special birds, such operation should only be carried out on the morning of a warm day.

It is pointed out that the treatment of perch joints and perches with wood-preserving oil (the crude cresote preparation used in the building industry), in addition to controlling infestation of a red mite, will help to prevent the spread of lice from bird to bird. Further, in the early stages after the application of wood-preserving oil, the heat of the bird's body will cause creosote fumes to rise through the plumage, thus killing many of the lice.

Poultry farmers should be careful to examine odd birds in their flock from time to time, spreading the feathers around the vent and breast quickly and making a careful scrutiny for the presence of lice.

Treatment is not expensive, but omission to treat heavy infestations will become very costly due to the depression in egg production.

Fungi.—Internal mycocis has already been dealt with. External infestation with fungi is noted from time to time, being referred to as favus.

Favus (Commonly known as "White Comb").

This disease occurs in New South Wales and is seen in commercial poultry farms, though it cannot be regarded as a common disease. When it does occur, its main effect is to spoil the appearance of the fowl and perhaps cause some minor loss in condition. Only in rare cases, and then usually from secondary complications, does the bird die.

Favus.

[*After Kaupp.*

Cause of the Disease—

It is not clear whether or not all cases of "white comb" are due to the fungus which typically causes this disease and is known as *Lophophyton gallinae*. It is quite conceivable that there may be an inflammatory condition of the skin caused by other species of fungi. One other that has been incriminated as causing such trouble is *Achorion schoenlenii*.

Symptoms—

The disease is first noted in the skin near the beak. Numerous small white papules (or small rounded elevations) will be observed. If these are examined under a hand lens, a fine downy covering will

be seen soon after they first appear. These elevations enlarge and coalesce, forming a layer of greyish material. The thickness of this crust gradually increases. The condition spreads over the skin of the face, around the eye, on the wattles and comb, and, in bad cases the whole comb and wattles surface may be involved. The thickness of the scaly covering varies in different places and may be scraped off in fragments or flakes. The condition often spreads to the surrounding feather portions, forming a dirty whitish encrustation around the feather bases. On pulling out feathers from the affected area they may break off or come out intact, in which case some of the fungoid encrustation will adhere to that portion of the feather on the level of the skin surface.

Favus or "White Comb."
[After Ward and Gallagher.

The disease is not drastic in its course, there being a tendency for the diseased area to recover, but apparently no immunity is developed by the bird; consequently recovered areas may become re-infected, and so the disease may go on indefinitely in a flock, unless careful preventive measures and treatment are adopted.

The infection may be transmitted from fowl to fowl by taking the scales from a diseased bird and rubbing them on the comb or wattles of another bird. The disease will then develop in about a fortnight on the fowl so infected.

Treatment—

Numerous drugs and mixtures are effectively used for the treatment of this disease. The critical factor in curing outbreaks in a flock is the frequency with which the treatment is carried out and the care with which cleaning and disinfection of houses, coops, yards, etc., is undertaken.

In treating, the scaly material should be softened with soap and warm water, and as much of it as possible scrubbed off. A solution of 1 drachm of salicylic acid in 1¼ oz. of methylated spirits should be scrubbed into the affected area (by means of a tooth brush). This treatment was found very effective for one outbreak dealt with by the author. Tincture of iodine is also effective at times, as is a 5 per cent. solution of resorcinol.

Ointments are sometimes used. Salicyclic acid ointment 10 per cent. and red mercuric oxide ointment 10 per cent. are both suitable. When the disease occurs in the feathered portions of the body, the most economical solution to use is perchloride of mercury, 1 part to 500 parts

of water. The only practical way of treating the disease is to make a careful examination of the heads of all birds at night time with an electric torch, remove all affected birds into rigid isolation and treat them at least once daily.

The main economic importance of this disease is due to the depreciated sale value of both adult and young stock, as naturally other farmers will avoid any possibility of introducing infection on to their farms.

Fleas.

The Fowl Flea—

Ceratophyllus gallinae may occur in dirty conditions of poultry husbandry. Such parasites cause irritation, loss of health, and depression of egg production. Control is by frequent cleansing and spraying the infested areas with kerosene emulsion. (Use the same solution as recommended for fowl tick eradication.)

The Stickfast Flea—

Echidnophaga gallinaceae is a very serious parasite where it occurs causing most severe irritation and upset of health.

It occurs in other States, but it is not certain whether infestation has ever been seen in this State. Other closely allied species do occur. Where infestation does occur, thousands of the insects may bury themselves deeply into the skin of the comb, wattles and face, producing severe irritation, leading to emaciation, ill-health and sometimes death.

In addition to control by cleanliness and spraying, affected birds should have the infested parts of the head and neck smeared with a mixture of 1 part of kerosene to 20 parts olive oil.

Mosquitoes.

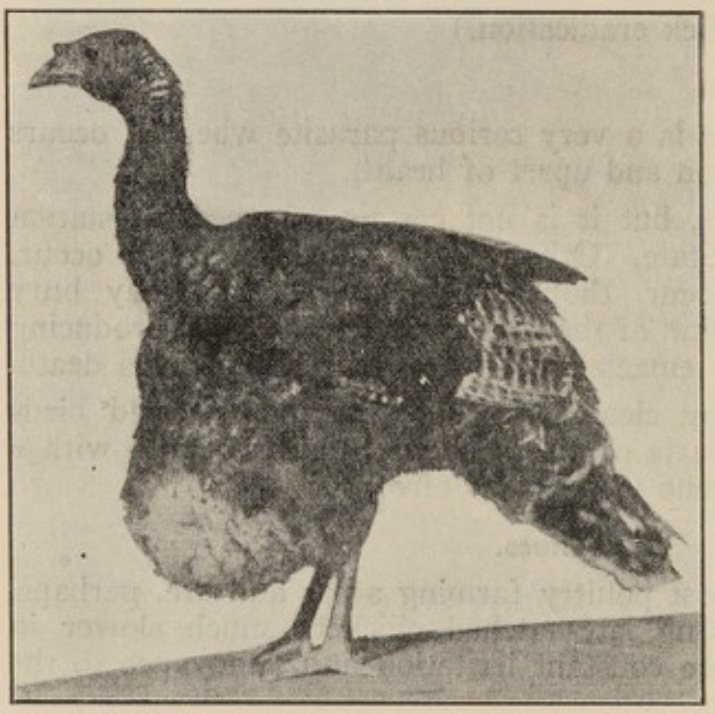
These are common in most poultry farming areas and are, perhaps, one of the causes in making late-hatched chickens much slower in development. They produce constant irritation and annoyance to the birds.

Unfortunately, mosquitoes play a prominent part in the transmission of fowl pox, and may also transmit leucosis, spirochaetosis and possibly other diseases as already mentioned.

TURKEY DISEASES.

Pendulous Crop of Turkeys.

Many turkey breeders in the inland districts are worried by the occurrence of this condition, which may be defined as a dilation of the crop (or ingluvies) with fluid and food materials. The dilation may sometimes reach enormous proportions, and lead to various other abnormalities, as described later. The condition may result in serious economical loss, both as a result of mortality of birds, and emaciation and poor development of those which do not succumb.



An Eight-months-old Female Turkey with a Pendulous Crop of about Five Months' Duration.

[After Hinshaw and Asmundson.

The condition has long been in evidence in this and other countries, but reference to it in this State has not been noticed in literature. A somewhat similar condition known as sour crop or water crop in fowls is of common occurrence, and has been fully described. In fowls however, there is seldom any serious economic loss, and the affected birds, with or without treatment, do not linger, but recover or die within about a week. Such is not the case in pendulous crop of turkeys.

The condition is referred to by various names, such as water bag, dropped crop, hanging crop, sour crop, baggy crop, and so on.

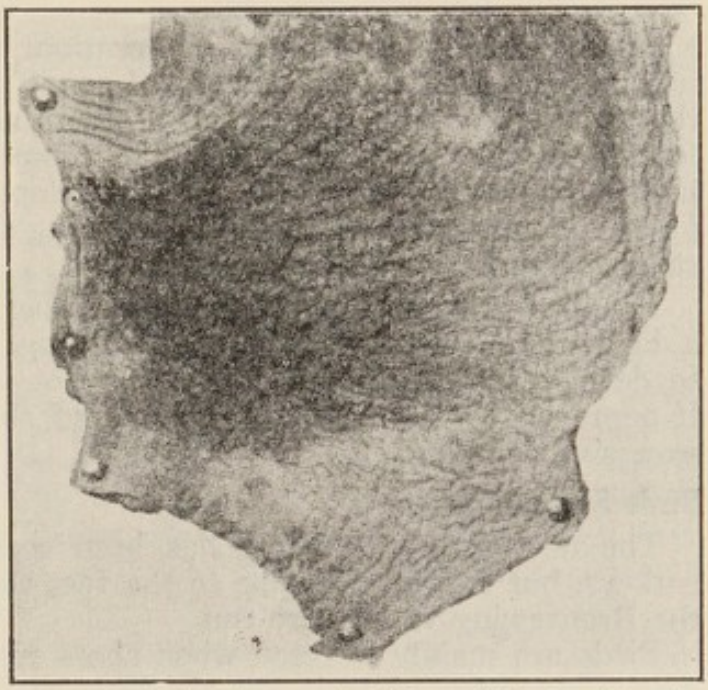
Cause—

The cause of the condition must be considered as twofold; inherited factors may pre-dispose the bird to the condition, but local factors, such as high temperature, cause this inherited tendency to become apparent.

(*a*) *Genetic or Predisposing Cause.*—The disease was investigated in California, and evidence collected to show that the tendency to be affected was inherited; and that birds which had inherited this

tendency would develop the condition when very hot weather occurred. Two investigators reported the results of further work, which showed clearly that the tendency to develop the disease is inherited.

As is well known, the inheritance of a particular characteristic may be "dominant" or "recessive." Thus, an individual may inherit two tendencies, such, for example, as a tendency for tallness, and another for dwarfness. If the factor for tallness is dominant, the animal which inherits these two tendencies will be tall, although it carries, a "recessive" factor for dwarfness. It is a fact of genetics that this recessive factor will not become apparent, unless such an animal is bred with another one which also carries a factor for dwarfness.



Necrotic (dead) Tissue on the Lining of the Crop Due to Distension and Pressure.

In the case of pendulous crop, it has been shown that a tendency to this disease is inherited as a "recessive" factor. This leads to some confusion. For instance, a good type of turkey gobbler which shows no tendency whatever to have a pendulous crop, may be brought into the flock, and this bird may carry a "recessive" (or hidden) factor for the condition. If he is bred with a turkey hen which also shows no tendency to have a pendulous crop, but actually carries a "recessive" (hidden) factor for this condition, the chance probability is that out of every four progeny one will be affected. Authors suspect that the factor may not be a simple, clear "recessive" factor, so that this proportion may vary somewhat from the expected result.

Further, a bird which carries genetic factors for pendulous crop may not develop this disease unless climatic conditions are such as cause this tendency to develop.

(*b*) *Aggravating or Actual Cause.*—It has been suggested by various parties concerned that faulty feeding, particularly in regard to the amount of proteins and roughage (fibre) in the ration, is the cause of the condition. Whilst faulty feeding may have some effect, so far there is no evidence to support this view. On the contrary, the environment of the bird seems to be the critical factor in bringing on the condition. This is particularly noticed in this State.

Odd cases have been seen on the coastal areas, but the condition is seldom severe, and does not seem to cause economic loss. In the dry,

hot, inland climate, it assumes economic importance, and the worst outbreaks usually occur after spells of hot, dry weather, which conditions induce copious drinking.

The amount of shade provided, and the distance which the birds have to come for water, also seem to be important factors. Thus, it has been noted that where the only water available is in a dam, not shaded by trees, the worst outbreaks of pendulous crop occur. This would seem to be due to the fact that on very hot days, the turkeys resent walking across the hot, sunlit area which separates their resting places from their drinking places. Trips for water are only made infrequently, due to the birds' disinclination to travel, and on these occasions, large quantities of water are ingested. This results in temporary dilation of the crop, and the inherited tendency of the crop to remain dilated thus becomes apparent.

In one particular overseas observation, where the temperature did not exceed 74 deg. Fahr., no cases of pendulous crop developed. During the same period, among the full brothers and sisters of these turkeys, located in another area, where the temperature was 20 deg. Fahr. higher, 67.2 per cent. of the birds developed the condition. The two groups of birds were obtained by dividing the progeny of a susceptible strain of turkeys into two even groups.

It will thus be clear from the above, that for the condition to occur, a bird must have inherited the tendency, which tendency is caused to develop into the actual disease by hot, dry conditions, particularly if aggravated by dry winds, and lack of shade, all of which stimulate excessive drinking.

Birds Susceptible—

The disease in this State has been seen only in the Bronzewing turkeys, but this may be due to the fact that few birds of other than the Bronzewing variety are run.

Birds are mainly affected when about nine to twelve weeks of age. This age susceptibility is not absolute, as in this State turkeys which have shown no symptoms until they are one year old have been seen to develop the condition suddenly.

Course of the Disease—

Birds may first show symptoms in a mild form, these symptoms becoming more marked over a period of weeks. The crop, when fully distended, may remain so for years unless death supervenes.

Symptoms—

Usually the first abnormality noticed is slight dilation of the crop, which contains stagnant fluid or semi-fluid matter. If the bird is caught, and held head down, fluid may flow from the mouth, even without any manipulation of the crop. Frequently, if the head is depressed, whilst the bird is still standing on its feet, fluid will run out of the mouth. When symptoms first develop this fluid is more or less odourless, but if the condition has been in evidence for some days, the crop develops a particularly acrid, unpleasant smell.

The abnormality often first appears in a number of birds on the first or second days of a heat wave, and if cool weather follows immediately some of these may recover. Birds which are badly affected suffer from

a progressive enlargement of the crop, which may develop into a pendulous mass, the size of a football, the diameter of which if measured as the bird is standing may sometimes be more than 10 inches, and the crop may reach to within an inch or two of the ground.

Due to the enormous distension, the lower portion of the crop is brushed against various objects, and quickly becomes denuded of feathers. Injuries to the organ are common and may be inflicted by the bird brushing against sticks or stones, etc., or may be self-inflicted usually by the bird clawing at the inflamed crop. When the bird is depressed and slightly injured, other flock mates may attack and destroy it.

Frequently, when the bird is affected with pendulous crop, some other condition, such as pneumonia or mycosis, occurs, and the symptoms of these other complaints are then in evidence.

The affected birds may linger on for months or even years, in which case they remain poor doers, being emaciated, and unfit for table consumption, and certainly unfit from every standpoint for breeding purposes.

Common Complications—

In many cases of the condition, foul stagnant fluid will run out of the crop as the bird lowers its head to eat, and it would appear that frequently the turkeys aspirate some of this highly infectious material into the wind-pipe, and so set up infection of the lungs, and air-sacs. This results in production of cheesy masses in these organs. While these features have been noted on a number of occasions in this State, only very rarely have specimens been submitted for laboratory examination, and it is therefore not certain what causes the production of this inflammatory condition of the air-sacs. Probably both bacterial and fungal parasites are common.

Overseas authors have commented on the fact that moulds may occur in the distended crop, and that the inflammation set up in the lungs, and air-sacs from contamination with the crop fluid, may be due to the growth of these moulds (*Monilia* spp. and *Aspergillus*). In one investigation overseas, it was found that 24 per cent. of birds affected with pendulous crop developed the symptoms in the air-sacs, or lungs, and that the combined effect of the lung or air-sac lesion, with the crop abnormality, resulted in the birds' death.

Post-mortem Appearances—

If the affected turkey is killed within a few days of the development of symptoms, it will be noted that the fluid in the crop has a more or less sour odour. The mucuous membrane of this organ will lack tone, is thinner than normal, but may not show any inflammatory changes.

If a bird has been affected for some weeks or months, the whole of the lining membrane of the crop may be congested (reddened), and the most dependent portion of it may be covered with dark necrotic material or cheesy material. Apart from this, in uncomplicated cases, no abnormality may be seen, except for the fact that the bird is emaciated or poorly developed. Where the lung or air-sacs have been affected, small portions or the entire organ may be filled with cheesy deposits.

Control—

In view of the fact that susceptibility to the disease is inherited, any birds that are affected in any degree whatsoever should be rigidly discarded from future breeding operations. If possible, birds should be selected from flocks in which the condition has never occurred.

The occurrence of even a mild degree of crop dilation should be considered an absolute disqualification for turkeys for show purposes, as it has frequently been noted that some of the very best types of birds may belong to strains which are particularly susceptible to this disease.

Individual Treatment—

Where birds are affected prior to market age it might be particularly important to take palliative measures so that birds may be reared to a marketable age. Particular care should be taken not to use such treatment for any specially treasured specimen which is being kept for breeding purposes, as, in spite of the birds' other excellencies, the predisposition of these birds should exclude their selection.

In mild cases a crop may be emptied out daily by holding the bird upside down, and manipulating the crop. By these means the further development of the condition may be avoided, and the affected bird may not become emaciated.

Another method of treatment is to tie up a portion of the skin over the most prominent portion of the crop and to ligate this skin so firmly that it sloughs off. The removal of this portion of skin increases the tension over the crop, thus tending to hold it in place. Following this treatment, the crop should be emptied out daily for some days.

Surgical removal of the dilated portion of the crop is effective, but quite impracticable, as this must be done, for humane reasons, under general anaesthetic, and is therefore a task for a veterinary surgeon. Removal of a portion of the crop, including the overlying skin, is carried out by crude surgical methods by some farmers, but this practice is to be strongly discouraged on account of its cruelty, and, further, its success is very doubtful in most cases, for frequently septic complications occur which result in the death of the treated bird. Trusses are sometimes adjusted to fit over the crop to prevent dilation, but are not satisfactory.

General Measures to Avoid Occurrence of the Condition—

Ample shade should be provided and water should be made available in this shade; reduction of fluid intake is sometimes resorted to, but is not effective. Following extremely hot weather careful watch should be kept for the first symptoms of dilation so that the crop may be emptied out. If this is done, and cool weather follows, the condition frequently clears up, allowing such birds to be reared for market. Care should also be taken to feed a correct ration to the birds. Full advice can be obtained on this from the Department, but particular care should be taken to provide ample fresh green feed. If this is not available, at least 1 per cent. (approximately 1 pint to 100 lb.) of a good sample of cod liver oil should be incorporated in the morning feed.

Crop Binding, Sour Crop, Distended Crop.

In all birds obstruction of the crop due to ingestion of fibrous substances such as long grass, bark and similar material is likely to occur. Feeding chickens on large quantities of whole corn may cause impaction. Sometimes partial obstruction, and fermentation of fluid contents will cause a condition of "sour crop." If administration of half a teaspoon of baking soda in several tablespoons of water followed by massage of the crop, with the bird held upside down, does not cause emptying of the crop, the latter should be incised with a sharp knife, the crop contents removed, the incision neatly sewed up with white thread or silk. The bird should be fed only light feeds of mash for a day or two.

Ingestion of fibre may also produce inflammation of the crop and gizzard, and should be avoided. Chickens will often playfully tear off bark strips from a tree such as a stringy bark or ti-tree, and a heavy mortality has been encountered from such a cause.

DUCK DISEASES.

The more important diseases affecting ducks in New South Wales are as follows:—

"White Eye" (see also Vitamin A deficiency disease, page 42).

"Cholera" (See Fowl Cholera, page 142).

"Mycosis" (see page 110).

Enteritis of Ducklings and Ducks.

Ducks may also be affected with fowl tick fever, coccidiosis, rickets, and botulism. These diseases are dealt with elsewhere in this book.

White Eye.

As already mentioned, deficiency of green feed or any other source of Vitamin A in the ration will cause disease in ducks (more particularly in young rapidly growing stock) as in the case of other classes of birds.

On numerous occasions, investigations have been carried out in reported outbreaks of "white eye." In each case the trouble has been dietetic, and was remedied by supplying green feed or cod liver oil in the mash. In such outbreaks of "white eye" there is first a mucoid discharge from the eye, which thickens into whitish flakey material, and the duckling dies. In adult laying ducks the complaint may also be seen and sometimes the infraorbital sinuses become puffed out with mucoid material (resembling sinusitis in turkeys).

On post mortem examination sometimes very few changes are seen.

The Bursa cloacae (of Fabricius) is frequently distended with yellowish white material.

Apart from the above symptoms of green feed deficiency which comprise the disease most commonly referred to as "white eye," there appears to be another infectious condition, or group of infectious conditions referred to by the same name.

Apparently the incidence of these conditions is favoured by allowing ducklings access to water before they are fledged, by damp, cold, dirty quarters, by absence of shellgrit, ashes, and sand, and by unsuitable management conditions.

Actually no precise research work has been done in Australia (so far as known) on the cause of the disease (Vitamin A deficiency) known as "White Eye."

Enteritis of Ducklings and Ducks.

Non-specific enteritis due to faulty feeding methods is not uncommon on duck farms, as food is collected from hotels, restaurants, etc.

Feeding almost entirely on bread, and feeding excess salt, have both been noted to bring on serious attacks of enteritis.

In outbreaks of duck cholera seen locally, enteritis has also been a prominent feature.

Apart from the above, there is a specific infectious disease which occurs in the duck farms of the metropolitan area of New South Wales and causes enormous losses from time to time in affected flocks.

General—

History gathered from farms indicated that the disease has occurred intermittently for many years (probably over twenty).

Owners have been loath to contact the Department as such farmers were also pig owners, who feared that action similar to that taken in the case of swine fever might be taken in the case of this disease. In 1938 an active outbreak of the disease was investigated.

Cause*—

The causal micro-organism has been determined as *Salmonella typhimurium*. This is a microscopic germ which may also cause disease in humans.

Infectivity—

It appears that the disease is mainly contracted by the birds eating the causal micro-organism. The latter may be spread on feeding utensils by the attendant's boots, or by the flies which swarm in countless millions in the yards of duck farms, on the particular area where the disease is encountered.

From overseas evidence, and also from the history of the local outbreaks it would seem certain that ducks which "recover" from the disease as ducklings remain carriers and may lay infected eggs. The disease is then transmitted to the next generation in the same way as pullorum disease.

Symptoms—

Ducklings may be affected at any age, but those from one week to about eight weeks seem to suffer most. They appear listless, refuse food, and die within about twelve hours.

Post-mortem Lesions—

In many cases little is seen on post mortem examination. The intestines may be inflamed, or show patchy congestion. The spleen is sometimes enlarged, and the liver may be enlarged and show minute haemorrhages under its capsule.

Pathogenicity—

Losses from the disease may be very severe. In one case on a farm where 10,000 ducklings were being reared, just over 2,000 died in one group of affected yards in less than a fortnight.

Control—

It is not known as yet how reliable the agglutination test is, to detect the adult carrier birds. Control should be the same as for pullorum disease (see page 81).

Usually it is best to dispose of all birds in affected yards, disinfect these premises thoroughly and leave the yards vacant for a season, if possible. The causal germ does not form spores, but will probably remain alive in moist shaded areas for many months.

*Work carried out by D. F. Stewart, B.V.Sc., Dip. Bact., at present (8.4.39) in course of publication.

POISONING OF POULTRY.

A full account of toxicology would constitute a volume on its own, and only a very brief mention will be made of cases of poisonings of considerable economic importance which have actually occurred in recent years in this State.

Of cases of poisoning encountered, the following have been most common agents involved:—

Common Salt, Arsenic, Ants, Mexican Poppy Seed, Phosphorus, Copper Sulphate, Strychnine, Hydrocyanic Acid.

Antidotes are not given, as being impractical for the most part. The main thing is to recognise the condition and to eliminate further poison intake. Antidotes can be ascertained from literature on human first-aid, it one desires to administer same.

Common Salt Poisoning.

This poisoning usually occurs due to feeding of excess amounts in the mash, either accidentally, due to incorrect mixing, or maliciously.

Usually the salt is dissolved in water and mixed with the bran which is then mixed with the rest of the mash. This obviates any bird getting a lump of salt. Where dry mash is used great care must be taken to eliminate all lumps (by sieving).

A great diversity of opinion occurs as to the proportion of salt in the mash that will produce poisoning. Thus one authority (Kaupp) states that 1¼ to 1½ per cent. may produce poisoning whilst others have found that 5 per cent. will produce great thirst but not poisoning. From ½ to 1 per cent. of salt in the total ration will prevent any deficiency of sodium or chlorine. Normally 1¼ per cent. or slightly more (22 oz. to 100 lb.) is fed in the morning mash and no symptoms of excessive supply are noted.

Symptoms—

Symptoms seem to vary greatly, apparently due to whether a large amount (e.g. a lump) is ingested or whether an excess is taken over a period.

In chickens, the bird is depressed, standing around chirping uneasily. It has a great thirst, usually a copious diarrhoea and may be weak or even partly paralysed. Loss of muscular power is sometimes present and may be accompanied by sleepiness which is intermittently punctuated by muscular spasms or twitchings.

Post-mortem—

Sometimes the tissues of the chickens will be oedematous (or sodden with watery fluid), the body pulling apart very easily on manipulation. This oedema may only affect the subcutaneous connective tissue and in other cases is absent. In adults it is seldom noted. There is frequently watery fluid in the abdominal cavity and often in the pericardial sac. The lungs are often oedematous. The proventriculus and intestines are

affected in varying degree and show changes varying from congestion to acute inflammation. Small haemorrhages are sometimes present in the heart muscle and the kidneys may be congested. In adults the mucous membrane of the mouth may be inflamed and covered with a yellowish layer. The linings of the crop, stomach and intestines are violently inflamed and may show small haemorrhages.

Arsenic Poisoning.

Such poisonings may be accidental due to rat poison, spray or dip materials gaining access to the food, or they may be malicious. Cases are very frequent in fowls and turkeys due to accidental access to old tins or packets of arsenic sheep dip. More care should be taken by people handling deadly poisons such as arsenic—the use of which is a routine necessity (e.g. in dipping sheep).

A fatal dose for an adult fowl of "white arsenic" is about four or five grains. Deaths have been recorded due to ingestion of 1½ grains of white arsenic.* Smaller doses (e.g., ½ grain) repeated will have a cumulative affect and may bring about death.

Symptoms—

Symptoms are much the same in all birds, though turkeys affected with this poison present a particularly dismal picture as they mope about, not eating, with drooped tail feathers and humped back.

In fowls and most other birds, appetite is suppressed, the affected bird mopes about near water, drinks freely and has a greenish to white diarrhoea. If the bird lingers for some days the body wastes and there may be marked leg weakness. The comb becomes dark and shrivelled. In cases where small amounts only are eaten over a period, birds may linger for weeks.

Post-mortem Appearances—

A notable feature in most cases is that the gizzard or muscular stomach is markedly affected.

The horny lining of this organ is at times separated from the muscular wall by a gelatinous layer which may at times be 1/3 in. thick. On removal of this layer, the muscular wall is seen to be intensely inflamed.

Post-mortem appearances vary from bird to bird. Any parts of the food canal such as the crop, proventriculus or intestines are likely to be severely inflamed. The heart muscles and liver may be congested, and the muscles of the body may be dark in colour. At times the mucous membrane of the intestines and of the crop may be ulcerated, and sometimes oedematous.

Poisoning by Insects.

Ants—

In the inland districts of the State frequent cases are reported where mortality occurs in the afternoon and evening of certain seasons of the year, particularly after rainy periods.

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*Work carried out at Research Station, Glenfield.

Such deaths have been caused * by ingestion of a greenish black ant identified as belonging to the genera *Crematogaster* and *Chalcoponera*. These ants swarm out in great numbers after rain and are eaten freely by poultry. Deaths only occur over a period of twenty-four to thirty-six hours at most. Similar mortalities (due to *Crematogaster* ants) have have also been recorded in other States of the Commonwealth. † In Queensland two species have been incriminated as causing poultry mortality namely *Chalcoponera metallica* (the green headed ant) and *Iridomyrmex detectus* (the meat ant).

Symptoms.—Affected fowls stay near the water and drink freely. Those fatally affected go into a convulsion and die.

Post Mortem.—No post mortem lesions with the exception of congestion of the crop are usually seen.

It would appear possible that the mortality is due to the ants stinging the crop after ingestion. Many hundreds of ants are usually found in the crop of each dead bird.

Bees—

Ducks, goslings and fowls are not infrequently killed as a result of stinging by bees. This stinging may be a result of the bird annoying a hive or on the other hand, the bird may eat bees which contrive to sting the gullet or crop on the way down.

Poisoning Due to Mexican Poppy.

Mexican Poppy (*Argemone mexicana*) is not an uncommon weed of wheatfields in some districts of New South Wales, and the seed of this plant may sometimes be seen in wheat fed to poultry. Normally, when this wheat is thrown down on the ground the very small poppy seed is not eaten by the fowls. When the soaked wheat feeding system came into popularity, however, all the poppy seed present in the wheat was incorporated in the mixture fed to the fowls, and in such cases symptoms of the harmful effect of the poppy seed were noted.

Symptoms‡—

One or a few doses of the seed may have no effect, but continued ingestion leads to abnormality. Cyanosis (darkening) of the tips of the comb, is sometimes the first symptom. It may take nearly an ounce of the seed to produce such symptoms and perhaps 2 oz. to kill a bird when eaten over a lengthy period. A watery swelling (*oedema*) of the wattles and tissues under the beak is probably the most prominent symptom. Egg-laying is seriously depressed and the birds are listless and obviously sick. Thirst is increased, diarrhoea is present, the bird loses weight, and in acute cases may become paralysed.

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*Accounts of these mortalities are as yet unpublished. Reports from Veterinary Officers and Stock Inspectors, particularly L. Rose, B.V.Sc., have been drawn on.

†Unpublished data received from F. H. S. Roberts, D.Sc., Animal Health Station, Yeerongpilly.

‡Feeding tests with this seed were carried out at Veterinary Research Station, Glenfield, by L. Hart, B.V.Sc., H.D.A., and much of the above is drawn from these unpublished findings.

Swelling of the joints of the legs and wings, swelling of the whole legs, and also the breast is sometimes noted. Jaundice is sometimes present. At times the birds have a peculiar action when walking, lifting the legs high in front without bending at the hock joint.

Post-mortem Lesions—

The heart may be enlarged and the liver is much reduced in size. Congestion, inflammation, or ulceration of the proventriculus, gizzard and small intestines may be noted.

Other Plant Poisonings.*

Other plants which have caused, or are suspected to have caused, posioning in poultry are:—

Acacia decurrens (Sally Wattle).—The seeds of this are under suspicion as causing poisoning in poultry.

Araujia sericifera.—This is a vine with a white bell-shaped flower and fruit resembling a small choko. It grows commonly on the coastal area around Sydney. Seeds from the fruit have produced poisoning in poultry.

Conium maculatum (Hemlock).—This seed has caused poisoning in ducks.

Cotyledon orbiculata (grown locally as a garden plant).—The flowers and leaves are poisonous.

Lathyrus sativa (Indian or Mutta Pea).—When eaten by pigeons, causes loss of power of flight.

Malva parviflora (Marsh Mallow).—Suspected to cause a pinkish discolouration of the egg whites sometimes called "blooded" eggs.

Melia azedarach (White Cedar).—The berries of this tree are under suspicion as causing poisoning of poultry. It has been stated that whilst these berries are poisonous to mammals, they are not poisonous to birds. However, mortalities have come under notice in which these berries are held under suspicion.

Ricinus communis.—Seeds are poisonous.

Robinia pseudo-acacia (Black Locust).—The leaves of the lower branches may cause poisoning if eaten by poultry.

Sida rhombifolia.—This plant has rough seed capsules which, when eaten, have produced enteritis in chickens.

Solanum sp. (Potato and Tomato family).—Various members of this genus have produced poisoning in ducks and chickens.

Phosphorus Poisoning.

Most commonly occurs in the accidental inclusion of rat or rabbit baits with the fowl food.

Symptoms—

Affected birds exhibit malaise, diarrhoea, trembling, and thirst.

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*List kindly supplied by Miss E. A. Mercer, B.Sc.Agr., after survey of literature.

Post-mortem—

On opening the crop, stomach and intestines, the characteristic smell of phosphorus will be noted. If opened in the dark there will be a luminosity.

Acute inflammation of the stomach and intestines is usually noted.

Copper Sulphate Poisoning.

Sometimes occurs when farmers endeavour to eradicate worms in fowls by the use of this drug.

If enough copper sulphate is administered in the food or drinking water to deal *effectively* with the worm infestation, cases of poisoning of the fowls are a probability.

Symptoms—

Thirst, malaise, weakness and purging are in evidence, followed by convulsions and death.

Post-mortem—

Gastro-enteritis as shown by acute inflammation of the stomach and intestines is the most marked symptom.

Strychnine Poisoning.

This usually occurs due to accidental disposal of rabbit bait material, or may be malicious. Fowls are fairly resistant, and it may take two or three grains of a drug such as strychnine sulphate to cause the death of an adult bird.

Symptoms are rapid breathing, stiffness in the leg movements, followed by unsteadiness. The bird then suffers from spasmodic convulsions, which cause stiffening out of the legs. In such cases a convulsion may end in death.

In cases which recover diarrhoea may be in evidence.

Hydrocyanic (Prussic) Acid Poisoning.

Hydrocyanic (prussic) acid poisoning, also called cyanide poisoning, may be the result of malicious poisoning, or, in areas where blue couch grass occurs, it may cause death if it is eaten freely by fowls. Blue couch can only be differentiated from ordinary couch grass by minute botanical features. If large quantities of blue couch be eaten in a small time, *e.g.*, when fowls are first let out, enough prussic acid may be liberated to cause their death. Suspicious cases have come under notice (proof is lacking).

Symptoms—

Fowls may gasp, flutter about and die. Few, if any changes, are observed post mortem.

DISEASES OF THE OVARY AND OVIDUCT.

Fowls have been so developed that their natural egg production has been increased enormously. All organs suffer from time to time with specific abnormalities, but due to the development of the ovarian function, diseases in the ovary and oviduct appear to be more common and of more economic importance. Of diseases dealt with separately, pullorum disease, vent gleet, cancerous growths and leucotic (see leucosis) tumors affect the oviduct. In pullorum disease, as already mentioned, the ovary of carrier birds has many misshapen and discoloured ova. On rare occasions pullorum infection may also cause inflammation of the oviduct with haemorrhagic discharges. Such a symptom of pullorum infection is not common, however.

Egg Bound.

Egg-bound, or obstruction of the oviduct, is a local condition, and is related to the formation of an abnormally large egg, or the passage of a normal egg through an abnormally small oviduct. Sometimes the condition is due to a failure of the mucous secreting glands of the oviduct to function. It is seen particularly when pullets of the light breeds come on to lay. The case is usually noted by the fact that the bird is restless, attempts to lay, strains continually and may evert the oviduct. Should this happen the other inhabitants of the yard immediately attack the fowl, which dies as the result of evisceration and haemorrhage. In birds which are worth treating, the egg may be manipulated within sight, pierced, the contents evacuated and the egg then broken and removed, after which the cloaca should be douched thoroughly with saline solution (a teaspoon of common salt to the pint).

Tumors of the oviduct and also various inflammatory conditions may bring about obstruction as above.

Nervous affections as in the case of Neuro lymphomatosis gallinarum, where the nerves supporting the oviduct are affected, may bring about this condition. In small birds of excitable temperament such as bantams and cage birds, retention of the egg is not uncommon. Placing the bird in a room or compartment at 105 deg. Fahr. appears to facilitate normal peristaltic contractions and laying of the egg.

Yolk Bound.

In this condition egg-yolks are liberated from the ovary and gain access to the abdominal cavity, where they may form a cheesy yellowish mass, or may break down to form a turbid yolky fluid which covers the peritoneum and abdominal organs, causing inflammation.

The yolks may escape due to the faulty function of the infundibulum, or funnel-shaped end of the oviduct. Due to faulty structure or function of this organ (*e.g.*, due to inflammation) the yolks, on being liberated from the ovary do not pass into the oviduct, but fall into the abdominal cavity. On other occasions the condition arises due to yolks bursting through the oviduct wall.

An egg partly or wholly formed may be retained in the oviduct due to poor nervous tone of the organ, overfatness of the fowl, or some other obstruction (*e.g.*, such as a tumor), and further yolks may accumulate behind this retained egg. The accumulation results in the formation of a weighty mass which dilates the oviduct and causes pressure. This pressure obstructs the blood supply of that particular part of the oviduct wall, resulting in necrosis, sloughing and liberation of the yolk mass into the abdominal cavity.

The rupture of the oviduct may at times be brought about by physical violence such as an attack by a dog.

It has been noticed that this condition is very common in egg-laying competitions, where strains of high layers are confined with relatively little exercise. The presence of yolk binding is frequently indicated by the bird adopting a "penguin" attitude with the vent and tail resting on the ground.

It would also appear that the tendency towards the condition may be hereditary. Providing ample exercise, keeping fowls as quiet as possible, avoiding disturbance whilst on the nest, breeding from fowls of good body size only, avoiding over-fatness, and avoiding "stimulating" rations (*e.g.*, spices) will all tend to minimise the incidence of cases of "yolk binding."

Infection of the Oviduct.

Apart from the specific infection which causes "floating yolks" (see later) there may be quite a variety of infectious inflammatory conditions of the oviduct which normally affect odd fowls only. Infection may be spread along the oviduct from a case of vent gleet. A condition known as "whites" in which there is a creamy white purulent material in the oviduct is not uncommon. This has in some cases been due to infection of the organ by *Salmonella pullorum* (the germ causing pullorum disease of chickens).

In some cases there may be cheesy false membranes formed along the lining membrane of the oviduct and in other cases the inflammation may be haemorrhagic.

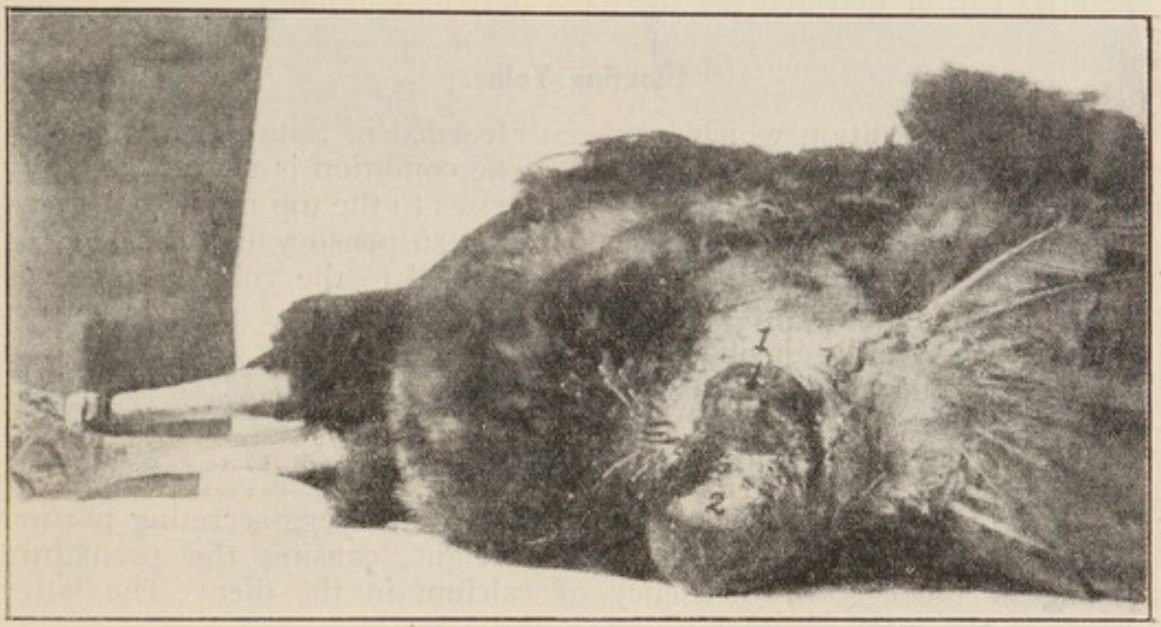
Depending upon which particular part of the oviduct is inflamed there will be varying abnormalities in the eggs which are produced. Thus the eggs may be putrid and offensive, they may have "blooded" yolks, due to inflammation and haemorrhage in the ovary, or blood spots, shell-less eggs, or blood-smeared shells, when the inflammation is in the different portions of the oviduct.

Inflammatory conditions of the oviduct may lead to eversion of the cloaca or to prolapse of the whole oviduct.

Affected birds are best destroyed. Under no circumstances should cases be left in a yard where a rooster is present, as the male may spread the infection from bird to bird.

Prolapse of the Cloaca.

This condition may be the result of inflammation of the oviduct, vent gleet, straining to lay an oversize egg, a severe fright during egg-laying, over-stimulating food, or the presence of abdominal tumors. Usually birds affected are best destroyed. A valuable breeding bird may be treated, but this is unwise, as there may be an inherited predisposition to the condition.



Eversion of the Cloaca and Oviduct.

(1) Eversion of the vaginal portion of the oviduct. (2) Eversion of the cloaca.

[After Kaupp.

Treatment may be carried out by washing with a saturated sugar solution for ten minutes to reduce the congestion, then washing with normal saline (one teaspoon of salt in one pint of water) and returning the everted portion, which is held in place by a wet (cold water) pad of cotton wool bandaged over the cloaca for one to two hours.

EGG CONDITIONS.

Abnormalities in eggs are, generally speaking, of trifling economic importance to the farmer, but on odd occasions large numbers of abnormal eggs may occur, and some comment as to the cause of some of the more common abnormalities may be of interest. These abnormalities can be detected for the most part by candling.

Floating Yolks.

This is a condition which has been recorded in South Australia, but is not known to occur in this State. The condition is characterised by an excessive mobility of the yolk which rises to the top no matter which way one turns the egg. The chalaza or suspensory ligament of the yolk breaks, portion of it remaining attached to the yolk. A germ is found located in the affected chalaza, and feeding of such infectious material to healthy laying hens causes them to lag eggs similarly affected within a few days.

Soft Shelled and Thin Shelled Eggs.

This condition may be due to a disease of the egg-secreting portion of the oviduct, over-fatness, severe fright, causing the premature laying of the egg, or deficiency of calcium in the diet. The latter cause is the most common.

The tendency to lay thin and soft-shelled eggs may be inherited.

Adequate calcium is supplied in this State by making shell and oyster grit available to the birds. Care should be taken to avoid placing out a large bulk of grit, which is depended on to supply the birds for many months. In this case birds may pick over all suitable grit, leaving the hard blue shells and large pieces. There may be a large bulk of such waste material present, while the birds are suffering from an acute grit (calcium) deficiency. Some farmers supply crushed calcium carbonate or crushed oyster flour at the rate of ½ to 1 per cent. in the morning mash.

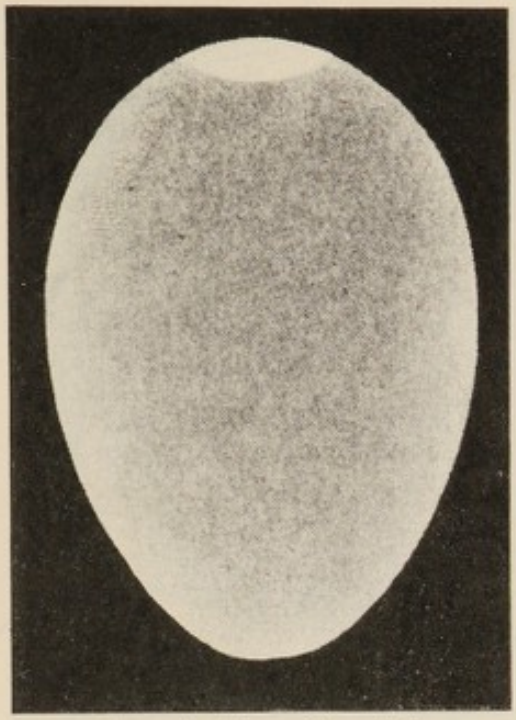
Where ample suitable shell and oyster grit is supplied this is not necessary.

Yolkless Eggs and Dwarf Eggs.

Such "eggs" are due to some piece of material such as a piece of hardened yolk, hardened albumen, coagulated blood, or some foreign body carried up by antiperistalsis, stimulating the albumen secreting portion of the oviduct to function. Thus an "egg white" and shell may be formed round this foreign body to produce a dwarf and yolkless egg.

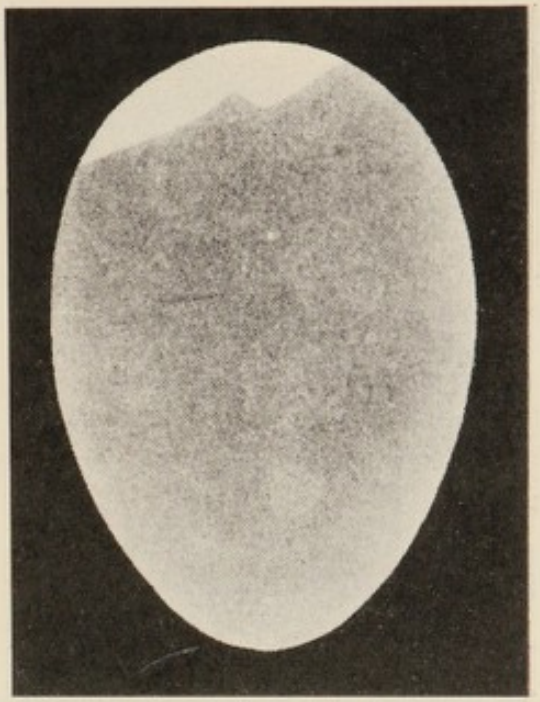
Double Yolked Eggs.

This results from the liberation of two yolks from the ovary which are carried down the oviduct together and are enclosed in the one shell.



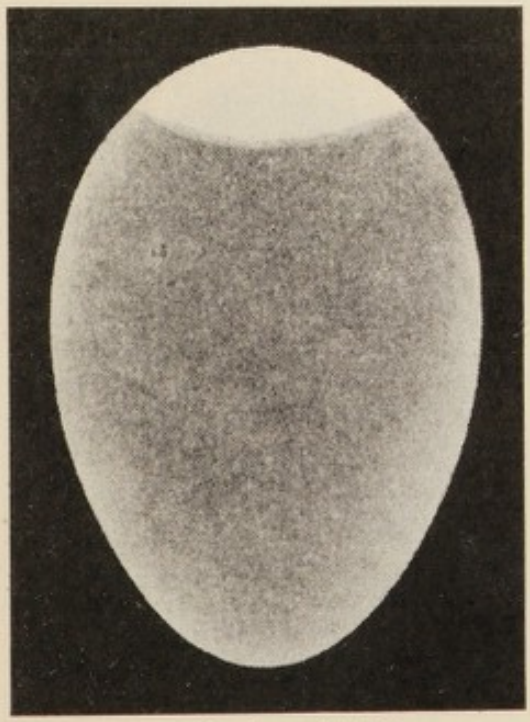
Standard Egg, showing a Normal Air Cell when Laid.

[After Platt.



Condition of Air Cell due to "Watery White" when tested at Farm.

[After Platt.



Egg with a Large Air Cell.

This is a condition which under ordinary marketing conditions is common to eggs over a week old.

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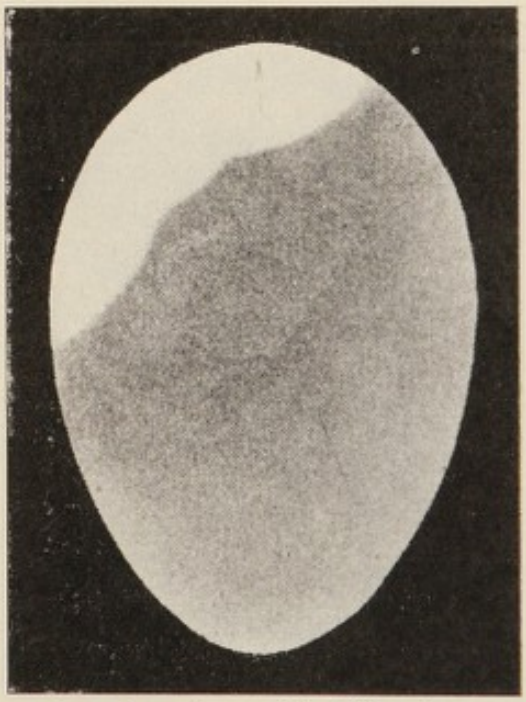

Same Egg as in preceding Photo. when Tested at Packing Floor.

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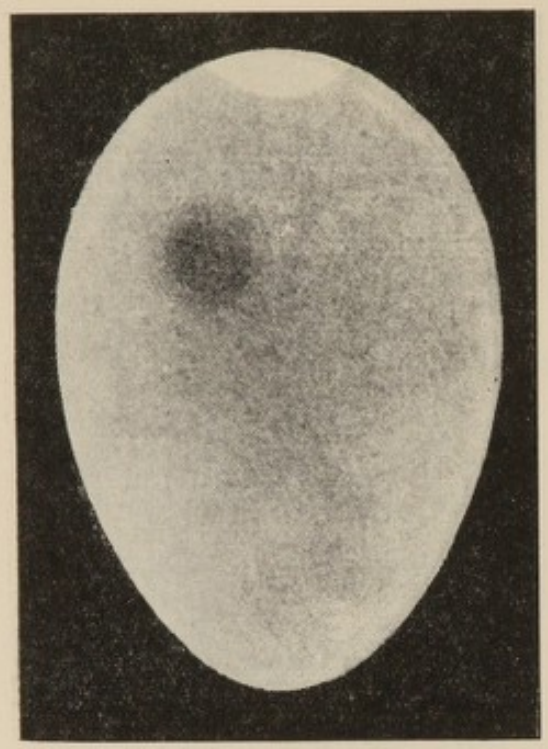
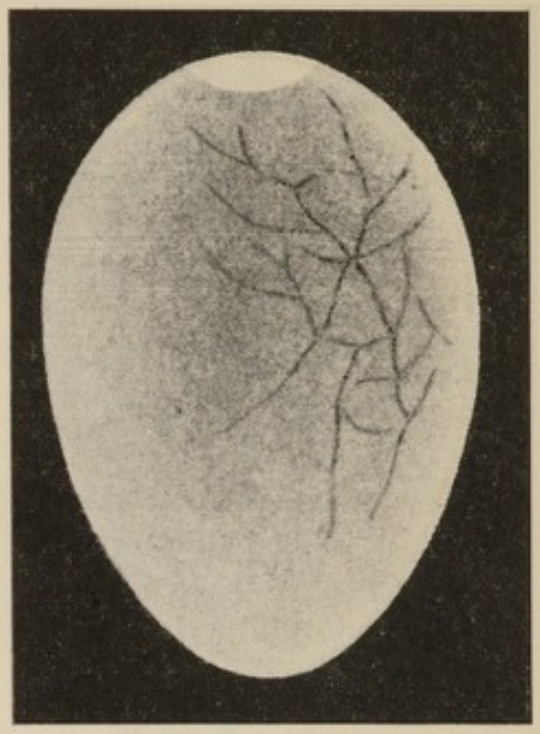
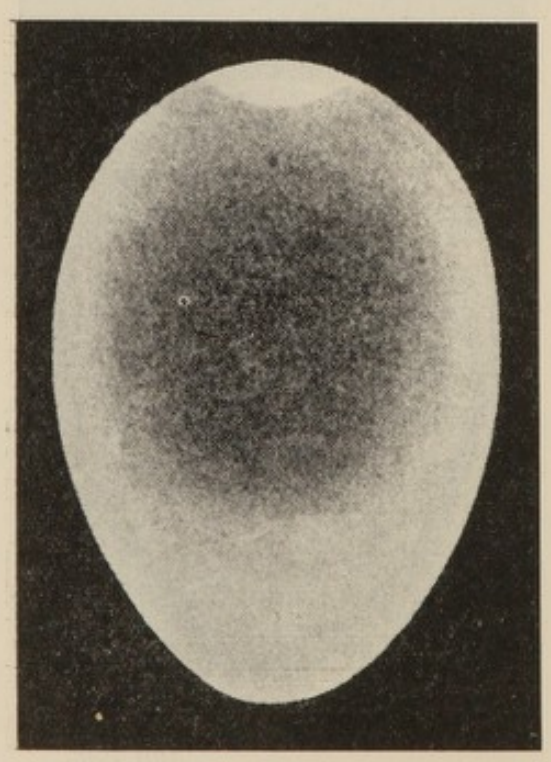


Egg, showing Blood Spot.
[After Platt.



Egg, showing Spider Cracks.
Due to weak shells and rough handling. Difficult to detect until the egg is candled.
[After Platt.



Egg, showing Cloudy Yolk.
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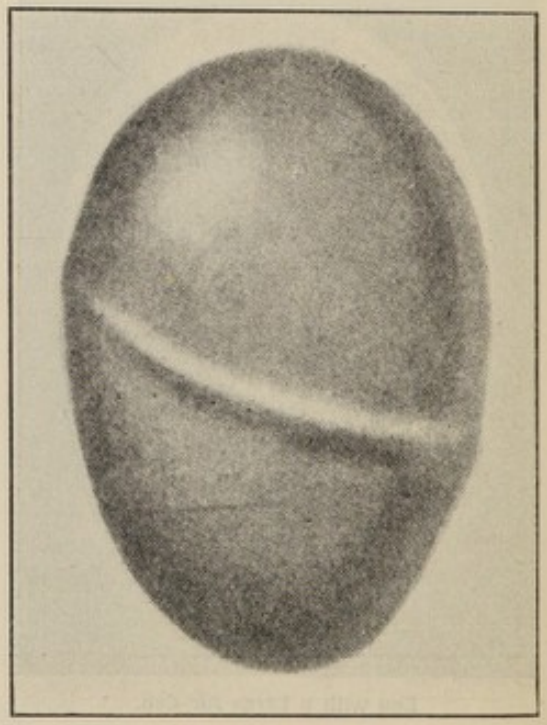


Misshapen Egg.
Sometimes caused by over-feeding.
[After Platt.

Double Shelled Eggs.

Such eggs occur when the egg which is ready to be laid is carried back by an antiperistaltic contraction to the albumen or shell-secreting portions of the oviduct a second time. The presence of the egg may stimulate the product of further shell membranes and a further shell.

Blood Spots, Blood Streaks, etc.

The location of a blood spot in an egg will depend on the site at where the haemorrhage occurred in the ovary or oviduct. Thus, if the ovary is congested or inflamed, and an oozing of blood occurs when the yolk is liberated this blood will occur as a blood spot on the surface of the yolk. Sometimes the whole yolk may be "blooded." Inflammation and haemorrhage in the albumen secretion portion is comparatively rare. When it does occur blood may be diffused through the albumen or "white" of the egg.

Haemorrhage may occur in the oviduct after formation of the egg, resulting in blood-smeared eggs. This is seen frequently in young laying pullets.

Egg Inclusions.

Not infrequently foreign bodies, such as a round worm or piece of manure, may be carried up into the oviduct by antiperistaltic waves and be included in the egg white. Sometimes a piece of grey, stone-like material due to abnormal function of the shell glands may be produced, and included in the egg, and at other times portion of shell membranes may be included.

Olive Yolks.

This is a condition which is noted from time to time in this State. The cause or causes are not certain, but it appears probable that the eating of citrus blossoms may cause the condition.

Careful experimental work has shown that pigments fed in the ration may influence the colour of the egg-yolk, and that this influence is first exercised about three to five days after feeding of the pigment begins.

The occurrence of olive yolks has never caused widespread trouble, but individual farmers have had quite a large number of eggs rejected (after candling) on intermittent occasions.

Watery Whites.

This is also referred to as "fractured or broken air cell," air bubbles, tremulous air cells, and by various other names.

The condition is detected, on candling, by the presence of a tremulous and irregular air space, or by the presence of loose bubbles in the white of the egg. Information as to all factors involved in the causation of this disease is incomplete, though comprehensive observations have been made, particularly in South Australia, where the condition is of particular importance due to the fact that the large proportion of eggs produced are for export.

The following facts are known. Newly-laid eggs may be affected with the condition. Pullet eggs are not so frequently affected as hen eggs.

Travelling long distances increases the proportion of eggs showing this abnormality, and the increased incidence due to travelling is more marked in the case of hen eggs than in pullet eggs. When padding or rubber mattresses are used to pack the cases of eggs in the vehicle the increase in percentage of eggs affected due to travelling is not nearly so great.

The condition occurs most frequently at the end of a period of heavy laying, and in South Australia the incidence increases from January to March, when the peak is reached.

Storing eggs for long periods will increase the number affected. The conditions can frequently be produced by shaking an egg very vigorously. There is some evidence that exposure of the egg to a warm temperature (*e.g.*, as where laying boxes are on the sunny side of the house) will favour development of the abnormality.

Packing eggs with the air cell downwards during transport has been blamed, and the feeding of excess green feed has also been cited as a cause, but there is little evidence to support the latter contention.

Control—

Should aim at eliminating all the causative factors as outlined above.

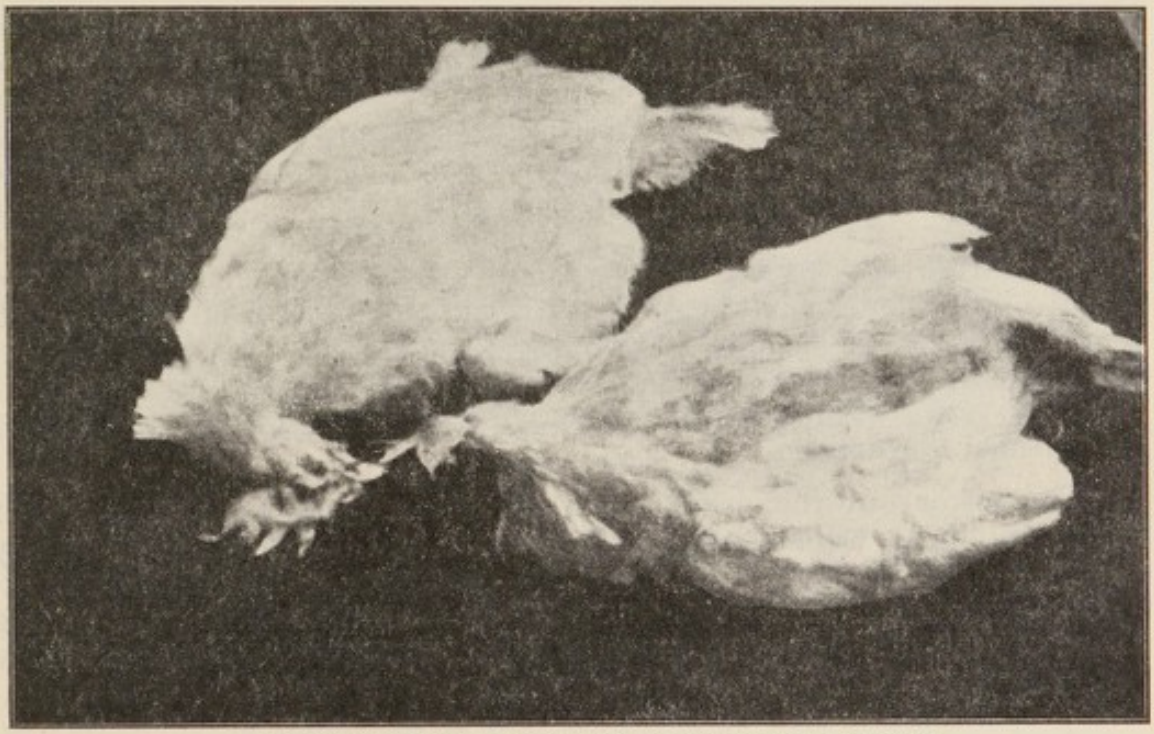
Thus the nests should be in a cool situation, and all broodies should be promptly eliminated. Ample shell and oyster grit should always be supplied. Care should be taken when collecting eggs not to bump, shake or agitate the eggs. Care in washing, packing and transport should be maintained. Wood wool should be used below the bottom and above the top "flat."

Eggs, if stored at all (*e.g.*, for two days) should be kept in a cool site. An underground egg room is easy to construct and is always cool. The construction of such rooms is of particular importance in the hot inland areas (*e.g.*, Wagga). Do not store the eggs in a draught. Maintain the condition of the stock by correct feeding and management.

OTHER DISEASE CONDITIONS.

Newcastle Disease and Fowl Plague.

These are two closely related diseases which are caused by viruses (micro-organisms too small to be seen under the microscope). Newcastle disease occurred in Victoria some years ago, but was stamped out by vigorous slaughter and quarantine. Neither disease, however, has ever been recorded in New South Wales. In both diseases mortality is usually extremely heavy (more than 90 per cent. in most cases). Affected birds may gasp and show copious mucous discharges from the mouth and nose. The diseases are very highly infectious and produce devastating mortalities. Further details are not give, as if there is the slightest doubt the matter should at once be reported to the Chief Veterinary Surgeon.



Two White Leghorns affected with Botulism or "Limberneck."

The left one is prostrate in a state of coma. The right one is prostrate, but able to hold the head up, resting the beak on the ground. The feathers pull out easily, which is a diagnostic symptom of the disease.

[After Bayon.

Botulism or Limber Neck.

This condition is not common in commercial poultry flocks, but has been recorded more frequently from wild birds living under natural conditions, particularly swamp-living birds. It may affect all classes of poultry such as fowls, ducks, turkeys and geese.

Cause—

The cause of the condition is the toxin produced by the specific spore-bearing anaerobes (germs) which maintain a saprophytic existence on rotting animal or vegetable matter. The organisms are divided

into *Clostridium botulinus* types A, B, and C, and *Clostridium parabotulinus*. There is some confusion between the terms botulism and ptomaine poisoning. In 1896, Van Ermengen demonstrated the true cause of botulism. Before this, botulism and similar conditions, together with infections of human food by micro-organisms known as the paratyphoid group, which caused gastro-intestinal upsets were referred to collectively as ptomaine poisoning. Botulism is commonly associated with depraved appetites in animals leading them to eat such material as rotting carcases, old bones, etc. In the case of poultry it is always referable to either swamp birds feeding on mud flats and locations where rotting organic matter provides a habitat for the growth of the causal toxin-producing bacterium, or to the feeding of mouldy foodstuffs. Maggots from rotting carcases may convey the organism and the toxin from the ingestion of such maggots may lead to symptoms developing.

Symptoms—

These are paralysis and weakness. This affection is apparently due to affection of the sciatic nerve, which supplies the leg. The wings are carried out from the body in a depressed position, but later become paralysed and trail on the ground. Depression and a tendency to sleepiness is a notable feature. Relaxation of muscular tone throughout the body is often marked, and actual flaccid paralysis of the muscles is sometimes present. The neck may lie in a flaccid condition on the ground, the head rolling from side to side. Mucous secretions and saliva are often much increased. Diarrhoea of a watery, greenish nature is a usual symptom. Symptoms usually occur about twenty-four hours after the ingestion of toxic material.

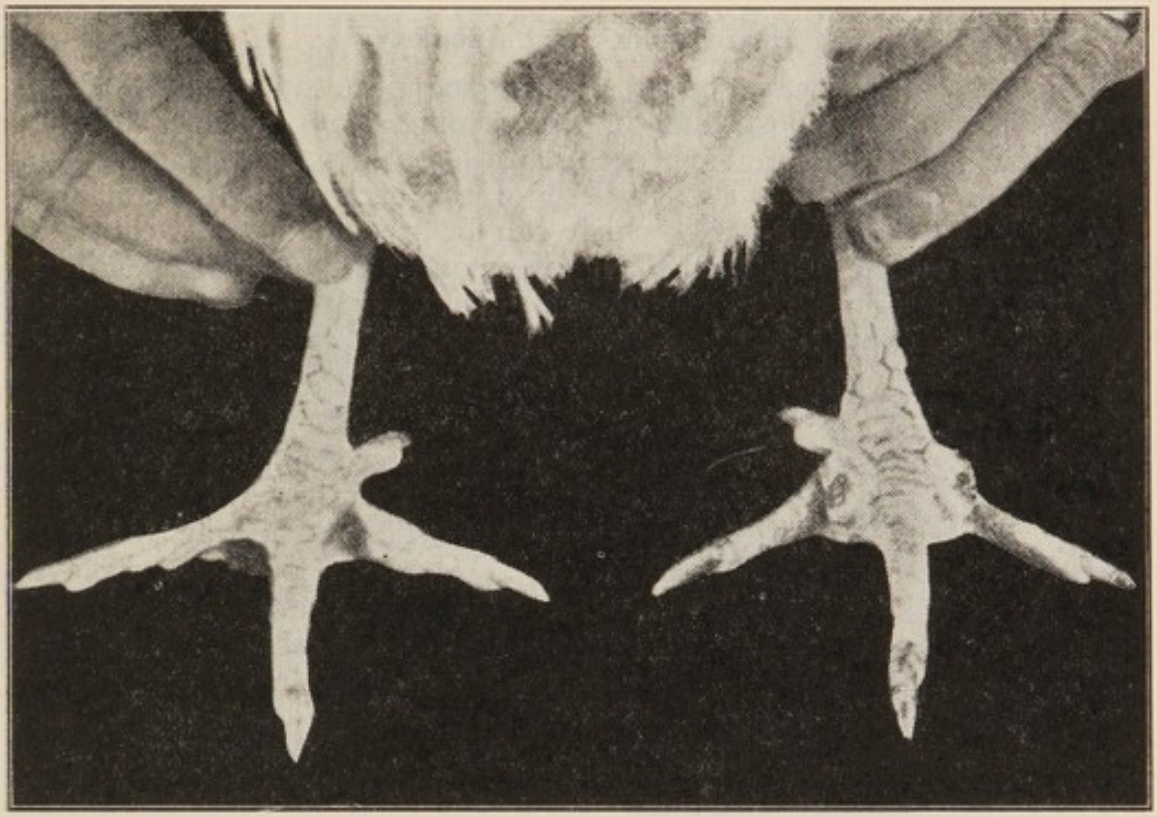
Differential Diagnosis—

The history of the disease (*i.e.*, widespread sudden occurrence of the symptoms) would point to this disease and no other. The history of having fed on likely food materials, tainted or decomposing is significant. Isolated cases when one bird dies from feeding on a rotting carcase may thoroughly confuse the diagnostician, who would probably classify the condition as of the leucosis group of diseases. For laboratory confirmation sick birds whilst still alive should be forwarded to the laboratory, together with recently dead birds, and some of the suspected food material. Gut contents should also be dried and forwarded if there is difficulty in sending the other specimens. Anti-toxin tests are the critical means of laboratory diagnosis, the mixed toxin (suspected) anti-toxin being administered to test animals. Guinea pigs are suitable as test animals.

Bumble Foot.

This is a condition affecting fowls, more particularly of the heavy breeds and is well known to all poultry men. The ball of the foot or surrounding structures become swollen and the bird is quite lame. Swelling of the foot may be due to articular gout or even to tuberculosis (not seen in this State). The true lesion of bumble foot comprises

necrosis (death) of some of the deeper structures of the foot, which is brought about by injury, often associated with infection. The necrotic material as usual in fowls, becomes "cheesy," and inflammation extends; in some cases the inflammatory exudates are forced along the tendons and ligaments of the legs. Surgical treatment by opening at the lowest point, making incision in the direction of the toes, and scraping away



Bumble Foot.
[*After Bayon.*

the necrotic material, followed by suitable wound treatment is justifiable in valuable birds. Bathing with hot dettol solution, followed by packing the cavity with a wad of cotton wool saturated in tincture of metaphen, held in place by a bandage is suitable treatment. It should be realised that in the case of gouty swelling (not true bumble foot) such treatment is valueless. By way of prevention, perches should not be too high from the ground, and birds should be prevented from perching in trees. Stony ground is often a predisposing cause of the condition. The tree-perching results in birds flying down and landing with considerable concussion, perhaps on some stony projection.

Psittacosis.

This is a disease which affects mainly birds of the parrot family, such as parrots, budgerigars, rosellas, etc., but fowls, geese, thrushes, pheasants and other birds may also be affected. So far in this State the disease has not been noted to occur in fowls run under commercial conditions. The importance of the disease is that when it does occur in aviary birds, it is a highly infectious and fatal disease for humans. Symptoms in affected birds are often indefinite. The following may

be evidenced: diarrhoea, nasal catarrh and malaise. On post-mortem examination the liver is usually enlarged and discoloured, being paler than normally. Its surface may be covered with white dots and sometimes there are large discoloured patches which are encircled by reddened areas. The spleen is nearly always enlarged. In doubtful cases in parrots, aviary birds, or domestic fowls kept in contact with such, veterinary advice should at once be sought.

Heat Stroke, Heat Apoplexy.

When severe heat waves occur, as happened in the summer of 1938-39, many birds may be lost due to heat stroke, heavy breeds suffering more than light. Where such events are likely it is a great advantage to have the bottom foot (12 inches) of the back wall of each house closed with a hinged board, which can be raised in hot weather to allow a free draught of air through the house. The usual opening of 4 to 9 inches below the roof at the back of the house and an open front should always be present.

A.S.C. Rhode Island Red Hen with an Abdominal Cyst.

The upright attitude is a diagnostic symptom of an abdominal tumor, ruptured oviduct with eggs in the abdominal cavity, or, of an injured spine.

[*After Kaupp.*

The factor of most importance is to have cool water available to the birds, as in a heat wave the fowls will not walk far for water, and are loath to walk over a sunlit area for it. Water troughs, if not usually situated in the houses, should be placed there during a heat wave, and the water should be changed frequently.

Affected birds gasp, become weak, depressed, dizzy, (apparently) are unable to stand, and die in a convulsion. On post-mortem examination such birds may show haemorrhages in the brain or within the cranial cavity.

Birds showing premonitory symptoms may be immersed in water. To save them, birds must be treated early in the course of the condition.

Obesity or Excess Fatness.

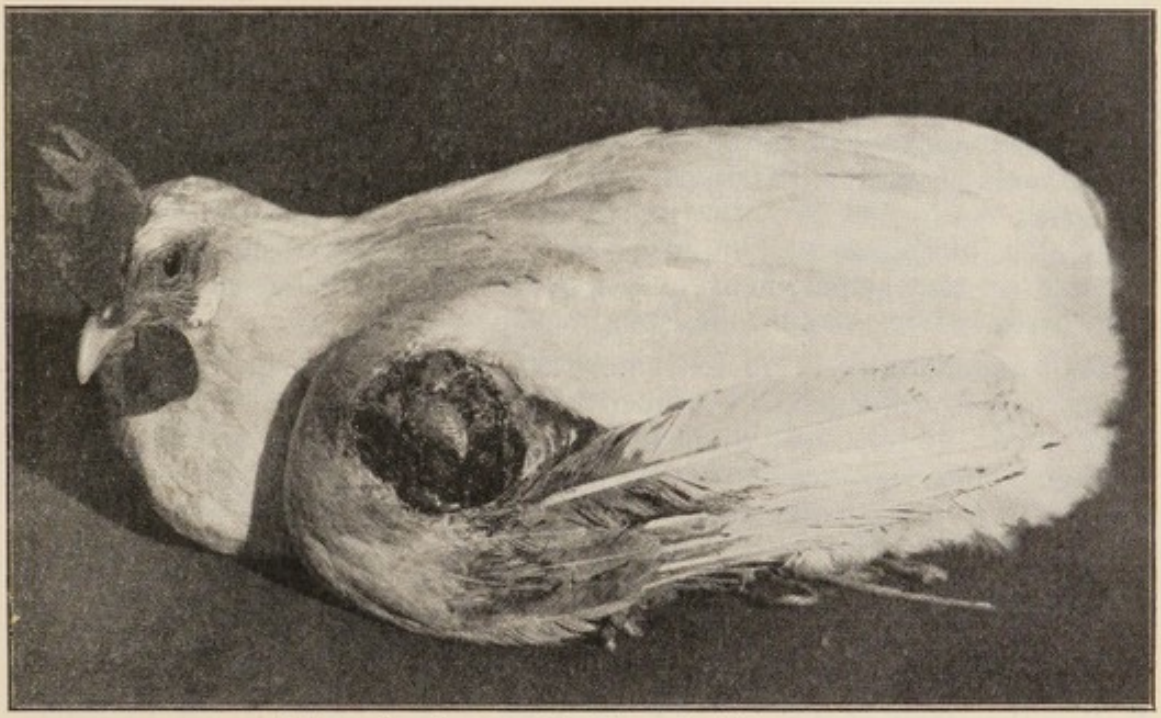
This is due to overfeeding, faulty feeding, or lack of exercise. The condition diminishes egg-laying, and may predispose to abnormalities such as visceral gout and prolapse of the cloaca.

To avoid this condition a standard ration with a nutritive ratio of about 1 : 4.5 (see "Poultry Farming in New South Wales") should be provided and, in addition, exercise provided either by open range, or by scratching litter, in which the grain ration is scattered.

Neoplastic (Cancerous) Condition of Birds.

All birds, and particularly fowls, are commonly affected with various types of cancerous growths.

Thus carcinomata, sarcomata, epitheliomata and many other tumors are commonly observed. In some cases it is difficult to differentiate a neoplastic tumor from a leucotic tumor (see leucosis).



A Case of a Cancerous Growth on the Wing.

Details concerning cancer are not given, as the only interest to the farmer is that birds affected or suspected to be affected with such condition should be killed promptly.

HATCHERY DISEASES.*

With the advent of mammoth incubators, precise methods of incubation have become possible. The mammoth incubator has facilitated mass production of poultry, which in turn has assisted and expedited the spread of various epidemic diseases. In no case are the maintenance of health and the control of disease more closely inter-related than in mass incubation.

Hatchability is a term which is used in a loose way to indicate the percentage of eggs which hatch. It is, however, now usually taken to mean the percentage of fertile eggs which hatch out, *i.e.*, the percentage infertility is not included in the percentage hatchability. In a consideration of the possible influence of disease on hatchability (its interest to the hatcheryman) all the factors of incubator management must necessarily be touched upon, as in any investigation of disease, factors of incorrect management must first be eliminated to arrive at a differential diagnosis. It may be that incorrect management and infectious disease are both concerned in some mortalities. For information concerning incubator management, inquiries should be directed to the Poultry Expert of the Department of Agriculture, and in disease problems veterinary advice should be sought.

Poor hatchability is an extremely complex problem as the hatching of an egg is influenced by numerous factors, many of which are still imperfectly understood, while there may be others not realised as yet. The economic significance of the problem of poor hatchability may be visualised by noting the fact that the value of the eggs which failed to hatch out in the United of America is stated to be about £3,500,000.

Mismanagement a Major Cause—

It has been noted repeatedly that where flocks have suffered from severe disease epidemics, hatchability has been affected materially. Before the effect of disease on hatchability is considered at all, however, the possible major factors of mismanagement must first be taken into account. These include: Temperature, humidity, incubator atmosphere, respiratory and thermal quotients of the chick, storage of the eggs prior to incubation, the turning of the eggs during hatching, egg weight, conformation and shell characters, and the position of the egg during hatching. Then the effect of disease on hatchability could be considered from such aspects as dietary deficiency of the hens, reproductive organ infection, and the effect of disease epidemics in the parent stock.

Incubator Temperatures—

In the old type of still-air cabinet machine, there was a temperature gradient from the top to the bottom of the egg of up to 2 deg. Fahr., which necessitated the temperature being run at 102 deg. Fahr. With an agitated air or forced draught mammoth incubator, where the atmosphere throughout the machine is uniform in temperature, it has been

*For full information readers should consult W. Landauer (1937), Storrs Agric. Exp. Stat. Bull. 216.

shown by many authorities that 100 deg. Fahr. is the ideal temperature, where humidity and other factors are optimum. In such machines it has been shown that at 102 deg. chickens are not as large, fluffy or lively, and many abnormalities such as crooked toes, sprawling legs and crooked necks occur. Mortality amongst such chickens might easily be confused with that produced by infection such as pullorum disease. It has been shown that periodic cooling in agitated air machines does not improve the hatch when modern equipment is used. The ideal temperature is not a constant, but varies slightly with the humidity, and this fact confuses many farmers.

From the above it will be seen that an incorrect or fluctuating temperature may account for a poor hatch, quite apart from disease.

Wide Humidity Variation May Be Detrimental—

When the temperature is kept at 100 deg. Fahr. the optimum humidity has been shown (Barott, 1937) to be about 61 per cent. Variations from 50 to 70 per cent. do not cause material reduction in hatchability, but outside these limits variations may have very detrimental effects. If the humidity is increased, the temperature should be slightly decreased. In recording humidity, hatcherymen are often at a loss because the recording instrument does not give actual percentages. A humidity of 60 per cent. means that there is actually present 60 per cent of the amount of water vapour which the air is capable of containing at that temperature. The most accurate means of measuring humidity is by means of wet and dry bulb thermometers. Providing the air velocity in the machine is 9 feet per second or more, the difference in the readings of the two thermometers indicates accurately the amount of moisture in the air, this amount being read off a humidity scale. Some authorities (*e.g.*, Palmer and Dykes, 1927), claim that humidity should be lowered slightly from the fourteenth to the nineteenth day for ideal hatches.

If the humidity is not kept at a reasonably correct level, it will be apparent that losses will occur quite apart from disease.

Incubator Atmosphere—

It has been shown that if the carbon dioxide content of the incubator air exceeds 0.5 per cent., or if the oxygen percentage decreases below 15 per cent. (normal, 21 per cent.) hatchability is seriously reduced. Thus, in one experiment overseas, when the carbon dioxide was allowed to accumulate in the incubator to 5.5 per cent., hatchability was 45 per cent.; when the carbon dioxide was allowed to accumulate to 10 per cent. hatchability was reduced to 16 per cent. When the carbon dioxide content of the air was kept from the first day throughout incubation at 4 per cent., only 20 per cent. of fertile eggs hatched. One authority found that if 6 per cent. of carbon dioxide was in the incubator throughout, no chicken embryo was able to exist longer than two weeks. Similar results have been obtained by many others.

These findings are in striking contrast to early impressions of various poultry authorities who about the year 1900, held that pullorum disease, then known as bacillary white diarrhoea, was the outcome of insufficient quantities of carbon dioxide being present in the incubator air. There

was in any case, no grounds for this contention, as the air under the broody hen seldom contains up to 0.5 per cent. carbon dioxide gas, until the last day of incubation.

The hatcheryman should have no cause for worry on any of these points, so long as the instructions issued by the maker of the machine are adhered to closely. If in doubt communication should be made with the Poultry Expert.

Respiratory and Thermal Quotients—

These are indications of the normality or otherwise of the chicken's digestion and assimilation of the yolk substance. They cannot be dealt with here.

Storage of Eggs Prior to Incubation—

Authorities (*e.g.*, E. M. Funk, 1934) concur in indicating that hatchability may be decreased and chick abnormalities increased by the keeping of eggs. Thus in one experiment—

Where eggs were kept 1-7 days, 76.2 per cent. fertile eggs hatched.

Where eggs were kept 8-14 days, 74 per cent. fertile eggs hatched.

Where eggs were kept 15-21 days, 64.6 per cent. fertile eggs hatched.

Where eggs were kept 22-28 days, 32.2 per cent. fertile eggs hatched.

Where eggs were kept 29-31 days, 0.0 per cent. fertile eggs hatched.

Further, it has been shown that the length of storage increases the incubation period, other things being equal. (Increase in incubation temperature within a small range decreases the incubation period.)

Air Velocity—

Air velocity in the incubator has little effect on hatchability providing other features are kept constant.

Turning of the Eggs—

This seems to have little significance, other than its function of preventing the embryo from sticking to the shell. Authorities, *e.g.*, Olsen (1930), emphasize the need of turning frequently to obtain good hatches. Lamson and Kirkpatrick (1918) found that when eggs were turned twice daily, 55.4 per cent. hatchability resulted; when turned five times, 61.1 per cent. hatchability was obtained. Many others have confirmed these findings.

There is little evidence that turning more than five times daily gives improved results. On the other hand, it has been shown that frequent turning in one direction only, may cause high embryo mortality, due to ruptured yolk-sac, disruption of the chorion, allantois, and shell membrane, twisting of the chalazae, rupture of the blood vessels, &c.

Thus, it will be seen that where turning is not suitably carried out about four times per day, hatchability may be reduced to some extent quite apart from any disease.

However, it is well known that some hatcherymen only turn the eggs twice daily. This is not ideal in mammoth machines with automatic turning devices.

Egg Characters—

Three egg characters at least may affect hatchability.

(*a*) *Weight.*—Authorities all over the world seem to be almost unanimous that only large eggs should be set, otherwise flock stamina may decrease. On the other hand, there is abundant evidence that eggs of only moderate size give best hatchability. For example, Landaeur (1937) and Dunn (1932) show that eggs 50 to 60 grams in weight (1 3/5th oz. to 2 oz.) give highest hatchability; and further, it would appear that those eggs which are larger than usual, are the ones that fail to produce a viable chick.

Excessively large eggs are notorious for poor hatchability, quite apart from any infectious disease condition.

(*b*) *Shape of Egg.*—Very long and narrow eggs give poor hatchability.

(*c*) *Faulty Shell Texture.*—Very porous, thin-shelled eggs rapidly lose moisture and give very poor hatchability.

Position of the Egg—

If the egg is placed the wrong side up (air cavity down) many chickens turn in the shell, placing their beaks in the small end away from the air cavity, and a heavy mortality results. There is little evidence to indicate that laying eggs on the flat is responsible for any mortality of this nature.

The above features are, of course, well understood by an efficient hatcheryman, but it is essential that their significance should be fully appreciated before the effect of disease is either mentioned or reviewed, as a possibility of decreased hatching results.

Condition of the Hen—

It is a well-known fact that the general condition of the hen is reflected in the egg character and quality. Thus, excess of potassium iodide in the diet will produce physiological changes in the hen so that she lays eggs rich in iodine. Deficiency of Vitamin A in the diet leads to eggs deficient in Vitamin A, and it will readily be appreciated that various deficiency and infectious diseases which affect laying hens will influence to a greater or lesser degree the composition of the eggs laid by them. In infectious diseases, such as laryngo-tracheitis, the birds are off their feed, and running a high temperature. Frequently they show symptoms of deficiency of Vitamin A, though still continuing to lay eggs.

Effects of Epidemic Diseases—

While experimental evidence is not available in this State, wide field clinical observations point to the fact that when eggs for incubation are collected from a flock suffering from a severe attack of coryza, hatchability is decreased. The same phenomenon is noticed, but to a less degree, with laryngo-tracheitis. Figures are not available, for the

reason that in the hatching season those in the poultry industry connected with the hatchery trade will not keep figures or undertake any observations which involve the expenditure of time in this peak labour period of the year. The fact remains, however, that hatcherymen who are buying eggs on a large scale for the supply of the day-old chick trade should be particularly careful to avoid flocks which are affected with catarrhal diseases such as coryza (common colds), infectious catarrh, infectious laryngo-tracheitis, and green feed deficiency diseases, if maximum hatchability is desired.

Diseases of the Reproductive Organs—

As is well known, birds which are affected as chickens with pullorum disease (once known as bacillary white diarrhoea) and "recover," remain carriers of the infection. Such carriers may transmit the disease gradually from bird to bird through the flock. The infection, for the most part, becomes localised in the sexual organs, that is, the ovary or testes. In most cases, hens affected with the disease lay from 10 to 15 per cent. of eggs which carry the germ (*Salmonella pullorum*). It has been shown that most of these eggs, when placed in the incubator, do not produce viable chickens. This is fortunate, as the hatching of an infected egg usually results in widespread infection of the other chickens in the incubator by means of the infected fluff which floats off the diseased chick, and is inhaled by other chickens, thus setting up infection.

In a flock heavily infected with pullorum disease it may be noted that hatchability is very low. If all the factors of incubation management already dealt with are in order, then the possibility of pullorum disease should be investigated, particularly if the flock of breeding hens is apparently healthy.

It has been noted on two occasions in this State that following testing and eradication of the "carrier" birds, hatchability has markedly increased (unpublished data). Losses due to pullorum disease can be eliminated by testing. All stock should be tested at intervals over a period, and reacting birds eliminated. This test can be carried out by the poultryman himself. A departmental publication on the subject is available.

The poor hatchability in the case of pullorum disease is explained by the fact that most of the infected embryos die during the incubation period.

A disease due to a special type of micro-organism has been found to be affecting the reproductive organs of fowls in Australia, and results in abnormality of the thick egg albumen, which presumably would cause decrease in hatchability. This disease is not known to occur as yet in New South Wales.

Other infectious diseases which cause inflammation of the oviduct are noted from time to time, and cause much reduced hatchability of the eggs from the particular hens affected.

Deficiency Diseases—

Deficiency diseases in adult birds and young growing stock take an enormous toll from the revenue of the poultry industry each year. The loss sustained is, however, not confined to the hatched bird only, but is often a major factor to the hatcheryman.

Although an inadequate diet supplied to the laying hen will often lead to a decline and cessation of egg production, rather than to the laying of deficient and unhatchable eggs, yet there is conclusive evidence that deficiencies in the diet will also cause poor hatchability of the eggs that are laid.

At present there is little evidence available that nutritional deficiencies in the cocks affect the hatchability of the eggs laid by the hens mated to these cocks.

Avitaminosis A.—

This is a well-defined disease condition in adult birds and chickens.

Fowls which are fed a ration deficient in Vitamin A lay eggs which have a very pale yolk. The yolk is the main reservior of Vitamin A in the egg, but on the other hand there is evidence to suggest that the small portion of Vitamin A contained in the albumen is the important part so far as hatching quality of the egg is concerned.

Sherwood and Fraps (1934) give the following results:—

BIRDS FED INADEQUATE AMOUNTS OF VITAMIN A.

No Lucerne Supplement—38.4% hatchability.
Plus 4% *Lucerne Meal*—46% hatchability.
Plus 8% *Lucerne Meal*—52.4% hatchability.

Similar results are reported by a number of authors. It should be noted that in most cases 8 per cent. of lucerne meal would not be an adequate Vitamin A supplement.

As previously mentioned, the percentage infertility is not taken into account in considering hatchability. It is an interesting fact that while hatchability is markedly decreased by deficiency of Vitamin A, percentage infertility does not seem to be affected in certain cases investigated.

Vitamin D.—

Deficiency of this vitamin causes marked symptoms (rickets) in chickens, and to a less extent in adult stock (abnormal eggshell production and bone conditions). Where a marked deficiency of this vitamin exists, due to the fact that birds are confined indoors, *e.g.*, in intensive sheds, and no Vitamin D supplement, such as fish liver oils, are supplied, the blood calcium falls below the normal level, the weight of the eggshell relative to the total egg weight is much reduced, as is its ash content. In such cases the hatchability is notably decreased, *e.g.*, quoting Landauer (1937):—

U.S.A. P. Units of Vitamin D per 100 Grams. of Feed.

	10	19	39	58	78	115	116	Range.
	%	%	%	%	%	%	%	%
Hatchability	50·4	44·5	74·6	77·4	84·9	87·8	88·1	91

It will be noted that in this experiment the birds on open range which had access to green feed (Vitamin A) and sunlight (Vitamin D) gave the greatest percentage of hatchability. Further, it was shown that embryos which had commenced to develop in the eggs which were deficient in Vitamin D were unable to obtain normal amounts of calcium and phosphorus.

To summarise the position concerning Vitamins A and D—where ample quantities of a good type of green feed are available to the birds and they are run in open sunlight, then no deficiency effects should occur. The green feed should preferably be fed separately at midday or after the evening feed. Pale-coloured green feeds and fibrous materials may provide inadequate amounts of Vitamin A. Where there is an absence of either sunlight or green feed, then breeding fowls should be supplied some supplement in their ration, such as 1½ per cent. of pure cod-liver oil, to maintain the hatchability of the eggs. The feeding of this cod-liver oil when ample green feed and sunlight are available is not advised. Care should be taken to measure carefully the amount of oil used, so that excess amounts are not fed. These excess amounts will cause dietetic disorders and will also result in unnecessary expense.

Other Vitamin Deficiencies—

Vitamin B is a complex vitamin, and under normal feeding conditions in New South Wales is not likely to be deficient, as wheat products are relatively rich in this food constituent. It has been shown with experimental rations (Bethke *et al.* (1936)) that this vitamin is essential for the normal development of the embryo.

Norris *et al.* (1936) showed that when a basal ration extremely deficient in this vitamin was fed, the provision of Vitamin B supplement caused an increase in hatchability proportionate to the amount of Vitamin B2 (or G).

Vitamin E is necessary for normal hatching results. It would appear that with the rations normally fed in this State the deficiency is not likely to occur, and the matter is therefore of experimental interest only. It has been shown by various workers (*e.g.* Card and others (1930)) that when pullets were raised and mated on a diet in which Vitamin E had been destroyed, hatchability was nil. Out of 317 fertile eggs set, none developed beyond the ninth day of incubation. It was shown that embryo mortality usually occurred at about the 84th to 96th hour of incubation.

Another apparent vitamin deficiency of undetermined nature was reported in 1935 in America. In this condition the chicks showed marked bone abnormality, the bones of the extremities being shortened and the skull being abnormal in shape.

A Problem for Husbandryman and Veterinarian—

To summarise, it is pointed out that when poor hatchability occurs, the diagnosis of the cause may be a complex matter, in which the close collaboration of poultry husbandrymen and a veterinarian is necessary.

The poor return of chickens from eggs incubated may be referable either to faulty incubator conditions or to the effect of disease or dietetic deficiency on the hens producing the eggs.

As the economic loss from unhatchable eggs is a matter of considerable economic importance, it is felt that the matter should be gone into more critically in this State, so that avoidable losses may be eliminated. It is obvious that the loss to the hatcherymen is only part of the total financial loss, in view of the fact that where poor hatchability is experienced, those chicks which are produced may have only a low livability, which loss is then borne by the poultryman purchasing the chicks.

Where the loss is associated with an infectious disease such as pullorum, the recurrence of such losses year after year becomes a probability until suitable veterinary preventive measures are adopted.

Diseases and Incubator Hygiene.

As pointed out elsewhere, pullorum disease, omphalitis and certain other diseases may be spread in the incubator. Absolute cleanliness and every possible precaution as to disinfection and fumigation *cannot* be effective in preventing spread of infective micro-organisms which are liberated (on contaminated fluff particles) into the incubator atmosphere by the hatching of the eggs. However, such measures may, and should, be employed to prevent spread of infection from one hatch to a subsequent hatch in the same machine. Unless rigid measures are taken diseases such as omphalitis and pullorum disease may be spread from hatch to hatch by infected dust, down, droppings and yolk and shell residues.

Disinfection—

After each hatch the machine should be thoroughly cleansed. This is best done using a disinfectant solution such as kresolvo, lysol, or some other soapy disinfectant solution, using about a tablespoon to the pint (stronger solutions may burn the hands of the operator). The machine may then be fumigated with a stronger dose of fumigant than indicated below for fumigation when eggs are in the machine, care being taken to leave trays of water in the working machine for some hours before fumigation in order to increase humidity.

Fumigation During the Hatch—

This procedure is by far the better because not only is the machine disinfected, but the egg shells also are treated.

It must be stressed that prior to fumigation thorough cleansing must be carried out as the gaseous disinfectant will not effectively penetrate dirt.

In most modern hatcheries, separate machines, or in some cases, separate compartments of a machine, are kept for hatching. These "hatchers" are obviously the machines that need thorough cleansing and fumigation. Usually the eggs are placed in the hatcher on the eighteenth day and fumigation may be carried out on this day, even though an odd egg may have chipped. Even though the formaldehyde

gas is highly lethal for the microscopic germ life and has little effect on the chicken in the recommended proportions, it is inadvisable to fumigate after the main hatch has really commenced (usually the twentieth day under usual commercial conditions). After the eighteenth day there may be definite danger in the procedure if the hatch is early.

Fumigation is an inexpensive method of disinfection and for thorough effectiveness, a hatch may be fumigated on the second or third day, about the tenth to twelfth day, and again on the eighteenth day. In large machines where eggs are added every few days fumigation may be carried out once weekly.

Methods of Fumigation—

Two methods are commonly employed. The first is apparently more commonly used in America and the second described has been almost exclusively used here. The latter is suitable for all types of machines.

(1) *The Formalin Method.*—For every one hundred cubic feet of incubator space, about 20 millilitres (16 to 25 may be used) of formalin (a 40 per cent. solution of formaldehyde) are taken and absorbed by cheesecloth. Enough cheesecloth is used to absorb all the moisture without dripping.

This wet cheesecloth is then placed in the machine in the direct air current. In fan-forced draught machines this will be directly under the fan; in other types, inside the machine and directly opposite the air intake part. The blast of air evaporates the formalin and disseminates it through the machine. The machine is run normally after the saturated cloths are placed in.

(2) *The Potassium Permanganate - Formalin Method.*—In this method the fumigation is effected by liberation through the mixing of the two ingredients. In old cabinet machines 0.35 millilitres of formalin and 0.175 grams of potassium permanganate are required for every cubic foot of incubator space.

In the larger forced draught machines the amounts needed are greater and 0.40 ml. of formalin and 0.2 grams of potassium permanganate should be used.

The quantities of potassium permanganate required (when calculated) should be weighed out by a chemist, and the amount of formalin should be carefully measured in a 100 millilitre measure.

Amounts are estimated by multiplying the height, width and depth of the machine, taking the full inside measurements and making no allowance for contents such as eggs and trays.

Example.—Inside measurements of incubator—10 feet long, 8 feet from front to back, 6 feet high.

Cubic Contents $= 10 \times 8 \times 6 = 480$.

Requirements—

Formalin 0.4 ml. $\times$ 480 = 192 millilitres.

Potassium permanganate 0.2 $\times$ 480 = 96 grams.

The potassium permanganate is now placed into one or more large plates or dishes (china, glass or enamel) which are then placed in the incubator on the floor or a ledge.

The formalin is then measured out, placed on the potassium permanganate, the door of the incubator closed quickly, and the machine allowed to run as normally. There is no need to close up the ventilation ports. Care should be taken to get effective mixing of the potassium permanganate and formalin. If the container is tipped to one side mixing will not be complete.

Other Factors—

The greater the humidity the more effective is the fumigation. It is an advantage to have the machine running at a normal temperature and to make sure that the humidity is near 100 per cent. at the actual time of incubation.

This high humidity should be obtained by placing wet sand or cloths in water trays, and leaving there for only about half an hour.

Effectiveness of Fumigation—

It has been shown that formalin in the above proportions will kill *Salmonella pullorum* (the causal germ of pullorum disease) in times varying from five to sixty minutes.

GLOSSARY OF TERMS.

Abdomen	The large body cavity bounded by ribs, pelvis and diaphragm.
Abscess	A localised collection of pus, usually associated with inflammation of the surrounding structures.
Acute	Severe. Having a rapid onset, short course and pronounced symptoms and termination.
Agglutination	A joining together. The phenomenon in which micro-organisms are clumped together by the action of the blood (or portion of the blood) of an animal affected with the disease produced by such micro-organism. Agglutination Test.—A biological test as above to detect carriers of a disease *e.g.*, pullorum disease.
Air sacs	The cavities in the thorax, abdomen and bones of birds, lined with mucous membrane, filled with air, and having direct communication with the lungs.
Albumen	The white of a fowl's egg is almost entirely albumin.
Albumin	A protein, found in egg white and other animal substances.
Allantois	One of the membranes developed during the growth of an embryo.
Amoebotaenia	A generic name for certain tapeworms.
Anaemia	A deficiency of blood, or deficiency of constituents of the blood, usually accompanied by pallor and other symptoms.
Antigen	A substance, such as bacteria, which, when injected into a bird, stimulates the production of "protector" substances known as an antibody. An antigen is used in testing to determine the presence of such antibodies in the blood of the bird under test. Antigen—stained antigen. An antigen (such as *Salmonella pullorum*) in which the bacteria are stained in order that the reaction may show up more clearly.
Arecoline	A drug sometimes used for treatment of tapeworm infestation.
Argas	The generic name for some ticks (such as fowl tick).
Argemone Mexicana ...	The botanical name of Mexican poppy.
Argyrol	A proprietary preparation of colloidal silver used in eye and other treatment.

GLOSSARY OF TERMS—*continued.*

Arsenic	A poisonous element. Usually when "arsenic" is spoken of in a popular sense, white arsenic or arsenious oxide is meant.
Apoplexy	A disease or symptom complex due to rupture or obstruction of a vessel in the brain or spinal cord.
Aspergillus	A type of fungus which may infect birds.
Atrophy	A wasting away.
Avitaminosis	A complete deficiency of a vitamin.
Bacteria	Microscopic forms of life, usually said to belong to the plant kingdom.
Bacteriology	The study of bacteria.
Blackhead	A common term for entero-hepatitis, a disease of turkeys and fowls, caused by *Histamonas meleagridis* a protozoan parasite.
Botulism	A disease due to ingestion of toxins produced by a special group of bacteria.
Bronchi	The two tubes into which the windpipe divides at its lower end.
Bumble foot	A disease affecting the foot and surrounding structures (see text).
Bursa	A small blind sac. Bursa of Fabricius is a small blind sac or pouch located just above the cloaca.
Butilydene chloride ...	A drug that has been used for treatment of worm infestation.
Cæca (plural) ... ... Cæcum (singular).	The two long, blind, paired intestinal pouches which can be easily located if the course of the intestines is followed down from the stomach.
Calcium	A mineral necessary for birds.
Canker	A popular term for yellow, cheesy, false membranes which form in the mouth and throat, etc., of fowls affected with so-called "roup diseases."
Cannibalism	A vicious habit or disease in which birds attack one another (see text).
Capillaria	A generic name for some worms of poultry.
Carbon bisulphide ...	A drug sometimes used for the treatment of worms in poultry.
Carbon tetrachloride ...	A drug used for the treatment of worms in poultry.
Carotene	A food substance which is associated with the supply of Vitamin A in the diet.
Carotenoid	Like, or associated with carotene.
Caseous	A term used to describe material cheese-like in colour and consistency.

GLOSSARY OF TERMS—*continued.*

Catarrh	Inflammation of a mucous membrane.
Ceratophillus	The generic name of a flea of poultry.
Chalcoponera	The generic name of an ant.
Chicken pox	The American name for fowl pox.
Choanotaenia	The generic name of a tapeworm of poultry.
Cholera	A disease of birds (see text).
Chorioallantoic	Relating to the chorion and allantoic membranes of the developing embryo of the egg.
Chronic	Long continued, of long duration. Opposed to acute.
Clinical	Pertaining to the features of disease as observed in the field.
Cloaca	Or "vent." The common opening of rectum, urethra and oviduct.
Cloacitis	Inflammation of the cloaca.
Clostridium	A genus of bacteria.
Cnemidocoptes	A type of mite.
Coccidia	A group of microscopic animal parasites which infest birds and other animals.
Coccidiosis	The disease caused by coccidia (see text).
colloidal	The state in which non-crystalline materials occur in very fine particles. Glue-like.
Congestion	An abnormal collection of blood in a part or organ. Reddening, often the first step in inflammation.
Copper sulphate	A drug sometimes used in treating worm infestations (see text).
Coryza	One of the so-called "roup" diseases (see text).
Crematogaster	The technical name for a type of ant.
Cresol	A disinfectant.
Crooked breast	A disease of birds (see text) thought by some to be due to deficiencies in the diet.
Crop	The dilated portion of the gullet which holds food.
Cubic centimetre (c.c.) ...	A measure of volume— 30 c.c. = 1 fl. oz., approx. 1 c.c. = 1 millilitre or m.l.
Davainea	The generic name of a tapeworm of poultry.
Dermanyssus	The generic name of red mite (*Dermanyssus gallinae*).
Diagnosis	Determination of the nature of a disease.
Diarrhoea	A condition characterised by increased frequency and more fluid consistency of the faecal evacuations.

GLOSSARY OF TERMS—*continued.*

Differential	Differential diagnosis is the determination of the nature and identity of a disease by means of noting and assessing the features in which it differs from other related conditions.
Diphtheritic	A term used to describe the yellow or greyish-yellow cheesy false membranes which develop on the surfaces of the mouth, tongue, throat and similar situations in birds affected with "roup" diseases.
Disinfectant	An agent that destroys the germs of disease, fermentation, or putrefaction.
Disinfection	The destruction of microscopic germ life (particularly disease pathogens).
Dominant	The term applied to a characteristic of an animal, which characteristic tends always to show out in the progeny when such animal is bred to the exclusion of the opposite recessive factor, *e.g.*, tallness (dominant) and dwarfness (recessive).
Douglas mixture	The name given to an iron tonic, which is made as follows;—4 oz. of ferrous sulphate and 4 oz. of magnesium sulphate are dissolved in 1 gallon of boiling water. Add half an ounce of dilute sulphuric acid. Two tablespoons of this solution are added to each gallon of drinking water.
Dropsy	A synoym for œdema. An infiltration of the tissues with lymph-like fluid, or the collection of such fluid in the body cavities.
Echidnophaga	The technical (generic) name for certain fleas.
Emaciation	Wasting, leanness, loss of fat and bodily "condition."
Endothelioma	A type of cancerous condition.
Enteritis	Inflammation of the intestines.
Enterohepatitis	A disease of fowls, turkeys and, perhaps, other birds.
Epidemiology	The science of epidemic diseases.
Epizootology	The science of epidemic diseases in animals.
Erythroblastosis	A condition which is one symptom of a type of leucosis.
Erythroleucosis	A type of leucosis—particularly affecting the red blood cells and blood-forming organs.
Exudate	The material which has passed through the walls of blood or lymph vessels into tissue spaces, or into the surface to form a discharge.

GLOSSARY OF TERMS—*continued.*

Favus	A fungous disease of birds.
Febrile	Pertaining to or characterised by fever.
Fever	A symptom of disease, the outstanding feature of which is a rise in temperature, accompanied by changes in pulse and respiration.
Formaldehyde	A formic aldehyde. A disinfectant substance.
Formalin	A solution of 40 per cent. formaldehyde.
Fowl plague	A disease of birds due to a virus.
Fowl pox	A disease of fowls (see text—"roup" diseases).
Fumigation	A method of disinfection employing fumes.
Fungus	A microscopic form of vegetable life (larger and more highly developed than bacteria).
Gapeworm	A parasite of birds, known technically as *Syngamus tracheæ* (see text).
Generic	A generic name is the first part of the technical name of a plant or animal and indicates the genus to which such plant or animal belongs.
Genus	A group of plant or animals arranged according to scientific classification of similarities.
Germ	A term sometimes used to describe a microscopic organism which causes diseases such as a bacterium, fungus, or protozoan parasite.
Glottis	The opening of the larynx.
Gout	A disease of birds (see text).
Grams (gms.)	A measure of weight. 30 gms. = 1 oz., approx.
Haemocytoblastosis ...	A type of leucosis. Some authorities hold that this is the first symptom which leads to any one of the many other forms of leucosis.
Haemorrhagic	Blood-stained.
Hatchability	A term used to define the capacity of fertile eggs to produce living chickens (thus 80 per cent. hatchability means that out of each 100 fertile eggs 80 chickens are produced).
Hatcher	The incubator used to hatch out the eggs, which are usually placed into it on the 17th or 18th day.
Hepatolymphomatosis ...	A type of leucosis.

GLOSSARY OF TERMS—*continued.*

Heredity	The transmission of characteristics from parent to offspring.
Histomonas meleagridis ...	The technical name for the micro-organism which causes blackhead in turkeys and fowls.
Hypovitaminosis	A deficiency of vitamin as supplied to the animal.
Icterus	Jaundice—a symptom of disease involving excessive formation or retention of bile pigments.
Impaction	Massing together of material to form an obstruction, *e.g.*, of food in the " crop " to produce impaction of the crop.
Immunisation	The stimulation of the animal to produce an immunity, *e.g.*, as by vaccination.
Immunity	The condition of the body wherein it resists the development of morbid processes.
Incubation	A term applied to the treatment of eggs such that hatching proceeds.
Incubation period... ...	A term applied (*a*) to the period required for eggs to hatch; (*b*) the period between infection of an animal by some micro-organism and the development of symptoms of disease as a result of infection.
Infection	(1) The communication of disease from one body to another. (2) The material or agent causing the disease.
Infectivity	Infectiousness—the quality of being infectious.
Inflammation	A symptom of disease, prominent features of which are heat, pain, redness and swelling.
Infra-orbital	Below the eye—infra-orbital sinus, the cavity in the head below the eye and lateral to the nasal cavity.
Ingestion	Eating; the taking in of food.
Intra-follicular	A term used to indicate location in a follicle, *e.g.*, when fowl pox vaccine is placed in a feather follicle, as in the intra-follicular method of vaccination.
Iodine	An element frequently employed in various forms as an antiseptic.
Jaundice	See icterus.
Kamala	A drug sometimes used for the treatment of tapeworm infestations.
Larynx	The top portion or entrance to the windpipe.
Laryngitis	Inflammation of the larynx.

GLOSSARY OF TERMS—*continued.*

Laryngo-tracheitis ...	A disease of birds (see text—"Roup" diseases).
Lesions	An injury, wound, or morbid structural change.
Leucosis	A term used in this book to include a whole group of allied diseases, which may or may not be due to one cause (see text).
Lipeurus	Generic name of a louse (*Lipeurus heterographus*).
Lymphatic	Pertaining or referring in some way to the lymphatic system.
Lymphocyte	A type of white blood cell.
Lymphocytoma	A type of leucosis.
Malaise	The feeling of depression and lack of "well being" which accompanies some cases of systemic disease.
Malefern	A drug sometimes used for the treatment of tapeworm infestation.
Manganese	A mineral necessary in trace amounts for the nutrition of poultry.
Menopon	The generic name for a type of louse.
Mesothelioma	A type of cancerous condition.
Micro-organism	A vegetable or animal form of life, microscopic in size.
Millilitre (m.l.)	A measure of quantity synonymous with cubic centimetre (c.c.). 30 m.l. (or c.c.) = 1 fl. oz. approx.
Mortality	Death rate.
Mucous	Having the nature of mucus. Mucous membranes are the membranes of the body (usually in contact with air) which secrete mucus.
Mucus	A viscid liquid (varying in consistency).
Mycosis	A disease of birds due to fungus (see text).
Myeloid leucosis	A type of leucosis (see text—diarrhœa and wasting diseases).
Naphthalene	A drug sometimes used for the control of external parasites
Navel	Or umbilicus. The connection between the body cavity of the chick and the egg cavity before "chipping" of the egg occurs. The yolk passes through this connection into the body cavity of the chick.
Necrosis	The death of cells surrounded by living tissues.
Necrotic	Pertaining to or characterised by necrosis.

GLOSSARY OF TERMS—*continued.*

Nemural	A drug sometimes used for the treatment of tapeworms.
Neoplastic	Cancerous in nature.
Neurolymphomatosis ...	A disease grouped with leucosis (see text).
Newcastle disease ...	A disease of birds due to a virus (see text).
Nicotine sulphate ...	A drug used for the expulsion of intestinal round-worms from poultry.
Nictitating membrane ...	The third eyelid present in birds. The function of which is to remove foreign bodies from the eyeball surface.
Obesity	Excessive fatness—particularly with reference in poultry to masses of abdominal fat.
Ocular	Pertaining to or in relation to the eye.
Œdema	Dropsy.
Œsophagus	The gullet or tube between the throat and crop.
Oil of chenopodium ...	A medicament sometimes used as a vermifuge.
Omphalitis	Inflammation of the navel.
Orbital	Pertaining to the orbit or structures surrounding the eye.
Ornithostrongylus ...	The generic name for a worm of pigeons.
Ova	Plural of ovum, an egg—refers to the " yolks " as produced in the ovary.
Ovary	The female glandular organ which produces the ova.
Oxyspirura	The generic name for a type of worm infesting the eye.
Paratyphoid	The group name for a certain type of related bacteria.
Paralysis	A loss of power, motion and/or sensation in a part.
Paresis	A slight paralysis, an incomplete loss of muscular power.
Pathogenic...	Disease-producing.
Pathogenicity	The condition or quality of producing disease.
Pathogens	Agents which produce disease.
Pectoral	Pertaining o the " chest " (or breast in the case of birds).
Pendulous	Drooping and dilated.
Peracute	Very acute.
Peritoneum	The membrane lining the abdominal cavity and surrounding the organs in that cavity.
Perosis	A deficiency disease (ee text).

GLOSSARY OF TERMS—*continued.*

Pharynx	The throat—continuous on the one side with the mouth and nose and on the other with the gullet (or Œsophagus).
Phosphorus	An element necessary for the nutrition of birds.
Pomegranate	A type of fruit. Pomegranate preparations are sometimes used as vermifuges.
Post mortem	After death. Post mortem examination—examination of the dead bird or animal.
Poult	A young turkey.
Predisposing	Rendering susceptible or liable to attack.
Protozoan	Pertaining to protozoa, which are microscopic animal parasites.
Proventriculus	The glandular stomach located in front of the gizzard.
Psittacosis	A disease of birds and humans due to a virus (see text).
Pullorum	A disease of birds, sometimes referred to as bacillary white diarrhœa (see text).
Pustule	A small circumscribed elevation containing pus.
Pyrethrum	A medicinal agent sometimes used as a vermifuge.
Raillietina	The technical (generic) name of certain tapeworm.
Reaction	The response of an organ or part of an animal to a stimulus.
Reactor	An animal that reacts.
Rearability	The capacity of the chick to live throughout the rearing period of its life.
Recessive	The reverse of dominant (see dominant).
Red mite	An external parasite of fowls (*Dermanyssus gallinæ*).
Rickets	A deficiency disease (see text).
Roup	A term popularly used to indicate a group of poultry diseases which closely resemble one another in their symptoms.
Salmonella	The technical term used to describe a class of bacteria grouped according to certain technical similarities.
Scalpel	A lance or surgical knife.
Scolex	The head of a tapeworm.
Serological	Pertaining to serology, which is the branch of science dealing with su h questions as diagnosis by means of blood tests and reactions.

GLOSSARY OF TERMS—*continued.*

Sinus	A cavity, *e.g.*, the cavity below the eye is the infraorbital sinus.
Sinusitis	Inflammation of a sinus. Also the name given to a disease of turkeys (see text).
Slipped tendon	A term synonymous with perosis.
Spirochætes	Microscopic animal parasites.
Spirochætosis	A disease of birds due to infection by spirochætes (see text).
Strobila	The body of the tapeworm.
Subacute	The stage of a disease when it is between acute and chronic.
Susceptible	Liable to become infected with a disease.
Symptoms	The change in a live bird occurring during disease, serving to point out its nature.
Syngamus trachea ...	The technical name for gapeworm.
Tetrachlor-ethylene ...	A drug used for the treatment of roundworms in poultry.
Tonics	Agents which are used in the hope of improving the health condition and production of poultry; for example see Douglas mixture.
Trachea	The windpipe.
Tracheitis	Inflammation of the trachea.
Treponema anserinum ...	A microscopic animal parasite which causes spirochætosis in fowls.
Trichomonads	Microscopic animal parasites which cause disease.
Tryparsamide	A drug (arsenical) used in the treatment of certain disease (such as entero-hepatitis—see text).
Tube agglutination test ...	A technical test used to detect carriers of certain diseases (*e.g.*, pullorum disease).
Tuberculosis	A disease of birds, animals and humans (see text).
Turpentine...	A medicament sometimes used as a vermifuge.
Ulceration	The formation of ulcers which are patches of tissue which die gradually and erode away.
Ureter	The tube from the kidney to the cloaca.
Vaccine	A strain (usually weakened) of an infective agent used for inoculation to produce an immunity.
Vaccination	Inoculation of a bird with a vaccine.
Vent gleet	A disease of fowls (synonym—cloacitis).
Vermicide	An agent that destroys intestinal worms.
Vermifuge	An agent that expels intestinal worms.

GLOSSARY OF TERMS—*continued.*

Viability	The capacity to live.
Viscera	Plural of viscus—an organ contained within the body cavities (abdomen, pelvis, thorax, or cranium).
Vitamins	Complex food substances necessary to support health. Knowledge of these ubstances is mainly confined to the effects produced by thei absence.
Warts	Small projections of the skin.
Waterbag	A popular name for several separate disease conditions (see text).
White comb	A popular term for favus (see text).
White eye	A popular term for a disease in ducks (see text).

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Sydney: Thomas Henry Tennant, Acting Government Printer—1939.