Antibiotics, particularly penicillin, are useful. Used early enough and at adequate levels, they usually give satisfactory results. One should not expect all sick birds to recover, however, and relapses may occur.

Erysipelas adsorbate bacterin has shown promise as a biologic for vaccination, but a final estimate of its value must await further reports from users. For a long time we have needed an immunizing agent that will protect the growing turkey right on through to market time.

Removal of the snood, or dewbill, of day-old turkey poults in areas where the disease is prevalent has been suggested by W. A. Billings, of the University of Minnesota. The basis for such a procedure is that the snood is especially apt to be injured because of its location; removing it would reduce the hazard of wound infection with the erysipelas organism. Debeaking at about 14 weeks also has been suggested in an effort to minimize the effect of fighting among the toms.

Segregation of sick birds, moving the remainder to a new range, and the use of antibiotics will help to control an outbreak. An infected range should not be used for turkeys for a year. Any low points should be landscaped to eliminate areas of standing water, and a cover crop should be put in. As a precaution, particularly in the places where erysipelas is known to exist in hogs or sheep, turkeys should be kept away from any contact with them.

One should remember that the causative organism of turkey erysipelas can infect people (erysipeloid). A person should therefore handle sick or dead birds only with proper care.

Richard D. Shuman is a veterinarian in the Bacterial and Mycotic Disease Section of the Animal Disease and Parasite Research Branch. He was graduated from the College of Veterinary Medicine of the State College of Washington in 1938.

O. L. Osteen joined the Department of Agriculture in 1932 and has engaged in research in diseases of animals and poultry.

Chronic Respiratory Disease in Chickens

J. F. Sullivan, Clarence H. Thompson, Jr., and O. L. Osteen

Chronic respiratory disease ( airsac infection), a respiratory disease of chickens, has a slow rate of spread and a persistent nature.

The average annual mortality from chronic respiratory disease (CRD) in chickens from 1942 through 1951 was more than 14 million birds. Each year additional losses are due to the decreased gains and lowered feed utilization that accompany the disease, extra time needed to bring broilers to market, a larger number of unmarketable birds, and lower egg production and hatchability.

A pleuropneumonia-like organism causes CRD. The organism is extremely small and, like viruses, can pass through certain filters designed to retain bacteria. Like bacteria, it can be seen, following appropriate staining, with an ordinary microscope. It grows readily in the yolk sac of 7-day-old embryonating chicken eggs and can be propagated in suitable cell-free cultural media. The organism has varying degrees of sensitivity to antibiotics, ranging generally from complete inhibition by streptomycin to total resistance to penicillin.
Observations made in the field and investigators’ inability to produce in the laboratory the entire picture of CRD as it occurs in nature suggest that various factors have a part in starting severe outbreaks—extremes of climate, lack of sanitation, mismanagement, or other diseases.

Sometimes it seems that Newcastle disease, infectious bronchitis, and vaccination against them provoke CRD.

Investigations by Canadian scientists attribute the occurrence of CRD to a specific virus acting simultaneously with the pleuropneumonia-like organism. This agent has not been recovered by American investigators.

CRD might be spread to healthy flocks by introducing apparently healthy carriers as flock replacements or by putting clean and infected flocks close together on the same premises. The unusually long duration of the disease once it has gained access to a flock makes its spread more certain.

The disease may be transmitted from infected hens to the chicks through the eggs. The agent causing CRD has been recovered from young cull chicks.

CRD has been observed in chicks hatched from eggs laid by infected hens and maintained in isolation about 15 weeks. Although this suggests that transmission by eggs is related to outbreaks in growing birds, the true importance of this means of spread has not been determined. The presence of the CRD organism in incubator eggs that are pipped but do not hatch at the usual time indicates that incubator transmission likewise may have an undetermined part in the general dissemination of the disease.

The course and severity of CRD reflect environmental factors and the individual status of the exposed bird. Aside from the slow rate of spread and tendency toward chronicity, CRD in many respects seems similar to Newcastle disease and infectious bronchitis.

A drop in the consumption of feed by broiler flocks usually is the first indication of infection. Soon generalized symptoms of respiratory distress appear. They may include nasal discharge, audible breathing, sneezing, and coughing. Coughing is more noticeable at night, when the cooler air makes it worse. In some cases the face may swell up in the area of the infraorbital sinus—below the eye.

Mortality in broilers may run as high as 10 or 20 percent of the flock. The heaviest losses occur in the first few weeks. The mortality among adult or laying birds is negligible, but the drop in their productivity means serious economic losses.

The most commonly observed post-mortem findings are inflammatory involvements of the air sacs and other serous membranes in the body cavity. These tissues, appearing as thin, transparent membranes in health, are somewhat thickened and may show varying degrees of opacity. Small curds of caseous material are often found in or on the air sacs or within the trachea. The serous membranes covering the lungs, heart, liver, and intestines often are involved. The organs themselves are also affected in some cases. The mucous membranes lining the upper respiratory passages are often inflamed and secrete abnormal amounts of mucous material.

Any diagnosis based entirely on clinical observations is subject to question because of the similarity of symptoms observed in CRD, Newcastle disease, and other common respiratory diseases of poultry. A definite diagnosis can be made only through certain laboratory techniques.

A tentative field diagnosis may be confirmed in the laboratory by isolating and identifying the causal agent of CRD in embryonating chicken eggs, where it is known to grow well, or in certain enriched bacteriological media. The microscopic examination of appropriately stained tissues, obtained from affected birds, also is an effective means of confirming a field diagnosis.

In other commonly employed laboratory procedures, the chickens and poults are given hypodermic injections of suitably treated tissues obtained
from birds suspected of having CRD. The development of characteristic symptoms and lesions in birds immune to Newcastle disease and infectious bronchitis is highly suggestive of CRD.

Diagnostic blood serum tests (similar to those used in the pullorum testing program), although still in an experimental stage have been used in some laboratories.

**Antibiotics** have been used to treat CRD, although opinions have differed as to their true worth. In view of the many known and unknown factors that may initiate an outbreak and the possibility of infection at the same time with other poultry diseases, the recorded variation is to be expected. Low levels of antibiotics incorporated in the feed promote growth; high or therapeutic levels, administered to individual birds or to flocks, are costly and in many instances fail to offer a worthwhile return.

The best way to control CRD is to keep it out of clean areas and flocks. Replacements should never be obtained from an unknown source or from a flock with a recent history of respiratory disease because of the danger of obtaining an apparently healthy carrier. Baby chicks should be obtained from a reliable hatchery where healthy breeder flocks are maintained under constant supervision. They should be raised in isolation. Eggs from flocks showing any active respiratory infection should never be set.

The numerous stress factors—climatic extremes, improper sanitation and management, and concurrent bacterial, viral, and parasitic diseases that may initiate a severe outbreak of CRD as well as the chronic nature of the disease—emphasize the need of adequate sanitation and management throughout the growing and laying period.

Birds should be raised in properly ventilated houses that provide adequate protection against extreme variations of temperature. Overcrowding should be avoided. Litter, whether fresh or built up, should be dry and crumbly. Waterers and other equipment should be cleaned and disinfected (with a 2-percent lye solution) periodically. Houses should be thoroughly cleaned and disinfected after one flock has used them and before another flock is put in them.

CRD can be eradicated by marketing the entire flock immediately. If conditions favor a less drastic method, the flock should be kept well isolated, and all birds known to be infected should be removed. Identification of infected birds is usually accomplished by physical examination of all birds in the flock and (in a limited number of instances) by the use of experimental blood tests. Unless extremely valuable birds are involved, the rest of the flock should be marketed as soon as that is possible.

In either instance, the entire premises should be cleaned thoroughly and disinfected before new birds are bought.

J. F. Sullivan joined the Department of Agriculture in 1953. As a veterinarian in the Agricultural Research Service, he has done major research in respiratory diseases of poultry.

Clarence H. Thompson, Jr., was formerly a veterinarian in the Animal Disease and Parasite Research Branch, where he worked on diseases of poultry. He joined the State Experiment Station Division of the Department in 1954.

O. L. Osteen received the degree of doctor of veterinary medicine from the University of Georgia in 1928. He joined the Department of Agriculture in 1932.

For further reading:

