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Progressive Pathologic Signs of Botulism in Pheasants

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Much has been written about the problem of botulism in wild ducks and geese where the number of deaths in a single area may be strikingly high. The importance of botulism in wild pheasants, however, is unknown, since scavengers and dense cover may conceal the numbers of dead birds. Since wild pheasants seldom form large flocks, the chances of many deaths in a single outbreak are slim.

Botulism has been reported as a problem in game farm pheasants since 1954. Outbreaks have been reported in Wisconsin,[8] Indiana,[3] New York,[2] California,[5] Ontario[4] and South Dakota.[7] Most often the birds are raised indoors until they are a few weeks old, at which time they are put out-of-doors in open-top pens. In order to give the birds protection from flying predators, grass and weeds in these pens are allowed to grow high. This forms an effective hiding place for the birds. When a pheasant becomes ill from any cause, it will crawl into a dense area and perhaps will die there. In the heat of the summer, the carcasses decompose rapidly, drawing flies and other insects. *Clostridium botulinum* may be in the insects or in the pheasant intestines, since it is most ubiquitous. Decaying flesh creates an ideal habitat for the anaerobic organism. As the bacterium grows and dies, it releases toxin. Fly larvae seem to have the capability of concentrating toxin, so as another pheasant eats the larvae it picks up a lethal dose, and the cycle is perpetuated.

In the South Dakota Animal Disease Research and Diagnostic Laboratory, our previous experience with botulism had been limited to domestic birds, and we could not equate signs in pheasants with those of chickens and turkeys. This study was undertaken to determine the clinical signs of botulism in pheasants.

Cultures of *Clostridium botulinum* types A, Cα, Cβ, D, and E were obtained from the American Type Culture Collection. Toxins were produced using the inverted dialysis tubing method of Sterne and Wentzel.[6] Each toxin was tested for potency by mouse inoculation, and all were found to be lethal for mice.

Each type of toxin was given to adult pheasants, both orally and intramuscularly. Of the strains used, only types A and Cα were found to be toxic to pheasants. 0.1 ml. of toxin inoculated intraperitoneally into mice gave an LD_{50} of $10^{4.5}$ for type Cα and $10^{6.5}$ for type A. The oral minimum lethal dose in adult pheasants was 0.3 ml. and the intramuscular dose was 0.1 ml. for type Cα. The oral minimum lethal dose for type A was 0.5 ml., while the intramuscular dose was again 0.1 ml.

Twenty-four 8-week-old birds were inoculated with each toxin, twelve intramuscularly and twelve orally. Clinical signs were observed closely and necropsies done on each bird that died. All the intramuscularly inoculated birds died, but two of the twelve receiving oral doses survived. Gross necropsy findings were non-specific. Many birds had excess fluid in the pericardial sac, and in some the cloaca was distended with urates. A few had enlarged spleens. These findings were not consistent among all birds. The

feathers did not become loose, and the nictitating membranes were not involved. Routine bacteriologic and histopathologic studies were made on brain, spinal cord and major organs of each bird. No histopathologic changes were found, and bacteriologic studies revealed the predominant organisms to be *Staphylococcus epidermidis, Escherichia coli* and alpha Streptococci, none of which may be considered pathogens.

Immunity was not produced by sublethal intoxication. Several birds had severe signs of botulism, recovered and then succumbed to a slightly larger dose of toxin given two weeks later.

Toxoid was produced from type Cα toxin and given in two injections, 3 weeks apart. Two weeks following the final injection these birds were challenged with toxin sufficient to kill control birds. Those protected with toxoid survived.

Toxoids may be useful in controlling botulism on game farms. However, it is possible that this protection is insufficient to save a pheasant from the amount of toxin it might pick up in a botulism outbreak. The best protection is removal of all carcasses before they can become a source of toxin.

Literature Cited

1. BOROFF, D. A. and REILLY, J. R. 1959. Studies of the toxin of Clostridium botulinum. V. Prophylactic immunization of pheasants and ducks against avian potulism. J. Bact. 77: 142-146.
2. CHEATUM, E. L., REILLY, J. R. and FORDHAM, S. C., JR., 1957. Botulism in game farm pheasants. Trans. N. A. Wildl. Conf. 22: 170-179.
3. DEMAREE, HERALD A., JR. 1968. Pheasant botulism and toxoid treatment investigation on the Jasper-Pulaski State Fish and Game Area in 1968. Rpt. of Indiana State Fish and Game Dept.
4. FISH, N. A., MITCHELL, W. R., and BARNUM, D. A. 1967. A report of a natural outbreak of botulism in pheasants. Can. Vet. Jour. 8: 10-16.
5. ROSEN, M. N. 1955. Some new pheasant diseases in California. Trans. N. A. Wildl. Conf. 20: 220-228.
6. STERNE, M. and WENTZEL, L. M. 1950. A new method for the large scale production of a high-titre botulinum formol-toxoid types C and D. J. Immunol. 65: 175-183.
7. SUTER, D. D. 1968. Personal communication.
8. VADLAMUDI, SRIKRISHNA, LEE, V. H., and HANSON, R. P. 1959. Case report — botulism type C outbreak on a pheasant game farm. Avian Dis. 3: 344-350.