SUSPECTED BOTULISM IN POULTRY

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B OTULISM has been recorded in most parts of the world in fowls and ducks. A clinical diagnosis of the disease has been made previously in Papua. Outbreaks can usually be traced to the ingestion of spoiled food, decaying vegetable matter, or carcases of dead animals. In the suspected outbreak described in this article a large commercial flock at Lae was involved.

Six thousand birds are kept on the farm where the outbreak occurred. All birds over six weeks of age are run on a modified colony house system. During the day free range into the surrounding bush is allowed the pullets in order to supplement the diet with green feed. It was amongst 1,000 of these pullets that the disease manifested itself. Unfortunately losses had been going on for five days before the owner became fully aware of the seriousness of the situation. It is a standard procedure on the farm for native employees to dispose of fowl carcases as soon as they are found. Over 100 carcases were disposed of in five days before it was reported to the owner that an abnormal number of deaths was occurring.

On the sixth day the author was called in to give advice. Four clinical cases were presented and the following clinical signs were seen. The affected birds were disinclined to move, appearing drowsy, with their eyes dull and partly closed. There was a progressive paralysis of the leg and wing muscles and the gait when the bird was forced to move was unsteady. The head was held in a characteristic fashion with the point of the beak resting on the ground. As paralysis developed the head hung loose and finally a comatose state was reached in which the affected birds died a few hours later. A looseness of the feathers was observed in some birds. Post-mortem signs were

generally negative. There was a slight enteritis in one bird and several lesions considered to be those of coryza were present.

The owner was advised to confine the birds to their houses and to search the surrounding area for carcases. Eighteen carcases in various stages of decomposition were found and destroyed.

Losses ceased within a day of the birds being confined and no further losses were suffered when the birds were again released on the ninth day.

In all 150 of the 1,000 pullets were Iost—a mortality rate of 15 per cent.

Botulism is caused by the ingestion of the toxin produced by the anaerobic bacterium Clostridium botulinum. The bacterium grows in decaying organic matter, when suitable conditions of acidity and temperature are present. As it grows it produces a powerful toxin which is very resistant to extremes of temperatures. Very small amounts of the toxin will kill birds or animals which eat material contaminated with it. Animals dead of botulism constitute a further hazard for other individuals which might happen to eat portions of them. This could conceivably have occurred in the outbreak described.

To confirm a diagnosis of botulism it is necessary to demonstrate the presence of the toxin in the intestinal contents of affected animals. This is done by injecting bacteria free filtrates of the material into experimental animals, some of which have been immunised against botulism. Death in non-protected and survival of immunized animals is used as the criterion diagnosis of the disease. The Lae outbreak was not confirmed in this manner but there is considered to have been sufficient evidence in the clinical signs seen and the disease history to support the diagnosis of botulism.

The outbreak emphasises the need for cleanliness and hygiene in poultry runs and the need for the rapid diagnosis and treatment of any disease which occurs.

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