

Ventricular Myopathy

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VENTRICULAR MYOPATHY or avitaminosis-E of turkeys is quite uncommon. The affection is rarely encountered in the field, the usual incidence being less than one percent in a given flock. However, when the incidence increases to 60 percent, it stimulates the interest of those people concerned with the turkey industry.

Affected gizzards were submitted to the Iowa Veterinary Diagnostic Laboratory by Dr. Marion H. Carter, Federal Veterinary Poultry Inspector for Fairmont Food Company, Inc., at Webster City, Iowa. Dr. Carter said he had not encountered this condition in the 1951 turkey run nor in the other flocks processed by the plant in 1952. The plant processes about 75,000 turkeys annually, and this particular flock numbered 4,000 birds. It was estimated that about 60 percent of the gizzards were affected to various degrees; this constitutes quite a loss.

The turkeys from which the gizzards were taken were in excellent condition at the time of marketing: their bodies were well-fleshed, firm, plump and large for their age. No clinical symptoms of disease were noted. However, in the eviscerating room Dr. Carter noted that many of the gizzards had small gray lesions on the cut surface of the muscle. These were irregular in shape and measured one or two millimeters in diameter; others had much larger gray areas, up to one centimeter in

size. There were some gizzards in which an entire half was grayish in color. It was easy to distinguish between the darker color of the normal muscle tissue and the light areas of necrosis and hyaline degeneration in these gizzards.

Although the gizzards retained their normal shape and tonus, it was quite evident that a degenerative change had taken place in the muscle tissue. These lesions were not just superficial but extended throughout the entire muscle. The gross lesions were present in only one of the muscles of the gizzard in all cases. The Iowa Veterinary Diagnostic Laboratory diagnosed the case as ventricular myopathy.

Ventricular myopathy has been produced experimentally in turkeys by Pappenheimer, Goettsch and Jungherr of Connecticut State College at Storrs, Conn. By eliminating vitamin E from the diet of young poults up to two weeks old, they were able to produce this condition in as high as 97 percent of those poults on test. If they waited until the poults were over two weeks of age and then eliminated vitamin E from the diet, the figure dropped to less than 10 percent. The results of their work showed that if the damage was not severe, the tissue would be repaired and macroscopic lesions could not be discerned. If the damage was quite severe, however, then the lesions were much like the ones described previously in this article.

In compiling this material the author was confronted with at least two explanations that may be the cause or which may contribute to the cause of ventricular myopathy. The first of these was that in

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feeding any animal for the purpose of marketing the meat, the animal is fed so that it gains at an abnormally rapid rate. In so doing, the animal's status is transformed from one that is physiological to one that is near pathological. Therefore, the metabolic balance of the animal may be disturbed. However, this does not appear to be a reasonable explanation in this case. In view of the fact that ventricular myopathy must be initiated in the first 14 days of the bird's life (according to experimental findings) and the time of marketing would be in the distant future, there would be no point in trying to fatten them at so few days of age. Furthermore, the diet was not of a fattening nature.

The second explanation states that it would be very difficult to prepare a feed formula from basic grains that did not contain an adequate source of vitamin E. This statement is quite correct. However, the feed reported consumed by these poults during the first two weeks of life contained only one constituent, dehydrated alfalfa leaf meal, which is generally recognized as being a significant source of vitamin E. There were no whole grains used in the formula. The workers at Connecticut State College conducted experiments in which they used 5 percent alfalfa leaf meal to supply vitamin E. They concluded that this level was insufficient to afford protection against nutritional myopathy. Furthermore, it was determined that turkeys require higher levels of vitamin E and, perhaps, of other vitamins than do chickens and rats. Therefore, it appears that foodstuffs which normally supply sufficient amounts of vitamin E to other animals are deficient for turkeys.

Other possible causes of ventricular myopathy may have a connection with disease produced by living organisms or a group of chemical compounds called antimetabolites.

An investigation in connection with the feeding and management of these birds was conducted by the author. The poults were started as day-old birds by the grower. He fed a commercial turkey mash in pellet form up to eight weeks of age. The grower did not feed any vitamin supplements. At eight weeks the birds

were fed whole and ground oats, and at 12 weeks the birds were changed to a growing mash in which there was not more than .04 percent yellow corn meal. From that time on the poults were fed whole corn, protein concentrate, and were ranged on a mixture of timothy and red clover pasture. According to the grower, the birds had not been sick at any time.

In view of the fact that many growers in that area employed similar feeding practices, one might expect to find the same condition in their birds. With this in mind, an inquiry was made at the local processing plant which handles some 300,000 turkeys annually. The inspector, a layman, had not noticed any gizzards so affected. Thus it appears that some factor, other than vitamin E deficiency, may be involved in ventricular myopathy.

In summarizing this disease, it is well to remember the following facts: (1) It is usually an individual rather than a flock condition; (2) Experimentally it can be produced in poults only in the first two weeks of life by withholding vitamin E from the ration; (3) It is not infectious and clinically the birds are healthy throughout life; (4) It is only diagnosed on post-mortem; (5) The chief economic significance is that affected gizzards are condemned; and (6) Possibly some factor other than vitamin E may be involved in ventricular myopathy.

References

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Disc protrusion affects principally the dog. It results from rupture or protrusion of the intervertebral disc with compression demyelination occurring in the spinal cord. It is more commonly observed in long-backed, short-legged dogs three years of age or older. Intervertebral disc protrusion causes a major share of the paraplegia observed in the dog.