kidneys, proventriculus, ventriculus, intestine, sciatic nerves and beak. Occasionally the ureters were seen to be whitish, injected and slightly distended. In gross lesion cases, constituting approximately 85 per cent. of the total, the cerebellum was found enlarged, edematous, grayish and affected with minute hemorrhages around the central white matter and on the occipital surface; protracted cases sometimes showed a greenish-brown hue in the affected areas. Encephalomalacic foci were occasionally seen in the cerebrum, usually in conjunction with cerebellar lesions, but not as yet in the optical lobes. Histologic examination of etherized chicks revealed extensive pyknosis and edema in the granular layer of the cerebellum, hyaline thrombosis, hemorrhages and necrosis of the Purkinje cells, in the affected areas. Similar areas of softening without hemorrhages occurred occasionally in the cerebrum, surrounded by fat-laden macrophages. Dr. Pappenheimer kindly examined sections of our material and regarded the lesions as closely resembling those of experimental nutritional encephalomalacia.

Etiologic studies on transmissibility of the condition and attempts at cultural isolation of causative organisms have been conducted with entirely negative results. Although many field cases occurred in poorly ventilated brooder houses, and positive CO blood tests could occasionally be obtained, the condition was later observed on range, and experimentally CO-poisoned chicks did not exhibit the pathognomonic lesions. Feeding experiments with commercial brands of feed associated with which a high incidence of the disorder has been observed will form the subject of a further report.

The differential diagnosis of paralytic conditions in growing chicks to which field encephalomalacia belongs is of importance, in view of the trade practice of trying to hold either hatchery or feed concerns responsible for chick losses not directly attributable to disease factors derived from the premises. In such controversies the laboratory report is frequently made the basis of settlement. A differential scheme has been presented by the author,[3] based upon the exclusion of microbial agents and grossly recognizable conditions such as osteoporosis and perosis, followed by histologic examination of the beak, liver, kidneys, pancreas, sciatics and the brain. Of the non-microbial paralytic disorders, field encephalomalacia tallies in clinical features with the condition described by Dunlap[4] as an ataxia associated with nephritis. Pathologically it differs from the latter in the absence of kidney lesions and the presence of brain lesions. Both the nephritic ataxia and the brain disorder go under the popular term "crazy chicks"; the confusion caused by this term can readily be appreciated. The existence, however, of nephritic ataxia as an entity is doubtful; it has not been definitely observed in a large number of chicks examined histologically in this laboratory, although slight autolytic changes have been seen which to some extent simulated the alleged characteristic tubular pyknosis in the kidneys. Of other non-microbial paralytic conditions in chicks, uremia, so-called visceral gout, vitamin A[5] and vitamin G[6] deficiencies, epidemic tremor,[7] neurolymphomatosis gallinarum[8] and possibly the lack of the antihemorrhagic factor[9] have to be taken into consideration, especially in atypical cases.

A brain disorder has been observed in brooder chicks kept on commercial rations which was indistinguishable in symptomatic and histologic manifestations from nutritional encephalomalacia inducible with simplified diets. The etiology of this condition is not definitely known. The popular term "crazy chicks" is applied to it, but is confusing, as it has been linked in the literature[10] to a nephritic ataxia without brain lesions, the nature of which is uncertain. Histologic examination of certain organs is necessary for the differentiation of field encephalomalacia from other non-microbial paralytic disturbances in growing chicks.

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