thyroid apparatus and vitamin D (this latter being chemically related to the sex hormones) were the prime supervisors of the utilization and deposit of lime. Remembering the more prolonged epiphyseal growth of the bones in human castrates the anatomist and anthropologist may also give consideration to the circumstance that all the several sex cycles of females probably involve temporary interruption of calcium deposit (actual extraction?) in the bones, and perhaps thus account for a lower stature in sister than in brother; and again, they may unite this and a further fact—namely, that in some individuals and races these sex cycles begin earlier than in others—to assist, even if slightly, our understanding of the complicated problem of the statural differences of individuals and races.

V

Summary: Adequate and sufficiently prolonged administration of gonad-stimulating hormone (F.S.H. or perhaps the luteinizer) of the anterior pituitary results in an increase of serum calcium in normal, hypophysectomized or thyroidectomized pigeons; but not in castrates. This action is apparently exerted on tissues producing sex hormone or chemically related substance; the parathyroids may or may not be essential to the response. This action is obtained more quickly in females with intact ovaries than in males or in operated animals of either sex. Prolactin, cortin, F.S.H.-free "growth hormone" and probably thyrotropic do not have this action on blood calcium.

Sufficient and prolonged dosage of certain female sex hormones have great power (theelin), others notably less power (dihydrotheelin, theelol, progesterone), to increase the serum calcium in normal, castrate, hypophysectomized, thyroidectomized pigeons and rats, and in normal doves, fowl and dogs (not obtained in rabbits). Androstenediol and the male sex hormones testosterone and its oxime have no similar or demonstrable power to increase the level of calcium of the blood in these animals.

Some significant applications of the fact that male and female sex hormones contrast sharply in their action on calcium metabolism of normal and suitably operated animals are noted.

OSCAR RIDDLE
LOUIS B. DOTTI
STATION FOR EXPERIMENTAL EVOLUTION,
CARNEGIE INSTITUTION OF WASHINGTON,
COLD SPRING HARBOR, N. Y.

A FIELD CONDITION RESEMBLING NUTRITIONAL ENCEPHALOMALACIA IN CHICKS

IN the course of vitamin E experiments in fowls fed a simplified but otherwise complete diet, Pappenheimer and Goettsch[1] observed a cerebellar disorder in chicks which was apparently of nutritional origin and which was characterized by ataxia and spasms, and histologically by ischemic necrosis in the affected areas. Further studies published in a series of papers culminated in the discovery by Goettsch and Pappenheimer[2] of a hitherto unrecognized protective factor against the experimental disease in certain vegetable oils.

A corresponding disorder has apparently not been recognized in the field. It is the purpose of this communication to record the occurrence of a brain disorder in chicks fed commercial rations which is indistinguishable in symptoms and in gross and histopathology from induced nutritional encephalomalacia.

The first definitely recognized case came to the writer's attention in September, 1935, in a lot of 1,200 Barred Rock chicks four weeks old which had shown symptoms of stupor during the preceding two weeks, with a mortality of 25 per cent. Recovery followed a change in feed. Since that time the disorder has been recognized in 40 specimen consignments comprising 175 chicks, 103 of which represented uncomplicated cases, 16 complicated by affections with *Eimeria tenella,* intestinal coccidiosis, non-specific ingluvitis or *Tetrameres americana;* 56 were histologically negative cases. The specimens came from an affected chick population of 49,800 having an average morbidity of 22.2 per cent. and mortality of 10.6 per cent., the mortality ranging from 0.1 to 42.8 per cent. Hearsay reports on the occurrence of this condition have also come from surrounding New England states. The disturbance was seen most frequently in Barred Rock chicks, probably due to the preference for this variety on the broiler market; it was observed also in Rhode Island and New Hampshire Reds and in White Leghorns. Comparatively large and fast-growing chicks between the ages of 14 and 59 days (average 32.5) appear to be the victims. As far as our records show, two well-known brands of feed were involved, the one 10, and the other 8 times; also home-mixed New England Conference rations, 9 times, and 8 other brands once each, making a total of 11 different feeds.

The symptoms varied from those of droopiness, as seen in the first case, to evidence of muscular incoordination, resting on the pelvic bones with a tendency to fall backwards, retraction of head, somersaults and rotation in a lateral prone position. Coarse tremor was observed occasionally in the legs. The condition was afebrile and ordinarily of rapid onset.

On gross and microscopic study of uncomplicated cases, no abnormalities could be detected in the liver,

[1] A. M. Pappenheimer and M. Goettsch, *Jour. Exp. Med.*, 53: 11, 1931.

[2] M. Goettsch and A. M. Pappenheimer, *Jour. Biol. Chem.*, 114: 673, 1936.

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.

ERWIN JUNGHERR

STORRS (CONN.) AGRICULTURAL EXPERIMENT STATION

[3] E. Jungherr, *Poultry Sci.*, 15: 415, 1936.

[4] G. L. Dunlap, *Jour. Am. Vet. Med. Ass.*, 80: 880, 1932.

[5] O. Seifried, "Lehrgang der Histopathologie," Berlin, J. Springer, 1934.

[6] L. C. Norris, G. F. Heuser, H. S. Wilgus and A. T. Ringrose, *Poultry Sci.*, 10: 93, 1931.

[7] E. E. Jones, *Jour. Exp. Med.*, 59: 781, 1934.

[8] A. M. Pappenheimer, L. C. Dunn and V. Cone, *Storrs Agr. Exp. Sta. Bul.* 143, 1926.

[9] D. Dam, *Biochem. Jour.*, 29: 1273, 1935.

[10] G. L. Dunlap, *loc. cit.*

BOOKS RECEIVED

Actualités Scientifiques et Industrielles. *No. 332, Analyse des Matières Cristallisées. Au Moyen des Rayons X.* M. E. NAHMIAS. Pp. 41. 16 figures. 4 plates. 15 frs. *No. 338, Exposés sur la Physique des Solides. II, Le Champ Moléculaire dans les Diélectriques. (Le Sel de Seignette).* I. V. KOURTSCHATOV. Pp. 46. 28 figures. 12 frs. *No. 334, Atomistique. VII, Spectres de Vibration et Structure des Molécules Polyatomiques.* M. RADU TITEICA. Pp. 61. 8 figures. 18 frs. Hermann & Cie, Paris.

BOWDEN, JOSEPH. *Special Topics in Theoretical Arithmetic.* Pp. vi + 217. Illustrated. Author, Garden City, New York.

CURIE, MADAME PIERRE. *Radioactivité.* Pp. 563. 170 figures. 26 plates. Hermann & Cie, Paris. 150 frs.

HERMANN, LOUIS G. *Passive Vascular Exercises and the Conservative Management of Obliterative Arterial Diseases of the Extremities.* Pp. xx + 288. 80 figures. Lippincott.

Report of the Secretary of Agriculture, 1936. Pp. iv + 115. U. S. Government Printing Office, Washington.

SHRYOCK, RICHARD H. *The Development of Modern Medicine.* Pp. xiv + 442. Illustrated. University of Pennsylvania Press. $4.00.