basis of Donaldson's values. The study here reported is based on observations on 125 animals.

The rats were given *ad libitum* a "synthetic" ration consisting of casein* 18 per cent, raw corn starch 51 per cent, lard 22 per cent, cod liver oil 5 per cent, salts[1] 4 per cent, and in addition 300 mg. dried yeast daily. This diet has been shown repeatedly to be adequate for maintenance and growth.

As may be seen from the chart, there is a rapid increase in the compensatory enlargement of the remaining kidney within the first 3 weeks. At this time the left kidney is about 20 per cent heavier than the control. From the 21st day to the 120th day there is a steady increase at a slower rate (approximately 3 per cent in 10 days) until the enlargement has reached 46 per cent of the control value. From the 120th day to the 150th day our data show no significant increase in size of the remaining kidney, which suggests that the limit of enlargement may have been reached in 120 days.

The enlarged kidneys have shown no gross or microscopic evidence of an anatomical injury.

3058

Chondrodystrophia in chicken embryos.

W. LANDAUER and L. C. DUNN.

[*From the Storrs Agricultural Experiment Station, Storrs, Conn.*]

During the routine examination of chick embryos which had died during incubation, we found in 1923 several embryos exhibiting a striking abnormality resembling the condition known in mammals as *Chondrodystrophia foetais* (Kaufman).* As far as our knowledge of the literature reaches, chondrodystrophia is here reported for the first time in bird embryos.

* The "washed" casein used contained 13 per cent nitrogen. The protein furnished approximately 13 per cent of the dietary calories.

[1] Osborne, T. B., and Mendel, L. B., *J. Biol. Chem.*, 1919, xxxvii, 557.

* Earlier terms used to designate the same condition are *achondroplasia* (Parrot) and *Micromelia chondromalacica* (Kirchberg-Marchand).

The abnormal embryos were found first in the eggs from one fowl, but subsequently some 50 embryos of the same type have been found among about 4,000 embryos from several strains and varieties of fowls which have been examined. Inheritance may play some part in the causation of this abnormality, although our evidence is conclusive that it is not a simple Mendelian trait. Such embryos (at least those showing this malformation in a recognizable degree) have never hatched. Embryos dying as early as the 12th to 14th day of incubation have shown some of the extreme characters of the chondrodystrophic condition. Most frequently the abnormal embryos die near the end of the incubation period (18th to 20th days), while some have been found still living, but unable to emerge from the shell on the 23rd day of incubation.

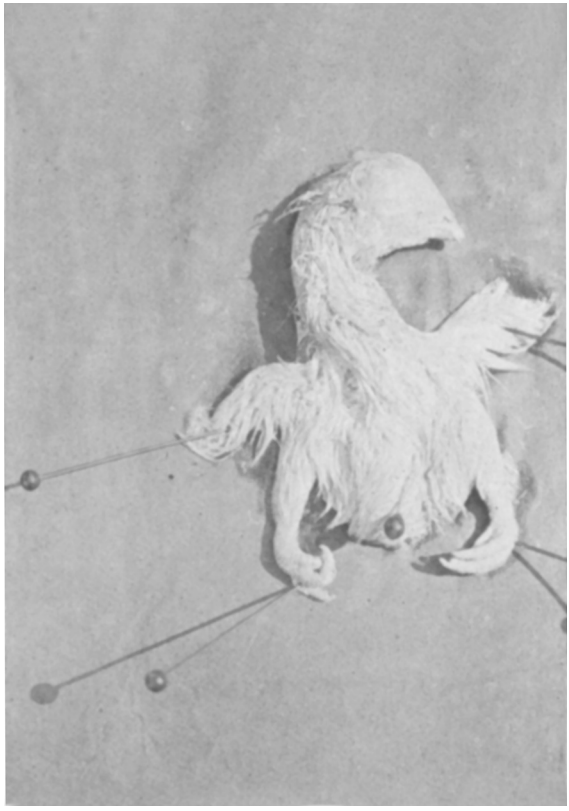

FIG. 1.

The external morphology of these embryos, and, as far as our observations go, the histological structure of the cartilage and of the bones as well, resemble very strikingly the characteristics reported for mammalian chondrodystrophia. A typical chondrodystrophic embryo, nineteen days old, is shown in Fig. 1. A comparison of this embryo with a normal embryo of the same age (Fig. 2) shows at once the extraordinary shortening of the legs and the abnormal conformation of the head. In the most extreme cases the legs scarcely protrude beyond the body, the plantar surface is directed towards the body, and the legs are bent towards the body, but are too short to reach each other. All the leg bones are shortened and thickened, but the tibia apparently is affected to a greater degree than the other bones. Even externally

FIG. 2.

the tibia shows in most cases a distinct bending. At this place a triangular plate of bone can often be seen on the inner side of the legs, protruding in a sharp angle towards the outside.

The second striking trait of these embryos is the conformation of the head. The base of the skull apparently is shortened, the parietal and the frontal bones are displaced forward, and the upper jaw shows a marked prognathism. The protruding upper beak is bent downward, giving the embryo a parrot-like appearance. The wings seem to show little or no abnormality. Curvatures of the spinal column have been observed in early chondrodystrophic embryos. In other respects the embryos seem to be normally developed, although the general growth is probably somewhat retarded. Besides the most typical cases we have found a greater number of slighter degrees of this malformation varying to an almost normal appearance. In some of these cases the head seems to be normal but the legs much shortened; in others even the legs do not show a very striking shortening.

Only the bones of the leg have thus far been studied histologically. Although there is considerable variation in the histological picture, yet it shows regularly many of the features found in the bones of chondrodystrophic mammals. The chief departures of the avian from the mammalian type of chondrodystrophia apparently are due to the differences in normal histogenesis in the two classes. The main histological features of the chondrodystrophic leg bones are as follows: The cartilage of the epiphyses shows many irregularities. The perichondrium frequently is thickened. The number of cartilage cells usually is decreased greatly in the peripheral layers of the epiphysial cartilage. The cells are enlarged, and have large cartilage-capsules, in this resembling the cartilage cells of deeper layers of the normal epiphysis. The cells, or if present, the cell-capsules, frequently are contiguous and flatten each other. As a consequence of this fact, little or no matrix is present. The zone of flattened cells, typical for the epiphyses of birds, usually is entirely missing, or only small parts of it are left, in which the arrangement of the rows of cartilage cells is very irregular. An invasion of connective tissue into the epiphysis usually can be seen in the region where the zone of flattened cells normally is situated. The fibrous connective tissue comes mostly from the periost of the inner side of the bone. This periosteal tissue interrupts the longitudinal growth of the bones. In the same region long bow-shaped vessels which in

some cases interrupt the epiphysis for a long distance frequently can be seen. There is probably a causal relationship between these vessels and the invasion of periosteal tissue. The rate of ossification is advanced on the side on which the connective tissue enters the epiphysis. In some cases the invasion of connective tissue from other places than the one stated above has been observed. The formation of columns of calcifying cartilage cells is disturbed. The epiphyses as a whole frequently although not regularly are much increased in size, at times showing a mushroom-like appearance. The periosteal bone reaches far into the epiphysis and surrounds the epiphyseal cartilage like a funnel.

The rate of ossification of the diaphysis is normal or advanced; the ossification itself is increased. The diameter is greater than normal. The tibia shows always, and usually in the same region, a striking bending towards the inner side of the leg. Femur and metatarsus too, frequently are bent, although in a slighter degree. At the place of bending, the trabeculae of the diaphyseal bone show a new arrangement in response to the bending (functional structure). The degree of bending probably depends upon the stage of development at which the formation of the cartilage at first was disturbed, the earlier embryos which we found dead in the shell having a greater bending than the later ones. The bending takes place in a cartilaginous stage of the extremities. The connective tissue seems to enter the epiphysis only in later stages; that is, after the bending of the bones has already taken place. Marrow is present, although frequently reduced in amount. The number of cells in the marrow is increased. Osteoclasts are rare. The number of erythrocytes in the marrow is decreased. A conspicuous alveolate structure fills the place between the marrow cells.

As in human material, the histological appearence of the epiphysial cartilage is very variable, frequently even the two epiphyses of one and the same bone showing considerable differences. The abundance of cartilage matrix in certain epiphyses, the entire absence of the matrix in other epiphyses even of the same embryo, and other observations not mentioned in this preliminary report probably will make it impossible to range the chondrodystrophia of chicken embryos in one of the three classes *(Chondrodystrophia foetalis hypoplastica, hyperplastica,* and *malacica)* which Kaufmann established for human embryos. A complete study of the histology of the skeleton of these embryos is in progress.