his first series of tumor transplantations the author obtained well growing tumors after injection of cystic tumor-fluid into rats. In such cases one or very few cells must have given rise to the tumor growth, and these tumors developed in a few cases quite rapidly. Such an explanation is, therefore, improbable. Further, we would have to consider the possibility that the means employed to decrease the virulence of tumor cells are favorable to the growth of bacteria, and that they inhibit in this way the development of tumors. It is certain that bacterial toxins frequently act unfavorably upon the growth of tumors. Against this explanation, however, the objections can be raised that tumors with experimentally diminished virulence did not show any sign of putrefaction, nor did they, after inoculation, cause a formation of abscesses, occurrences which are frequent after transplantation of infected material.

It is, therefore, most likely that the cause of this decrease in virulence is the result of the direct decrease of the vitality of the tumor cells as expressed in their energy of growth. It is, however, desirable to further analyze these facts in future experimental work on tumors, especially as the character of such work necessarily limits greatly the number of experiments a single observer can make. With this restriction it may be stated that the observations here recorded point to the conclusion that it is possible to cause an experimental increase or decrease in the energy of tumor growth, that these variations may be caused by a direct stimulating or depressing influence upon the tumor cells, and that such a stimulating effect may be cumulative.

36 (82). **"Demonstration: Photographs and plumage-charts of hybrid poultry,"** with remarks: **CHARLES B. DAVENPORT.**

Dr. Davenport exhibited photographs and plumage-charts of four hybrids between different races of poultry, and also of their parents, and remarked on the nature of the inheritance illustrated by each example.

37 (83). **"Experimental cirrhosis of the liver": RICHARD M. PEARCE.** (Presented by **EUGENE L. OPIE.**)

The experimental studies upon which this communication is based were suggested by an investigation of the necrosis produced

in the liver of the dog as the result of injecting hemolytic immune sera of high hemagglutinative power.[1] These necrotic lesions, which are due apparently to an obstruction of the circulation by thrombi composed of fused red blood-corpuscles, vary in position and extent, according to the dose of serum administered. Small doses cause more or less isolated lesions which may occupy any portion of the lobule; large doses produce a diffuse necrosis which spares only the tissue about the larger portal spaces. The uniformity of this necrotic lesion suggested the importance of a study of the repair process which would naturally follow in animals recovering from the acute toxic effects of the injected serum. The extent of the destruction precluded complete regeneration of liver parenchyma, and if the defect was repaired by connective-tissue proliferation, the resulting histological picture would be, except for a difference in the relation between the new tissue and the remainder of the lobule, analogous to cirrhosis in man.

Methods. — Dogs were injected either in the smaller branches of the femoral vein, or in the abdominal cavity, with serum obtained from rabbits which had received repeated injections of red blood-corpuscles of the dog. The dose usually employed was 1 c.c. of serum to from 500 gm. to 800 gm. of body-weight, and the animals were killed at intervals varying from 48 hours to 36 days

Results. — A majority of the animals die after intervals varying from 4 minutes to 48 hours. In those surviving, hemoglobinuria was a constant phenomenon usually appearing within 18 to 24 hours, persisting 3 to 4 days, and followed for several days by the presence of bile pigment in the urine. The first evidence of repair was mitosis of the liver cells lying at a slight distance from the necrotic areas. The earliest period at which this was seen was 38 hours after injection. At 48 hours the poliferation of endothelial and connective-tissue cells was evident, and this increased so rapidly that by the fifth day the necrotic tissue was largely replaced by young granulation tissue in the midst of which dividing liver cells could be found in considerable number. The young tissue later assumes a more fibrous appearance, the new blood-vessels become prominent, and newly formed bile ducts appear in the midst of the stroma. A development of liver cells from these

[1] *Journal of Medical Research*, 1904, xii, 329.

new bile ducts is readily demonstated. Multinucleated liver cells containing four to twelve nuclei are very abundant in the late stages. An interesting pheomenon is the englobing and removal of the hyaline remains of necrotic liver cells by large multinucleated masses of protoplasm. These giant cells, essentially foreign body giant cells, are derived in part from endothelial cells, but many have all the characteristics of true hepatic cells and are, undoubtedly, multinucleated liver cells with phagocytic properties.

The oldest lesion obtained (thirty-sixth day) presented an appearance analogous in histological structure to early cirrhosis as seen in man, differing only in that the new connective tissue surrounded the island of liver tissue persisting about the portal spaces, instead of having a distinctly perilobular arrangement. Macroscopically, this liver was much firmer than normal, deeply bile stained, and had a finely granular surface. Thus we have a form of experimental cirrhosis affecting the liver in a diffuse but uniform manner, and more typical than any previously described in the literature.

The observations thus briefly outlined, while of importance in explaining the histogenesis of cirrhosis, and incidentally of various processes of repair in liver tissue, do not aid in the elucidation of the etiology of cirrhosis in man, nor do they explain the peculiar arrangement of the connective tissue in human cirrhosis. They demonstrate, on the other hand, however, that cirrhosis may follow extensive primary destructive lesions, a view not yet fully accepted, and thus support the contention of Kretz that cirrhosis is essentially the result of a series of repair processes following repeated injuries of liver parenchyma.

The earlier lesions closely resemble acute yellow atrophy of the liver in man and appear to be of considerable importance in explaining the pathogenesis of this process.

38 (84). **"Experimental arteriosclerosis": RICHARD M. PEARCE** and **E. McD. STANTON.** (Presented by **J. E. SWEET.**)

Within the past two years several French and German writers (Josué, Erb and others) have described under the various names of calcification, atheroma or arteriosclerosis, a lesion of the aorta of rabbits produced by the intravenous injection of adrenalin.