

growth of the tumors appeared to be the reduced caloric intake rather than the heptaldehyde. This was demonstrated by feeding stock ration to tumor-bearing mice in the small amounts consumed by the animals receiving 2% heptaldehyde. Under these conditions the growth rate of the tumors was also markedly restricted.

When 0.8% heptaldehyde was fed, food consumption was more nearly normal. This level approximates the amount fed by Strong. However, in our experimental animals the tumors grew at the same rate as those in the controls. The feeding of heptaldehyde failed to prolong the life of the tumor-bearing animals; in fact in several groups it appeared to hasten death (Table I). Nor did it alter the character of the tumors. Of 97 tumor-bearing mice treated, only one small spontaneous tumor regressed and the animal in question died shortly thereafter. Heptaldehyde also failed to inhibit the growth of the Flexner-Jobling rat carcinoma. The discrepancy between our results and those of Strong demonstrate that heptaldehyde is not a universal tumor inhibitor.

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Production of Cirrhosis in Fatty Livers with Alcohol.*

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It was previously shown that depancreatized dogs develop cirrhosis of the liver when maintained with insulin and a diet containing meat, sucrose, bone ash and vitamins. The cirrhosis was observed as early as 2.6 years after pancreatectomy and was preceded by the infiltration of massive amounts of fat, the latter appearing as a rule within 20 weeks after excision of the gland. It was concluded that the scar tissue developed in response to the presence of the large amounts of fat in the liver. In the present study cirrhosis in normal dogs is shown to occur when the feeding of large amounts of alcohol is superimposed upon a previously established fatty liver.

For 30-35 days each dog received daily 10 g lard and 7 g lean meat per kilo. Vitamin sources and Cowgill's salt mixture were fed throughout the experiment. At the end of this period, the adminis-

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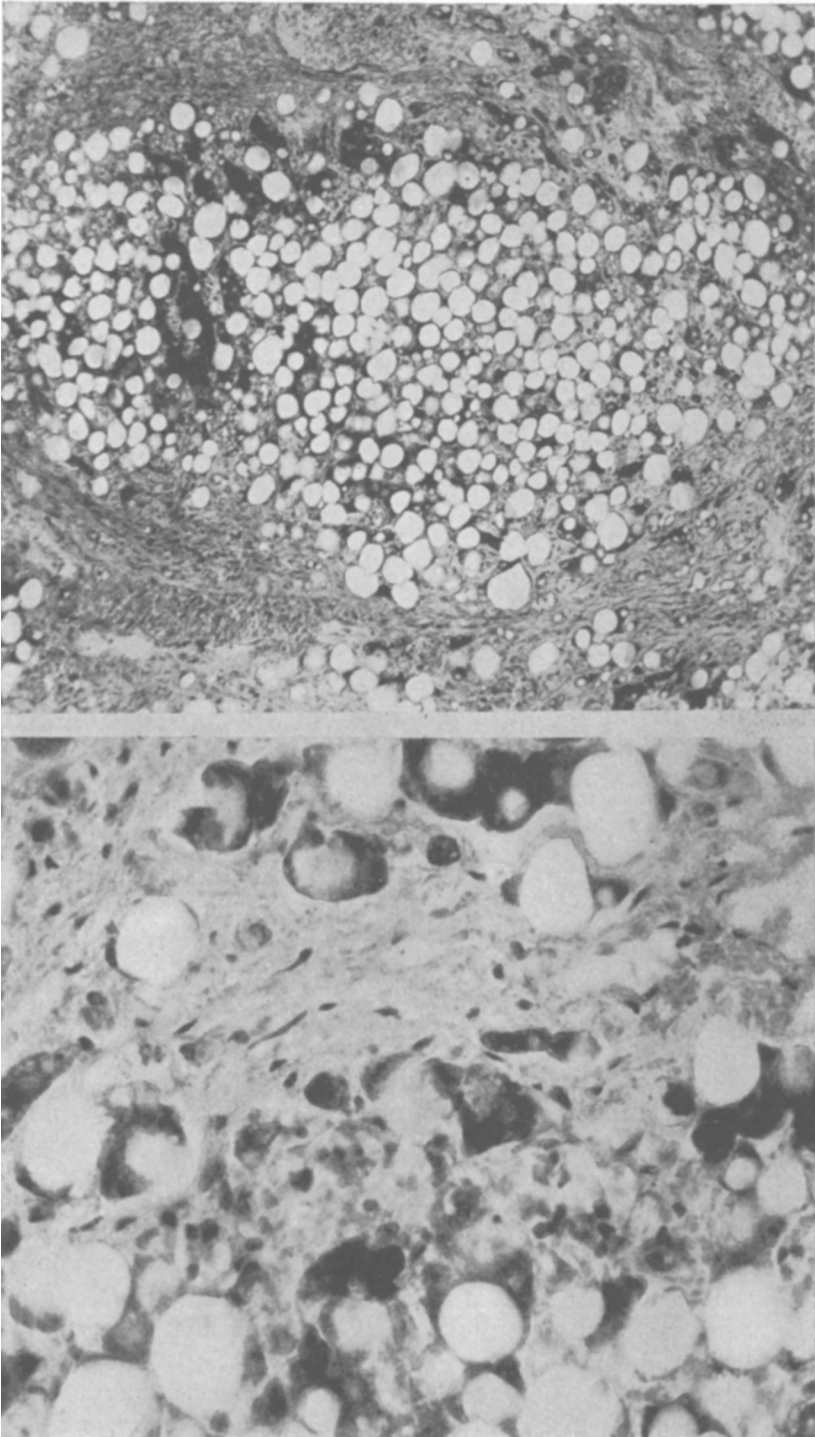
tration of alcohol was begun and the diet changed to approximately 30 g of lean meat. Ten cc of 22.5% alcohol per kilo were administered twice daily but the amount was varied somewhat with the response of the dog. The animals became comatose as a rule within an hour after the alcohol administration. The period of alcohol feeding was not unduly prolonged, 4-7 days of alcohol feeding being alternated with a similar period of fat feeding.

At various intervals after the alcohol treatments were begun, the animals died or were sacrificed and the tissues subjected to histological examination. Sixteen alcohol-treated dogs have so far been studied and the liver findings on these may be summarized as follows: 1. Excessively fatty livers were present in all animals, which resembled those found in chronic severe alcoholism in man. 2. A number of these fatty livers were greenish in color and had intrahepatic obstruction. Atrophy of liver cells was found at the periphery and in these cells hyaline degeneration was observed. 3. Four of the 16 animals showed definite cirrhosis, moderate in degree, but in all ways resembling that found in early fatty cirrhosis in the liver of man.

A typical protocol follows:

Dog F14 weighed 13.3 kilos at the start of the experiment, December 6, 1937. On this day the feeding of the high fat diet was begun and continued until January 10, 1938, at which time it weighed 14.6 kilos. The alcohol feeding was now started and the diet changed to the high protein diet which was supplemented with salts and vitamin sources. Approximately 260 cc of 22.5% alcohol were administered daily and this was increased slowly to 320 cc. In the course of 3 weeks the weight increased to 15.2 kilos, when anorexia developed. For the next 3 weeks alcohol administration was intermittent, partly because its effect appeared to be greater, and partly because of the poor condition of the animal. In another 3 weeks the weight dropped to 13.5 kilos and alcohol was omitted from the diet for several periods, the longest being 7 days. The alcohol kept the animal drunk much of the time and part of the time so sick that he vomited frequently. Ten days before death it was noted that the 2 large superficial epigastric veins on the lower abdomen were markedly dilated. The animal died March 21, 1938, *i.e.*, 106 days after the beginning of the experiment.

Autopsy: Weight 13.5 kilos, weight of liver 980 g. There was no ascites but the intestinal tract was filled with bloody material. The liver was mottled in appearance with very large lobules, some of which measured 3 mm in diameter. Fatty and fibrous streaks ran through the liver which was quite friable. Blocks were taken for



FIGS. 1 AND 2.

Low power photomicrograph showing a representative liver lobule surrounded by proliferating fibrous tissue, and (below) higher power showing fatty and coagulative degeneration of liver cells, atrophy of cells, and fibroblastic reaction.

histological examination and the remainder ground up for fat estimation. (Total fatty acid, 30%.)

Microscopic Description: The liver shows extensive fatty infiltration. This is central in location, although scattered patches are present in different portions of some lobules. Many cells which do not contain much fat are undergoing some rather rapid degeneration of a coagulative hyaline type. Around the periphery of many lobules, cells have become atrophied and have been caught in a meshwork of proliferating fibrous tissue. This has proliferated around lobules forming a characteristic retaining wall. In other lobules it ramifies throughout, breaking the normal lobule up into several smaller lobules. This is better seen in phosphotungstic acid hemotoxylin sections where the picture of cirrhosis of the liver is reproduced. Rather wide bands of fibrous tissue surround the lobules and pass through lobules to connect others and form islands of fatty liver. The picture is that of a well advanced fatty cirrhosis such as can be duplicated by many similar cases called alcoholic cirrhosis in man. (Figs. 1 and 2.)

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Pseudopregnancies from Electrical Stimulation of the Cervix in the Diestrum.*

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The artificial induction of a pseudopregnant state in the rat by means of electrical or mechanical stimulation of the cervix uteri is a common laboratory procedure. It has long been supposed that these stimuli to be effective must be applied at or near the time when the female is in a sexually receptive mood (*i. e.*, late proestrus or estrus). We wish to report some observations on the application of an electrical stimulus to the cervix of adult female rats during the diestrous phase of the cycle.

The cervix was exposed with a speculum and the electrodes, spaced at 2 mm, were applied at any place on the body of the cervix. The stimulus, a faradic current taken from an induction coil, was moderately intense and always produced convulsive contractions in the hind quarters. The stimulus was applied continuously over a period of 5 to 10 sec.

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