cent of the total dietary) was added to the new balanced dietary given above. These rats have died in less than one-half the time of the rats on a minus vitamin A dietary (Curve M-7, Fig. 1).

The rats in all the cages, excepting where the very small doses of mineral oil were given, died without showing any xerophthalmia. In Cage 7, two rats showed for a short time before death the narrowing of the pupils and clouding of cornea, which is typical of this affliction.

As is evident, therefore, the mineral oils, quite different from the vegetable oils, not only remove the fat soluble vitamins from the food, but produce other toxic effects as well. From our studies of the effects of these oils on the tissues and in the production of cancer, it may be assumed that their toxic effect is due to the fact that they act on the mucous membrane of the intestines to further strip these tissues of the body of their fat soluble vitamin reserve.

This is a preliminary report.

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Studies on Fatty Infiltration of the Liver Induced by Lipoid Solvents.

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In a previous paper, Johnston and the author[1] reported that fatty infiltration of the liver occurs a certain period of time after the injection of a mixture of mineral oil and coal tar into the subcutaneous tissue of rats fed a diet rich in vitamin A. We thought at that time that this mixture of lipoid solvents alone produced the fatty infiltration in these animals. More recent studies have shown that

this is not true. It was a time factor only which brought about the action of the mixture of mineral oil and coal tar. Later experiments have shown that other lipoid solvents will produce the same effect in the liver after a longer interval of time. A mixture of coal tar and paraffin produces the change 30 days after the injection, coal tar in 90 days, mazola oil in 60 to 90 days, mineral oil in 45 to 60 days, and a mixture of coal tar and mazola oil in 40 to 60 days. Thus, the time of occurrence of the fatty infiltration is governed by the particular kind of lipoid solvent used. At the times given above there is an extensive infiltration involving the entire parenchyma of the liver. Previous to this time in certain of the above series a few scattered areas of vacuolization were found in a few of the livers.

The infiltration does not occur in the animals fed a ration deficient in vitamin A, and only to a slight degree in the animals fed a well balanced stock ration. It does not occur in rats fed a rich vitamin A dietary that are not injected with the substances listed above. These substances are lipoid solvents. It is in a system saturated with ergusia (vitamin A) that these lipoid solvents induced the transportation of fat to the liver. As Burrows[2] has shown with the tissue culture, the migration of fat is a simple process. The fat droplet migrates from an area poor to one rich in ergusia. In these terms fatty infiltration can be understood as being due to a disturbance in the ergusia balance of the system induced by the introduction of a lipoid solvent into it. The same amount of fat is included in the rations deficient in vitamin A, but the only ergusia present in these animals is that stored by the body previous to the beginning of the deficient vitamin A diet.

It is well known that chloroform causes a necrosis of the liver. Certain authors have reported cases of fatty infiltration following chloroform inhalation. The protective action of a high carbohydrate, low fat dietary against chloroform poisoning has been emphasized by many authors.[3] In our studies we find that chloroform (2 cc. subcutaneously or a small amount by inhalation) causes an extensive necrosis of the liver within 7 days, and on a fat diet deficient in carbohydrate, the lesion occurs within 24 to 36 hours. With the dietary high in carbohydrates the lesion is less extensive and a certain number of the livers show a vacuolization associated with the necrosis. However, this vacuolization occurs in areas that have undergone a certain degree of necrosis. The cells in these areas are granular and the nuclei are centrally placed. In the animals injected with the lipoid solvents mentioned above, the nucleus is peripherally placed as if pushed to one side by the fat taken up by the cell. The

cells are not granular. This does not mean necessarily that chloroform acts differently, but probably only more drastically than coal tar, paraffin and mazola oil, or mixtures of them.

This is a preliminary report.

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Contractures Caused by Tenotomy and by Tetanus Toxin.

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The term, contracture, is here used to designate a state of permanent shortening of a muscle which persists after section of the motor nerve or after death of the animal. Since under these conditions no nerve impulses can reach the muscle it is obvious that when once developed this sort of shortening is independent of the nervous system and is due to a more or less permanent alteration of the muscle. These contractures develop too quickly to be due to fibrosis and histological sections show no increase in connective tissue. The muscle fibers themselves have acquired a new and shorter length.

When a resting muscle is made to support a moderate load it undergoes in addition to the elastic stretch, which disappears promptly when the load is removed, also a more or less permanent elongation (Nachdehnung of Blix,[1] permanent deformation of Langelaan[2]). In the contractures caused by tendon section and by tetanus toxin the elasticity remains approximately normal but the resistance to permanent elongation is greatly increased.

In addition to its action on the spinal cord, tetanus toxin also exerts a direct action on muscle, causing a shortening and increased resistance to permanent elongation when injected after section of the motor nerve.

This is a preliminary report.

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