reactions depend upon the metabolism of the living tissue and furthermore that it is possible to dissociate through graded heating the activity and the life of the cartilage from that of the perichondrium.

The results are entirely different in heterotransplantation of cartilage. While here the untreated cartilage tissue may remain alive up to 28 days (Loeb and Harter[3]), even the lowest intensity of heating employed in my experiments killed the cartilage and the surrounding fat tissue. Yet the reaction of the host against the heterotransplants remained very active; lymphocytes and polymorphonuclear leucocytes as well as connective tissue surrounded and invaded the transplants and the intensity of these reactions on the part of the host were very strong. We may then conclude (1) that heterotransplanted tissue is injured through heating more markedly than homoiotransplanted tissue, owing to a summation of the injurious effects of heating and of heterotoxins and (2) that in contradistinction to homoiotoxins the production of which depends upon living, metabolizing tissue, heterotoxins may also be active even in necrotic tissue.

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Hepatic Lesion in Eclampsia (An Experimental Study).

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The hepatic lesion in Eclampsia in humans is peculiar in that it does not occur in any pregnant animal or in any other human disease. The lesion is essentially a thrombosis occurring in the capillaries of the portal vein in the periphery of the liver lobule with hemorrhage into the adjacent tissues, resulting in necrosis. It is our belief that whatever the substance is which causes this thrombosis, it must be in greater concentration in the portal system.

From clinical experience we know that curtailment of protein together with intestinal elimination will in almost all cases prevent Eclampsia. We also know that Eclampsia does not occur unless the woman is pregnant, that is, living chorionic villi must be present. The fetus may be dead for true Eclampsia has occurred with Hydatidiform Mole.

In late pregnancy placental fragments are constantly entering the

[3] Loeb, Leo, and Harter, J. S., *Am. J. Pathol.*, 1926, ii, 521.

blood stream. This placental tissue is rich in tissue fibrinogen, a blood coagulant found in tissue extracts, which must be neutralized or destroyed by substances in the blood.

Mills[1] has demonstrated that tissue fibrinogen, a cephalin-protein complex can pass through the intact intestinal wall and shorten the clotting time of the blood. If this is true it seemed to us that we could explain the production of the hepatic lesion of Eclampsia as follows: If too much of the neutralizing substance is used up by the placental tissue, it is conceivable that tissue fibrinogen from meat, by mouth, requiring the same detoxifying substance, could reach such a concentration in the portal system that thrombosis would occur.

We first wished to see if we could produce portal thrombosis. We used tissue fibrinogen prepared by the Merrel Drug Company according to Mills'[2] directions. Thus we ruled out the possible effects of other substances which are found in tissue extracts. In order to get as great a concentration as possible in the portal system we made use of the Blankenhorn cannula, through which we could daily inject tissue fibrinogen into the portal vein, at the same time injecting the same substance in the peripheral circulation, thus simulating pregnancy. Doses of 2 to 5 ml. were used peripherally and 3 to 12 ml. for the portal. Eight dogs were handled in this way, 3 of which showed lesions typical of Eclampsia. The remainder all showed marked portal vein thrombosis and hemorrhage into the liver tissue, but only to some extent from the periphery of the lobule. The fact that marked thrombosis occurred was due to over dosing resulting in coagulation of the blood in the larger branches of the portal vein, which, therefore, interfered with the production of hemorrhage. This work demonstrated that tissue fibrinogen used in this way in proper doses could cause portal coagulation and produce a liver lesion similar to that of Eclampsia.[3]

The present report is based on the combination of the peripheral injection and oral feeding of tissue fibrinogen. Seven dogs were fed fibrinogen in 3 ml. doses through a stomach tube and at the same time injected 1 to 5 ml. peripherally. These experiments varied from 3 to 7 days in duration. In 7 cases, 3 showed liver hemorrhage in the gross, similar to that in Eclampsia, 4 showed beautiful Eclamptic lesions and 6 showed portal vein thrombosis within the liver. One was entirely negative.

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[1] Mills, C. A., *Am. J. Physiol.*, 1923, lxiii, 484.

[2] Mills, C. A., *J. Biol. Chem.*, 1923, lv, 17.

[3] Reported before the American Association of Obstetricians and Gynecologists, Toronto, 1928.

The fact that limitation of protein prevents the development of Eclampsia in many cases and, furthermore, that a method of starvation and intestinal elimination gives the best results in curing the condition suggests strongly that substances which may be toxic to the pregnant woman are absorbed from the intestinal tract.

In this report we are primarily concerned with the hepatic lesion of Eclampsia; but it may be noted briefly that some of the dogs had convulsions, tonic and clonic, and some became comatose. The development of these conditions depended on the dosage. Sections of the kidney in some animals showed lesions similar to those found in humans dying of Eclampsia.

Our findings, we believe, explain in a measure why the hepatic lesion in Eclampsia can be averted by limiting the protein intake of the patient in the last months of pregnancy.

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Correlation between Threshold and Conduction Rate in Myelinated Nerves.

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In observing the effects of stimulating a nerve in the body, it is possible to lead off the cut end of the nerve into the oscillograph and thus correlate the potential form with the functional result of stimulation. Since the different fibers of a nerve are stimulated at different threshold strengths, if the difference in thresholds of different fibers were known, the oscillograph or other potential recorder could be dispensed with except for an occasional observation of threshold for the most irritable fibers, and the experimental procedure thus simplified. With this end in view, we have examined the ratios between the threshold of the first fibers stimulated in a nerve and that of other fibers, taking as criteria the thresholds of the first fibers in the various potential waves which represent fiber size groups. Gasser and Erlanger[1] have shown that the conduction rates of different fibers tend to vary as the fiber diameters, and by correlating the thresholds and conduction rates of different waves it should be possible to find the relationship between threshold and fiber size.

[1] Gasser, H. S., and Erlanger, J., *Am. J. Physiol.*, 1927, lxxx, 522.