

Effect of Graded Degrees of Heat Upon Cartilage in Homoiotransplantation and Heterotransplantation in Guinea Pig.

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In his series of transplantations Loeb concluded that the production of homoiotoxins depends upon the metabolism of the living transplants, whereas heterotoxins may be given off even by non-metabolizing, dead tissue.¹ He further had observed that through graded heating of homoio tissue it was possible to diminish its growth energy before the point is reached where the tumor cells are killed as the result of heating.²

In the following investigations we exposed xyphoid cartilage of the guinea pig to graded intensities of heat before transplanting it into subcutaneous pockets of other, non-related guinea pigs. Thus we tested the effect of heating on homoiotransplanted cartilage. In other experiments we treated rat cartilage in a similar way, before transplanting it into the guinea pig and thus tested the effect of heating on heterotransplanted tissue. In both series we exposed the cartilage to temperatures which in different experiments varied between 43° and 51° and the time of heating varied between 15 and 45 minutes. In every case the transplant remained in the host for a period of 20 days. In the case of homoiotransplantation we found that heating the cartilage to 47° C. for 30 minutes represents the critical point. Tissues exposed to a lower intensity of heating remained alive and elicited the typical lymphocytic reaction on the part of the host tissue; the connective tissue reaction was also present. Transplants exposed to greater intensities of heat were killed and no longer elicited lymphocytic reactions. At about the critical point the cartilage proper became necrotic after transplantation, while in a number of cases the perichondrium remained alive and produced new cartilage and the surrounding fat tissue was still preserved. At this point the lymphocytic reaction was much diminished or lacking altogether and the connective tissue reaction also was absent or much diminished. We may then conclude that in homoiotransplantation the lymphocytic and connective tissue

¹ Loeb, Leo, *J. Med. Research*, 1918, xxxvii, 353; *Biol. Bull.*, 1921, xl, 143.

² Loeb, Leo, *J. Med. Research*, 1902, viii, 44; *Virchow's Archiv.*, 1903, clxxii, 345; *Am. Med.*, 1903, v, 412, 1905, x, 265.

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 Harter³), 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.