1811. PATHOLOGIC CHANGES FOLLOWING EXPOSURE TO A SYNTHETIC LUBRICANT CONSIDERED FOR USE ABOARD SUBMARINES. M. E. Tracht,* R. Bland Williams, Jr., J. F. Failing, Jr.* and E. L. Elworth.* Naval Med. Research Inst., Bethesda, Md.

The lubricant contained 67% by weight of 2-ethylhexyl diphenyl phosphate and 23% chlorinated diphenyls. Rats and hamsters were exposed for 1, 19 and 50 days to an aerosol of about 13 μg/l. of the lubricant with a flow rate resulting in 2 complete changes of air per min. An increase in liver-weight body-weight ratio, hepatic fatty metamorphosis, and increased susceptibility to a second hepatotoxin were demonstrated despite normal weight gain, appearance and 100% survival. 2-Ethylhexyl diphenyl phosphate is used in the manufacture of food wrappers, shower curtains and wearing apparel and has been considered innocuous. Applied to the skin of rats equal volumes of this substance and the chlorinated diphenyl compound produced the same degree of increase in liver-weight body-weight ratio, even in starved animals, while inanition controls lost 50% of their liver weight. Both substances produced acneiform lesions, but hepatic fatty metamorphosis and fatalities following skin application of 1.5 cc to rats occured only with the chlorinated diphenyl mixture. Effects following oral or subcutaneous administration of the lubricant or either of its principal components were less marked than after inhalation or direct skin application. Quantitative studies on the composition of the liver will be presented.

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1812. AN ENTITY RESEMBLING HOMOLOGOUS DISEASE INDUCED BY TRANSPLANTATION OF LYMPHOID TISSUE IN UNIRRADIATED MICE. John J. Trentin (intr. by A. Kirschbaum). Baylor Univ. College of Medicine, Houston, Texas.

Mice protected against otherwise lethal doses of x-irradiation by homologous bone marrow (from an unrelated strain of mice) are thereby rendered tolerant of the injected marrow elements, as well as skin grafts, from the donor strain. There follows a secondary wasting disease, without marrow depression, that often results in death. This "homologous disease" is not seen in mice protected with isologous marrow (from the same inbred strain). There is evidence that the secondary disease may result from an immune reaction of the transplanted homologous marrow elements (lymphoid) against the tolerant host (Proc. Soc. Exper. Biol. & Med. 96:139, 1957). If this were so, then irradiation is an essential component of the disease only insofar as it renders the host tolerant, and a similar disease should be obtained by transplantation of homologous lymphoid elements into unirradiated, genetically tolerant mice. The F_1 hybrid mouse is genetically tolerant of tissue grafts from either inbred parent strain (but not vice versa). Unirradiated F_1 hybrid mice were injected with spleen and lymph node from either or both parent strains (homologous) or from other F_1 hybrids (isologous). Some combinations of parent lymphoid tissue into unirradiated F_1 hybrids caused wasting disease and death in up to 80% of the mice. No such entity was observed in controls receiving isologous lymphoid tissue.

1813. SYNERGISM AND ANTAGONISM OF HYPERCHOLESTEREMIA AND HYPERVITAMINOSIS D IN EXPERIMENTAL ATHERO-ARTERIOSCLEROSIS. Richard E. Trueheart,* George M. Hass, Ann Hemmens* and Marjorie Counts.* Presbyterian-St. Luke's Hosp., Chicago, Ill.

Permutation of hypervitaminosis D and dietary hypercholesterolemia in rabbits led to the following conclusions. Hypervitaminosis D produced medial degeneration and calcification with fibrous intimal proliferation. Hypercholesterolemia produced lipid intimal plaques. The distribution of disease in both conditions was similar in some arteries but not in others. When hypercholesterolemia was induced during early hypervitaminosis D, lipids accumulated rapidly and electively at sites of active fibrous intimal proliferation but not at sites of active medial calcification. When hypervitaminosis D was induced during well-established hypercholesterolemia, medial calcification and fibrous intimal proliferation in arteries ordinarily susceptible to both diseases were inhibited while lipid deposition became conspicuous in arteries ordinarily involved only by hypervitaminosis D. After calcific medial disease was well established, the superposition of hypercholesterolemia led to accelerated resorption of calcium from the media while overlying preformed fibrous intimal plaques often showed less susceptibility to lipid deposition than adjacent normal intima. All chronic forms of separate or combined arterial disease eventually showed a decreasing rate of progression with increasing time. This was due partly to reparative tissue reactions but mostly to undisclosed mechanisms which with increasing time created increasing resistance of the animal to undesirable effects of hypercholesterolemia and/or hypervitaminosis D. These observations may bear upon reasons for variations in athero-arteriosclerotic lesions in different people.

1814. BIOCHEMICAL ASPECTS OF THE PAPAIN INDUCED CHANGES IN CARTILAGE. Theodore I. Tsaltas (intr. by Lewis Thomas). Dept. of Pathology, New York Univ. College of Medicine, New York City.

The reversible morphologic changes observed in cartilage following the intravenous injection of papain in rabbits (L. Thomas, J. Exper. Med., 104:245, 1956) have been investigated from the biochemical point of view. Thus 24 hr after the injection of papain, the amount of mucochondroitin sulfate (M.C.S.) isolated from the ear cartilage was 35-40% lower than that in the control animals. The S35 activity in the isolated M.C.S. decreased by approximately 30%. The S35 activity in the organic and inorganic fractions of the serum increased considerably, the most marked being the increase in the organic fraction. Urinary excretion of glucuronic acid and S35 containing compounds increased in the same period manyfold. In a large series of experiments, animals were sacrificed at regular time intervals after the injection of papain in order to follow the rate of these changes. Thus the maximum depletion of M.C.S. from cartilage occurred 12 hr after the injection. This depletion was maintained for 24 hr, thereafter increasing gradually to normal levels after 60 hr. The S35 activity in the serum organic and inorganic fractions reached a maximum at the same time that the depletion of the cartilage M.C.S. was maximal. A very important change was observed in the Hexosamine:N2 ratio of the M.C.S. This increased markedly and reached the highest point 24 hr after the injection of papain and returned to approximately the normal levels 60 hr later.

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