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High Temperature Liver Death Syndrome.

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High temperature liver death has been an unexplained postoperative complication following cholecystectomy and bile duct surgery. Clinically there is an immediate postoperative rise in temperature. This elevation of temperature is rapid and progressive; reaching 107° to 109°F. (41.7°-42.8°C.), and death occurs in 48 hours or less. Postmortem examination of the liver shows passive congestion, disorganization of the cords of liver cells and widespread focal necrosis.

The nature of these lesions and the finding of hepatic arteries in close relation to the cystic duct in several recent operations upon the gall bladder suggested vascular damage as an etiological factor in this syndrome. To establish the possibility of such vessel injury at operation the hepatic vessels, the bile ducts and the gall bladders of 29 cadavers were dissected. In 16 of these 29 bodies the right hepatic artery was in such close relation to the cystic duct that it might easily have been injured during cholecystectomy.

Fourteen dogs were used to determine experimentally the clinical course and the liver changes following ligation of the hepatic arteries. Cholecystectomy was performed in all and at the same time dissections were carried out isolating the hepatic arteries. The largest artery was invariably ligated and in many, smaller vessels were also clamped. Six of the dogs showed immediate postoperative temperature rise to 104° to 105.8°F. (40° to 41°C.). Five of these animals died within 36 hours and one died during the fourth postoperative day. Examinations of their livers show diffuse nec-

rosis, disorganization of the liver cords and focal necrosis. These lesions are similar to those found in human livers after high temperature liver death. In the 8 dogs which did not die within 36 hours and which did not show typical elevation of temperature, injections of bismuth or starch mixtures demonstrated adequate collateral circulation to all lobes of the liver. The reaction to ligation of the hepatic arteries appears to be quantitative; if the liver is deprived of sufficient arterial blood the experimental animal develops the syndrome, high temperature liver death.

The essential lesion of this syndrome appears to be acute necrosis of the liver. Experimentally this lesion is produced by ligation of the hepatic arteries; clinically arterial occlusion may be due to thrombosis, embolism, or accidental ligation. On the basis of this work it is suggested that this postoperative complication be called acute postoperative necrosis of the liver.

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Decomposition of the Group A Substance in Horse Saliva by a Myxobacterium.

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The results of chemical investigation of preparations of the group A substance in horse saliva[1] which indicated that their activity may depend upon carbohydrate groupings suggested testing the effect upon the saliva substance, of a microorganism known to attack carbohydrates.

The discovery of a bacterium producing an enzyme which destroys the polysaccharide of Pneumococcus type III was made by Avery and Dubos.[2] Several other bacteria capable of splitting Pneumococcus polysaccharides have been found since by Sickles and Shaw.[3] While the action of the Avery and Dubos bacterium appeared to be specific, a Myxococcus which decomposes a variety of

[1] Landsteiner, K., *Science*, 1932, **76**, 351.

[2] Avery, O. T., and Dubos, R., *Science*, 1930, **72**, 151; *J. Exp. Med.*, 1931, **54**, 51.

[3] Sickles, G. M., and Shaw, M., *J. Inf. Dis.*, 1933, **53**, 38; PROC. SOC. EXP. BIOL. AND MED., 1934, **31**, 443; *J. Bact.*, 1934, **28**, 415.