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Effect of Mechanical Obstruction of the Hepatic Veins upon Guanidine Content of the Blood.

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Increase in the guanidine content of the blood has been reported in tetany following parathyroidectomy (Burns and Sharpe,[1] Koch,[2] and Paton and Findley[3]); in hypertension (Major and Weber[4]); in Laennec's cirrhosis of the liver and in Banti's disease (Ellsworth[5]); in eclampsia (Minot and Cutler[6]); and in poisoning with carbon tetrachloride and chloroform (Minot and Cutler[7]). In the last 3 of the above conditions there is an acute severe damage to the liver. Injury to the liver can be induced by mechanical obstruction of the hepatic veins. It appeared to be worth while, therefore, to compare the effects of injury to the liver induced by this method on the guanidine content of the blood with those obtained by Minot and Cutler in cases of liver injury caused by acute poisoning and eclampsia.

Under complete ether anesthesia and with rigid aseptic precautions we mechanically obstructed the hepatic veins by the method described by Simonds and Brandes.[8] Samples of blood were taken before the operation and 24, 48 and, in some instances, 72 hours after the operation. Guanidine was determined by the method described by Major and Weber.[9] The average of our normal determinations was 0.347 mg. of guanidine per 100 cc. of blood, with a maximum of 0.417 mg. and minimum of 0.303 mg. The figures given by Minot and Cutler[7] are the only determinations on normal dogs that we have been able to find. Their average was 0.374, with a maximum of 0.48 and a minimum of 0.29.

Our experiments involved a study of 16 dogs. Of these, 8 showed either a definite decrease or no change in the guanidine content of the blood; 4 showed increase of less than 0.1 mg. in 24

[1] Burns and Sharpe, *Quar. J. Physiol.*, 1916, **10**, 345.

[2] Koch, *J. Biol. Chem.*, 1912, **12**, 313.

[3] Paton and Findley, *Quar. J. Physiol.*, 1916, **10**, 202, 315.

[4] Major and Weber, *Arch. Int. Med.*, 1927, **40**, 891.

[5] Ellsworth, *Johns Hopkins Hosp. Bull.*, 1930, **46**, 296.

[6] Minot and Cutler, PROC. SOC. EXP. BIOL. AND MED., 1929, **26**, 607.

[7] Minot and Cutler, *J. Clin. Invest.*, 1928-29, **6**, 369.

[8] Simonds and Brandes, *Am. J. Physiol.*, 1925, **72**, 201.

[9] Major and Weber, *Johns Hopkins Hosp. Bull.*, 1927, **40**, 87; *Arch. Int. Med.*, 1927, **40**, 891.

hours with a return to normal in 48 hours; one showed a slight decrease in 24 hours with a return to approximately normal in 48 hours; while only 2 showed a definite increase (from 0.340 to 0.444 mg. and 0.340 to 0.466 mg., respectively). In each of these latter dogs peritonitis was present, and one of them being quite ill at the time it was sacrificed. One animal (No. 8) died 4 hours after the operation with symptoms of hypoglycemic convulsions; its blood sugar dropped from 104 mg. to 41 mg. and the guanidine from 0.409 to 0.337 during the 4 hours. All of these dogs showed liver injury on microscopic examination and most of them showed an increase in blood esterase.

From these experiments it appears that injury to the liver due to mechanical obstruction of the hepatic veins does not affect the guanidine content of the blood in the same manner as injury to the liver induced by acute poisons. It is suggested, although without any experimental basis, that the poisons used by Minot and Cutler may have injured the parathyroids as well as the liver.

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Skin Reactivity of Mothers and Infants to Staphylococcus Aureus Filtrate and Vaccine.

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The negative response to the Dick and Schick tests in the new born and young infant gave rise to the supposition that neutralizing antibodies were present. These antibodies were considered to be of transplacental origin at birth, and then later might be obtained from the breast milk. Further clinical support is found in the apparent lack of diphtheria, scarlet fever, or other infectious diseases in early infancy. However, the Dick and Schick tests are often negative in the newborn although their mothers may be definitely positive (Cooke,[1] Ruh and McClelland[2]). Furthermore, the blood serum of the young infant also lacks the antitoxins that might neutralize the toxin of the scarlet fever streptococcus (Cooke[1]) or the diphtheria

[1] Cooke, J. V., *Am. J. Dis. Child.*, 1927, **34**, 969.

[2] Ruh, H. C., and McClelland, J. E., *Am. J. Dis. Child.*, 1923, **25**, 59.