ments 3 and 4 did not prevent the nearly complete inhibition of photosynthesis by the high concentrations of cyanide, it did completely protect the photosynthetic mechanism against injury in .38 x 10^{-2} mol. KCN because recovery in pure sea water was practically complete and no bleaching of the pigments or loss of turgor occurred. Even in experiment 4 with a concentration of .76 x 10^{-2} the added CO_2 afforded almost complete protection except to one of the strips.

3119

Changes in the excretion of uric acid produced by experimental hepatic insufficiency.

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Destruction of uric acid, which is rapid and marked in the normal dog,[2] does not occur if the liver is entirely removed. Complete removal of the liver in the dog produces a very great increase in the uric acid content of the blood and tissues, and also in the urine. Uric acid injected into the dehepatized dog remains unchanged in the blood and tissues and is excreted unchanged in the urine. The destruction of uric acid in the dog seems to be entirely dependent on the presence of the liver,[1] since no uric acid is destroyed in the absence of the liver and no other means of influencing the destruction of uric acid has been demonstrated. Intravenous injection of standard amounts of uric acid into dogs with hepatic insufficiency is followed by a delay in the disappearance of the excess uric acid from the blood, and by an increase in the amount of uric acid excreted in the urine. Both the delay in the disappearance of the excess uric acid from the blood and the amount of uric acid appearing in the urine are greater, the greater the amount of damage or reduction of hepatic tissue. Two

[2] Folin, O., Berglund, H., and Derick, C., *J. Biol. Chem.*, 1924, lx, 361-471.

[1] Bollman, J. L., Mann, F. C., and Magath, T. B., *Am. J. Physiol.*, 1925, lxxii, 629-646.

objections should be raised to the use of injections of uric acid as a test of hepatic function. First, injections of large amounts of uric acid produce severe lesions in the kidneys and interfere with excretion; and second, lesions of the kidneys may produce retention of uric acid, although in the dog the presence or absence of the kidneys is without effect on the rate of disappearance of uric acid from the blood.

When a diet rich in purines is fed to the normal dog the excretion of uric acid may be measurably increased. Following an eighteen-hour fast, animals fed 175 gm. of fresh pancreas excrete from 80 to 160 mg. of uric acid during the twenty-four hours after feeding. The average amount of uric acid excreted by normal dogs on this diet is about 120 mg. Animals with definite hepatic damage show an increase in the amount of uric acid in the urine. Following ligation of the common bile duct the excretion of uric acid remains within normal limits for about four weeks. After the fourth week of obstructive jaundice the excretion of uric acid increases up to from 200 to 500 mg. of uric acid, following a diet of 175 gm. of fresh pancreas. There is no apparent relationship between the duration of obstructive jaundice and the excretion of uric acid, although the amount of uric acid excreted is roughly proportional to the estimated hepatic atrophy found on exploration or necropsy of animals with obstructive jaundice.

Animals with an Eck fistula also show a marked increase in the amount of uric acid excreted on a diet of pancreas. They excrete from 120 to 750 mg. of uric acid or an average of about 450 mg., the amount of uric acid excreted being roughly proportional to the amount of hepatic atrophy as estimated at exploration. However, there was no apparent relation between the duration of the fistula and the amount of uric acid excreted. Individual animals varied considerably with regard to the amount of uric acid excreted at different periods following the production of an Eck fistula, and some have approached normal after periods of rather high excretion of uric acid. The gross and microscopic appearance of the liver with an Eck fistula also varies considerably, and it appears that the amount of uric acid excreted increases with the damage of the liver.

Surgical removal of portions of the liver from animals with an Eck fistula is followed by only slight regeneration of hepatic tissue; thus we have been able permanently to reduce the

amount of hepatic tissue to only a small percentage of the normal. By careful dietary measures animals so treated may be maintained in apparently good condition for several years, and after a short time there seems to be but little change in the gross or microscopic appearance of the hepatic tissue. Following the ingestion of 175 gm. of fresh pancreas these animals excrete from 600 to 900 mg. of uric acid, or an average of about 700 mg. The amount of uric acid excreted was quite constant for the individual animals of this series, and increased in proportion to the reduction of hepatic tissue.

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The prenatal growth and natal involution of the human uterus.

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The human uterus undergoes a marked reduction in length and weight in the first few weeks following birth. This was first described by Lyubetski,[1] and later, independently, by Bayer[2] and by Conte.[3] This reduction takes place through hypoplasia and hypotrophy of the uterine muscle, together with a disappearance of the marked natal hyperemia of the organ. It is supposed to be caused by the withdrawal at birth of a hormone produced by the placenta, the ovary or the tissues of both of these structures, (Aschner,[4] Herrmann,[5] Fellner,[6, 7] Frank,[8, 9] and

[1] Lyubetski, N. S., Anatomical changes in the uterus in children. Diss. St. Petersburg, 1900.

[2] Bayer, H., *Deutsch. Arch. klin. Med.*, 1902, lxxiii, 422.

[3] Conte, G., *Atti Soc. Ital. d. Ostet e Ginecol.*, 1903, ix, 670.

[4] Aschner, B., *Arch. f. Gynäkol.*, 1913, xcix, 534.

[5] Herrmann, E., *Monatsschr. f. Geburtsh.*, 1915, xli, 1.

[6] Fellner, O. O. *Zentralbl. f. allg. Path. u. path. Anat.*, 1912, xxiii.

[7] Fellner, O. O., *Arch. f. Gynäkol.*, 1913, c, 641.

[8] Frank, R. T., and Rosenbloom, J., *Surg. Gynecol. and Obstet.*, 1915, xxi, 646.

[9] Frank, R. T., *ibid.*, 1917, xxv, 329.