4139

On the Vitality of Dental Enamel.

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Various common pathological changes in the dental enamel of fully erupted human teeth, which destroy its protective function, are ascribed by some investigators to intrinsic nutritional alterations, rather than to extrinsic influences. The proof that enamel contains protein[1, 2] has induced several investigators to regard its presence as indicative of nutritional capacity, despite the fact that, although permeable to water and various substances, enamel completely lacks power of self-repair after injury and has no cellular nuclei. That the contained protein (probably keratin) is not associated with intrinsic nutritional changes in the enamel of erupted teeth is indicated by the writer's further observation that conventional tests for proteolytic and amylolytic enzymes, applied to extracts of pulverized normal enamel, were completely negative. These tests, to be extended, will include studies of abnormal enamel.

4140

Occurrence of Cystine in Sweat of Cystinurics.

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In 1871, Dewar and Gamgee[1] stated that, in some cases, cystine was present in the sweat of cystinurics, apparently basing their conclusions largely upon the observation that, in cases where cystine was present in the urine, silver coins carried in the pocket were observed rapidly to become blackened. Garrod,[2] however, was unable to obtain evidence of the presence of cystine in the sweat in the one case studied by him.

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[1] Bödecker and Gies, PROC. SOC. EXP. BIOL. AND MED., 1924, xxii, 175.

[2] Gies, *J. Dental Res.*, 1926, vi, 143.

[1] Dewar, J., and Gamgee, A., *J. Anat. Physiol.*, 1871, v, 142.

[2] Garrod, A. E., "Inborn Errors of Metabolism," London, 1923, 2nd edition, p. 103.

We have had under observation a cystinuric whose daily cystine excretion (determined by the colorimetric method of Folin and Looney) varied from 1.0 to 1.8 gm., whose urine frequently contained large amounts of cystine crystals, and who occasionally passed cystine calculi. From this patient we collected by the use of pilocarpine and heat, 100 cc. of sweat in 50 minutes.* No cystine could be detected by the colorimetric method of Folin and Looney. 35 cc. of sweat were deproteinized by the use of heat and alumina cream and the filtrate was evaporated to dryness. The residue was extracted with dilute ammonium hydroxide and the extract treated with acetic acid. No cystine crystals were observed microscopically. Wollaston's test (formation of cystine hydrochloride crystals) was also negative.

4141

Factors in the Control of Ascites.

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Ascites associated with portal cirrhosis of the liver is ordinarily controlled in one of 3 ways: by parcentesis, by variations of the Talma-Morison operation, or by the diuretics. All 3 procedures were employed in this case-study—a man of 58 with a long alcoholic history; a slight jaundice, malaise and some edema of the legs during the past 7 months; and an ascites of 2 months' duration. Paracentesis was made 5 times in all in the 8 weeks previous to operation, from 9 to 14 liters of fluid being removed each time. Analyses were made of the fluid. Omentopexy was accomplished 10 weeks after the onset of the ascites. A large fan of the omentum was brought out through a mid-line incision in the upper abdomen, spread out and sewn in place in a prepared properitoneal bed. The peritoneum was closed about the omental stalk with wide-apart mattress sutures. Convalescence was uneventful. The diagnosis of portal cirrhosis was confirmed at operation.

The reaccumulation of fluid in the peritoneal cavity subsequent to various attempts to establish additional collaterals is a frequent ex-

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