chicks which has been described in the text. Apparently the syndrome can be ascribed to the fish meals used in that they contained objectionable materials and/or lacked some accessory factor. It was not possible to produce this syndrome quantitatively by the use of other protein supplements.

8294 P

Metabolism of Benzene, Anthracene and Phenanthrene in Adult and Growing Dogs.

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In view of similarity in the metabolism of bromobenzene and naphthalene in rabbits[1] and in adult and growing dogs,[2] it seemed of interest to extend the comparison of metabolism of aromatic hydrocarbons in dogs to benzene, anthracene and phenanthrene. Each substance was fed on 4 occasions to each of the 4 growing and 2 adult dogs in 1.0 gm. doses. The pups and dogs were maintained on a diet of constant composition and the urine collected every 24 hours. The diet, the general experimental procedure, methods of analysis of urine were the same as described by us elsewhere.[3] The pups were fed benzene, anthracene and phenanthrene at the age of 2, 3, 4, and 7 months at sufficiently large intervals (7-8 days) to allow the animals to return to the original nitrogen and sulfur balance. The analysis of urine collected after feeding the hydrocarbons to pups and dogs indicated the formation of ethereal sulfates from benzene and anthracene; benzene, in addition, and especially phenanthrene, raised the output of neutral sulfur. All 3 hydrocarbons increased the output of glycuronates in the urine.

Hele[4] on the basis of experiments with benzene, similar to those described here, suggested that benzene probably yields a mercapturic acid, although the latter could not be isolated by Baumann-Preusse method.[5] Pending the isolation of detoxication products of benzene, anthracene and phenanthrene from the urine of dogs and pups, now

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1 Bourne, M. C., and Young, L., *Biochem. J.*, 1934, **28**, 803; Nakashima, T., *J. Biochem.* (Japan), 1934, **19**, 281.

2 Stekol, J. A., *J. Biol. Chem.*, 1935, **110**, 463.

3 Stekol, J. A., *J. Biol. Chem.*, 1934, **107**, 641; 1935, **109**, 147.

4 Callow, E. H., and Hele, T. S., 1926, **20**, 598.

5 Baumann, E., and Preusse, C., *Ber. deutsch. chem. Ges.*, 1879, **12**, 806.

in progress, it is assumed that benzene and phenanthrene are conjugated in the animal body to yield, perhaps, a mercapturic acid type product, similar to p-halogen-phenylmercapturic and l-α-naphthalene mercapturic acids.

8295 C

Effects of Various Anesthesias on Autoxidation Rate of Surviving Brain Tissue.

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It has previously been shown that ether anesthesia in rats brings about a condition, demonstrable in brain tissue removed immediately after termination of one hour of deep surgical anesthesia, such that the rate of autoxidation of available carbohydrate and lactic acid in the surviving brain decreases more rapidly than is usual for tissues taken from untreated rats.[1] Since both the total carbohydrate and glycogen content of rat brain decreases in ether anesthesia[2] despite the marked hyperglycemia maintained at the same time, it was suggested that the inhibitory effect on rate of autoxidation was due simply to limiting of oxidizable carbohydrate in the excised tissue. The ability of such tissue to metabolize added glucose at a normal rate substantiates this.

Examination of data on blood and urine chemistry shows marked resemblance of biochemical effects of epinephrine and ether. The possibility that many of the physiological side actions of ether are mediated through stimulation of adrenin output is supported by the weight of much evidence,[3] and studies on intestinal chemistry[4] and ether ketosis[5] show marked parallelism of many effects of the 2 agents. Unequivocal direct evidence of the supposed action of ether on the suprarenals is as yet lacking.[6] The present communica-

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1 Emerson, G. A., *J. Tenn. Acad. Sci.* In press.

2 Uchida, S., *Biochem. Z.*, 1926, **167**, 9.

3 Knoefel, P. K., *California and Western Med.*, 1933, **39**, 5.

4 Emerson, G. A. To be published.

5 Emerson, G. A., *J. Pharm. Exp. Therap.*, 1935, **54**, 90.

6 Elliott, T. R., *J. Physiol.*, 1912, **44**, 374; Schlossmann, H., and Mügge, H., *Arch. Exp. Pathol. Pharmacol.*, 1929, **144**, 133; Fujii, I., *Tohoku J. Exp. Med.*, 1925, **5**, 566; Kodama, S., *Tohoku J. Exp. Med.*, 1924, **4**, 601.