14°C. showed an excess oxygen consumption 70% of normal when treated with brom-acetic acid of 0.04 to 0.2%. At this temperature the muscles retained their irritability for several hours after poisoning.

Normal muscles electrically stimulated for 5 sec. at 14°C. gave an average value (6 determinations) of 0.0035 for the quotient

$$\frac{O_2\text{mm}^3}{\text{Tension (gm.)} \times \text{length (cm.)} \times \text{time (sec.)}}$$

where the oxygen represents the excess consumption by the weight of muscle used. Muscles poisoned ½ hour with brom-acetic acid in concentrations of 0.01 to 0.2% gave an average value of 0.0024 for this quotient or approximately 70% of the normal value.

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Influence of Carbon Dioxide upon the Respiration of Nerve.*

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The influence of carbon dioxide upon the respiration of the lateral line nerve of the smooth and spiny dogfish has been studied by the tonometric method. Fifteen cc. tonometers which contained besides the nerves one-tenth of a cc. of isotonic solution were placed in a water bath, the temperature of which varied from 20.5 to 23.7°C. in the different experiments. The gas mixture present in the tonometers was analyzed at the beginning and at the end of each experiment on a modified Haldane-Henderson analyzer. The measurement was begun 2 to 3 hours after the removal of the nerves and lasted for 4 or 5 hours. Eight or more hours after the isolation of dogfish nerves the rate of oxygen consumption rapidly increases.

The tonometric method has been checked by burning ethyl alcohol in the tonometers and determining the respiratory quotient. These quotients vary from 0.630 to 0.678 with an average of 0.655.

Ten control experiments showed that the maximum difference in the rate of respiration of matched nerves was 16%. Forty-five experiments were carried out on nerves in air-oxygen gas mixtures. The rate of oxygen consumption varied from 25.1 to 157.0 cu. mm. per gm. of fresh weight per hour, depending upon the size of the

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nerve. The average was 64.1 cu. mm. The rate of carbon dioxide production fluctuated between 17.3 and 176.0 cu. mm. per gm. per hour with an average of 59.2. The apparent respiratory quotients averaged 0.92.

Calculation of the oxygen tension necessary to supply these nerves by Fenn's[1] or Gerard's[2] formula indicated that in all cases sufficient oxygen was present to prevent asphyxia. This has been tested directly in a few experiments.

The influence of carbon dioxide upon the respiration of one of a pair of matched nerves has been studied in 24 experiments. The rate of oxygen consumption decreases in approximately a linear manner to 50% of the control value in the presence of 150 mm. Hg carbon dioxide tension. Above 150 mm. Hg carbon dioxide tension, the rate of oxygen consumption decreases more slowly to about 40% of the control at 300 mm. Hg. The rate of carbon dioxide production is depressed to a slightly greater extent than the rate of oxygen consumption. Above 150 mm. Hg carbon dioxide tension the rate of carbon dioxide production was not constant although in 4 of 5 experiments a depression was present.

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Excretion of Xylose.

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The constancy of the rate of disappearance of xylose from the blood of animals[1] and the change in the rate of elimination after experimental damage to the kidney membrane[2] suggested the possibility of its use as a delicate indicator of damaged renal function. The present availability of xylose would render it an excellent substance for kidney function tests in analogy to the liver function tests which employ such sugars as galactose, levulose, etc. Xylose is particularly suited for this purpose since its elimination is practically independent of the liver.

On the oral administration of 50 gm. of xylose, 25±5% is elim-

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[1] Fenn, W. O., *J. Gen. Physiol.*, 1926, **10**, 767.

[2] Gerard, R. W., *Am. J. Physiol.*, 1927, **82**, 381.

[1] Fishberg, E. H., *J. Biol. Chem.*, 1930, **86**, 665.

[2] Fishberg, E. H., and Dolin, B. T., *Arch. Int. Med.*, 1930, **46**, 321.