not from sodium chloride disturbance. The results appear to refute the theory that sodium chloride metabolism is specially regulated by the adrenal cortex. They furnish further strong evidence, however, in favor of our first-proposed carbohydrate theory of cortico-adrenal function.

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THE PERIPHERAL ACTION OF BARBITURATES[1]

LIEB and Mulinos[2] and other authors[3,4] described the action of amytal in impairing vagus cardiac inhibitory effects. According to Swanson and Shonle,[5] this action was regarded as characteristic for amytal, since a closely related barbiturate, iso-amytal (1-methyl-butyl ethyl barbituric acid or pentobarbital), failed to produce a similar peripheral depression of the cardiac vagus in doses of 5 to 20 mgm per kgm.

In a large series of experiments, using dogs and rabbits, we confirmed the original observation of Lieb and Mulinos with amytal. We found that amytal in doses ranging from 50 to 60 mgm per kgm abolished the typical cardiac vagus effect, *viz.*, the cardiac inhibition which resulted from stimulation of the peripheral vagus. Experiments of a similar nature revealed the fact that this peripheral action of amytal is also exhibited after very large doses of pentobarbital-sodium and other barbiturates. The animals used in these experiments were kept alive with picrotoxin. Doses of pentobarbital-sodium varying from 40 to 100 mgm and barbital-sodium in doses of 500 to 1,100 mgm per kgm completely abolished the typical vagus response to faradic stimulation. Phenobarbital-sodium in doses of 100 to 350 mgm per kgm impaired but did not abolish the cardiac vagus effect. In all these instances where the peripheral vagus effect had been abolished by barbiturates, the central vagus effects, *e.g.*, the respiratory inhibition following the faradic stimulation of the central end, remained unimpaired.

The mechanism of the depression of the cardiac vagus function by barbiturates was disclosed by another series of experiments. The following facts relative to this problem have been ascertained:

(a) Pilocarpine (1 mgm per kgm) slowed the heart of barbiturate-treated animals which did not respond to peripheral vagus stimulation.

(b) Large doses of acetyl choline (0.1 mgm per kgm) produce vasodilation and cardiac inhibition in the animals which had received barbiturates.

(c) Doses of physostigmine (0.2 to 0.35 mgm per kgm) which were insufficient to produce slowing of the heart rendered the cardiac vagus sensitive to faradic stimulation. Also large doses of acetyl choline had the same effect after the blood pressure and heart rate returned to the original level.

These experiments suggest that the cardiac vagus impairing action of barbiturates is not an atropine-like action but probably a nicotine-like effect on the vagus ganglia.

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SCIENTIFIC APPARATUS AND LABORATORY METHODS

A SIMPLE TYPE OF ERGOMETER

IN order that the greatest value may be obtained from many laboratory experiments in human physiology it is desirable that students, particularly those in the preclinical years of medicine, understand the relationship between basal manifestations and known amounts of activity. Ergometers, when constructed to give a reasonably accurate measure of the work performed, are usually so costly that one piece of apparatus is to be found in a laboratory. Hence experiments must be either done by very large groups or the experiment conducted as a demonstration.

It has been the policy in the author's laboratory to correlate to a maximal degree the effect of known amounts of activity with the various physiological systems. In order to do this a simple yet efficient type of ergometer has been devised of such a nature that each table of a special laboratory devoted to human physiology is equipped with this device. The arrangement has proven of great convenience, since students have an opportunity to evaluate the effect of graded activity upon such physiological manifestations as blood pressure, pulse rate, metabolism and respiration. A further definite advantage of the apparatus rests in the fact that preliminary measurements can be taken under nearly basal conditions, which is not true of many common forms of ergometer.

The ergometer is constructed at one end of a table, 72×30 inches, with legs of sufficient height that the usual type of clinical metabolism apparatus may be

[1] From the Department of Pharmacology and Materia Medica, Georgetown University, School of Medicine, Washington, D. C.

[2] Lieb and Mulinos, *Proc. Soc. Exper. Biol. and Med.*, 26: 709, 1929.

[3] Shafer, Underwood and Gaynor, *Am. Jour. Physiol.*, 91: 461, 1930.

[4] Garry, *Jour. Pharmacol. Exper. Therap.*, 39: 129, 1930.

[5] Swanson and Shonle, *Jour. Lab. and Clin. Med.*, 16: 1056, 1931.