

Fig. 2. Calibration curve obtained by plotting meter reading against the log of exposure time.

meter reading was plotted against the log of correct exposure time in seconds (see curve Fig. 2 and Table 2).

The exposure time was found to be constant for any particular meter reading, when the B Wratten (green) filter was used and when objectives were changed.

Table 2. Log of exposure time.

Meter reading	Exposure time (sec)	Log of exposure
1	1/25	1.398
5	1/125	2.017
10	1/200	2.301
15	1/320	2.500
20	1/500	2.699

This was also true for living tissue in agarslant tubes. After calibrating the meter, 36 exposures were made on Plus-X film at various meter readings using the exposure time indicated by the graph (Fig. 2). The resulting negatives were of equal density displaying the same printing qualities.

Some improvements were suggested after the meter was constructed. (i) The brass plate and steel mounting box could be made of aluminum. (ii) A 0-20 microammeter, 4-in. scale, with resistance shunts, may be used to some advantage. (iii) If additional sensitivity is required, a 1½-v dry cell can be wired in series with the microammeter.

This meter can also be used on bellows-type cameras when it is properly calibrated to the conditions employed. The calibrations should be carried out on newly constructed meters.

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Effects of Adrenal Medullary Hormones on Antidiuretic Substance in Blood Serum

Frederic A. Giere* and W. J. Eversole

Department of Biology,
University of New Mexico, Albuquerque

O'Connor and Verney (1) suggested that adrenaline inhibits the release of the posterior pituitary antidiuretic hormone (ADH), but their evidence was indirect in nature since they made no attempt to measure antidiuretic substance (ADS) levels in blood. The present investigation (2, 3) represents an attempt to answer the question: Are the diuretic effects of adrenal medullary hormones in the rat (4, 5) due in any part to an inhibition of the release of posterior pituitary ADH?

The design of the experiment was patterned after Ames and van Dyke's (6) modification of the intravenous assay method of Jeffers *et al.* (7). The assay animals were rendered diuretic by the administration of water and ethanol, which anesthetized the rats and probably suppressed the secretion of endogenous antidiuretic substance from the posterior pituitary of the test animal (8). The rats used for assay, as well as the serum donors, were fasted for 18 hr. At the zero hour each assay animal was given 5 ml per 100 g of a 10 percent ethanol solution by gavage. After 30 min, 3 ml water per 100 gm body weight was given in the same manner. Within the following 45 min, the urethra was ligated, the bladder was cannulated, and a hypodermic needle was inserted into the saphenous vein. The needle was left in the vein, and a mandrel was kept in the bore of the needle except during the intravenous injection of the test material. The rat was placed (ventral side down) on a board equipped with a hole that supported a tuberculin syringe barrel (graduated to 0.01 ml) and a hardware cloth cage that served as a restraining device. A polyethylene cannula drained urine from the urinary bladder into the mouth of the syringe barrel whose tip was fitted with a three-way stopcock.

Urine collections were made and recorded at 10-min intervals. Test samples of serum or Pitressin were injected via the previously prepared saphenous vein. Estimations of antidiuretic activity were made by comparing the antidiuretic effect of material to be assayed with that obtained from known amounts of Pitressin in the same assay rat. Duplicate assays of the same dose in the same animal were in agreement.

Serum obtained from blood collected by draining the trunk of decapitated gentle rats did not exhibit antidiuretic properties. Antidiuretic substance (ADS) equivalent to less than 0.11 milliunits (mU) Pitressin per milliliter of serum, was present in blood obtained by decapitation from tranquil rats that had been injected with morphine sulfate, 1 mg/kg (9). No serum ADS was detected in the blood of animals that had been injected with diuretic doses of epinephrine or nor-epinephrine (5) 5 min prior to injection of morphine sulfate. ADS release that follows morphine ad-

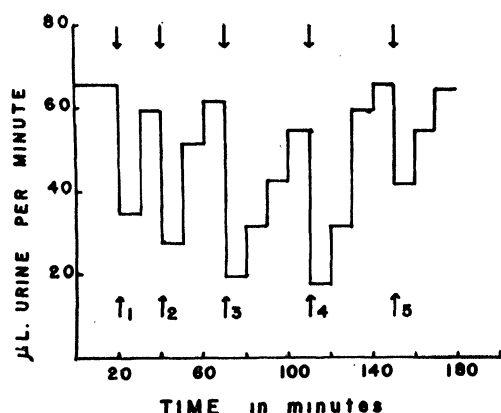


Fig. 1. Typical curve from assay procedure. Intravenous injections were made at the numbered arrows as follows: (1) 20 μ U Pitressin; (2) 30 μ U Pitressin; (3) 40 μ U Pitressin; (4) 50 μ U Pitressin; (5) 0.2 ml serum from rat exposed to ether for 5 min. The response indicates that the serum contained less than the equivalent of 0.1 mU Pitressin per milliliter.

ministration was not inhibited by a nondiuretic dose of epinephrine (20 μ g/100 g). ADS release caused by a 5-min exposure to ether could not be blocked by the previous administration of medullary hormones in the doses used here.

Figure 1 illustrates the manner in which the values shown in Table 1 were estimated. At this time only relative values of ADS in blood serums may be assigned to these results. Although the method now employed is only semiquantitative, we regard it as an

Table 1. Effects of medullary hormones on ADS in blood serum of rats.

Treatment	Cases	No. showing ADS	Estimated ADS (mU Pitressin/ml serum)
Decapitation	8	0	0.00
Morphine, 1 mg/kg			
Decapitation	8	8	.11
Epi., 20 μ g/100 g			
Morphine 1 mg/kg			
Decapitation	5	4	.10
Epi., 100 μ g/100 g			
Morphine 1 mg/kg			
Decapitation	9	0	.00
Nor-epi., 20 μ g/100 g			
Morphine 1 mg/kg			
Decapitation	7	0	.00
Ether anesthesia			
Heart puncture	13	11	.18
Epi. 100 μ g/100 g			
Ether anesthesia			
Heart puncture	15	11	.10
Nor-epi., 20 μ g/100 g			
Ether anesthesia			
Heart puncture	6	6	.13

accurate index for the detection of ADS in blood serums. We interpret these data as indicating that (i) 5-min exposure to ether is a stronger stimulus in causing release of ADH than is the injection of 1 mg/kg morphine sulfate; and (ii) the diuretic action of the adrenal medullary hormones in the rat may, in part, be due to their blocking of the release of posterior pituitary antidiuretic hormone.

References and Notes

- * Present address, Department of Biology, Luther College, Decorah, Iowa.
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2. This investigation was supported by a research grant (A-202) from the National Institute of Arthritis and Metabolic Diseases of the National Institutes of Health, U.S. Public Health Service.
3. Acknowledgment is made to M. L. Tainter of the Sterling-Winthrop Research Institute, for the pure forms of the adrenal medullary hormones, and to D. A. McGinty of Parke Davis Co., for the standard Pitressin. An explanation of some terms used in this paper follows: epinephrine, pure 1-epinephrine bitartrate; nor-epinephrine, pure 1-arterenol bitartrate monohydrate.
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A Single Diet for All Living Organisms

Thomas D. Luckey

Department of Biochemistry, School of Medicine,
University of Missouri, Columbia

Certain aspects of nutrition as a common denominator of biology are presented to indicate that diet can be considered as a single unit in the variables of biological research. A broad concept of comparative nutrition could be considered if different species could be reared on the same diet. Such a possibility may be induced from two considerations, the first being the great extent to which nutritional principles may be translated from one species to another. This is well illustrated by the fact that some of the individual B-vitamins, essential to the life of animals, were first discovered as microbial growth factors. Literature shows a striking similarity in the semisynthetic diets fed to monkeys, rats, mice, dogs, and chicks. Second, the qualitative nutritional requirements of most animals, plants, and microbial forms studied can be expressed in common terms as 15 to 50 nutritional elements (minerals, amino acids, vitamins, and a few unidentified factors). The suspected role of intestinal microorganisms in the production of vital unidentified factors for the host has been contraindicated by maintaining germfree chicks and rats (1) through one and six generations, respectively. This gives some assur-