EFFECT OF DIAPHYLLINE ON SODIUM EXCRETION IN THE SALIVA

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Injection of diaphylline into the arterial blood flow to the parotid and submandibular salivary glands of anesthetized dogs caused a definite increase in the sodium concentration in the saliva from both glands. Vasopressin, injected into the blood flow of the glands against the background of diaphylline action, caused a more marked increase in the concentration and content of sodium in the saliva than that produced by administration of the hormone alone.

Investigations have shown that xanthine derivatives (theophylline and diaphylline) have the property of stimulating active sodium transport through the wall of the amphibian urinary bladder [5-8], and also of increasing the excretion of sodium in the urine [1, 2]. The sodium-excreting effect of theophylline and diaphylline is most marked in cases when an antidiuretic hormone is present in the circulating blood. Xanthine derivatives are known to potentiate the sodium-excreting action of pituitrin [1, 2]. Pituitrin increases the elimination of sodium not only via the kidneys, but also via the salivary glands [3, 4].

It was therefore decided to investigate whether the action of xanthine derivatives is restricted to the osmoregulatory organs or whether it extends also to other organs engaged in the transfer of electrolytes and water from the internal to the external milieu of the body, viz., the salivary glands. The present investigation is concerned with the effect of diaphylline on the excretion of sodium in the saliva.

EXPERIMENTAL METHOD

In experiments on eight dogs, anesthetized with chloralose (100 mg/kg), saliva was collected through thin polyethylene catheters introduced into the common duct of the right parotid and right submandibular salivary glands. The secretion of saliva was stimulated by pilocarpine, which was made up in isotonic dextrose solution and injected at a constant rate throughout the experiment. The initial level of secretion of the parotid gland was 0.3 ml/min, and of the submandibular gland 0.6 ml/min. After the rate of secretion of saliva by the animals had become stable, 1 ml of isotonic dextrose solution containing vasopressin in a dose of 0.2 milliunit (m.u.) per kilogram body weight was injected into the blood flowing through the right carotid artery. The rate of secretion of saliva and the concentrations of sodium and potassium in it were determined. The experiment was continued after an interval of 1 h. The same level of saliva secretion as at the beginning of the experiment was maintained, and against this background a 2.4% solution of diaphylline was injected into the right carotid artery at the rate of 0.2 ml/min for 10 min. After the injection of diaphylline had continued for 6 min, vasopressin was injected in a dose of 0.2 m.u./kg body weight. The synthetic hormone lysine vasopressin (SPOFA, Czechoslovakia) anddiaphylline (Gedeon Richter, Hungary) were used in the experiments.

EXPERIMENTAL RESULTS

Injection of vasopressin into the arterial blood flow to the parotid and submandibular salivary glands did not produce any significant change in the rate of secretion of saliva or in its sodium content, and the

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excretion of potassium in the saliva was only slightly affected. The results of the second series of experiments showed that diaphylline, if injected into the arterial blood flow to the salivary glands, caused a regular increase in the sodium concentration in saliva excreted both by the parotid (from 26.4 ± 4 to 38.4 ± 3.6 meq/liter; $P < 0.05$) and the submandibular (from 19.9 ± 3.6 to 28.8 ± 3.5 meq/liter; $P = 0.05$) salivary glands. The sodium content in saliva from the parotid gland was increased by 32%, and in saliva from the submandibular gland by 42%; diaphylline had no effect on the volume of saliva secreted.

Injection of vasopressin after diaphylline, for instance, increased the sodium concentration in the saliva from the parotid gland to 49.2 ± 7.5 meq/liter (by 86%), and in the saliva from the submandibular gland to 38.2 ± 4.7 meq/liter (by 97%), whereas injection of vasopressin alone increased the sodium concentration in the saliva by not more than 35%. The sodium content in the saliva was increased only slightly after injection of vasopressin plus diaphylline. The reason for this is that vasopressin reduced the rate of secretion of saliva.

The results of these experiments thus showed that xanthine derivatives increase the excretion of sodium in the saliva and also potentiate the corresponding action of vasopressin on the salivary glands.

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