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Diurnal Variations in Excretion of Chloride in Normal Subjects.* (23327)

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A number of investigators have reported a diurnal rhythm in the excretion of sodium or chloride. Most of these authors were interested in the effects of sleep(1-7). Some studied the salt excretion for consecutive time intervals of different lengths(8,9). The effects of posture(10) and of varying degrees of muscular work(11) also have been considered. In all studies most of the normal subjects excreted more sodium chloride/hour during the day than during the night. However, the number of cases which have been studied is relatively small. Since derangement of the diurnal rhythm in excretion of sodium chloride is sufficiently characteristic in certain diseases to be an aid in diagnosis, and possibly in prognosis and evaluation of therapy, more information is needed on the variations for normal subjects(10-13).

Methods. Forty-one normal subjects were studied, 40 of whom were between 18 and 33 years of age. The subjects were university students or laboratory workers. There was no regulation of their salt intake or their degree of activity. No attempt was made to standardize or in any way to regulate their sleeping and eating habits. They were required simply to furnish a consecutive day and night urine. Each subject recorded his time of retiring and of arising. The night urine was that produced during the recumbent period after retiring at night, and the day urine was that of the up-and-about period between arising and retiring. Chloride was determined by the Volhard-Harvey method. Chloride excretion is expressed as NaCl excretion/hour. NaCl excretion/hour during the day divided by NaCl excretion/hour during the night is the ratio which is calculated.

Results. The chloride excretion/hour during the day, expressed as NaCl, ranged from .15 to .96 g with a mean of .48 g and standard deviation of .21. The chloride excretion/hour during the night, expressed as NaCl, ranged from .04 to .64 g with a mean of .25 g and standard deviation of .14. For the day to night ratios the range was 0.71 to 4.58 with a mean of 2.31 and standard deviation of 1.09. Creatinine excretion determined in 25 of the subjects ranged from 1.16 to 2.73 g per 24 hours(14).

The correlation coefficient for the total amount of salt excreted per 24 hours and the day-to-night NaCl excretion ratio was +0.026. The distribution of the ratios of the 41 subjects studied did not differ significantly from that of the ratios of 28 subjects for whom data necessary for calculation of the ratios had been reported by other investigators. The distribution of the day-to-night Na or Cl excretion ratios for the 69 subjects is shown in Fig. 1. The lowest ratio found

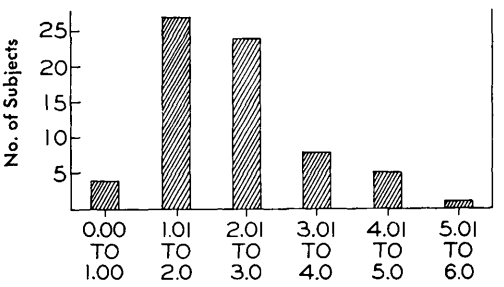

FIG. 1. Distribution of day-to-night NaCl excretion ratios of normal subjects.

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in a normal subject was 0.71. This is to be contrasted with the low ratios seen in subjects with congestive heart failure, cirrhosis of the liver and orthostatic hypotension(10-12).

Summary. The day-to-night ratio of urinary chloride excretion has been determined for each of 41 normal subjects and has been calculated for 28 subjects reported in other papers. No restrictions of diet or activity were imposed. In the great majority of the subjects more chloride was excreted/hour during the up-and-about period than during the period of recumbency. This was true regardless of the amount of chloride excreted/24 hours.

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Mechanism of Reflex Bradycardia which Follows Sudden Closure of an Arteriovenous Fistula.* (23328)

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The bradycardia which follows sudden closure of an arteriovenous fistula was first described by Nicoladoni(1), and independently by Branham(2). There have since been numerous attempts to explain the mechanism of this response. Carotid-aortic pressoreceptors(3), pressoreceptors of the right atrio-caval junction(4), and reflexes arising in the vessels at the site of the fistula(5) have all been discussed as possible sites of origin of the reflex bradycardia. Lewis and Drury (6) showed that atropine will abolish the bradycardia, and concluded that the efferent limb of the reflex is the vagus. This work was later confirmed by Kramer and Kahn(7). The purpose of this report is to describe the afferent limb of the reflex and to define the mechanism which initiates the reflex bradycardia.

Methods. Dogs, weighing 11 to 27 kg, were premedicated with 1-2 mg/kg of morphine sulfate, and anesthetized with 70-90 mg/kg of chloralose, intravenously. Heparin, 5 mg/kg, and Manuronate, 20 mg/kg, were used as anticoagulants. Heart rates were measured from electrocardiograms recorded on a Sanborn visocardiette, or in some dogs from systemic pressure pulse tracings recorded by a strain gauge or Gregg manometer. Systemic arterial pressure was measured through a brachial artery cannula, and in some experiments right atrial pressure was measured through a catheter passed into the atrium by way of the jugular vein. The carotid sinuses were denervated either by adventitial stripping and phenol painting, or by complete extirpation. The aortic arch was denervated according to a modification of the method of Comroe(8). The left vagus nerve was cut high in the neck, and all branches running medially and laterally from the right main vagal trunk were cut down to the level of the aortic arch, leaving the right main

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