had been omitted the decrease in acuity failed to occur.

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Sweat Sodium Levels in Congestive Heart Failure. (19292)

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Evidence has been presented suggesting that overactivity of adrenal cortical electrolyte hormone may be partially responsible for the sodium retention characteristic of congestive heart failure. Biological methods have indicated increases in corticoids(1) and DOCA-like material(2) in the urines of congestive failure patients. Salivary sodium concentrations have been reported to be low in congestive failure(3), also suggesting adrenal hyperactivity. On the other hand Lasche, Perloff, and Durant found low urine 11-oxysteroid and 17 ketosteroid concentrations in the majority of 23 cardiac failure patients(4).

The work of Conn indicates that the activity of the adrenal "electrolyte-influencing" steroids can be correlated with the sweat sodium or chloride concentration(5). Low sweat sodium concentrations (below 10 meq/L) are found in hyperadrenal cortical states such as Cushing's syndrome and high concentrations (above 80 meq/L) in Addison's disease. Merrill in 1949 stated that several of his congestive failure patients had low sweat sodium concentrations(6); this appears to be the only published reference to the use of the sweat test in evaluating adrenal salt hormone activity in congestive heart failure. In the following study sweat sodium concentrations were investigated by a method applicable to patients with heart failure.

Methods. A plastic jacket with tight elastic neck, arm, and waist bands ensures collection of sweat on the trunk without evaporation. The subject, whose back has been thoroughly cleansed with distilled water, sits in the Fowler position in bed, and is heated by immersing one arm to the elbow in a tub of hot water and by placing several blankets over the body. In from 15 to 90 minutes sufficient sweat will be present on the back to ensure the collection of from 3 to 5 cc when the jacket is opened. A tablespoon is used to transfer the sweat to a test tube. To avoid evaporation it is necessary to wait until enough sweat has accumulated so that the desired amount can be obtained the first time the jacket is opened. Two cubic centimeters are usually sufficient for sodium and potassium determination. The jackets, towels, test tubes, and spoons are prepared by soaking in 3 changes of distilled water, which is sufficient to lower the concentration of sodium in the final wash-water to a negligible level. Blood eosinophil counts using Randolph's stain, were done immediately before the sweat tests. Sodium and potassium were determined with the flame photometer using lithium as the internal standard.

Material. The patients tested were in obvious, severe, congestive failure without evident complicating factors such as infection. All had gross pitting edema. Their ages ranged from 37 to 83 years. The majority of the patients had hypertensive or rheumatic heart disease or cor pulmonale. Some were studied immediately after admission to the hospital for symptoms of congestive failure. Others were studied towards the end of a 24-hour control period designed to standardize as far as

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TABLE I. Results of the Sweat Test in 3 Groups of Congestive Failure Patients.

Patient	Date	Avg temp.†	Na^+ intake, meq.	Urine Na^+, meq.	Fluid intake, cc	Urine vol., cc	Wt change lb	Sweat Na^+, meq.	Sweat K^+, meq.
				Group A, sodium retention					
1. S.	10/4/50	73	31	8.2	2400	430	+1	45.5	6.7
2. C.	9/22	69	22	10	1200	500	0	27	4.9
3. T.C.	11/2	77	22	.65	1700	500	0	69	2.1
4. M.	11/16	58	43	12.6	2500	600	0	49	7.7
5. T.	11/9	61	33	21.5	3000	500	0	55.2	7.6
6. J.	1/6/51	58	33	12.5		600		87	6.4
7. V.	1/6	58	22	15		500		77	5.1
8. D.	1/14	52	22	5.3	1500	700	— .25	37.8	7.8
9. R.	2/6	66	18	6.3	1250	330	+ .5	64	5.1
10. W.	4/3	60	22	3.4	2000	420	+ .75	34	9.2
*	4/14	63	76	112.8	2000	2450	—1	29.5	8.2
11. M.M.	4/12	67	44	23.5	2200	500	+1	51.3	6.4
12. G.	5/6	62	28	17.1	1750	300		45	6.7
*	5/14	61	33		1750	900	0	55	5.4
13. C.R.	6/5	64	55	6.8	5000	800	+3	40	11.6
				Group B, water retention					
14. H.	11/22/50	73	11	46.2	1500	370	+ .25	22	5.6
*	11/30	55	97	122.5	2250	2400	— .5	13.5	5.6
15. D.	1/15/51	55	60		4000		+2.5	31	8.7
16. S.	1/25	73	22	28.8	2500	720	+1.8	22.6	8.2
17. M.S.	4/12	66	33	69.5	2000	600	+ .5	38.3	5.2
18. A.	6/5	64	44	114	3250	1000	+ .8	77	5.2
*	6/11	62	35		2000		0	29	1.2
				Group C, diuresis of both water and sodium					
19. H.	10/20/50	63	55	208	3000	3200	—4	62	11.5
20. F.H.	1/18/51	55	27	62.1	1750	1150	— .25	34	5.6
21. G.	2/5	57	33	51.7	1500	600	— .5	75	6
22. B.	5/13	59	33	130	2500	2000	0	41.7	5.4
*	5/21	61	33		2500	1800	0	22.7	6.1

* Clinically compensated. During the previous 24 hr 4.5 g sodium chloride were given without sodium retention or wt gain.

† Avg of high and low temp. for the day.

possible the fluid and salt intake. During this control period the patients were in bed and received no medications, other than oxygen and opiates when necessary, and, occasionally, maintenance digitalis. Food intake was limited to measured amounts of milk (usually 1500 cc daily) so that the sodium intake could be calculated. The sodium content of milk closely approximated 22 meq per liter. The total fluid intake was recorded and, when possible, the patients were weighed at the beginning and end of the 24-hour periods. The urine volume and sodium content were measured. In 10 patients the test was repeated during a consecutive 24-hour period following the injection of 2 cc of mercuhydrin intramuscularly. Sodium and fluid intakes were essentially unchanged. Sweat tests were performed 8 hours after the injection to coincide with the peak of diuresis. Whenever possible, patients who regained compensation, through rest in bed and the use of digitalis, were restudied during a third 24-hour control period. These patients, on the day before the test, were able to handle a moderate salt load (4.5 g) without sodium retention or weight gain. The *control group* consisted of apparently normal hospitalized patients awaiting hernia repair or biopsy of breast lesions that proved to be non-malignant. They ranged in age from 24 to 60. They were studied during a similar 24-hour control period in which the fluid intake approximated 2500 cc and the food intake consisted of 1500 cc of milk. Three patients receiving ACTH in amounts sufficient to cause a mild Cushing's syndrome, and one patient with Addison's disease, untreated for a week, were tested during 24-hour control periods.

Results. In congestive failure, sweat studies should be most significant if performed when edema fluid is actually being retained. For

TABLE II. Comparison of Sweat Sodium Levels in the Various Groups Tested.

Group	No. of cases	Sweat Na^+, meq./L, mean and S.D.
Controls	18	34.3 ± 17.3
Congestive failure		
Group A	13	52.4 ± 16.5
B	5	38.2 ± 20.2
C	4	53.2 ± 16.2
D	11	33 ± 20.1
After mercuhydrin	10	48 ± 23.3
Compensated	5	29.9 ± 15.4
ACTH treated	3	7 ± .7
Addison's disease	1	117

this reason the congestive failure patients were evaluated by means of the 24-hour control periods. There were 4 groups among the patients studied (Table I). Group A consisted of those patients who were retaining sodium, with oliguria and a low urine sodium concentration, and usually a weight gain during the control period. In Group B were those who were in negative sodium balance with relatively high urine sodium concentrations but who were retaining water as evidenced by oliguria and weight gain during the control period. Group C contained those who, though they entered the hospital in congestive failure, were not actually retaining either sodium or water at the time of the test. The patients who entered the hospital in congestive failure and who were studied immediately without a control period made up Group D (Table II).

In none of the groups of congestive failure patients did the sweat sodium concentration offer evidence of adrenal cortical hyperactivity (Table II). The mean value for the patients with both sodium and water retention (Group A) was, in fact, significantly higher ($P > .02$) than that of the control group, suggesting adrenal hypoactivity. The mean value for Group C was approximately the same although sodium and water were being lost rather than retained. In the few patients who achieved relative "sodium compensation" following rest and treatment, there was a fall in the average value of the sweat sodium concentration. This is further evidence against the presence of adrenal hyperactivity during edema formation.

Mercuhydrin apparently does not affect the sweat sodium. There was no significant difference in the mean sweat sodium concentration of 10 patients before (47.4 meq per liter) and after (48.0 meq per liter) mercuhydrin. The concentrations of sodium below 10 meq per liter in the 3 ACTH-treated patients and the value of 117 meq per liter in the Addisonian patient are similar to the findings of others in such states(5,7).

Blood eosinophil counts and serum sodium levels were determined on all subjects. No correlation was evident between either of these values and the sweat sodium level.

Sweat potassium concentrations are included in Table I. Some desquamation often occurred with sweating even though the back had been thoroughly washed. The solid material is separable from the sweat; however, it is possible that dissolution of some of it may alter the potassium concentration. For this reason the significance of the sweat potassium levels is uncertain.

Discussion. In addition to Conn other investigators have reported the results of analysis of sweat in normals and various disease states(7-10). Sodium and chloride concentrations are roughly parallel so that the concentration of either probably serves as an index of electrolyte hormone activity. Clinically demonstrable disease of the adrenal cortex has been associated with concentrations of sodium below 10 or above 80 meq/L. The normal range of sodium concentration is usually considered to be from 15 to 60 meq/L, but many of the patients in our control group, as well as those of others, have exceeded these limits. This wide range of values in normals serves to emphasize the probability that many factors, both endocrine and non-endocrine, affect the sweat electrolyte composition.

The amount of salt in the diet appears to influence the sodium concentration of sweat (10). In the present study the sodium intake was kept as near 33 meq as possible during the day on which the test was done.

The environmental temperature is a modifying influence on the sweat sodium concentration, presumably through variation in the "tone" of adrenal cortical control. Normals tested in the summer averaged 26 meq/L lower than when tested in the winter in St. Louis,

Missouri(9). The present study was carried out in a relatively mild climate where there was a difference of only 25°F between the average temperatures of the warmest and coldest days on which sweat tests were performed (Table I).

The rate of production of sweat appears to be an important factor affecting its electrolyte concentration. The rate of sweating can be measured on the arm by the method described by Locke *et al.*(10). However, many of our patients were observed to display markedly different rates of sweating on different body surfaces. Some perspired profusely on the forehead and lightly on the back, and others vice versa. If the sodium concentration of sweat is relatively constant on different body areas, as has been assumed, then measurement of the rate of sweat production in any one area might be misleading. Total body sweat volume has been measured by Dill, Hall, and Edwards(11), but the method is not easily applicable to bedridden patients. The back was used in our patients rather than the arm because it was felt that sweat production on the back usually occurred with less heat exposure. The back is poorly adapted to measurement of sweat volume, however. A very gross estimate of the rate of sweating was made by recording the time necessary for the back to become wet. This measurement did not appear to correlate with sweat sodium concentrations.

Many other factors which may affect the volume and composition of sweat, such as parasympathetic and sympathetic tone, blood vessel tone and state of hydration, remain to be evaluated. Therefore it is felt that, at least at the present time, unless the sweat sodium concentration is very high or very low, it probably should not be assumed to be a reliable index of adrenal electrolyte hormone activity.

Summary. (1) Sweat sodium concentrations were determined by a method applicable to sick patients confined to bed. (2) Congestive heart failure patients were studied while retaining edema fluid, before and after mercuhydrin injection, and, in a few instances, when recompensated. There was no evidence that increased adrenal electrolyte hormone activity contributed to the sodium retention of heart failure. The mean sweat sodium concentration of one group of congestive failure patients was significantly higher than that of the controls. (3) Mercuhydrin did not affect sweat sodium concentration. (4) In addition to the adrenal cortex, there may be numerous non-endocrine factors that affect sweat electrolyte composition.

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