

in the weasel. Petrides(4) has recently discussed the problem of aging various fur bearing mammals by such characters as the baculum and the value of obtaining data of this type in the management of these species. If the results of this study do apply to other closely related species such as the marten, fisher, wolverine, badger, etc. whose sexual cycles are not fully understood, it would mean that those animals in the wild with adult type bacula were animals which had been sexually mature during the previous breeding season.

4. Petrides, G. A., *Am. Midl. Nat.*, 1950, v43, 355.

This would be of value in estimating age ratios of these animals taken by trappers.

Summary. The bacula of castrated immature male long-tailed weasels developed to the adult type after the animals were implanted with pellets of testosterone propionate. The bacula of untreated castrated immature males did not develop to the adult type after several months. Once the baculum develops to the adult type it does not regress after castration.

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Renal Function in Decompensated Cirrhosis of the Liver.* (18358)

FREDERICK H. EPSTEIN, GERSON T. LESSER, AND EUGENE Y. BERGER.
(With the technical assistance of Roslyn Jackenthal.)

From the Research Service, New York University Medical Division, Goldwater Memorial Hospital, Welfare Island, and Department of Medicine, New York University College of Medicine.

Patients with decompensated cirrhosis of the liver have, as is well known, an impaired capacity for excreting sodium(1-5). Studies of glomerular filtration rate and renal plasma flow have, however, been few and the data obtained contradictory (Table I). The present study was intended to expand existing information and report simultaneous observations on glomerular filtration rate and electrolyte excretion, with a view toward drawing

TABLE I. Summary of Inulin, Mannitol, and Para-aminohippurate Clearances in Patients with Cirrhosis of the Liver Reported from Various Laboratories.*

Author	No. of subjects	Accumulating fluid	Presence of ascites	Inulin clearance, ml/min.	Mannitol clearance, ml/min.	PAH clearance, ml/min.
Patek(6)	1	Yes	Yes	91		434
	1	"	"	142		600
	1	"	"	172		709
Leslie(7)	5	"	"	69†		371
	2	No	"	136†		704
	6	"	No	201†		846
Farnsworth(1)	1	Yes	Yes		61	415
	1	"	"		74	476
Goodyer(5)	1	"	"		60	298
	1	"	"		88	449
	1	"	"		94	526
	1	"	"		102	492
	1	"	"		123	418
	1	"	"		124	647

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1. Farnsworth, E. B., and Krakusin, J. S., *J. Lab. Clin. Med.*, 1948, v33, 1545.
2. Falloon, W. W., Eckhardt, R. D., Cooper, A. M., and Davidson, C. S., *J. Clin. Invest.*, 1949, v28, 595.
3. Eisenmenger, W. J., Ahrens, E. H., Jr., Blondheim, S. H., and Kunkel, H. G., *J. Lab. Clin. Med.*, 1949, v34, 1029.
4. Hilton, J. G., *Am. J. Med.*, 1950, v9, 108.
5. Goodyer, A. V. N., Relman, A. S., Lawrason, F. D., and Epstein, F. H., *J. Clin. Invest.*, 1950, v29, 973.
6. Patek, A. J., Jr., Mankin, H., Colcher, H., Lowell, A., and Earle, D. P., Jr., *J. Clin. Invest.*, 1948, v27, 135.
7. Leslie, S. H., Johnson, B., and Ralli, E. P., *J. Clin. Endocrin.*, 1949, v9, 682.

* The data of Patek and Goodyer are corrected to 1.73 sq.m., the data of Leslie and Farnsworth apparently are not corrected for surface area.
† Mean.

some conclusions concerning the mechanism of salt and water retention in cirrhosis of the liver and to correlate the degree of liver im-

TABLE II. Clinical and Laboratory Data on Patients with Cirrhosis of the Liver.

Name	Age, yr	Sex	Wt gain per wk, lb.†	Inulin clearance,* ml/min.	PAH clearance,* ml/min.	Plasma Na, mEq/min.	Urinary Na ex.,† mEq/min.
T	51	M	6	93	705	131.4	1.5
K	59	F	2	98	324	135.6	88.0
WS	50	M	9	103	565	127.8	0.7
H	46	M	13	105	425	139.7	2.6
F	41	F	7	106	700	141.6	13.7
G	67	M	7	127	488	138.4	1.9
NS	53	M	8	127	710	135.2	58.0
NK	58	F	4	132	521	—	89.0
BW	46	M	8	132	610	140.6	90.0
WT	36	M	0‡	141	660	136.7	70.0
BG	54	M	8	141	618	132.4	1.5
O	34	M	4	169	1110	144.0	12.0

* Avg of 3 consecutive collection periods corrected to body surface area of 1.73 Sq.M.

† The wt gain per wk cannot be correlated with the urinary sodium excretion since the dietary sodium intake was not strictly controlled in all patients.

‡ WT was undergoing a spontaneous diuresis at the time of measurement.

pairment with renal function.

Methodology. Twelve patients suffering from Laennec's cirrhosis of the liver confirmed by history and by clinical findings were studied. All showed diminished liver function on the basis of cephalin flocculation, thymol turbidity, bromsulfalein retention, and plasma albumin level. Subjects were selected to exclude those with findings indicative of cardiac or renal disease. All patients had required abdominal paracentesis during a period of illness which ranged from 3 to 60 months (average 16 months). All had ascites and edema of varying extent; 11 were accumulating fluid and one (WT) was undergoing a spontaneous diuresis at the time of measurement (Table II). The patients were ambulatory in the hospital. They received a high protein, high caloric diet containing at least 3 to 5 g (50 to 80 mEq) of sodium chloride per day. Mercurial diuretics and abdominal paracenteses were withheld for several days prior to study. Glomerular filtration rate was measured as the inulin clearance and renal plasma flow was measured as the p-aminohippurate clearance. After an appropriate priming dose, 5% glucose containing inulin and sodium p-aminohippurate was administered intravenously at a rate of 1 ml per minute. This solution was freshly prepared and the glucose did not remain in contact with the p-aminohippurate longer than 3 hours(8). If a reaction product formed

between the two, the blood flows would be still higher than the reported values. Urine was collected in periods of 20 to 30 minutes through an indwelling catheter. Venous blood was collected at the mid-point of each urine collection period. All clearance data are reported as the mean of 3 consecutive collection periods. Inulin was measured in urine and plasma by Harrison's modification(9) of Alving's method(10). p-Aminohippurate was measured in diluted urine and trichloroacetic acid filtrates of plasma by the Bratton-Marshall reaction(11). Sodium was determined with an internal standard flame photometer (12).

Observations. The inulin clearance varied from 93 to 169 ml/min. which is within the normal range of published data(13). The mean for all subjects was 123 ml/min. (Table

8. Baldwin, D. S., Schreiner, G. E., Breed, E. S., Wesson, L. G., Jr., and Maxwell, M. H., *J. Clin. Invest.*, 1950, v29, 614.

9. Harrison, H. E., *PROC. SOC. EXP. BIOL. MED.*, 1942, v49, 111.

10. Alving, A. S., Rubin, J., and Miller, B. F., *J. Biol. Chem.*, 1939, v127, 609.

11. Bratton, A. C., and Marshall, E. K., Jr., *J. Biol. Chem.*, 1939, v128, 537.

12. Barnes, R. B., Richardson, D., Berry, J. W., and Hood, R. L., *Indust. Engin. Chem. (Anal. Ed.)* 1945, v17, 605.

13. Smith, H. W., *Lectures on the Kidney*. University Extension Division, University of Kansas. Lawrence, Kansas, 1945.

II). Likewise, the individual clearances of p-aminohippurate fell within normal limits apart from the lowest (324 ml/min.) and the highest (1110 ml/min.). The mean renal plasma flow was 595 ml/min.

Plasma sodium values were normal except for 3 patients. In view of a normal filtration rate the load of sodium presented to the tubule can be considered essentially normal. The actual urinary excretion of sodium, however, was practically negligible in 5 patients (1 to 2 micro equivalents/min.), while the others excreted sodium in amounts which could be considered normal. It is evident, therefore, that the inability to excrete sodium in cirrhosis is not a function of the filtration rate.

The degree of abnormality of the liver function tests was not related to the rate at which the patient was accumulating fluid. Moreover, in the same patient spontaneous diuresis may occur prior to any significant change in the liver function tests, as was subsequently observed in patients WS and WT. This confirms the well known clinical observation that the degree of liver damage measured by the usual tests does not necessarily parallel salt retention.

Comment. The data presented demonstrate

that in cirrhosis of the liver profound salt retention can occur in the presence of a normal glomerular filtration rate. It is not doubted that for a given patient the capacity to eliminate sodium will be greater at higher than at lower rates of filtration(14) but this does not imply that a low filtration rate is the cause of sodium retention. The present data indicate that sodium retention in cirrhosis of the liver is due to excessive tubular reabsorption since a normal sodium load is presented to the tubule. This observation is in accord with the findings of Goodyer and his group(5). In addition, they were able to demonstrate that patients with cirrhosis still failed to eliminate sodium in the normal fashion even though the sodium load was increased by the intravenous infusion of 5% saline.

Summary. Glomerular filtration and renal plasma flow were found to be normal in eleven patients with cirrhosis of the liver who were accumulating edema fluid indicating that the retention of sodium is due to increased tubular reabsorption.

14. Berliner, R. W., *Am. J. Med.*, 1950, v9, 541.

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Compensatory Hypertrophy of the Intact Adrenal of Fetal Rats Subjected to Unilateral Adrenalectomy.* (18359)

RALPH L. KITCHELL. (Introduced by L. J. Wells.)

From the Department of Anatomy, University of Minnesota.

There are several lines of evidence that the mammalian adrenal begins to function prior to birth. The suprarenal cortex of the human fetus becomes markedly enlarged(1). Obviously this is only circumstantial evidence, and, as has been pointed out(2), the physio-

logical significance of this hypertrophy is not understood. It has been demonstrated that pregnancy prolongs the life of adrenalectomized dogs(3,4). The removal of the adrenals of pregnant rats causes an increase in the weight of the adrenals of the fetuses (5,6). The removal of the hypophysis of fetal rats by decapitation inhibits the growth of the adrenal(7,8), as does also the destruction of the hypophysis of fetal mice by ir-

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1. Scammon, R. E., *The Measurement of Man*, Univ. of Minn. Press, Minneapolis, Part IV, 1930.

2. Windle, W. F., *Physiology of the Fetus*, W. B. Saunders Co., 1940, xiii + 249.