TABLE II.
Blood P Fraction, Readily Hydrolysable by N Mineral Acid, in Infants with Rickets.

Case No.	Red cells vol. %	Mg. P per 100 cc. Serum Ortho-phosphate	Whole Blood Ortho-phosphate	Whole Blood Acid hydro-lysable phosphate	Red Cells Ortho-phosphate	Red Cells Acid hydro-lysable phosphate
1	36	4.4	4.1	3.9	3.6	10.8
2	30	1.6	1.0	2.3	—0.4	7.7
3	32	3.1	2.2	3.8	0.3	11.9
4	24	3.1	2.9	3.3	2.3	13.8
5	29	3.0	2.4	3.6	1.0	12.4
6	33	5.1	3.3	4.5	—0.3	13.5
7	33	—	2.1	4.4	—	13.2
8	33	2.4	1.6	3.9	0.0	11.7
9	26	2.1	1.2	3.2	—0.2	12.3
10	44	1.8	1.3	5.0	0.7	11.4
11	28	1.4	1.2	2.8	0.7	10.0
12	28	3.0	2.7	3.6	1.7	12.8
13	33	5.3	4.4	4.2	2.7	12.6
14	35	3.1	2.3	4.7	0.9	13.4
15	41	2.4	1.7	2.8	0.7	6.8
16	33	3.6	2.4	3.1	0.0	9.3
17	35	1.7	1.3	3.4	0.6	9.7
18	39	1.7	1.4	4.6	1.0	11.8
19	33	3.3	2.6	3.8	1.2	11.4
Average		2.95	2.28	3.76	1.03	11.40
Standard deviation		1.14	1.24	0.69	1.05	1.89
Coefficient variation %		38.7	54.3	18.4	102.0	16.6

No. 10 was an older child.

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Experimental Production of Chronic and Sub-acute Adrenal Insufficiency in Dogs and Cats.

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Chronic adrenal insufficiency, clinically and pathologically comparable with Addison's disease, has not heretofore been successfully produced in experimental animals. This has been attempted by various procedures, especially excision or denervation of the adrenals, destruction by mechanical, thermal, bacterial or chemical agents and by radiation, and ligation of blood vessels.

In 1917, I performed a number of experiments, upon cats and dogs, in which I practised mass ligation, ligation of the adrenal vein and ligation of the lumbo-adrenal vein on both sides of the gland. In general, my results were similar to those of others.[1, 2]

[1] Torrini, U. L., *Sperim.*, 1910, **64**, 342.

[2] Hartman, F. A., and Blatz, W. E., *Endoc.*, 1919, **3**, 137.

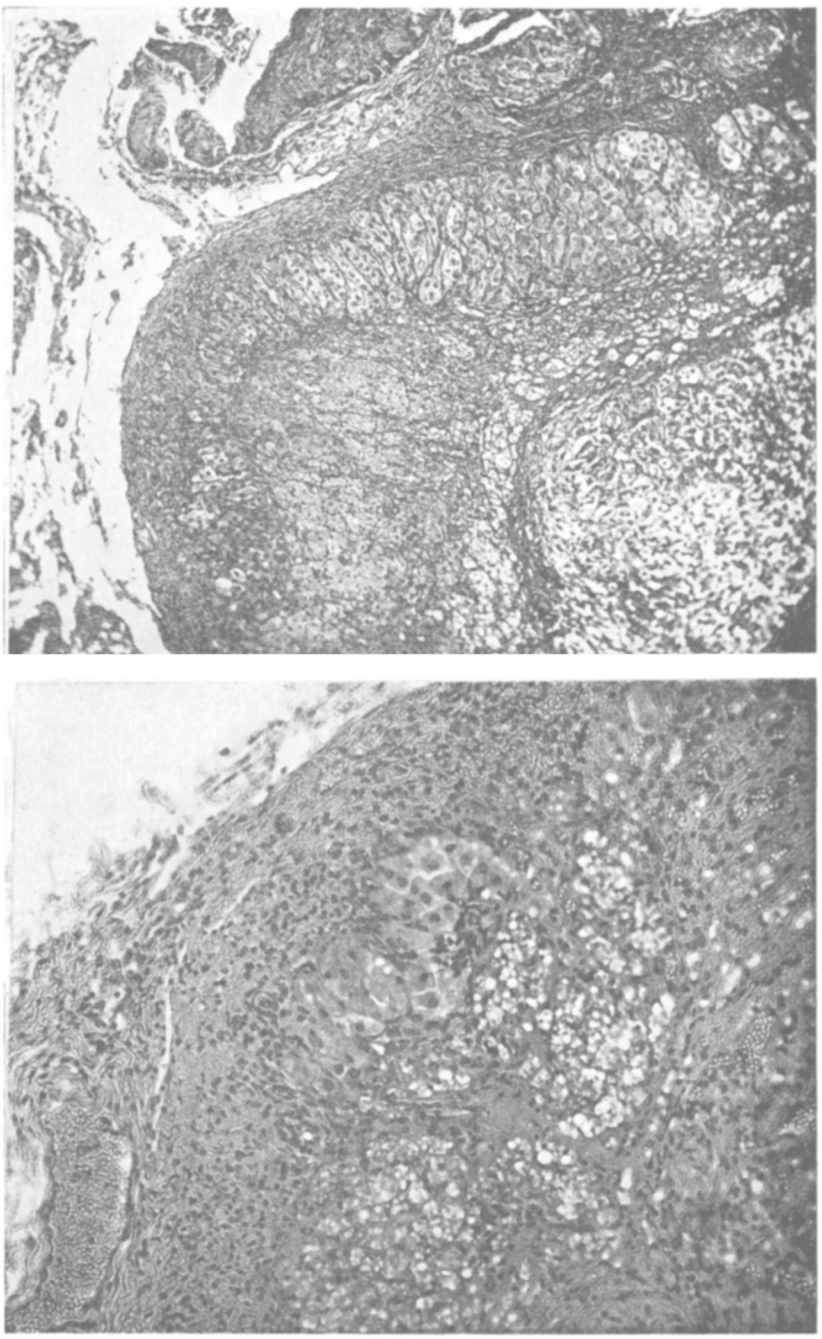

FIG. 1.

Photomicrographs of sections of cat's adrenals showing (left gland, above) small zone of surviving (regenerated) cortical tissue, degenerated and necrotic cortex, well preserved medulla, 83 ×; right gland (below) nodule of regenerated cortical cells, lymphocytic infiltration and fibrosis of cortex, degenerated and necrotic cortex, well preserved medulla, 180 ×.

I have observed a number of cases of Addison's disease among which were found at autopsy, pathological changes in the adrenal glands similar to those described by Kovács,[3] as "Cytotoxische Schrumpfnebenniere" and "Vaskuläre Schrumpfnebenniere". The excellent discussion by Kovács, together with my observations on adrenal insufficiency in laboratory animals and on Addison's disease, renewed my interest in the possibility of inducing circulatory and other changes in the adrenals which might lead to chronic insufficiency.

Physiological and clinical observations suggested the possibility that certain types of adrenal atrophy, associated with Addison's disease, may be results of the immediate effects of circulatory disturbances associated with thrombosis and anemic infarction. Accordingly, attempts were made to create comparable lesions by *subtotal* ligations of adrenal blood vessels. This was accomplished, sometimes in a single operation (bilaterally, or unilaterally after excision of the opposite gland), sometimes following the first operation by one or more subsequent operations, to create additional circulatory disturbances by ligating vessels of the collateral circulation (which often become enlarged after the first operation). A number of animals developed clinical manifestations of sub-acute and chronic adrenal insufficiency, in most instances comparable with Addison's disease. The physiological and anatomical significance of the collateral venous anastomoses in relation to the adrenals has been considered by Cow[4] and others. Its relation to thrombosis of the suprarenal vein has been studied by Seecof.[5]

Survival periods of the animals, in the group observed, range from about 2 to 14 months. Among those surviving long periods we have observed acute phenomena (anorexia, vomiting, asthenia and nervous symptoms) at intervals and finally an acute condition which terminates fatally. During these periods of acute (or subacute) adrenal insufficiency we have observed blood changes similar to those occurring in adrenalectomized animals, though apparently less severe. Laparotomy, performed during the survival of some animals, revealed the same remarkable congestion of the pancreas observed by us following double adrenalectomy.

At autopsy, the adrenal glands showed various degrees and extent of degeneration and necrosis. In most cases, the pathological changes in the adrenals consisted of *selective* degeneration of the cor-

[3] Kovács, W., *Ziegler's Beitr.*, 1928, **79**, 213.

[4] Cow, D., *J. Physiol.*, 1914, **48**, 443.

[5] Seecof, D. P., *Proc. N. Y. Path. Soc.*, 1926, **26**, 126.

tex, the medulla remaining intact (in others the medulla shared in extensive degeneration and necrosis) and zones of regenerated cortical cells were present, presenting a picture (Fig. 1) comparable, if not identical, with that seen in so-called "cytotoxic atrophy" associated with Addison's disease.

In a few experiments it was possible to create pathological changes in the adrenals leading to purulent infiltration or areas of purulent necrosis, by superposing infection on the degenerative processes induced by sub-total ligation. Frequently, this procedure leads to total acute purulent necrosis of the gland.

In some experiments the glands were removed for examination during periods of good health, in others when clinical symptoms were present. These and other observations are still in progress. The experiments thus far performed constitute a foundation for studies which may lead to a better understanding of Addison's disease and of the function of the adrenal cortex.

It is a pleasure to acknowledge my gratitude to Doctors D. P. Seecof and H. Goldblatt for valuable aid in preparation and interpretation of much of my experimental and human pathological material.

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Studies in Hypertension. I. Injection of Various Substances, Especially Amino-Acids, upon Blood Pressure of Rabbits.*

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The smallest dose of the selected substances which would produce histological changes in the kidneys of rabbits was first determined. Doses approximately one-half this amount were then injected daily intramuscularly and the effect upon the blood pressure studied. Uranium nitrate, lead acetate, phosphorus, ergot, viosterol, cholesterol, lecithin, putrescin, tyramine, arginine carbonate, asparagine, glutamic acid, histidine, tyrosine, tryptophane, succinic acid, aspartic acid, and guanidine carbonate were used. Of this entire group aspartic acid and guanidine alone produced progressive increase in systolic blood pressure.

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