11016 P

Existence of an Endocrine Gland in the Media of the Renal Arterioles.

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The juxta glomerular apparatus is composed mainly of smooth muscle cells devoid of myofibrils called afibrillar cells (abbr: a. f. cells).[2,4] In the superficial cortical zone of the normal kidney of the rabbit, these cell-groups show a glandular cycle culminating in the formation of acidophil or basophil secretion granules intermingled with minute vacuoles (Bouin-Hollande or Zenker-formol fixation). They are in close contact with the lumen of the vas afferens or with capillaries. The endocrine features of these cells are as definite as those of the chromophil cells of the anterior pituitary. Three weeks of moderate constriction of the left renal artery of young rabbits (Drury technic[1]) which causes the shrinkage of a few glomeruli, are followed by an increase in number and size of the granulated cells in the superficial juxta glomerular apparatuses. (R. 31-91; 31-92; 31-93.) After the same lapse of time a more pronounced constriction causes the hyalinization of the superficial glomeruli and the regression of many tubuli, but leaves the renal arterioles patent. (R. 31-96; 31-97; 31-97). In these kidneys the increase in number of the granulated, a. f. cells is remarkable. Not only do they exist at the vascular pole of the intact or hyalinized glomeruli but they invade the latter and become conspicuous in the wall of all the arterioles of the cortex; a considerable number of smooth muscle cells still spindle-shaped, change into granulated a. f. cells. The granules become basophil. (Masson's trichrome technic.)

Identical changes occur in the ischemic kidney of the dog, but are not so readily recognized because the a. f. cells do not contain secretion granules normally. From a renewed survey of the ischemic kidneys of the 12 dogs referred to in a previous communication[3] the behavior of the a. f. cells in acute or subacute experiments can be summarized as follows: hypertrophy, hyperplasia and vacuolation of the a. f. cells of the juxta glomerular apparatus; occasional pro-

1 Drury, D. R., *J. Exp. Med.*, 1938, **68**, 693.

2 Goormaghtigh, N., *J. Physiol.*, 1937, **90**, 1263.

3 Goormaghtigh, N., and Grimson, K., PROC. SOC. EXP. BIOL. AND MED., 1939, **42**, 227.

4 Goormaghtigh, N., and Handovsky, H., *Arch. Path.*, 1938, **26**, 1144.

trusion of the a. f. cells in the glomerular tuft; hyperplasia of the a. f. cells of the vas afferens followed by glomerular regression; transformation of ordinary smooth muscle cells into a. f. cells: a process which is accompanied by mitotic activity. In chronic cases of eight and seventeen months 'duration all the juxta glomerular apparatuses are hyperplastic and secretion granules appear in the a. f. cells while no qualitative changes occur in the tubules.

Since renal ischemia causes hyperplasia and hypertrophy of the a. f. cells which in the control rabbit have cytologic features of endocrine activity; since carefully graduated ischemia stimulates the a. f. cells at the exclusion of any qualitative changes in the tubules and favors the apparition of secretion granules in the a. f. cells of the dog, normally devoid of them, it must be concluded that the endocrine activity of the a. f. cells is related to the production of the hypertensive substance present in the ischemic kidney. It is suggested that in normal conditions the a. f. cells regulate the tonus of the renal arterioles.

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Effect of Added Glucose on Rate of Appearance of Free Sugar in Liver Brei.

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Previous work has shown that a rise in the blood sugar level in the normal animal causes a compensatory decrease in the sugar output of the liver.[1, 2] The operation of this homeostatic mechanism to maintain the normal blood sugar level depends upon the presence of the equal and opposite influences of insulin and other hormones. But, providing this endocrine balance is normal, no extra insulin need be secreted for each regulatory action.[1] Indeed, the regulation has been demonstrated in the "Houssay animal," in which insulin and the anterior pituitary hormones are entirely lacking.[3] The ab-

[1] Soskin, S., Allweiss, M. D., and Cohn, D. J., *Am. J. Physiol.*, 1934, **109**, 155.

[2] Soskin, S., Essex, H. E., Herrick, J. F., and Mann, F. C., *Am. J. Physiol.*, 1938, **124**, 558.

[3] Soskin, S., Mirsky, I. A., Zimmerman, L. M., and Heller, R. C., *Am. J. Physiol.*, 1936, **114**, 648.