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Rôle of Liver in Depression of Plasma Potassium Level by Epinephrine.

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The decline in blood potassium occurring shortly after beginning infusion of epinephrine has been shown not to be due, probably, to depletion of the liver of its potassium stores since the rapid injection of 0.02 mg epinephrine during the infusion transiently raises the blood potassium level.[1] It seems most likely that this decline of potassium level in the blood is due to its entry into the tissues, probably muscle and possibly also back into the liver itself.

In order to investigate this latter possibility epinephrine was infused, over periods up to 45 minutes, into the femoral vein of cats under sodium pentobarbital anesthesia. Blood samples were taken simultaneously from the portal vein and hepatic vein at various times during the course of the infusion. The serum was analyzed for potassium by the method of Kramer and Tisdall;[2] the results are shown in Table I.

TABLE I.
Comparison of Blood Potassium from Hepatic Vein and Portal Vein During Infusion of Epinephrine 1:75,000 at Rate of 1 ml per Minute.

Animal No.	After 15 min infusion		After 30 min infusion		After 45 min infusion	
	Hepatic mg%	Portal mg%	Hepatic mg%	Portal mg%	Hepatic mg%	Portal mg%
1	13.9	16.0	—	—	—	—
2	—	—	12.2	12.5	11.4	12.3
3	10.3	11.7	11.0	12.9	10.0	11.0
4	13.6	13.6	12.4	13.9	14.1	14.3
5	9.0	13.5	12.6	13.9	—	—
6	12.0	14.8	11.3	14.6	—	—

It will be seen that there is almost invariably a difference, small but occurring consistently and probably significant, between hepatic and portal blood potassium concentration. Further, it is the portal blood that contains the larger amount and the hepatic the lesser. This would seem to signify that the liver is removing at least some of the potassium being carried to it by the blood.

[1] Brewer, George, Larson, P. S., and Schroeder, A. R., *Am. J. Physiol.*, 1939, **126**, 708.

[2] Kramer, B., and Tisdall, F. F., *J. Biol. Chem.*, 1921, **46**, 339.

It would seem that if the liver is the only source of the potassium mobilized in response to the stimulation of epinephrine[3] it probably continues to release it during the entire time when the epinephrine level of the blood is elevated. However, the liver, as well as the other tissues, very rapidly begins removing potassium from the blood and at such a rate that the blood potassium level is depressed appreciably below the resting normal. The mechanism by which this is accomplished is not clear and whether it depends upon metabolic or purely vascular factors cannot be said at this time.

Summary. During infusion of 1:75,000 epinephrine for as long as 45 minutes at the rate of 1 ml per minute the potassium content of the blood entering the liver, although actually below the resting concentration still exceeds that of blood leaving the liver. This strongly suggests that the cell source from which potassium is mobilized by epinephrine takes part, at least, in removing the excess from the circulation.

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Thymus and Lymph Nodes Following Adrenalectomy and Maintenance with NaCl in the Rat.*

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The existence of a relationship between the thymus gland and the adrenal cortex has been well established. Adrenalectomy has been reported as causing thymic regeneration,[1] and it has been shown that the inability of the thymus to undergo involution in response to alarming stimuli after this operation is not altered by the administration of sodium chloride.[2] Cortin[3] and adrenocorticotropic hormone[4] have been shown to cause thymic involution. Clinical reports

[3] D'Silva, J. L., *J. Physiol.*, 1936, **87**, 181.

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[1] Jaffe, H. L., *J. Exp. Med.*, 1924, **40**, 325.

[2] Selye, H., *Brit. J. Path.*, 1936, **17**, 234.

[3] Ingle, D. J., PROC. SOC. EXP. BIOL. AND MED., 1938, **38**, 443.

[4] Evans, H. M., Moon, H. D., Simpson, M. E., and Lyons, W. R., PROC. SOC. EXP. BIOL. AND MED., 1938, **38**, 419.