Quick;[3] there is still controversy as to results obtained by its use in thyrotoxicosis.

Conclusions. Increased thyroid activity is accompanied by decrease in the amount of the blood amylase, and vice versa. There is a pronounced fall in the level of blood amylase within 8 hours following thyroidectomy in thyrotoxic patients; this does not occur after operations for non-toxic goiter. The return of blood amylase to normal values following thyroidectomy lags behind the improvement in clinical status and the restoration of basal metabolism to normal level. These events are considered to be evidence of impaired liver function in thyrotoxicosis.

9423

Action of Atropin and Eserin on Adrenalin Secretion Caused by KCl and CaCl.*

GERHARD KATZ AND GERTRUD KATZ. (Introduced by J. T. Halsey.)

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The introduction of $CaCl_2$ or KCl into the adrenal circulation is known to cause a secretion of adrenalin (Bacq and Rosenblueth,[1] Feldberg,[2] Katz and Katz[3]). In the present investigation we have endeavored to determine whether this adrenalin secretion in cats can be influenced by previous injections of atropin or eserin, assuming that atropin may decrease the amount of adrenalin secreted upon $CaCl_2$ or KCl injection, while eserin may enhance it. Such results would justify the working-assumption that the salts act on the adrenal medulla by means of an acetylcholin-like transmitter, as has been shown for the action of KCl on other organs such as salivary glands, tongue, sweat glands (Feldberg and Guimarais[4]), or for the transmission of splanchnic stimulation to the adrenals (Feldberg and coworkers[5, 6]).

Our method of recording the adrenalin output effected by the

[3] Quick, A. J., Arch. Int. Med., 1936, **57**, 544.

* Aided by a grant from the David Trautman Schwartz Research Fund.

[1] Bacq, Z. M., and Rosenblueth, A., *Am. J. Physiol.*, 1934, **108**, 46.

[2] See 4.

[3] Katz, G., and Katz, G., *J. Pharm. and Exp. Ther.*, 1937, **59**, 284.

[4] Feldberg, W., and Guimarais, J. A., *J. Physiol.*, 1936, **86**, 306.

[5] Feldberg, W., and Minz, B., *Pfluegers Arch.*, 1933, **233**, 657.

[6] Feldberg, W., Minz, B., and Tsudzimura, H., *J. Physiol.*, 1934, **81**, 286.

salts was the one developed by Feldberg and coworkers[7] and used in our previous investigations on $CaCl_2$ and KCl.[3] It consisted in comparing the effects on blood pressure and nictitating membrane produced by the salt when injected into a vein or into the central stump of the coeliac artery. The animal was eviscerated and with restricted circulation (aorta and vena cava tied at kidney level, the kidney vessels tied close to the kidneys). A small dose of the drug causes little or no effect upon intravenous injections, while the same amount injected into the central end of the coeliac artery reaches the adrenals in more or less concentrated form and may cause an adrenalin secretion. This latter adrenalin secretion manifests itself by a rise in blood pressure and a retraction of the denervated nictitating membrane after some latent period. These effects may be abolished by removal of the adrenals or enhanced by cocaine administration. They persist in decerebrated and pithed animals. Cats under dial anesthesia were used.

It was found that 0.2 mg. of atropin i.v. diminish the adrenalin secretion caused by Ca or K for a period of about 5 minutes (Fig. 1), while the effects of injected adrenalin remained unaltered. This

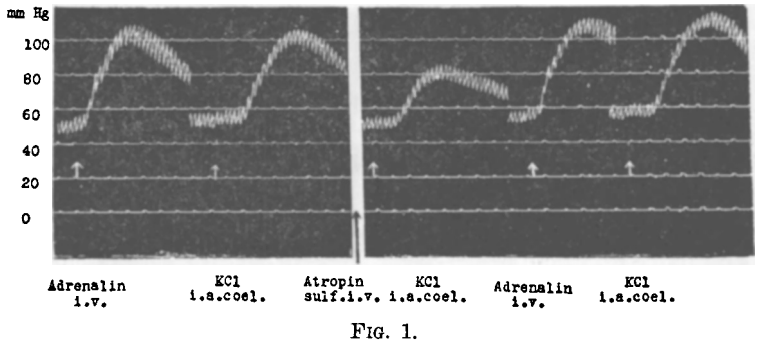

FIG. 1.

Eviscerated cat, Dial anesthesia, restricted circulation. Effects on blood pressure of 2.5γ adrenalin, intravenously, and 1.25 mg. KCl into central end of coeliac artery before and 2-10 min. after intravenous injection of 0.2 mg. atropin.

short action of the atropin on the adrenals corresponds to the observation of Feldberg and Minz on adrenalin secretion caused by injected acetylcholin.[7] They found that an injection of acetylcholin discharged a decreased amount of adrenalin after intravenous injection of 2 mg. of atropin and that this effect lasted for about 2 minutes only.

In our experiments, incidentally, we have often observed that atropin (1-2 mg. given intravenously) relaxes the denervated nic-

[7] Feldberg, W., and Minz, B., *Arch. Exp. Pharm.*, 1931, **163**, 66.

titating membrane. This observation is in contrast to Rosenblueth's report,[8] which stated that atropin has no action *per se* on the nictitating membrane. An explanation for this divergence may be suggested by the fact that we observed this effect only in membranes which were not completely relaxed at the time, while Rosenblueth may have studied the drug on relaxed membranes only.

The effects of a previous injection of eserin (0.4-0.8 mg., intravenously) on the adrenalin secretion caused by injections of $CaCl_2$ or KCl have not been constant. In some experiments we have found no increase in response of blood pressure or nictitating membrane, while in others the reactions of both were enhanced, either in height or in length, although the effects of injected adrenalin were not modified (Figs. 2 and 3). However, in some experiments eserin unexpectedly enhanced the effects of injected adrenalin as well.†

The inconsistent findings in this part of our investigation, there-

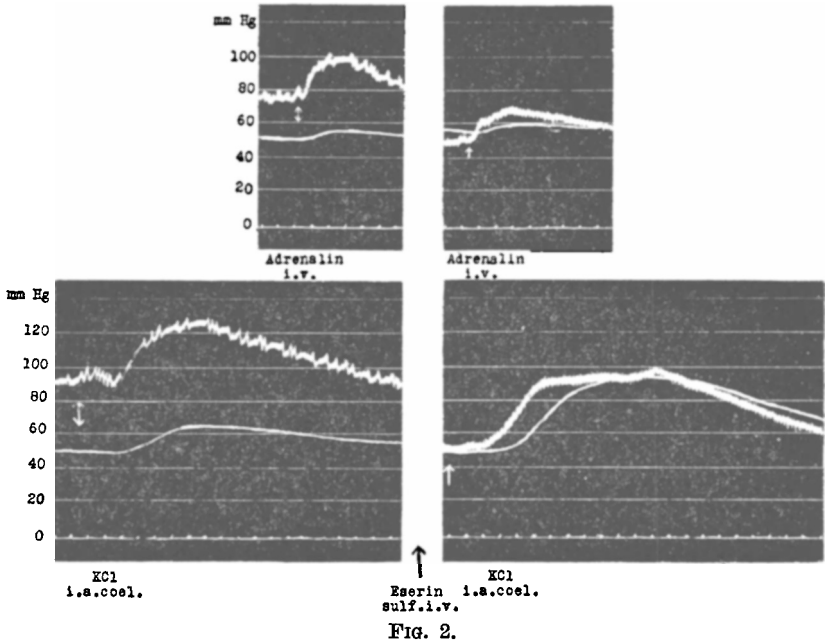

FIG. 2.

Upper curves: blood pressure; lower curves: acutely denervated nictitating membrane. Cat, Dial anesthesia. Preparation as in text. Effects of 5γ adrenalin, intravenously, and 5 mg. KCl into coeliac artery before and after intravenous injection of 0.5 mg. eserin sulf.

[8] Rosenblueth, A., *Am. J. Physiol.*, 1932, **100**, 443.

† After this work was completed J. Secker (*J. Physiol.*, 1937, **89**, 296) reported that the action of adrenalin (like the effects of nervous stimulation) on the nictitating membranes of cats can be enhanced by previous administration of eserin; we observed this effect only occasionally.

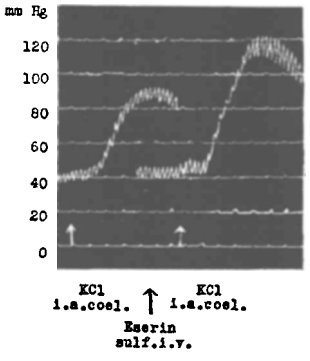

FIG. 3.

Cat. Preparation as in text. Effects on blood pressure of 1.25 mg. KCl injected into the central end of coeliac artery before and after intravenous injection of 0.4 mg. eserin sulfate.

fore, do not yet allow us to conclude that $CaCl_2$ or KCl discharge adrenalin by a cholinergic mechanism. Moreover, we do not believe that in studies of the autonomic nervous system, the effects of eserin and particularly those of atropin by themselves prove whether or not a mechanism is cholinergic. In our experiments, a cholinergic action of $CaCl_2$ or KCl on the adrenals can be definitely proved only by actual demonstration (by bioassay) of the liberation of acetylcholin in the glands following injection of the salts.

9424 P

Neurotoxic Action of Aluminum Salts.

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In the course of experiments involving the fractionation of herpes virus preparations by aluminum salts it was observed that rabbits which had received an intracerebral injection of certain fractions developed a syndrome indicative of a lesion of the central nervous system. The possibility of herpes encephalitis was excluded by the dissimilarity in symptoms and in cellular pathology. Nevertheless, several features of this syndrome, which had a uniformly fatal termination, were suggestive of a neurotropic virus disease, and a further investigation was made of its etiology.