adrenals and perhaps via adrenalin secretion. It is hoped that further studies on adrenal demedullated rats may throw some light on this point.

The mechanisms which account for the difference between the mono and dicarboxylic amino acids and between glutamic and aspartic acid are obscure at present. It may be of interest to note that Awapara(10) has reported that the adrenals appear to play some significant role in the metabolism of the dicarboxylic amino acids.

Summary and conclusions. 1. A study was made to determine whether the oral administration of glutamic acid produced an increased secretion of adrenalin by employing the changes in the blood eosinophil and lymphocyte level as indicators. 2. Glutamic acid, aspartic acid, glycine, alanine, and water or saline were administered to a group of white rats before and after adrenalectomy and the effects on the levels of the blood eosinophils and lymphocytes were determined. 3. Only the dicarboxylic amino acids produced a statistically significant depression of the eosinophils in the normal and adrenalectomized animals. However, in the latter animals the lymphocytes also were depressed by aspartic acid but not by glutamic acid. 4. The results obtained in adrenalectomized rats showed that the levels of the blood eosinophils and lymphocytes could be influenced by other mechanisms than the pituitary-adrenal system. 5. The possible significance of the small difference observed between normal and adrenalectomized rats regarding eosinophil changes is discussed.

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Effects of Adrenal Ligation and Adrenergic Blockade on Pressor Responses to Acetaldehyde in Dogs.* (19725)

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The use of tetraethylthiuram disulfide (Antabuse) in the treatment of chronic alcoholism has stimulated interest in the mechanisms involved in the Antabuse-alcohol reaction. At the present time, it is believed that the acetaldehyde formed from ingested alcohol produces the symptoms of the Antabuse-alcohol reaction in which hypotension is characteristic(1). Nelson(2) has shown that acetaldehyde causes a transient pressor response in dogs after both carotid arteries are clamped, the vagus nerves sectioned, the adrenal vessels ligated, and after ganglionic blockade induced by nicotine. Reversal of the pressor response by the adrenergic blocking drug 2-(N, p-tolyl-N [m′ oxyphenyl] aminomethyl) imidazoline (C-7337) has been demonstrated by Christensen(3) to occur in the intact anesthetized dog.

These observations indicate that acetaldehyde is a peripherally acting drug. The present experiment was undertaken to confirm 2 aspects of the evidence: 1) that the pressor response is not due to the release of epinephrine from the adrenal medulla; 2) that the pressor response is reversed by an adrenergic

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TABLE I. Effect of Adrenal Ligation and SY-28 on Blood Pressure Responses to Epinephrine and Acetaldehyde in Dogs.

Procedure	Mean dose & S.D. l-epinephrine tartrate, γ/kg	Mean pressor response & S.D., mm Hg	Mean dose & S.D. acetaldehyde, mg/kg	Mean pressor response & S.D., mm Hg
Before adrenal ligation	4.67 ± 2.76	70 ± 41.8	9.37 ± 1.76	45.7 ± 15.9
After adrenal ligation	4.07 ± 3.31*	57.4 ± 41.8*	9.37 ± 4.96	24 ± 7.73
		Mean depressor response & S.D., mm Hg		Mean depressor response & S.D., mm Hg
After† adrenal ligation & after SY-28	1.1 ± .86	28.5 ± 19.2	8.25 ± 3.46	22.5 ± 5.9

* Seven observations.
† Four experiments; all other data represent 8 experiments.

blocking agent after ligation of the adrenal vessels.

Procedure. In 4 male and 4 female dogs anesthetized with pentobarbital sodium (approximately 30 mg/kg) the right common carotid artery and right femoral vein were cannulated. Blood pressure was recorded by a mercury manometer. Prior to and following a one-stage bilateral ligation of the adrenal vessels, acetaldehyde (Mallinckrodt), and l-epinephrine tartrate were given intravenously as *statim* injections. After the second series of injections, the adrenergic blocking agent N-(2-bromoethyl)-N-ethyl-1-naphthalenemethylamine • HBr (SY-28) was administered intravenously over a period of 10 minutes to 2 male and 2 female dogs at a dosage of 1 mg/kg. Fifteen minutes later presence of adrenergic blockade was tested by injecting l-epinephrine tartrate. When blockade was complete, as shown by epinephrine reversal, acetaldehyde was given. The completeness of the adrenal ligation was confirmed at autopsy in all animals.

Results. The results of these experiments, presented in Table I, indicate that acetaldehyde causes a pressor response in dogs before and after the adrenal vessels are ligated. Following the adrenal ligation, the pressor response is reversed by an adrenergic blocking agent.

Discussion. This experiment confirms the observation of Nelson(2) that the pressor response to acetaldehyde is not wholly due to the release of epinephrine from the adrenals. Moreover, the pressor response is reversed by SY-28 after the adrenal vessels are ligated. The fact that statim injections of acetaldehyde and its continuous administration as a volatile anesthetic(4) produce a rise in blood pressure in dogs is at variance with the present belief that acetaldehyde causes the hypotension of the Antabuse-alcohol reaction in humans.

Summary. 1. The pressor response to acetaldehyde in dogs is not wholly due to the release of epinephrine from the adrenals. 2. The pressor response to acetaldehyde is reversed by the adrenergic blocking drug SY-28 following bilateral ligation of the adrenal vessels.

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