made it clear that no rise in systemic arterial pressure occurred during or following the adrenalin injections. At the same time it was noted that the accelerating action of adrenalin upon the heart was not inhibited by the Dibenamine premedication. As a control, two dogs were given Dibenamine hydrochloride alone, 20 mg/kg, intravenously, each day for 3 days.

Autopsies were performed promptly with the termination of each experiment and histologic study of the tissues was carried out.

Animals of the first two groups—which received massive doses of adrenalin alone—showed striking changes in the coronary and pulmonary arteries, aorta, and myocardium. There were no comparable cardiovascular lesions, except for a few scattered areas of myocardial necrosis in the animals of the third group, which received Dibenamine followed by adrenalin. No cardiac or arterial changes were found in the dogs of the fourth group, given Dibenamine alone.

Segmental necrosis of many of the small coronary arteries and arterioles, as well as extensive hemorrhages and necrosis of the pulmonary artery and aorta, was present in the dogs given adrenalin. Hemorrhage was prominent also in the coronary vessels, often occurring, as in the aorta, at the origin of branches. Medial necrosis of the coronary arteries was frequently accompanied by periarteritic cellular inflammatory exudate or by perivascular fibrosis, depending on the age of the lesion. Necrosis of the aortic adventitial vasa vasorum was prominent. An occasional necrotic arteriole of the gastric submucosa was also encountered. There were no renal or cerebral arterial lesions. Interstitial myocardial edema and focal myocardial necroses were observed. Ten of the 12 dogs receiving adrenalin alone had many lesions of the types described. Two had only minimal changes.

The necrosis of coronary arteries observed in the present experiments following injections of adrenalin reproduces faithfully the acute arterial changes often encountered throughout the body in rapidly developing hypertension in man. Similar arterial necroses are well known in dogs and other animals following certain types of experimentally produced renal insufficiency (*5*).

In the present experiment pretreatment with Dibenamine abolished the rise in systemic pressure of subsequent adrenalin injections and prevented the development of arterial lesions. This suggests that in the untreated groups the severe hypertension was a critical factor in the etiology of these changes. Recent experiments of Byrom and Dodson (*1*) have suggested a direct relationship between increased intra-arterial pressure and necrosis of renal arterioles. Experiments are now in progress to see if adrenolytic drugs will prevent the development of acute arterial damage that follows other experimental procedures.

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The Presence in Animal Organs and Human Blood of a Peptide Detected by Paper Chromatography

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Recently, Borsook *et al.* (*1*) reported the existence of a peptide fraction in various tissues of several animal species. This fraction was obtained from extracts of tissues by means of chromatography on starch columns. In this laboratory we have detected a similar or perhaps identical fraction in several organs of the rat, in human blood and tumors, and in Witte's peptone. The fraction was detected first, and isolated later, exclusively by means of paper chromatography (details for the isolation technique to be reported later). Analytical data revealed that this peptide fraction contains 14% nitrogen and is made up of at least the following amino acids: tyrosine, methionine, proline, arginine, hydroxyproline, leucine and/or phenylalanine, alanine, serine, glycine, threonine, glutamic acid, aspartic acid, and lysine.

At present there are insufficient data to warrant the assumption that this fraction is a single peptide. The chromatographic behavior of the fraction does not vary with the tissue of origin. In every case the R_f values were found to be 0.95 in phenol and 0.05 in 2,4-lutidine. Furthermore, the ratio of the value of amino nitrogen after hydrolysis to the value before hydrolysis averages 14.5, which is in close agreement with the value of 14.2 reported by Borsook for "peptide A."

It is conceivable that we are dealing with the same peptide detected by Borsook in various organs of several animal species.

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