ped phenylhydrazones or osazones were transformed in the corresponding 2,4-DP derivatives. The mixture was allowed to stand for some hours, then filtered, washed, and dried. The material was treated with boiling benzene, filtered, dried, and dissolved in 4 ml of nitrobenzene. To the solution 20 ml of a mixture of benzene, containing 25% of benzine (90–100°C), were added. After filtration, the solution was chromatographed in a Brockmann's alumina column. Development of the chromatogram was carried out with a mixture containing 67·5% of benzene, 22·5% of benzine, and 10% of nitrobenzene. Some red products remained on top of the column, the glyoxal 2,4-DP-osazone in the central portion, and methylglyoxal 2,4-DP osazone in the lower portion. The methylglyoxal arose from the triose phosphates of the endogenous respiration[11]. It was always present in all experiments in which the yeast cells used were not treated with 2,4-dinitrophenol. The glyoxal osazone was eluted with acetone and after partial evaporation of the solvent, it was precipitated with ethyl alcohol. The product melted at 325°C and no depression was observed on admixture with synthetic glyoxal-2,4-DP osazone. Amounts found, mg 20–40/l. Failure to trap larger amounts of glycolaldehyde depended on the fact that it is oxidizable also in the presence of phenylhydrazine, as mentioned above (see also[10]).

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Riassunto

Dall'ossidazione dell'acetato con cellule intatte di lievito, in presenza di un eccesso di fenilidrazina, è stato isolato l'osazone del gliossale, che rappresenta probabilmente l'aldeide glicolica. Si ritiene perciò che l'aldeide sia un intermedio dell'ossidazione dell'acetato a glicolato secondo la formulazione riportata nel testo.

Catecholamines and Histamine in Vascular Tissue of Normal and Depancreatised Dogs

Since the observations of von Euler[1], the presence of noradrenaline was demonstrated in different tissues, thus also in the walls of the blood vessels[2]. Special significance was attributed to this substance in the arteries by Burn and Rand[3], who have found that an artery deprived of its noradrenaline content by pretreating the animal with reserpine does not react in the usual way to vasoconstricting stimuli. Raab[4] supposes that the adrenaline content of the arteries is in some way connected with atherogenesis.

It seemed of interest to study the adrenaline and noradrenaline concentration in the arteries of normal and diabetic dogs, since the occurrence of late and even early vascular changes in human diabetes mellitus is well known[5].

The dogs were bled in morphine-chloralose narcosis through the carotid artery and segments of different arteries were excised. After washing with saline, drying on filter paper and weighing, the tissues were ground with quartz-sand and trichloracetic acid containing ascorbic acid. The suspension was centrifuged and the catecholamines were chromatographed on aluminium oxid. After washing, the catecholamines were eluted with acetic acid and the Euler-Floding reaction was performed. The reaction is specific for adrenaline at pH 3·45, while both catecholamines give the reaction at pH 5·45 as described previously[6]. The results are expressed in μg/g wet weight.

The results in normal dogs are summarised in Table I. With the exception of the femoral artery, the sum of the two catecholamines is 1·0–1·5 μg/g, adrenaline representing only 5–6% of the total.

Totally depancreatised dogs were maintained for 27–55 days on insulin. The treatment was abandoned three days prior to the experiment. The results are given in Table II. It is evident that, with the exception of the femoral artery, the adrenaline concentration increased significantly compared with the vessels of normal dogs ($p < 0{\cdot}05$). The noradrenaline concentration showed a decrease, but according to statistical analysis this decrease did not prove significant ($p > 0{\cdot}05$). The sum of the concentration of the two catecholamines showed a small decrease too. The most significant finding is the change in the percentage of the adrenaline fraction which proved to be 10–35% in

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Table I

	Number of dogs	Adrenaline (A)	Noradrenaline (NA)	Sum of A+NA	Percentage of A
Femoral artery	10	0·039 ± 0·005	0·470 ± 0·072	0·509 ± 0·077	7·6
Carotid artery	12	0·056 ± 0·006	1·112 ± 0·129	1·168 ± 0·135	4·8
Renal artery.	11	0·073 ± 0·007	1·445 ± 0·221	1·518 ± 0·228	4·8
Coeliac artery	7	0·075 ± 0·011	1·440 ± 0·284	1·515 ± 0·295	4·9
Abdominal aorta	9	0·093 ± 0·015	1·368 ± 0·305	1·461 ± 0·320	6·3

Mean adrenaline and noradrenaline concentrations expressed in μg/g tissue with standard deviations, in normal dogs.

Table II

	Number of dogs	Adrenaline (A)	Noradrenaline (NA)	Sum of A+NA	Percentage of A
Femoral artery	9	0·053 ± 0·007	0·307 ± 0·067	0·360 ± 0·074	14·7
Carotid artery	12	0·102 ± 0·022	0·890 ± 0·145	0·992 ± 0·167	10·2
Renal artery	12	0·191 ± 0·042	0·986 ± 0·214	1·177 ± 0·256	16·2
Coeliac artery	9	0·225 ± 0·045	0·697 ± 0·183	0·922 ± 0·228	24·5
Abdominal aorta	10	0·317 ± 0·060	0·602 ± 0·224	0·919 ± 0·284	34·6

Corresponding data on diabetic dogs, 27–55 days after pancreatectomy.

Table III

	Number of dogs	Adrenaline (A)	Noradrenaline (NA)	Sum of A+NA	Percentage of A
Femoral artery	4	0·045 ± 0·017	0·464 ± 0·030	0·509 ± 0·047	8·9
Carotid artery	4	0·058 ± 0·019	0·735 ± 0·234	0·793 ± 0·253	7·3
Renal artery	3	0·139 ± 0·039	1·718 ± 0·300	1·857 ± 0·339	7·5
Coeliac artery	4	0·095 ± 0·024	1·704 ± 0·204	1·799 ± 0·228	5·3
Abdominal aorta	3	0·169 ± 0·040	1·194 ± 0·565	1·363 ± 0·605	12·4

Corresponding data on diabetic dogs, 7–9 days after pancreatectomy.

Table IV

	Normal dogs (8)	Diabetic dogs 7–9 days after pancreatectomy (2)	Diabetic dogs 27–55 days after pancreatectomy (10)
Femoral artery	1·3 ± 0·2	0·9	0·9 ± 0·1
Carotid artery	1·1 ± 0·3	1·3	0·9 ± 0·1
Renal artery	4·2 ± 1·2	5·6	3·9 ± 0·6
Coeliac artery	6·3 ± 1·2	–	3·2 ± 0·4
Abdominal aorta	2·9 ± 0·7	2·6	3·1 ± 0·7

Mean histamine concentrations expressed in μg/g tissue with standard deviations. Number of animals in each group in parenthesis.

the different vessels of the pancreatectomised dogs. These values reached only 5–7% in normal dogs.

Pancreatectomy *per se* does not lead to similar alterations: only slight changes were found in the adrenaline concentration of the arteries of diabetic dogs killed 7–9 days after the operation (Table III).

For the determination of histamine—due to the presence of substances causing relaxation of the guinea pig ileum—some inadequacies were observed with the classic Barsoum-Gaddum-Code-Method. Therefore, before the biological test was performed, an extract was made with an ionic exchange resin[7]. The average concentration of histamine in the vessel walls was found to be 1–6 μg/g, no significant difference occurring between the three groups of animals (Table IV).

The importance of the above findings is as yet not clear. Whether adrenaline plays any rôle in atherogenesis is open to debate. According to RAAB[2], adrenaline may promote the fatty infiltration of the intima by causing local hypoxia. Its administration intensifies the deposition of cholesterol in the intimal tissue; catecholamine releasing mechanisms produce or aggravate vascular lesions. Although the physiological significance of the constancy of the adrenaline-noradrenaline ratio is as yet not well known, the biochemical abnormality described above may be in some correlation with the development of functional derangements and, after long standing, probably even with morphological changes of the blood vessels.

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Zusammenfassung

Der Adrenalin-, Noradrenalin- und Histamingehalt wurde in verschiedenen Arterien normaler und pankreaektomierter Hunde bestimmt. Die Konzentration von Adrenalin nahm in den Gefässen der seit 27–55 Tagen diabetischen Tieren zu, die von Noradrenalin mässig ab. Der Histamingehalt blieb unverändert. Der Anteil von Adrenalin am gesamten Katecholamingehalt der Gefässe war 5–6% in normalen, 10–35% in diabetischen Hunden. Es wird auf die Möglichkeit hingewiesen, dass die beschriebenen Veränderungen mit den diabetischen Gefässleiden im Zusammenhang stehen.

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