

Catecholamine Content of the Arterial Walls in Experimental Hypertension

The pathogenesis of the vascular lesions occurring in hypertensive disease is an unsolved problem. Among the different theories concerning this question, the assumption of RAAB¹ should be mentioned, according to whom the changes in the catecholamine content of the blood vessels may play a significant role in the development of hypertensive vascular disease.

In the following set of experiments, the consequences of long standing hypertension were studied. Hypertension was produced in adult mongrel dogs by clamping one renal artery, according to GOLDBLATT. The blood pressure of the animals was controlled by direct arterial puncture during the next months. After an interval of 3–8 months, the animals were sacrificed and the catecholamines of different arterial segments were determined, using the EULER-FLODING reaction, as described previously².

In one group of animals, the GOLDBLATT-technique was successful: the systolic blood pressure rose to 170–190 mm Hg. These animals gave the results summarized in Table I. It can be seen that the adrenaline concentration increased in all the arteries studied, while the noradrenaline concentrations were lower than the corresponding figures in normal dogs. According to statistical analysis, these changes are highly significant. The percentage of adrenaline in relation to the sum of the two catecholamines increased consequently from 5–8% of normal canine arteries to 15–42%.

In some animals, the clamping of one renal artery was not followed by a lasting hypertension. The adrenaline concentration was also elevated in some arteries of these animals, but not in others; the noradrenaline concen-

tration was not changed significantly (Table II). Comparing the results obtained in the two groups of operated animals, it can be seen that the adrenaline concentration is of the same order of magnitude, while the noradrenaline concentration is much lower in the hypertensive dogs.

Since the adrenaline concentration of the blood was found to be elevated in the hypertensive dogs (4–5 µg/l vs. 0.5–2.5 µg/l in normal controls), it may be supposed that the increased adrenaline concentration of the arteries was derived from the blood stream³. It is probable that substances liberated from the ischemic kidney are the causative agents in the increased production of adrenaline. From our data, we may conclude that the increase of arterial wall adrenaline is not in causative relation to the establishment of a lasting hypertension. There is no acceptable explanation at present for the decrease of the noradrenaline concentration seen in the arteries of the hypertensive dogs.

Zusammenfassung. In Hunden mit GOLDBLATT-Hypertonie ist die Adrenalin-konzentration der Arterien stark erhöht, diejenige von Noradrenalin aber – verglichen mit den Werten der Kontrolltiere – erniedrigt. Die Adrenalin-erhöhung in der Arterienwandung ist wahrscheinlich nicht die Ursache des Hochdruckes, da sie auch in Hunden ohne postoperative Blutdruckerhöhung gefunden wurde.

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¹ W. RAAB, *Amer. J. Cardiol.* 1, 133 (1958).

² I. FARE DIN, M. WINTER, B. TANOS, and G. HETÉNYI, *Exper.* 15, 389 (1959).

³ W. RAAB and W. GIGEE, *Angiology* 9, 283 (1958).

Tab. I. Results in hypertensive dogs

	Number of dogs	Adrenaline (A) conc.	Noradrenaline (NA) conc.	Sum of A + NA	% of A
Femoral artery	8	0.096 ± 0.022 (0.039 ± 0.005)	0.131 ± 0.026 0.470 ± 0.072	0.227 ± 0.048 0.509 ± 0.077	42.3 7.6)
Carotid artery	8	0.126 ± 0.015 (0.056 ± 0.006)	0.427 ± 0.090 1.112 ± 0.129	0.553 ± 0.105 1.168 ± 0.135	22.8 4.8)
Renal artery	7	0.305 ± 0.066 (0.073 ± 0.007)	0.512 ± 0.160 1.445 ± 0.221	0.817 ± 0.226 1.518 ± 0.228	37.3 4.8)
Coeliac artery	9	0.207 ± 0.030 (0.075 ± 0.011)	0.647 ± 0.140 1.440 ± 0.284	0.854 ± 0.170 1.515 ± 0.295	24.2 4.9)
Abdominal aorta	7	0.112 ± 0.021 (0.093 ± 0.015)	0.295 ± 0.082 1.368 ± 0.305	0.407 ± 0.103 1.461 ± 0.320	27.5 6.3)
Mean values and standard deviations of catecholamine concentrations in µg/g tissue wet weight. In parentheses corresponding values of 10 normal animals are given.					

Tab. II. Results in operated dogs failing to increase their blood pressure

	Number of dogs	Adrenaline (A) conc.	Noradrenaline (NA) conc.	Sum of A + NA	% of A
Femoral artery	3	0.058 ± 0.014	0.607 ± 0.077	0.665	8.7
Carotid artery	2	0.141 ± 0.045	0.777 ± 0.319	0.918	15.3
Renal artery	3	0.179 ± 0.031	1.388 ± 0.304	1.567	11.4
Coeliac artery	2	0.186 ± 0.061	2.105 ± 0.275	2.291	8.1
Abdominal aorta	3	0.106 ± 0.012	0.759 ± 0.282	0.865	12.2
Mean values and standard deviations of catecholamine concentrations in µg/g tissue wet weight.					