(PST) showing refractory periods of 1.0–1.8 msec. Its short latency is also indicative of its fibre-like nature. Moreover, no somatic unitary responses were recorded which were associated with the early component. This component most likely reflects the activity of the relatively coarse fibres of Zuckerkandl's bundle[10].

In this probe the negativity (N) associated with the synaptic activation of nDBB neurons appears at a depth of 3000 μ. Here, the latency to peak is 7.5 msec. As the probe descends the negativity steadily increases in amplitude and decreases in peak latency. At 5000 μ, the peak negativity has decreased to 5.5 msec. The relative stability of the peak latency with changes in stimulus intensity (not shown) and relative stability of the peak latency when recovering from paired-stimulus testing (PST) (see Figure B) is an indication of the monosynaptic nature of the N negativity. The conduction distance, for this instance, was estimated to be approximately 6 mm. A calculated conduction velocity for MFB fibres of 0.9–1.2 m/sec supports the notion that such field responses probably represent the monosynaptic excitation of cells.

The typical behavior of the field response with PST is illustrated in Figure B. Record specimens of test responses from 20 msec, 50 msec, and 100 msec interstimulus intervals (ISI) are presented. There is no substantial recovery in the 20 msec ISI, but the response is almost completely recovered in the 50 msec ISI. At 100 msec, there is actually a slight facilitation of the test response.

Extracellular unitary discharges appear out of the negative envelope (Figure C) of the field response. After penetration of the cell whose discharge was shown in Figure C, and after deterioration of the spike potential, a depolarizing-hyperpolarizing potential sequence remains. The intracellular recording shown in Figure D is shown at two different gains and with two different sweep speeds. The EPSP nature of the depolarizing potential is shown in Figure F.

The behavior of such depolarizing potentials was then examined by systematically changing the stimulus intensity (Figure F). With a stimulus intensity a 4.0 T (4 × threshold), a spike is issued. As the stimulus intensity is reduced the spike disappears and the depolarizing potentials decrease in amplitude. However, there is no significant increase in the latency to onset (the arrow indicates the onset at 2.3 T). The wave form, graded nature, and lack of latency shift for these depolarizing potentials indicate that they represent monosynaptic EPSPs.

Some of the cells which were orthrodromically activated by MFB input could be antidromically activated by IFim stimulation. An example is shown in Figure H and I. Figure I shows a unitary discharge recorded from the same cell, with a latency of 2 msec following IFim stimulation. The antidromic nature of this unit is evidenced by recovery in a 3.0 msec ISI.

The data presented here indicate that at least some of the projections coursing via the MFB are excitatory with respect to their target cells in the nDBB. This is in agreement with other electrophysiological studies[11,12] where MFB stimulation leads to the appearance of extracellular unitary activity in the nDBB.

Summary. The electrophysiological characteristics of the medical forebrain bundle (MFB) projections to the nucleus of the diagonal band of Broca (nDBB) were studied in acutely prepared cats. MFB stimulation evoked field potentials which consisted of a large negative wave followed by a shallow positivity. Extracellular unitary discharges appeared out of the negativity. In addition, intracellularly recorded EPSPs showed no significant shift in the latency to onset with changes in stimulus intensity. These observations indicate that at least some of the MFB projections to the nDBB are excitatory with respect to their target cells.

J. F. DEFRANCE and H. YOSHIHARA[13,14]

Morin Memorial Laboratory, Department of Anatomy, Wayne State University School of Medicine, 550 East Canfield Avenue, Detroit (Michigan 48201, USA), 10 March 1975.

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Blood Pressure Regulation in Spontaneously Hypertensive Rats[1]

Spontaneously hypertensive rats (SHR) from a Wistar strain[2], exhibit a sustained increase in vascular resistance, at least when they are 18 weeks or older[3]. Studies on SHR, as well as on human essential hypertension, suggest that an increased media thickness, partly encroaching upon the vascular lumen of resistance vessels, forms the main background of the increased flow resistance[4,5].

Increased vascular reactivity in SHR is also reported and attributed to an increased ionic permeability regulating smooth muscle tension[6–8]. In spite of the high blood pressure, efficient baroreflex regulation occurs. We described some histological aspects of intimal and medial vascular hypertrophy in the carotid arteries and aortic arch of our SHR colony[9].

The present study provides morphometric data on the medial vascular hypertrophy, which will diminish the transmission of the pressure pulse signal to the baroreceptors, located between the media and the adventitia[10].

Material and methods. Male SHR were compared to normotensive Wistar rats (NWR) of our own permanent laboratory colony established in 1956. The animals were transiently anaesthetized with ether and the femoral artery was catheterized under local anaesthesia with lidocaine. 1 h after recovery from the ether anaesthesia, heart rate, systolic and diastolic blood pressures were recorded. Thereafter, the rats were anaesthetized with Hypnorm® (haloanisone, 10 mg/kg and fentanyl, 0,1 mg/kg s.c.). Under free venous outflow, the arterial circulation was fixed retrogradely in situ with 2% glutaraldehyde in 0.1 *M* cacodylate buffer at a sustained perfusion pressure of 120 or 200 mm Hg. The internal carotid artery was taken at 2 mm from its origin and the aortic arch at 1 cm from the heart. The samples were postfixed, embedded, stained and transversely cut as previously described[9].

The perimeters of the inner and outer side of the media were measured with a curvimeter (precision = 1 mm) on

Matched pairs of normotensive (NWR) and hypertensive rats (SHR).

Strain	Age (months)	SBP (mmHg)	DBP (mmHg)	HR (beats/min)	Aortic arch			Left internal carotid artery			Right internal carotid artery		
					R_i (μm)	WT (μm)	WT/R_i	R_i (μm)	WT (μm)	WT/R_i	R_i (μm)	WT (μm)	WT/R_i
NWR	3	145	100	440	—	—	—	327	32	0.098	331	40	0.121
	3	160	110	390	1165	125	0.107	301	33	0.110	—	—	—
SHR	3	280	170	420	1049	164	0.157	337	50	0.148	333	54	0.160
	3	240	165	410	1185	164	0.139	363	45	0.121	289	60	0.208
NWR	7	130	90	390	1257	110	0.087	336	36	0.107	327	35	0.108
	7	150	95	420	1410	121	0.086	426	38	0.091	427	51	0.118
	7	90	55	390	1410	131	0.093	375	51	0.137	375	46	0.123
SHR	7	225	160	470	1059	143	0.135	352	65	0.183	334	65	0.148
	7	200	150	390	1194	173	0.145	425	60	0.142	375	71	0.189
	7	240	155	370	1143	165	0.144	387	69	0.178	471	62	0.131
NWR	12	150	95	420	1154	125	0.108	326	35	0.109	368	42	0.115
	12	125	85	360	1216	105	0.086	364	46	0.126	372	35	0.095
	12	135	85	450	—	—	—	350	37	0.106	327	30	0.092
	12	150	100	420	1704	118	0.069	391	41	0.104	382	46	0.120
	12	155	95	440	1329	142	0.107	467	46	0.098	373	43	0.114
	12	155	85	420	1124	141	0.125	359	39	0.108	384	44	0.115
median values:		150[a]	90[a]	420	1216	125[a]	0.107[a]	361[a]	40[a]	0.107[a]	372[a]	42[a]	0.115[a]
SHR	12	220	145	390	1274	195	0.153	582	68	0.117	413	63	0.156
	12	200	150	400	1330	151	0.114	482	62	0.129	361	52	0.143
	12	170	95	420	1122	177	0.158	407	66	0.164	—	—	—
	12	250	185	350	1384	223	0.168	495	73	0.150	532	71	0.134
	12	245	175	400	1308	188	0.144	482	69	0.143	491	64	0.132
	12	260	180	400	1200	199	0.166	460	65	0.140	471	67	0.142
Median values:		232[a]	162[a]	400	1291	191[a]	0.155[a]	484[a]	67[a]	0.141[a]	471[a]	64[a]	0.142[a]

[a] Significant difference between NWR and SHR group ($p \leqslant 0.05$, Mann-Whitney-U-test); sample lacking for technical reasons. SBP, systolic blood pressure; DBP, diastolic blood pressure; R_i, inner radius; WT, wall thickness.

photographs of the arterial cross-sections. Magnifications ranged from 25 times for aortic arches up to 110 times for internal carotid arteries. The perimeter values were divided by 2 Π and the difference between outer and inner radius (R_o–R_i) was taken as a measurement of the medial wall thickness (WT).

Results. Respectively, 2, 3 and 6 pairs of experiments were carried out with SHR and NWR at the age of 3, 7 and 12 months. The results are shown in the Table. In both types of arteries and at all ages, the medial wall was approximately 50% thicker in SHR than in NWR. In all instances, evaluation in groups with 5 or more samples showed that this difference was highly significant. Since WT/R_i is influenced by the perfusion pressure at the moment of fixation, measurements with different perfusion pressures were performed on internal carotid arteries of 3 month old SHR. The median values of WT/R_i were 0.162 ($n = 8$) at 120 mm Hg and 0.149 ($n = 6$) at 200 mm Hg and this difference was not statistically significant. Nevertheless, it must be kept in mind that the morphometric data of the Table were obtained at equal intraluminal fixation pressures. Consequently, the reported difference between NWR and SHR regarding WT and especially WT/R_i will be slightly less important in the living animal at the prevailing blood pressure level. Of course, the medial hypertrophy in SHR is not restricted to the level of the baroreceptor areas but occurs also at other sites, such as the common carotid arteries and the abdominal aorta[9]. According to morphometric data reported on the ascending aorta of 5 to 12 month old rats[11], the medial aortic wall thickness was 0.160 μm for NWR and 230 μm for SHR. In our experiments with 12-month-old rats the medial thickness of the aortic arch was 125 μm (median value) for NWR and 191 μm for SHR. We attribute the difference of the absolute values mainly to the fact that our samples were fixed under a perfusion pressure of 120 mm Hg, whereas YURUKOVA and KIPROV[11] used immersion fixation in the absence of any intraluminal pressure. Other published findings on the thoracic aorta reported a wall thickness of 80 to 100

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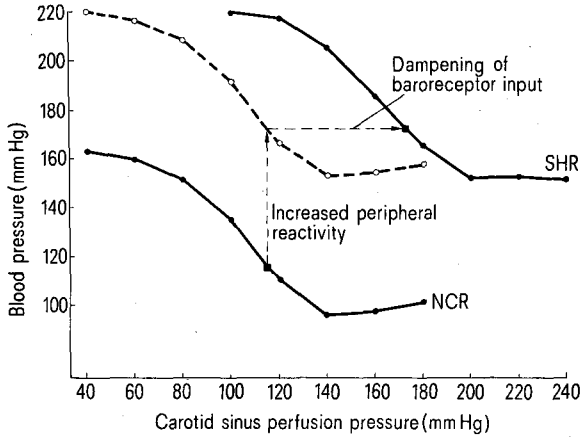
μm for 16- to 28-week-old normotensive rats and of 120 to 140 μm when the animals were made hypertensive[12]. In this latter study, fixation of the arteries was performed in vitro at varying pressures according to the blood pressure level. We assume that the extensive manipulation of the arteries in vitro and in the absence of functional sympathetic innervation has led to unphysiological dilatation.

In our experiments, the systolic and diastolic blood pressures of SHR were much higher than those of NWR, whereas the heart rate values were not different. This was a general feature observed in our SHR colony.

Discussion. Our findings suggest the following interpretation of blood pressure regulation in SHR. The baroreceptor nerve-endings of SHR are protected against excessive stimulation in spite of the hypertension, since the transmission of the pressure signal through the medial smooth muscles is dampened to a larger extent as in NWR. This explains why the efferent sympathetic tone is not depressed at the level of the vasomotor center and why the heart rate is not slowed down in the hypertensive rats. On the contrary, some available data indicate that efferent sympathetic discharge rate is rather increased in SHR, as compared to normotensive[13,14] or renal hypertensive rats[15]. In particular, our findings explain an important aspect of the modified baroreceptor functions reported in SHR[16,17]. NOSAKA and WANG[17] have tabulated their measurements, describing the relationship between systemic arterial pressure and carotid sinus perfusion pressure. We have represented their results in a diagram (Figure) and we have drawn the theoretical curve, which would be obtained if SHR had only a greater 'effector response' in comparison with NWR. These factors, whether they result from a greater amount[4,5] or from an increased reactivity[6,7] of the vascular smooth muscle cells in the resistance vessels, would only elicit an upward shift of the relationship-curve. It is clear that the results of NOSAKA and WANG with SHR also reveal an important shift of the curve from left to right, showing that a greater carotid sinus pressure is necessary before baroreceptor regulatory function comes into play. These authors speculate upon an increased rigidity of the sinus wall. Our morphometric studies on the medial hypertrophy in vascular stretch-receptor areas of SHR provide direct evidence for a structural basis of this horizontal shift of baroreceptor function due to dampening of the input signal.



●—●, experimental values published by NOSAKA and WANG[17]. ○—○, theoretical curve of NCR with increased peripheral reactivity, whithout modification of the baroreceptor setpoint.

Zusammenfassung. Morphometrische Daten bei Ratten mit genetischem Hochdruck zeigen, dass die Media der Gefässwand um etwa 50% dicker ist als bei normotonen Tieren. Diese Verdickungen sind im Aortenbogen und in der Nähe des Karotissinus gemessen worden, wo Pressorrezeptoren an der Aussenseite der Media liegen. Die hervorgerufene Verschiebung der Pressorrezeptor-Steuerung wird im Zusammenhang mit der veränderten Blutdruckregulierung bei genetisch hypertonen Ratten diskutiert.

D. WELLENS, A. VERHEYEN and M. BORGERS

Janssen Pharmaceutica, Departments of Cardiovascular Research and Cell Biology, Koninklijke Laan 17, B–2340 Beerse (Belgium), 11 September 1974.

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Bilateral Resection of Superficial Rat Kidney Cortex: Effect on Sodium Balance

Functional heterogeneity of the nephron population has been postulated since some years[1]. There was no experimental model, however, for direct studies on juxtamedullary nephrons in conscious animals. RASHID et al.[2] recently developed such an experimental model, producing unilateral necrosis of the outer rabbit kidney cortex by surface hyperthermia. We present here a similar model: bilateral surgical ablation of the whole superficial kidney cortex. Sodium balance was studied in rats subjected to this type of partial nephrectomy.

Material and methods. Male Wistar rats weighing 350–450 g were used.

Operative procedures. All rats were anesthetized with ether. Both kidneys were exposed from dorsal and the renal pedicles were clamped for a few minutes. The rats were then divided into 4 groups and each group was treated in a different way. Group I: no further treatment (sham operation). Group II: nephrectomy on the right side. Group III: nephrectomy on the right side + pole resection on the left kidney = approximately 4/6 nephrectomy[3]. Group IV: resection of the whole outer kidney cortex using a sharp scissor. Thus, about 50% of kidney tissue was removed. The wound surfaces were thoroughly dried with filter paper, and then covered with Histoacryl®, Braun, Melsungen. Seconds later the renal pedicle clamp was removed. Generally no bleeding occurred.

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