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Regional Cutaneous and Visceral Sympathetic Activity during Asphyxia in the Anesthetized Rabbit

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Summary. The changes of activity in cutaneous and visceral sympathetic efferents during asphyxia were simultaneously examined in anesthetized, paralyzed, artificially ventilated rabbits. In all cases an increase of activity in the splanchnic nerve and a simultaneous decrease of activity in the cutaneous (ear) sympathetic branch were observed during asphyxia. The decrease of cutaneous sympathetic activity was closely related with an increase of ear blood flow.

The decrease of cutaneous sympathetic activity represents a regional sympathetic response which is antagonistic to the hitherto known excitation of the sympathetic system by asphyxia. This antagonism is, however, not a manifestation of an invariable pattern of sympathetic activity. A stronger asphyctic stress led to an increase of activity in the cutaneous sympathetic branch as in other sections of the sympathetic system. An increase of activity in the cutaneous sympathetic branch was likewise observed during asphyxia, if its activity had been reduced before by a central heat stimulus.

Key-Words: Asphyxia — Cutaneous Blood Flow — Regional Sympathetic Activity.

Schlüsselwörter: Asphyxie — Hautdurchblutung — Regionale Sympathicusaktivität.

Early observations of Zuntz (1878) and of Dastre and Morat (1884) have shown that antagonistic changes of blood flow in cutaneous and visceral vascular beds are elicited in the rabbit by hypoxia and asphyxia. The same response has been found in the dog (Kullmann, 1969, unpublished observation). Within the scope of the research work done on the circulatory performance during hypoxia, only minor attention has been paid to this phenomenon (Korner, 1959). However, in a recent series of investigations this antagonism has been thoroughly studied, and interest has been focused on the cooperation of local metabolic, humoral and sympathetic vasoconstrictor influences in determining the regionally different circulatory responses in the skin and in other vascular areas (Chalmers and Korner, 1966; Chalmers *et al.*, 1967).

Recent experiments dealing with the thermoregulatory control of cutaneous blood flow (Kullmann *et al.*, 1970; Walther *et al.*, 1970) have

shown that the sympathetic system is capable of producing antagonistic changes of activity in its diverse sections. This finding raised the assumption that a similar performance of the sympathetic system might be involved in the circulatory adjustments during asphyxia. In order to clarify this question, the discharges in sympathetic efferents supplying cutaneous and visceral vascular beds were simultaneously recorded in rabbits which were submitted to asphyxia of variable duration.

Material and Methods

The experiments were performed in 29 albino rabbits of either sex, weighing 1.7—4.0 kg, from November 1969 to May 1970. Sodium pentobarbital was given intraperitoneally at a dose of 40 mg/kg. If necessary, small amounts of the anesthetic (10 mg/kg) were re-injected i.m. or i.v. The animals were placed on their right sides on a heating pad in order to maintain normal body temperature. Ambient air temperature was kept at constant values between 22 and 29° C. After cannulation of the trachea the animals were artificially ventilated with 300—400 ml/(kg · min) of air with the aid of a Starling pump. Muscular paralysis was produced by intravenous injection of succinyl choline and was maintained by continuous infusion of 0.1—0.2 mg/(kg · min) of the relaxant.

Measurements and Recordings. For bipolar recording of regional sympathetic activity, a sympathetic nerve twig accompanying the left retroauricular artery (ear sympathetic) and a branch of the splanchnic nerve were dissected free and were protected by a paraffin pool. The discharges were picked up with fine stainless steel hooks and were fed into differential amplifiers with impedance-matched outputs (Tönnies, No. 0329). The discharges were recorded a) directly and b) after summation during 5- or 10-sec intervalls (Tönnies, "amplitude adder" No. M 8) on a UV-direct writing oscillograph (CEC, Galvomat). Arterial blood pressure was measured with a Statham pressure transducer (Type P 23a) after cannulation of one femoral artery. One core temperature (vertebral canal temperature) and skin temperatures of both ears were measured by means of miniature-NTC bridge circuits. The temperatures and arterial pressure were recorded simultaneously with the sympathetic discharges on the oscillograph. Ambient air temperature and rectal temperature were measured with an electric thermometer (Ellab, Kopenhagen).— In 6 experiments, PaO_2, $PaCO_2$ and pH were followed up during the experiments. Small samples of arterial blood were taken in which these parameters were immediately measured directly with electrodes of Radiometer (Kopenhagen). Corrections for the differences between electrode and body temperatures were made for pH according to Rosenthal (1948) and for PaO_2 and $PaCO_2$ according to Nunn *et al.* (1965).

Calculations and Statistics. The courses of the activities of the investigated sympathetic efferents during the experiments were determined by comparing the integrator deflection amplitudes with the average amplitude during the pre-stimulation periods. Mean arterial pressure was calculated after its numerical evaluation from the recordings as $P_{diast.} + (P_{syst.} - P_{diast.}) \times 0.3$. Heart rate was determined from the pulse pressure recordings. The temperature recordings were numerically evaluated every 30 sec. For statistical presentation of the data, from a total of 52 experimental periods performed in 23 rabbits, one period for each animal was randomly selected (pre-stimulation phase: 1 min; asphyxia: 2 min; post-stimulation phase: 3 min). For statistical comparison of samples of paired observations the Wilcoxon matched-pairs signed-ranks test was applied.

Results

The courses of the activities of the ear sympathetic and of the splanchnic nerve branch during asphyxia as evaluated from the integrator deflections are shown in Fig. 1 A together with the ear skin temperatures, mean arterial pressure and heart rate. Immediately after the start of asphyxia, the activity of the ear sympathetic started to decrease and stayed at a lower level. The steep rise of ear skin temperature indicates vasodilatation which corresponds well with the reduced regional sympathetic activity and is in conformity with observations of other authors. In contrast to the ear sympathetic, the activity in the splanchnic branch continuously increased during asphyxia. Mean arterial pressure increased slightly during respiratory arrest, while heart rate markedly decreased to half of the pre-asphyctic value. After the end of asphyxia, heart rate and splanchnic activity returned to their previous levels within 1 min. Blood pressure showed a temporary overshoot. The activity of the ear sympathetic only gradually returned to the starting level, and consequently, ear skin temperatures did not tend to decline during the time of experimental observation.—The antagonistic changes of activity occurring in the ear and the visceral sympathetic branches are demonstrated in detail by sections from the original recording in Fig. 1 B. During asphyxia the discharge of the ear sympathetic seemed to vanish completely, while the predominantly respiratory bursts of activity in the splanchnic branch increased, changing at first to a mainly pulse-synchronous and then to a continuous elevated discharge. Immediately after the end of the asphyxia a prominent post-asphyctic inhibition occurred in the splanchnic discharge.

While in the experiment of Fig. 1 the response of the ear sympathetic to asphyxia can be termed as „monophasic", a "biphasic" response of this sympathetic efferent occurred in other experiments, one of which is demonstrated in Fig. 2. As shown in part A of the figure, splanchnic activity, arterial pressure and heart rate principally responded in the same manner to the asphyctic stimulus. Only the decrease of heart rate seemed to be more prominent. At first, the ear sympathetic also responded, as in the example of Fig. 1, with a decrease of activity during the 1st min of asphyxia, which was followed by a steep increase of the ear skin temperature. However, during the 2nd min of asphyxia the discharge increased again and, at the end of the asphyctic phase, it exceeded the pre-stimulation activity. Consequently, ear skin temperatures started to decline again during this time. After the end of asphyxia the activity of the ear sympathetic temporarily dropped to a low value and then gradually returned to its pre-stimulation level. Part B of the figure again demonstrates in detail the regional changes of sympathetic activity. In this case the pulse-synchronous rhythm was

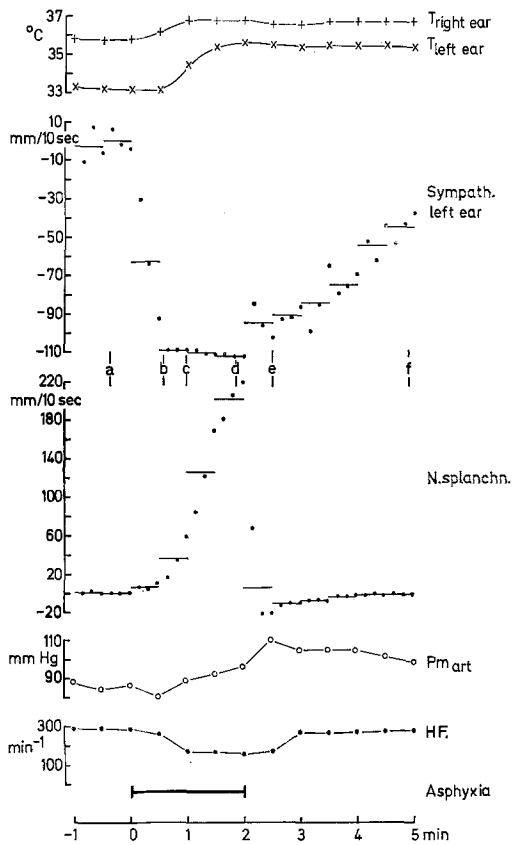

Fig. 1 A. Regional sympathetic activity during asphyxia in the anesthetized, paralyzed rabbit—"monophasic" response. Time courses of regional cutaneous (sympath. left ear) and visceral (N. splanchn.) sympathetic activity as influenced by asphyxia of 2 min duration (black bar). The integrated discharges per 10 sec and the mean discharge levels during 30 sec-intervals are plotted against time; reference value: mean discharge level during the last 30 sec before asphyxia. Further parameters: Ear skin temperatures (T right ear, T left ear), arterial mean pressure (Pm art.), heart rate (HF). Air temperature 29° C

more prominent in the splanchnic branch during the pre-stimulation phase and remained visible during the asphyctic excitation. Postasphyctic inhibition was distinct in the splanchnic nerve branch. The ear sympathetic also showed a rhythmic component in its discharge, which could however not be clearly attributed to either respiration or pulse pressure.

In 11 out of 23 experiments, a "monophasic" response of the ear sympathetic, i.e. a permanent decrease of activity, was observed during

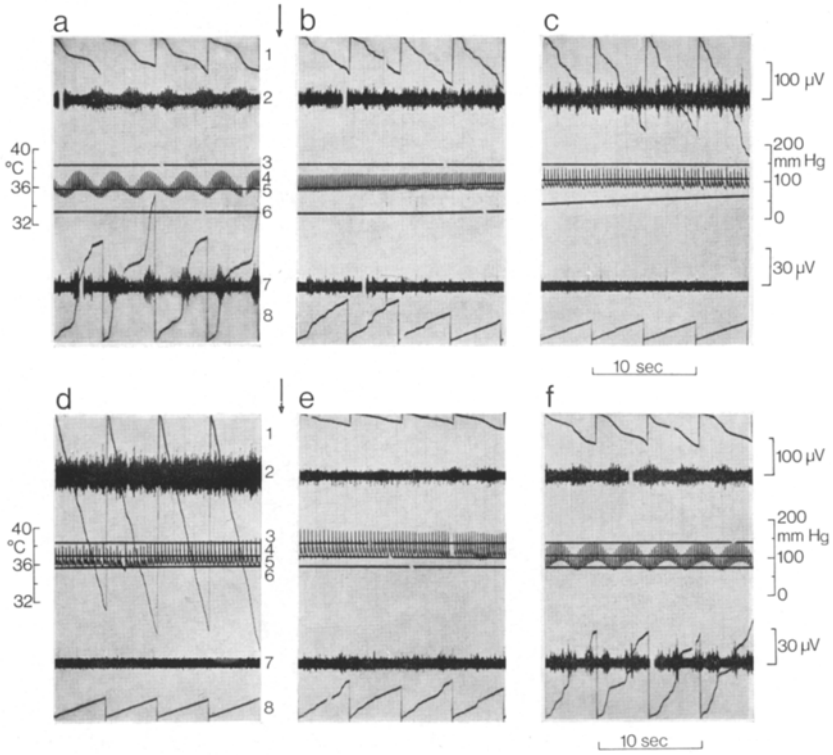

Fig. 1 B. Sections from the original recordings before, during and after asphyxia according to the letters in Fig. 1 A; arrows indicate start and end of asphyxia. Integrated discharges (downward deflection) (*1*) and discharges (*2*) of visceral sympathetic, core (vertebral canal) temperature (*3*), arterial pressure (*4*), skin temperatures of the right (*5*) and left (*6*) ears, discharges (*7*) and integrated discharges (upward deflection) (*8*) of cutaneous sympathetic

asphyxia, while in the remaining 12 cases a "biphasic" response, i.e. a re-increase of activity after an initial depression, occurred. These two groups of results are collected in Table 1 on the basis of randomly selected periods, one for each experiment. The data show that only the ear sympathetic and consequently the ear skin temperatures behaved differently in both groups. While in the monophasically responding sample the ear temperatures showed a slight but significant further rise after the end of asphyxia, this was not the case in the biphasically responding animals. Apparently the rise of ear skin temperatures was stopped by the increase of sympathetic activity during the 2nd min of asphyxia. All other values, i.e. blood pressure, heart rate and splanchnic sympathetic activity showed an identical behaviour. The greater average rise of splanchnic

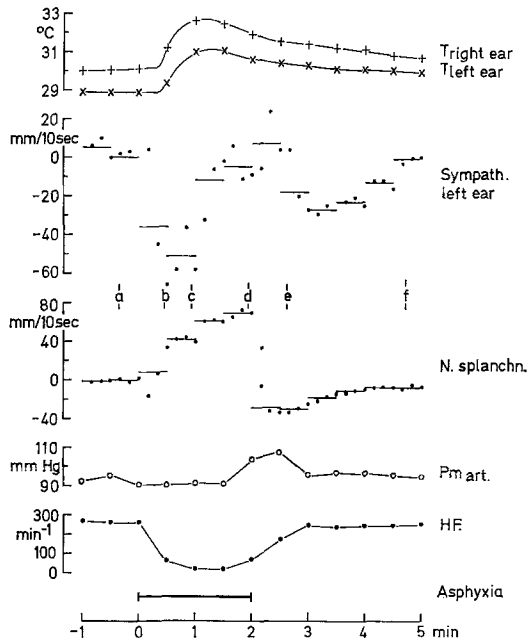

Fig. 2 A. Regional sympathetic activity during asphyxia in the anesthetized paralyzed rabbit—"biphasic" response. Time courses of regional cutaneous (sympath. left ear) and visceral (N. splanchn.) sympathetic activity as influenced by asphyxia of 2 min duration (black bar). Parameters as in Fig. 1 A. Air temperature 27° C

activity in the biphasic group may be meaningful but the method of evaluation is only semi-quantitative.

In 3 monophasically and 3 biphasically responding animals experimental periods with analysis of PaO_2, $PaCO_2$ and pH before, at the end and after the end of asphyxia were performed. The results are collected in Table 2. In these cases, regional sympathetic activity behaved according to the results shown in Table 1. The blood gas analyses revealed a difference between both groups which existed already during the pre-stimulation phase; the biphasic group showed a lower PaO_2, higher $PaCO_2$ and lower pH. This difference persisted during asphyxia and also during the post-stimulation phase. Apparently, a greater asphyctic stress developed in the biphasically responding group. The assumption that this greater stress was, in any way, connected with the re-increase of activity of the ear sympathetic was confirmed in two experiments, one of which is shown in Fig. 3. In these animals an asphyxia of 2 min duration had evoked a monophasic response. However, when an asphyxia of 3 min duration was applied, a re-increase of the discharge in the ear sympathetic

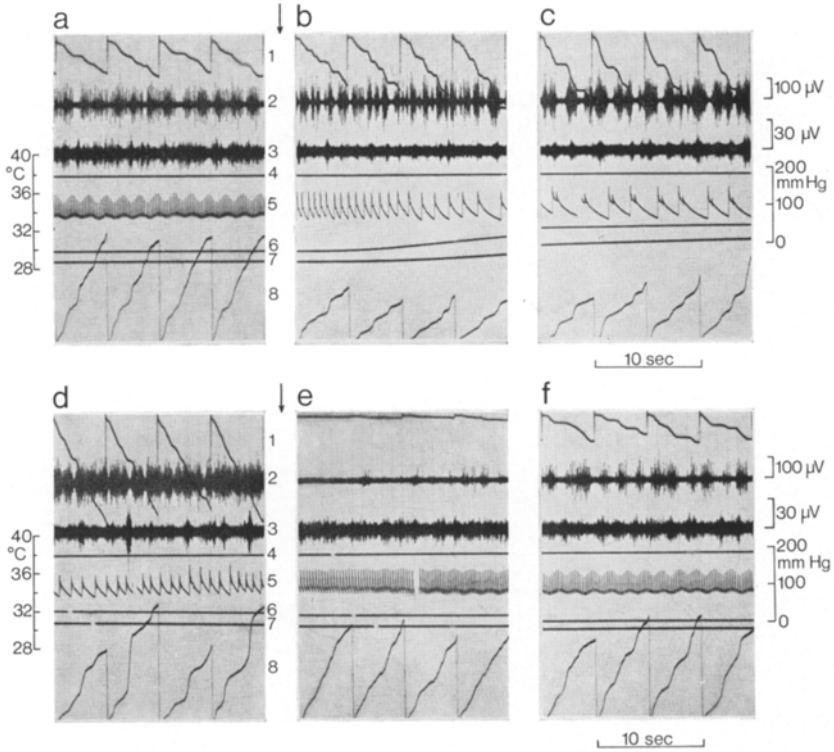

Fig. 2B. Sections from the original recordings before, during and after asphyxia according to the letters in Fig. 2A; arrows indicate start and end of asphyxia. Integrated discharges (downward deflection) (*1*) and discharges (*2*) of visceral sympathetic, discharges of cutaneous sympathetic (*3*), core (vertebral canal) temperature (*4*), arterial pressure (*5*), skin temperatures of the right (*6*) and left (*7*) ears, integrated discharges of cutaneous sympathetic (upward deflection) (*8*)

occurred during the 3rd min and the splanchnic sympathetic was further activated. As shown by the example, PaO_2 and pH further decreased during this time reaching values below 20 mm Hg, and 7,36 respectively.

In the early report of Zuntz (1878) and later by Chalmers and Korner (1966) it was mentioned that a reduction of ear blood flow rather than an increase occurred during asphyxia, if the animals were examined under heat stress, which presumably meant that the ear skin vessels were already dilated. The behaviour of the ear sympathetic in this situation was tested in 3 animals. A central heat stimulus was applied by spinal cord heating, which is known to induce a strong cutaneous vasodilatation (for details see Walther *et al.*, 1970). The experimental period shown in

Table 1. *Changes of regional sympathetic activity, ear temperatures, arterial mean pressure and heart rate during asphyxia of 2 min duration. A: "monophasic" response, 11 animals, rectal temperatures 36.5—38.1° C, air temperatures 22—29° C. B: "biphasic" response, 12 animals, rectal temperatures 36.5—38.1° C, air temperatures 23—29° C*

A

Time after start of asphyxia	min			During asphyxia							
			−0.5—0	0—0.5	0.5—1.0	1.0—1.5	1.5—2.0	2.0—2.5	2.5—3.0	3.5—4.0	4.5—5.0
Sympath. left ear	mm/10 sec	$\bar{x}$	0	− 6.5	− 16.4	− 18.2[a]	− 23.8[a]	− 16.2[a]	− 16.6[a]	− 10.9	− 6.9[b]
		$S_{\bar{x}}$		0.6	10.2	10.5	10.3	8.2	7.9	6.7	4.3
N. splanch.	mm/10 sec	$\bar{x}$	0	+ 6.0[a]	+ 26.2[a]	+ 53.7[a]	+ 66.4[a]	+ 3.8[b]	− 6.0[a b]	− 3.1[a b]	− 1.2[b]
		$S_{\bar{x}}$		1.5	7.1	14.4	19.6	4.4	3.0	2.0	1.2
Time after start of asphyxia	min		0	0.5	1.0	1.5	2.0	2.5	3.0	4.0	5.0
Temp. left ear	° C	$\bar{x}$	28.3	28.6	28.9	29.4[a]	29.9[a]	30.7[a b]	31.0[a b]	31.2[a b]	31.0[a b]
		$S_{\bar{x}}$	1.0	1.1	1.1	1.2	1.2	1.2	1.2	1.2	1.3
Temp. right ear	° C	$\bar{x}$	29.5	29.7	29.9	30.5[a]	31.4[a]	32.9[a b]	32.2[a b]	32.2[a b]	32.0[a b]
		$S_{\bar{x}}$	0.9	1.0	1.0	1.1	1.1	1.2	1.1	1.1	1.2
Pm art.	mm Hg	$\bar{x}$	87.0	93.6	95.8	99.4[a]	98.1	109.2[a b]	97.7[a]	89.2	82.7
		$S_{\bar{x}}$	2.9	3.7	4.8	5.1	3.9	5.0	4.1	2.7	2.4
Heart rate	min^{-1}	$\bar{x}$	242.1	136.0[a]	90.0[a]	78.5[a]	71.9[a]	139.7[a b]	179.8[a b]	208.8[a b]	220.5[a b]
		$S_{\bar{x}}$	8.7	25.1	19.4	14.3	9.2	16.0	17.8	13.5	13.2

B

Time after start of asphyxia	min		— 0.5—0	During asphyxia 0—0.5	0.5—1.0	1.0—1.5	1.5—2.0	2.0—2.5	2.5—3.0	3.5—4.0	4.5—5.0
Sympath. left ear	mm/10 sec	$\bar{x}$	0	— 6.2[a]	— 9.9[a]	— 10.7[a]	+ 4.9	— 2.7	— 5.4	— 9.8[b]	— 8.5[b]
		$S_{\bar{x}}$		3.5	4.6	5.3	6.7	6.6	6.0	5.5	5.0
N. splanchn.	mm/10 sec	$\bar{x}$	0	+ 8.8[a]	+ 40.9[a]	+ 82.2[a]	+ 93.6[a]	+ 0.5[b]	— 12.0[a b]	— 6.3[a b]	— 3.1[a b]
		$S_{\bar{x}}$		4.2	7.2	21.2	21.5	4.5	3.0	2.0	1.4
Time after start of asphyxia	min		0	0.5	1.0	1.5	2.0	2.5	3.0	4.0	5.0
Temp. left ear	° C	$\bar{x}$	28.1	28.1	28.5[a]	29.7[a]	29.9[a]	29.8[a]	29.8[a]	29.6[a]	29.4[a]
		$S_{\bar{x}}$	0.9	0.8	0.9	0.9	0.9	0.9	0.8	0.8	0.8
Temp. right ear	° C	$\bar{x}$	29.7	30.0	30.8[a]	31.8[a]	32.0[a]	31.9[a]	32.0[a]	32.1[a]	31.8[a]
		$S_{\bar{x}}$	0.7	0.8	0.8	0.9	0.9	0.9	0.8	0.8	0.8
Pm art.	mm Hg	$\bar{x}$	85.1	91.9	99.9[a]	98.8[a]	99.9[a]	115.4[a]	100.8[a]	84.3[b]	83.8[b]
		$S_{\bar{x}}$	2.8	3.7	3.5	4.2	6.0	4.8	6.0	3.0	2.8
Heart rate	min^{-1}	$\bar{x}$	248.8	122.4[a]	89.1[a]	83.3[a]	87.5[a]	143.0[a b]	206.4[a b]	213.3[a b]	227.3[a b]
		$S_{\bar{x}}$	10.7	21.9	15.9	11.0	8.7	10.7	21.0	13.1	13.2

Sympathetic activity expressed in millimeters of integrator deflection per 10 sec; the data of the table were calculated as average values for 30 sec-intervalls, reference value: discharge level during 30 sec before start of asphyxia.—$\bar{x}$: mean values, $S_{\bar{x}}$: standard deviations of mean values. [a] Data significantly different from the values before asphyxia, [b] data significantly different from the values at the end of asphyxia ($p < 0.05$, Wilcoxon matched-pairs signed-ranks test).

Table 2. *Blood gas data (PaO_2, $PaCO_2$, pH) from 3 animals with "monophasic" activity evaluated as in*

		N. splanchn. mm/10 sec				Sympath. ear mm/10 sec			
	Animal no.	1	2	3	$\bar{x}$	1	2	3	$\bar{x}$
"monophasic" asphyxia	before (min)								
	−0.5—0	0	0	0	*0*	0	0	0	*0*
	during (min)								
	1.5—2.0	+22.0	+51.0	+ 2.7	*+25.2*	−58.0	− 8.3	−11.3	*−25.9*
	after (min)								
	4.5—5.0	− 0.7	− 6.3	− 2.0	*− 3.0*	+ 3.0	+ 9.0	−10.3	*+ 0.6*
	Animal no.	4	5	6	$\bar{x}$	4	5	6	$\bar{x}$
"biphasic" asphyxia	before (min)								
	−0.5—0	0	0	0	*0*	0	0	0	*0*
	during (min)								
	1.5—2.0	+57.3	+18.0	+98.7	*+58.0*	+ 7.3	+40.7	+40.9	*+29.6*
	after (min)								
	4.5—5.0	+ 6.7	− 2.3	− 3.0	*−0.5*	+ 5.3	+12.3	−32.0	*− 4.8*

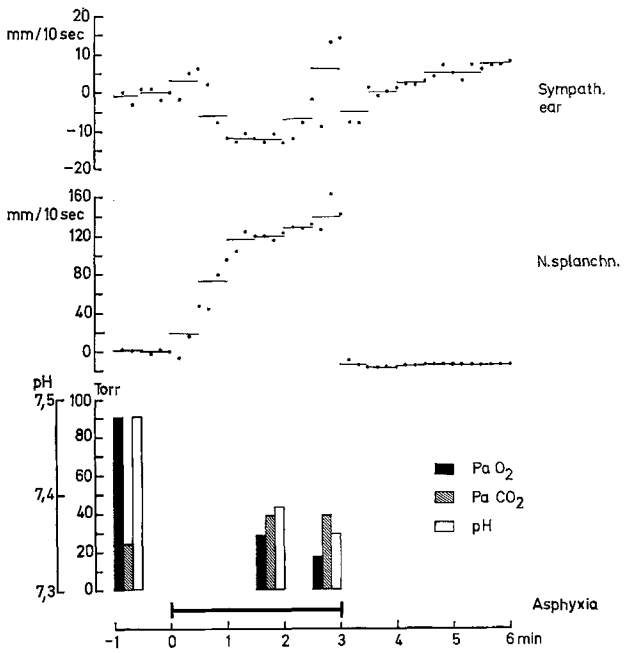

Fig. 3. Time courses of regional cutaneous (sympath. left ear) and visceral (N. splanchn.) sympathetic activity and changes of blood gases (PaO_2, $PaCO_2$, pH) as influenced by asphyxia of 3 min duration (black bar). This animal had shown a "monophasic" response to asphyxia of 2 min duration. Rectal temperature 37.6° C, air temperature 22° C

and 3 animals with "biphasic" responses to asphyxia of 2 min duration. Sympathetic Table 1. $\bar{x}$: Mean values

PaO_2 Torr				$PaCO_2$ Torr				pH			
1	2	3	$\bar{x}$	1	2	3	$\bar{x}$	1	2	3	$\bar{x}$
65.5	89.4	98.1	*84.3*	30.2	28.0	21.8	*26.7*	7.42	7.40	7.36	*7.39*
21.5	32.3	22.5	*25.4*	42.5	36.0	36.7	*38.4*	7.35	7.34	7.23	*7.31*
65.6	93.2	92.5	*83.8*	32.0	26.7	23.3	*27.3*	7.38	7.41	7.30	*7.36*
4	5	6	$\bar{x}$	4	5	6	$\bar{x}$	4	5	6	$\bar{x}$
54.9	75.0	81.3	*70.4*	31.7	32.9	25.7	*30.1*	7.22	7.37	7.31	*7.30*
16.0	13.3	18.0	*15.8*	40.5	55.3	37.5	*44.4*	7.13	7.23	7.22	*7.19*
49.0	64.9	79.4	*64.4*	33.6	41.1	27.5	*34.1*	7.16	7.32	7.28	*7.25*

Fig. 4 was performed during such a spinal heat stimulation, by which the activity of the ear sympathetic had been greatly reduced. Consequently, the ear vessels were dilatated as indicated by the high ear skin temperatures. In this situation asphyxia led to an immediate increase of activity in the ear sympathetic, which thus paralleled the response of the splanchnic sympathetic. A slight decrease of ear skin temperatures reflected the increase of activity in the ear sympathetic. As shown in Table 3, the remaining two experiments gave consistent results and thus offered a reasonable neuronal correlate for the findings of Zuntz (1878) and of Chalmers and Korner (1966).

Discussion

There are many reports on changes of sympathetic activity during asphyxia which indicate a general activation. Own occasional tests performed in cats and rabbits confirmed that the sympathetic activity increased in the splanchnic, lienal and renal nerves, in sympathetic branches running from the superior cervical ganglion to the upper cervical spinal nerves and in the cervical sympathetic trunk as a whole. It was ascertained in the present investigation by the recordings from the splanchnic branch that an increase of sympathetic activity always occurred during asphyxia, at least in certain regions. In view of many other additional findings which all indicate more or less uniform responses of the sympathetic system to any relevant stimulus (Weidinger *et al.*, 1961;

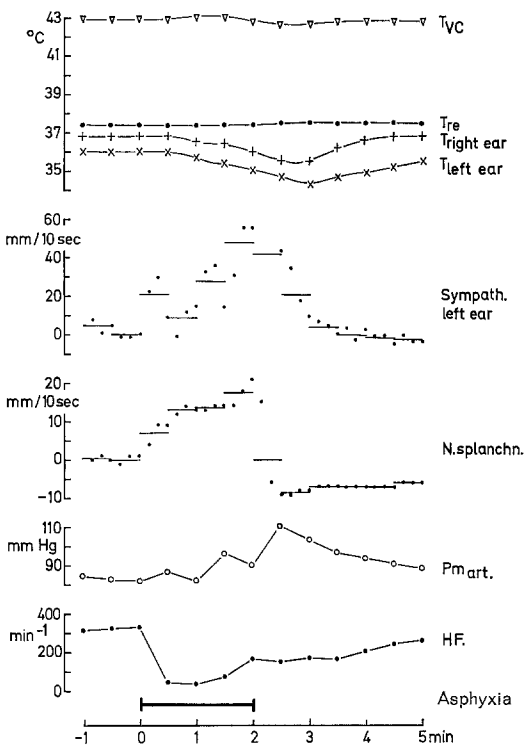

Fig. 4. Time courses of regional cutaneous (sympath. left ear) sympathetic activity as influenced by asphyxia of 2 min duration (black bar) during central heat stress (spinal cord heating). Air temperature 28° C. Vertebral canal (T_{VC}), rectal temperature (T_{re}), remaining parameters as in Fig. 1.—Parameters before spinal cord heating: T_{re}: 37.3° C, T_{VC}: 38.8° C, both ear temperatures: 28.7° C, integrated activity of the ear sympathetic as compared with the pre-asphyctic level: + 73.3 mm/10 sec

Peiper and Hauck, 1961), differences observed in the regional circulatory adjustments have been explained by various hypothetical mechanisms. Folkow *et al.* (1965) mentioned that regional differences of blood flow might partly be due to the fact that the tonically active vasomotor centers might exhibit differences in the excitability of its diverse neuron pools controlling functionally different vascular circuits. Further, regional differences in the frequency-response characteristics of the local vascular beds or a competitive balance between the local humoral vasodilatator effects and the nervous constrictor effects were discussed as possible contributing factors. With respect to the vasodilatation in the rabbit's ear during hypoxia, Chalmers *et al.* (1966, 1967) assumed at

Table 3. *Changes of sympathetic activity and of left ear temperature in 3 animals during asphyxia under central heat stress (spinal cord heating). Sympathetic activity evaluated as in Table 1; duration of asphyxia: case No. 1: 1.5 min, cases No. 2 and 3: 2 min*

	Animal no.	Before spinal warming	Spinal warming				After spinal warming	Spinal cord temperature	
			Before asphyxia	During asphyxia		After asphyxia		Before spinal warming	During spinal warming
				First half	Second half			° C	° C
Ear sympathetic activity mm/10 sec	1	+ 67.7	0	+ 60.0	+ 34.0	− 0.3	+ 104.3	38.7	41.8
	2	+ 75.7	0	+ 9.0	+ 48.0	− 1.7	+ 54.3	38.8	43.0
	3	+ 24.7	0	+ 4.7	+ 51.3	− 2.7	+ 17.0	38.3	42.4

	Animal no.	Start of spinal warming	Spinal warming				After spinal warming	Rectal temperature	Air temperature
			Start of asphyxia	During asphyxia		End of spinal warming			
				Middle of asphyxia	End of asphyxia				
								° C	° C
Ear temperature ° C	1	30.5	37.3	36.6	36.3	37.1	33.2	38.8	23.5
	2	28.7	36.0	35.7	35.0	35.5	31.7	37.3	28.0
	3	30.0	35.7	35.5	34.5	35.5	33.6	38.1	28.0

first that the increase of sympathetic activity during asphyxia in this special vascular region was much less marked than in other regions. They later raised the suggestion that the vasodilatator response might be the result of a central inhibition of regional vasoconstrictor tone.

Besides some indirect observations indicating *quantitatively* different responses in diverse vascular regions, there are few reports in the literature which give a more direct evidence for such a mechanism participating in the asphyctic vasomotor response. Dowing and Siegel (1963), when perfusing the isolated carotid sinus with hypoxic blood, observed a behaviour of the discharges recorded from the left inferior cardiac nerve which was different from that known from other sympathetic sections. Green and Heffron (1966) reported quantitative differences between the simultaneously recorded discharges of the inferior cardiac nerve and of a ramus from the stellate ganglion to the brachial plexus. They stated this with special respect to the time differences between the discharge bursts in these sympathetic branches. In the present investigation similar or even greater shifts were occasionally observed between the investigated nerves but this question was not systematically examined.

No reports seem to exist on *qualitatively* different responses of the sympathetic system in diverse regions, especially with respect to the regionally different vasomotor adjustments during asphyxia. Direct evidence for this type of sympathetic response is offered in the present investigation. The explanation of the antagonistic changes of blood flow in the skin and in other regions during asphyxia by a likewise antagonistic innervation is confirmed by the close correlation between the activity of the ear sympathetic and the ear skin blood flow as indicated by the ear temperature measurements.

An antagonism between ear sympathetic and the splanchnic and probably other sympathetic nerves has also been observed during thermoregulatory vasomotor responses (Walther *et al.*, 1970). However, it has been pointed out already in this preceding communication that this antagonism represents, by no means, an invariable pattern of the sympathetic outflow. This could be confirmed also in the present investigation. In about half of the examined animals only temporary depression of the ear sympathetic was observed during asphyxia which was replaced by a re-increase, i.e. by a reaction paralleling the course of activity in other sympathetic efferents. From the blood gas analyses it may be inferred that during a stronger asphyctic stress another mechanism comes into play, exerting a generalized drive on the sympathetic system. Further investigation is necessary to evaluate the factors responsible for this generalized uniform reaction.—Another situation in which parallel changes of activity occurred in the ear sympathetic and the splanchnic

nerve, was found during heat stress. In conformity with the earlier observations made on blood flow, an immediate increase of activity in the ear sympathetic accompanied by a decrease of ear skin temperature occurred, if its activity had been reduced before by activation of heat loss mechanism. This finding may be explained in different ways. Two types of efferent sympathetic fibers might exist in the ear sympathetic, a greater portion which is depressed by heat stress as well as by asphyxia, and a second, smaller portion which is activated by asphyxia. The discharge of the latter could, then, be observed during asphyxia only if the activity of the former was reduced before, as it is the case during heat stress. This hypothesis would imply that a functional difference exists between the diverse peripheral sympathetic neurones of the cutaneous sympathetic. Another explanation would presuppose that several pathways of information converge on the cell bodies of the sympathetic preganglionic neurones supplying the ear; inhibiting ones which are driven e.g. by heat stress and by asphyxia, and exciting ones which are driven e.g. by asphyxia. In this case, functional differentiation would be brought forth by a specific pattern of activity impinging upon the preganglionic vasomotor neurones.

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