Résumé. Des suspensions de mastocytes péritoneaux isolées de rat furent incubées avec 10 substances différentes, connues ou presumées être des neurotransmitteurs. Seulement l'adenosine triphosphate (ATP) aux concentrations supérieures à $2.64 \times 10^{-6} M$ causèrent la dégranulation des mastocytes. L'ATP cause également la dégranulation des mastocytes dans le mésentère. L'action de l'ATP peut être responsible de la dégranulation des mastocytes cutanées observée après la stimulation antidromique des nerfs sensorieux, alors que de l'ATP est libéré dans la peau [23].

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[23] The technical assistance of Miss CHRISTINE GOLDING and Miss JO MALLON is gratefully acknowledged. The summary was kindly translated by Dr. J. PAPAIOANNOU. Most of the work was carried out during the tenure by the author of a Stanley Elmore Senior Research Fellowship of Sidney Sussex College, Cambridge.

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Electrocardiographic Changes in Anaphylactic Shock of the Rabbit

Anaphylactic shock due to a challenge with heterologous proteins induces a predominantly histamine independent overall arterial vasoconstriction in intact animals, as well as in isolated organs which also includes the coronary arteries[1-9,11]. The anoxic effects of coronary constriction can be electrocardiographically demonstrated[5,7,8,11]. In man, most concern is directed towards the often dramatic decrease of arterial blood pressure, and clinical treatment mainly consists in an attempt to increase vascular resistance and to enhance cardiac activity by β-receptor stimulating substances such as epinephrine or isoprenaline, or even by the use of vascoconstricting agents such as norepinephrine or angiotensin. It therefore seemed of interest to reproduce the decrease of arterial blood pressure under experimental anaphylactic shock conditions and to establish the nature of its origin.

Materials and methods. Six white rabbits were sensitized twice in an interval of 10 days by 1.0 ml/kg horse plasma subcutaneously. The challenge was performed 3 weeks after the second injection by 2.0 ml/kg of plasma of the same horse given intravenously. The animals were anaesthetized by 22.5 mg/kg of Na-pentobarbital intravenously.

The arterial blood pressure was measured by direct puncture of the carotid artery with an Autocath-Teflon 3.6 F catheter which was connected to an electromanometric blood pressure unit with continuous rinsing of the catheter. Simultaneously with the blood pressure the electrocardiogram was recorded on a Cardiopan III T of F. Liechti AG, Ostermundigen (Switzerland).

Results. The results are summarized in the Table. Blood pressure values and electrocardiographic changes are reported together with the time elapsed after the challenge. The electrocardiographic changes mainly consisted in negativization of ST-T and/or ventricular arrhythmia. They were interpreted as signs of myocardial anoxia. In 3 cases, ventricular arrhythmia occurred at first while in 3 cases deformation of ST-T was first observed. In no case did the blood pressure decline more than 5 mm Hg systolically and/or diastolically at the onset of electrocardiographic changes. On the contrary, in 2 animals (No. 1 and 2) the blood pressure values had increased by 20/10 mm Hg and 30/25 mm Hg, respectively, at the onset of cardiac alterations. It may also be seen that a notable blood pressure decrease of at least 10 mm Hg occurred only after electrocardiographic changes had persisted for at least 20 sec.

Discussion. Our results obtained in intact rabbits in an early phase of anaphylactic shock indicate that heart reactions as evidenced by electrocardiographic changes are initially not due to a decrease of arterial blood pressure and coronary perfusion pressure, respectively. These ex-

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Carotid blood pressure in anaphylactic shock before and after challenge with reference to the time of onset of electrocardiographic changes

Animal No.	Blood pressure before challenge (in narcosis)	Blood pressure and time at the onset of				Blood pressure and time			
		Ventricular arrhythmia		Negativization of ST-T		at the earliest decline		at the lowest level	
	mm Hg	mm Hg	sec	mm Hg	sec	mm Hg	sec	mm Hg	sec
1	100/80	120/90	62	120/90	70	90/70	120	0/0	140
2	110/90	110/80	110	140/115	70	95/75	130	70/50	140
3	135/105	135/110	14	130/115	40	110/90	90	60/40	300
4	165/130	165/135	67	150/120	82	125/105	90	60/40	180
5	120/80	120/85	1080	115/80	65	—	—	(115/80)	(65)
6	130/80	120/70	45	130/75	35	100/50	55	50/25	110

periments do not elucidate the cause of the anaphylactic electrocardiographic changes. However, many observations suggest that coronary vasoconstriction is the main cause of myocardial reaction in anaphylactic shock although anoxemia due to constriction of pulmonary arterial vessels and bronchospasms may also play an important role in this connection[1-8, 11]. A direct anaphylactic reaction of the myocardial tissue has also to be taken into consideration since arrhythmias have been observed in anaphylaxis in isolated papillary muscle[10].

Although blood pressure eventually decreases because of diminished venous blood return resulting from peripheral vasoconstriction which was demonstrated by many investigators[1, 9], it seems most likely that diminished cardiac work also contributes to the blood pressure decrease occurring in experimental and clinical anaphylactic shock. This point of view is supported by observations made in isolated guinea-pig hearts in anaphylaxis giving evidence of strong coronary vasoconstriction followed by a significantly decreased cardiac output[5, 6].

Under the aspect of the electrocardiographic changes observed by us and others[5, 7, 8, 11], an undifferentiated therapy of anaphylactic shock with epinephrine and similar substances should be reconsidered despite the fact that it may decrease pulmonary resistance[3] and overcome peripheral vasoconstriction[1, 9]. Epinephrine as well as isoprenaline may eventually increase arterial blood pressure and peripheral tissue perfusion, but they may also induce ventricular extrasystolias by themselves and/or increase myocardial oxygen deficiency as detectable in the electrocardiogram[12]. It is also questionable whether the use of pressoric substances such as norepinephrine or angiotensin can be of value in a situation in which maximal vasoconstriction already prevails[9, 12].

Zusammenfassung. Die simultane Registrierung des EKG und des Karotisblutdrucks am Kaninchen in der Frühphase des anaphylaktischen Schocks lässt auf eine primäre Herzreaktion schliessen, welche nicht auf einer Verminderung des Blutdrucks, bzw. des koronaren Perfusionsdrucks, sondern auf einer vorwiegend infolge Koronarkonstriktion eintretenden Abnahme der Herzleistung zu beruhen scheint.

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Amine and Amino Acid Microanalysis of Two Identified Snail neurons with Known Characteristics

Because of the known heterogeneity of neurons it would seem that precise biochemical information on the functioning of nervous systems is best obtained from experiments on individual neurons. Certainly we will need to learn more of the cellular biochemistry occurring in neurons during adaptive behavioural activity before fully comprehending the underlying processes controlling these events.

In the present work we have used a recently devised micromethod[1] to analyse the amine and amino acid composition of two specified giant neurons in the snail (*Helix pomatia*) brain. The method involves the reaction of amines and amino acids with ^{14}C-labelled dansyl chloride to form compounds which can be separated by thin layer chromatography and detected under UV-light or by autoradiography. One of the neurons studied, the giant metacerebral, or giant serotonin cell (GSC)[2] receives both excitatory (cholinergic)[3] and inhibitory innervation. The inhibitory effect is mimicked by glutamic acid[4]. There are two identical GSCs in each snail brain; one is located in each cerebral ganglion. Evidence that these cells contain serotonin comes from fluorescence histochemical and bioassay data[2]. The other cell analysed, the so called posterior buccal cell receives an excitatory (serotonergic) input from each GSC[5, 6]. This cell lacks the capacity, shown by the GSC, to form serotonin from 5-hydroxytryptophan[7] and histochemical studies suggest that the cell lacks serotonin. There is one posterior buccal cell in each buccal ganglion.

The main objectives of our study were as follows: 1. To determine whether it is possible to obtain consitent data when analysing a small number of GSCs or posterior buccal cells dissected from different animals. 2. To obtain biochemical data on the content of serotonin in both types of neurons. 3. To establish whether there are any differences in the amino acid composition of the two types of cells. 4. To obtain further data on the effect of optic tentacle ablation on the level of serotonin in the GSC, since previous results suggested that this procedure causes a reduction in serotonin[8].

The identified neurons were dissected by hand from ganglia removed from live snails. The cells were transferred to a Drummond Microcap containing 3 µl of cold 0.05 N $NaHCO_3$ (adjusted to pH 9.5 with 1 N NaOH). When sufficient cells were collected they were homogenised, 3 µl of acetone was added and the mixture cooled to −5°C for 1 h. After centrifuging at 4,000 g for 30 min the supernatant was mixed with 4 cl of a 2 mg/ml solution ^{14}C labelled dansyl chloride ((1 dimethyl ^{14}C) aminonaphthalene-5-sulphonyl chloride)[9] in acetone. Each sample was incubated at 37°C for 1 h and spotted in the corner of 3 × 3 cm polyamide layer chromatography sheet (Carl, Schneider and Schüll, F 170 Mikropolyamide). Chromatograms were developed in water/formic acid (100:3) in one dimension and in benzene/acetic acid (9:1) in the second dimension, and viewed in UV-light. Autoradiograms were prepared using Afga-Gevaert Denture Ultra Rapid L film.

Best results were obtained with extracts from 8 cells although satisfactory chromatograms were also obtained when 4 cells were used. Examination of 16 pairs of chromatograms, each prepared from 8 cells showed that it is indeed possible to obtain consistent results for each cell type. Figure 1 shows the autoradiograms of 3 pairs of chromatograms, those of the posterior buccal cells above,

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