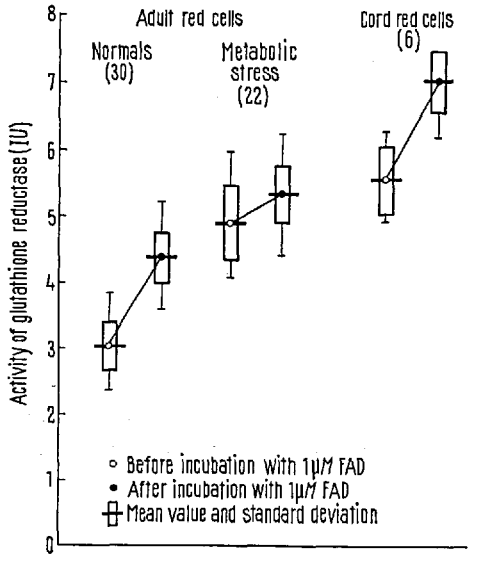
glutathione (GSSG), and 0.29 mM NADPH using a Gilford Model 2000 Automatic Recording Photometer. Total GR activity in hemolysates was measured after 30 min of incubation at 37 °C with 1 μM FAD according to the method of BEUTLER[3]. The amount of the inactive form of GR represents the increase in GR activity after incubation with FAD. The saturation percentage was calculated by dividing the GR activity of the active form by the total activity obtained after incubation with FAD.

Results are shown in the Figure. Mean GR activity in red cells from 30 normal adults was 3.04 ± 0.36 IU (the active form). After the addition of FAD to the hemolysates, this increased to 4.38 ± 0.37 IU, an increase of 1.34 ± 0.37 IU (the inactive form). Thus, in normal adults 69.4% of GR is in the active form, and 30.6% of GR is in the inactive form. In the red cells of 22 adult patients with severe metabolic disorders, as described above, total GR activity was 5.35 ± 0.43 IU and 91.8% of GR was in the active form.

In contrast, mean GR activity in 6 samples of normal cord red cells was 5.57 ± 0.51 IU (active form). After addition of FAD to the hemolysates, this increased to 7.03 ± 0.45 IU, an increase of 1.46 ± 0.65 IU (the inactive form). Thus, in normal cord red cells, 79.2% of GR is in the active form, and 20.8% of GR is in the inactive form.

The percentage of GR in the active form in cord red cells is only moderately greater than that in the red cells of normal adults, and is not as elevated as in severe metabolic disorders. However, it is noteworthy that the total amount of GR in normal cord red cells is considerably higher than that in the red cells of normal adults or even of patients with severe metabolic disorders. This increased total GR activity cannot be ascribed to a younger red cell population in cord blood since GR activity is not related to the mean age of the red cell population[7]. Thus, the increase of total GR activity in cord red cells may be related to at least 2 possible causes; 1. increased synthesis of the enzyme, or 2. the presence of an isozyme of GR in cord red cells different from that of adult red cells. The latter is a consideration in light of the work of SCHROETER and TILLMANN[8] with hexokinase (HK). They have demonstrated that the higher HK activity of red cells of new-borns is due to the presence of HK isozyme type I, which is different from that of adult red cells (type III). Although, to our knowledge, studies on isozymes of GR in human cord red cells have not been reported, it is possible that normal cord red cells have an isozyme (a fetal type) of GR that is analogous to that for HK. This fetal type GR isozyme may then account for the increased activity, as in the case with HK[9].



Activation of glutathione reductase by FAD in normal adult red cells, adult red cells from patients with metabolic stress, and cord red cells.

Zusammenfassung. Nachweis, dass foetales Blut mehr Erythrocyten-Glutathionreduktase enthält als Erwachsenenblut und auch der prozentuale Anteil der aktiven Enzymform etwas grösser ist. Diese Befunde sind ein erster Hinweis auf das Vorkommen eines besonderen foetalen Isoenzyms.

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25 January 1971.

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Neurogenic Responses of Resistance and Capacitance Vessels

Besides data providing the similar patterns of reflex responses in resistance and capacitance parts of skeletal muscle vascular bed[1,2] there are studies[3,4] revealing these responses to be opposite in direction under some pressor reflexes. Directional differences for resistance and capacitance responses were also shown to be possible in the abdominal aorta vascular bed under depressor reflex initiated from the right heart and the pressor synocarotid reflex[4]. Resistance vessels both in skeletal muscle and in splanchnic vascular beds are known to be constricted under pressor reflexes[5,6]. As to the responses of capacitance vessels recording simultaneously

in a number of vascular zones under these reflexes, there is a lack of data in the literature. This study was intended to reveal the pattern of capacitance vessel reflex responses in skin-muscle and splanchnic vascular zones and to compare these responses with those of resistance vessels in the same zones.

Method. Experiments were performed on 17 cats anaesthetized with urethane (1 g/kg). The constant blood flow perfusion of splanchnic and skin-muscle vascular zones was installed to investigate responses of resistance vessels responses in the both zones simultaneously. The study of capacitance vessels responses was carried out by means of parallel registration of venous outflow from these zones, the method described elsewhere[7] being used. The resistance vessels responses were identified by the changes of perfusion pressure, and the responses of capacitance vessels – by the value of maximal output or pooling of blood[7].

Results. 2 main types of vasomotor responses in skin-muscle and splanchnic vascular zones were observed under pressor synocarotid reflex (26 experiments). The first one (11 experiments) was constriction of both resistance and capacitance vessels (Figure 1). The response latency was longer for capacitance vessels (7.5 ± 2.2 sec) than for resistance ones (0.6 ± 0.4 sec) in skin-muscle vascular zone, the duration of the capacitance vessel response (134.9 sec) being longer than one of the resistance vessel response (83.1 sec). Similarly, the response of capacitance vessels in splanchnic area arised later than and was of greater duration than resistance vessel response.

As to the other 11 experiments, a duration of capacitance vessels in skin-muscle vascular zone and their constriction in splanchnic region (Figure 2) were observed under the same reflex, resistance vessels of the both zones being constricted. Response latency and duration for capacitance vessels were longer than for resistance ones both in skin-muscle and splanchnic areas.

The remaining 4 experiments of this series showed various capacitance vessels responses on the background of resistance vessel constrictor responses under the pressor synocarotid reflex. The capacitance responses were as follows: constriction in the skin-muscle vascular zone and dilatation in the splanchnic one in 2 experiments; constriction in the skin-muscle zone and unchanged capacity in the splanchnic one in 1 experiment; dilatation of capacitance vessels in the both vascular zones in 1 experiment. Systemic arterial pressure increased by $57.7 \pm 2.9\%$ in this series experiments.

Effect of electric stimulation (15 v, 30 imp/sec, 5 msec) of brachial plexus afferents on resistance and capacitance vessels of investigated vascular zones being studied (19 experiments). the above-mentioned 2 types of vasomotor response were observed again. They were as follows: constriction of resistance and capacitance vessels of vascular zones under study (5 experiments); constriction of resistance vessels in the both zones and constriction of capacitance vessels in splanchnic zone, capacitance vessels of skin-muscle zone, being dilated (8 experiments). The resistance vessel response being constrictor in the both vascular zones (6 experiments); constriction of splanchnic capacitance vessels and unchanged vascular capacity of the skin-muscle zone were observed in 2 experiments; the others (4 experiments) showed the capacitance vessel dilation in the both zones. Interrelation of response latency and duration for resistance vessels with those for capacitance ones under electric stimulation of brachial plexus afferents was analogus to that observed under pressor synocarotid reflex.

The resistance and capacitance vessel constriction in skin-muscle and splanchnic vascular zones occurred in

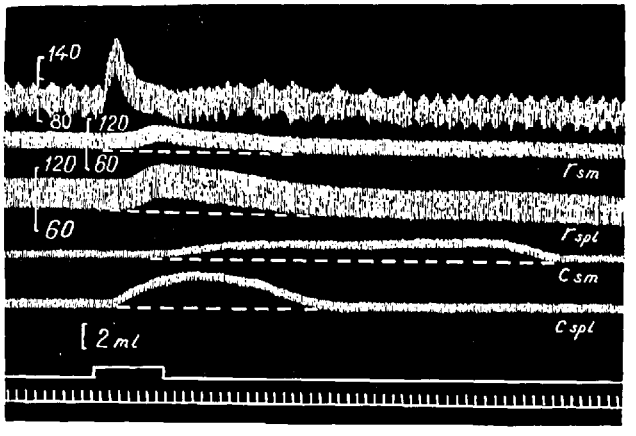

Fig. 1. The constriction of the skin-muscle and splanchnic resistance and capacitance vessels under pressor synocarotid reflex. Designations. Records from up to down: systemic arterial pressure, resistogramms of the skin-muscle (r sm) and splanchnic (r spl) region vessels, changes of venous outflow from the skin-muscle (C sm) and splanchnic (C spl) regions, the stimulation mark, time mark (5 sec). The scales on arterial and perfusion pressure curves – in mm Hg, the scale of venous outflow changes in ml.

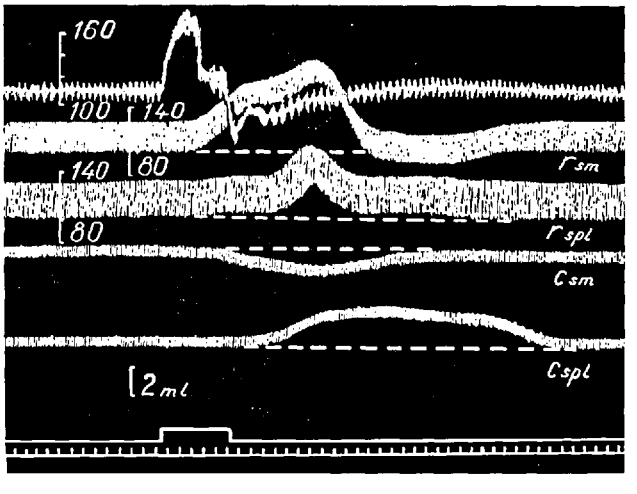

Fig. 2. The constriction of the skin-muscle and splanchnic resistance vessels under increase of the vessel capacity in the skin-muscle region and under its decrease in the splanchnic region under pressor synocarotid reflex. Designations same as in Figure 1.

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all experiments[7] as a result of electric stimulation of the thorax sympathetic chain (6 v, 20 imp/sec, 5 msec). The pattern of the neurogenic responses of resistance and capacitance vessels, recorded simultaneously in skin-muscle and splanchnic areas is illustrated in Figure 3.

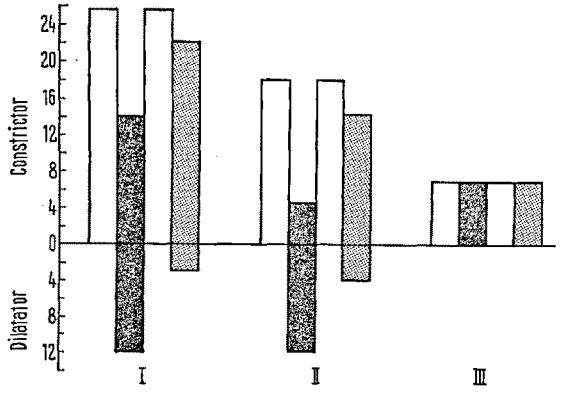

Fig. 3. Directivity of resistance and capacitance vessels responses in the skin-muscle and splanchnic regions under pressor synocarotid reflex (I), electric stimulation of the brachial nerve afferents (II), electric stimulation of the thorax sympathetic chain (III). Designations: on the ordinate, the quantity of experiments; white rectangles, responses of resistance vessels; black rectangles, responses of skin-muscle capacitance vessels; shade rectangles, responses of splanchnic capacitance vessels; above abscissa, constrictor response; below – dilatator response.

Conclusion. Electric stimulation of the thorax sympathetic chain resulted in a constriction of resistance and capacitance vessels in skin-muscle and splanchnic vascular zones. Simultaneous registration of resistance and capacitance vessel reflex responses in skin-muscle and splanchnic areas revealed resistance vessel responses to be always constrictor, while the capacitance ones might be either identical or different their direction being considered. Furthermore, the responses of capacitance vessels in the same vascular zone may differ directionally from the responses of resistance vessels.

Выводы. Электрическая стимуляция грудной симпатической цепочки вызывает констрикцию резистивных и емкостных сосудов в кожно-мышечной и спланхнической областях. Изучение рефлекторных реакций резистивных и емкостных сосудов одновременно в кожномышечной и спланхнической областях показало, что реакции резистивных сосудов всегда имеют однонаправленный констрикторный характер, тогда как реакции емкостных сосудов указанных областей могут быть как одинаковыми, так и различными по знаку. Кроме того, реакции емкостных сосудов одной и той же области могут отличаться от реакций резистивных по направленносси.

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Water and Sodium Chloride intake Following Microinjection of Carbachol into the Septal Area of the Rat Brain

Several studies have been published giving evidence that the activity of the septal area is mediated via cholinergic pathways, supporting the hypothesis that the septal area contains a cholinergic neuronal system (GROSSMANN[1], HAMILTON et al.[2], KELSEY[3]).

In the present study, this hypothesis was tested using the free water and sodium chloride intake as criteria of altered septal function. Adult male Wistar rats, of 200 to 300 g body weight, were kept in individual cages with a food cup filled with dry mixed diet and 2 graduated drinking bottles filled with 1.5% NaCl and unfiltered tap water respectively; daily readings were made of the intakes. After a control period of 2 weeks a stainless-steel cannula (O.D. 0.71 mm) was stereotaxically implanted into the septal area and after a further week injections were made through a dental stainless steel cannula (O.D. 0.31 mm) into the conscious and unrestrained rats. Carbachol (Carbamylcholine Chloride) and atropine sulfate were delivered by a 10-µl microsyringe in a standard volume of 2 µl of isotonic saline (0.15 *M*) by way of a polyethylene plastic tube connected to the inner cannula which was placed inside and advanced to the tip of the implanted cannula.

At the end of the experiment the rats were sacrificed and their brains were sectioned and stained. The end of the cannula was considered to represent the stimulation site. No particular localization was found and it was not intended to make any mapping, but a tendency was observed for more positive results with the cannula at an anterior placement.

Effect of carbachol. Immediately after the injection the rat was returned to its cage and the intake of fluids were measured after 10, 30, 60 and 360 min. Drinking started after a latency of between 3 to 5 min. Doses of 0.06, 0.125, 0.50, 1.0 and 2.0 µg were effective but routinely the dose of 1.0 µg was adopted. Figure 1 depicts the results obtained: it can be observed that carbachol

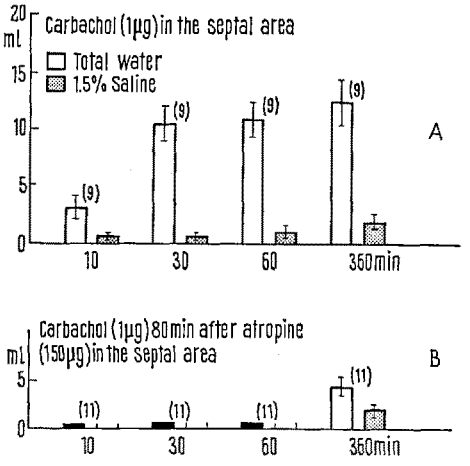

Fig. 1. A) Cumulative intake of total water and NaCl 1.5% following the injection of 1 µg of carbachol in the septal area. B) Inhibition produced by 150 µg of atropine upon the drinking effect of carbachol. The bars indicate the standard deviation of the mean; the number of rats are indicated between parentheses. Stereotaxic coordinates: F, 8.0; L, 0.3; H, +1.0. Total water: tap water plus water of saline solution.