ducts were exposed in the neck and cannulated. In two of the dogs the parotid duct was also cannulated. The following drugs were given. Methacholine (1–10 μg/kg), isoprenaline (0.1–2 μg/kg), salbutamol (1–2 mg/kg). Methacholine was injected between the injections of the other drugs as a control of the functional state of the gland. In 7 of the rats and 4 of the dogs, the right chorda-lingual nerve was cut in order to sensitize the gland cells to secretory agents.

Results and discussion. The results are summarized in the Table. It can be seen that in rats salivary secretion was obtained from all submaxillary glands when isoprenaline 0.5 μg/kg was given. In the normally innervated glands, no salivary secretion was obtained when salbutamol 20 μg/kg was injected. In 1 out of 7 of the decentralized glands, salivary secretion was obtained when salbutamol 20 μg/kg was given. With salbutamol 10 μg/kg, no secretion was obtained in this case.

In the normally innervated submaxillary gland of the dog, salivary secretion was obtained in 3 glands out of 6, when isoprenaline 0.5 μg/kg was given. Salivary secretion was obtained from the other 3 glands when isoprenaline 1–2 μg/kg was given. Salbutamol 20, 50 and 100 μg/kg did not cause secretion in these glands. When salbutamol was found to cause salivation in submaxillary glands of rats and dogs, the effect was abolished by propanolol showing that it was mediated by β-receptors. It is not surprising that salbutamol was unable to evoque any flow of saliva from parotid glands of dogs, which lack β-receptors[2]. The secretory effect on the submaxillary gland was very small; it was at most 1/100 of that of isoprenaline and usually much smaller. It is obvious that, in its responsiveness to salbutamol, the salivary glands studied resemble the heart rather the bronchioles or the blood vessels. Using the terminology of LANDS, LUDUENA and BUZZO[3] their β-receptors should in other words be described as belonging to the β_1 subgroup.

Zusammenfassung. Nachweis an Ratten und Hunden, dass Salbutamol, ähnlich wie Noradrenalin, nur in höherer Dosierung, die Speichelsekretion der Submaxillaris, nicht aber die der Parotis erhöhen kann. Diese Wirkung kann durch Propanolol aufgehoben werden, was zeigt, dass sich in der Submaxillaris offenbar β_1-Rezeptoren befinden.

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Responses of the Resistance and Capacitance Vessels Reflexly Induced from the Cardiac Chambers

NEIL[1] suggested that the receptors of the right side of the heart may have a predominant influence on capacitance vessels, the receptors of the left heart having predominant influence on resistance vessels. This hypothesis was supported by ÖBERG[2]. However, there are no experimental data which could conclusively confirm or disprove this conception. While there is certain information on the responses of resistance vessels connected with cardiac reflexes, no information on capacitance vessels is practically available. Distension of the left atrium in cats is known to be followed by dilation of capacitance vessels in extremities and intestine[2]. An increase of blood pressure in the left ventricle in dogs results in a decrease of the venous blood return to the heart[3–6]. This investigation concerns the responses of resistance and capacitance vessels as reflex effects of the distension of the heart chambers.

Method. Experiments were performed on cats under urethane anaesthesia (1 g/kg), with the thorax open, under artificial breathing. Heparine was administered i.v. to prevent blood coagulation. Vasomotor responses resulting from a distension of the left atrium or ventricle were studied in preparations with the right heart bypass. Through catheters introduced into both venae cavae blood was passing to an extracorporal reservoir. By means of a perfusion apparatus the blood was pumped into the pulmonary artery through a catheter introduced in the succession via the right auriculum, atrium and ventricle. The pulmonary artery and the right auriculum were tightly ligatured on to the catheter. Blood entering the right heart chambers through the coronary sinus and thebesian veins was also diverted to the extracorporal reservoir through another catheter. The distension of the right heart chambers was made by inflation of a rubber balloon introduced via the central end of the anterior vena cava into the right atrium. In the course of the experiment the balloon was moved to the right ventricle.

A distension of the left heart chambers was made in the preparations with the bypassed left heart, a donor cat being used. The main pulmonary artery of a recipient cat was ligatured and the venous blood flowed to the donor's lungs through a catheter introduced into the right atrium via the auriculum. The level of the donor cat was 50 cm lower than that of the recipient cat. Blood oxygenated in the donor's lungs moved through a catheter into the extracorporal reservoir; from here it was taken by means of a perfusion apparatus and pumped into the thoracic aorta of the recipient cat through a T-shaped tube. The distension of the left heart chambers was made by an inflation of a rubber balloon introduced into the left atrium or ventricle via the left auriculum.

The responses of the resistance and capacitance vessels in the hindquarter preparations were studied with a method described in a previous communication[7].

Results. The distension of the right heart chambers (9 animals) was found to produce dilation of the resistance vessels and constriction of the capacitance vessels (Figure 1). Distension of the right atrium resulted in a decrease of vascular resistance (by 15.4 ± 3.6% on the average) in 13 experiments out of 14. In one experiment no changes were found. In 9 of 14 experiments an increase of venous outflow (by 6.2±1.2% on the average) was observed, in the other 5 experiments no changes of the venous outflow

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being found. The latent period for the resistance decrease was 4.0 ± 0.6 sec and for the increase of the venous outflow – 9.1 ± 1.5 sec. Distension of the right ventricle caused the resistance decrease (by $13.3 \pm 4.7\%$ on the average) in all 7 experiments. The venous outflow increased in most of these experiments (5 out of 7) by $8.3 \pm 1.6\%$ on the average. In the other 2 experiments no changes of the venous outflow were observed. The latent period for the response of the resistance vessels (3.5 ± 0.7 sec) was shorter than that for the increase of the venous outflow (11.6 ± 2.2 sec) both in experiments with the right ventricle distension and those with the stretching of the right atrium. The distension of the right ventricle resulted in a slightly greater increase of the venous outflow and less pronounced resistance decrease as compared with the distension of the right atrium. However, the difference found was not statistically significant.

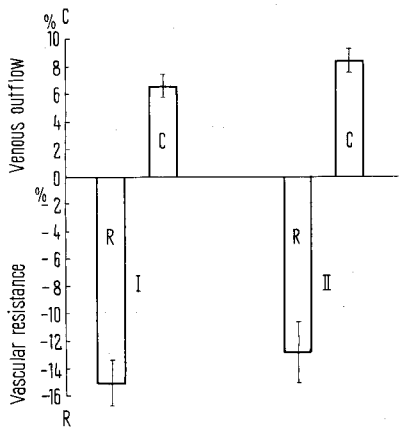

Fig. 1. Decrease of vascular resistance and increase of the venous outflow in a hindquarter preparation after distension of the right atrium (I) and ventricle (II). Designations: R, changes of vascular resistance in percent to a control; C, changes of the venous outflow in percent to a control.

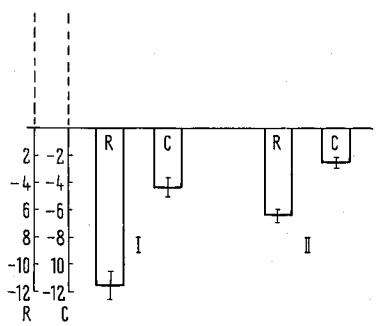

Fig. 2. Decrease of vascular resistance and venous outflow in the hindquarter preparation induced by distension of the left atrium (I) and ventricle (II). Designations are the same as in Figure 1.

The distension of the left heart chambers (5 animals) caused dilation of the resistance and capacitance vessels (Figure 2). As a result of the stretching of the left atrium, the vascular resistance and the venous outflow were shown to decrease by $11.8 \pm 1.7\%$ and $4.3 \pm 0.7\%$ respectively. The latent period for the responses of the resistance vessels (1.9 ± 0.3 sec) was much shorter than that for the shifts of the venous outflow (11.3 ± 3.3 sec). A distension of the left ventricle induced the same vasomotor responses, the vascular resistance being decreased by $6.5 \pm 0.9\%$ and the venous outflow (in 5 experiments of 6) by $2.9 \pm 0.6\%$. In one of the experiments the venous outflow remained unchanged. The latent period for the responses of the resistance vessels was 2.7 ± 0.6 sec and that for the venous outflow changes was 4.4 ± 1.8 sec. The quantitative difference between the responses of the resistance and capacitance vessels nduced by the distention of the left heart chambers was statistically insignificant.

Thus, the experiments performed showed the stimulation of any heart chamber to be followed by identical responses (dilatation) of the resistance vessels while the responses of the capacitance vessels were found to depend on the site of the stimulation of the cardiac receptory zones. The vasomotor responses resulting from the distension of the right chambers were more pronounced than those elicited from the left chambers. The experiments failed to reveal closer relationship between the left heart receptors and the resistance vessels on one hand and between the right heart receptors and the capacitance vessels on the other hand.

Conclusion. The distension of the bypassed right heart in a hindquarter preparation elicited a dilatation of the resistance vessels and mostly the constriction of the capacitance vessels (dilatation of the capacitance vessels was not observed in this series of experiments). The distension of the left heart chambers in the same experimental conditions resulted in dilation of both resistance and capacitance vessels.

Выводы. Растяжение камер правой половины сердца в условиях ее гемодинамической изоляции вызывает дилатацию резистивных сосудов препарата задних конечностей и в большинстве случаев констрикцию емкостных сосудов этой области (дилатации емкостных сосудов в этом случае не наблюдалось). Растяжение камер левой половины сердца в аналогичных условиях опытов ведет к дилатации как резистивных, так и емкостных сосудов препарата задних конечностей.

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Einfluss des luftelektrischen Feldes auf das Haut-Potential von *Rana esculenta*

Bei der Froschbauchhaut liegt ein Membransystem vor, das aus zwei Schichten besteht. Von aussen nach innen findet man als erste Membranschicht das Stratum corneum und das Stratum germinativum. Die zweite Membranschicht wird vom Corium gebildet. Die erste Membranschicht ist für Natrium- und Kaliumionen eine Diffusionsstrecke. Durch den zweiten Teil des Membransystems werden Natriumionen aktiv transportiert. Der Kaliumionentransport erfolgt hier nicht durch aktiven Transport, sondern durch einfache Diffusion. Auf dieses Membransystem