The tectoria is connected to the stereocilia which bridge the gap to the reticular lamina. These transmitter pins act on the cuticular plate which closes the upper side of the hair cells, and this diaphragm piston displaces the cytoplasm of the transducers and strains the fine nerve endings at their lower side.

The transducers are mounted in a lattice girder which consists of lower and upper rigid plate (basilar membrane and reticular lamina), the first one supporting the lower side of the transducers by means of the phalangeal cells, and the second one holding the upper rim of the transducers. These plates are connected by the rows of pillars towards their center, and at the edges by border cells and cells of Hensen. The 2 outermost transducer rows are shifted relative to the first and to the second transducer row, and this angle of shift corresponds to the oblique arrangement of the reinforcing fibers within the tectorial membrane. This contributes to the precision of the instrument. Tuning of first and of second order neurons and behaviour of synaptic connections further enhance the analising properties of the instrument described.

The experiments established 132 essential dimensions of parts as function of the distance of the place of measurement from the stapes, and some of the most important dimensional changes of the instrument along the cochlear partition, as well as natural frequencies of the tectoria of *Anoa bubalis* determined during the investigations and compared to the frequency function of the human ear, are shown in Figure 2.

Zusammenfassung. Basierend auf eingehenden Versuchs- und Messreihen am Hörorgan des Wasserbüffels (*Anoa bubalis*), dessen Innenohr dem menschlichen nahezu gleicht, wird ein kybernetisches Modell des Hörinstruments geschildert, das eine einfache und widerspruchslose Erklärung der Eigenschaften des Ohres gestattet.

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Changes in the Lumen of Coronary Vessels under Oligemic Hypotension

A number of papers describe the study of myocardial blood supply under oligemic hypotension[1–8]. Nevertheless data available are not sufficient to reveal whether active reactions of cardiac vessels contribute to changes of coronary blood flow in this case. Furthermore one cannot judge from these data of the shifts in the coronary vascular bed, arising immediately after start of hermorrhage, since coronary blood flow has not been recorded until the withdrawal of blood had been completed.

The present study was intended to reveal whether the active changes of coronary vessel lumen were possible under hypotension resulting from decrease in the circulating blood volume in cats.

Method. Experiments were carried out on cats (33) anaesthetized with urethane (1 g/kg), with the thorax open, under artificial breathing. Perfusion pressure in the left common coronary artery, into which blood was impelled by a constant blood flow perfusion pump from the same animal's femoral or carotid artery, revealed changes in the lumen of the coronary arteries. The catheter for perfusion was inserted via the left subclavian artery and the aortic arch into the left common coronary artery and fixed therein by ligature. Contractile force of the left ventricle was recorded with a miniature strain gauge similiar to one described elsewhere[9]. The strain gauge was sutured to the left ventricle surface. Pressure in the right atrium was recorded with a similiar strain gauge attached to the Marey tambour. Systemic blood pressure (in axillaris artery) and the coronary perfusion pressure were measured with mercury manometers. A decrease in the circulating blood volume was gained by withdrawal with a syringe of some blood from the animal through catheters inserted either into the inferior vena cava or left atrium, or femoral artery. After the systemic blood pressure had been reduced to 40–50 mm Hg (it would take 15 sec on the average) the blood removed was returned to the animal. The majority of experiments were performed on animals with denervated carotid sinuses.

Results. The decrease of circulating blood volume caused a fall of systemic blood pressure in all experiments, and in 19 experiments out of a total of 33 the increase of coronary perfusion pressure also occurred (Figure 1A), the latter having the latency of 4 sec on the average. Coronary vessel responses were accompanied with unaltered heart rate. The cardiac contractile force either increased or decreased, or did not change at all. The right atrial pressure fell in all animals.

The diminution of circulating blood volume in 7 other experiments out of a total of 33 brought about an increase of the coronary perfusion pressure with a latency of 45.3 sec on the average (Figure 1B). Coronary responses of this type were accompanied with unaltered heart rate and occurred with the shifts of systemic arterial and central venous pressure being very small or absent.

In 4 experiments out of a total of 33, the responses of both types were observed, one following the other (Figure 2).

The coronary responses of both types were observed in animals with bilateral cervical vagotomy. Atropinization did not exclude them.

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The data obtained showed that the decrease of circulating blood volume could evoke immediately, or in a short while, constriction of the coronary vessels. Since such a response was not preceded by any changes of the heart rate and since the ventricular contractile force shifts were either inconsistent in various experiments or absent, one can infer that the constriction of coronary vessels arising immediately after start of oligemic hypotension was not a result of an increase of the extravascular support. As the constriction of the first type had a short latency, one can suppose its neurogenic origin. On the other hand, the experiments showed that other types of coronary responses could also be observed as a result of oligemic hypotension. These responses, like ones of the first type, were not the effects of increased extravascular compression, because their occurrence did

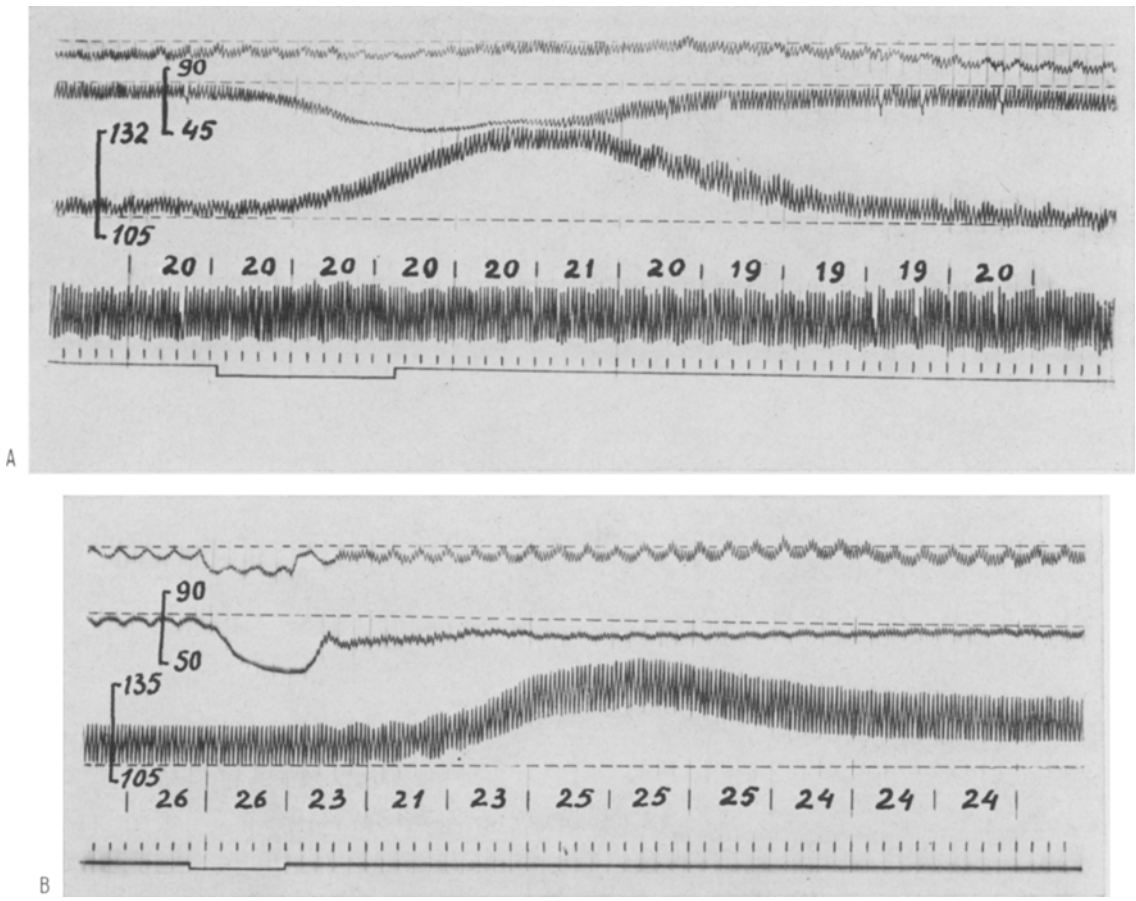

Fig. 1. Responses of the coronary vessels resulting from the oligemic hypotension: A, with a short latent period; B, with a greater one. From up to down: right atrial pressure, systemic blood pressure, coronary perfusion pressure, left ventricle contractile force (on Figure 1A only), the mark of start and finish of the extraction of blood. Scales in mm Hg, figures under coronary perfusion pressure – number of heart beats for 10 sec, vertical short lines – time mark (2 sec).

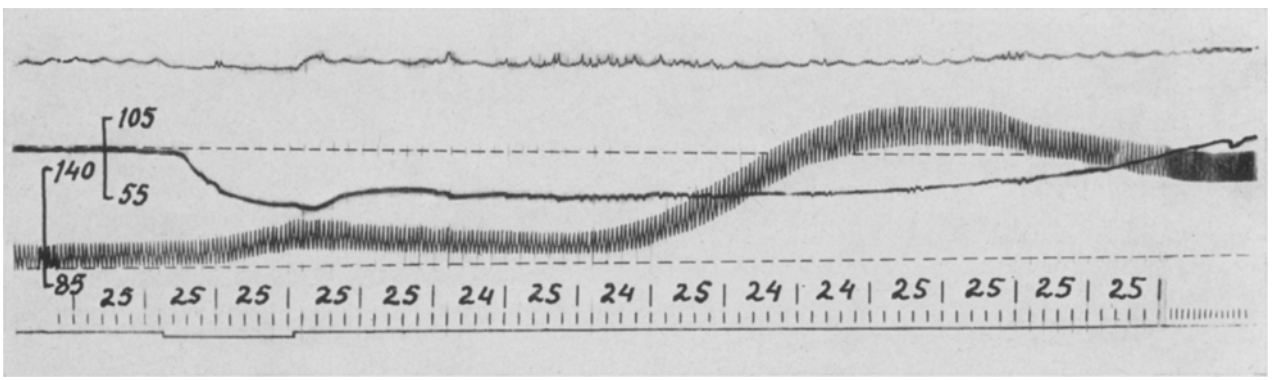

Fig. 2. Two-phase response of coronary vessels resulting from the decrease of circulating blood volume. Designations the same as on Figure 1B.

not coincide with the changes of cardiac activity and significant shifts of the systemic blood pressure. The great latency and duration characteristic for this type of coronary responses permit us to suppose them to be of humoral origin.

Conclusions. The decrease of circulating blood volume, resulting in oligemic hypotension, may evoke in anaesthetized cat the active constrictory coronary vessel responses of 2 types, one occuring immediately after start of the hypotension and the other having a significantly prolonged latency.

Выводы. В острых опытах на кошках с аутоперфузией левой общей коронарной артерии постоянным объемом крови показано, что уменьшение объема циркулирующей крови, достигавшееся удалением некоторого количества ее из организма, вызывает снижение общего артериального давления и активные констрикторные реакции коронарных сосудов двух типов, из которых первые возникают практически одновременно с началом гипотензии или с небольшим латентным периодом (около 4 секунд), а вторые имеют длительный период (около 45 секунд).

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Intrarenal Circulation in Hemorrhagic Hypotension

Hemorrhagic hypotension was supposed to be connected with extreme renal ischaemia, as judged by the clearance of PAH, even if the extraction ratio of PAH is taken into consideration (PHILLIPS et al.[1]). It is now well established that the decrease in clearance values is in part a technical consequence of the oliguria and considerable renal blood flow can be revealed by applying some direct method for its evaluation (SELKURT[2], BÁLINT[3], CARRIERE et al.[4], TRUNINGER et al.[5]).

The aim of this study was the determination of the intrarenal distribution of blood flow during hemorrhagic hypotension. Our observations were made on dogs narcotized by chloralose. The following methods were simultaneously applied: (1) The left kidney was approached by laparatomy and the left renal vein was connected by means of a plastic catheter to the external jugular vein. A T-extension of the tube made the direct measurement of renal blood flow (RBF total) and the taking of renal venous blood samples possible. (2) Extraction ratios (at plasma PAH levels of about 2 mg/100 ml) and Tm_{PAH}-values (at plasma PAH levels of about 20 mg/100 ml) were assessed. (3) Blood flow through various parts of the kidney was determined by SAPIRSTEIN's[6] method (as modified by HÁRSING and PELLEY[7]) based on the fractional distribution of i.v. injected ^{86}Rb.

Having determined RBF and E_{PAH} in the normotensive (control) periods, the dogs were bled to arbitrarily set blood pressure levels of 60–80 mm Hg; this level was kept constant by further bleeding or retransfusion, respectively. 45 min after inducing the hemorrhagic hypotension RBF_{total}, E_{PAH} and Tm_{PAH} were determined. The suitable dose of ^{86}Rb was then rapidly injected and serial arterial blood samples were taken. 60 sec after the ^{86}Rb injection the animals were killed by the i.v. injection of a concentrated KCl solution.

The appropriate arterial and renal venous blood samples were analysed for PAH-, creatinine- and ^{86}Rb-activity. Slices of renal cortex, outer and inner medulla were cut and their activity was measured. The acitivity of the remaining kidney substance was assessed as well. Details of the various techniques are published elsewhere[8].

The directly measured RBF is considered as the total blood flow: RBF_{total} (calculated for 100 g kidney). The results of the ^{86}Rb-fractionation yield the blood flow passing through the capillaries socalled nutritive flow (RBF_{nutr}). The nutritive flows through cortex, outer and inner medulla, are calculated for 100 g kidney under the assumption that cortex, outer medulla and inner medulla weigh 70 g, 20 g and 10 g, respectively, in 100 g kidney. The fact that $RBF_{cort} + RBF_{o.m.} + RBF_{i.m.} + RBF_{non\ nutr} \sim RBF_{total}$ (Table I), proves that our assumption concerning the distribution of kidney weight is essentially correct.

Table I. Intrarenal distribution of blood flow

	Control conditions		Hemorrhagic hypotension	
	ml/min per 100 g kidney $\bar{x} \pm s_{\bar{x}}$ $n = 12$	ml/min per 100 ml RBF_{total}	ml/min per 100 g kidney $\bar{x} \pm s_{\bar{x}}$ $n = 11$	ml/min per 100 ml RBF_{total}
Arterial pressure mmHg	133 ± 4		71 ± 3	
RBF_{total}	421 ± 37	100.0	163 ± 24	100.0
RBF_{nutr}	400 ± 34	95.0	130 ± 15	79.8
RBF_{cort}	338 ± 29	80.3	106 ± 11	65.0
$RBF_{o.m.}$	56 ± 8	13.3	19 ± 4	11.6
$RBF_{i.m.}$	8 ± 1	1.9	3 ± 0.6	1.9
$RBF_{non\text{-}nutr.}$	21 ± 18	5.0	34 ± 12	20.9
	423	100.5	162	99.4

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