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).

V. I. OVSYANNIKOV and B. I. TKACHENKO

Laboratory for Circulation, Institute of Experimental Medicine, Leningrad (USSR), 30 December 1968.

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

[1] R. A. PHILLIPS, V. P. DOLE, P. B. HAMILTON, K. EMERSON, R. M. ARCHIBALD and D. D. VAN SLYKE, Am. J. Physiol. *145*, 314 (1946).
[2] E. E. SELKURT, Am. J. Physiol. *145*, 699 (1946).
[3] P. BÁLINT, A. FEKETE and J. STURCZ, Acta physiol. Acad. Sci. Hung. *17*, 287 (1960).
[4] S. CARRIERE, C. D. THORBURN, C. C. C. O'MORCHOE and A. BARGER, Circulation Res. *19*, 167 (1966).
[5] B. TRUNIGER, S. M. ROSEN and D. E. OKEN, Klin. Wschr. *44*, 857 (1966).
[6] L. A. SAPIRSTEIN, Am. J. Physiol. *193*, 161 (1958).
[7] L. HÁRSING and K. PELLEY, Pflügers Arch. ges. Physiol. *285*, 302 (1965).
[8] P. BÁLINT, J. BARTHA and A. FEKETE, Acta physiol. Acad. Sci. Hung., in press.

The intrarenal distribution of blood flow in control conditions is shown in the left columns of Table I and II. The difference between RBF_{total} and RBF_{nutr} is *not* significant, i.e. practically the whole blood flow of the kidney passes through the peritubular capillaries. The ratio of cortical to total blood flow amounts to 0.80. Extraction ratio of PAH shows a mean of 0.79. The identity of these figures (i.e. 0.80 and 0.79) favours the conception of REUBI[9] that in the cortex $E_{PAH} \sim 1$ and in the medulla $E_{PAH} \sim 0$.

In hemorrhagic hypotension (right columns of the Tables) both total and nutritive blood flows are diminished; the decrease in blood flow is more pronounced than that of arterial pressure and the calculated renal resistance is somewhat increased. There is a significant difference between the total and nutritive flow values with about 20% of the total flow passing through non-nutritive channels. The cortical (nutritive) blood flow is about 15% less than in control conditions. The ratio cortical/total blood flow and the extraction ratio of PAH amount to 0.66 and 0.64, respectively.

It seems to be an obvious conclusion that in hemorrhagic hypotension about 15% of total blood flow avoid the cortical peritubular capillaries. Similar conclusions were drawn by GÖMÖRI et al.[10,11], who demonstrated morphologically arterio-venous shunts in corrosion preparations.

It is assumed that, in analogy to many kinds of tissues (ZWEIFACH[12]), the 'microcirculation' within the kidney consists of arterio-venous capillaries (thoroughfare channels), from which the true capillaries branch off; at the origin of the outflowing branches 'precapillary sphincters' control the flow in the true capillaries. It is further assumed that the enhanced sympathetic discharge caused by bleeding leads to arteriolar constriction within the kidney and the augmented tone of the precapillary sphincters shunts off part of the blood from peritubulary capillaries.

Zusammenfassung. Durch simultane Ermittlung der totalen und der nutritiven Nierendurchblutung, sowie der PAH-Extraktion und -Sekretion, wurde festgestellt, dass während einer hämorrhagischen Hypotonie (1) der Nierenwiderstand ansteigt, (2) der relative Anteil der Markdurchblutung unverändert bleibt und (3) etwa 15% der gesamten Durchblutung in der Rinde durch arteriovenöse Anastomosen geleitet wird bzw. die peritubulären Kapillaren vermeidet.

Table II

	Control conditions $\bar{x} \pm s_{\bar{x}}$ (12)	Hemorrhagic hypotension $\bar{x} \pm s_{\bar{x}}$ (11)
C_{creat}	48 ± 4	5 ± 2
Tm_{PAH}	12.5 ± 1.3	6.1 ± 1.1
$E_{PAH}{}^{x}$	0.79 ± 0.02	0.64 ± 0.07
$\frac{RBF_{cort}}{RBF_{total}}$	0.80 ± 0.04	0.67 ± 0.04

$^{x}L_{PAH}/Tm_{PAH} < 0.7$.

P. BÁLINT, J. BARTHA and AGNES FEKETE

Physiologisches Institut, Medizinische Universität, Budapest (Ungarn), 8. Januar 1969.

[9] F. REUBI, Helv. med. acta *25*, 516 (1958).
[10] P. GÖMÖRI, S. MUNKÁCSI, Z. NAGY, L. TAKÁCS and K. KÁLLAY, Acta med. Acad. Sci. Hung. *18*, 119 (1962).
[11] P. GÖMÖRI, Z. NAGY and I. JAKOB, Acta med. Acad. Sci. Hung. *20*, 153 (1964).
[12] B. W. ZWEIFACH, Am. J. Med. *23*, 684 (1957).

Über den Einfluss von Natriumsulfit, Natriumtellurit und Natriumselenit auf die Retention von Zink, Kadmium und Quecksilber im Organismus

Bei akuten Versuchen an Mäusen haben wir nachgewiesen, dass Natriumselenit, das gleichzeitig mit $HgCl_2$ appliziert wurde, die Retention des Quecksilbers im Organismus bewirkt, wobei es zu wesentlichen Veränderungen in der Verteilung des Quecksilbers in Organen kommt[1].

Bei Versuchen an Mäusen (♂, Gewicht 18–20 g) wurde die Retention der Metalle im Organismus nach i.v. Applikation von $^{65}ZnCl_2$, $^{115m}CdCl_2$ und $^{203}HgCl_2$ (mit Träger) sowie der Einfluss der gleichzeitigen s.c. Applikation von Natriumsulfit, Natriumtellurit und Natriumselenit auf diese Retention vorfolgt. Alle Verbindungen wurden in der Dosis 3×10^{-3} mM/kg (Aktivität der mit Radioisotopen markierten Chloriden etwa 3 μCi per Maus) appliziert. Jede Kombination der verabreichten Stoffe wurde immer einer Gruppe von 8 Mäusen appliziert. Am 1., 7. und 28. Tage nach der Applikation wurde die Retention der Metalle mittels Gesamtkörpermessung der Tiere mit dem Szintillationskristall NaJ (Tl) und unter Anwendung von Gammaspektrometrie festgestellt; bei Kadmium wurde die Bremsstrahlung X gemessen. Die Ergebnisse sind in der Tabelle zusammengefasst.

Am wirksamsten zeigt sich Natriumselenit, das nicht nur die Retention des Quecksilbers, sondern auch des Kadmiums bewirkt. Natriumtellurit verursacht nur die Retention des Quecksilbers, wobei seine Wirkung schwächer ist als die des Selenits. Natriumsulfit hat keine erhöhte Retention der benützten Metalle im Organismus zur Folge. Die Ausscheidung von Zink wurde von keinem der verwendeten Stoffe wesentlich beeinflusst.

Natriumselenit und Natriumtellurit werden im Organismus reduziert[2,3]. Das Redoxpotential der benützten

[1] V. EYBL, J. SÝKORA, M. HRNČÍŘOVÁ und F. MERTL, Czech. Fysiol. *17*, 156 (1968).
[2] F. HOFMEISTER, Arch. exp. Path. Pharmak. *33*, 198 (1894).
[3] I. ROSENFELD und O. A. BEATH, J. biol. Chem. *172*, 333 (1948).