unchanged. Data on the activity of $(Na^+ + K^+)$-ATPase in whole homogenate after uninefrectomy have not been published before. In Table II it is seen that the activity per mg kidney remained unchanged. These findings suggest a preferential increase in the specific activity of enzyme in cellular constituents collected in the microsomal fraction. $(Na^+ + K^+)$-ATPase seems not to be selectively induced in the enlarging kidney[1]; but it increases in amount, along with the increase in kidney weight, after uninefrectomy.

After uninefrectomy and bilateral adrenalectomy, the activity of $(Na^+ + K^+)$-ATPase both in whole homogenate and in the microsomal fraction was significantly lower than the corresponding activity in kidneys of sham-operated and uninefrectomized rats (Table II). This decrease in activity dissociated not only from the increase in kidney weight (Table I), but also from a moderate increase in activity of glucose-6-phosphatase in the microsomal fraction (Table II). As the level of Mg^{++}-ATPase in homogenate and in the microsomal fraction was unchanged, it is unlikely that the changes in activity of $(Na^+ + K^+)$-ATPase are due to unspecific alterations in the composition of the preparations[6].

Thus, the stimulus causing compensatory hypertrophy of the remaining kidney after uninefrectomy could not prevent the decrease in activity of $(Na^+ + K^+)$-ATPase after adrenalectomy. On the contrary, a dissociation between compensatory renal enlargement and the level of $(Na^+ + K^+)$-ATPase was found. It is therefore unlikely that a causal relationship exists between compensatory renal enlargement after uninefrectomy and induction of $(Na^+ + K^+)$-ATPase. The enzyme activity seems to be under control of the adrenal cortical steroids. It is, however, not possible at present to decide whether this control is exerted directly, or whether the changes in activity reflect adaptation to sustained changes in the functional demands on the enzyme[12].

Zusammenfassung. Nach Uninephrektomie und bilateraler Adrenalektomie entsteht eine Dissoziation zwischen der Nierenvergrösserung und der $(Na^+ + K^+)$-ATPase Aktivität. Es besteht wahrscheinlich kein Zusammenhang zwischen der Nierenvergrösserung und der Induktion von $(Na^+ + K^+)$-ATPase. Auch in der vergrösserten Niere sind die Nebennierenhormone notwendig, um die $(Na^+ + K^+)$-ATPase zu erhalten.

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Reciprocal Autoregulation of Blood Flow and Blood Pressure

The following results led us to the conclusion that the peripheral regulation of blood flow through different arterial beds depends on an equilibrium of 2 reciprocally acting autoregulatory control mechanisms. The arterial input impedance

$$R_i(p, \dot{Q}) = p/\dot{Q}\,, \qquad 1$$

as defined in the frequency domain[1,2], is the quotient of the complex arterial pressure p and the complex arterial flow $\dot{Q}$. The existence of local control mechanisms is expressed by assuming R_i in equation 1 to be an implicite function of pressure and flow[2]. Considering only small perturbations of pressure and flow, the following relation can be derived from equation 1:

$$dp/d\dot{Q} = R_{iin} = R_{io}\frac{1 + G_q}{1 + G_p}. \qquad 2$$

R_{iin} is the 'linearized' input impedance. R_{io} is the impedance set value around which small perturbations of pressure and flow are examined. Q_o and p_o are the corresponding set values of flow and pressure.

$$G_q = (\dot{Q}_o/R_{io})\,\delta R_i/\delta\dot{Q} \qquad 3$$

is defined as the gain of the autoregulation of flow and reflects the controlling influence of arterial flow on the impedance R_i.

$$G_p = -(p_o/R_{io})\,\delta R_i/\delta\dot{Q} \qquad 4$$

is defined as the gain of the autoregulation of pressure and reflects the controlling influence of the arterial pressure on the impedance. The choice of a negative sign in the definition of G_p is suggested by the direction of the expected response, as explained in Figure 1.

Equation 2 was used to examine the autoregulatory frequency response of different arteries. We perfused the A. mesenterica sup., the A. femoralis and the A. renalis of different anesthetized dogs (Morphin-Chloralose) with arterial blood. The blood flow was provided by a peristaltic

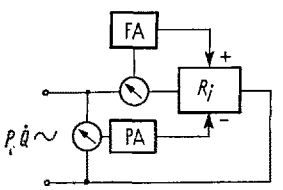

Fig. 1. Schematic diagram demonstrating possible controlling influences of autoregulation of flow (FA) and hypothetic autoregulation of pressure (PA) on the input impedance R_i of an arterial bed.

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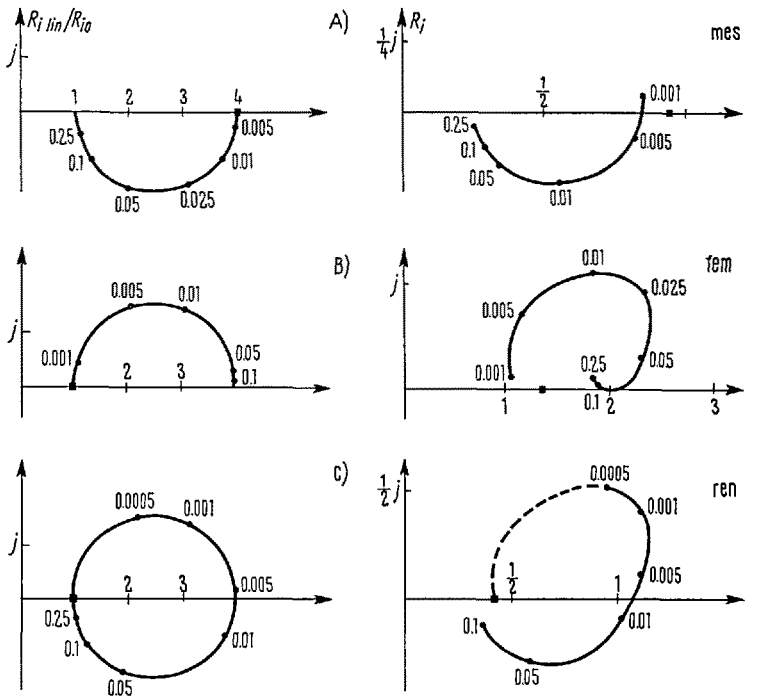

Fig. 2. Left column: Calculated Nyquist diagrams of the normalized input impedance according to equation 2. A) Pure autoregulation of flow, $G_p = 0$, $G_q = 3/(1 + j\omega\, 5)$, where $j = (-1)^{1/2}$ and ω: circular frequency (rad. sec^{-1}). B) Autoregulation of pressure, $G_p = 3/(1 + j\omega\, 100)$. A constant gain factor $G_q = 3$ was assumed for proper scaling of the diagram. C) Autoregulation of pressure and flow. $G_q = 3/(1 + j\omega\, 5)$ and $G_p = 3/(1 + j\omega\, 10^3)$. Right column: Measured Nyquist diagrams of the input impedance of the mesenteric artery (MES, units mm Hg/ml/min), femoral artery (FEM, units mm Hg/ml/min) and renal artery (REN, units mm Hg/ml/100 g/min). Average dog weight 20 kg. Flow was used as modulated input signal with the modulated amplitude being about 40% of the mean flow value. Parameters frequency in Hz. Squares: Zero frequency resistance as measured during constant perfusion with mean flow value.

pump from a cannula inserted in one femoral artery. The pump rate could be modulated. Using a TR-48 analog computer in the control circuit both, the arterial perfusion flow or local arterial pressure could be modulated sinusoidally in the frequency range between 0.0005 Hz and 0.25 Hz according to signals from a function generator. The responses of pressure and flow, respectively, were recorded. Each arterial bed was examined in at least 3 different animals. The responses were reasonably linear in the examined range.

The right column of Figure 2 shows characteristic results from the A. mesenterica sup. (MES), the A. femoralis (FEM) and the A. renalis (REN) in the form of Nyquist diagrams of the input impedance. The left column of Figure 2 shows Nyquist diagrams calculated according to equation 2 for the following assumptions: A) pure autoregulation of flow; B) autoregulation of pressure and C) reciprocal autoregulation of pressure and flow. First order transfer functions were assumed for G_q and G_p, as indicated in the figure.

From these results it can be concluded that there exists an autoregulation of pressure besides the wellknown autoregulation of flow[3] in all 3 examined beds. Although the experimental results certainly are distorted by nonlinear effects, it can be seen that the effect of the autoregulation of pressure is most marked in the femoral artery, whereas the mesenteric artery exhibits nearly pure autoregulation of flow. The renal artery shows both autoregulation of flow and pressure. The time constant of the latter is in the order of 1000 sec.

The existence of an autoregulation of pressure indicates that local control mechanisms take part in the dynamic regulation of the arterial pressure and may do so even in the absence of neural control[4].

Zusammenfassung. Der Eingangswiderstand verschiedener Arterien wurde im Niederfrequenzbereich (0,0005 bis 0,25 Hz) mittels sinusförmig modulierter Pumpen-Perfusion mit arteriellem Blut an narkotisierten Hunden gemessen. Die Resultate lassen die Existenz zweier reziprok wirkender Autoregulationsmechanismen vermuten, deren Frequenzcharakteristik mit Hilfe eines einfachen linearen Modells simuliert werden konnte.

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Effects of L-Dopa Alone and in Combination with Dopa Decarboxylase Inhibitors on the Arterial Pressure and Heart Rate of Dogs

L-3,4-Dihydroxyphenylalanine (L-dopa) is known to produce clinical improvement in patients with Parkinson's disease[1]. Side effects of L-dopa given orally include hypotension[1–3], while by i.v. administration L-dopa elevates arterial pressure in man[4]. In experimental animals the acute effect of L-dopa on the arterial pressure is species dependent. By i.v. administration L-dopa elevates arterial pressure in cats[5] but lowers it in rabbits[6].

The possible mechanisms of the hypotensive action of L-dopa include: 1. replacement of norepinephrine at the peripheral sympathetic nerve endings with dopamine, which is a weaker α-adrenergic stimulant than norepine-