THE CORTICAL AND MEDULLARY BLOOD FLOWS
OF THE ISOLATED DOG'S KIDNEY

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Since TRUETA and associates (1947) published that under certain experimental conditions there might be a deviation in the distribution of blood through the cortex and medulla of the kidney several investigators have reported that the cortical and medullary blood flows behave differently. For example: a) CARGIL (1948) reported that during the intravenous administration of serum albumin the flow through the medullary circuit increased in a higher magnitude than the cortical flow, b) KRAMER et al. (1960) found that during a sudden rise in the perfusion pressure, the blood flow through the medullary circuit increased while the cortical and total renal blood flow remained constant, c) THURAU et al. (1960) showed that the medullary flow increased proportionately more than the cortical flow during osmotic diuresis and finally d) Pilkington et al. (1965) have shown that acetylcholine (50-80 μ moles/min) injected into the renal artery produced a greater increase in the medullary flow than in the cortical flow. The latter authors also reported that norepinephrine reduced cortical flow markedly, while medullary flow was not significantly changed.

In the present study we are reporting further evidences which show that in the kidney the cortical and medullary blood flows behave differently.

METHOD

The experiments were performed on twenty eight anesthetized (pentobarbital 30 mg/kg) dogs weighing 17-25 kg. The left kidney was approached through a flank incision, without opening the peritoneal cavity. The renal artery, the renal vein and the ureter were dissected. After heparinization (1000 I.U./kg) the kidney was removed and its artery, vein and ureter were cannulated. The kidney was then immersed into a mineral oil bath at 38°C. To perfuse the kidney the renal artery was connected to both femoral arteries of a donor dog (heparinized). To induce a sudden increase

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in the perfusion pressure, a reservoir containing heparinized blood and a large volume of air at a given pressure was used (Fig. 1). This system was connected to the renal artery of the isolated kidney and simultaneously the flow from the donor dog was stopped. The perfusion pressure was kept at a constant level until the renal blood flow became steady. Under these conditions the perfusion pressure was suddenly increased in about 30–80 mm Hg above the initial value and after 30–60 seconds it was lowered to the control level. The venous return was drained against a hydrostatic pressure of 8 mm Hg and afterwards by gravity to a flask. The blood was returned by a multiple finger pump to the donor animal (Fig. 1).

The changes in cortical and medullary blood flow were measured with heated thermocouples (Fig. 2). This circuit is a modification of the one first employed in tissue by Gibbs in 1933. The thermojunctions used by us were made out of constantan and cooper wires. A constantan wire of 1.3 cm in length and 88 μ in diameter was divided into two sections by soldering two copper wires in a point where the

![Diagram of the experimental set-up.](image)

Fig. 1. *Diagram of the experimental set-up.* The isolated and perfused dog kidney was placed into a mineral oil bath at 36–38°C. A non pulsatile perfusion pressure system consisting in a reservoir containing heparinized blood and a large volume of air at a given pressure was used. The sudden increase in the perfusion pressure was produced by connecting the air reservoir to a higher pressure air tank (HPS). Between two experimental trials the valve (2) was closed and the kidney was perfused with blood coming from a heparinized donor dog. The blood from the renal vein was drained against a hydrostatic pressure (8 mm Hg) and afterwards by gravity to a flask. The blood was returned by a multiple finger pump (P) either to the donor dog (FV) or to the blood reservoir flask. The local changes in blood flow was measured by thermocouples inserted in the cortex (C) and in the medulla (M). The total blood flow was measured by a rotameter (R) connected in series to the renal artery. The perfusion pressure (PP) and the venous pressure (VP) were measured by transducers connected to the renal artery and to the renal vein, respectively. The urinary flow (U) was estimated by an electrical drop counter.
FIG. 2. Circuit used to measure the local blood flow changes. 1) Source for heating; a 6 volts storage battery was connected in series with a rheostat of 40Ω and an ammeter (A). 2) The thermojunction formed by 50 μ diameter copper wires (broken line) and a 88 μ diameter constantan wire (solid line). The section of 0.6Ω was used as a heater filament and another of 0.2Ω as the recording element. 3) Balancing circuit and 4) a high gain DC amplifier (5Pl, Grass) to be connected to the poligraph.

Two thermopairs of this type were implanted, one in the cortex and the other in the medulla, being cautious to insert both the heater as well as the recording ends in each one of these regions. The changes in blood flow were expressed as a percentage of the initial flow. The difference between the thermocouple voltage output produced by the initial level of the flow and the voltage acquired when the flow was momentarily stopped by clamping the perfusion system, was considered as 100 per cent. The locations of the thermocouples were verified macroscopically immediately after each experiment and histologically by hematoxylin eosine staining afterwards.

The total renal blood flow was measured by a rotameter connected in series to the renal artery. The perfusion and venous pressures were measured by transducers (Statham P23 AC and P23 DC) connected in parallel to the renal artery and vein respectively. The urinary flow was measured by a drop counting system (Wheatstone bridge).

RESULTS

In the present communication we chose those kidneys which showed a blood flow over 1.8 ml/g/min during the whole experiment when perfused with a pressure of 100 mm Hg.

Changes in the regional flow produced by sudden increase in the perfusion pressure.

The sudden increase in the perfusion pressure, from 100 mm Hg of initial perfusion level to 180 mm Hg, produced an increase of the total renal blood
flow (Fig. 3). In this particular case, an increase of 80 per cent in the perfusion pressure produced only a 30 per cent increment in total flow. The local cortical and medullary flow also increased as a result of the abrupt increment in the perfusion pressure. The increase in the cortical flow showed three different phases. a) an initial increase in flow which reached its peak simultaneously with the maximal value of total flow. b) The flow started to decrease without reaching the control level. This decrease was more evident approximately 10 to 20 seconds after the perfusion pressure was elevated. c) The third phase consisted of a further increase in flow which lasted as long as the perfusion pressure was kept high (Fig. 3).

The double-humped cortical response appeared only when the initial perfusion pressure level was above 70 mm Hg and less than 160 mm Hg. If the initial perfusion pressure was not within these values the cortex responded with a monophasic increase. The pressure gradient necessary for evoking a double-humped response varied between 30 to 80 mm Hg.

In contrast to the cortical response, the medullary flow always responded with an increase when the perfusion pressure was abruptly raised. This increase was relatively greater than the increase in both the total renal and

![Graph showing perfusion pressure (PP), total renal blood flow (RBF), changes in medullary flow, changes in cortical flow, and venous pressure (VP).](image)

**Fig. 3.** Effects of the sudden increase in the perfusion pressure on the regional blood flows. Up to down the records represent the perfusion pressure (PP) in mm Hg, the total renal blood flow (RBF) in ml per minute per gram of kidney, changes in the medullary flow, changes in the cortical flow and the venous pressure (VP). Note that the cortical flow showed two humps in contrast to the medullary flow that only was increased. The total renal blood flow increased (30 per cent) although the increment in the perfusion pressure was of larger magnitude (80 per cent).
cortical blood flows (Fig. 3) and it occurred independently of the initial perfusion pressure values.

The venous pressure also increased during the sudden raise in perfusion pressure (lowest trace in Fig. 3).

When the perfusion pressure was lowered rapidly to the initial level, the total renal flow and the cortical flow diminished abruptly below the control level, while the medullary flow decreased slowly and sometimes remained slightly higher than the control level. The venous pressure returned to the control value approximately with the same temporal course as the medullary flow.

The increase in the perfusion pressure produced a late increase in the urinary flow which appeared simultaneously with the late increment in cortical flow (Fig. 4-I). When the perfusion pressure was abruptly returned to the control level the urinary flow gradually recovered its initial value.

**Effects of the KCN on both the cortical double-humped response and the urinary flow.**

When the kidney was perfused with blood containing 4 mM/L of KCN,

![Fig. 4. Effects of the KCN on the regional flows.](image)

The tracings show: A, perfusion pressure; B, the total renal blood flow; C, the medullary flow; D, the cortical flow and E, urine drops. I, before and II after KCN (4 mM/L added to the perfusing blood). Note that the cortical flow changed from the two-humped form in I to a monophasic response parallel to the perfusion pressure in II. The urinary flow after KCN increased (9-10 folds) in comparison to the control value (I-E). The sudden increase in the perfusion pressure also increased abruptly the urinary.
the total renal blood flow increased (20 to 50 per cent). The urinary flow increased substantially (8 to 12 times) and in addition, the urine output became continuous (Fig. 4-II).

In the KCN poisoned preparation, the sudden increase in the perfusion pressure produced increments in the medullary, the cortical and the total renal blood flows. It is important to mention that the cortical double-humped response, described above, was transformed into a monophasic pattern, parallel to the changes in the perfusion pressure (Fig. 4-II). During the increased perfusion pressure there was a considerable increment in the urinary flow (lowest trace in Fig. 4-II).

When the perfusion pressure was lowered to the control level the total renal flow, the cortical and the medullary flow decreased slightly below the control values. The urinary flow returned slowly to the control level (Fig. 4-II).

Renal blood flow changes produced by acetylcholine injections.

Acetylcholine injected into the renal artery produced different responses depending upon the doses. An increment in total renal blood flow and in the cortical and medullary flows was evoked by low doses of acetylcholine

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**Fig. 5.** Effects of the intra-arterial injection of acetylcholine (2.5 µg/g of kidney). The tracings represent (up to down) the total renal blood flow (RBF) in milliliters per minute per gram of kidney, the changes in the medullary blood flow and those in the cortical blood flow. Note that the medulla and the cortex changes have a temporal course in a mirror image. At the arrow the acetylcholine was injected into the renal artery.
(0.5 µg per gram of kidney), as reported previously by PilKington et al. (1965). In contrast, larger doses (2.5 µg per gram of kidney) decreased the total renal blood flow, as shown by McGiff et al. (1967). Concerning to the responses of the cortical and medullary blood flows they differed significantly from one another. The cortical flow showed an initial decrease which occurred concomitantly with the diminution in the total blood flow. When the total flow settled to the control value, the cortex showed a prolonged increase in blood flow. On the other hand the medullary blood flow increased simultaneously with the decrease of the total and cortical blood flows. When the total blood flow returned to the control value the medullary blood flow showed a marked decrease. The decrease in blood flow in the medulla and the increase in flow in the cortex occurred approximately at the same time (Fig. 5).

Effects of an α adrenergic blocking agent (MA 1277) on the responses produced by the sudden increase in the perfusion pressure and by the intra-arterial injection of acetylcholine.

The α adrenergic blocking agent MA 1277 (Miles laboratories, Rodríguez et al., 1965) was added to the perfusing blood in such doses (3-5 mg/l) that the intra-arterial injection of one microgram of adrenaline did not change the cortical, medullary or total renal blood flows. In these conditions the basal flow did not change and the cortical and medullary responses produced by the sudden rise in perfusion pressure also were unaffected. However, the injection of acetylcholine at a dose of 2.5 microgram per gram of kidney produced an increase in the total renal blood flow, that is, the response was similar to that elicited by the small doses of acetylcholine (0.5 microgram per gram of kidney).

DISCUSSION

The sudden increase in the perfusion pressure augmented the total blood flow. However, this modification was not proportional to the magnitude of changes in the perfusion pressure as mentioned by several authors (see review Smith, 1939-40). The local responses of the medulla and the cortex to a sudden increase in pressure are significantly different; the medullary flow increased while the cortical flow showed a double-humped response. This latter response resembles that obtained by Thurau et al. (1959). In their experiments the kidney weight changes were accompanied by a transient increase in the capillary volume and when the total flow settled to lower levels, the capillary blood content decreased to a certain extent. From these findings they concluded that the initial rapid increment was due to the filling of the vessels; the transient drop to the myogenic response, that is
to vasoconstriction and the secondary rise probably due to the increased transudation of fluid through the capillaries, because of the elevated pressure and increased flow. Our results support to certain extent the interpretation given by Thuraus et al. (1959) for the double-humped response. The initial rapid flow increment may be due to the filling of vessels with blood. The transient decrease which does not reach the control value, could be originated by the vasoconstriction of the arteriolar muscle, since the transient decrease in the cortical flow was only observed when the sudden increase in the perfusion pressure reached a certain value (threshold) and when the initial perfusion level was above 70 mm Hg and below 160 mm Hg. The fact that under 70 mm Hg of initial pressure level, the pressure gradient applied produced only a monophasic increase, suggests that this stimulus does not reach the threshold to elicit the muscular response and therefore the thermocouple only tends to cool by the filling of vessels. If the initial perfusion level was over 160 mm Hg, the vascular bed was already distended and when the pressure gradient was applied the muscular contraction would be isometric, giving therefore a monophasic increase. The above mentioned vasoconstriction does not seem to be mediated by the sympathetic activity since the use of the $\alpha$ adrenergic blocking agent did not affect the transient decrease in the cortical flow. The transient decrease in the cortical flow might be due to an active contraction elicited by the sudden distention of the arteriolar muscles. Regarding to this possibility, it is well known since Bayliss (1902) that the muscular coat of arteries contracts when stretched. In our experiments, the fact that the KCN abolished the cortical double-humped response also supports this possibility. Electrophysiological studies on the smooth muscle have shown that when the preparation was stretched the taenia coli fibers depolarized and the burst of spike potentials were prolonged (Bulbring, 1955). Although this phenomenon has not been demonstrated in the smooth muscle fibers of arteries it could be possible that the arterial muscle behave in a similar way. Concerning to the late increase in the cortical flow, it may be attributed to a relaxation of the preglomerular arteriolar musculature, since the urinary flow increased just at the moment of the appearance of the late cortical increase (Fig. 4).

Whichever the mechanism of the cortical double-humped response would be, the site in which this phenomenon occurred might be the preglomerular arteriole, since during the double-humped response of the cortex, the urinary flow presented a slight and late increase (Fig. 4-I) in contrast with the immediate and considerable increase shown when KCN was added (Fig. 4-II). Forster et al. (1947) and Selkurt et al. (1949) postulated the afferent arteriole as the responsible for the renal autoregulation; their postulation was based on calculation of the renal afferent and efferent arteriolar resistance during the changes in kidney perfusion pressure.
THURAU et al. (1962) have demonstrated that the afferent arteriole to the glomerulus is the site of autoregulation in the rat kidney.

In order to explain the considerable difference between cortical and medullary flows during the sudden increase in the perfusion pressure we can mention the data reported by KRAMER et al. (1960). They suggested that the medullary vessels were not participating in the kidney autoregulation. Our results are in agreement with those of Kramer, since the medullary flow increased proportionally to the perfusion pressure when it was raised. There is however, another possible explanation for this phenomenon. Considering the afferent arteriole to the glomerulus as the main site of the autoregulation of flow and the fact that the medullary blood flow did not react in a double-humped manner, one can assume that in this condition the medullary blood flow supply comes from another source in addition to the efferent blood vessels of the juxtamedullary glomeruli, such as the arteria rectae verae (CHRISTENSEN, 1952), Ludwig's arteriole and the spiral vessels (BAKER, 1959). These vessels could play a role in the diversion of blood above discussed. On the other hand, arterio-venous anastomoses have been described between the spiral arteries and the sinusoides (BARRIE et al. 1950), the latter draining into the interlobar veins. These arterio-venous shunts may accelerate the blood flow running through the medulla.

In our experiments the total renal blood flow pattern did not resemble that of cortical flow (Fig. 3). There are some data showing that the cortical blood flow amounts between 80% (THORNBURN et al., 1963) and 90% (REUBI, 1958). Although in our experiences it was difficult to measure the absolute value of the blood flow through either the cortex or the medulla, it is important to mention that the transient halfway back in the cortical flow represents only a 10% of the cortical flow (Fig. 3). In other experiments, the above mentioned transient halfway back in the cortical flow was of smaller magnitude; the average of 24 experiments being 3.5%. The fact that the medullary flow increased relatively more than the increase in both the total renal and cortical blood flows, might suggest a diversion of cortical blood to the medullary circuit that additionally would contribute to mask the cortical double-humped phenomenon in the total renal flow trace (Figs. 3 and 4–1).

Another circumstance under which the cortical and medullary flows showed a different behaviour was the response elicited by the intra-arterial injection of acetylcholine in such doses (2.5 µg/g) that the total renal blood flow and the cortical flow decreased but the medullary flow increased (Fig. 5). McGIFF et al. (1967) have shown that the acetylcholine in certain doses produces constriction of the renal blood vessels by the release of catecholamines in the sympathetic nerve terminals of the kidney. Our results are in agreement with this interpretation since the use of an α adrenergic blocking agent (MA 1277) prevented the vasoconstrictor effect of acetylcho-
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line. Concerning the initial increase in the medullary flow produced by acetylcholine injections, it could be mentioned that the majority of the kidney nerves are innervating the cortex (MITCHELL, G.A.G., 1951). According to these findings the acetylcholine should change in a different magnitude the cortical and medullary resistance since the amount of sympathetic transmitter released would be also different. The fact that both small (0.5 μg/g) and large (2.5 μg/g) doses of acetylcholine produce the same effect under the α adrenergic blocking agent, indicates that acetylcholine could act also directly on the arterial muscles eliciting a vasodilation.

SUMMARY

The changes in the medullary and cortical blood flows of isolated dog kidney were measured by means of a light and small thermocouple inserted in each region.

When the perfusion pressure rose abruptly, the medulla responded increasing the flow independently of the initial perfusion pressure, while the cortical flow varied according to the initial perfusion pressure value. From 70 to 160 mm Hg of initial perfusion pressure, the cortex showed a double-humped response, that is, an initial increase in blood flow followed by a decrease that did not reach the control value and finally a late increase. The urinary flow increased concomitantly with the late increase in the cortical blood flow. When the initial perfusion pressure was under 70 mm Hg or over 160 mm Hg the cortex responded similarly to the medulla.

The α adrenergic blocking agent (MA 1277) did not change any of the responses above mentioned but potassium cyanide (4 mM/L in the perfusing blood) suppressed the decrease in blood flow shown by the cortex, in such a way that only an increase was observed. Under KCN action the urinary flow increased simultaneously with the increase in the perfusion pressure.

The intra-arterial injection of acetylcholine, in doses of 2.5 μg/g of kidney tissue, produced a decrease in the cortical blood flow while the medullary blood flow responded by an initial increase followed by a decrease. These responses were concomitant with a decrease in the total renal flow. When the MA 1277 was added (5 mM/L) to the perfusing blood the response to acetylcholine mentioned above, changed in such a manner that only increases in the cortical, medullary and total renal blood flows were observed. The different effects of large doses of acetylcholine on cortical and medullary resistance are discussed in terms of different amount of sympathetic transmitter released.

The present findings suggest that during an abrupt increase in the perfusion pressure the diminution phase of the double-humped cortical flow response was caused by an active contraction of the vascular smooth
muscles; while the increment in medullary flow could be due to the blood coming from another source different from the efferent blood vessels of the juxtamedullary glomeruli.

**APPENDIX**

The heated thermocouple (GIBBS, 1933) was based on the Seebeck effect. This effect relates the electromotive force developed in a circuit made up of different conducting elements, when not all of the contacts are at the same temperature. The main advantage of heated thermocouple is the possibility to measure local changes in blood flow. There are, however, some problems inherent to the thermocouples, namely the spatial distribution of the heat generated by the heated element. This point is crucial since the changes in flow were measured modifications in temperature of the object under investigation. We will consider some of the parameter involved in this problem.

The heat transfer between the heated filament and the surroundings is assumed to be proportional to the temperature gradient existing between the surface of the heated filament and the surroundings (Newton law of cooling). This transfer is characterized by a heat transfer coefficient $h$ expressed in cal/(cm²) (sec)(°C) in such a way that the normal component of the heat transferred in unit time across the vector element of area dS is $hT$. If $n$ is the outward drawn unit normal vector to the surface, the boundary condition at the surface may be expressed as $n \, \text{grad} \, T = \frac{h}{k} \, T$. The nature of the thermal contact is thus characterized by a parameter $\frac{k}{h}$ (k is the thermal conductivity of the heated filament and $h$ is the heat transfer coefficient). When $\frac{k}{h} \rightarrow 0$ the boundary is assumed to be maintained at a fixed temperature $T$. This assumption can be applied in our experiments because when the renal blood flow was steady the thermocouple output also was steady. On the other hand, as the heat production in the heated thermocouple was kept constant, under steady state condition, the corresponding temperature distribution in the cylinder formed by the tissue, that is, the element surrounding the thermocouple, may be expressed as $T = T_1 + (T_2 - T_1) \frac{\ln(r_2/r_1)}{\ln(r_2/r_1)}$, where $T$ is the corresponding temperature distribution, $T_1$ is the temperature of the heated filament of radius $r_1$, $T_2$ is the temperature at the limit considered by the radius $r_2$, and $r$ is the distance at any interval between $r_1$ and $r_2$. This formula of temperature distribution (INGARD and KRAUSHARR, 1961) assumes the homogeneity of the material and that the heat transfer coefficient in the heated filament-tissue system remained constant. In our
thermocouple-kidney system only the last condition is fulfilled at least for the control values. The heat transfer coefficient is a function of the liquid circulating around the thermojunction, therefore, the changes in the voltage output of the thermojunction reflect changes in the heat transfer coefficient of the kidney. The difference in the thermocouple output, due to the circulating blood was referred by Grayson (1951) as "conductivity increment". It seems to us a more appropriate term "changes in the heat transfer coefficient", since according to the above mentioned considerations we have shown that at the boundary of our thermocouple-tissue system the heat flowed to the tissue was characterized by a coefficient named heat transfer coefficient. On the other hand, Grayson (1952) also suggested that the difference in thermal conductivity, between living animal tissue and the same tissue after the death of the animal, was due to the circulating blood. This difference was called "conductivity increment" and quantitatively it was a linear function of the rate of flow. Furthermore, he attempted to correlate the total flow with the "conductivity increment" and calculate the factor that correlates the increase in flow with the rise in the "conductivity increment" according to the volume of the organ. This relationship was named "correlation factor" and multiplying this factor by the value of the "conductivity increment" he obtained the total blood flow. This was true for the spleen or the liver but he could not determine the correlation factor for the kidney. Grayson himself pointed out that this correlation factor is based on the assumption of homogeneity of flow through the whole organ but his assumption has not been confirmed in the kidney. The other problem is that the "conductivity increment" recorded by an electrode is only applied to a small volume of tissue and it may or may not be the representative of the whole organ. The heterogeneity of renal tissue does not permit the application of the temperature distribution formula. This is a serious inconvenience when a quantitative evaluation is required. For this reason we have estimated the local changes in flow as a percentage of the base values.

In order to calibrate the thermocouples and to evaluate the distance at which the heat of the filament spreads we have measured it in an agar block (4%) by means of another recording thermocouple calibrated with ice water as a reference. The real temperature of the heated filament was held at 1°C above the surrounding agar and in this condition we did not record any temperature change at a distance of 1.0 mm. It means that the thermocouples used were sensitive only in a radius less than 1.0 mm. The accurate determination of these limits in the living tissue is difficult to accomplish but one could assume or extrapolate that the tissue could behave in a manner similar to the agar. This extrapolation would be based on Grayson's reports. For the gelatin in 10%, Grayson (1952) reported a drop of 80% of the temperature gradient (1°C) at 1.5 mm distance from the heated filament, while
at 3 mm the temperature increment was negligible. GRAYSON determined the thermal conductivity in the rat liver, when the thermocouples were surrounded by a ring of 1.5 to 3.0 mm of the tissue. In these conditions he obtained similar values to those just mentioned for the gelatin and the liver, and concluded that the practical limits are less than 3 mm for the gelatin in 10%. In our test in agar (4%) the radius was smaller than that of GRAYSON's. According to the general equation of heat conduction, if the radius is greater, the heat loss will be greater, too. It seems reasonable, therefore, that the electrode used by us records only the temperature changes of the tissues within a radius of 1.0 mm. Since the thickness of cortex of the dog kidney weighing 60 g is about 8 mm, one can conclude that the changes recorded in the present experiments, correspond to the cortical and medullary tissues, respectively, in which the thermocouples were located. In the cases in which records from the medulla and the cortex were practically similar, it was found by histological examination of the kidney that the thermocouple inserted into the medulla was near a vessel irrigating the cortex.

It is known that the total mass of the electrode placed in the kidney plays a key role in the sensitivity and in the time taken to establish an equilibrium, since the total caloric energy necessary to heat-up the heater elements is a function of the heater filament mass. For this reason we have considered the use of light and small electrodes (2 to 2.5 mg/in weight) to be indispensable.

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