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Renal effect of dopexamine hydrochloride in patients with chronic renal dysfunction

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Summary. Dopexamine hydrochloride, a dopamine analogue, has been reported, both experimentally and clinically, to increase renal blood flow (RBF) and improve renal function in normal kidneys. The availability of computer-enhanced radionuclide scintigraphy, which provides accurate non-invasive measurement of changes in RBF, enabled us to study the renographic effects of dopexamine hydrochloride in patients with chronic renal dysfunction (CRD). Ten patients suffering from CRD and ten normal kidney donors were the study population. Renography was performed, heart rate (HR) and blood pressure (BP) measured, and hematological and biochemical tests carried out before and after intravenous infusion of dopexamine 2 μg kg^{-1} min^{-1} for 60 min. The patient population displayed signficant increases in total cortical and medullary RBF and renographic clearance rate (CR), while in kidney donors the RBF was increased in all kidney regions with no change in CR. HR increased in both groups, while BP showed no significant changes. The hematological and biochemical changes were transient and returned to preinfusion levels after 24 h. It is concluded that dopexamine hydrochloride 2 μg kg^{-1} min^{-1} increases RBF and CR in patients with CRD.

Key words: Inotropes: dopexamine – Kidney: failure – blood flow – Clearance rate

Dopexamine hydrochloride is a dopamine analogue which increases renal blood flow and improves renal function. This has been reported experimentally in conscious [7] and pentobarbitone-anesthetized dogs [3, 6, 7]. In clinical studies conducted in volunteers [12], and in patients with mild to moderate hypertension [11] or low-output congestive heart failure [4, 10], and in the critically ill [5], increases in renal blood flow and improvement in kidney function have been reported during intravenous infusion of dopexamine hydrochloride. These studies were reported in subjects who did not have preexisting chronic renal disease.

Radionuclide methods utilizing a bolus injection of a suitable tracer, coupled with the development of computer-enhanced techniques, provide an accurate measurement of changes in renal blood flow [2, 8, 15]. This noninvasive technique enabled us to study the renal effects of dopexamine hydrochloride in chronic renal insufficiency. This was carried out by open comparative design investigating the renographic data before and after intravenous infusion of dopexamine hydrochloride 2 μg kg^{-1} min^{-1} for 60 min in patients with chronic renal dysfunction (CRD) compared with those obtained from normal kidney donors.

Materials and methods

Study population

The study group contained members of both sexes and included ten patients and ten kidney donors (Table 1). They were consecutive subjects from the population admitted for routine renographic investigations. Kidney donors were due to donate one of their kidneys to a relative, and patients were suffering from chronic renal dysfunction in need of surgical intervention (Table 2).

Patient/donor entry criteria

All subjects included in the study were aged 18–60 years with systolic blood pressure between 80 and 160 mmHg and serum potassium concentration above 3.5 mM l^{-1}. Kidney donors were not suffering from any clinically known disease, or taking regular medication. Their hematological and biochemical parameters were within the normal ranges. Patients had to demonstrate at least an increase in serum creatinine (> 1.5 mg dl^{-1}) and a decrease in creatinine and nucleotide clearance rates (< 60 ml min^{-1}). Entry into the study was precluded by pregnancy or possibility of pregnancy, abnormal cardiac function or rhythm, unstable diabetes mellitus, and taking

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Table 1. Demographic and basal biochemical variables of the study population. Values are mean (range) or mean ± SD

	Patients	Kidney donors
Number of subjects	10	10
Age (years)	39.3 (23–59)	32.7 (21–55)
Sex (M/F)	8/2	4/6
Weight (kg)	68.9 (46–97)	72.2 (56–96)
Height (cm)	169.2 (160–180)	165.3 (158–178)
Serum creatinine (mg dl^{-1})	2.60 ± 0.776*	0.84 ± 0.178
Serum sodium (mM l^{-1})	141.6 ± 1.32*	144.2 ± 2.02
Serum potassium (mM l^{-1})	3.96 ± 0.328	3.92 ± 0.370
Creatinine clearance (ml min^{-1})	37.9 ± 15.80*	106.6 ± 20.95
Radioisotope clearance (ml min^{-1})	27.7 ± 10.33*	116.0 ± 19.57

* Significant difference, $P < 0.05$

Table 2. Urological pathological findings in the patient group

Patient no.	Pathological diagnosis
1	Bilateral staghorn calculi, hypertension.
2	Bilateral calculi, hypertension.
3	Bilateral calculi.
4	Bilateral hydronephrosis, urethral diverticulum.
5	Bilateral hydronephrosis, renal calculi, cancer of the bladder.
6	Urethral stricture.
7	Bilateral renal calculi.
8	Proteinuria and hematuria, hypertension.
9	Ureteric isolated ileal diversion (left kidney), pelvic aliceal drainage catheter (right kidney).
10	Bilateral staghorn calculi, hypertension.

monoamine oxidase inhibitors in the previous 2 weeks. All participants gave written informed consent, and the study protocol was approved by the responsible authorities.

Trial drug

Dopexamine hydrochloride was supplied as 1% solution in 5-ml ampoules with disodium edetate 0.01% and pH adjusted to 2.6. The contents of one ampoule was diluted with glucose 5% to make 50 ml, with a dopexamine hydrochloride concentration of 0.2 mg ml^{-1}. The drug was infused via a large peripheral vein by infusion pump at a rate of 2 μg kg^{-1} min^{-1}.

Radionuclide study

The radionuclide study was performed by intravenous bolus injection of 10 mCi (370 MBq) technetium-99m-diethylene triamine pentaacetic acid (^{99m}Tc-DTPA), with the patient or kidney donor in a prone position. A gamma camera (Picker International) with 61 photomultiplier tubes, and fitted with a low-energy, parallel-hole, highly sensitive collimator, was used. A flow study of 60 1-s images followed by 57 20-s images for 19 min was taken to complete the scan.

Dynamic acquisition of analogue images on X-ray film and digitalization of data on the magnetic disk of a computer system enabled visual interpretation of the analogue images which correlated with the computer-generated data.

Images obtained during the 1st min were used, through a frame addition program, to generate the blood flow curves. The latter were used by another computer program for calculation of the perfusion index (PI). For each kidney, the global PI was obtained by selection of the whole outline of the kidney and the related area of the aorta. Areas of interest were drawn by the use of computer light pen. The renal cortical PI was calculated by flagging the cortex, comprising 25% of the transverse diameter of the kidney, usually 2–3 pixels in width. Areas under the normalized aortic and renal curves up to the time of the aortic peak caused by the first passage of the radionuclide bolus were used to calculate the PI. As relative blood flow through the kidney falls, the area under the vascular phase of the renal curve becomes smaller, increasing the index. Conversely, as the renal blood flow improves, the PI falls.

At the end of the scintigram, a heparinized 3-ml venous blood sample was taken, centrifuged, and 1 ml plasma was counted on a scale-rate meter for 60 s. This plasma count together with the net injected dose count and patient's body weight and height were computed to get the isotope clearance rate (CR), total and for each kidney [13].

Monitoring

Heart rate and ECG were monitored continuously. Systolic, diastolic, and mean blood pressure were measured every 15 min.

Laboratory investigations

Hematological and biochemical variables were measured before and after dopexamine hydrochloride infusion. The hematological variables included: red blood cell count (RBC), total and differential white blood cell counts (WBC), platelet count, and estimations of hemoglobin (Hb) and hematocrit (Hct). The blood cell counts and Hb and Hct calculations were carried out by a Coulter Counter Model S770 (Coulter Coultronics, France) using Isoton II and lyse S as diluter counting. Differential WBC were done manually using Giemsa and Leishman staining. Platelets were counted manually by counting chambers (Hemocytometer) using ammonium oxalate 1% as a reagent. The biochemical variables included: blood glucose, serum creatinine (L508 Electrolyte and Chemistry Analyzer; Instrumental Laboratory, USA), total plasma protein, (proteins kit, ref. 61602, Bio Merieux, France), serum albumin (albumin kit, ref. 61051, Bio Merieux), serum sodium, serum and urine potassium (Nova I Sodium/Potassium Analyzer, Nova Biochemical, USA), ionized and total calcium (calcium kit, ref. 11001, Bio Analytics, USA), phosphorus (phosphorus kit, ref. 61599, Bio Merieux), direct and total bilirubin (bilirubin kit, ref. 123927, Boehringer, Germany), and transaminases (Bio Analytics kits, refs. 23001 and 26001 for SGOT and SGPT respectively).

Study protocol

The day before dopexamine hydrochloride infusion, all patients and kidney donors underwent a medical examination including ECG. Venous blood samples were withdrawn for hematological and biochemical screening and a baseline renogram was obtained.

Table 3. Renal perfusion indices[a] calculated for 10 patients and 10 kidney donors before and during infusion of dopexamine 2 μg kg^{-1} min^{-1}. Values are mean ± SD

		L kidney	R kidney	Total
Patients				
Globe:	basal	41.6 ± 29.8	82.4 ± 55.9	62.0 ± 24.5
	dopexamine	35.3 ± 18.4	70.4 ± 40.8	52.8 ± 16.6
Cortex:	basal	57.3 + 46.2	88.3 ± 42.0	72.8 ± 22.1
	dopexamine	46.2 ± 30.0	*79.5 ± 35.0	*62.9 ± 14.4
Medulla:	basal	105.5 ± 94.2	170.8 ± 77.0	138.2 ± 36.5
	dopexamine	88.7 ± 66.5	*148.2 + 73.4	*118.5 + 30.4
Kidney donors				
Globe:	basal	31.3 ± 6.2	28.6 ± 5.0	30.0 ± 5.4
	dopexamine	*26.7 ± 8.4	*23.6 ± 3.7	*25.2 ± 4.9
Cortex:	basal	40.7 ± 10.6	39.4 ± 8.1	40.0 ± 9.0
	dopexamine	*31.7 ± 5.1	*32.1 ± 3.7	*31.9 ± 4.2
Medulla:	basal	68.2 ± 14.1	60.1 ± 11.5	64.1 ± 11.6
	dopexamine	*53.9 ± 9.2	*50.6 ± 9.4	*52.3 ± 8.4

* Significant change from basal value, $P < 0.05$

[a] Perfusion index

$$= \frac{\text{area under the aortic curve, integrated to peak}}{\text{area under the renal curve}} \times 100$$

On the day of the dopexamine hydrochloride infusion, patients and kidney donors had ECG electrodes and a BP cuff attached, and were allowed to rest for 15 min before control readings of HR and BP were taken. A cannula was inserted into a forearm vein on both arms, and a blood sample was taken for glucose estimation. Infusion of dopexamine 2 μg kg^{-1} min^{-1} for 1 h was performed at approximately the same time the following day. At 40 min into the infusion a renogram was obtained simultaneously by injection of the isotope through the other arm.

After completion of the dopexamine hydrochloride infusion, venous blood samples were taken for assessment of hematological and biochemical variables, and this was repeated 24 h after dopexamine hydrochloride infusion. Another blood sample was taken at the end of the scintigram for nucleotide CR calculations.

Statistical analysis

Parametric statistical methods were used. Comparison of data for each group was made using a two-way analysis of variance. Where an overall significant difference between two time points was seen, pairwise comparisons of mean values was carried out using the least significant difference method. Comparison between the two groups was done by means of unpaired *t* tests. All statistical tests were two-tailed and carried out at the 5% significance level. Statistical analyses were carried out using BMDP and a Fortran program written to perform Mann-Whitney U tests.

Results

Renal blood flow

During the administration of dopexamine hydrochloride, the cortical and medullary PI calculated for both kidneys of the patient population showed significant decreases from basal values, denoting a significant increase in renal perfusion (Table 3). When calculated for each kidney, the PI showed a significant decrease in the right kidney, while the left kidney was approaching the significance level ($P = 0.08$). There was no significant change in global PI, although a decrease was observed.

The kidney donors displayed significant decreases in PI for both kidneys and in the calculated regions, denoting significant increases in renal blood flow to all regions of the kidney during intravenous infusion of dopexamine hydrochloride.

Table 4. Renographic clearance rate in 10 patients with chronic renal dysfunction and 10 normal relative kidney donors before and after infusion of dopexamine 2 μg kg^{-1} min^{-1} for 60 min. Values are mean ± SD

	L kidney	R kidney	Total
Patients			
Basal	18.5 ± 9.9	9.2 ± 7.5	27.2 + 10.3
Dopexamine	22.3 + 10.8	12.0 + 11.1	34.3 + 11.4
t Value	3.41	2.19	4.34
P	0.008	0.063	0.002
Kidney donors			
Basal	51.9 ± 9.5	64.1 ± 11.5	116.0 ± 19.6
Dopexamine	51.0 ± 10.7	61.3 ± 10.8	112.3 ± 20.2
t Value	0.41	0.76	0.65
P	0.681	0.469	0.534

Clearance rate

The radionuclide CR calculated for the patient group displayed a significant increase in the left kidney, while the increase in the right kidney was close to significance ($P = 0.064$; Table 4). However, total CR was significantly increased. The change in CR for the kidney donor group was insignificant.

Hemodynamic changes

Intravenous infusion of dopexamine hydrochloride in patients with chronic renal dysfunction and kidney donors demonstrated significant increases in HR during the period of infusion. These decreased to preinfusion levels 15 min after cessation of the infusion (Table 5). There were no significant changes in BP measurements in patients and kidney donors during dopexamine infusion (Table 5).

The increase in HR was paroxysmal in one patient and one kidney donor during the first 15 min of dopexamine hydrochloride infusion. Atrial premature beats occurred in one donor and two patients with chronic renal failure. These were transitory and occurred at a rate of less than 4 min^{-1}. Transitory premature ventricular contractions occurred in one kidney donor and two patients, at a rate of less than 3 min^{-1}.

Table 5. Changes in heart rate (beats/min) and blood pressure (mmHg) during and after dopexamine infusion 2 μg kg^{-1} min^{-1} in 10 patients with chronic renal dysfunction, and 10 normal kidney donors. Values are mean ± SD

		Zero time	During infusion (min)				After infusion (min)
			15	30	45	60	15
Patients							
Heart rate		78 ± 14	*93 ± 10	*95 ± 18	*102 ± 17	*103 ± 14	88 ± 11
Blood pressure:	systolic	130 ± 12	134 ± 8	140 ± 14	136 ± 14	137 ± 10	131 ± 10
	diastolic	81 ± 8	72 ± 7	77 ± 7	74 ± 13	73 ± 14	79 ± 12
	mean	93 ± 10	84 ± 6	92 ± 11	86 ± 11	87 ± 9	91 ± 12
Kidney donors							
Heart rate		73 + 9	*88 + 11	*95 + 12	*101 + 10	*103 + 11	84 ± 12
Blood pressure:	systolic	126 ± 14	128 ± 11	129 ± 6	129 ± 10	130 ± 10	125 ± 9
	diastolic	72 ± 9	67 ± 11	70 ± 9	70 ± 11	69 ± 13	76 ± 7
	mean	85 ± 8	79 ± 9	82 ± 7	82 ± 8	82 ± 12	86 ± 7

* Significant change from zero time values, $P < 0.05$

Table 6. Mean hematological, serum electrolyte, blood glucose, and urine potassium variables before and after dopexamine hydrochloride infusion (2 μg kg^{-1} min^{-1}) for 60 min in 10 patients and 10 kidney donors. Least significant difference (LSD) is stated when a significant difference ($P < 0.05$) was found

	Hematological variables			Serum electrolytes (mM l^{-1})			Urine potassium (mM l^{-1})	Blood glucose (Random)
	RBC ($\times 10^{12}$ l^{-1})	Hb (g dl^{-1})	Platelets ($\times 10^{9}$ l^{-1})	Sodium	Potassium	Phosphorus		
Patients								
Before infusion	4.77	11.8	222.3	141.6	3.96	4.13	14.3	115.7
After infusion	4.58	11.3	183.4	141.4	3.67	3.39	11.2	119.6
24 h after infusion	4.80	11.8	248.3	143.4	3.89	3.91	12.2	95.1
S deviation (df)	0.14 (18)	0.34 (18)	42.29 (18)	1.55 (18)	0.35 (18)	0.63 (18)	4.86 (18)	27.67 (12)
LSD	0.134	0.32	39.74	1.45		0.588		
Kidney donors								
Before infusion	4.76	12.6	187.6	144.2	3.92	3.62	26.5	104.6
After infusion	4.36	11.9	216.9	143.3	3.39	3.05	21.9	129.2
24 h after infusion	4.64	12.2	227.2	143.8	3.73	3.82	26.0	109.9
S deviation (df)	0.31 (18)	0.55 (18)	51.32 (18)	1.15 (18)	0.30 (18)	0.46 (18)	12.14 (14)	29.25 (16)
LSD	0.289	0.51			0.278	0.436		

RBC, Red blood cells; Hb, hemoglobin

Hematological changes

Dopexamine hydrochloride infusion, in patients and kidney donors, resulted in significant decreases in RBC and Hb (Table 6). These returned to preinfusion levels after 24 h. Other hematologic variables did not display significant changes.

Biochemical variables

The infusion of dopexamine hydrochloride in the patient group was associated with a decrease in serum phosphorus, while kidney donors demonstrated decreases in serum potassium and phosphorus (Table 6). These variables returned to preinfusion values after 24 h. However, the serum sodium in the patient group showed a significant increase 24 h after dopexamine infusion. There were no significant changes in random blood glucose determinations after dopexamine infusion in both patients and kidney donors.

Discussion

Computer-enhanced radionuclide scintigraphy provides accurate noninvasive measurements of changes in renal blood flow. The use of ^{99m}Tc-DTPA, a chelate cleared by

glomerular filtration, improves this technique. This tracer has a short half-life and a high photon flux which gives good external counts after bolus injection [2].

The development of computer-enhanced facilities enables the scintigraphic technique to measure changes in renal blood flow at short time intervals. The use of PI calculation, and its application to the regions of the kidney, has enabled investigators [2, 8, 15] to compare changes in renal blood flow against changes in aortic perfusion, and to detect early alterations in renal cortical blood flow, a sensitive monitor for early renal vascular changes [2, 15].

Dopexamine is an afterload-reducing agent with agonist activity at DA_1 and DA_2 dopaminergic receptors and β_2-adrenoceptors [6]. The intravenous infusion of dopexamine in kidney donors resulted in an increase in renal blood flow. This renal vasodilating property was not unexpected, and has been demonstrated experimentally in pentobarbitone-anesthetized dogs through intrarenal artery administration [3, 6], and by intravenous infusion [7]. Clinical studies attributing a renal vasodilating property to dopexamine hydrochloride have been conducted in healthy volunteers [12] and in patients with mild to moderate hypertension [11].

However, the infusion of dopexamine hydrochloride in the present patient population also resulted in a significant increase in renal perfusion. Although this was just near the significant level for the left kidney, the total perfusion for both kidneys was significantly increased. More importantly, there was an increase in renal cortical blood flow, a sensitive indicator of early changes in renal blood flow [2]. This was associated with an increase in renographic CR, indicating improvement in their compromised kidney function. Improvements in renal function with dopexamine hydrochloride have previously been reported [4, 5, 10]. Patients in the present study were suffering from chronic longstanding irreversible renal damage, and that dopexamine hydrochloride produced a renal vasolidating effect and an increase in renographic CR in these patients is interesting, since dopamine was reported to have no effect on renal function in patients with varying degrees of renal insufficiency [17].

The infusion of dopexamine hydrochloride in patients and kidney donors resulted in a significant increase in HR. This has been reported in experimental [3, 6, 7] and clinical studies [4, 9, 11], and is attributable to its agonistic activity at β_2-adrenergic receptors [6, 7]. The effect of dopexamine hydrochloride infusion on systolic BP is variable, demonstrating no significant change in patients with chronic renal insufficiency in the present study or in chronic congestive heart failure [4], although an increase was obtained in hypertensive patients [11] and in cardiac surgery patients following cardiopulmonary bypass [9]. The effect of dopexamine hydrochloride on diastolic BP is similarly variable: diastolic BP was reduced in patients with congestive heart failure [4], but did not show significant changes in patients with mild to moderate hypertension [11] or in the present study in patients with chronic renal dysfunction. The circulatory effects of dopexamine hydrochloride are mediated through several receptors, namely postjunctional dopaminergic (DA_1), prejunctional dopaminergic (DA_2), and β_2-adrenergic receptors and also through potent inhibition of uptake-1 [16]. Due to the effect of dopexamine on neuronal uptake of norepinephrine, it is possible that the effects of dopexamine infusion on the BP are modified by the pathophysiological setting of the patient.

The infusion of dopexamine hydrochloride in kidney donors resulted in a decrease in serum potassium level, associated with no significant changes in random blood glucose and urine potassium concentrations. This hypokalemic effect can possibly be attributed to β-adrenergic activity, which causes a decreased in serum potassium concentration by driving potassium into the cell [14]. The infusion of dopexamine in the patient group did not significantly change serum potassium levels. It seems that potassium movement across the cell membrane is limited in chronic renal failure. The concentration of serum phosphorus decreased in both patients and kidney donors following the infusion of dopexamine. An explanation of this finding could not be traced in the available literature, and it is presumed that phosphorus was driven by dopexamine into the cells.

In conclusion, the infusion of dopexamine hydrochloride $2\,\mu g\ kg^{-1}\ min^{-1}$ in patients with chronic renal insufficiency resulted in increases in renal blood flow and improvement in renographic renal clearance.

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