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ABNORMAL RENAL FUNCTION IN HYPERTENSIVE PREGNANT WOMEN: REVERSAL BY LEFT LATERAL DECUBITUS

M Labeeuw, N Pozet, A Hadj Aissa, M Vincent, P Zech

INSERM U 80, E Herriot Hospital, Lyon, France

Summary

The influence of the left lateral position on renal function was assessed in non-pregnant normotensive and hypertensive women. In a supine position, the expected changes in renal function were observed in pregnant compared to non-pregnant normotensive women, but the influence of assuming a lateral position was comparable in the two groups. In hypertensive pregnant women, the abnormal renal function observed in a supine position was reversed in the left lateral position.

Subjects and methods

Twenty-four pregnant women, seven hypertensive patients (blood pressure above the 90th percentile of normal distribution for the same gestational age according to Friedman [1]) and 17 normotensive patients were studied. There were no differences between the two groups for age (27.5±5.1 versus 29.7± 5.5), gestational age (19.8±5.7 versus 19.4±4.8 weeks) and parity (1.72 versus 1.76). None had a previous history of hypertension or had proteinuria at the time of the study. Seven non-pregnant women (36.3±6.3 years, previous pregnancies: 1.14) served as controls. All subjects were studied for two consecutive 60-minute periods: period I in the supine position, period II in the lateral decubitus position. Controls were studied a second time for two successive periods in the supine position. Systemic haemodynamics, creatinine and uric acid clearances, urinary flow rate, sodium and potassium excretions, plasma electrolytes, aldosterone and renin activity were determined for each period.

Results

Period I The detailed results are summarized in Table I, which shows the expected findings in normotensive patients: tachycardia, low serum creatinine, uric acid,

TABLE I. Data in period I

	C	NP	HP	HPvsNP
Blood pressure (mmHg)	109±7/62±8	118±8/66±6	135±8/79±5[b]	0.025
Heart rate/min	67±9	84±10[a]	97±14[b]	0.05
Blood				
Creatinine (μmol/L)	76±7	60±8[a]	62±9[a]	NS
Uric acid (mmol/L)	238±26	216±45[a]	209±30[a]	–
Sodium (mmol/L)	140±0.8	139±1.6[a]	138±1.5[b]	–
Potassium (mmol/L)	4.4±0.3	4.1±0.3[a]	4.0±0.7[a]	–
Osmolality (mOsm/L)	289±4	285±5	281±6[b]	–
Plasma renin activity (ng/L/min)	42.6±32.9	246±88[b]	231±126[b]	–
Aldosterone (pmol/L)	296±130	638±432[b]	673±301[b]	–
Renal function				
Urinary flow rate (ml/s)	0.036±0.024	0.028±0.020	0.024±0.020	–
Creatinine clearance (ml/s)	2.07±0.34	2.78±0.72[b]	2.04±0.29	0.025
Uric acid clearance (ml/s)	0.23±0.05	0.31±0.09[a]	0.24±0.10	0.10
Na excretion (μmol/s)	2.04±0.91	2.80±1.42	2.15±1.19	NS
Na fractional excretion (%)	0.72±0.34	0.73±0.33	0.73±0.33	–
K excretion (μmol/s)	1.68±0.36	1.23±0.52[a]	0.82±0.21[a]	0.025
K fractional excretion (%)	18.4±2.9	10.7±3.4[a]	10.3±2.3[a]	NS

Pregnant versus controls: a=$p<0.05$; b=$p<0.01$
C=control; NP=normotensive; HP=hypertensive

sodium, potassium and osmolality, high renin activity and aldosterone. Hypertensive patients differ from normotensive patients by higher systolic and diastolic blood pressure, heart rate, lower creatinine and uric acid clearances, sodium and potassium excretions, but identical fractional excretion of sodium, potassium and uric acid.

TABLE II. Data in period II

	C	NP	HP	HPvsNP
Blood pressure (mmHg)	100±12/53±7**	110±11/58±11**	117±9/65±8**b	0.025
Heart rate/min	69±10	84±10a	91±11*a	NS
Plasma renin activity (ng/L/min)	31.3±24.2	220±100b	227±145b	–
Aldosterone (pmol/L)	166±80**	408±268*a	385±139**b	–
Renal function				
Urinary flow rate (ml/s)	0.027±0.013	0.027±0.021	0.022±0.011	–
Creatinine clearance (ml/s)	1.66±0.19*	2.40±57**b	2.34±0.39*b	–
Uric acid clearance (ml/s)	0.21±0.04*	0.28±0.09a	0.30±0.09*a	–
Na excretion (μmol/s)	2.32±1.1*	3.15±1.64*	3.31±1.34*	–
Na fractional excretion (%)	0.99±0.43**	0.94±0.34**	1.03±0.45**	–
K excretion (μmol/s)	1.36±0.30	1.14±0.48	1.20±0.30	–
K fractional excretion (%)	18.5±3.3	11.4±3.6	13.3±3.6**	–
Relative changes %				
Heart rate	+2.6±7.7	−0.4±6.1	−5.6±5.2a	0.01
Creatinine clearance	−18.1±14.2	−12.3±13.2	+18.6±36.1a	0.05
Uric acid clearance	−9.1±8.6	−7.1±19.6	+35.6±38.3a	0.01
Na excretion	+22.4±32.6	+15.3±32.7	+59.8±63.8a	0.05
K excretion	−14.5±26.9	−3.9±24.2	+46.8±34.6a	0.01

Pregnant versus controls: a=$p<0.05$; b=$p<0.01$. Period II versus period I: *=$p<0.05$; **=$p<0.01$. C=control; NP=normotensive; HP=hypertensive

Period II Values of most parameters are given in Table II, together with their percentage relative changes from period I. No changes in urinary flow rate and renin activity occurred in any cases.

In period II, for controls, changes were similar whatever the position, supine or lateral decubitus, for all parameters except blood pressure which did not decrease when the second hour was completed supine. In lateral decubitus, relative changes from period I were identical in normotensive patients and controls: significant decreases in blood pressure, aldosterone and creatinine, significant increases in sodium excretion and sodium fractional excretion; there were no variations in other parameters. Hypertensive patients exhibited a markedly different pattern: blood pressure tended to decrease more but remained higher than in normotensive patients. Only two of seven hypertensive patients showed a decrease in mean arterial pressure greater than 20mmHg. For all subjects, the relative changes in sodium fractional excretion correlated with the d% of aldosterone (d% FeNa=17.61 - 0.54 d% A, r=0.51, $p<0.01$).

Discussion

This study documents the changes in systemic haemodynamics and renal function after assumption of the left lateral decubitus position during the second trimester of normotensive and hypertensive pregnancy. Lateral decubitus position induced a significant decrease in blood pressure in all cases, even in non-pregnant controls, but tachycardia was moderately reduced only in hypertensive patients (Table II). The pitfalls in clearance measurements in pregnancy have been reviewed [2]. Urinary flow rates were similar in normotensive patients and hypertensive patients and all parameters normalized in period II without increases in urinary flow rate. Therefore there is no evidence that dilatation of the urinary tract or incomplete bladder emptying were a significant problem.

Normotensive patients showed in the supine position the well known changes in blood parameters and renal function [2,3]. The low potassium excretion and potassium fractional excretion despite a marked hyperaldosteronism, presumably reflect the positive balance for potassium and the influence of progesterone [2]. In controls, assumption of the lateral decubitus position led to similar variations whether the period II was completed in the supine or in the lateral decubitus position, suggesting that they depend on time rather than on position. Similar findings (using inulin clearance as a measure of GFR) were reported by Dunlop [4]. This indicates a need for careful re-evaluation of previous reports on the influence of posture on renal function in pregnancy [2,4].

Compared to normotensive patients, hypertensive patients exhibited several abnormalities in period I: low creatinine clearance, uric acid clearance, sodium excretion and potassium excretion, which were abolished in period II. These data indicate that low GFR (and probably low renal plasma flow) [5] in hypertensive pregnancy can be reversed. The respective influence of the lateral position and time in this improvement remain to be further clarified. However our data might explain discrepancies on the influence of position, depending on whether pre-eclamptic women are included or not [4,5]. Interestingly, in a study of

essential hypertension, we found that the influence of position on GFR and sodium was exaggerated [6], compared to normotensive controls: this suggests that the sensitivity of renal vasculature might be increased in both essential hypertension and hypertension of pregnancy. The decreasing values of aldosterone, while plasma renin activity did not vary are likely to be a consequence of the different kinetics of these parameters [7]. Accordingly, the changes in aldosterone could only be a delayed consequence of the suppression of the stimulation induced by orthostatism in the pre-study period. Changes in aldosterone correlated with changes in sodium fractional excretion but did not explain the divergent changes in potassium excretion; these variations should be interpreted in view of the multiple influences on renal handling of potassium [8].

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