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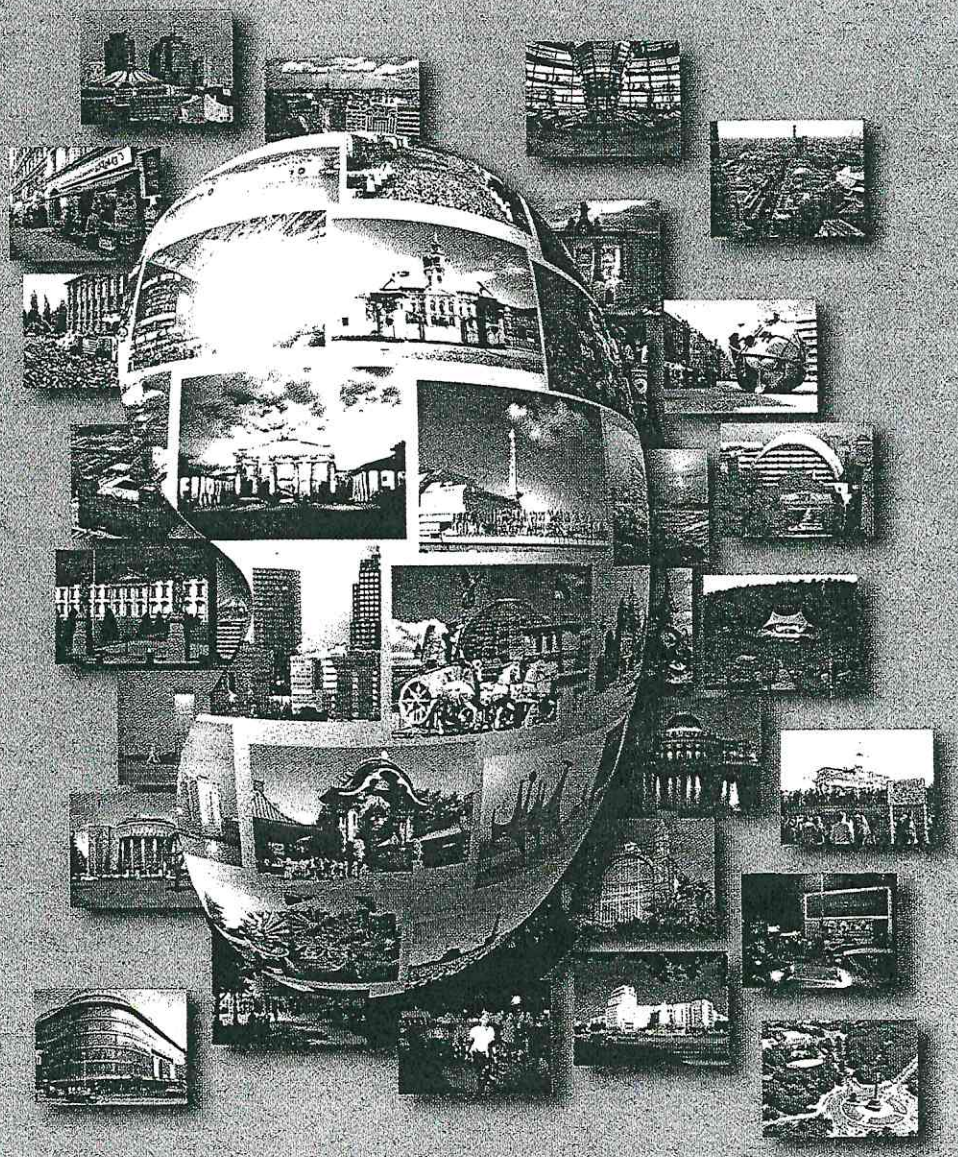

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The following data (Mean ± SD) were received (Table 1).

Renal haemodynamic response to acute captopril test

		Vs	Vd	Vmean	PI	RI
RA	before C	97.5±23.5	36.7±8.7	52.0±12.9	1.16±0.19	0.64±0.05
	after C	115.5±39.1**	42.8±3.7**	62.9±18.3**	1.13±0.29	0.63±0.05
SA	before C	64.2±15.8	27.8±7.7	40.3±9.2	0.94±0.14	0.58±0.05
	after C	74.8±24.8**	32.6±11.4**	46.8±15.3**	0.93±0.19	0.50±0.06
IA	before C	38.1±9.9	17.1±4.7	24.4±6.3	0.90±0.16	0.56±0.06
	after C	44.6±13.5**	21.1±6.9**	28.4±9.1**	1.18±1.6	0.56±0.07
AA	before C	29.7±8.1	15.2±4.2	20.2±5.3	0.75±0.18	0.50±0.09
	after C	33.9±7.7**	16.7±3.4*	22.1±4.7*	0.84±0.13	0.53±0.05

* - P<0.05; ** - P<0.01 before/after C intake by Wilcoxon test

In total the increase of blood flow velocities without changes of PI and RI in all investigated arteries was observed after C intake. Significant blood flow velocities rise was found both in pts with normal and deteriorated renal function. Blood flow velocities increase correlated with young age (P<0.01) and systolic blood pressure reduction (P<0.05). 5 pts which yilded no blood velocities increase in C test had significantly older age (in 4 it was over 52) but they did not differ from the other pts by renal function. We conclude that renin-angiotensin system blocade by C improved renal haemodynamics in GN including pts with mild CRF. The older age was associated with risk of poor haemodynamic response to C.

M252 HIGH DOSAGES OF ARA 1 AND ACE INHIBITORS IN RENAL PROTECTION

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Angiotenzin have much higher kidney tissue concentrations than in blood. We assumed that retardation of kidney function loss need more higher blocking of this substrate. The aim of the study was to estimate high dosages of ARA 1 and ACE inhibitors in renoprotection. During 5 years follow-up we conducted the randomized prospective multi-centre study of usage combined therapy with ARA 1 and ACE inhibitors in 126 pts aged 4-62 with glomerulonephritis (GN). Impaired renal function in the disease onset was proved in 23 pts. The dosage of losartan was up to 3 mg/kg or irbesartan up to 8 mg/kg or telmisartan up to 4 mg/kg in combination with enalapril up to 2 mg/kg or fosinopril up to 1 mg/kg or diltiazem up to 10 mg/kg. A criterion for dosage individualization was hypotension. 131 pts with GN on a traditional dose of ACE inhibitors and ARA 1 was enrolled in the study as control group. The data obtained demonstrated rapid effect in decrease BP. In 3-6 months period of combination therapy the reducing of proteinuria from 2,1±0,3 g/l to 0,3±0,2 g/l was noted. 4-5 years follow-up of ACE inhibitors + ARA 1 led to improvement renal function in 9 pts (39%) and its full normalization in 5 (21%) pts. No side effects were revealed except for reversible serum creatinine raise during starting the therapy. Comparing with the traditional usage of ACE inhibitors or ARA 1 showed less positive effect in proteinuria reduction (2,0±0,3 g/l to 0,9±0,1 g/l) and no significant improvement of renal function. We confirm 3 possible dosages of ARA 1 and ACE inhibitors. First (traditional doses) – hypotensive effect. Second (middle doses) – antiproteinuric effect, third – high doses – renoprotective effect with possible improving of impaired renal function. We also proved the safety of high doses ACE inhibitors and ARA 1 usage.

M253 CYCLOOXYGENASE (COX)-2 SELECTIVE INHIBITORS + ACE INHIBITORS: A NEW PROTOCOL IN THE TREATMENT OF HEAVY PROTEINURIA

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Non steroidal anti-inflammatory drugs (NSAIDs) are surely useful in the treatment of proteinuria, but these agents may cause deleterious effects on kidney function as maintenance of renal perfusion and glomerular filtration; moreover often they induce gastrointestinal toxicity. Recent studies have demonstrated that COX-2 is constutively expressed in renal tissues; this isoform is intimately involved in prostaglandin-dependent renal omeostatic processes. Drugs that selectively inhibit COX-2 might therefore be expected to produce effects on renal function similar to non selective NSAIDs.

Aim of our study was to observe the efficacy of a COX-2 selective inhibitor (rofecoxib) associated with an ACE inhibitor (ramipril) on proteinuria and on renal function of chronic glomerular diseases; we compared this group with another omogeneous pool of patients previously treated with ramipril and a NSAID (meclovanate) with good results on proteinuria, but 36.6% of side effects and 3 patients in drop-out.

Eighteen patients affected with primitive chronic glomerulonephritis were treated with rofecoxib (25 mg/day) + ramipril (10 mg/day) for six months. All of them were not responsive to single treatment with ramipril. We observed daily proteinuria, renal function as corrected creatinine clearance, mean arterial pressure (MAP), serum creatinine and electrolytes. During all the period of our observation no changes were in diuretic and immunosuppressive therapy.

Our results show a significative reduction of proteinuria from 4.9 to 1.1 gr/day (p<0.002); no modification in MAP, serum electrolytes. Creatinine clearance moved from 95.3 to 97.8 ml/min, with no significative statistical modification. No patient showed gastrointestinal side effects induced from drugs.

In conclusion, we propose this combined therapy (rofecoxib + ramipril) in the treatment of chronic proteinuria in patients resistant to single therapy (ramipril), without important side effects. We considerer this protocol a better and safer treatment in front of NSAID + ACE inhibitor.

M254 CYCLOSPORINE A (NEORAL) C2 MONITORING IN THE TREATMENT OF SEVERE NEPHROPATHIES: A PILOT STUDY

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Cyclosporine A (CyA) is used as an essential part of a pathogenic treatment for some immune glomerulopathies, with concentration controlled dosage for optimal therapeutic management. Neoral C_2 monitoring is superior to C_0 monitoring in renal and liver transplant patients. The aim of the present study was to compare the monitoring of C_2 against C_0 for the Neoral formulation of CyA in 10 patients with severe course of immune and autoimmune glomerulonephritis. They were also receiving corticosteroids and cyclophosphamide in conventional and pulse doses. Patients were followed for 3 to 12 months. Whole blood CyA levels were measured 5 minutes before and 2 hours post dose of Neoral at the steady state and drug levels were checked more than one, so 33 pairs of C_2 and C_0 concentrations were included in the study. Results as mean±SD (min-max) were as follows: Neoral daily dose 2.34±0.64 mg/kg (1.2 to 3 mg/kg); C_0 level 93.9±90.8 μg/L (25 to 230 μg/L); C_2 754.8±324.7 μg/L (362 to 1500 μg/L). Individual concentrations were rated as therapeutic, low or high, applying a target range of 75-150 μg/L and 600-1200 μg/L for C_0 and C_2 respectively. Among the 13 low C_0 concentrations observed, 8 were paired with optimal C_2 levels; 4 of the 17 "optimal" C_0 levels were paired with low C_2 concentrations; and 4 of the 11 low C_2 levels were paired with "optimal" C_0 values. There were not significant differences in serum creatinine, creatinine clearance, cholesterol, serum albumin and blood pressure at the beginning and at the end of the study. Proteinuria and episodes of hematuria were reduced significantly. No adverse effects were established during Neoral therapy. We conclude that C_2 monitoring of Neoral might have a place in the effort to optimise the therapy and avoid nephrotoxicity in patients with immune nephropathies.