a. Purpose of study. Among side effects of corticosteroid, much attention has not been paid to steroid cataracts (posterior subcapsular cataracts) in patients with renal diseases. In addition, there were few works concerning serial ophthalmologic observation. This study was undertaken to evaluate the incidence, advance and improvement of steroid cataracts in patients with renal diseases of childhood.

b. Materials and methods. Slit-lamp examinations were done by the same two ophthalmologists for 214 patients with renal diseases who were given steroid.

c. Summary of results. The incidence of steroid cataracts was 29.9 % in patients with renal diseases. According to Crews' classification, the incidence of steroid cataracts was 15.0 % in grade I, 6.5 % in grade II, 7.9 % in grade III, 0.5 % in grade IV in total 214 patients. While 20.8 % incidence was found in total 24 patients without renal diseases. The advance of steroid cataracts by Crews' classification was obtained in 16.3 % of total 98 patients who were followed up for 22 months on the average. No improvement of steroid cataracts was obtained in the observation period.

d. Conclusions. The incidence of steroid cataracts was very high especially in patients with nephrotic syndrome. The importance of periodic ophthalmologic examinations is emphasized in patients on long-term steroid therapy.

THE EFFECT OF INDOMETHACIN ON RENAL FUNCTION IN CYSTINOSIS. Haycock, G.B., Al-Dahhan, J. and Chantler, C. Guy's Hospital, London, England.

Prostaglandin synthetase inhibitors have been shown to enhance tubular sodium reabsorption in normal subjects and in patients with salt losing renal disorders such as Bartter's syndrome. We examined the effect of indomethacin (I) on renal function in 3 cystinotic children aged 6-8. After induction of maximal water diuresis, inulin clearance (GFR) and tubular handling of sodium (Na), potassium (K) and water were studied before and after saline volume expansion (VE). The studies were performed under control conditions and after 2 weeks treatment with I 2-3 mg/kg/day. Na and K supplements were not given during the study period. I caused GFR to fall from an average of 28.9 ml/min/1.73 m² to 19.9, the fall being abolished by VE. Absolute and fractional Na excretion were markedly reduced by I particularly after VE. The most striking change was in calculated fractional proximal tubular Na reabsorption after VE, which rose from 53.8% to 74.5% of the filtered load. Plasma Na rose from 128 to 132 mEq/l, plasma K from 2.57 to 3.17. Polyuria, polydipsia and salt craving were greatly reduced. An average weight gain of 7% was observed over the study period, presumably as a result of expansion of extracellular fluid volume. The primary effect of I appears to be facilitation of proximal tubular Na reabsorption, leading to reduced Na delivery to distal nephron segments and reduced distal Na-K exchange. I is a useful aid in the management of the tubular defect of cystinosis.

RENAL PROSTAGLANDIN E₂ IN NEPHROGENIC DIABETES INSIPIDUS : EFFECTS OF INHIBITION OF PROSTAGLANDIN SYNTHESIS BY INDOMETHACIN. Usberti M., Dechaux M., Pavlovitch H. Sachs C., Broeyer M.- Hôpital des Enfants Malades, Paris, France.

Urinary prostaglandin E₂ (PGE₂) and cyclic AMP (AMPC) were determined in two children with nephrogenic diabetes insipidus (NDI) under basal conditions, after intranasal DDAVP and after 3-day treatment with indomethacin (3mg/Kg b.w./day). In one case PGE₂ was very high, rose after DDAVP and fell to approximately normal values after indomethacin (-73%). In this case basal AMPC was high, showed minimal modifications after DDAVP (+12%), but decreased sharply after indomethacin (-50%). In the second case PGE₂ were slightly above normal values, rose after DDAVP (+28%) and were undetectable after indomethacin. Basal AMPC was high, fell after DDAVP (-21%) and rose after indomethacin (+35%). In both cases there was a marked fall of diuresis, C_{H2O}, E_{Na} and of the distal delivery (C_{H2O}+C_{Na}) after indomethacin, while GFR fell slightly. Distal fractional reabsorption of sodium (C_{H2O}/C_{H2O}+C_{Na}) was unaffected by the drug.

We suggest 1) that the adenylyl-cyclase system is intact in NDI and the PGE₂, as well as ADH, is responsible for the variations in urinary AMPC. A very high level of PGE₂ can itself stimulate AMPC and may increase the AMPC production induced by exogenous ADH. In contrast, concentrations of PGE₂ slightly above normal values probably have a minimal effect on basal AMPC, but they reduce the stimulation of AMPC mediated by exogenous vasopressin. 2) That the antidiuresis induced by indomethacin treatment in NDI is due to enhanced proximal tubular reabsorption.

NEPHROGENIC DIABETES INSIPIDUS (NDI) TREATED WITH INDOMETHACIN

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5 boys with congenital NDI have been treated with indomethacin 3mg/kg/24 hours for 12-33 months. All experienced a 20-40% reduction of urine output and three reported increased well-being. A 10% mannitol loading test was performed before and after treatment for 1 week with the following results:-

Indomethacin	Before		After	
	Alone	+DDAVP	Alone	+DDAVP
10% mannitol				
GFR(Ccr.)	128±14	120±12	139±24	123±16
V(ml/100ml GFR)	22±2	33±4	22±4	29±5
Cosm(ml/100ml GFR)	9.7±1.5	16±2.8	9.4±2.6	15.3±3.2
TcH ₂ O(ml/100ml GFR)	-11.9±.8	-16.8±1.5	-12.2±2.2	-13.7±2.2

After treatment for up to 33 months plasma creatinine and serum electrolytes remained normal. One boy was found to have symmetrically distributed non-calcified cerebral opacities unlike those of tuberosc sclerosis but they were not found in two others. We conclude that the concentrating defect in NDI is not mediated by increased prostaglandin production and that indomethacin does not exert its beneficial effect by altering collecting duct permeability to water.

LONG-TERM EFFECT OF CO-TRIMOXAZOLE (CT) ON RENAL FUNCTION

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A decrease in creatinine clearance has been described after CT administration. Plasma creatinine (umol/l), inulin and PAH clearances (ml/min x 1.73 m²) were studied prospectively in 16 children, at the start and the end of long-term CT therapy. The drug was given for 4 - 6 months for curative or preventive treatment of urinary tract infection, at a dose of 6 mg/kg/day trimethoprim and 30 mg/kg/day of sulfamethoxazole.

GROUP 1: In 4 children aged less than one year, CT did not interfere with normal functional renal maturation. Values before/after treatment: P-creat 56/62, C-in 89/110, C-PAH 347/529. GROUP 2: In 5 children aged 7 years with recurrent urinary tract infection, CT did not significantly affect renal function: P-creat 54/61, C-in 132/150, C-PAH 614/540. GROUP 3: In 7 children aged 5 years with operated vesico-ureteric reflux, CT did not significantly affect renal function: P-creat 72/73, C-in 112/100, C-PAH 520/502.

We conclude that CT does not alter renal function when it is normal at the start of treatment and does not interfere with normal functional maturation.

DEPAKENE^(R) (DPA) : a new aetiology for proximal tubulopathy. LENOIR G., PERIGNON J.L., GUBLER M.C., BROYER M. Hôpital des Enfants-Malades. PARIS. FRANCE.

A 9 year-old boy was referred on Nov. 1977 for a complete Fanconi syndrome. Prior history was unremarkable, except for a petit mal epilepsy treated with DPA (200 mg T.I.D.) since Oct. 1976. On Dec. 1976, he developed anorexia and abdominal pain persisting after appendectomy, and rapidly associated with drowsiness. Pancreatitis was suspected and in April 1977 serum transaminases were elevated (SGOT : 92 IU/l, SGPT : 33 IU/l). On May 1977 muscle weakness and alopecia were noticed. An urinalysis showed glucosuria and proteinuria. On Nov. 1977, he presented with complete growth arrest for the preceding year, and marked weakness in all limbs with normal reflexes. Evaluation disclosed in the serum : Ca : 2,2 mmol/l, P : 0,6 mmol/l, Bicarbonate : 17 mmol/l, creatinine : 70 umol/l and in the urines : proteins : 0,6 g/day, glucose : 5 g/day, Ca : 7 mmol/day with a generalized important aminoaciduria (8 times normal level). Renal biopsy showed giant mitochondria in proximal tubular cells. Ultrastructural study revealed within tubular cells an abnormal granular material in which no heavy metal was detected using Castaing's probe, glomeruli were normal. Muscle biopsy showed non specific lesions. Leucocyte cystine and serum ceruloplasmin were normal. All symptoms persisted until May 1978, when DPA was stopped : Clinical improvement was dramatic, renal function returned to normal within a few months and a catch-up growth was observed. We conclude that DPA is possibly an aetiology for Fanconi syndrome.