

## Abstracts

### Société Française de Néphrologie, Nancy, France, November 3, 1973

**Role of "physical" and natriuretic factors in pathogenesis of edemas in glomerulonephritis.** *J. P. Godon. Université de Liège, Institut de Médecine, Secteur de Néphrologie, Liège, Belgium.* (Summary of a lecture at French Society of Nephrology, Nancy 3/11/73.) In a previous work, we have demonstrated that rats with experimental glomerulonephritis (GN) were unable to decrease overall fractional sodium reabsorption and increase sodium excretion after an i.v. saline load. Unspecific factors as GFR, RPF, blood dilution, particularly pre- and postglomerular protein concentration, have no influence because they evolve with the same pattern in normal rats and those with GN. We have studied urine and plasma extracts of normal salt-loaded rats, normal rats not salt-loaded and salt-loaded rats with experimental GN by bioassay in normal rats. Natriuretic material is present in urine and plasma of normal salt loaded rats but absent in other conditions, particularly in rats with GN. The difference between the natriuretic responses of the first group and the other two groups is highly significant at  $2P < 0.001$ . Moreover, injection of natriuretic material into the renal artery of rats with GN does not induce a natriuretic effect. We conclude that natriuretic factor(s) disappear in experimental GN. If this factor or its co-factor is of renal origin, it apparently is not produced in GN. If it is of extrarenal origin, it can be destroyed by the kidney with GN, the lone organ which is diseased in experimental GN. Thus, sodium retention by the kidney with GN is due to its inability to respond to all known determinants of saline diuresis.

**Nephronophthisis and tapeto-retinal degeneration.** *J. P. Fillastre, P. Marx, R. Laumonier, J. Metayer and D. Dubois. Hôpital Charles Nicolle. Rouen, France.* We report a new case of nephronophthisis associated with tapeto-retinal degeneration in a 22-year-old man who was investigated after the discovery of proteinuria. He had had polyuria and polydipsia for some years. His blood pressure was 110/70 mm Hg; all other findings on clinical examination were normal. Proteinuria varied between 0.60 and 1.20 g/24 h and was not selective on electrophoresis. Urinary sediment and Addis count were normal. Maximum urine specific gravity after water restriction was 1.008, and the urinary pH did not fall below 5.4 after loading

with ammonium chloride. Chromatography of the urinary amino acids was normal. Blood urea was 60 mg/100 ml; creatinine 2.5 mg/100 ml; and creatinine clearance, 49 ml/min. Biopsy revealed severe interstitial fibrosis with heavy cellular infiltration. Some tubules were atrophic, others had cyst-like dilatations. The glomeruli were hyalinised. Electron microscopy revealed the fibrosis to be rich in collagen fibers and the cellular infiltration to be composed of lymphocytes and plasmocytes. Ophthalmic examination revealed nystagmus, a marked lowering of visual acuity and pigmentation affecting only the two lower parts of the chorioretinal layers. The electro-retinogram was flat and confirmed the diagnosis of tapeto-retinal degeneration. There was a superior hemianopsia. The mother and father were both free from ocular and renal disease; they were not blood relations. A sister died at the age of 12 years from chronic renal failure resulting from a histologically proved nephronophthisis; she had no ocular involvement. The other sister was normal. Only 25 other cases of this rare association have been reported. In some there were additional symptoms, namely cerebellar ataxia, mental deficiency and bone or skin abnormalities. The essentials of the syndrome are, however, the ocular and renal lesions. The renal involvement is often clinically silent, polyuria and polydipsia being the only symptoms of defective renal function. High blood pressure appears only when the renal parenchyma is considerably damaged. Urine analysis shows only proteinuria. The inability to concentrate urine appears early in the course of the disease. The prognosis is poor and, unlike the particular case of one patient, end-stage renal failure occurs within a few years. Findings of all histological studies show tubulo-interstitial lesions. The ocular involvement is usually much more serious and invalidating than in our patient and leads rapidly to blindness. The superior hemianopsia is to be noted; it is due to a tapeto-retinal degeneration of sector development involving the inferior part of both chorioretinal layers. Nephronophthisis and tapeto-retinal degeneration represent an example of hereditary disease affecting two widely separated ectodermal tissues: the renal tubular epithelium and the neuro-epithelium. The transmission of this disease seems to be autosomal recessive, the ocular and renal involvement being due to a single gene of inconstant expression.

### The Renal Association, Portsmouth, England, October 25, 1973

**Single nephron function in experimental glomerulonephritis in rats.** *M. E. Allison, C. B. Wilson and C. W. Gottschalk. Department of Medicine, University of North Carolina School of Medicine, Chapel Hill, North Carolina, and Scripps Clinic and Research Foundation, La Jolla, California, U.S.A.* Micropuncture techniques were used to study single nephron function in rats with autologous immune complex (Heymann's) nephritis (AICN), rats with anti-GBM nephritis and control rats (C). Marked

heterogeneity of superficial SNGFR (AICN, 0 to 93 nl/min; anti-GBM, 0 to 49 nl/min) and of proximal tubular hydrostatic pressure (AICN, 3 to 42 mm Hg; anti-GBM, 5 to 47 mm Hg) was found. Despite this heterogeneity late proximal (F/P) inulin levels were constant, although less than in controls, indicating that absolute proximal reabsorption varied directly with SNGFR, resulting in almost perfect glomerulotubular balance on a single nephron basis. Superficial SNGFR was related to