Further Section

Nephron 1970;7:91-95

Varia

Renal Association (May 8, 1969)

Continuous creatinine, urea and PAH clearances were measured along with urinary electrolytes in seventeen patients before and up to the sixth post-operative day following aortic valve replacement. Simultaneously, serial measurements of cardiac output, intra-cardiac pressures and plasma volume were made. Creatinine and inulin clearances were compared on fifteen occasions in three patients. In seven patients, renal vein blood was obtained throughout the study. Creatinine clearance predicted inulin clearance well, but urea clearance predicted creatinine clearance rather poorly. Creatinine clearance was normal pre-operatively, fell in the immediate post-operative period and gradually rose to normal values by the sixth post-operative day. PAH clearance was low before operation, further reduced after, but rose above pre-operative values by the sixth day. In the seven patients where renal vein blood was obtained, PAH extraction was incomplete, both before and after operation, being well below 50% on occasions. The RPF was usually higher than the PAH clearance, and both tended to move in parallel. The filtration fraction was raised before operation, rose higher on the second post-operative day and fell again by day six.

Urinary sodium concentrations below 20 mEq/l or 10 mEq/min were associated with low renal plasma flows and urine sodium was inversely related to filtration fraction. Cardiac output changed little across operation, the percentage of the cardiac output perfusing the kidneys being further reduced post-operatively. Cardiac output correlated rather poorly with renal perfusion taking the group as a whole. Neither cardiac output nor blood volume correlated well with urinary sodium.


It has been stated that children admitted to hospital with malnutrition are unable to concentrate their urine and that this function has materially improved by the time of discharge. This has been confirmed in Kampala, using conventional methods on the Bantu population, but further observations have been made: (1) the concentrating capacity on admission can be raised to that on discharge by priming the children suitably with urea; (2) although the concentrating capacity on discharge is as good as that of the local adults, these capacities, both on admission and discharge, are highly abnormal by European and American standards; (3) this is not a genetic difference. In the adults it is due to the local dietary practices and in the children to the application of an unsatisfactory test. Both the children and the adults have kidneys with normal (Caucasian) concentrating capacities, and this fact has found an application in treatment.

Papillary urinary urea concentration ratios exceeding unity have been reported in the rat kidney. The present experiments suggest that some of these published data may have been obtained during transient, non-steady state conditions, and may not constitute reliable evidence for active urea transport: (a) During the onset of water diuresis, a fall in urinary urea concentration more rapid than that in papillary concentration caused reversal of the urinary-papillary difference characteristic of antidiuresis. Even after prolonged diuresis papillary urea remained significantly higher. The qualitative pattern of change was similar to that of the papillary-urinary osmolal concentration difference, (b) Mannitol infusion produced rapid dissipation of the cortico-medullary concentration gradients. After 15-30 min, any small differences between urinary and papillary values were not significant, (c) Continuous ADH infusion produced marked increases in the cortico-medullary urea concentration gradients; urinary urea concentration increased more rapidly than papillary, and after a few hours became considerably higher.

These data are explicable in terms of passive handling: with equilibration across a freely permeable collecting duct membrane during osmotic diuresis and ADH infusion, and incomplete equilibration across a collecting duct membrane of decreased permeability in water diuresis.

Eisinger, A.J.; Jones, N.F.; Barraclough, M.A. and McSwiney, R.R.: Effect of vasopressin on renal excretion of phosphate in normal man. The renal tubular handling of phosphate, as assessed by the ratio of phosphate clearance to creatinine clearance was studied in normal subjects on varying phosphate intakes under the following conditions: (1) Hydration (maintained water load 15 ml/kg); (2) Dehydration + vasopressin (overnight fluid restriction+i.m. 5U Pitressin); (3) Hydration+vasopressin (maintained water load 15 ml/kg-[i.m. 5U Pitressin]). When phosphate excretion was low, vasopressin caused an increase in the phosphate clearance to creatinine clearance ratio, indicating that the hormone decreased net tubular reabsorption of phosphate. Vasopressin had a similar effect on phosphate reabsorption in a patient with hypoparathyroidism, suggesting that this effect does not depend on the presence of functionally active parathyroid glands.

Ngu, J.L.; Barratt, T.M. and Soothill, J.F.: Immunoconglutinin in acute nephritis and the nephrotic syndrome. Immunoconglutinin (Ik), antibody to reacted C’3 and C’4, has been found to be elevated in Nigerian adult patients with the nephrotic syndrome and to fall during remission induced by cyclophosphamide—evidence suggestive of an immuno-pathogenic basis of this disease. Sequential studies of Ik, haemolytic complement (C’hso), βSic and immuno-electrophoresis for altered complement components in European children with the acute nephritis syndrome have revealed that the rise in Ik titre follows after the fall in haemolytic complement and βSic and the development of altered complement components that occur during the early phases. Ik, and altered components, were detected in one patient in whom the complement levels were not depressed during the period of study. The Ik was the last parameter to return towards normal. Ik was raised in relapse in steroid sensitive, highly selective proteinuric nephrotic children and returned to normal on steroid or cyclophosphamide induced remission. As a group, there was no significant difference between the βSic and C’hso values in relapse and in remission, but the
increase of Ik in relapse was highly significant. This provides positive evidence to suggest an immunopathogenic basis for this disease.

Varia

Antoine, B.: A special form of proteinuria in renal transplantation: histuria. Some urine specimens from human transplanted kidneys contain an abnormal output of macromolecules bearing the antigenic features of kidney tissue (histuria). This has been established by analyzing 142 urine specimens from 29 allotransplanted and one isotransplanted patients. A standardized immunoprecipitation technique was used, with antisera precipitating only tissue components, without any effect on plasma and erythrocyte constituents. Abnormal histuria was found in 48% of all specimens (P < 0.001), and decreased progressively with time following transplantation: 0-2 months, 64%; 2-6 months, 50%; 6-18 months, 36%; 18 months or longer, 0%. No correlation exists with classical proteinuria, fever, pyuria, impaired renal function or drugs.

Abnormal histuria was very common during the course of acute rejection crises, especially during their first 7 days (88%). Histuria was also noted for the first days following an isotransplantation and operative ischaemia is a possible cause of early histuria.

Histuria has a significant correlation with pathological lesions of the kidney consisting of interstitial oedema, cellular proliferation and tubular alterations.

Abnormal histuria was frequent during the jaundice which sometimes occurs during the course of kidney transplantation, and here a concomitant hista-emia was occasionally observed. Apart from postoperative ischaemia, acute rejection crisis and jaundice, an unexplained histuria often precedes late chronic lesions of the transplant.

Transplantation histuria may be interpreted as a leakage of organic proteins directly from altered renal cells. This phenomenon may help to forecast and prevent late rejection lesions of the transplant.

Floyer, M.A.: Measurement of selective capillary permeability (SCP) to protein in rats; the effect of total nephrectomy and angiotensin.

After bilateral nephrectomy, a small increase in blood volume causes a relatively large rise in blood pressure; a similar increase does not affect the blood pressure in intact animals or following anastomosis of ureters with vena cava. Asscher [1963] has demonstrated a factor of renal origin which increases capillary permeability to protein. Therefore we studied the effect of bilateral nephrectomy on capillary permeability.

Selective capillary permeability (SCP) to protein is proportional to:

\[ \text{Ealb/EGamma glob.} \]

\[ \text{Palb/PGamma glob.} \]

Where Ealb, EGamma glob, Palb, PGamma glob are the concentrations of albumin and gamma globulin in extracellular fluid (ECF) and plasma, provided that bulk flow from capillary to ECF remains constant.

ECF samples were obtained from a capsule implanted subcutaneously [Guy-ton, 1963]. Bulk flow was measured by successive counting over capsule after injection of 131I-tagged gamma globulin.

No change in SCP was observed four days after unilateral nephrectomy, but a marked rise (i.e. fewer large protein molecules in ECF) occurred four days after bilateral nephrectomy. Four days after uretero-caval anastomosis SCP fell. Angiotensin (30 µg/kg/day) given after bilateral nephrectomy prevented the rise in SCP.

A renal factor, possibly renin, affects capillary permeability to protein.

94

Varia

Third Annual Meeting of the European Society for Paediatric Nephrology

The third annual meeting of the European Society for Paediatric Nephrology was held at the Children’s Hospital, Helsinki on June 18th and 19th, 1969. Virtually all European countries were represented and a number of prominent guests from the USA and Mexico attended. The meeting began with a lecture by Jack Metcoff of the Michael Reese Hospital and Medical Center, Chicago, who spoke in a masterly fashion about ‘Kidney metabolism and renal function in childhood’. Sixteen scientific papers were read and discussion meetings on ‘Haemolytic uraemic syndrome’ and on ‘Familial nephrotic syndrome’ were held. The guest lecture entitled ‘The development of pediatric nephrology in Latin America’ was delivered by Dr. Gustavo Gordillo P. of the Hospital Infantil de Mexico.

President for the coming year is Professor Horst Bickel, Universitäts-Kinder-klinik, 69 Heidelberg, West Germany. The next meeting of the European Society for Paediatric Nephrology will be held at Heidelberg, West Germany on September 24th and 25th, 1970.

Scientific papers:

Potassium, a factor influencing renal bicarbonate threshold in man. O.H. Oetliker, Universitäts-Kinderklinik, Inselspital, 3008 Bern, Switzerland.

Column-chromatographic determination of the organic acids of the citric acid cycle. K.J.M. van Acker, Akademisch Ziekenhuis, Kinderkliniek, Gent, Belgium.

3. Effect of hydrochlorothiazide on rickets and on renal tubular acidosis in two patients with cystinosis.


4. The effects of diuretics on the metabolic acidosis of a Fanconi syndrome patient.

D. Santos, Hospital de Pediatria, C.M.N., Mexico 7, D.F.

Effect of a cystine and methionine low diet in two patients with cystinosis. H. Holl, State University, Dept. of Pediatrics, Groningen, the Netherlands.

Bicarbonate loss in urinary ileoplasty.

H. Mathieu, Hopital Bretonneau, Paris XVIII, France.

7. Renal aspects of the respiratory distress syndrome of newborns. Therapeutic trial with peritoneal dialysis.

D. Boda, Children’s Clinic University, Szeged, Hungary.

8. Renal function in hyaline membrane disease.

A. Torrado, Hopital Cantonal Universitaire, Clinique Infantile, Lausanne, Switzerland.

9. Renal Phosphate excretion during creatinine loading.

F. Egli, Kinderspital, Basel, Switzerland.


F. Démant, Children’s University Hospital, Kosice, C.S.S.R.

11. The Addis count revalued.


Varia

95

Modified techniques for percutaneous renal biopsy in children. M. Mydlík, Children’s University Hospital, Kosice, C.S.S.R.

In vitro evidence for cellular immunity to glomerular basement membrane antigens in human glomerulonephritis.
E.J. Lewis, Robert B. Brigham Hospital, Boston, Mass. 02120, U.S.A.

16. Induction of immune tolerance to experimental autoimmune nephrosis in rats.
W. Heymann, Western Reserve University School of Medicine, Dept. of Pediatrics, Cleveland, Ohio, U.S.A.