Abstracts

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Direct renal actions of dopamine. J. L. Ader, J. P. Girolami, P. Seres, M. P. Cavalleri, T. Tran Van, and J. M. Suc. Inserm U 133. Department of Nephrology and Department of Physiology, CHU Toulouse-Rangueil, Department of Isotopic Functional Investigations. CHU Toulouse-Purpan, Toulouse, Cedex, France. The actions of dopamine on the kidney differ in man and in animals with the dose, the injection route, and the hemodynamic state. Ten new cases of glomerulonephritis, and the effects of dopamine were investigated on isolated dog kidneys, perfused at different pressures. Both kidneys of 10 different animals were perfused with heparinized blood under identical conditions. No difference was found between hemodynamics and functions of the two kidneys. One kidney was intraarterially infused with 3 \( \mu \)g/min of dopamine (D kidney) and was compared with the other kidney of the pair which was considered as the control (C kidney). They were subjected to three periods of investigation of 30 min each at a perfusion pressure of 120 mm Hg for the first 30 min, 110 for the next 30 min, and 100 for the last 30 min. Dopamine infusion induced significant increases in the total renal blood flow (176 ± (SD) 72 ml/min/100 g), the rapid component flow rate estimated with \(^{133}\)Xenon wash-out technique (87.5 ± (SD) 45.5 ml/min/100 g), the GFR (11.5 ± (SD) 7 ml/min/100 g), the total and the fractional sodium excretions (58.5 ± (SD) 45.5 mEq/min and 1.3% ± 0.7) of the D kidneys. These data remained higher than those of the C kidneys at all perfusion pressures. The second component flow rate, the glomerular filtration fraction, and the PAH extraction ratio of the D kidneys were lower than those of the C kidneys at all pressures. The venous plasma renin activities of the D kidneys were never different from those of the C kidneys. Dopamine exerts direct vasodilator and natriuretic actions on isolated kidneys with a relative increase in the cortical component of the blood flow at all perfusion pressures studied.

Membranous glomerulonephritis with malignant tumor: Report of seven cases. P. Aubert, H. Beaufils, M. Luecko, and J. Guédon. Department of Nephrology, CMC, Foch, Suresnes, France. Membranous glomerulonephritis (MGN) is idiopathic or secondary to various diseases. In this respect, the association of MGN with cancer is of great interest. From a pathogenic point of view, it is consistent with the immunologic mechanisms presumably involved in this type of glomerulonephritis. For the clinician, such a nephropathy can lead to discovery of the causal neoplasm. We report seven new cases of this association. Renal symptoms (nephrotic syndrome in all cases) antedated the discovery of the malignant tumor in five patients. Adenocarcinoma was found in six cases, and histiocytoma in one case. Four patients underwent surgery. In the only case with a survival time allowing a long enough follow-up, tumoral ablation was followed by progressive disappearance of proteinuria. In no case was renal insufficiency observed. The relative frequency of the MGN-cancer association and its severe prognosis (five patients died within 30 months following appearance of the nephrotic syndrome) emphasize the importance of an active search for an occult cancer in patients exhibiting MGN.

Metabolic fate of lactate and pyruvate in isolated human kidney cortex tubules. G. Baverel, M. Bonnard, and M. Pellet. Laboratoire de Physiologie Rénale et Métabolique, Faculté de Médecine Alexis Carrel, Lyon, France. The metabolism of lactate and pyruvate, which are normal substrates for the human kidney in vivo, was studied in isolated human kidney cortex tubules prepared by collagenase treatment from normal cortex obtained after total nephrectomy. The tubules were incubated at 37°C for 15, 30, 45, and 60 min in 4 ml of Krebs-Henseleit medium (pH 7.40) with 95% oxygen and 5% carbon dioxide as gas phase, with 1 mm lactate or pyruvate as substrate. The formation of glucose, pyruvate, lactate, and alanine, as well as lactate or pyruvate utilization were determined. Lactate and pyruvate utilization, glucose formation from both substrates, and lactate formation from pyruvate were linear with time. Pyruvate formation from lactate peaked at 45 min; after 45 min, pyruvate reutilization was observed. Alanine formation from lactate was linear with time, but the rate of alanine formation from pyruvate decreased with time. With lactate (N = 4) or pyruvate (N = 4) as substrate, and after 60 min, glucose formation was 118.9 ± 14.9 (μmol/kg/g dry wt of tubules; mean ± SEM) or 92.3 ± 16.1. pyruvate formation was 64.7 ± 12.2. lactate formation was 266.6 ± 22.0, and alanine formation was 91.2 ± 4.6 or 116.8 ± 10.7. Lactate and pyruvate utilization was 420.1 ± 41.9 and 778.5 ± 104.2, respectively. Carbon balance studies revealed that the lactate utilized was accounted for by glucose (56.6%), pyruvate (15.4%), and alanine (21.7%). The pyruvate utilized was accounted for by glucose (23.7%), lactate (34.3%), and alanine (15.0%). These results show that, in human kidney cortex tubules, the major fates of 1 mm lactate or pyruvate can be accounted for by nonvolatile products. They also suggest that there is almost no room for complete oxidation of lactate.

Renal pathological and immunofluorescent findings in Behçet's disease: A report of 11 cases. H. Beaufils, B. Cassou, J. C. Roujeau, B. Wechsler, and G. Herreman. Service de Néphrologie, and Service de Médecine Interne, Groupe hospitalier Pitié-Salpétrière, Paris, Cédex, France. Renal biopsy specimens of 11 patients with Behçet's disease (followed for 6 months to 15 yr) have been studied by light and immunofluorescence microscopy. In all cases, blood pressure and renal function were normal. Proteinuria was present in five patients. By light microscopy, amyloidosis could not be demonstrated in any case. In one patient, a focal and segmental glomerulonephritis (GN) was associated with fibrinoid mesangial and irregular subepithelial deposits. These deposits were also detected in seven other patients but to a lesser degree. Arteriosclerosis was present in all cases. By immunofluorescence, small scattered granules of C3 were observed in 10 patients in the mesangium and along the capillary basement membrane. They were diffuse in six cases, focal in four cases. Small focal deposits containing IgA and/or IgG, C3 were also observed in four cases. Rare cases of focal and segmental GN, and amyloidosis have been reported.
in Behcet’s disease. To our knowledge, glomerular C3 deposits have not been yet reported. These findings suggest that renal symptoms occasionally observed in Behcet’s disease could be related to immune complex deposition.

Diagnosis of renal hypoplasia with nephron hypertrophy in young adults. F. C. Berthoux, S. Khalil, J. C. Sabatier, S. Colon, D. Lyonne, and C. Veyrel. Unité de Néphrologie et Service de Radiologie, Hôpital de Bellevue, Saint Etienne, and Clinique de Néphrologie et Service de Radiologie, Hôpital Edouard Herriot Lyon, Cedex, France. Renal hypoplasia with nephron hypertrophy is a congenital, juvenile disease which progresses into chronic renal failure. We have observed four young adults with this disease and describe the clinical, radiological, or pathological criteria used for diagnosis. All patients presented at between 12 to 21 yr of age with a glomerular proteinuria (heavy in two cases) associated with reduction of GFR (serum creatinine, above 1.4 mg/dl). None of these patients had had acute urinary tract infection, but all cases were associated with congenital defects of morphogenesis: multicystic hydronephrosis in the family (case 1), girdle and pelvic bone hypoplasia (case 2), bilateral simian crease (case 3), and probable osteochondrodysplasia (case 4). IVP revealed small kidneys but asymmetric in size, without collected cystic dilatation of the collecting system. Retrograde cystogram demonstrated a normal, selective renal angiography, performed with x-ray tube allowing high resolution, showed the following features: 1) the smallest kidney is hypoplasic (hydropolycystic artery), and 2) There is no major scar on the cortex or renal shrinkage. 3) At the nephrographic phase, there is a “finely spotted” cortex, which was interpreted as a density decrease and an increased volume of glomeruli. Renal biopsy, performed in all cases, confirmed the decreased density (mean number of glomeruli per biopsy = 15) with 50% of the glomeruli sections exceeding 260 μm (normal size ranging from 150 to 200). There was also hypertrophy of other nephron parts, with glomerular lesions of focal hyalinosis. We conclude that renal hypoplasia with nephron hypertrophy can be seen in adults and that it differs from all other interstitial nephritis. Radiological and pathological correlations have isolated a specific angiographic sign, the “finely spotted nephrographic cortex,” which indicates scarce and hypertrophic glomeruli.

Acute pulmonary and cardiac calcifications after kidney transplantation: In vivo diagnosis by scintigraphy with pyrophosphates of Tc 99. J. Guenel, Ch. Fontenaille, J-P. Soudillon, and J. Chat- al. Nantes. France. A 29-yr-old patient hemodialyzed for 10 yr received a cadaver kidney transplant. Three weeks later, he became dyspneic and had cardiac arrhythmia. Chest x-ray showed dense opacities in both lung fields, and roentgenogram of hands and feet showed extensive calcifications of arterial vessels and soft tissues. Scintigraphy with pyrophosphates Tc 99 demonstrated lung opacities to be massive calcifications, with similar calcium deposits in the myocardium. Parathyroidectomy was done on the 45th posttransplant day. Pulmonary calcifications did not improve, but myocardial calcifications disappeared. Although secondary hyperparathyroidism appears to be a factor enhancing this rare complication related to calciphylaxis, elevation of calcium x phosphate product probably plays the main pathogenic role.

Prostaglandins and renin-aldosterone system in borderline and sustained hypertension. A. Horwich, G. Lukom, M. Safar, Y. Weiss, A. Simon, T. T. Guericke, J. Barry, and P. Molie. Hôpital Broussais, U-28, Inserm, Paris, France. Plasma prostaglandins (PG) E2, F2 alpha plasma renin activity (PRA), plasma aldosterone, and plasma and urinary electrolytes were measured in seven borderline and six sustained essential hypertensive patients; this data was compared with 13 control normotensive age-matched subjects. PRA was increased in both groups of hypertensive patients, but the increase was significant only in borderline hypertension. PGF2 alpha was not significantly increased in sustained hypertension. Pulmonary inactivation of both prostaglandins was decreased, suggesting systemic effects as circulating hormones. Sustained hypertensives differed significantly from borderline hypertensives by higher arterial blood pressure (BP), higher plasma sodium (PNa), and lower plasma volume. In the overall population of hypertensive patients, PGF2 alpha was positively correlated with arterial BP (P < 0.05) and PNa (P < 0.002) and positively correlated with plasma volume (P < 0.01) and urinary volume (P < 0.02). PGF2 alpha was positively correlated with arterial BP (P < 0.02) and PNa (P < 0.025), and negatively correlated with PRA (P < 0.005). The results provide data about the vasodilator and natriuretic effects of PGF2 alpha with an inhibitory effect on renin release. This last effect may explain the distribution of hypertensive patients according to levels of PRA. The basic disorder in clinical hypertension could be a defective renal sodium chloride excretion compensated by increased levels of prostaglandins.

Adult polycystic disease associated with the oral-facial-digital syndrome: Report of two cases. J. P. Méry, P. Simin, H. Houitte, T. Tanquerel, R. Toulet, and A. Kanfer. Centre Pasteur-Valley-Rado, Paris et Centre Hospitalier, Saint-Brieuc, France. The oral-facial-digital (OFD) syndrome described in 1954 by Papillon-Lefèvre and Psammoma is characterized by congenital anomalies of the face, oral cavity, and hands, including a typical profile with a narrowed upper lip and hooked nose, hyperplasia of gingival and buccal frenula, cleft palate, lobulated tongue, syndactyly and/or brachydactyly. OFD syndrome occurs only in females; transmission appears to be dominant, with the disease being lethal during fetal life in males. The postmortem discovery of adult polycystic kidney disease (PKD) in a mother and daughter (Dooge, 1964) and infanteile PKD in a newborn female (Tucker, 1964), all affected with the OFD syndrome, called attention to the possible association of these two hereditary diseases. The two additional cases we report provide evidence against its fortuitous nature. Both patients (EG and RR), belonging to unrelated families, show a typical and complete OFD syndrome. Both in childhood underwent several surgical procedures in an attempt to correct a minor cleft palate. In the two patients, development of severe renal failure at 22 yr (EG) and 53 yr (RR), respectively, led to the discovery of PKD of the adult type. The course of PKD had no particularity. Peritoneal dialysis was started in EG 17 months ago; hemodialysis will be started soon in RR. Neither patient had hepatic cysts. Absence of aneurysms of the cerebral arteries was documented in EG. Karyotype of leukocytes was normal in both patients. No other case of the OFD syndrome or PKD was found in either of the two families. Association of the OFD syndrome and PKD must therefore be added to the list of disorders associated with hereditary anomalies of the bones and renal disease.

Pheochromocytoma with hyperreninism. L. Monnier, A. Mimron, G. Deschodt, C. Jaffiol, and J. Miroze, Department of Metabolism and Endocrine Diseases, St. Etio Hospital, Montpellier, Cedex, France. The authors report the case of a 16-yr-old Caucasian female exhibiting hypertensive crisis and a permanent tachycardia due to a pheochromocytoma associated with a marked increase in plasma renin activity (55.4 ng/ml/hr), suspect- ed early on a persistent hypokalemia (3 mEq/liter). A catecholamine-secreting tumor arising from the left adrenal gland and extending along the medial side of the left kidney was revealed during surgery. A stenosis of the left renal vein was found by preoperative phlebography, and was confirmed during surgery. The renin activity in the renal veins (right vein, 100.8 ng/ml/hr; left vein, 65.5 ng/ml/hr) was greatly increased. There are three possible mechanisms for the hypertension: 1) catecholamine overproduction, since the sulphide test produced major increases in arterial blood pressure, plasma epinephrine, and noradrenaline levels; 2) angiotensin-dependence, based on the significant drop in blood pressure observed during the angio- tensin test (i.e., the angiotensin receptor blockade); and 3) aldoste- rone hypersecretion, highly probable since the plasma aldoste-
rone concentration was elevated: 23.7 ng/100 ml. For these reasons, the increase in renin release was certainly responsible for the overproduction of angiotensin and aldosterone, which in turn resulted in aggravation of the hypertension. On the other hand, the hyperreninemia was probably due to the juxtapaglomerular stimulating effect of the renal vein stenosis and of excessive catecholamine release.

Hypokalemic myopathy with rhabdomyolysis and acute renal failure in the course of chronic licorice ingestion. G. Mourad, P. Gallay, R. Oules, A. Mimran, and C. Mion. Department of Nephrology, C.H.U., Nimes, France. The occurrence of oliguric acute renal failure due to rhabdomyolysis in the course of chronic potassium (K+) depletion induced by long-term licorice ingestion has seldom been reported. A 51-yr-old man, with hypertension of 6 month's duration, was admitted because of acute oliguria with symptoms of acute myopathy. This patient had ingested 5 g of licorice daily for 4 yr. Upon admission, arterial pressure was 180/110 mm Hg; severe generalized weakness and oliguria were found. Blood urea was 140 mg/100 ml, plasma creatinine was 4.3 mg/100 ml, serum sodium and potassium were 146 and 1.4 mEq/liter, serum chloride was 69 mEq/liter. Uncompensated metabolic alkalosis was found; pH, 7.6; bicarbonate, 51 mEq/liter; P(O2), 54; and P(O2), 45 mm Hg. Hematost was positive in the urine. In addition, serum CPK was 9,264 mU/ml (normal, 16 to 35) and LDH was 1,840 mU/ml (normal, 110 to 160). Supine plasma aldosterone concentration was 3.6 ng/100 ml (normal, 4 to 12). Peritoneal dialysis treatment was undertaken, and urine output reappeared within 3 weeks. Complete recovery of renal function was subsequently observed. Muscle biopsy showed signs of macrovascular degeneration and myolysis. Renal biopsy, performed on the 12th day, showed extensive tubular lesions and intratubular casts. Among the factors proposed to explain this syndrome, the role of chronic potassium depletion by licorice, associated with furosemide ingestion and muscular exercise 2 days before anuria, is possible. The finding of low-renin aldosterone hypertension is compatible with an important role of licorice.

Inhibition by indomethacin of the natriuretic response to an acute saline load in the dog. A. Nizet. Institut de Médecine, Bvd de la Constitution, Liège, Belgium. We have observed that the kidneys of a dog previously submitted to a sodium-enriched diet ("Na+") demonstrate an intensive natriuretic response to an acute saline load after transplantation to the neck vessels of a sodium-deprived dog ("Na-"), moreover, the response of the kidneys in situ becomes positive. These results demonstrate an adjustment of kidney sensitivity depending on the previous sodium balance. We have transplanted to the neck vessels of "Na-" dogs the kidneys of "Na+" donors which had received an i.v. injection of indomethacin 30 min before the transplantation (4 mg/kg). In these conditions, the natriuretic response to the acute saline load is suppressed in the transplanted kidneys, and it is not potentiated in the kidney in situ. The results suggest that the sensitivity of the kidney to hemodilution, depending on the previous sodium balance, is related to renal prostaglandins which might play a direct role or might be required for the release of other active factors.

First clinical experience with sorbent regeneration of ultrafiltration (SRU) in hemofiltration (HF) in end-stage renal disease (ESRD). S. Shalvon, M. C. Beau, G. Claret, G. Deschot, R. Oules, P. Rampey, and C. Mion. Service de Néphrologie, CHR de Nimes, Nimes, Cédex, France. The encouraging results obtained with postdilution hemofiltration stimulated the development of a closed loop sorbent regeneration system for use with hemofiltration. The Amicon Difilter TM-30 yielded 5 to 6 liters/hr of ultrafiltrate, which was pumped through a D11 chloride Redy sorbent cartridge, previously saturated with 1/6 m sodium bicarbonate (5 liters at 200 ml/min for 30 min). The UF was then rewarmed and degassed by exposure to a hydrophobic gas permeable membrane. It was reconnected to the venous bubble trap after passage through a polysulphone filter to remove urease, pyrogen, bacteria, and carbon emboli. Excess UF (patient weight loss) was removed at a constant rate by a variable speed peristaltic pump. A constant infusion of magnesium, calcium, and potassium chloride was added to UF after the cartridge. Two anuric ESRD patients (body wt, 78 and 59 kg), previously treated by hemodialysis (6 months) and hemofiltration (20 liters; acetate, 40 mmoles/liter; three times per week for 3 months) were treated for 3 months, three times per week for 4 to 5 hr. Clinical status and tolerance of treatment, compared to hemofiltration, remain unchanged, and there have been no febrile reactions. Small and middle molecular solute control was similar to that of hemofiltration. Mean serum urea was: pre 220 ± 18.6, post 145 ± 15/ mg/100 ml. Creatinine was: pre 13.5 ± 1.4, post 9.1 ± 1.3 mg/100 ml. Serum electrolytes were similar except for an improvement in acidosis with the sorbent system. Serum bicarbonate with HF was: pre 16.3 ± 2.0, post 19.5 ± 1.5 mEq/liter; with SRU, it was: pre 19.6 ± 1.8, post 24.7 ± 2.1 mEq/liter. SRU permits HF without the need for expensive sterile replacement fluid and sophisticated balancing equipment. Limited experience suggests equivalent results with a better correction of acidosis.

Renal granulomatous changes in sarcoidosis. F. P. Wambargue, P. Duchatelle, P. Riberti, G. Routier, T. M. Pidiaiz, B. Gosselin, P. Dequet, G. Lelièvre. Clinique du Bois et Hôpital Calmette, Lille, France. We report four cases of renal granulomatous changes associated with renal failure. In two cases, the diagnosis of sarcoidosis was documented on specific extrarenal pathologic findings. In the two other patients, extrarenal granulomatous changes were not detected. The follow-up ranged from 1 to 5 yr. Steroid administration proved to be effective, improving renal function in two patients.