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Cadaveric renal transplantation: An analysis of 65 cases
Sixty-five patients with end-stage renal disease received 76 cadaveric kidney transplants between July 1, 1965 and June 30, 1968. Thirty-two patients were still alive including 22 patients at more than one year. Of the patients surviving more than a year (including 5 survivors from an earlier series), renal function was steady in 18 and deteriorating in 9; 14 had moderate-to-severe proteinuria. In the 33 fatal cases, sepsis was the most significant contributing factor, followed by ureteric leaks and vascular complications. The duration of peritoneal dialysis, length of ‘warm’ ischaemia-time of the grafted kidney, development of acute tubular necrosis, and the number of blood transfusions received did not affect kidney function or survival.
Author’s address: Dr. P. Pletka, Department of Medicine, St. Mary’s Hospital, London, W2 (England)

A collaborative scheme for tissue typing and matching in renal transplantation
The requirements and organisation for improving the matching of random cadaver kidneys by prospective tissue typing and exchanging of kidneys between centres are described. Computer analysis of the frequency of 13 HL-A specificities in a population of 180 renal patients and healthy volunteers in the London region has shown that a pool size of greater than 120 is required to enable close matching (1 or less differences between donor and recipient) of all kidneys which become available. In a series of 32 transplants it was possible to reduce the degree of mismatching between donor and recipient from 3.9 specificities, when the pool size was 4 or less, to 1.8 when the pool size was between 50 and 60.
Author’s address: Dr. H. Festenstein, London Hospital Medical College, London E.1 (England)

Investigation of ureteric function by isotope renography with particular reference to patients with unexplained renal pain
Transient episodes of complete or partial interruption of ureteric flow were found in 75% of 200 patients with otherwise normal renograms. This diminution of ureteric urine flow was sometimes quite prolonged, lasting over 2 ½ min in 28 of the patients. The majority of these subjects had no pathological or radiological evidence of renal or urinary tract disease. There was a history of recurrent attacks of loin pain, urgency, and frequency without consistent laboratory evidence of urinary tract infection in 14 of these cases, all but one, women. Repeated work-ups with negative results had been performed on 4 patients in an attempt to explain their symptomatology. The authors suggest that symptoms in these patients
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may be due to contractions of the ureters and the trigone, a functional condition which they compare to spastic colon. Anxiety was commonly present in these patients. The authors recommend that the diagnosis of spastic or ‘irritable’ ureter be considered as a cause of loin pain in such patients, and suggest that the isotope renogram offers a useful diagnostic aid in identifying them. They stress, however, that the diagnosis should be made only after careful examination and investigations have shown no other urinary tract abnormality.

Author’s address: Dr. R.F. Harvey, Institute of Nuclear Medicine, Middlesex Hospital Medical School, London W. 1 (England)

Syndrome of incomplete renal tubular acidosis after cadaver kidney transplantation

Renal capacity to excrete acid and lower urinary pH was tested in nine patients after cadaver kidney homotransplantation by the short, oral ammonium chloride loading test of Wrong and Davies. The control population consisted of 15 healthy subjects. The patients had a defect in acid excretion resembling incomplete renal tubular acidosis. This defect could not be accounted for by the moderate reduction in glomerular filtration rate. Impaired ability to dilute and concentrate the urine was also shown in the patients. The presence of these abnormalities suggested the occurrence of distal tubular dysfunction in the kidneys transplanted from cadavers. On the basis of these findings it is proposed that the defect in renal acid excretion contributes to the genesis of the metabolic acidosis that occasionally complicates rejection episodes in such patients.

Author’s address: Dr. O.S. Better, Department of Nephrology, Rambam Hospital, Haifa (Israel)

Coagulation studies in the hyperacute and other forms of renal allograft rejection.

Nine renal-allograft recipients were studied for consumption coagulopathy and fibrinolysis, which should be present if rejection is associated with the generalized Schwartzman phenomenon. One of the two patients with hyperacute rejections had renal arterial and venous blood sampled during transplant nephrectomy. Peripheral blood samples from three patients with acute and one with hyperacute rejections were examined before and after transplantation. Serial observations of fibrinogen, platelets and factors II, V and VIII failed to show consumption coagulopathy in either hyperacute or acute rejections despite morphologic and immunohistochemical evidence of extensive renal cortical vascular thrombosis. Euglobulin lysis times, plasminogen levels and fibrinogen split products did not provide evidence for systemic fibrinolysis. In one patient with chronic refractory rejection, renal venous blood contained small amounts of fibrinogen split products, suggesting localized fibrinolysis.

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It is concluded that intravascular coagulation during acute forms of rejection does not appear to be an analogue of the generalized Shwartzman phenomenon but rather a localized reaction confined within the graft.

Author’s address: Dr. Robert W. Colman, Hematology Research Lab., Massachusetts General Hospital, Boston, MA (USA)

Hyperacute rejection in human-kidney allografts: Shwartzman or Arthus reaction
The course of three patients indicated that both types of reaction, Shwartzman and Arthus-like, can occur in hyperacute rejection of human kidney allografts and that the late histologic appearances may be indistinguishable. In the first patient hyperacute failure of the kidney occurred in 4 hours, in association with the development of a bleeding diathesis. Sequential histologic appearances were those of progressive fibrin deposition resembling a generalized Shwartzman reaction.

In the second patient, two consecutive kidneys from the same donor were rejected within minutes of revascularization despite prophylactic heparinization. There was neutrophil infiltration without fibrin deposition. At four days there was cortical necrosis, fibrin thrombi in glomerular capillaries and minimal cellular infiltration. A third patient without prophylactic heparin, shewed both neutrophil infiltration and fibrin deposition at one hour and cortical necrosis at 5 days.

Author’s address: Dr. J.A. Myburgh, Department of Surgery, University Medical School, Esselen St., Johannesburg (South Africa)

Renal tubular acidosis, acidosis due to hyperkalaemia, hypercalcaemia, disordered citrate metabolism and other tubular dysfunctions following human renal transplantation


Twenty-one patients with cadaver-donor renal allografts were studied during the third to twenty-second week after transplantation, and three patients between one and one-half and two and one-half years after operation. In the short-term transplant group, three patients had transient classical acquired renal tubular acidosis with moderate to severe systemic hyperchloremic acidosis. Ten patients had a mild acidification defect causing a mild reduction in total acid excretion. Of the 18 short-term transplant patients with normal or only mildly defective urinary acidification, seven had mild to moderate extracellular acidosis which was not accounted for by normal or slightly raised serum lactate concentrations. Maximum urinary concentration was significantly reduced in all transplants, but was normal when corrected for increased urine flow and reduced glomerular filtration rate except in four short-term patients. Plasma calcium concentration was significantly elevated in the group as a whole, and was persistently raised above the normal range in 8 of the 24 patients. This syndrome, although demonstrable in experimental situations has only recently been recognized in clinical medicine.

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Hyperchloremic acidosis in early diagnosis of renal allograft rejection


Hyperchloremic renal tubular acidosis was observed to occur in a surprisingly high percentage of episodes of rejection in patients who had had cadaver renal allografts. Impairment of hydrogen ion excretion was the commonest defect, although multiple defects in tubular function were also encountered. In those rejection episodes that were not associated with the development of hyperchloremia, the pattern of acid excretion resembled that associated with simple loss of functioning nephrons. In over half of the cases hyperchloremia became evident before rejection became definable by conventional criteria (sometimes even 1 to 2 week before). The authors feel that elevation in serum chloride in patients who have had cadaver renal allografts is a valuable and reliable early sign of rejection. Furthermore, it may indicate
smouldering rejection activity even when serum creatinine has come back to prerejection levels during reversal of the episode. Reestablishment of normochloremia may be a good prognostic guide to the efficacy of treatment.

Author’s address: Dr. B. Mookerjee, Department of Medicine and Nephrology, Royal Victoria Hospital, Montreal, P.Q. (Canada)

Physiologic responses of the transplanted human kidney: Sodium regulation and renin secretion

The denervated, transplanted kidneys of 6 patients who had received kidneys from live, related donors (one identical twin) were able to maintain normal sodium balance. Peripheral plasma renin activity on a high-salt diet was 354 ± 28 ng of angiotensin II (±SE) in the donors and 291 ± 39 ng per 100 ml in the recipients. Renin activity (low-salt diet) averaged 620 ± 50 ng in the donors and 700 ± 52 ng per 100 ml in the recipients. Aldosterone secretory rates were normal in the patients studied as were exchangeable sodium, potassium and body water. The denervated, transplanted human kidney can maintain clearance comparable with the donor kidney and sodium regulation and renin secretion as evaluated in this study appear to be normal.

Author’s address: Dr. M. Donald Blaufox, Albert Einstein College of Medicine, 1300 Morris Park Avenue, Bronx, NY 10461 (USA)

Calcium metabolism and osteodystrophy after renal transplantation

The biochemical, roentgenological, clinical and pathological manifestations of secondary hyperparathyroidism have been investigated in 150 kidney transplant recipients 3 to 60 months after transplantation. In 84% of these patients renal disease had been present for at least three years and in 67%, at least five years prior to transplantation.

Although persistent hypercalcemia (serum calcium levels > 5.5 mEq/liter) was rare (4 patients), transient elevations were detected in 18 individuals at some time in the postoperative period. In only 1 case did the serum calcium level fail to become normal with time and parathyroidectomy was required. The possible influence of steroids, antacids, and actinomycin was difficult to determine.

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Manifestations of secondary hyperparathyroidism noted on x-ray film responded variably to transplantation and were related to the state of renal function. Patients with severe hyperparathyroidism underwent parathyroidectomy prior to transplantation (11 cases). In the remaining, demineralization of the spine (½ of patients) and milder degree of osteitis fibrosa cystica (½ of patients) present prior to transplantation, improved in the majority of individuals with good renal function within 1 year. Vascular calcifications were very slow to resorb in some patients and remained unchanged in the majority.

Eighty-eight percent of the parathyroid glands (56) and of bone specimens (43) examined at autopsy (the majority of these individuals died within 3 months of transplantation) were abnormal. The predominant histological change in the parathyroid glands was diffuse chief cell hyperplasia and the glands were between 2 and 50 times their normal size. Bone changes consistent with osteitis fibrosa cystica were the rule.

Despite chemical, roentgenological, and histological evidence of secondary hyperparathyroidism, clinical manifestations were rare in the post-transplantation period. It is
concluded that if left to their own resources, the vast majority of patients will be euparathyroid within 6 to 18 months of successful transplantation.

Author’s address: Dr. C.L. Hampers, Peter Bent Brigham, 721 Huntington Avenue, Boston, MA (USA)

Le chlorambucil dans le traitement du syndrome néphrotique idiopathique sans lesion glomérulaire chez l’enfant: A propos de 30 observations

Les auteurs rapportent leur experience du chlorambucil dans le traitement du syndrome néphrotique à lesions glomérulaires minimes chez l’enfant, à propos de 30 observations, 24 d’entre elles comportant une biopsie, les 6 autres corres-pondant à des syndromes néphrotiques purs, idiopathiques pour lesquels les signes cliniques étaient suffisamment nets pour éliminer toute possibilité de lesions glomérulaires.

Le chlorambucil donné à la dose de 0,2 mg à 0,3 mg/kg/24 h pendant une durée de 6 à 18 mois, et même plus pour quelques observations, a toujours été associé à la corticothérapie prescrite selon les regies de traitement hormonal prolongé discontinu. Les résultats sont particulièrement intéressants dans les formes cortico-dépendantes et cortico-résistantes, où les auteurs ont obtenu une proportion im-portante de remissions: 27 sur 30, dont la durée moyenne s’établit à 12 mois.

Les limites de l’efficacité du traitement sont tracées par la possibilité d’échec total (1 sur 30), de remission incomplete (2 cas sur 30), et de rechute (4 rechutes observées en 3 ans par 4 malades différents), ces rechutes ayant d’ailleurs été sen-sibles à une deuxième cure de chlorambucil. La tolerance du produit pendant la durée d’observation, a paru bonne en tenant compte des modifications habituelles, mais toujours regressives, du taux des leucocytes et surtout des lymphocytes. L’éventualité d’effets mutagène et cancérigène éloignés ne peut actuellement être encore définitivement rejetée.

Dans l’ensemble et en accord avec les travaux publiés, le bilan de l’action du chlorambucil sur le syndrome néphrotique à lesions glomérulaires minimes peut être considéré comme positif.

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Renal biopsy study of juxtaglomerular cells in renal failure. Analysis of 1609 biopsies performed in 1450 cases

A study on the renal biopsy juxtaglomerular cell counts (1609 biopsies) in patients with renal diseases (1450 cases) has been performed. Only the biopsies showing at least 6 juxtaglomerular bodies have been considered as ‘significant’ (52% of the cases). The quantitative analysis was carried out by counting the cells of 6 JGB (afferent arteriolae and Polkissen). The cells were counted and grouped in 2 categories: granulated and degranulated. The ‘normal’ JGCC was 33,6 cells ± 9,06σ. In acute renal failure either ‘glomerular’ (acute cortical necrosis, acute glomerulone-phritis) or ‘tubular’ (acute tubular necrosis, acute papillary necrosis) an average JGCC increase was found. The percentage of granulated JG cells was higher in acute hypertensive in respect to normotensive renal failure. After extracorporeal baemodialysis serial renal biopsies demonstrated a normalisation in JG cells. In chronic renal failure only rarely does RB demonstrate a significant increase in JGCC, either when the degree of the functional impairement or the presence of arterial hypertension are considered. A marked increase was
observed in malignant hypertension, and, above all, in reno-vascular hypertension. This was the only clinical situation showing an increase in the cells three-fold the normal, with a percentage of granulated cells above 25%. Serial renal biopsies carried out in cases of reno-vascular hypertension before and after surgery (aorto-renal artery by-pass) characteristically showed a morphological normalisation in JG cells. In 2 cases of kidney transplantation, finally, an increase in JGCC with a moderate hypergranularity was found in one of them 2 days after the transplant during an early rejection crisis with an abrupt increase in BP. A third renal biopsy after 1 month demonstrated a normal JGCC. The blood pressure also returned to normal. In the other case no demonstrable JGCC changes in the graft during a similarly rejection crisis was found.

Author’s address: Dr. V. Bonomini, S. Orsola Hospital, Nephrological Department, Bologna (Italy)

Transient and permanent deafness following treatment with Ethacrynic Acid in renal failure

Five uremic patients in whom deafness followed ethacrynic acid treatment are described. The dose of ethacrynic acid received varied but deafness occurred with a single 200 mg intravenous dose in one instance. The other patients had been taking the drug for longer periods. In three cases, the deafness was permanent. The cause of the deafness is unknown in these instances but it is speculated that it may be related to the retention of congeners of ethacrynic acid. The authors warn that ethacrynic acid should be used cautiously in uremic patients and the drug stopped at the first sign of a hearing deficit.

Author’s address: Dr. V.K.G. Pillay, Department of Medicine, University of Illinois College of Medicine, Chicago, IL· (USA)

Studies on the control of sodium excretion in experimental uremia

A study of the mechanisms governing the high rate of sodium excretion per ne-phron characteristic of patients with chronic renal disease was made in dogs. A ‘remnant kidney’ was produced by 85% infarction of the left kidney while the right kidney was left intact.

In a group of dogs fed 3 or 5 g of salt per day, sodium excretion by the remnant kidney averaged 6.5 µEq/min while the intact kidney was present and 53.7 µEq/min when the animals became uremic after the intact kidney was removed. The increased sodium excretion per nephron by the remnant organ often occurred within 18 h after contralateral nephrectomy and persisted despite experimentally induced acute reductions in the glomerular filtration rate to below prenephrectomy levels. A second group of animals studied in the same manner but receiving 1 g of salt per day or less failed to develop a natriuresis after contralateral nephrectomy despite high grade uremia. Thus an increased impermeable solute load per nephron was not a regulatory factor in the production of the natriuresis. The increased rate of sodium excretion per nephron in uremia resembles that after saline loading in that it may occur without an increase in glomerular filtration rate or a reduction in mineralocorticoid stimulation. It follows that an additional factor or factors must be involved in the genesis of the natriuresis.

In contrast to the natriuresis that is seen in normal animals subjected to saline loading, these uremic animals were found not to have a detectable increase in extracellular fluid volume or blood volume in the presence of high fractional sodium excretion rates.

Sodium excretion in response to a small salt load by the remnant organ in uremia was 30% greater than the response of both kidneys in the preuremic state despite a markedly reduced total
GFR. These data are consistent with the view that the volume control mechanism becomes more responsive in uremia.

Author’s address: Dr. Raymond G. Schultze, Renal Division, Dept., Internal Medicine, Washington University School of Medicine, St. Louis, MO (USA)

Guanidinosuccinic acid in renal failure, experimental azotemia and inborn errors of the urea cycle


The similarity of guanidine intoxication to the symptoms of uremia has led to a number of investigations of guanidino compounds in pathologic states due to renal insufficiency. An improved method of isolation and quantitation, using Dowex-1 ion-exchange chromatography and paper electrophoresis has permitted efficient recovery of guanidinosuccinic acid from serum, urine and cerebrospinal fluid. Mean concentration of this acid in normal urine is $1.71 \pm 1.60$ (SD) mg/100 ml and $5.35 \pm 2.68$ mg/100 ml in urine from uremic patients. Serum and cerebrospinal fluid levels also increase in uremia. In patients undergoing peritoneal dialysis, guanidinosuccinic acid was dialyzable. The metabolic pathway for synthesis of guanidinosuccinic acid is unknown. The increased excretion of this compound in rats treated with arginine, coupled

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with the failure to demonstrate the metabolite in the urine of patients suspected of having arginine deficiency resulting from genetic defects of the urea cycle, suggests that arginine is an intermediate in guanidinosuccinic acid synthesis.

Author’s address: Dr. Israel M. Stein, Section of Metabolism, Department of Medicine, Bronx-Lebanon Hospital, Bronx, NY (USA)

Increase in proteinuria due to steroid medication in chronic renal disease


Considerable increases of proteinuria caused by steroid medication have been noted in 10 of 21 patients with the nephrotic syndrome. Two had systemic lupus erythematosus, one had Schonlein-Henoch purpura nephritis, and 18 had the nephrotic syndrome in its idiopathic form. These children had the disease for 3 months to 14 ½ years and were maintained on an intermittent regimen of steroid administration. Proteinuria increased on days of steroid administration two to tenfold, abruptly from one day to another. Periods of steroid-dependent proteinuria were noted for various lengths of time. They did not necessarily last as long as the proteinuria persisted. The mechanism of this steroid effect remains unclarified. The practically important implication of this observation is that, if unrecognized, periods of steroid treatment may be unduly prolonged and doses unnecessarily increased. It also obscures the interpretation of results obtained with newly added agents, if steroid medication is simultaneously reduced.

Authors’ address: Dr. Walter Heymann and Dr. Warren E. Grupe, Department of Pediatrics, Case Western Reserve University, Cleveland, OH (USA)

Renal function in patients with chronic bacteriuria: A longitudinal study


Endogenous creatinine and urea clearances, and 15-minute PSP excretion values were obtained annually in forty-seven patients, forty-one of whom had additional evidence of chronic pyelonephritis either by renal biopsy findings or characteristic radiographic features. Patients
were followed from 3 to 6-1/2 years (mean 5-1/6 years) and their renal function was examined with regard to the presence or absence of continued bacteriuria. Because only patients who could be followed for 3 years or more were reported on, no patients with severely depressed renal function were included since they did not survive this 3-year period. Twenty-six patients lost their bacteriuria during the period of study, and twenty-one had persistent bacteriuria. Twenty-three patients showed no change in creatinine clearance in the study period, and three showed an improvement in clearance values. The remaining twenty-one patients showed some deterioration of renal function. This did not relate to the presence or absence of continued infection. The following table summarized the comparisons of patients with cleared and uncleared infections:

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<th>Infection</th>
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<td>Diabetics</td>
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<td>Creatinine clearance, %</td>
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<td>Number of positive urine cultures</td>
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There was no significant difference in the incidence of urologic abnormalities, duration of symptoms, number of positive cultures, duration of therapy, number of months the patients had bacteriuria, number of episodes of acute urinary tract symptomatology, number whose hypertension was treated or the patients’ ages to separate those whose renal function deteriorated and those whose did not. The authors conclude that the presence or absence of bacteriuria is not a factor of major importance in determining the loss of renal function in this series of patients with established pyelonephritis. They do not imply that chronic bacteriuria does not deserve to be treated, but point out that factors in addition to bacteriuria must determine the rate of nephron destruction in chronic urinary tract infection.

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The role of a renal thirst factor in drinking induced by extracellular stimuli

Much of the drinking which follows certain extracellular stimuli of thirst seems to depend on secretion of a renal thirst factor. Thus drinking after haemorrhage, hyperoncotic peritoneal
dialysis, caval ligation, or constriction of the abdominal aorta above the renal arteries, is considerably reduced by previous nephrectomy. There is a potent dipsogen in the renal cortex. This is probably renin because: (1) it is impossible to separate dipsogenic and pressor activities of extracts of kidney during the different stages of fractionation which lead to the production of renin; disappearance of one activity is invariably accompanied by disappearance of the other; (2) dipsogenic and pressor actions are more marked in nephrecto-mized rats; (3) both extractable dipsogenic factor and extractable pressor activity are reduced by treating the rat with saline and DOCA for several weeks beforehand; (4) angiotensin administered by continuous intravenous infusion causes rats in water balance to drink water, the effect being greater in nephrectomised rats; (Fitzsimons, J.T. and Simons, B. J.: J. Physiol. 203: 45-57 [1969]); and (5) angiotensin injected directly into the hypothalamus in doses several hundred times smaller than threshold intravenous doses also causes hydrated rats to drink water. (Epstein, A.N.; Fitzsimons, J.T. and Simons, B. J.: J. Physiol. 200: 98-100P [1969]).

The role and importance of the renin angiotensin system in thirst is uncertain. It is worth noting however, that all the known extracellular causes of thirst also provoke secretion of renin. The dipsogenic action of angiotensin is not mediated through the adrenal cortex since adrenalectomy does not impair the response, but precisely how angiotensin exerts its effect on drinking is not clear. There are at least 3 possibilities: (1) in large doses renin and angiotensin may increase capillary permeability sufficiently to convert an existing but perhaps dipsogenically sub-threshold hypovolaemia into an adequate stimulus to thirst; (2) activation of a vasoactive system as powerful as the renin angiotensin system may sensitize vascular receptors to an existing hypovolaemia; and (3) in view of the effect of angiotensin on the hypothalamus, its function may be to sensitize hypothalamic drinking centres to nervous information coming from thirst receptors elsewhere in the body.

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Hygroton-induced myopia


In this short report, attention is payed to the rather uncommon association of transient but significant acute myopia and chlorthalidone (Hygroton®) ingestion. Four previous cases of chlorthalidone-induced myopia have been reported and are reviewed briefly in this paper. Acetazolamide and hydrochlorothiazide also have been reported to have similar ocular effects, so that this is not a unique effect of chlorthalidone itself. In this report, a 36-year-old woman developed blurring of vision and supraorbital headache with normal near vision after taking three 50 mg doses of chlorthalidone over a six day period. Bilateral periorbital edema, conjunctival injection and chemosis were present and visual acuity was reduced markedly. The optic fundi were normal. As in other cases reported, the signs and symptoms caused by chlorthalidone disappeared completely within a week after discontinuance of the drug.

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Neuropsychologic outcome of children whose mothers had proteinuria during pregnancy

This paper has resulted from a collaborative study between the Perinatal Research Branch of the National Institute of Neurologic Disease and Blindness and fourteen cooperating hospitals. The neurologic and psychologic performance of 53 children whose mothers had heavy proteinuria during the second half of pregnancy was compared with the performance of the same number of children whose mothers had not had proteinuria. The proteinuric mothers did not have symptomatic renal disease or renal insufficiency and none was hospitalized for renal disease during pregnancy. The children were matched for race, sex, hospital of birth, socio-economic status, birth order, duration of gestation and maternal age. No renal function studies were done on the mothers and the cause of their proteinuria is unknown. Sixteen were diagnosed as having urinary tract infection, and one was given the diagnosis of chronic pyelonephritis. The children of the mothers with proteinuria scored significantly less well in the Bayley mental and motor scores at 8 months of age, and the neurologic posturing rating scale at 12 months of age. Neurologic abnormalities other than the posturing scale occurred in 5 offspring of proteinuric and in 1 of non-proteinuric mothers. They also had a significantly lower Binet IQ score at age 4. The authors suggest that the neurologic deficit of the offspring of mothers with heavy proteinuria may be related to protein deprivation, but also consider the possibility that proteinuria may signify an alternative illness factor which is itself the cause of the poorer neurologic performance of the offspring.

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Renal tubular acidosis in cirrhosis. A determinant of susceptibility to recurrent hepatic precoma
This report calls attention to the concurrence of cirrhosis and an acquired deficiency in hydrogen excretion in response to an acid load. Of fifteen cirrhotic patients studied, nine had clearly low urinary titratable acidity after a standard acid load of calcium chloride or hydrochloric acid. Urinary pH fell normally in four subjects, and the response was borderline in two others. Ammonium excretion increased to supernormal values relative to urine pH, even in those whose urine pH fell normally. Six of the nine patients with acidifying defects had impaired renal potassium conservation, excreting excessive quantities of potassium in the urine with serum concentrations of 2.0 to 3.2 mEq/1. Impaired potassium conservation was independent of the patients’ acid-base status, and aldosterone excretion rates measured in three individuals were normal. Four patients had reduced creatinine clearance values (45-50 ml/min) but Ccr was 45-117 ml/min in three others. Eight of the nine patients with acidifying defects were considered to be in hepatic precoma. The authors conclude that the co-existence of renal tubular acidosis and cirrhosis may explain why some cirrhotic patients readily become hypokalemic and remain so despite treatment with potassium salts. Renal tubular acidosis, through its effects on ammonium metabolism, may also make some patients susceptible to recurrent episodes of hepatic precoma.

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Amyloidosis in childhood
Amyloidosis in children is found in three basic underlying diseases. Of 76 cases reviewed 33 occurred in association with juvenile rheumatoid arthritis, 14 with familial Mediterranean fever, and 29 with chronic suppurative diseases. Only two cases in the chronic suppurative disease...
group have been reported since the development of antibiotics. Although the usual age of onset is in early adolescence, amyloidosis has been reported in a child only three years of age, and it has appeared when the primary illness has been clinically apparent for less than one year.

The manifestations of amyloidosis are similar whatever the underlying disease. Proteinuria is the hallmark, and its appearance in a child with chronic inflammatory disease should alert the physician to the possibility of amyloidosis. Other manifestations of renal disease are absent until terminally, when they may be present as part of the uremic syndrome. Enlargement of the liver and/or spleen is present at the time of diagnosis in over one half of the cases; however, liver failure is rare. The use of diagnostic procedures including percutaneous biopsy is discussed.

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