Further Section

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Summaries – Résumés

Micropuncture study of the effect of various diuretics on sodium reabsorption by the proximal tubules of the dog
The effect of various diuretics on fluid reabsorption in the proximal tubule of the dog was investigated by micropuncture technique using repeated collections from identical tubule segments during control and experimental phases.
Mannitol infusion produced consistent reductions in reabsorption in the proximal tubule when urine flow exceeded 4% of the rate of glomerular filtration.
After the administration to hydropenic dogs of ethacrynic acid (6 dogs) or chlormerodrin (3 dogs) there was a significant increase in the fraction of the glomerular filtrate reabsorbed in the proximal tubules. This increase in reabsorption was markedly reduced or abolished when the fluid lost as a result of the diuresis was replaced with isotonic saline solution.
Furosemide and hydrochlorothiazide in hydropenic animals also produced significant increases in proximal reabsorption.
There was a small but significant decrease in reabsorption after the administration of acetazolamide to hydropenic animals, but the change was not significantly greater than observed in repeated punctures in control hydropenic dogs.
The authors concluded that depressed proximal reabsorption does not contribute significantly to the diuresis produced in dogs by ethacrynic acid, chlormerodrin, furosemide, and hydrochlorothiazide. The increased proximal reabsorption observed appears to be a consequence of acute depletion of extracellular volume, a response opposite to that produced by infusion of saline.

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Osmolality of distal tubular fluid in the dog
Surface segments of the distal convoluted tubule were identified in the dog, thus permitting the first application of renal micropuncture techniques to an investigation of distal nephron function in this species.
The osmolality of distal tubular fluid was measured either during hydropenia and antidiuresis (5 dogs) or water diuresis (4 dogs) to evaluate the contribution of distal nephron segments to urinary concentration and dilution. In contrast to previous micropuncture observations in rodents, the distal tubular fluid was found to be markedly dilute along the entire length of the distal tubule during both antidiuresis (average osmolal tubular fluid to plasma ratio, 0.41 ± 0.13) and moderate water diuresis (average osmolal tubular fluid to plasma ratio, 0.24 ± 0.07). Despite the presence of adequate antidiuretic hormone, osmotic equilibrium between tubular fluid and plasma was never achieved at any site along the distal tubule.
The authors conclude that the water-impermeable segment of the dog nephron during antidiuresis must include the ascending limb of Henle’s loop and the entire
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distal tubule. The normal entrance of hypotonic tubular fluid into the cortical collecting duct provides a satisfactory explanation for previous observations on the excretion of hypotonic final urine at high rates of solute excretion in the dog (and perhaps in man) despite the presence of antidiuretic hormone in adequate amounts.

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Potassium reabsorption in the proximal tubule of the dog nephron

Proximal tubular fluid to plasma potassium in inulin ratios were determined by micropuncture techniques in anesthetized dogs under four experimental conditions: (1) Hydropenia, (2) hydropenia with renal arterial constriction, (3) potassium loading with acetazolamide administration, and (4) isotonic saline loading. The plasma potassium concentrations were comparable in all groups except the potassium loaded animals in which the values were elevated. In the potassium loaded, acetazolamide infusion experiments, urine pH was alkaline in all samples. There was no relationship between TF/Pk and position of sampling within the proximal tubule. The mean ratio for TF/Pk for the groups were as follows: Hydropenia-1.16, renal arterial constriction-1.12, potassium loaded, acetazolamide-infused-1.00, isotonic saline infusion-0.88. It was noted that in spite of wide variations in the renal excretion of potassium, fractional reabsorption in the proximal tubule remained relatively constant with approximately 50% of the potassium reabsorbed by 6.0 mm of the proximal tubular nephron.

The experiments with saline loading demonstrated that sodium and potassium reabsorption in the proximal tubule can operate independently. During saline loading the proximal reabsorption of sodium in the proximal tubule was decreased whereas fractional reabsorption of potassium remained constant. It is suggested that the potassium secretion occurring with saline loading may be the result of increased delivery of sodium to the distal secretory site as a result of proximal rejection with subsequent sodium for potassium exchange.

In the experiments with acetazolamide administration, inhibition of proximal tubular hydrogen ion secretion was achieved. This was associated with proximal inhibition of sodium reabsorption while potassium reabsorption in the proximal tubule was unaffected. The author suggests that the proximal tubule, therefore, unlike the distal tubule does not demonstrate a reciprocal relationship between hydrogen and potassium ion secretion. In summary, the author states that the dissociation of sodium and potassium transport in the proximal tubule on the one hand, and the constant relationship between filtered load and proximal reabsorption for both ions on the other suggests that physical factors rather than humoral are responsible for glomerular tubular balance. Conversely, continued potassium reabsorption in the face of inhibited sodium reabsorption favors a specific (possibly humoral) effect on the active transport site for sodium during the volume expansion by isotonic saline loading. The results obtained support the concept that potassium is extensively reabsorbed in the proximal tubule and that nephron segments more distal than the accessible portions of the proximal tubule are responsible for adjustments in the rate of potassium excretion.

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The effects of chronic acid loads in normal man: Further evidence for the participation of bone mineral in the defense against chronic metabolic acidosis

The authors carried out metabolic balance studies in five normal subjects to investigate the nature of the buffers utilized in the defense against chronic ammonium chloride acidosis and to determine the mechanisms responsible for the ultimate disposal of the administered acid.
Ammonium chloride acidosis was generated by the administration of 700 to 1300 mEq of oral NH₄Cl over six-day experimental periods.

Initially, as in studies of acute metabolic acidosis, acid was retained and appeared to titrate extra- and intracellular buffer systems. As acid retention continued, intracellular and bone buffers and, finally, bone mineral alone, appeared to provide additional buffer reserves. When the acid load was stopped, extra- and intracellular buffers appeared to be promptly restored, but less than one-third of the observed calcium losses were replaced during observations that lasted as long as 42 days after NH₄Cl loading was stopped.

The constant whole food diets used in the experiments provided a significant quantity of potential alkali as combustible anions, and during acid loading the fecal excretion of organic anions declined significantly.

During NH₄Cl loading, net fixed acid production was increased by an average of 3,425 mEq. Most of this acid load was excreted by the kidneys, but at the end of 12 recovery days an average of 193 mEq of the acid fed had not been excreted in the urine, despite the return of the serum bicarbonate to stable control levels. The simultaneous calcium balances averaged –185 mEq, supporting the previous suggestion that bone mineral is an important buffer reservoir in the defense against chronic metabolic acidosis.

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Ammonia metabolism in renal failure

The ammonia concentration of arterial blood was compared in control subjects and azotemic patients with and without congestive heart failure. The purpose of the investigation was to determine the effect of decreased functioning renal mass on blood ammonia levels. Renal failure was due to varied causes. One patient was anephric. The mean ammonia level (±SEM) in uremic patients without liver disease, infection or congestive heart failure (33 ± 1.4 µM/l) was below that found in control subjects (54 ± 1.8 µM/l). This difference was significant (p < 0.01). Patients with renal failure and congestive heart failure demonstrated higher arterial ammonia levels (88 ± 3.0 µM/l) than did the control subjects. Arterial ammonia levels increased from subnormal values in individual uremic patients to high levels when heart failure occurred. In one patient an ammonia tolerance test was found to be abnormal when the patient had right sided heart failure and normal after disappearance of all clinical evidence of heart failure. In three patients who developed asterixis (also had right-sided congestive heart failure) the arterial ammonia levels were above normal. Three patients with uremic twitching had low arterial concentrations of ammonia. The authors conclude that the kidney is an important 186

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contributor to the normal arterial ammonial concentration. Despite decreased renal production of ammonia, low levels of ammonia are not seen if liver function is abnormal as in congestive heart failure. Therefore, arterial concentration of ammonia in renal failure can vary over a wide range. 

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Renal tubular acidosis, hypokalemia, and acid phosphatase


A 30 year old Negro woman presented with renal tubular acidosis, chronic pyelonephritis, nephrocalcinosis, and hypokalemia. Sections of the patient’s renal cortex obtained by needle biopsy and stained with hematoxylin and eosin showed cyto-plasmic granulation most marked in proximal convoluted tubules. Sections stained by Gomori’s method showed markedly increased acid phosphatase activity (lyso-somal and non-lysosomal) primarily in distal tubules and cortical collecting ducts. Available evidence suggests that this increase in distal tubular acid phosphatase activity was caused by potassium depletion. While the proximal tubular granulations probably have a similar origin, they are not lysosomes, and their nature remains obscure. Thus, the case study does not provide supporting evidence that hypokalemic nephropathy (vacuolization and/or granulation in proximal convoluted tubules) is due to an increased number of renal lysosomes.

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The bleeding disorder of uraemia. A qualitative platelet defect


Hemostasis, platelet numbers and function, and coagulation were investigated in 19 severely uremic patients, 7 of whom had abnormal bleeding. Disordered platelet function was the commonest abnormality demonstrated. Patients with evident bleeding invariably had a prolonged Ivy bleeding time and impaired platelet aggregation and clot retraction, and in about half of them, in vivo platelet adhesiveness and availability of platelet factor 3 were also abnormal. About half of those patients who were not bleeding had reduced platelet aggregation and clot retraction, but the Ivy bleeding time, in vivo platelet adhesiveness, and tests of platelet factor 3 were usually normal. The authors note that the simple, frequently used screening tests for hemostasis, such as capillary fragility, Duke’s bleeding time, and whole blood platelet count were rarely abnormal even in the presence of uremic bleeding. They recommend that in uremic patients, these tests be discarded in favor of measurements of the bleeding time, platelet aggregation, or clot retraction and one of the tests for platelet factor 3—preferably either the prothrombin consumption index or the recalcification clotting time after Kaolin activation, both of which are simple yet give reproducible results.

The authors were able to correct clinical manifestations of bleeding and abnormal platelet function tests after treatment of the uremia in two cases by dialysis and in one instance by successful renal transplantation. In vitro disruption of platelet by freezing and thawing corrected abnormal tests of platelet factor 3 availability. They conclude that uremic bleeding is due to a reversible, qualitative defect of the platelet resulting from the abnormal biochemical environment of renal failure.

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Renal regulation of serum alpha lipoproteins. Decrease of alpha lipoproteins in the absence of renal function
Serum lipoproteins of 64 patients having renal transplantation at the Cleveland Clinic were studied; determinations were made of some of them before, immediately after, and at intervals for as long as two years after renal transplantation. Proteins were fractionated by paper electrophoresis. Lipoproteins were stained with oil red-0, and proteins with bromophenol blue. The amount of alpha lipoproteins and beta lipoproteins present was estimated by visual inspection of the strips stained with oil red-0 and graded 0–4+. The percentage of the total lipoprotein migrating as alpha lipoprotein and beta lipoprotein was estimated by scanning of the stained strips, and lipoproteins were also determined ultracentri-fugally at a density of 1.21 on some sera.

In the absence of kidneys, alpha lipoprotein levels were greatly decreased, but beta lipoproteins were relatively normal. After renal transplantation alpha lipoprotein levels returned to normal and remained there as long as the kidney was normal. Beta lipoproteins were unchanged. In those in whom rejection occurred, or removal of the kidney was necessary, alpha lipoprotein again decreased to low levels. These studies demonstrate a function of the kidneys in regulating alpha lipoprotein levels.

Author’s address: Prof. Dr. L.A. Lewis, Research Division, Cleveland Clinic Foundation, Cleveland, Ohio (USA).

Decreased responsiveness of hematopoietic tissue to erythropoietin in acutely uremic rats

The erythropoietic response to 25.0 standard A units of erythropoietin was evaluated in hypertransfused normal and hypertransfused uremic rats by means of $^{59}$Fe uptake in the spleen and red blood cells and in histological studies. Rats made uremic by means of bilateral nephrectomy or by unilateral nephrectomy with contralateral ligation of the ureter showed a significant decrease in the erythroid responsiveness to erythropoietin when compared to normal animals. In rats made uremic by means of bilateral nephrectomy, there was a relationship between the severity of azotemia and the erythropoietic response to erythropoietin. The authors suggest that some toxic factor in uremia might act as a depressant of erythro-poiesis.

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The metabolism of tritiated digoxin in renal insufficiency in dogs and man

The metabolism of tritiated digoxin was studied in dogs with acute renal insufficiency induced by bilateral nephrectomy as well as bilateral ureter ligations. Eight-day metabolic studies were obtained in patients with chronic renal disease after a single intravenous injection of tritiated digoxin. In animals with renal insuff...
activity correspond to digoxigenin-mono- and bis-digitoxoside. Only one peak of activity which migrated with digoxin was found in kidney tissue. Two patients with renal disease had an impaired ability to eliminate a single intravenously administered dose of digoxin. The total urinary activity was \( \frac{1}{2} \) that excreted by control subjects. Fecal excretion of digoxin and metabolites was increased, but not sufficiently to compensate for the diminished renal excretion. The difference in bio-transformation of digoxin in renal patients as compared to control subjects was detailed. The findings of a diminished urinary excretion of tritiated digoxin as well as renal retention of this drug in tissues provides a metabolic basis for the observed sensitivity to digoxin in renal failure.

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The irradiated hamster test. A new method of donor selection for homo-transplantation in man

It has been shown that homologous rodent lymphoid cell mixtures of binary origin evoke delay reactions when injected intracutaneously in irradiated hamsters. The intensities of these reactions appear to be related to the degree of immuno-genetic disparity between the cell donor as revealed by the survival time of skin homografts exchanged between members of the strains concerned. This report is an attempt to apply this test to the human being as a possible means of donor selection for homo-transplantation. Lymphocyte suspensions were prepared from two sources: 1. From lymph nodes removed at the time of surgery or from cadavers (1–16 h post mortem), and 2. From peripheral blood.

Using this model, it was found that (A) Cell suspensions from lymph nodes of unitary origin consistently failed to elicit significant skin reactions. (B) Peripheral blood lymphocytes also did not induce significant skin reactions in the irradiated hamsters when sufficient care was taken to purify the lymphocyte suspension. (C) When mixtures of lymphocytes from pairs of identical twins were injected into the skins of the animals, there was no appreciable difference in intensity between the lesions thus induced and lesions evoked by injection of cell suspensions of the unitary origin. (D) When mixtures of lymph node cells were injected, cutaneous lesions developed with peak scores at 48–72 h. These findings applied to genetically disparate cell suspensions prepared from lymph nodes removed from random cadaveric donors. (E) When peripheral blood lymphocyte suspensions from two unrelated individuals were mixed and injected into the skins of irradiated hamsters, an inflammatory lesion similar to that induced by human lymph node cell suspensions developed at the site of inoculation. In this regard it was apparent that high scoring results were more frequent in the genetically unrelated group than among the genetically related pairs. (F) Experiments in which the test was matched against the survival of skin homograft to indicate the degree of histocompatibility demonstrated that the irradiated hamster test was, in three of five instances, capable of selecting relatively compatible pairs of unrelated human volunteers. The authors conclude that the irradiated hamster test seems to warrant a trial of its ability to select relatively compatible donors for kidney homotransplantation in man. It is acknowledged that in this present form, however, it suffers from the fact that two-way reactions are induced by lymphocyte mixtures, and of more practical import a relatively long time is necessary to provide an answer (96 h), thereby excluding most cadaver donors sources.
Author’s address: Dr. J. W. Streilein, Department of Medical Genetics, University of Pennsylvania, School of Medicine, Philadelphia, Pa. (USA).

Chronic pyelonephritis: Relative incidence in transplant recipients
The authors have analyzed the type of renal disease in 315 uremic patients who were recipients of renal transplants in North America and Europe of whom 95% were less than 50 years of age. It was found that the ratio of pyelonephritis to glomerulonephritis was 1:2.7. This finding is at variance with conclusions based on autopsy studies of the relative frequency of these two disorders as a cause of death. The authors considered the influence of sex, age, congenital or neurologic abnormalities, methods of diagnosis and management and diagnostic criteria on these figures. None of these factors would seem to account for the high incidence of glomerulonephritis found in this group. The data collected suggest that although chronic pyelonephritis may be a common renal disease and that many patients die with this disorder, relatively few under the age of 50 die because of renal failure due to chronic pyelonephritis. Since evidence suggests only 15 to 30% of all deaths from uremia occur during the first five decades, it is quite possible that pyelonephritis is the commonest cause of death from renal failure in later decades, and perhaps even when all ages are considered together.

Author’s address: Dr. M. H. Gault, Director, Renal Service, Queen Mary Veterans Hospital, Montreal, Quebec (Canada).

Protection of the donor kidney during homotransplantation
Twenty-eight patients received a total of 31 kidney homografts from living consanguinous donors. All patients were given corticosteroids, beginning 30 h before transplantation and continuing during the posttransplantation period. In one group (10 patients), the donors received routine pre-operative treatment. In the second group (21 patients), a program of donor ‘renal protection’ was used, including: (1) induction of diuresis by infusion of 15 ml/kg body weight of 5% dextrose and 0.45 normal saline for one hour before induction of anesthesia; (2) strict maintenance of normal blood pressure during operation and continuous fluid replacement; (3) maintenance of diuresis by intermittent injections of Mannitol. In the first two months after transplantation, the incidence of failure was 40% in the group in which renal protection was not performed, as compared with the failure rate of 5% in the ‘renal protective’ group. An analysis of the failures in the first group revealed patterns of renal damage secondary to ischemia despite an average ex vivo ischemia of 19.9 min. The overall mortality was 60% in the first group, but only 19% in the ‘renal protective’ group. Immunologic factors did not appear to be responsible for the early renal damage. Renal injury was initiated while the kidney still remained with the donor during nephrectomy. It is felt that this type of damage can be prevented by prehydration and Mannitol-induced diuresis in the donor.

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Membranous glomerulonephritis in two human renal homotransplants
Two human kidney homotransplants are described in whom proteinuria above 3.5 are G/24 h was noted 2½ and 13 months after transplantation. One patient had all the stigmata of the
nephrotic syndrome. Renal biopsy demonstrated the histologic picture of chronic rejection and thickening of the glomerular capillary basement membranes. Electron microscopic study of the biopsy material from one patient showed thickened basement membranes and fusion of the podocytes.

In one of the patients the original disease in the kidneys before transplantation was chronic pyelonephritis; in the other it was chronic glomerulonephritis. The authors suggest that these glomerular lesions found in the transplanted kidney are due to the immunological rejection processes and do not depend on the pre-existence of glomerulonephritis in the host prior to transplantation. It is also suggested that the early cellular infiltration, the arterial obliteration, and the glomerular capillary basement membrane thickening represents different phases of a homograft reaction which has been modified but not halted by treatment.

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Hyperacute rejection of kidney allografts, associated with preexisting humoral antibodies against donor cells


In this report two cases of acute accelerated rejection of cadaveric renal allo-transplants in multiparous women are described. In both cases urine secretion was active following opening of the arterial anastomosis at surgery, however, rapidly fell off within ten minutes and completely ceased within one hour. Histologic examination of the transplanted kidney revealed microthrombi throughout the glomerular architecture and was almost identical to that seen in renal cortical necrosis associated with a generalized Schwartzman reaction. The serum from both patients contained complement-fixing thrombocyte and kidney antibodies beside leukocyte agglutinins. The antibodies were active against antigens from the cadaver donors used. The antibodies were further characterized as to their immunoglobulin characteristics and were found to be a combination of IgG, IgA, and IgM. The authors postulate that these patients had preexisting humoral antibodies active against antigens in the graft, and this may have had a decisive effect on the fate of the transplant. It was further postulated that the reaction between these antibodies and graft antigens functions as a trigger mechanism in the formation of microthrombi leading to impairment of the cortical blood flow and subsequent cortical necrosis.

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Transmission of glomerulonephritis from host to human kidney allo-transplant


This paper reports the clinical, serologic, and pathological findings in a patient in whom glomerulonephritis developed in a kidney transplant received from her mother. The recipient was anuric because of far advanced glomerulonephritis, which had developed from an acute poststreptococcal glomerulonephritis six months before. Gross hematuria occurred a few days after transplantation, and hematuria along with heavy proteinuria persisted during the entire course, which terminated with deteriorating renal function and death after four months. The kidney allograft showed typical subacute glomerulonephritis with epithelial crescents, in addition to obliterative vascular disease. The serum from the recipient did not containing leukoagglutinins or complement-fixing thrombocyte antibodies active against antigens from eight normal donors,
but two samples of recipient serum contained (before transplantation) weak leukoagglutinins active against leukocytes from the mother. These agglutinins disappeared after transplantation. The authors believe that this case represents a transmission of glomerulonephritis from the recipient to the donor kidney owing to persistent hypersensitivity on the part of the host at the time of transplantation.

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Serum erythropoietin after renal homotransplantation

In this study serum erythropoietin was assayed in seven patients at various times before and after renal homotransplantation. Pretransplant erythropoietin was either undetectable or minimal. All patients had significant serum erythropoietin at some time after transplantation. In several cases there was considerable delay between improvement in renal excretory function and the appearance of serum erythropoietin. The levels of erythropoietin were influenced by hematocrit level, presence of rejection crises, and possibly by renal infection. Reticulocyte counts paralleled, but lagged behind erythropoietin changes. Late rises in erythropoietin with accompanying reticulocytosis occurred in three patients at times when their hematocrit reading was normal or above normal, and their blood pressure was elevated. In the latter instances it is postulated that an ischemic mechanism may have increased renal production of erythropoietin. It is concluded that the transplanted kidney produces erythropoietin in response to both anemia and possibly renal ischemia.

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Peripheral plasma renin activity in renal homotransplant recipients

In order to study the relationship of the hypertension occasionally occurring after renal homotransplantation, peripheral plasma renin activity was measured in 11 patients with terminal renal failure supported by chronic hemodialysis while awaiting homotransplantation and in 3 of these patients the assays were repeated after bilateral nephrectomy. Serial renin assay were performed on 7 patients after renal transplantation. The renin activity ranged from 38 to 1105 ng/100 ml. A percent in the patients with chronic renal failure before transplantation. Two patients had elevated renin activity before transplantation; both these patients suffered from chronic pyelonephritis. After bilateral nephrectomy renin-like activity was present in very low levels in the 3 cases tested. Four transplant recipients were observed to have elevated plasma renin levels during their postoperative course. One of these patients had congestive heart failure and another, malignant hypertension with congestive heart failure. This study suggested that the hypertension in kidney transplant recipients is not necessarily on the basis of elevated levels of plasma renin. Renin elevations may occur in hypertensive recipients at the time of severe hypertension and congestive heart failure. However, there is no consistent relation between the renin levels and the degree of hypertension.

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Comparative results of cadaver and related donor renal homografts in man, and immunologic implications of the outcome of second and paired transplants


Since August, 1962, 89 non-twin renal homotransplants have been performed at the Medical College of Virginia in 82 recipients with an overall survival of 66% up to 3½ years after transplantation. Recipients with related donor transplants demonstrated somewhat better survival, function, and immunologic acceptance of the graft than those with cadaver donor transplants. Both related and cadaver donor grafts are capable of anatomic and functional hypertrophy to the same extent seen in the living donor’s own kidney. If a second transplant is performed after failure of the first, a better state of acceptance is usually achieved. The interval between removal of the first and grafting of the second kidney may be of importance to the outcome of the transplant.