Summaries – Résumés

Étude par la microscopie électronique à balayage de la surface apicale des cellules du tube contourné distal du rein de rat
Le rein de rat, fixé au glutaraldéhyde, est découpé en tranches traitées par l’acide phosphotungstique à 1% dans de l’acide chlorhydrique, et métallisées immédiatement avant l’examen. Dans la lumière des tubes contournés distaux et des collecteurs, on distingue deux types de cellules dont la surface apicale est différente. Les unes sont lisses, parsemées par quelques microvillosités en doigt de gant, moins rares au pourtour de la cellule. Les autres, surtout nombreuses en aval, sont caractérisées par de très abondantes villosités serrées, en feuilletés, donnant un aspect en madrepore. Des états intermédiaires sont aisément identifiés. Le deuxième type de cellules, à surface tomenteuse, correspond aux cellules sombres ou intercalaires (dark cells).
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Early metabolic alterations in renal allografts
The isolated perfused kidney was used to evaluate metabolic activity in normal kidneys and in isografted and allografted kidneys 48 h after transplantation. In normal kidneys, palmitic 1-14C free fatty acid was rapidly incorporated into tissue lipids, primarily phosphatidyl choline and was oxidized to 14CO2. Isografted kidneys were similar to normal kidneys in metabolic activity. In contrast, allografted kidneys incorporated less radiolabeled palmitic acid into phospholipid and had a threefold increase in oxidation of fatty acid. The shift from phospholipid synthesis to oxidation was apparently not due to infiltration with foreign cells or major changes in blood flow, but probably resulted from shifts in tissue enzyme activities.
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Renal transplantation for childhood cystinosis
Four cystinotic children with renal failure received a renal allograft from a parent. The follow-up durations for the four patients were 8, 14, 16, and 32 months; final creatinine clearances were 115, 66, 130 and 41 ml/min/1.73 m2 respectively. No decrease in cystine crystals was visible on corneal and bone-marrow examinations. Although the allografts accumulated intracellular cystine, the distribution of cystine was different from that in the natural disease. Cystine crystals were plentiful in interstitial cells of both the patients’ original and their transplanted kidneys, but crystals were found in glomerular and tubular epithelial cells only in the original kidneys. Moreover, changes in renal function correlated with complications of transplantation and clinical rejection episodes rather than with the amount of accumulated...
Cystine. The Fanconi syndrome did not appear to be developing at the time of this report, and all patients had returned to full activity.

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Circulating lymphocyte depletion: effect on renal allograft survival in calves

Thirteen calves were prepared by either one week or two weeks of lymphocyte depletion via a continuously draining thoracic duct fistula. All lymph was reinfused after removal of the cells. Marked lymphocyte depletion was demonstrated within five days in the lymph, blood and lymphoid tissues. Renal allografts failed to survive beyond 14 days in the animals depleted for 7 days prior to transplant, regardless of the duration of depletion after transplantation. Four of eight calves with 14 days of depletion survived for 21, 60, 95 and 315 days. Two of these animals died from urinary tract infections and two were alive at 60 and 315 days. The four other animals in this group lost their grafts from urinary tract infections and ureteral obstruction. Analysis of data between the two groups of blood and lymph lymphocyte counts, number of cells removed, lymph-flow rates, and lymphoid tissue morphology did not reveal any significant differences. The single determinate of prolonged graft survival was the duration of depletion of lymphocytes prior to allotransplantation.

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Clotting changes including disseminated intravascular coagulation during rapid renal-homograft rejection

One of two patients in whom early homograft rejection developed after renal transplantation had many antidonor antibodies before operation. By the measurement of gradients across intracorporeal and extracorporeal homografts in this patient, the new kidneys were shown to sequester host immunoglobulins, platelets, white cells and clotting factors. Moreover, the renal venous blood then contained fibrinolytic activity. This presensitized recipient, as well as a second patient who did not have detectable preformed humoral antibodies, gave evidence from clinical observation and from the various clotting tests of disseminated intravascular coagulation with fibrinolysis and a severe bleeding diathesis. Immuno-fluorescent and histologic studies revealed a laying down of fibrin in the homo-graft vessels that continued in some cases to cortical necrosis of the transplanted kidneys or, alternatively, receded at the time fibrinolysis occurred. The variety of rejection seen in these patients has been characterized as an immunologically induced coagulopathy.

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In-vitro evidence for cellular hypersensitivity to glomerular-basement-membrane antigens in human glomerulonephritis

The macrophage inhibition assay was used to study in vitro the reactivity of human blood lymphocytes to a soluble preparation of glomerular-basement-membrane antigen. Twenty-one
patients with various forms of renal disease, 10 with diseases not affecting the kidney and
13 normal subjects were evaluated. The lymphocytes from 6 of 14 patients with glomerulonephritis
exhibited cellular hypersensitivity to glomerular-basement-membrane antigen, whereas, with one
exception, lymphocytes from the other subjects failed to do so. Immunofluorescence studies on 4
of the 6 patients with positive macrophage inhibition tests revealed that 3 had concomitant
evidence of antibodies directed against the glomerular-basement-membrane. These results
indicate that cellular hypersensitivity is present in certain forms of human glomerulonephritis
and suggest that sensitized lymphocytes may be involved in the pathogenesis of renal damage in
this disease.

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Antilymphocyte-serum preparations in treatment of renal allograft rejection

Seven renal homograft patients with rejection episodes were treated with anti-lymphocyte serum
(ALS) or antilymphocytic globulin (ALG) besides steroids and azathioprine. Only two patients
overcame the rejection episode. These results were no better than would have been expected had
antilymphocytic preparations not been added to the steroids and azathioprine. Thrombocytopenia
was seen in most patients receiving ALS or ALG and frequently limited the course of treatment.
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