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Transport of folate compounds through the membrane of normal and transport-deficient lymphoid cells.

The active transport of folic acid (FA) 5-methyltetrahydrofolate (5-MeFH4) and methotrexate (MTX) was investigated in mouse leukemia L 1210 and in a human lymphoid cultured cell line. In both cell types exist two different transport systems, one for 5-MeFH4 which transports also MTX and one for FA which is much less effective. The first system can be inhibited by blocking sulfhydryl groups in the membrane with organic mercurial compounds. But no SH-groups seem to be involved in the transport of FA. Iodoacetate, however, leads to an increased total uptake of MTX.Resistance to MTX can be due to decreased uptake of the folate antagonist. A cell strain with impaired transport of MTX takes also up less 5-MeFH4, where as the transport of FA is not decreased. Competition experiments show that folinic acid shares the transport system with MTX and 5-MeFH4. This system seems to be the important pathway for the uptake of folate and antifolate compounds into these cells, while FA is probably taken up by a much less specific system.

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14 children, 5 to 14 years old, received cadaveric renal kidneys during 1973. Donors were 2 to 41 years old. Kidney size was greater than normal for the recipient in 9 cases, less in 3 cases, and equivalent in 2 cases. When too large to be placed in the pelvis, the kidney was placed in a paravertebral position. Ureteroureteral anastomosis was performed in most cases. Treatment was Azathioprine and corticotherapy. High doses of Furosemide (10 mg/kg) during operation prevented ischemic acute renal failure, despite cold ischemia as long 23 hours. One child died after 1 week from intracerebral hemorrhage due to severe hypertension. A second died after 5 weeks from a diffuse hemorrhagic syndrome. Primary thrombosis of the kidney vessels led to transplantectomy in 1 case. Kidney function is normal in 9 cases, chronic rejection is present in 2. No urologic complications were observed. Hypertension developped in half of the children. One case was associated with renal artery stenosis, another with chronic rejection. Hypertension was more frequent in those who had severe hypertension, leading to binephrectomy, before transplantation. No correlation was found between occurence of hypertension and kidney size. Although follow-up is too short, growth appears possible in prepubertal children, when steroid doses are low. Rehabilitation was total within 3 months in 10 cases. Cadaveric kidney transplantation seems to be a valuable therapeutic procedure in children with chronic renal failure.

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The concentrations of immunoglobulins and complement components in the sera of children with glomerular disease.

Various complement components are estimated in the sera of patients with renal disease for the purposes of diagnosis and investigation. Deviation from normality may reflect involvement in the pathogenetic process, or may be a secondary consequence of proteinuria. We have therefore systematically measured plasma C1q, C4, C3, GBG, IgG (and specifically subclass 3), IgA and IgM, and have related them to the plasma concentration of albumin and to the urine albumin/creatinine concentration ratio.

GBG correlates positively with the plasma albumin concentration, and negatively with the urine albumin/creatinine ratio, indicating that its serum concentration is affected by urinary loss. Plasma IgG, but not IgG subclass 3, correlates positively with plasma albumin, indicating a different clearance and/or turnover rate of IgG3 from other IgG subclasses.

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Complement studies in nephrotoxic serum nephritis (NSN) and aminonucleoside nephrosis (AN) of the rat.

Total complement (CH50) and C1-C9 have been determined in sera and urines of rats with NSN and AN. In NSN, 3 hrs. after injection of antiserum, a significant drop of CH50 and C1-C8 in serum was observed, whereas C9 remained normal. At day 12 CH50 and the complement factors were normal except C1, which was still slightly diminished. In urine, at day 1 only C8 and C9, at day 12 small amounts of C1,C4,C8 and C9 and, less frequently, of C3-C7 could be measured. In AN, at day 12 CH50 and C1-C9 were strikingly diminished in serum. In urine, at day 12 C3,C5,C7,C8 and C9 could be determined regularly, whereas urinary excretion of C1,C4,C2 and C6 was observed in about 50% of the animals. In NSN, the consumption of complement in serum reflects the ongoing immunological process. The low clearances of complement components, compared to AN, may be due to their fixation and inactivation in the kidney. In AN, the reduction of complement in serum may be the result of loss of complement factors in oedema, ascites and urine, and/or of impaired synthesis of these proteins.