into the extracellular fluid when they are destroyed. The principal site of katabolism of extracellular lysozyme is the kidney: it enters the glomerular filtrate, and is reabsorbed and catabolized by the proximal tubule.

The fractional excretion of lysozyme (clearance of lysozyme/clearance of creatinine, \( C_L/C_C \)) was measured in healthy adults:

\[
C_L/C_C = 1.05 \pm 0.96 \times 10^{-3} \quad (\text{mean } \pm \text{SD}, n = 16).
\]

\( C_L/C_C \) showed a positive correlation with the rate of urine flow, but no correlation with albumin excretion was observed.

In healthy full-term newborns, \( C_L/C_C \) was not significantly increased:

\[
C_L/C_C = 1.44 \pm 1.02 \times 10^{-3} \quad (n = 8).
\]

This aspect of proximal tubular function is therefore normal in the neonate.

\( C_L/C_C \) was normal in the nephrotic syndrome in children:

\[
C_L/C_C = 0.76 \pm 0.68 \times 10^{-3} \quad (n = 12).
\]

These data indicate that the presence of high concentration of albumin in proximal tubular fluid does not interfere with lysozyme reabsorption, and support the hypothesis that small and large proteins do not compete for the same transport system.

In 4 children with the Fanconi syndrome due to cystinosis, the excretion of lysozyme was much increased, confirming proximal tubular dysfunction:

\[
C_L/C_C = 56 \pm 10^{-3} \quad (\text{mean}; \text{range } = 20 - 84 \times 10^{-6})
\]

The possibility of local production of lysozyme was examined in children with pyuria. In such children with structurally normal kidneys and normal blood ureas the urine lysozyme/creatinine concentration ratio did not differ from that of the normal adults.

The excretion of lysozyme therefore offers a simple test of proximal tubular function, and, in contrast to techniques of assessment of amino acid excretion, provides easily obtained quantitative data to which statistical techniques can be applied to assess the natural history and response to therapy of tubular disorders.

Use of Rat Fetus in Experimental Teratology.

C. L. Berry (Institute of Child Health, Guilford Street, W.C.1). Abnormalities of cell growth, differentiation, or morphogenetic movements during organogenesis represent the principal cause of major malformations in man. In teratological experiments it is difficult to observe organogenesis directly in mammals. The rat fetus may be grown in organ culture throughout the period of organogenesis, and this experimental model is of considerable value. It enables the possible effects of maternal metabolism on teratogens to be excluded, and the ability to initiate the direct absorption of antibody without the possibility of maternal cross-reaction is useful. The relations of growth and differentiation in this system, and the effects of trypan blue, methotrexate, and specific antisera have been studied.

Porosity of Placenta in Mouse to Maternal Cells. R. D. Barnes (Department of Haematology, Institute of Child Health, London). There have been occasional reports of maternal cells in the cord blood of newborn infants; however, it is commonly accepted that this is perhaps an infrequent occurrence. In theories concerning the immunological significance of the feto-maternal barrier the placenta has been considered to protect the fetus (an allograft) against maternal rejection.

Using an ovum transplantation model it has now been shown that a substantial number of nucleated cells from the mother are present in the young mice. Fertilized ova from normal CFW mice have now been successfully uterine-nurtured to term in the uterus of pregnant CBA/T6T6 mice, having cells with a characteristic chromosomal marker. Surprisingly, CFW mice derived in this way have up to 30% of maternal cells with the chromosomal marker—in fact these animals are chimeras. In these animals there is no evidence for the rejection of this 'graft' and no apparent evidence of any graft-versus-host reaction. The morphological nature of these foreign maternally derived cells is as yet unknown, but it seems likely that they represent nucleated blood cells. If these cells include maternal lymphocytes then their apparent immunological inactivity as a graft towards a genetically foreign host needs explanation. Chimerism here has been demonstrated in uterine-nurtured animals, and conceivably the surgical procedure of ovum transplantation itself might be held responsible. Preliminary data, however, suggest that chimerism in mice is a natural phenomenon since normally derived mice have maternal cells.

The significance of these observations might well influence theories of genetic transmission and development of immune tolerance. In addition, maternally derived cells may play a part in the development of both autoimmune disease and neoplasm, and these cells could conceivably be utilized in an active immunotherapy programme of neoplasms.

Blood Viscosity in Newborn Infant and Diagnosis and Treatment of Hyperviscous States. T. Mackintosh (Royal Infirmary, Dundee). Viscosity is the factor that determines how a fluid will behave when it flows. The viscosity of blood is not fixed but varies with the rate of shear, gradually increasing as shear rate decreases.

The normal range of blood viscosity was determined by studying 110 full-term, singleton, normal infants weighing over 2600 g., using a Wells-Brookfield synchro-electric microviscometer model L.V.T. (shear rate range 1·16 to 232 sec.\(^{-1}\)), with a special attachment for small blood samples. This confirmed that blood was a non-Newtonian fluid, with a viscosity varying from 5·47 c.p.s. at a shear rate of 232 sec.\(^{-1}\) to 33·6 c.p.s. at 1·16 sec.\(^{-1}\). A close correlation exists between blood viscosity and the venous PC\(_v\), but there is no relation to birthweight or time from birth.

Further studies showed that respiratory distress (20 infants) and prematurity (21) were not usually associated with increased viscosity, but the mean for 12 dysmature infants is only just within the normal range. 16 infants had signs attributable wholly or in part to