POLYSULPHONE MEMBRANE MIMICKING HUMAN GLOMERULAR BASEMENT MEMBRANE

SIR,—A membrane dialyser that does not exclude molecules in the higher molecular weight range has long been sought, and the development of haemofiltration was a direct result of the search for such a membrane. However, haemofiltration requires the administration of copious fluids: a membrane that could also function with a high-molecular-weight cut-off in the dialysis mode (diffusive mass transfer) would have advantages for routine haemodialysis. We have evaluated such a dialyser (Fresenius Co, Bad Homburg, West Germany) in vivo, looking at ultrafiltration capacity, clearance data, and sieving coefficients (see table) and biocompatibility.

PLASMA CLEARANCE DATA (ml/min) AND SIEVING COEFFICIENTS (S_c) IN TWELVE HAEMODIALYSES

Mode*	Urea	Cr	P	Inulin	β_2M
HD	189	168	157	85	56
HDF	191	173	165	93	81
HF	119	118	117	120	95
S_c	*0·995*	*0·967*	*1·003*	*1·056*	*0·791*

Cr = creatinine; P = phosphate; $\beta_2M = \beta_2$-microglobulin.
*Conditions were as follows: Diffusive procedure (HD): Q_B 200, Q_D 500, Q_F 0 (ml/min). Mixed procedure (HDF): Q_B 200, Q_D 500, Q_F 50 (ml/min). Convective procedure (HF): Q_B 200, Q_D 0, Q_F 120 (ml/min). Sieving coefficients were determined during the HF mode.

Clearance of important uraemic solutes in the diffusive (HD), convective-diffusive (HDF), and pure convective (HF) transport modes were similar except for substantial increases in inulin and β_2-microglobulin clearance rates during the purely convective mode, with significant retention of β_2-microglobulin (as shown by the low sieving coefficient). We know of no other artificial membrane with such a high diffusive mass transport capacity. Dialysis was not associated with a fall in leucocyte or platelet count, and there was no activation of the complement components C_3 and C_4.

Department of Nephrology
and Hypertension,
Katharinenhospital,
7000 Stuttgart 1, West Germany

E. STREICHER
H. SCHNEIDER

AIDS IN 1959?

SIR,—The acquired immunodeficiency syndrome (AIDS) is so far unexplained. It is widely presumed to be caused by transmission of an infective agent—if so why were cases not reported earlier? We therefore wish to draw attention to the patient with cytomegalic inclusion disease and *Pneumocystis carinii* infection we described in *The Lancet* in 1960.[1]

His clinical history was of gingivitis, non-irritant lesions on the skin, breathlessness, nocturnal sweats, anorexia, weight loss, tiredness, and fever. Later a large painful anal fissure developed and in April, 1959, a small papule appeared in one nostril.

In April, 1959, he was admitted here but investigations[1] failed to provide a clinical diagnosis. The anal lesion progressed remorselessly and the nasal lesion broke down into a chronic ulcer, eroding the nasal cartilages and spreading to the lip. He died in September, 1959.

Histological examination revealed large cells in the lungs typical of cytomegalic inclusion disease and scattered cysts of *P carinii*. We could not explain this patient's decreased resistance to infection. Could he have had AIDS? He had previously been well. While in the navy (1955–57) he had travelled abroad. He was not married and we know nothing of his sexual orientation.

Perhaps AIDS is not a new disease; rare examples may in the past have masqueraded under various diagnoses.

Manchester Royal Infirmary,
Manchester M13 9WL

GEORGE WILLIAMS
T. B. STRETTON
J. C. LEONARD

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VIRUSES AND SCHIZOPHRENIA

SIR,—A variety of insults (metabolic, toxic, traumatic, or infectious) may result in schizophrenic symptoms[1] and *The Lancet*'s letters pages have lately carried genetic evidence both for[2,3] and against[4,5] a viral aetiology of schizophrenia. Our immunological findings may help to evaluate the role of viruses.

Albrecht and others,[6] using an enhanced neutralisation test, demonstrated a higher cerebrospinal fluid (CSF) to serum ratio of antibody against cytomegalovirus (CMV) among schizophrenics than in former narcotic addicts and hospital staff. Torrey and others,[7] using an enzyme-linked immunosorbent assay (ELISA), found increased CSF but not serum IgM against CMV in 11% of schizophrenics and 18% of manic-depressives but in only 3% of neurological and in no volunteer controls. Both studies are consistent with local antibody production in the central nervous system. A third study,[8] however, based on indirect immunofluorescence, did not detect antibodies against CMV in the CSF of any of 19 schizophrenics.

35 inpatients at Saint Elizabeths Hospital (mean age 31, range 19–62) meeting DSM-III criteria for schizophrenia were studied, along with 6 inpatients at George Washington University Hospital (mean age 53, range 27–78) with various non-viral neurological disorders. CSF was analysed, "blind" to diagnosis, by an ELISA similar to that used by Torrey for IgM against CMV antigen. Total plasma and CSF IgM levels (immnofluorescence assay) were available for 21 schizophrenics.

6 of 35 schizophrenics (17%) and 1 of 6 neurological controls were IgM positive—ie, they had CMV antibody levels more than 2 SD above the mean for 38 children at Johns Hopkins University Hospital, aged 1–16, with suspected meningitis. Among the schizophrenics, IgM positive and negative patients did not differ by age, duration of illness, length of hospital stay, or blood-brain barrier permeability, as assessed by the CSF to plasma ratio of total IgM (mean±SD 0·11±0·11 and 0·11±0·22, respectively). Of the 19 patients off neuroleptics for three weeks or longer, 2 were IgM positive; of the 9 patients receiving neuroleptics, 1 was IgM positive. Unexpectedly, IgM status appeared to be associated with the frequency of CSF sampling. Thus, 3 of 29 patients who had had only one lumbar puncture were IgM positive, while 3 of 6 patients studied twice were IgM positive in one of two samples taken 112 days apart on average ($p<0·05$, Fisher's exact test). Unlike Torrey's patients, all of whom remained IgM positive if restudied within 49 days, 1 of our patients converted from IgM positive to negative within 36 days.

IgM antibody to CMV also seemed to be associated with structural brain lesions. 4 of 6 IgM-positive schizophrenics showed signs of brain atrophy on computerised tomographic scan compared with 6 of 28 IgM-negative schizophrenics ($p<0·05$, Fisher's exact test). Brain atrophy, defined as ventricular size outside the normal range and/or clear-cut cortical *and* cerebellar vermian atrophy, was assessed "blind" to the immunoglobulin results.

CSF IgM against CMV does not seem to be an artifact of age, length of hospital stay, alteration in blood-brain barrier, or neuroleptic drug treatment.[6,7] The probability of finding CSF IgM may depend on how often one looks. Frequent samples may be more likely to coincide with antibody production during brief episodes of

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