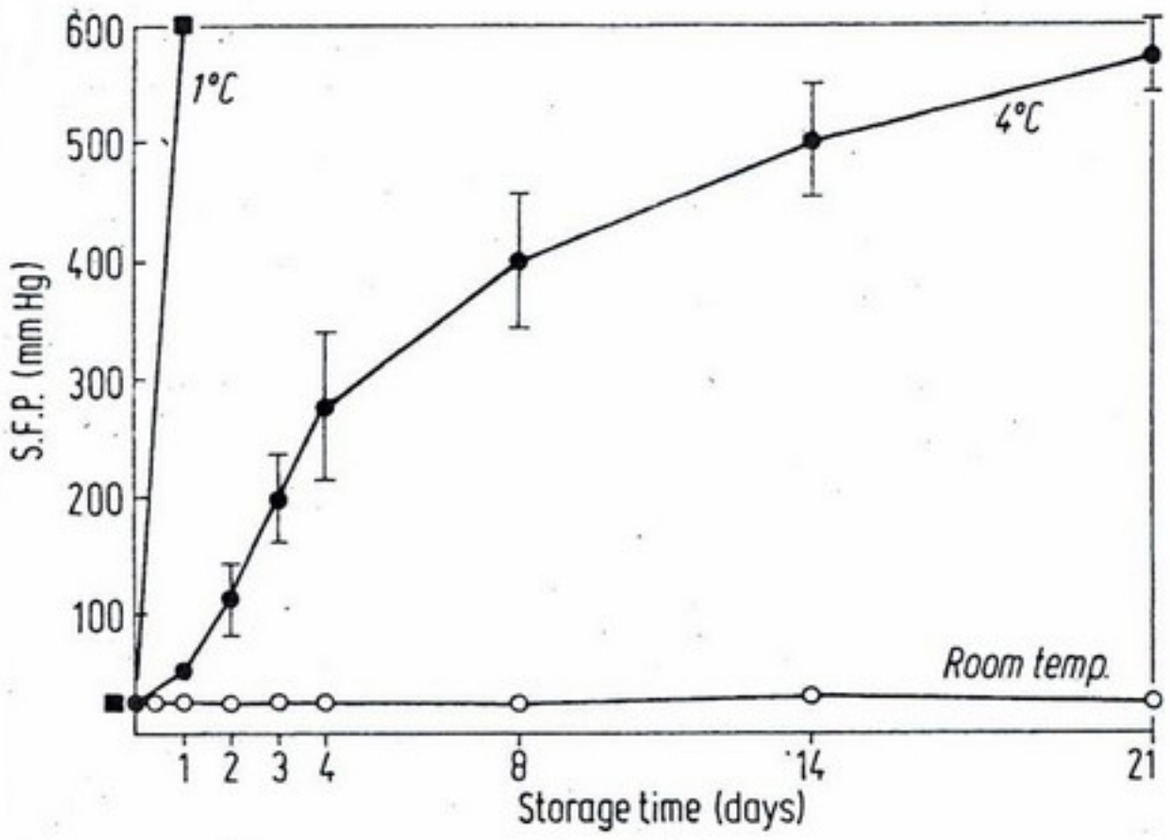

Screen filtration pressures (±S.E.M.).

usually reaching the measurable maximum between 4 and 21 days. The results were the same whether S.F.P. was measured on samples maintained at storage temperature or on samples allowed to warm to ambient temperature first.

Measurements of catecholamines, serotonin, and A.D.P. concentrations and pH suggest that the pattern of platelet release and/or degradation was similar at all three temperatures, but that it was more striking at 1°C. One explanation for the lack of aggregation at room temperature may be that, in the absence of the cold stimulus, platelet release is slower so that by the time the plasma concentrations of released A.D.P., catecholamines, and serotonin are high enough to induce platelet aggregation, the platelets are unaggregable, perhaps due to a much reduced pH or loss of viability.

With efficient microfiltration, platelet aggregation may be of little consequence for many routine transfusions of whole blood. However if cold storage is to continue, the striking difference in aggregation seen between 1° and 4°C suggests that the storage temperature should be no lower than 4°C.

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HB$_s$Ag IN RENAL DISEASE

SIR,—Renal disease has often been associated with hepatitis-B-antigen positive liver diseases. Brzosko et al.,[1] however, identified HB$_s$Ag-antibody complexes in 50% of sera and 56% of renal-biopsy samples of 32 children with immune-complex glomerulonephritis (G.N.) and/or nephrotic syndrome but without clinical liver disease.

In the past four years we have examined 276 patients with G.N., nephrotic syndrome, and other kidney diseases for the presence of HB$_s$Ag in serum and kidney. All patients were adults, 171 being males and 105 females. 7 had histories compatible with acute icteric hepatitis, 6 had received transfusions, and 6 had been treated with steroids and immunosuppressives. Only 5 patients had increased transaminases during the period of observation. We tested sera for HB$_s$Ag by passive hæmagglutination ('Hepanosticon') and for anti-HB$_s$Ag-antibody by immunoelectroosmophoresis. Kidney-biopsy tissue was studied by indirect immunofluorescence first with rabbit anti-HB$_s$Ag antiserum (Behring, Marburg) and, then with fluorescein-labelled goat anti-rabbit-7*S*-globulin (Cappel, Downingtown). Specificity was checked by blocking experiments and by simultaneous staining of a known HB$_s$Ag-positive liver section. Immunohistochemical analysis of kidney tissue was done by direct immunofluorescence using fluorescein-labelled anti-human IgG, IgM, IgA, IgE, C3, and fibrinogen (Hyland, Costa Mesa).

TABLE I—HB$_s$Ag IN SERA AND KIDNEY BIOPSIES OF 276 ADULT RENAL PATIENTS

Diagnosis	No.	HB$_s$Ag positivity in		
		Serum	Kidney	Both
Primary G.N.:				
Diffuse				
Minimal proliferative intercapillary	37	0	0	0
Endocapillary/acute/ proliferative	1	0	0	0
Mesangioproliferative	55	11	6	3
Mesangioproliferative with crescents	7	0	1	0
Membranous	17	3	4	3
Membranoproliferative	26	6	6	3
Berger disease	32	5	10	3
Focal	31	3	0	0
Focal sclerosing	9	2	1	0
G.N. associated with systemic disease:				
S.L.E.	7	0	2	0
Schönlein-Henoch	11	2	3	1
End-stage kidney	12	1	2	1
Other renal diseases				
Chronic pyelonephritis	10	1	0	0
Chronic interstitial nephritis	4	1	2	1
Secondary amyloidosis	3	0	0	0
Primary amyloidosis	2	0	0	0
Hereditary nephritis	9	1	0	0
Multiple myeloma	3	0	0	0
Total	276	36	37	15

The broken lines encompass immune-complex G.N.

Table I summarises the results. HB$_s$Ag was identified in 13·0% of sera and in 13·4% of kidney tissue. These figures were slightly higher in 196 patients with immune-complex G.N. (primary G.N. without minimal change and G.N. associated with systemic diseases), being 16·3% for serum and 16·8% for kidney, and much lower (5%) and 5%) for the patients with "other renal diseases". In a parallel study HB antigenæmia was identified in 45 (0·45%) of sera from 10 000 healthy blood-donors. Except in 1 patient, no anti-HB$_s$Ag antibody was noted in the sera. In 20 of the 32 immune-complex glomerulonephritic patients with HB$_s$ antigenæmia we re-examined sera after 6–10 months and found only 10 positive cases. In contrast to what Brzosko et al.[1] found in children, there was

TABLE II—COMPARISON OF DATA ON THE HB$_s$Ag POSITIVITY IN GLOMERULONEPHRITIS

Series	HB$_s$Ag positivity (%) in	
	Serum	Kidney
Brzosko et al.[1]*	50	56·2
Vos et al.[2]	20·3	N.D.
Reznikoff-Etievant and Lagrue[3]*	6·8	N.D.
Conte and Fournic[4]	0	31
Guardia et al.[5]	7·8	N.D.
Powell et al.[6]	14·2	N.D.
Nagy et al.	16·3	16·3

N.D.=not done
*Only cases of immune-complex glomerulonephritis were considered.

1. Brzosko, W. J., Krawczynski, K., Nazarewicz, T., Morzycka, M., Nowosawski, A. *Lancet*, 1974, ii, 477.
2. Vos, G. H., Grobbelaar, B. G., Milner, L. V. *S. Afr. med. J.* 1973, **47**, 911.
3. Reznikoff-Etievant, M. F., Lagrue, G. *Lancet*, 1971, ii, 234.
4. Conte, J. J., Fournie, G. J. *Nouv. Presse méd.* 1975, **4**, 429.
5. Guardia, J., Pedreira, J. D., Martinez-Vázquez, J. M., Vidal, M. T., Vilardell, M., Caralos, A., Ferrer, E., Bacardi, R. *ibid.* 1975, **4**, 2923.
6. Powell, K. C., Meadows, R., Anders, R., Draper, C. C., Lauer, C. *Aust. N. Z. Jl Med.* 1977, **7**, 243.

no predominance of HB_sAg positivity in our adult membranous and membranoproliferative patients (table I).

Table II compares our data on HB_sAg positivity in G.N. with those obtained by other workers. Differences in age, geographic location, methods, and immune sera used may account for the different results.

Besides HB_s antigenæmia we found renal HB_sAg positivity in some patients with immune-complex glomerulonephritis. Deposits of HB_sAg were seen in the same location as immunoglobulins and C3 suggesting that HB_sAg immune complexes may be implicated in the pathogenesis of G.N. in adults as well as children. The infection was anicteric in most of our cases. We recommend examination of the sera and kidney in all nephritic patients in order to detect HB_sAg components of immune-complex G.N.

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AGEING KIDNEY

SIR,—I read Professor McLachlan's paper (July 15, p. 143) with interest. My study in elderly women suggests that one factor which may accelerate renal ageing is bacteriuria.[1] Two groups of elderly women were compared: one group had a very high frequency of significant bacteriuria (75%) and the other had a frequency of 24%. The kidneys of the group with a 75% incidence of significant bacteriuria were radiologically shorter, weighed less at necropy, and, on histological examination, had a greater percentage of completely obsolescent glomeruli.

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SCREENING FOR REFLUX

SIR,—You conclude (July 1, p. 23) that there is, as yet, no strong case for widespread radiological screening in young children. We agree, but would like to add some comments on the radiological investigation of children with urinary-tract infection (U.T.I.).

While few people now subscribe to the view that reflux by itself damages the kidney and that the incidence of reflux warrants general population screening, most would agree that other major urological abnormalities can severely damage the kidney. U.T.I. is often the first (only) early symptom of such an abnormality, which should be detected and corrected early in life if renal function is to be preserved.[2]

In a screening survey in the neonatal intensive-care unit of Lausanne University Hospital[3] we found bacteriuria in 43 out of 1762 neonates, 36 being males. Clinical symptoms suggestive of U.T.I. were present in 34, while only 9 were symptom-free. Radiological examination of the kidneys and urinary pathways demonstrated abnormalities in 15 out of 31 patients studied. Reflux was present in 11 and major malformations in 4.

Since asymptomatic U.T.I. is rare in the newborn, and since, on the other hand, major abnormalities of the urinary tract are a threat to the morphological and functional integrity of the kidney, we concluded that routine survey of bacteriuria in symptom-free neonates was not essential, that a thorough search for symptoms suggestive of U.T.I. in neonates would be more helpful than routine screening for bacteriuria, and that all neonates presenting with proven U.T.I. should be investigated radiologically.

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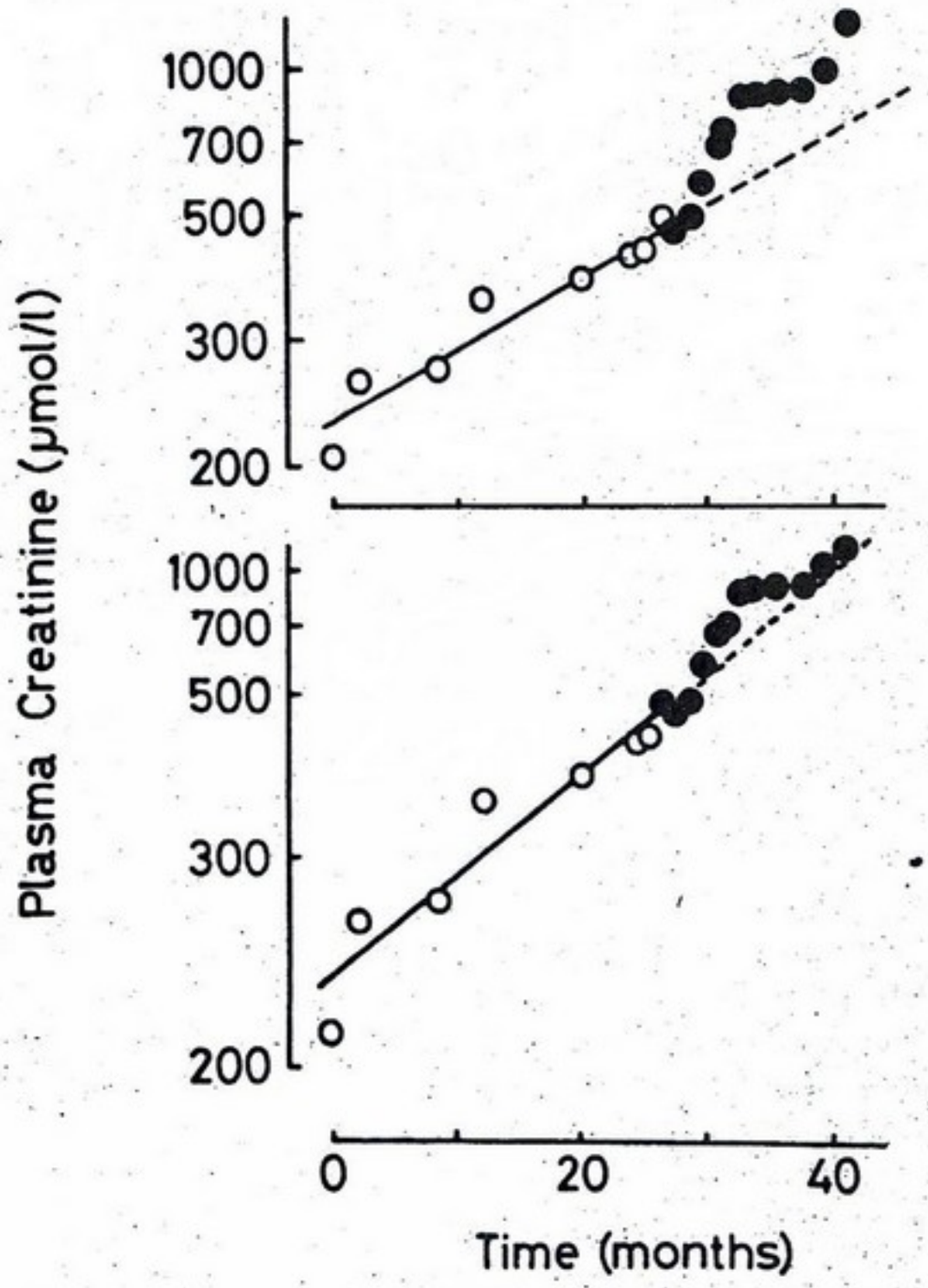
1α-HYDROXY DERIVATIVES OF VITAMIN D_3 AND RENAL FUNCTION

SIR,—Dr Winterborn and his colleagues (July 15, p. 150) suggest that administration of 1α-hydroxyvitamin D_3 (1α-OHD_3) adversely affects renal function in patients with chronic renal failure. Since this agent has now found a place in the treatment of renal bone disease, their conclusions, if correct, have important clinical implications, particularly if 1α-OHD_3 and 1,25-dihydroxycholecalciferol ($1,25[OH]_2D_3$) become widely used.

There are several problems in evaluating the effects of a drug on renal function in patients with progressive renal failure, including difficulties in judging the natural history of the kidney disease, the precision of measurements of renal function, and difficulties in separating direct effects of the drug



Serial measurements of plasma-creatinine in an adolescent with progressive renal failure due to membranoproliferative glomerulonephritis.

Plasma-creatinine is plotted against time on logarithmic (top) and exponential coordinates (bottom) before (0) and during treatment (●) with 1α-OHD_3. The upper diagram shows that the rate of change of plasma-creatinine accelerated during treatment with 1α-OHD_3. Assuming that deterioration of renal function would have continued exponentially had 1α-OHD_3 been withheld, then the onset of end-stage renal failure was apparently hastened by one year. The lower figure shows that if the rate of renal impairment is assumed to be hyperbolic, then any effect of 1α-OHD_3 on renal function is less evident. Because of the limited number of observations and the variation in plasma levels of creatinine, the natural history in this, and most patients, is difficult to predict.

1. Curtis, J. R. M.D. thesis, University of London, 1968.
2. Mayor, G., Genton, N., Torrado, A., Guignard, J.-P. *Pediatrics*, 1975, **56**, 740.
3. Maherzi, M., Guignard, J.-P., Torrado, A. *ibid.* (in the press).