

tion or evidence of damage. The dialyser assembly was then put into use in a SMAC in a routine chemical pathology laboratory and performed in a completely satisfactory manner.

The outcome of these tests confirmed that, if necessary, it is possible to decontaminate the exterior of complex equipment effectively without any detrimental effects on sensitive components. Such decontamination would only be required in the most exceptional circumstances and it is not envisaged as a routine procedure prior to maintenance.

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Isolation of *Pseudomonas fluorescens* after suprapubic catheterisation

A 64-year-old woman underwent a partial vulvectomy for malignant melanoma; a suprapubic bladder catheter was inserted in the operating theatre. The night before the operation, and for six days afterwards, she received cephradine and metronidazole. A urine sample taken on the third postoperative day revealed only large numbers of red blood cells.

By the tenth postoperative day, the patient was complaining of dysuria and although she was afebrile it was thought that she had a urinary tract infection. Microscopy of a urine sample showed large numbers of Gram-negative bacilli, small numbers of leucocytes and a moderate number of red blood cells. The organism failed to grow on Cysteine Lactose Electrolyte Deficient medium at 37°C overnight but was later found to grow at temperatures ranging from 4° to 30°C. It was identified as *Pseudomonas*

fluorescens. The same organism was isolated from further specimens of urine taken on the 15th and 17th postoperative days. Cotrimoxazole, to which the organism was resistant, was prescribed on clinical grounds from the 12th postoperative day. On the 18th day, treatment was begun with tetracycline, to which the organism was very sensitive in disc tests, and the suprapubic catheter was removed.

The patient's progress was interrupted by several days of urinary retention which necessitated the introduction of a Foley catheter *per urethram*. However, a further urine specimen taken on the 24th day after operation was sterile.

At no time were any bladder washouts given. Urine specimens were drawn by syringe from a sideport of the catheter and not from the drainage bag.

Can a psychotropic organism cause a urinary infection in these circumstances? We presume that the presence of the suprapubic catheter in a patient who was ambulant in the ward produced the lower temperature which this organism required in order to multiply. The absence of large numbers of leucocytes in the urine plus the fact that the patient was never pyrexial lead us to question the relevance of this isolate. Nevertheless, her clinicians were convinced that she was suffering from a urinary infection, and the same organism was isolated on three occasions over a period of eight days.

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Inhibition of direct binding of enzyme-conjugated antihuman IgG to C1q using dextran sulphate in solid phase assays for immune complexes

In the solid-phase C1q-binding assay for

the demonstration of circulating immune complexes the amount of immunoglobulin bound to the solid-phase C1q can be measured by radio- or enzyme-labelled anti-immunoglobulins.^{1,2} In these tests false-positive results due to non-immunoglobulin C1q-binding substances are avoided, but the solid-phase assays may be hampered by the direct binding of the anti-IgG enzyme conjugate to the solid-phase C1q, especially if the conjugate contains immunoglobulin aggregates.¹

Using purified human C1q (0.05-25 µg/ml) in a solid-phase enzyme-linked immunosorbent assay (ELISA) we noticed that the heavy-chain specific swine antihuman IgG alkaline phosphatase (ALP) conjugate used (Orion Diagnostica, Helsinki, Finland) reacted directly with the solid-phase C1q giving absorbance values in the range of 0.7-1.5 units. Various polyanions inhibit the binding of C1q to IgG complexes.³ We investigated the effect of the addition of dextran sulphate (DS) in concentrations of 100-0.01 µg/ml to the anti-IgG-ALP conjugate and found that this polyanion inhibited the direct binding of the conjugate to the solid-phase C1q (Table), whereas it did not affect antigen-antibody reactions in other ELISAs. In this modification of the solid-phase C1q-binding assay we have subsequently used a C1q-coating concentration of 1 µg/ml and have added DS at a concentration of 0.5 µg/ml to the enzyme-conjugate working dilution (1/500). In the assay aggregated human IgG (AHG) can be detected at concentrations above 2.5 µg/ml while deaggregated IgG fails to bind to the solid-phase C1q in concentrations up to 40 µg/ml. Forty-five normal blood donor sera tested in the assay gave a mean value of 33 ± 45 µg AHG eq/ml. Values above 120 µg AHG eq/ml were observed in 10/16 (62.5%) SLE patients, in 59% (26/44) of patients with rheumatoid arthritis and in 6/20 (30%) of patients with

The effect of dextran sulphate on the binding of the anti-IgG-ALP conjugate to solid-phase C1q*

Coated wells incubated with:	Anti-IgG-ALP conjugate	
	with DS (0.1 µg/ml)	without DS
20 µg AHG/ml phosphate-buffered saline-Tween	0.35†	1.15
Phosphate-buffered saline-Tween	0.01	0.79

*Coating concentration 1 µg/ml.

†Absorbance value (units).

ALP = alkaline phosphatase.

DS = dextran sulphate.

urogenital carcinoma.

We conclude that the addition of dextran sulphate to the anti-IgG enzyme conjugate provides a simple way of avoiding direct binding of commercially available enzyme-labelled anti-immunoglobulin conjugates to solid-phase C1q in ELISAs for the demonstration of immune complexes.

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IgA deficiency in Israeli blood donors

Immunoglobulin A deficiency occurs rarely, yet potentially fatal anaphylactic reactions may occur if such individuals are transfused with plasma-containing blood components from donors who are not absolutely deficient of IgA (aIgA).^{1,2}

Several national registers of IgA deficient blood donors have been established and large scale screenings to identify IgA deficient individuals have been reported.³⁻⁵

Our study was based on the application of a latex agglutination inhibition test (LAI) for the determination of the frequency of IgA deficiency in the Israeli population. The primary advantages of LAI, compared with the conventional radial immunodiffusion (RID), are rapidity and the ability to perform individual tests as required.

Sera were obtained from 4075 healthy Israeli blood donors, comprising 2192 Ashkenazi Jews originating from European communities and 1883 Sephardi Jews originating from Mediterranean or Middle Eastern communities. Known IgA deficient sera were obtained from 10

immunodeficient patients and from 32 newborns.⁶

Two test methods were used: (i) LAI—based on the inhibition of agglutination of (human) IgA-latex spheres by goat anti-IgA when an IgA-containing serum or other biological fluid is added. Portions (2.0 ml) of standardised goat antihuman IgA serum were mixed mechanically with 20 µl of the serum to be tested, then incubated at 37°C for 10 min. Subsequently, 100 µl of the IgA-latex conjugate were added and after brief mechanical mixing the test-tube was reincubated at 37°C for 90 min in dry heating blocks. Sera with IgA concentrations below 80 µg/ml do not inhibit the agglutination of the IgA-coated latex spheres and the end point of the reaction is a clear solution with white flocculates and sedimented latex spheres. (ii) RID—1803 (44.2%) of the 4075 blood donor sera were tested in duplicate by conventional radial immunodiffusion.^{7,8}

Of the donors screened, only 0.09% (4/4075) were found to be IgA-deficient (<80 µg/ml) by means of the LAI, while 2.7% (49/1803) were deficient by the RID method. All cases which turned out to have low level IgA as detected by the regular RID tests were retested in low level RID plates; only one serum sample was found to contain <40 µg/ml. The 42 known IgA-deficient sera samples were used as controls. The frequency of IgA deficiency was found to be markedly lower among Sephardi Jews—0.053% (1/1883)—than among those of Ashkenazi origin—0.13% (3/2192). In both ethnic groups, the higher sensitivity of the LAI as compared to the RID test was evident.

An attempt was made to relate the frequency of low IgA concentrations to the age of blood donors tested. The highest frequency of IgA deficiency was observed in the 17-19 yr age group (7.7%). However, all the four cases of complete IgA deficiency as determined by the LAI tests occurred in the 30-39 yr age group.

The immediate onset of a severe transfusion reaction—which cannot be ascribed to red cell incompatibility—may be due to the response to drugs in the transfused blood.⁹⁻¹¹ Another possible cause for immediate reaction is the transfusion of IgA-containing blood or blood components to an IgA-deficient patient; only a few millilitres of blood or plasma may be sufficient to evoke urticaria, throbbing headache, increased

warmth and flush, severe prostration, chest pains and shortness of breath.

In 1968 Fudenberg *et al*¹² first reported the presence of anti-IgA in human sera, applying the haemagglutination inhibition method.¹³ Anti-IgA occurs in subjects with a selective absence of IgA.¹⁴ In such subjects, anti-IgA may occur without a previous history of transfusion. In contrast to Fudenberg's method, a direct approach for the determination of IgA deficiency was used in this study. Both the radial immunodiffusion and latex agglutination tests provide a far more sensitive method for the detection of IgA deficiency, thus making the search for the cause of a transfusion reaction simple and reliable.

Total IgA deficiency (<80 µg/ml) was found in four of 4075 donors (1:1000). This frequency is far lower than that of 1:360 or 1:700 as estimated by Collins Williams¹⁵ and Bachman¹⁶ respectively. The incidence of IgA deficiency seems to be even lower among Sephardi Jews (1:1800). Nevertheless, once the presence of allergy-causing drugs in the transfused blood has been eliminated, the possibility that immediate transfusion reaction results from IgA deficiency in the patient must be investigated.

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