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 apyrexial 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 psychrotropic 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.

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