Letters to the Editor

Decontamination of automated laboratory equipment

In the Howie Code of Practice (1978)\(^5\) paragraph 25(a) lays down procedures for the routine decontamination of the parts of automated equipment used in chemical pathology laboratories that are in direct contact with samples. These procedures, involving the washing of liquid lines with either water or the manufacturer’s wash fluid are considered adequate for dealing with infectious agents in normal routine work. When dialyser membranes have to be changed the system should be washed with a solution of strong hypochlorite followed by a water wash.

Recently we have been asked to advise on a suitable procedure for dealing with microbial contamination that might occur in a laboratory housing large expensive automated equipment such as that found in many chemical pathology laboratories. Although this is an unlikely event, it might, depending on the nature of the micro-organism, or the level of contamination, call for the decontamination of the exterior of the equipment as well as decontamination of the liquid lines. Liquid lines can be dealt with by the use of a suitable disinfectant such as strong hypochlorite. The exterior of the equipment can be decontaminated by sealing the laboratory and then decontaminating the room and its contents with formaldehyde. This is a well established procedure for space decontamination. More limited decontamination could be carried out by ad hoc arrangements such as enclosing the equipment within a polythene film tent fabricated on site using polythene sheeting and adhesive tape and generating formaldehyde within the tent.

The point of interest here is whether exposure to formaldehyde would have any detrimental effect on components of the equipment such as electrodes, electric circuits etc necessitating expensive refurbishing after decontamination. Experience in the Microbiological Safety Reference Laboratory in which many pieces of equipment such as an automatic image analyser, cameras, microscopes and electronic control boxes have been repeatedly exposed to formaldehyde have not shown any detrimental effects from this treatment. An example of an item of equipment that might be considered vulnerable to treatment with formaldehyde, a cartridge, including dialyser assembly, from a Technicon SMAC (Sequential Multiple Analyzer with Computer) has been subjected to tests.

The SMAC cartridge was placed inside a sealed chamber (16 cubic feet) and 10 ml of formalin solution (40% wt/vol formaldehyde) plus 10 ml of water evaporised by heat. The chamber was opened after about 15 h and aired for 3 h to remove residual formaldehyde. This procedure was carried out on four occasions. On the first occasion, two flat surfaces of the cartridge were heavily contaminated with about $2 \times 10^7$ spores of Bacillus globigii suspended in either water or neat horse serum. These inocula were allowed to dry before exposure to the formaldehyde. The surfaces were sampled after one cycle of decontamination. After fumigation no viable spores were recovered from either of the deliberately contaminated surfaces demonstrating that the exposure to formaldehyde was effective for sterilising surfaces heavily contaminated with a resistant micro-organism.

Visual inspection of the SMAC cartridge after four cycles of decontamination showed no obvious changes, discolora-
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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References


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 cephadine 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 Clq using dextran sulphate in solid phase assays for immune complexes

In the solid-phase Clq-binding assay for the demonstration of circulating immune complexes the amount of immunoglobulin bound to the solid-phase Clq can be measured by radio- or enzyme-labelled anti-immunoglobulins.1 In these tests false-positive results due to non-immunoglobulin Clq-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 Clq, especially if the conjugate contains immunoglobulin aggregates.

Using purified human Clq (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 Clq giving absorbance values in the range of 0-7-1-5 units. Various polyanions inhibit the binding of Clq to IgG complexes.2 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 Clq (Table), whereas it did not affect antigen-antibody reactions in other ELISAs. In this modification of the solid-phase Clq-binding assay we have subsequently used a Clq-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 degaggereated IgG fails to bind to the solid-phase Clq 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 demonstration of circulating immune complexes the amount of immunoglobulin bound to the solid-phase CIq can be measured by radio- or enzyme-labelled anti-immunoglobulins.1 In these tests false-positive results due to non-immunoglobulin Clq-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 Clq, especially if the conjugate contains immunoglobulin aggregates.

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