

Letters to the Editor

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Needleless connectors—the way forward in the prevention of catheter-related infections?

Sir,

Intravascular catheters have become integral in patient management. Indeed, in the UK, 200 000 central venous catheters (CVC) are used annually.¹ However, indwelling intravascular devices are also associated with many complications including infection. Micro-organisms, in particular coagulase-negative staphylococci (CNS), originating from the skin of healthcare workers or patients may gain access to intravascular catheters via contamination of hubs and stopcock entry ports. The micro-organisms migrate to the catheter distal tip resulting in colonization and subsequent infection. In a recent study, 22% of stopcock entry ports and 31% of arterial line hubs were contaminated with micro-organisms within 72 h of placement.² In an attempt to reduce hub colonization, daily disinfection with 70% ethanol and the use of a povidone-iodine gauze hub cover have been employed.^{3,4} Needleless connectors have recently been introduced into clinical practice to reduce the risk of needlestick injuries. These devices may also facilitate aseptic technique and reduce the time spent on manipulation of intravenous connections. The potential infection risk of needleless connectors is, at present, unclear with studies yielding conflicting results.^{5,6}

We have investigated the external and internal microbial contamination rates of two types of needleless connector—Clave[®] and Posiflow[®] (Becton Dickinson, Utah, USA)—in a prospective randomized study (Figure 1). Thirty-eight patients from the cardiac and haematology departments at the University Hospital Birmingham NHS Trust who required a CVC as part of their clinical management were recruited into the study and assigned to have either the Clave or Posiflow attached to the hubs/stopcocks of their CVC. The needleless connectors were disinfected with chlorhexidine gluconate 0.5% w/v in industrial methylated spirit BP 70% w/w spray (Adams Healthcare, Leeds, UK) prior to and following each manipulation. All administrations and

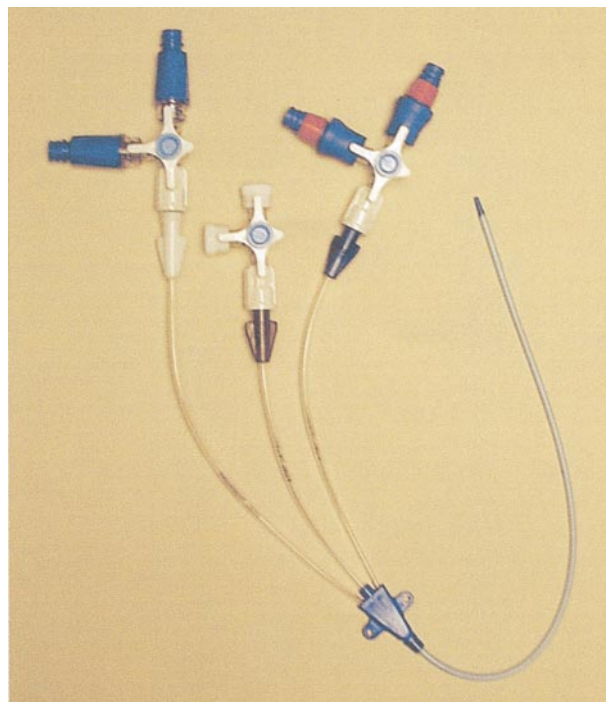


Figure 1 Triple lumen central venous catheter complete with 3-way taps and needleless connectors. From left to right; Posiflow[®], standard luer caps and Clave[®].

aspirations made through the connectors 72 h post CVC insertion were recorded. Each device remained in situ attached to a CVC for 72 h after which they were removed and investigated for microbial contamination. Two hundred and fifty-five needleless connectors (128 Clave, 127 Posiflow) were studied. The external surface of the silicone compression seals were pressed onto 7% blood agar (Oxoid, Basingstoke, UK) 10 times after which the plates were incubated at 37°C in air for 48 h. The silicone compression seals were then cleaned with a 70% v/v isopropyl alcohol swab (Sterets[®], Seton Prebble Ltd, Merseyside, UK). The internal microbial contamination rate of the needleless connectors was assessed by flushing with 100 µL of brain–heart infusion broth (Oxoid, Basingstoke, UK) which was inoculated on to 7% blood agar (Oxoid) and incubated in air at 37°C for 48 h. The external and internal microbial contamination rates of the Clave and Posiflow were compared by Fisher's exact test.

There was no significant difference between the rate of external microbial contamination of the Posiflow (28.3%) and Clave (39%) ($P=0.085$). Skin and environmental micro-organisms were recovered from the external silicone compression seal of the connectors including CNS (91.9%), environmental Gram-negative rods (8.1%), *Staphylococcus aureus* (4.7%) and Gram-positive rods (predominantly *Corynebacterium* spp.) (3.5%). There was also no significant difference between the internal microbial contamination rates of the devices, Posiflow (5.5%) versus Clave (4%) ($P=0.571$). Micro-organisms recovered from the internal cultures of the connectors included CNS (91.7%), *Staph. aureus* (8.3%) and Gram-positive Rods (predominantly *Corynebacterium* spp.) (8.3%). All of the Clave devices with internal microbial contamination also had external contamination, however identical micro-organisms were not always recovered. In comparison, 57.1% of Posiflow connectors were both internally and externally contaminated. The mean number of manipulations per connector for each type of device during the 72 h period was 1.6 (range=0 to 17). There was no correlation between the number of manipulations and the presence of internal microbial contamination ($P=0.5$, Spearman Rank Test) and no correlation between the number of manipulations and the extent of internal microbial contamination ($P=0.5$, Spearman Rank Test). Seventy-five percent of the devices with micro-organisms present internally were used for continuous infusions or for central venous pressure (CVP) monitoring, which would have resulted in the connectors being continually in use during the study period. Drugs and fluids including propofol, adrenaline, dopamine, 5% dextrose and blood were administered through the contaminated devices and may, therefore, have supported microbial growth.⁷

The number of needleless connectors through which each drug was administered was similar for both types of device. Although the average internal contamination rate of the devices was 4.8%, this was significantly lower than the contamination rates of open ports reported in a previous study² which suggests that the devices, if cleaned appropriately, may reduce contamination and subsequent infection rates. Furthermore, the Posiflow device has the added feature of maintaining positive pressure in the catheter. Therefore, when the syringe is removed, backflow of fluid up the catheter is prevented, maintaining catheter patency.

The microbial contamination rate associated with needleless connectors may be further reduced if the devices are used in conjunction with pre-filled syringes.⁸ The reduced levels of microbial contamination associated with pre-filled syringes and needleless connectors compared with conventional systems is encouraging, and the use of these devices may be the way forward in preventing sepsis associated with intravascular catheters.

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An outbreak of vancomycin-resistant enterococci associated with major ward refurbishment

Sir,

During 1998, an outbreak of infection caused by vancomycin-resistant enterococci (VRE) occurred on a renal unit, following major ward refurbishment. In January 1998, patients were relocated temporarily to allow the refurbishment, which included re-siting of the sluice and toilets, installation of new handwash basins and showers, and the provision of a new bathroom. The refurbished ward was re-opened over the last weekend in May 1998, but members of the infection control team (ICT) were unable to inspect the ward because this took place over a Bank Holiday weekend, and access had been restricted on the preceding Friday due to ongoing work. When patients were moved back, however, some work was still in progress.

Whilst on the temporary ward, just before the move back to the refurbished unit, VRE were cultured from peritoneal dialysis effluent in one patient with peritonitis and from the urine of another. In early June, VRE were isolated from two more patients on the refurbished ward and an outbreak control group was formed.

Several major problems were identified on the refurbished ward. Handwashing facilities were poor: several new handwash basins were not plumbed in and others had no taps. At functioning handwash basins, no liquid soap was available because new dispensers, which could not accept the soap cartridges used throughout the remainder of the hospital, had been fitted. At some basins, paper towel dispensers had not been fitted and paper towels were held on the rim of basins where they could be easily contaminated by splashing during hand washing. The ward cleaners' room was undergoing conversion to an office (not included in the original plans), necessitating removal of cleaning equipment to the sluice, where it was stored next to the bedpan washer. Floor polisher buffer pads were being cleaned in the sluice with a toilet brush.

Infected patients were isolated, and non-functional handwash basins were reinstated. Soap compatible with the new dispensers was ordered urgently and alcohol hand rub was provided in the interim. An intensive ward clean was initiated using neutral detergent and water, followed by a 1000 ppm aqueous hypochlorite solution. The cleaners' room was reinstated. Use of vancomycin as first-line therapy for *Clostridium difficile* infection and for empiric therapy for suspected vascular access device-associated infection was prohibited.

Environmental screening for VRE on the refurbished ward, the maintenance haemodialysis unit and temporary ward, and screening for faecal carriage of VRE in renal inpatients was instituted. Isolates were typed using pulsed-field gel electrophoresis (PFGE) of *Sma*I-digested DNA.¹

All outbreak isolates were identified as *E. faecium* and displayed the VanA phenotype. The isolate from the presumed index case with CAPD peritonitis yielded a PFGE banding pattern designated 'A'. A blood culture from a patient with haematological malignancy and peritoneal fluid from another episode of CAPD peritonitis also yielded isolates which were 'closely related' and 'possibly related' to outbreak strain A, respectively using the criteria of Tenover *et al.*² Strain A was not recovered from any environmental swabs. In the second chronological case, VRE was cultured from multiple ulcer swabs, multiple superficial sites and faeces as well as urine. The PFGE banding pattern of this strain differed from that of the index case and was designated 'B'. This was isolated subsequently from the blood and pleural fluid, respectively, of two additional patients.

Of the 51 environmental sites sampled initially on the refurbished ward, 13 (25%) were positive for VRE (strain B only). Only one site was positive on the haemodialysis unit and none on the temporary ward. Screening for faecal carriage of VRE among acute renal inpatients identified two further carriers who had not previously been detected by routinely obtained clinical samples.

Before the outbreak described in this report, no faecal carriers of VRE had been identified among 19/70 CAPD patients and 91/120 maintenance haemodialysis patients. However, not all of these patients in this incomplete screening exercise had been in recent contact with the ward in which the outbreak occurred. Nevertheless, the apparent lack of faecal colonization and the rarity of VRE from samples obtained for routine diagnostic purposes,