


Prevention of infection after vascular reconstruction

Wound infection after vascular reconstruction may cause considerable delay in hospital discharge and therefore be expensive in resources (Johnson et al., 1988). However, the real hazard is that the infection may involve prosthetic material. Graft infection is
hazardous both to life and limb with amputation rates of 17–79% and mortality of 17–26% recently reported (Lorenzen et al., 1985; Edwards et al., 1987; Kikta et al., 1987). Graft infection follows 1–6% of vascular reconstructions and may become apparent months or even years after initial surgery (Liekweg & Greenfield, 1977). Late graft sepsis may follow prolonged infection with an organism of low virulence implanted at or near the time of surgery or alternatively may be due to seeding following bacteraemia. Neointimal formation is unusual in prosthetic grafts which may explain this susceptibility to bacterial seeding. Early graft infection within a month of surgery often follows local wound sepsis (Moore, 1982). Prevention of wound sepsis may be the most important way of reducing graft infection.

The origin of vascular sepsis is widely believed to be endogenous implantation at or soon after, surgery of pathogenic organisms present on the skin of vascular patients. Although vascular surgery is classified as 'clean surgery', pathogenic bacteria can frequently be isolated from the skin, particularly in patients with ulcers or gangrene (Eamshaw et al., 1988). The proximity of the anogenital region to vascular incisions increases the risk of endogenous infection and may be one explanation why the groin wound is most frequently affected. An alternative theory, that infection arises in the lymphatics of limbs with distal skin sepsis, has not been supported by clinical studies (Weaver, Chatterpadhay & Angel, 1973; Eamshaw et al., 1989).

Reported rates of wound infection after vascular reconstruction vary widely, from 1 to 22%. In clinical studies wound infection is most frequently associated with distal skin necrosis (Liekweg & Greenfield, 1977). This may explain the wide variation in reported frequency, some studies even including patients having varicose vein surgery (Pitt et al., 1980). Uniformity of reporting has been a problem, but Szilagyi et al. (1972) introduced a grading system of vascular infection which has been used to standardize many series.

Though the elderly, malnourished and diabetic patients have shown to have an increased susceptibility to infection (Edwards et al., 1987), deficient surgical technique is probably the most vital factor in the aetiology of vascular sepsis. Hazards of surgery associated with increased infection rates include undercutting skin edges with subsequent necrosis, haematoma or seroma formation (particularly if heparin is used), damage to lymphatics particularly in the groin, long leg incisions in femoro-popliteal bypass, inaccurate skin apposition, reoperative surgery and bypass graft thrombosis (Goldstone & Moore, 1974; Hasselgren et al., 1984; Lorenzen et al., 1985; Edwards et al., 1987). Not only are the hazards of infection greater in patients with prosthetic grafts but the artificial material itself may enhance bacterial infectivity (Johnson et al., 1988).

Just as the aetiology of infection is multifactorial, there is no single solution to the prevention of vascular surgical sepsis. If the major cause is endogenous infection then reducing the number of skin pathogens should be beneficial. Though many surgeons use pre-operative antiseptic regimens empirically, the clinical evidence of benefit is sparse. In a single Swedish study, pre-operative chlorhexidine baths reduced the rate of infection from 17.5% to 8% in vascular patients (Holm, 1985), though this benefit was not confirmed in a recent study from the UK (Eamshaw et al., 1989). The best antiseptic agents and regimens for use both before surgery, and for skin cleansing at operation remain to be determined, though there is some evidence from a retrospective study that iodine-based agents may be superior (Kaiser et al., 1978). In future it will be important to establish whether the regimens actually reduce skin colonization prior to surgery. Daily application of antiseptic cream to post-operative wounds is also worthy of further study.

Parenteral prophylactic antibiotic therapy has been shown in randomized trials to reduce the frequency of wound, though not of graft, infection, whether cephalolin (Kaiser et al., 1978), cefotaxime (Salzmann, 1983), cefuroxime (Hasselgren et al., 1984) or netilmicin plus methicillin (Worning et al., 1986) is used. It has been demonstrated that prolonged courses of antibiotics are unnecessary and may even encourage proliferation of resistant organisms (Hasselgren et al., 1984; Eamshaw et al., 1988). However, a broad spectrum agent is required to cover the wide range of skin pathogens present on vascular patients and the use of an anti-staphylococcal agent such as flucloxacillin alone is unacceptable. Current recommendations are that four peri-operative injections of cephalolin 1 g are adequate (Kaiser, 1986), although amoxycillin-clavulanate has also been shown to be suitable (Eamshaw et al., 1987). Continuation of therapy until all central venous lines and catheters are removed may be appropriate,
particularly in aortic surgery, although prolonged cephalosporin administration may encourage colonization by resistant bacteria, such as Pseudomonas aeruginosa. It is unlikely that the direct effect of peri-operative antibiotic prophylaxis on graft sepsis will ever be known as studies would require thousands of patients followed for many years to identify late infections. Antibiotic prophylaxis has been clearly shown to reduce wound sepsis, a major risk factor for graft infection, therefore it is logical to expect that prophylaxis is also beneficial in preventing graft infection, an assumption well supported by animal experiments (Moore, Rosson & Hall, 1971). The evidence favours mandatory prophylaxis for patients receiving prosthetic grafts and for the same reason control groups are no longer ethical in studies of these patients. Antibiotic prophylaxis may be less vital where autogenous vein is used (Walker et al., 1984).

Other methods shown to prevent vascular wound infections include direct peri-operative installation of kanamycin, neomycin or cephalothin solutions (Lord, Rossi & Daliana, 1977), although, in a randomized study, local cephadrine instillation was not superior to parenteral administration (Pitt et al., 1980). Currently it is accepted that autogenous vein is the graft of choice wherever possible, although in the future antibiotic bonded synthetic grafts may become standard issue in vascular reconstruction when vein is not suitable.

Finally patients with prosthetic grafts in situ should be considered ‘at risk’ from bacteraemia for life and receive prompt treatment of any infections and antibiotic prophylaxis for invasive procedures in a similar manner to those with heart valve replacements (Working party of the British Society for Antimicrobial Chemotherapy, 1982). Skin staphylococci and faecal coliforms are the organisms most frequently isolated from vascular graft infections, though it is not known whether this pattern is similar for infections following bacteraemia. Therefore a broad spectrum antibiotic should be employed, and a single oral dose of amoxycillin/clavulanic acid (750 mg) would be a logical choice.

In conclusion, although a short peri-operative course of prophylactic antibiotic is mandatory in patients receiving prosthetic vascular grafts, careful surgical technique is probably a more important determinant of outcome.

References


