The Future of Vascular Access: Will the Benefits Be Worth the Risk?

Marcia Ryder

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Editorial

The Future of Vascular Access: Will the Benefits Be Worth the Risk?

Marcia Ryder, MS, BSN, RN
Department of Physiological Nursing, University of California, San Francisco

The introduction of the central venous catheter (CVC) has had a significant role in shaping modern medicine as we know it today. Complex medical and surgical interventions, such as parenteral nutrition, cancer therapies, hemodynamic monitoring, hemodialysis, bone-marrow and organ transplantations, and abdominal, cardiothoracic, and trauma surgery, would not be possible without the use of CVCs. Intravascular catheterization is the most common invasive procedure performed today. More than 150 million intravascular catheters, 5 million of which are CVCs and pulmonary artery catheters, are purchased annually in the United States.[1] Continued increase in the use of these devices is reflected in the American market for intravascular catheters, which is currently at $840 million and estimated to reach $1.4 billion by 2003.[2]

Unfortunately, these devices are not without significant risk of morbidity and mortality among all patient populations and age groups. Despite almost 50 years of experience with vascular catheters, practitioners continue to struggle with methods to prevent, diagnose, and treat complications associated with these devices. In a recent article, Collin et al listed 38 potential complications of CVCs, and more could be added.[3] Catheter-related infection, thrombosis, and catheter malposition are interrelated common events that often precipitate additional complications. Arnow et al found that catheter-related bloodstream infection (CRBSI) was accompanied by other major complications such as septic shock, suppurative phlebitis, metastatic infection, endocarditis, or arteritis in 32% of 102 reported cases.[4]

Infection and thrombophlebitis were recognized as major complications of venous catheterization from the earliest reports in the 1950s. However, surveillance of nosocomial infections in the United States did not begin until the 1970s. The scope of this problem was realized when reports from the Centers for Disease Control and Prevention (CDC), from 1980 to 1989, indicated that the overall bloodstream infection (BSI) rate in the United States increased by 70% at large, teaching hospitals and by 279% at small, nonteaching hospitals,[5] 90% of which were associated with intravascular devices in intensive care units (ICUs).[6] The increased incidence of BSIs was concomitant with the dramatic increase in the use of short- and long-term CVCs over this time period.

Currently, the CDC estimates that 200,000 hospital patients contract BSIs each year in the United States, with CVCs now accounting for about 90% of these BSIs.[7] On average, CRBSIs prolong hospital stays by 6.5 days in ICU at a cost of $29,000 per infection.[8] National Nosocomial Infection Surveillance (NNIS) data collected from January 1990 to April 1995 indicated that CRBSI rates (per 1000 catheter days) at hospital ICUs range from 4.9 in medical–surgical ICUs to 15.6 in burn ICUs.[9] In the most recent report, NNIS data from 1992 to 1998 indicated the range to be from 4.5 in medical–surgical ICUs to 12.8 in burn ICUs.[10] Although it is difficult to evaluate overlapping data, this comparison suggests that the incidence of CRBSI in adult, pediatric, and neonatal ICUs has slightly improved or at least remained relatively stable, albeit high, over the last 8 years.

Few data regarding nosocomial BSI rates exist in developing countries; however, European and US studies indicate that the risk of nosocomial BSIs is increased significantly in patients with CVCs (odds ratio, 4.6; 95% confidence interval [CI] range, 3.1–6.8).[11] The global increase in the use of CVCs has not only increased the incidence of BSIs but has dramatically influenced pathogen prevalence throughout the world. Until the 1970s, reports of *coagulase-negative staphylococci* (*CNS*) BSIs in the United States were very low, because they were almost exclusively regarded as skin contaminants. According to NNIS data, between 1980 and 1989 a 754% increase in the incidence of bacteremia that was due to *CNS* and *staphylococcus aureus* (176%) was noted at large, academic, medical centers in the

Correspondence and reprint requests: Marcia Ryder, MS, BSN, RN, Department of Physiological Nursing, University of California, San Francisco, 1504 Forge Road, San Mateo, CA 94402. Electronic mail may be sent to ryder1234@aol.com.

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United States.[5] *Candida albicans* BSIs increased as much as 487% over the same time period. The coexistence of a pathogen population with an ever-increasing resistance to many antibiotics, a patient population characterized by increasingly complex clinical problems, and the concomitant increase in the use of CVCs contributed to the increase in gram-positive bacteremias and fungemias.[12] *CNS*s now represent 32.2% of all primary BSIs in ICUs and are associated with 30% to 60% of CRBSIs.[12] Among the nosocomial bacteremias caused by *CNS* strains, 74% to 92% are *S. epidermidis*.[13] The trend of *CNS* bacteremia ($r = .54$, $p = .0014$) continues to increase, measured both as a proportion of BSI and as an incidence. *Staph aureus* represents 16.6% of primary BSIs and yeast represents 8.1%.[14]

Overall, the associated mortality rate for BSIs in the United States is 35%, involving up to 50,000 patients per year.[8,15] As of 1994, the number of deaths per year from intravascular catheter–related infections in Australia was greater than the number of deaths per year from acquired immunodeficiency syndrome.[16] The risk of death is dependent on the infecting organism. Mortality rates associated with (1) *CNS* bloodstream infection vary between 4.9% and 28%;[14] (2) *S. aureus,* 16% to 43%;[17] and (3) *candidemia,* 50% to 80%, with attributable mortality as high as 38%.[18]

Data reported by the NNIS system measure nosocomial infection rates in ICUs only. As many as 500,000 tunneled and implanted catheters are used per year in the United States on an inpatient and outpatient basis, primarily for cancer therapies and parenteral nutrition.[19] No national database currently exists to document catheter-related infection rates in long-term patients. Most recent studies report a mean infection rate of approximately 2.0 per 1000 catheter days,[20] a much lower rate than that for the short-term catheters used in ICU patients. This suggests a lower risk with the use of long-term catheters. However, a review of four recent studies of adult cancer patients with tunneled and implanted catheters indicated that between 19% and 42% of catheters were removed because of a complication.[21–24] The percentage of catheters removed because of CRBSIs ranged from 13% to 33%, and from 19% to 27% because of local infection. Interestingly, the percentage of catheters removed because of thrombotic events was of a similar range: 1% to 34% removed because of catheter occlusion and from 3% to 25% removed because of catheter-related thrombosis. Moukarzel et al found even higher complication rates in the University of California, Los Angeles, home pediatric total parenteral nutrition (TPN) population, with 78% of catheters removed because of complications, 45% with CRBSI, 17% with local infection, and 24% to catheter occlusion (venous thrombosis rates were not reported).[25]

Tolar and Gould examined the timing and sequence of complications in 356 tunneled Groshong catheters.[26] Overall, 397 complications were identified in 62% of catheters, and 31% of those experienced two or more sequential complications. "Ball-valve effect," defined as continued inability to aspirate blood after urokinase instillation, was the most frequently identified complication (30% of events) and occurred the earliest and most repeatedly in the life of the catheter. Catheter-related sepsis and catheter-related venous thrombosis (CRVT) were the second and third most common complications, at 19.4% and 16.6%, respectively. CRVT was the most frequent secondary complication and was followed by catheter sepsis. This study and others have established a causal relationship between catheter-related infection and thrombosis.[23,27–29]

CRVT and thrombotic intraluminal occlusions compose 25% to 40% of all catheter-related complications.[30] The rate of thrombotic events is reported to be as high as 32.5 per 1000 catheter days.[32] Approximately 57% of persistent access failures are related to thrombus in or around the catheter tip.[31] Symptomatic CRVT occurs in only about 6% to 10% of patients.[30,33] When asymptomatic patients were studied by venogram, CRVT was found in 66% of patients with both short- and long-term catheters.[33–35] Andrew et al found a 75% incidence of CRVT in a cross-sectional study of pediatric home TPN patients.[36]

Complications of upper extremity CRVT are similar to those of lower extremity deep vein thrombosis (DVT): pulmonary embolism (PE), postthrombotic syndrome, and venous gangrene (rare).[37] The rate of PE has been reported to be as high as 36% in patients with CVC[38] and 59% in pulmonary artery catheters.[38] Hingorani et al found that after 6 months of follow-up, patients with upper extremity DVT had a much higher rate of PE than did patients with lower extremity DVT (17% *vs* 8%, respectively).[40] Another surprising finding was that pulmonary embolisms that were due to upper extremity DVT conferred a higher rate of death than those that were due to lower extremity DVT (48% *vs* 13%, respectively). Compared with catheter-related infections, the morbidity and mortality associated with catheter-related thrombotic events have not been well appreciated, even though they occur with greater frequency.

The issue of risk in the use of CVCs changes perspective when combining the risks of catheter-related infection with catheter-related thrombosis. However, there is an even greater evolving crisis to consider. In the past 15 years, practitioners have seen the emergence of gram-positive microorganisms, not only as major nosocomial pathogens but with a corresponding increase in their antibiotic resistance. Approximately 25% of the *S. aureus* bloodstream isolates in the United States today are resistant to methicillin (MRSA).[12] The incidence of methicillin resistance among *CNS* has also dramatically increased. Methicillin resistance among clini-

cal isolates of *CNS* (MRCNS) increased from 20% to 60% between 1980 and 1989.[5] In a 1995–1996 SCOPE survey, 79% of *CNS* strains were found to be resistant to methicillin.[41] The majority of MRSA and MRCNS strains are cross-resistant to all beta lactams, macrolides, lincosamides, tetracyclines, aminoglycocides, and now quinolones.[12]

Vancomycin has effectively treated infections caused by gram-positive resistant organisms since the early 1980s. During the last two years, *S. aureus* strains with reduced susceptibility to vancomycin have been recovered from patients in Japan and in three patients in the United States.[42] Five cases of reduced or complete resistance of *S. epidermidis* to vancomycin, one from the United Kingdom and four from the Slovak Republic, have been reported over the last several years. The case in the United Kingdom involved peritonitis associated with a peritoneal dialysis catheter. In the other four patients, CVCs, neutropenia, prophylaxis with ofloxacin, and prior vancomycin therapy were common antecedents.[42] The first case of BSI associated with an *S. epidermidis* strain with decreased susceptibility to vancomycin has now been reported in the United States. Prolonged exposure to vancomycin and the presence of a Groshong CVC were predisposing factors in this patient.[13]

Over the last 10 years, many new compounds with activity against drug-resistant, gram-positive bacteria have been investigated. The most promising of these drugs appear to be the parenteral streptogramins, quinupristin/dalfopristin (Synercid, Rhône-Poulene-Rorer, Collegeville, PA), the oxazolidinone linezolid, and a new topical antibiotic, MBI 226.[8,43] In phase III clinical trials, Synercid has been comparable in efficacy with vancomycin for catheter-related bacteremia caused by *S. aureus* and *S. epidermidis*.[44] On the basis of its activity *in vitro* and clinically, Synercid has been submitted to the Food and Drug Administration for approval.[45] Synercid's parent compound, pristinamycin, has been used in Europe for several years with no significant increase in resistance over time;[46] however, recent reports of resistance to streptogramins of *Enterococcus faecium* and *staphylococci* are of concern.[45] Even chlorhexidine, an antiseptic shown to be superior in prevention of catheter-related infections, has demonstrated low-level, plasma-mediated resistance in antibiotic-resistant strains of *S. aureus* and *S. epidermidis*; however, the clinical relevance of this is in question.[47–50]

Increases in the rate of antimicrobial resistance will continue to result in the use of much more expensive drugs, more prolonged hospitalizations, higher death rates, and larger increases in health care costs. Yearly expenditures incurred from drug resistance in the United States are estimated to approach $4 billion and are rising.[51] The emergence of *S. aureus* and *S. epidermidis* vancomycin–resistant strains is a considerable threat to patients with CVCs. The prospect of developing an untreatable staphylococcal infection could adversely tilt the risk-to-benefit ratio for many moderate- to high-risk therapies that have become routine standards of care.[12]

The pathogenesis of catheter-related infection and thrombosis is a highly complex, multifactorial, physiologic response to the presence of a foreign body in biologic tissue. The future of VADs lies in the understanding of these complex physiologic events, the modification of catheter surfaces to interrupt the processes of the host–biomaterial response, and the ability to outwit the antibiotic-resistant capacity of both bloodborne and biofilm embedded bacteria. The time from development of new drugs to market takes, on average, 8 to 10 years. The new science of biomaterial surface modification holds much promise but is evolving slowly. Antibiotic- and antiseptic-coated catheters have already demonstrated the ability to significantly decrease the risk of catheter-related infections in short-term catheters.[52,53] This capacity may be greatly enhanced by the development of antithrombogenic surfaces in the future.

Although we are awaiting scientific developments, implementation of aggressive prevention and treatment strategies must be our priority. Two evidence-based documents are now available to assist clinicians in the prevention and management of catheter-related infection and thrombosis: the CDCs "Guideline for Prevention of Intravascular Device-Related Infections"[54] and the American Pharmaceutical Association's "Using Drug Treatment Protocols to Aid in Managing Patients With Venous Access Devices," which includes three protocols for managing patients with CVC-related thrombotic events, venous thrombosis, and infection.[55–57] Simply acquiring or reading these state-of-the-art documents will not be enough, however. The complexity and enormity of the problem require the establishment of a dedicated, multidisciplinary, quality-improvement team that is responsible for the implementation of these recommendations, individualized to their patient population.[54,58–60]

The vascular access team should involve members who are knowledgeable in the area of vascular access; this might include a medical director, team coordinator (nurse), certified IV nurse or certified nutrition support nurse, vascular access nurses, surgeon, interventional radiologist, home care nurse, pharmacist, staff nurse, infection control, and a risk-management representative. The role and responsibilities of this group should include a process for early assessment of patients for appropriate device selection, insertion and management of devices, implementation of policies and procedures, selection of VADs and associated products, ongoing education of physicians and nurses, quality-improvement program, patient education program, outpatient follow-up, and clinical research.[61–65] This special issue of *NCP* provides a comprehensive review of topics that are critical to the development

of strategies for the institution of vascular access programs in inpatient and outpatient settings.

The escalating trend of antimicrobial resistance among the most common pathogens that are responsible for catheter-related infections has produced a dangerous situation on both a national and international scale. With the existing threat of the loss of efficacy of vancomycin against staphylococcal infections in the absence of therapeutic alternatives, an increase in morbidity and mortality attributable to nosocomial bloodstream infections is certain to occur.[12,66] This, along with the high rate of thrombotic complications associated with the use of vascular catheters, may threaten the benefit of these devices in the future unless practitioners become proactive and meet the challenge to implement aggressive prevention and treatment strategies before it is too late.

References

1. Raad I. Intravascular-catheter-related infections. Lancet 1998; 351:893–7, March 21.
2. Spera G. A healthy future for catheter manufacturers. Medical Device & Diagnostic Industry. 1998;17, November.
3. Collin GR, Ahmadinejad AS, Misse E. Spontaneous migration of subcutaneous central venous catheters. Am Surg 1997;63:322–6.
4. Arnow PM, Quimosing EM, Beach M. Consequences of intravascular catheter sepsis. Clin Infect Dis 1993;16:778–4.
5. Banergee SN, Emori TG, Culver DH, et al. Secular trends in nosocomial primary bloodstream infections in the United States, 1980–1989. Am J Med 91 1991;(Suppl 3B):86S–9S.
6. Jarvis WJ, Edwards JR, Culver D, et al. Nosocomial infection rates in adult and pediatric intensive care units in the United States. Am J Med 91 1991;(Suppl 3B):185S–91S.
7. Pearson ML, Abrutyne E. Reducing the risk for catheter-related infections: a new strategy. Ann Intern Med 1997;127:304–6.
8. Micrologix Biotech Inc. Micrologix successfully completes phase I clinical trial for the prevention of bloodstream infections. [Online] *http://www.mbiotech.com*, April 1999:27.
9. Centers for Disease Control, National Nosicomial Infections Surveillance System. National Nosicomial Infections Surveillance (NNIS) Semiannual Report, May 1995. 1995;AJIC 23:377–5.
10. Centers for Disease Control, National Nosicomial Infections Surveillance System. National Nosicomial Infections Surveillance (NNIS) report, data summary from October 1986–April 1998. AJIC 1998;26:522–3.
11. Jarvis WR, Cookson ST, Robles B. Prevention of nosocomial bloodstream infections: a national and international priority. Infect Control Hosp Epidemiol 1996;17:272–5.
12. Linden PK. Clinical implications of nosocomial gram-positive bacteremia and superimposed antimicrobial resistance. Am J Med 1998;104(5A):24S–33S, May 29.
13. Garrett DO, Jochimsen E, Murfitt K, et al. The emergence of decreased susceptibility to vancomycin in staphylococcus epidermidis. Infect Control Hosp Epidemiol 1999;20:167–70.
14. Thylefors JD, Harbarth S, Pittet D. Increasing bacteremia due to coagulase-negative staphylococci: fiction of reality? Infect Control Hosp Epidemiol 1998;19:581–9.
15. Pittet D, Li N, Woolson RF, et al. Microbiological factors influencing the outcome of nosocomial bloodstream infections: a 6-year validated, population-based model. Clin Infect Dis 1997;24: 1068–78.
16. Collignon PJ. Intravascular associated sepsis: a common problem. Med J Aust 1994;161:374–8.
17. Conterno LO, Wey SB, Castelo A. Risk factors for mortality in staphylococcus aureus bacteremia. Infect Control Hosp Epidemiol 1998;19:32–7.
18. Nucci M, Colombo AL, Silveira F, et al. Risk factors for death in patients with candidemia. Infect Control Hosp Epidemiol 1998; 19:846–50.
19. Sotir MJ, Lewis C, Bisher EW, et al. Epidemiology of device-associated infections related to a long-term implantable vascular access device. Infect Control Hosp Epidemiol 1999;20:187–91.
20. O'Grady NP, Barie PS, Bartlett J, et al. Practice parameters for evaluating new fever in critically ill adult patients. Crit Care Med 1998;26:392–408.
21. Craft PS, May J, Dorigo A, et al. Hickman catheters: left-sided insertion, male gender, and obesity are associated with an increased risk of complications. Aust N Z J Med 1996;26:33–9.
22. Ray S, Stacey R, Imrie M, et al. A review of 560 Hickman catheter insertions. Anaesthesia 1996;51:981–5.
23. Nightingale CE, Norman A, Cunningham D, et al. A prospective analysis of 949 long-term central venous access catheters for ambulatory chemotherapy in patients with gastrointestinal malignancy. Eur J Cancer 1997;33:398–403.
24. Goey SH, Verweij J, Bolhuis RLH, et al. Tunnelled central venous catheters yield a low incidence of septicaemia in interleukin-2-treated patients. Cancer Immunol Immunother 1997;44:301–4.
25. Moukarzel AA, Haddad I, Ament ME, et al. 230 patient years of experience with home long-term parenteral nutrition in childhood: natural history and life of central venous catheters. J Ped Surg 1994;29:1323–7.
26. Tolar B, Gould JR. The timing and sequence of multiple device-related complications in patients with long term indwelling Groshong catheters. Cancer 1996;78:1308–13.
27. Timsit JF, Farkas JC, Boyer JM, et al. Central vein catheter-related thrombosis in intensive care patients. Incidence, risk factors, and relationship with catheter-related sepsis. Chest 1998; 114:207–13.
28. Raad II, Luna M, Khalil SM. The relationship between the thrombotic and infectious complications of central venous catheters. JAMA 1994;71:1014–16.
29. Didisheim P. Current concepts of thrombosis and infection in artificial organs. ASAIO 1994;40:230–7.
30. Cobos E, Dixon S, Keung YK. Prevention and management of central venous catheter thrombosis. Current Opinion Hematology 1998;5:355–9.
31. Cassidy FP, Jajko AB, Bron KM, et al. Noninfectious complications of long-term venous catheters: Radiologic evaluation and management. AJR 1987;149:671–5.
32. Beck C, Dubois J, Grignon A, et al. Incidence and risk factors of catheter-related deep vein thrombosis in a pediatric intensive care unit: a prospective study. J Pediatrics 1998;133:237–41.
33. Karnik R, Valentin A, Winkler WB. Duplex sonographic detection of internal jugular venous thrombosis after removal of central venous catheters. Clin Cardiol 1993;16:26–9.
34. Chastre J, Cornud F, Bouchama A, et al. Thrombosis as complication of pulmonary artery catheterization via the internal jugular vein: prospective evaluation by phlebography. N Engl J Med 1982;306:278–81.
35. DeCicco M, Matovic M, Balesteri L, et al. Central venous thrombosis: an early and frequent complication in cancer patients bearing long-term silastic catheter: a prospective study. Thrombosis Research 1997;86:101–13.
36. Andrew M, Marzinotto V, Pencharz P, et al. A cross-sectional study of catheter-related thrombosis in children receiving TPN at home. J Pediatrics 1995;126:358–63.
37. Carman TL, Fernandez BB. Issues and controversies in venous thromboembolism. Cleveland Clinic J Med 1999;66:113–23.
38. Prandroni P, Polistena P, Bernardi E, et al. Upper extremity deep vein thrombosis. Risk factors, diagnosis, and complications. Arch Intern Med 1997;157:57–63.
39. Connors AF, Castele RJ, Farhat NZ, et al. Complications of right heart catheterization: a prospective autopsy study. Chest 1985; 88:567–72.
40. Hingorani A, Ascher E, Hanson J, et al. Upper extremity versus lower extremity deep vein thrombosis. Am J Surg 1997;174: 214–7.
41. Jones RN. Impact of changing pathogens and antimicrobial susceptibility patterns in the treatment of serious infections in hospitalized patients. Am J Med 1996;100(Suppl 6A):3S–12S.
42. Strausbaugh LJ. Vancomycin-intermediate Staphylococcus epidermidis: curio or omen? Infect Control Hosp Epidemiol 1999;20: 163–5.

43. Raad I, Airahwan A, Rolston K. Staphyloccocus epidermidis: Emerging resistance and need for alternative agents. Clin Infect Dis 1998;26:1182–7.
44. Raad I & the Global Synercid CRGPB Study Group. Dose ranging randomized multicenter open phase I study of quinupristin/dalfopristin versus vancomycin in the treatment of catheter-related gram-positive bacteremia. Final Programme and Book of Abstracts, Twentieth International Congress of Chemotherapy, Sydney, Australia: June 1997 [Abstract 2319].
45. Leclercq R, Courvalin P. Streptogramins. An answer to antibiotic resistance in gram-positive bacteria. The Lancet 1998;352:591–2, August 12.
46. Barriere JC, Bouanchaud DH, Paris JM, et al. Antimicrobial activity against Staphylococcus aureus of semisynthetic injectable streptogramins: RP 59500 and related compounds. J Antimicrob Chemother 1992;30(Suppl A):1–8.
47. Russell AD, Tattawasart U, Maillard JY, et al. Possible link between bacterial resistance and use of antibiotics and biocides. Antimicrob Agents & Chemo 1998;42:2151.
48. Haines KA, Klein DA, McDonnell G, et al. Could antibiotic-resistant pathogens be cross-resistant to hard-surface disinfectants? AJIC 1997;25:439–41.
49. Eng JA, Valena F, Dever LL, et al. Could antibiotic-resistant pathogens be cross-resistant to hard-surface disinfectants? Reply. AJIC 1997;25:441.
50. Eng JA, Shsh L, Valena F, et al. Effectiveness of disinfectants for vancomycin-resistant Enterococcus faecium [Abstract]. AJIC 1996;24:101.
51. US Congress, Office of Technology Assessment: Impacts of antibiotic-resistant bacteria (Doc. No. OTA-H-629). US Government Printing Office, Washington, DC, 1995.
52. Veenstra DL, Saint S, Saha S, et al: Efficacy of antiseptic-impregnated central venous catheters in preventing catheter-related bloodstream infection. JAMA 1999;281:261–7.
53. Darouiche RO, Raad II, Heard SO, et al. A comparison of two antimicrobial-impregnated central venous catheters. N Engl J Med 1999;340:1–50.
54. Pearson ML. Guideline for the prevention of intravascular-device-related infections. Infect Control Hosp Epidemiol 1996;17:438–72.
55. American Pharmaceutical Association. Managing patients with venous access device-related thrombotic occlusion. IN APhA Guide to Drug Treatment Protocols: A Resource for Creating and Using Disease-Specific Pathways. Author, Washington, DC, 1998, pp 9–20.
56. American Pharmaceutical Association. Managing patients with venous access device-related venous thrombosis. IN APhA Guide to Drug Treatment Protocols: A Resource for Creating and Using Disease-Specific Pathways. Author, Washington, DC, 1998, pp 21–30.
57. American Pharmaceutical Association. Managing infectious complications in patients with venous access devices. IN APhA Guide to Drug Treatment Protocols: A Resource for Creating and Using Disease-Specific Pathways. Author, Washington, DC, 1998, pp 31–9.
58. Ryder M. Peripherally inserted central venous catheters. Nurs Clinics N Am 1993;28:937–72.
59. Ryder M, Scott WL, Helm A. Is downsizing and disbanding specialty care teams a counterproductive strategy for cost reduction in healthcare? Nutrition 1998;14:725–8.
60. Lange BJ, Weiman M, Feuer EJ, et al. Impact of changes in catheter management on infectious complications among children with central venous catheters. Infect Control Hosp Epidemiol 1997;18:326–32.
61. Sherertz R. Look before you leap: discontinuation of an infusion therapy team. Infect Control Hosp Epidemiol 1999;20:99–100.
62. Abi-said D, Raad I, Umphrey J, et al. Infusion therapy team and dressing changes of central venous catheters. Infect Control Hosp Epidemiol 1999;20:101–5.
63. Meier PA, Fredrickson M, Catney M, et al. Impact of a dedicated intravenous therapy team on nosocomial bloodstream infection rates. AJIC 1998;26:388–92.
64. Buggs C, Valadez AM, Lund CA. Evaluating the effectiveness of an intravenous therapy team. Nursing Economics 1993;11:249–55.
65. Christensen L, Greenough B. Centralized I.V. team provides inpatient and home-care treatment. Nurs Manage 1998;29:69–71.
66. Sherertz RJ, Westbrook DM, Gledhill KS, et al. Description of an infection control course for house officers and medical students. Abstract presented at the eighth annual meeting of the Society for Healthcare Epidemiology of America, Orlando, FL, 1998, April (Abstract 12).
67. Stephenson J. Worry grows as antibiotic-resistant bacteria continue to gain ground. JAMA 1997;278:2049–50.

Vascular Access Organizations and Government Agencies

Centers for Disease Control and Prevention
1600 Clifton Road, NE
Atlanta, GA 30333
(404) 639-3311
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Rockville, MD 20857
(888) 463-6332
Website: www.fda.gov/cdrh/
Email: webmail@oc.fda.gov

National Home Infusion Association
205 Daingerfield Road
Alexander, VA 22314
Phone: (703) 549-3740
Fax: (703) 683-1484
Email: nhia@vais.net

Intravenous Nurses Society
Fresh Pond Square
10 Fawcett Street
Cambridge, MA 02138
Phone: (617) 441-3008
Fax: (617) 441-3009
Website: www.ins1.org