Anesthesiology
47:195–202, 1977

The Role of Infection in Critical Care

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A BROAD AND COMPLEX NETWORK of host defense mechanisms maintains a normal host remarkably free of symptomatic infection. Body-wise defense mechanisms normally operative include polymorphonuclear leukocytes, circulating immunoglobulins, and cell-mediated immune mechanisms. In addition, specific organ defense mechanisms are increasingly being elucidated. For example, the principal components of respiratory tract defense mechanisms include the cough reflex, the filtration effect of the progressively narrowing diameters of the trachea, bronchi, and bronchioles, the mucociliary escalator system, local immunoglobulin production, and alveolar macrophages. Gastrointestinal tract defense mechanisms include gastric acid, motility, secretory immunoglobulins, and the normal microflora of the gastrointestinal tract. Organ defense mechanisms within the urinary tract, the skin, and within other organ systems are increasingly being appreciated.

For obvious reasons, patients undergoing critical care rarely, if ever, have completely functional host defense mechanisms. It is important to understand the extent of compromise of body-wide or of organ-specific host defense mechanisms induced by underlying disease, trauma, or diagnostic or therapeutic modalities, in order to appreciate both the risk of and the prevention of infection in critical care units.

This review deals with the three major categories of infection seen in critical care units, that is, septicemia, pneumonia, and urinary tract infection. Surgical wound infections are, of course, also a major category of infection seen in surgical intensive care units; postoperative wound infections are not, however, uniquely associated with critical care, and therefore are not considered further. Interested readers are referred to an excellent monograph recently published by the American College of Surgeons.[1]

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I. Bacteremia

A. Pathogenesis and Epidemiology

Bacteremia commonly occurs as a secondary manifestation of infection elsewhere, such as pneumonia, deep abscesses, and the like; of particular concern in critical care areas are bacteremias occurring during intravenous therapy. Plastic intravenous catheters were introduced in 1945, and have been widely accepted in medical practice. The advantages of ease of insertion, durability, and the ability to deliver large volumes of fluid rapidly are well known. It was not long after their introduction, however, before intravenous catheters were recognized as a cause of thrombophlebitis and septicemia.

Septicemia may occur during intravenous therapy as a result of infected venisection sites or septic thrombophlebitis, or directly from infusion fluid or the delivery system itself.

The weight of evidence suggests that flora of the skin are the major source of catheter-associated bacteremia.[2] Organisms most commonly isolated from in-use catheters, as well as from catheter-associated bacteremias, include *Staphylococcus aureus; S. epidermidis*; gram-negative bacilli, including *Klebsiella, Enterobacter*, and *Serratia*; and enterococci. These organisms are regularly found on the skin of hospitalized patients. A number of investigators have reported a strong similarity between cutaneous flora and organisms identified on catheter tips[3–6]; this suggests that such flora may contaminate the tip of the catheter either at the time of insertion or subsequently by migrating at the interface between tissue and catheter. Since a loose fibrin mesh regularly forms around the intravascular portion of plastic catheters, some have suggested[6,7] that this fibrin mesh may serve as a filter for circulating organisms. Thus, a transient bacteremia from a distant source could theoretically seed a catheter with pathogenic organisms. Although such a route of infection may, in fact, occur at times, it is unlikely to be a major source, since the microbiologic profiles of catheter-associated bacteremia and bacteremia associated with distant foci of infection differ.[2]

Good evidence exists that catheter-associated phlebitis, defined as evidence of venous inflammation with or without a palpable cord, occurs primarily on a physicochemical basis. Factors considered significant in the development of phlebitis include the type of

catheter used, the material used in the catheter, the size of the catheter, the anatomic location and position of the catheter, duration of cannulation, the nature of the infusion fluids given, and intravenous medications given through the delivery apparatus. Although prospective studies of catheter-associated infection have generally failed to find an association between phlebitis and positive catheter-tip cultures, Maki *et al.*,[2] in reviewing the relevant studies, have suggested that patients who have catheter-associated phlebitis have a significantly increased risk of septicemia.

The third major route of infection results from contamination of the infusion fluid itself, the delivery system, or the additives and intravenous medications administered through the delivery system. Although long recognized as a theoretical possibility, a nationwide outbreak of *Enterobacter agglomerans* septicemia traced to intrinsic contamination of fluids produced by a major manufacturer in the United States underscored the importance of this route of infection. Subsequent experience has identified other, albeit less dramatic, episodes of intrinsically contaminated intravenous fluids, as well as the risks of in-use contamination of fluids and of the delivery system itself. Maki and associates‡[8] have reported that in one study, 15 per cent of intravenous systems in use for more than 48 hours were contaminated, whereas only 3 per cent of systems in use for less than 48 hours were contaminated. Bacteria found were fortunately relatively avirulent, and were usually present in small numbers. Addition of solutions to the bottle, direct injection of medication into the tubing, the introduction of stopcocks, manometers, or other devices into the line, the common practice of irrigating occluded catheters, and the generally ill-advised practice of withdrawing venous blood samples from the intravenous delivery system all increase the risk of contamination. It is likely that extrinsic contamination from such sources has long been a source of sporadic septicemia associated with intravenous therapy.

Rates of positive catheter cultures and catheter-associated septicemia have been reported in most studies to increase with increasing duration of catheterization. In studies of plastic catheters left in place for more than 48 hours, the associated septicemia rates have varied between 2 and 5 per cent.[2] In some hospitals, however, rates as high as 7–8 per cent have been reported.[9,10] Observations of this nature have led to the generally accepted recommendation that, when feasible, a plastic catheter be removed and a new one reinserted at another site after 48 hours.

There is substantial evidence that the use of steel needles, especially scalp-vein needles, is associated with lower rates of septicemia than the use of plastic catheters.[11–13] The extensive use of scalp-vein needles in the critical care context is not feasible, however, and some of the relative safety of steel needles may simply reflect the fact that they infiltrate more readily, thus requiring more frequent changes.

It must be recognized that both physicians and nurses may carry nosocomial pathogens on their hands and thus may contaminate the catheter–skin junction at the time of routine medical or nursing care.[14] A vivid illustration of this route of transmission was provided by an outbreak of *Klebsiella* septicemia in an intensive care unit, traced to contaminated hand cream used by nurses following hand washing.[15]

B. Prevention

Because of the intrinsic nature of critical care, insertion of intravenous needles, cannulas, or catheters must often be carried out with all deliberate speed. It is widely recognized, however, that catheters inserted under the stress of an emergency, without adequate preparation of the skin and other aseptic precautions, have a significantly greater risk of catheter-associated septicemia. It is strongly recommended, therefore, that catheters inserted under such conditions be removed and reinserted using appropriate aseptic technique as soon as the patient's condition permits.

Adequate preparations of the skin consists of thorough and vigorous application of a cutaneous disinfectant such as tincture of iodine, or one of the currently available iodophors. Tincture of iodine must be washed off with 70 per cent alcohol to reduce the chance of cutaneous burns, but iodophor antiseptics should not be washed off after application, since the continued release of free iodine results in sustained broad-spectrum germicidal activity. Gentleness and strict attention to aseptic detail are necessary during insertion of the needle or catheter. The needle or catheter and hub, together with the proximal segment of the delivery line, should be secured after insertion to prevent to-and-fro motion, which might further irritate tissue and facilitate contaminants' being carried into the subcutaneous tissues or into the vein itself.

The topical application of antimicrobial agents at

‡ Maki DG, Rhame FS, Mackel DC, et al: Nosocomial septicemias subsequent to contaminated intravenous fluid. Presented at the Annual Meeting of the American Society for Microbiology, Minneapolis, 5 May 1971.

the skin–catheter junction has been extensively studied, but evidence for effectiveness in preventing catheter-associated septicemia is conflicting.[16–18] Moreover, there is evidence that topical antibiotic ointments select for antibiotic-resistant strains of bacteria or fungi. The use of an iodophor ointment applied at the skin–catheter junction has been recommended, but the safety and efficacy of this procedure remain to be established by controlled clinical trials.

The entire area, including the hub and adjacent tubing, should be adequately covered by a dressing, and changed at regular intervals. Totally occlusive dressings have been recommended,[1] but the possible significance of the change in underlying cutaneous flora that regularly occurs has not yet been adequately explored.[2]

All intravenous tubing and solutions should be changed every 24 hours, and intravenous catheters and sites of insertion should, when feasible, be changed every 48 hours. This is not always possible, of course, but the justification for leaving a catheter in place longer than 48 hours should appear in the patient's chart. The importance of hand washing between patient contacts must be emphasized over and over again; physicians appear to be particularly refractory to accepting this as the single most important measure in preventing cross-infection in critical care areas.

Contamination of an intravenous system should always be considered in the differential diagnosis of fever or other evidence of sepsis in the patient who has such a system in place. When there are local signs of phlebitis, the catheter at that site should be removed. Contaminated catheters or infusion fluids may result in bacteremia even though there is no local evidence of inflammation; therefore, when sepsis is suspected in a patient with an indwelling intravenous line, the catheter and delivery system should be removed, and a new one inserted at another site. When there is reason to suspect intrinsic contamination of infusion fluids or the delivery system, the identity and lot numbers of suspect products should be recorded, and all unused infusion fluid saved for microbiologic study. Such circumstances should be reported promptly to the Hospital Infection Committee.

Specially trained intravenous therapy teams have been recommended to achieve more uniform adherence to standards of asepsis. Such teams may develop great proficiency in establishing and maintaining intravenous lines, and it seems likely that such an approach might significantly reduce infusion-related sepsis, as well as increase efficiency, within critical care areas.

II. Lower Respiratory Tract Infections

A. Pathogenesis

Bacteria may invade the alveolar level of the lung in sufficient density to produce pneumonia by three routes: 1) by way of the vasculature; 2) in bacteria suspended in an inhaled gas; 3) by aspiration from the pharynx. It is generally felt that most pneumonias asquired in intensive care units are due to microorganisms that make up the flora of the pharynx.[19] Several studies have pointed out the frequency with which aspiration occurs in hospitalized patients, particularly those who have airway instrumentation.[20,21] Thus, of major importance are the quantity and types of organisms that are present in the oropharynx, as well as pulmonary antibacterial defenses.

The normal oropharynx is populated by a varied yet relatively constant population of aerobic and anaerobic bacteria. Of note, aerobic gram-negative rods are absent or present in very small numbers in oral cultures from normal volunteers, and normal individuals will clear large numbers of aerobic gram-negative rods when they are deposited by gargle challenge.[22,23] Chronically or severely ill patients, those most likely to be housed in intensive care units, are far more likely to become colonized with aerobic gram-negative rods.[22,24] Clinical and microbiologic studies have shown that persons bedded in intensive care units rapidly become colonized with aerobic gram-negative rods, and that oral colonization with gram-negative rods invariably precedes the development of pneumonia.[19,24]

The types of organisms that tend to colonize these patients are the usual gram-negative rod flora that we associate with intensive care units. Often there are local strains, some with unusual antimicrobial susceptibility patterns, that prove to be more important than others.

Only a few patients whose oral cavities become colonized with aerobic gram-negative rods will develop gram-negative rod pneumonia.[24,25] This suggests the importance of another variable, namely, pulmonary antibacterial defenses. The most important pulmonary antibacterial defenses against aspiration of oral flora are: 1) the cough reflex; 2) the mucociliary escalator system; 3) the alveolar macrophage defense network.

In normal subjects cough is a very effective means of expelling foreign bodies and secretions. However, in the presence of severe disease, cough can be remarkably inefficient and result in movement of secretions from one lung to another, rather than total clearance out of the bronchial tree. For a variety of

physiologic and pharmacologic reasons, intensive care unit patients often have severely depressed cough reflexes and are unable to utilize this mechanism to clear secretions.

Particles aspirated from the oropharynx are deposited on airways lined with ciliated epithelium. Smaller particles are then removed by the mucociliary transport mechanism. Beating cilia set overlying mucus in motion at rates that increase progressively from peripheral airways towards the trachea. Linear velocities range from rates of .5–1 mm/min in small airways to as much as 5–20 mm/min in the trachea in main bronchi.[26] Virtually all material deposited on ciliary epithelium in normal persons appears to be removed in less than 24 hours in a coordinated and sequential fashion.[27] Tracheal cuffs and trauma from repeated tracheal suction severely damage delicate ciliary epithelial cells.[28] Patients who have chronic bronchitis clear abnormally because of areas of squamous metaplasia, and uncoordinated ciliary activity. Finally, some agents, such as 100 per cent oxygen, are ciliotoxic.

Particles that get to the distal recesses of the lung are inactivated by the alveolar macrophage defense network. Alveolar macrophages actively phagocytize and kill these organisms, and their functional ability constitutes an important cellular defense mechanism.[29] Alveolar macrophage bactericidal activity is compromised by a number of exogenous and endogenous factors such as alcoholism, corticosteroid therapy, oxygen toxicity, uremia, acidosis, and pulmonary edema; these factors are commonly present in patients admitted to intensive care units.[30]

B. Clinical and Microbiologic Characteristics

The recognition of pneumonia in intensive care unit patients is often difficult. Many of these patients have chronic or non-infectious pulmonary diseases that make roentgenographic interpretations of infiltrates particularly difficult. Second, all patients who have airway instruction will develop an exudative bronchitis, which will become colonized, usually with aerobic gram-negative rods. Thus, Gram stains of material from tracheal aspirates will invariably show many polymorphonuclear leukocytes and gram-negative rods. Culture of this material will often yield a bewildering array of aerobic gram-negative rods. The tendency of some physicians is to treat these episodes of tracheitis. We, and others, would not agree, and feed that antimicrobial therapy should be reserved for those patients who demonstrate one or more of the following: new or increasing pulmonary infiltrates, fewer, leukocytosis, worsening pulmonary function, or suggestive evidence of septicemia. Because bacteremia frequently accompanies pneumonia, blood cultures are often valuable in establishing an etiologic diagnosis. Similarly, patients suspected to have pneumonia and pleural effusions should have thoracocentesis to exclude the presence of empyema. The reflex response of adding antibiotics for "positive" sputum cultures without a careful evaluation of the patient is to be discouraged.

In general, the microbiology of serious pneumonias acquired in intensive care units reflects the local ecology of aerobic gram-negative rods. As a group, these are hardy organisms (*Escherichia coli, Klebsiella pneumoniae, Proteeae, Serratia marcescens*, and *Pseudomonas aeruginosa*) that can survive in the environment and have the potential for developing resistance to commonly-used antimicrobial agents. Which of these organisms gains ascendancy may vary from hospital to hospital. That is, in one hospital *Pseudomonas aeruginosa* may predominate, whereas *Serratia marcescens* may be an important pathogen in yet another unit. The main point to be emphasized is that any aerobic gram-negative rod, given the proper environment, has the potential for causing lower respiratory tract infections in patients hospitalized in an intensive care unit.

C. Epidemiology

Perhaps the most detailed analysis of an intensive care unit experience with pneumonia is that of the Beth Israel Hospital in Boston. From 1967 to 1970, among 711 patients admitted to their intensive care unit, there were 158 episodes of bacterial pneumonia—an incidence of 21.6 per cent.[31] A prospective study by Johanson *et al.* documented an incidence of hospital-acquired pneumonia of 12.2 per cent in patients admitted to a medical intensive care unit.[24] Thus, pneumonia in intensive care units is frequent.

Mortality rates in patients who develop gram-negative rod pneumonia are depressingly high. In the Beth Israel study, the mortality rate for the pneumonia-free population was 3.8 per cent, while 5 per cent of gram-positive pneumonias were fatal, a figure not significantly different from that of the pneumonia-free group. However, mortality rates of about 70 per cent were regularly documented in two groups of patients where *Pseudomonas aeruginosa* was isolated, either as the predominant bacterium or as one of several organisms found on sputum culture.[31] This experience is certainly not unique, and has led to the general conclusion that aerobic gram-negative rod pneumonia, because of its formidable mortality rate, is the most important infectious disease problem within intensive care unit populations.

Contaminated nebulization equipment has been

associated with severe epidemics of gram-negative rod pneumonia. In the mid-1960's investigators in Dallas showed a relationship between autopsy evidence of necrotizing pneumonia and the use of nebulizer therapy.[32] Extensive microbiologic studies showed that aerosol reservoirs became extensively contaminated with aerobic gram-negative rods.[32] After routine acetic acid rinsing of the nebulizers was introduced, the autopsy incidence of necrotizing gram-negative pneumonia declined to a level similar to that seen prior to the introduction of widespread use of inhalation equipment.[33] Epidemics of pneumonia due to *K. pneumoniae, P. aeruginosa*, and *S. marcescens* have also been related to contaminated ultrasonic and reservoir nebulizers.[34-36]

In a prospective study of oral colonization and pneumonia in an intensive care unit at the Denver V.A. Hospital, we found acquisition of *Proteae* after exposure to adjacent-bed patients colonized with the same strain. This finding suggests patient-to-patient transmission, probably via the contact within the intensive care unit setting.§ Similar results have been reported by Lowbury *et al.*, who consistently recovered epidemic strains of *P. aeruginosa* from the hands of intensive care unit personnel.[37] They concluded that the hands of staff members presented a special hazard of transferring infection.

D. Prevention

Since an impressive number of intensive care unit fatalities are pneumonia-related, efforts at prevention have emphasized: 1) cleanliness of respiratory equipment; 2) prevention of oral colonization; 3) heightening of pulmonary host defenses.

As has been previously mentioned, contaminated respiratory equipment, particularly nebulizers, has been related to an increased incidence of pneumonia. Therefore, strict attention to the cleanliness of this equipment is critical. Guidelines for the care and sterilization of this equipment have been published and need not be reviewed.[38] Intensive care unit personnel must be aware of the infectious hazards of this equipment and must be specifically trained so that contamination is minimized. Routine microbiologic sampling of equipment has been suggested as a means to assess cleanliness, but we and others feel that strict adherence to proven protocols for decontamination and sterilization represents the most satisfactory approach.

Several approaches have been used to prevent colonization. The most obvious has been rigorous attention to suction techniques, particularly in patients who have some instrumentation of the respiratory tract. Equipment and medication used in the care of other patients should not be shared. Aseptic technique should be used for suction, and the use of sterile gloves or "no-touch" technique is essential. Fresh, sterile catheters should be used for each suction. Catheters may be flushed during the suction procedure with small amounts of sterile water, which should always be discarded immediately after use. Strict adherence to these techniques will not totally prevent pneumonia, but may be expected to decrease the incidence. Similarly, hand washing between patient contacts is essential if contact spread of aerobic gram-negative rods among patients is to be minimized.

Some investigators have studied the effects of local antibiotics to prevent oral or tracheal colonization with gram-negative rods. In an extensive series of studies, investigators have shown that intermittent daily aerosolization with polymyxin decreased the incidence of colonization and pneumonia due to *P. aeruginosa* in an intensive care unit.[39,40] However, when polymyxin was used continuously on all unit patients, pneumonia caused by polymyxin-resistant organisms was found.[41] Thus, prophylactic local installation of polymyxin to prevent colonization and pneumonia was felt to have limited value as a general measure, but might be useful in epidemics caused by polymyxin-sensitive organisms.

Similarly, Klastersky *et al.* have shown that endotracheal administration of gentamicin significantly reduced colonization of tracheal secretions by gram-negative rods.[42] Not surprisingly, bacteria isolated from gentamicin-treated patients were more resistant to gentamicin than organisms from the respiratory tracts of controls. Development of resistance to gentamicin may be too high a price to pay for the decrease in infections expected with this therapy, but only carefully controlled studies will answer this question.

Currently, the concept of strengthening pulmonary antibacterial defenses is largely investigational. Published data using a *Pseudomonas* vaccine in a surgical intensive care unit showed protection against *Pseudomonas* colonization in those patients intubated for less than a week, and some evidence that mortality due to *Pseudomonas* pneumonia was decreased.[43] Further studies in this general area are necessary.

Of more immediate applicability is effort directed at minimizing the use of techniques or drugs known to compromise pulmonary defense mechanisms. For example, aerodynamic filtration, a very effective pulmonary defense mechanism, is bypassed in patients

§ Tenney JH, Hopkins JA, Wang W-LL, et al: Pneumonia and pharyngeal colonization in a medical intensive care unit (unpublished data).

who have tracheal intubation. Likewise, the presence of a nasogastric tube facilitates aspiration. Thus, important and sometimes neglected questions relate to whether continued instrumentation is necessary in the individual patient. Similarly, antibiotics and corticosteroids, often extravagantly overused in intensive care unit patients, have been shown to predispose patients to colonization with aerobic gram-negative rods. A conscientious effort to reduce these factors to an essential minimum might be expected to decrease colonization rates.

III. Infections of the Urinary Tract

A. Pathogenesis and Epidemiology

Infections of the urinary tract merit special treatment in a consideration of infection and critical care, for three reasons: 1) the often documented observation that infections of the urinary tract account for a third or more of all nosocomial infections; 2) the observations of Altemeier and associates[1] that more than 50 per cent of gram-negative septicemias studied on their surgical service had their origin within the urinary tract; 3) the necessity for indwelling urethral catheterization of many patients in critical care areas.

Because of the need to monitor closely the urinary outputs of many patients in critical care areas, the frequent use of indwelling urethral catheters must be accepted as a medical fact of life. Other justifiable indications include major neurologic dysfunction involving the bladder, or major structural abnormality within the lower urinary tract. Uncooperative, obtunded, or comatose patients may also require indwelling catheters for variable lengths of time. Nevertheless, it must be appreciated that the close association of instrumentation or catheterization of the urinary tract and subsequent infection is beyond dispute.

The epidemiology and pathogenesis of urinary tract infection related to catheterization are the same in patients in critical care areas and in those in other areas of hospital care; the only difference is the higher proportion of patients in critical care areas who are at risk. The risks of infection of the urinary tract under conditions of open catheter drainage, and under conditions of sterile closed drainage, have been well defined, particularly by Kunin.[44] The majority of such infections result from 1) spread of bacteria from the perineum along the catheter–urethral interface and into the bladder; 2) ascending infection resulting from contamination of urine in the drainage tube, with subsequent extension directly into the bladder.

There is also a significant risk of nosocomial transmission of urinary tract infection, particularly those infections caused by multidrug-resistant gram-negative bacilli. Schaberg, Weinstein, and Stamm[45] recently summarized seven outbreaks of such infections caused by *Klebsiella pneumoniae, Serratia marcescens,* and *Proteus rettgeri*. Their investigations suggested that the organism was transmitted from patient to patient on the hands of personnel in all seven outbreaks. In the *Serratia marcescens* and *Proteus rettgeri* outbreaks, the major reservoir appeared to be the genitourinary tracts of catheterized patients. Spatial clustering of patients was especially prominent in the outbreaks caused by *Serratia marcescens* and *Proteus rettgeri*. In one of the *Serratia marcescens* outbreaks, a higher attack rate was found in catheterized patients sharing a room with an infected catheterized patient. Thus, the possibility of nosocomial transmission must be kept in mind, particularly in the environment of critical care areas.

Garibaldi and associates,[46] in an extensive study of factors predisposing to bacteriuria during indwelling urethral catheterization, observed that risks were significantly greater for patients who were female, elderly, or critically ill. These investigators did observe, however, that patients in intensive care units had no higher rates than patients in private or semiprivate rooms.

B. Prevention

A decision to insert an indwelling urethral catheter should not be a reflex reaction, but rather a balanced decision arrived at on the basis of risk–benefit considerations. Due consideration should also be given to the use of alternative methods of urinary tract drainage, including suprapubic bladder drainage and condom drainage. Under conditions of critical care, however, it is likely that the indwelling urethral catheter is, at this time, the most reasonable alternative.

Two considerations are of utmost importance: 1) the technique of insertion, and 2) catheter management following insertion. Appropriate techniques of insertion of indwelling urethral catheters have been well outlined by Kunin,[44] and by Stamm.[47] It should be pointed out that, although there is occasionally justification for the insertion of intravenous catheters under conditions of extreme emergency with minimal aseptic care, the same cannot be said for the insertion of an indwelling urethral catheter. There is rarely, if ever, justification for insertion of such catheters under circumstances that short-cut acceptable standards of asepsis. Garibaldi and associates documented the im-

portance of adequate training of the person who inserts the catheter, and observed that female patients catheterized by licensed practical nurses had almost twice the rate of acquired bacteriuria during the first 48 hours of catheterization as that found in patients catheterized by registered nurses or physicians.[46]

Major emphasis must be put on catheter management. Appropriate standards of catheter care have been reviewed by Kunin, and the referenced monograph[44] is an extraordinarily useful guide. Attention should be given to gentle washing of the perineal area with soap and water at least twice daily, taking care not to traumatize the perineal tissue unnecessarily, or to move the catheter to and fro unnecessarily in the urethra. The drainage bag should never touch the floor, and should always be kept below bladder level. Urine for urinalysis or culture should be obtained from the catheter itself using a needle and syringe, with the same care and aseptic precautions as for a venipuncture. Routine irrigation of catheters is not recommended. The balance of evidence suggests that other adjunctive measures, such as urethral irrigation with an antiseptic or ointment prior to catheter insertion, the addition of disinfectants to collection reservoirs, the topical application of antimicrobial ointments to the perineal–catheter junction, or the use of antimicrobial-impregnated catheters, are of little if any value in preventing bacteriuria. Similarly, systemically administered antimicrobial drugs are of no value in preventing infection within the urinary tract with a catheter in place.

The possibility of nosocomial transmission within the critical care setting must always be kept in mind; this again re-emphasizes the necessity of thorough hand washing between patient contacts by both physicians and nursing personnel.

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