TABLE 99-3 MODIFIED CLINICAL PULMONARY INFECTION SCORE (CPIS)

CLINICAL CRITERIA	INFORMATION	POINTS*
Temperature (°C)	≥36.5 and ≤38.4	0
	≥38.5 and ≤38.9	1
	≤36 or ≥39	2
Leukocyte count (per μL)	≥4,000 and ≤11,000	0
	<4,000 or >11,000	1
	<4,000 or >11,000 + ≥500 bands	2
Tracheal secretions	Absent/rare	0
	Abundant/nonpurulent	1
	Abundant + purulent	2
PaO_2/FIO_2 (mm Hg)	>240 or ARDS	0
	≤240 and no evidence of ARDS	2
Chest radiographic findings	No infiltrate	0
	Diffuse or patchy infiltrate	1
	Localized infiltrate	2
Microbiology†	Negative	0
	Positive	2

Data from Fartoukh M, Maitre B, Honore S, et al: Diagnosing pneumonia during mechanical ventilation: the clinical pulmonary infection score revisited, Am J Respir Crit Care Med 168:173–179, 2003.

ARDS, Acute respiratory distress syndrome.

*With the use of these clinical criteria, a clinical pulmonary infection score of 6 or greater has an 85% sensitivity for detecting pulmonary infection.

†Gram stain results of directed or blind, protected endotracheal aspirate.

One definition of HAP or VAP includes clinical, radiographic, and microbiologic criteria. The Clinical Pulmonary Infection Score (CPIS) system is useful in determining when antimicrobial therapy is necessary (Table 99-3). A score of 6 or greater has 85% sensitivity in diagnosing a pulmonary infection. Signs and symptoms indicating an infection include fever (≥38° C), peripheral leukocytosis, purulent sputum, and worsening respiratory status. A tracheal aspirate for Gram stain and culture provides the last piece of diagnostic information. When several of these signs and symptoms exist in the absence of a pulmonary infiltrate, alternative diagnoses should be considered, including ventilator-associated tracheobronchitis.

Duration of hospitalization is a significant factor to consider when initiating empirical therapy because of the increasing likelihood of MDRO colonization with prolonged length of stay. In early HAP (≤4 days), more community-acquired organisms may be targeted, except when the patient has certain qualifiers for HCAP (see Table 99-2) or is known to be colonized with resistant organisms. However, in late HAP (including VAP and HCAP), the IDSA guidelines recommend adding empirical coverage for resistant gram-positive organisms (including MRSA) and for multidrug-resistant Enterobacteriaceae (level II evidence according to the American Thoracic Society [ATS]/IDSA guidelines for HAP). Dual coverage for multidrug-resistant *P. aeruginosa* should also be considered. An example of an empirical regimen for late-onset HAP is vancomycin or linezolid plus an antipseudomonal β-lactam/β-lactamase inhibitor, carbapenem, or cephalosporin.

INFECTIONS ASSOCIATED WITH VASCULAR CATHETERS

The NHSN collects data on CLABSIs, and public reporting is required for CLABSIs in ICUs. In 2011, the incidence of CLABSIs ranged from 0 to 3.7 cases per 1000 catheter days, compared with 1 to 5.6 cases per 1000 catheter days in 2006 through 2007. Although CLABSIs have the lowest prevalence among HAIs, the cost per episode and morbidity rate remain high. The estimated additional cost of an infection related to an intravenous catheter is $4000 to $56,000 per episode. The attributable increase in length of stay has been between 6.5 and 22 days, and the attributable mortality rate is about 10% among hospitalized patients.

The most common pathogens that cause primary CLABSIs are flora arising at the percutaneous insertion site or from contamination of the catheter hub. Hematogenous seeding from a gastrointestinal or other endovascular source occurs but is less likely. The most common pathogens that cause CLABSIs are coagulase-negative staphylococci, *Candida* species, *S. aureus,* and *Enterococcus* species (see Fig. 99-1). The risk factors for CLABSI are provided in E-Table 99-1. The rising proportion of infections caused by *Enterococcus* species and *Candida* species since the 2006-2007 period suggests that skin colonization is being adequately addressed by the adoption of evidence-based prevention strategies and that an increasing fraction of CLABSIs are caused by secondary hematogenous seeding. Patients who are more severely ill, are neutropenic, have burns, or are on total parenteral nutrition are also at increased risk for candidemia. Other types of catheter-related infections include phlebitis, exit site infection, and pocket and tunnel infection.

Many CLABSIs are preventable through the use of evidence-based prevention practices for line insertion and maintenance. Strategies include appropriate decolonization of the skin before insertion with chlorhexidine plus alcohol, use of maximal sterile barriers (i.e., proceduralist wears sterile gloves and gown, cap, and mask, and a large barrier drape is placed over the patient), hand hygiene, and sterile technique (see Table 99-1). Appropriate maintenance of the central line mandates scrubbing the hub with antiseptic and discontinuing the catheter as soon as it is not needed.

For a patient with a fever or systemic symptoms who has a central venous catheter, a bloodstream infection should be suspected. The diagnostic evaluation should begin with paired peripheral and catheter blood samples for culture before initiation of antimicrobial therapy. In a suspected case of bloodstream infection, the exudate at the exit site should be cultured.

The type of device (e.g., peripheral vs. central, short term vs. long term), associated infectious complications, and the implicated organism all play a role in treatment. For CLABSIs associated with short-term, nontunneled catheters and no complicating factors (e.g., suppurative thrombophlebitis, endocarditis, intravascular hardware), it may be appropriate to treat for 7 to 14 days after removal of the catheter. However, for long-term catheters, salvage may be attempted with systemic plus antibiotic lock therapy (level B-II evidence, indicating a moderate amount of evidence from well-designed clinical trials or cohort or case series). Salvage of catheters associated with *S. aureus* bacteremia and fungemia have largely been unsuccessful, and it is not recommended. In the setting of an endovascular complication, removal of the catheter is strongly recommended, and systemic antibiotic therapy should be prolonged (i.e., 4 to 6 weeks) (level B-II). In many cases, septic thrombophlebitis may require surgical attention. Tunnel and pocket infections may also require débridement, but after the catheter is removed, 7 to 14 days of antimicrobial therapy should be sufficient.