could not be extrapolated to all practices in the United States.

We would like to point out that the 63 cases of allograft-associated infections cited as “alleged” were actually definite: all were ascertained through strict case definition, active case finding, detailed tissue trace-back investigation, and microbiological confirmation. Notwithstanding the excellent safety record of allograft tissues, we believe that there is a low yet tangible risk of infection associated with implantation of aseptically processed tissues.

Our report serves only to highlight areas where patient safety could be enhanced. This is consistent with previous discussions that tissue banks need to consider new tissue sterilization technologies for enhancing patient safety [3–5]. It is unfortunate, therefore, that this safety message has been interpreted as an “advertisement for sterilized tissue.”

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**References**


The findings and conclusions in this letter are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention.

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**Gram-Negative Bacterial Surgical Site Infection and Intensive Care Unit Patients**

Sir—The recent article by Gaynes and Edwards [1] of the Centers for Disease Control and Prevention (CDC) provides their analyses of clinical microbiological data from a 19-year time period. More than 400,000 bacterial isolates were obtained from patients who received treatment in intensive care units, and Gaynes and Edwards [1] constructed secular trend plots for the relative proportions of gram-negative and gram-positive organisms isolated from nosocomial infections each year during the period 1986–2003. The 4 major types of nosocomial infection referred to were pneumonia, urinary tract infection, bloodstream infection, and surgical site infection (SSI).

The concept of SSI was formally defined by the CDC in a 1992 article by Horan et al. [2]. This category contains 4 phenotypically unique types of infection: superficial incisional, deep incisional, organ, and space. It is very troublesome to find, in table 1 of the article by Gaynes and Edwards [1], a representation that pathogens were isolated from patients with SSI in 1975. It is even more troubling to see a graphic in the article (Gaynes and Edwards’s figure 1 [1]) that purports to show data for SSI pathogen isolates from the time period 1986–1992, which is ~37% of the time period during which isolates were obtained.

It is my understanding that, before 1993, hospitals participating in the National Nosocomial Infections Surveillance system gathered information regarding surgical-wound infections (i.e., incisional infections) along with information concerning other nosocomial infections. All CDC publications prior to 1992 are consistent with that interpretation. The major reason for the creation of the SSI concept as defined by the CDC in 1992 was to broaden the scope of surveillance to include nonincisional infections that uniquely affect surgical patients.

How could microbiological data have been gathered either during calendar year 1975, or during the 7-year period ending in December 1992, for a category of infection not even defined by the CDC until October 1992? Gaynes and Edwards [1] report National Nosocomial Infections Surveillance data for “SSI” in 1975 and data from 1986 to 1993 that must pertain only to incisional infections (i.e., surgical-wound infections). If so, this nontrivial fact should have been clearly explained, especially because the article is the product of CDC personnel. Unless I have seriously misinterpreted the authors’ descriptive terminology, it seems that Gaynes and Edwards [1] want readers to believe that the CDC and National Nosocomial Infections Surveillance volunteers were coherently using the SSI concept from 1975 until 1993.

Language is powerful, and confusion regarding SSI nomenclature persists in articles written by our colleagues in surgery, epidemiology, and infectious diseases [3]. It is unsettling when CDC authors appear to misuse SSI terminology. Health care workers who care for critically ill surgical patients would have been curious to see the ratio of gram-negative and gram-positive isolates for each of the 4 types of infection subsumed under the category label SSI since 1992. Moreover, did the change to a broader National Nosocomial Infections Surveillance scope in 1993 (i.e., the introduction of the SSI concept) have any link to the peculiar crossover of SSI pathogen isolate frequencies that began in...
the mid-1990s but that was not seen for any other nosocomial infection category?

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References


Reply to Lee

Sir—In response to Dr. Lee [1], I must begin with the 1992 article [2] that he refers to in his letter. That article described a modification of the definitions devised by the Centers for Disease Control and Prevention that had subdivided postsurgical infections into site-specific categories but did not change the universe of what comprises those infections. Rather, the modification, done in cooperation with the American College of Surgeons, changed the subdivisions (termed specific sites of infection) of all infections in the surgical field from 2 categories (superficial and deep surgical-wound infections) to 3 (superficial incisional, deep incisional, and organ and/or space infections). When combined with the 1 major category of infection, that is, surgical site infections (SSIs, which had been previously referred to as surgical wound infections [SWIs]), no changes occurred. The modification was made with the express desire to allow trend analysis of postsurgical infections. Thus, Dr. Lee is incorrect when he states, “It is my understanding that, before 1993, hospitals participating in the NationalNosocomialInfections Surveillance system gathered information regarding surgical wound infections (i.e., incisional infections) along with information concerning other nosocomial infections” [1, p. 577]. Deeper organ and/or space infections, such as intra-abdominal abscesses, were included in the definition of SWIs, a difficulty to which the Centers for Disease Control and Prevention attempted to respond in 1992 with the modification of the definition to include an additional specific site. As a result, the pathogen analysis beginning in 1975 remains valid with the definition of SWIs used before 1992 and with the definition used in 1992 and after.

The trend analysis of pathogens associated with SSIs and SWIs may be problematic if the distribution of specific sites, such as deep incisional–or organ and/or space–specific sites, compared with superficial incisional–specific sites, changed during the period 1975–2003 [3]. Additionally, the trend analysis of SSIs and SWIs could have been affected by changes in the distribution of surgical procedures as well as by changing surgical methods and procedures, for example, the use of laparoscope, or changes in the microbiology laboratory identification procedures. The site of surgical infection is not the only site where other factors could have influenced the trend analysis; the bloodstream and the respiratory and urinary tracts may have had similar factors influencing their analysis. Determining the possible effect of changes in these factors was beyond the scope of my colleagues and my overview article [3] and is the subject of ongoing analysis for each major site.

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Kikuchi’s Disease or Kikuchi’s Syndrome?

Sir—In the 15 October issue of Clinical Infectious Diseases, Noursadeghi et al. [1] report on an interesting and well-documented case of Kikuchi’s disease involving a patient who presented with fever and aseptic meningitis and who responded to steroid treatment. The differential diagnosis of tuberculosis meningitis is opportune and has been thoroughly discussed. However, another particular infectious disease to be discussed further in-depth is cat-scratch disease caused by Bartonella henselae [2]. Although rare, CNS involvement in acute B. henselae infection, including aseptic meningitis, has been reported, in addition to the typical regional lymphadenitis [2]. The patient improved on receipt of antituberculous treatment (probably including rifampin) and steroids. Of note is that rifampin is very active against B. henselae [3], and response to steroids has also been reported in the treatment of B. henselae encephalitis [4].

In the case under discussion, previous contact with cats is not specifically mentioned, and, although negative serologic