District (Ben Taub and Lyndon B. Johnson hospitals) and several private institutions care for more HIV-positive patients than our institution. Several primary care physicians at these private institutions have self-declared "acquired immunodeficiency syndrome (AIDS) practices."

There is a consensus that voluntary HIV testing to identify infection patients should be accomplished. The best approach to testing-who, how, where, and when-has not been established. We believe the authors took exception to the word "widespread" (not used in the first line of the abstract). As discussed in our article, we also believe in a targeted approach, but how best to select the population to test deserves further study.

The statistical difference between the seroprevalence of patients agreeing to and declining HIV screening reached a *p* value of .12-a value that is generally interpreted as not statistically significant. Before this study, our hypothesis was that we would see a statistically significant difference in the HIV seroprevalence of these groups; we did not. Whether the difference between 0.26% and 0.60% is medically significant, even though not statistically significant, is left to the reader. With a bigger sample size or different population, statistical difference might be shown, but it was not in our study. It takes a leap of faith to believe "these data clearly suggest that persons at risk will selectively refuse participation."

As we stated, the screening process did discover 12 patients not previously known to be HIV positive by the admitting physician. Even the patient who knew he was HIV-positive did not convey this information to healthcare workers until he was told of the positive serology. Some of these patients would have been found to be HIV-positive at some time during hospitalization, but when and how many are not known.

In assessing the financial aspects of HIV testing, it is important to distinguish between cost and charges. Many variables must be included in any financial equations, and several were discussed in the article. Certainly, the discovery of one HIV-positive patient and the subsequent prevention of one hospitalization for *Pneumocystis carinii* pneumonia or the prevention of transmission to one sexual partner would save huge sums of money. The cost analysis of an HIV screening program is very complex. HIV testing has not been a financial loss for our institution, although it might be for a public hospital. We agree that counseling high-risk patients is a valuable approach in controlling the AIDS epidemic and that screening programs are a golden opportunity for counseling. More work needs to be done in this area.

As we discussed in our article, there is considerable difference in the interest demonstrated by various TMH physicians and other healthcare workers in this screening program. The majority of physicians are supportive of the program, but vary in their degree of active participation. Few physicians are against the screening program. The scope of this article did not include long-term follow-up on HIV discoveries; however, each of these patients was counseled by physicians with expertise in the care of HI&elated disease and given the opportunity for prompt and appropriate medical care.

The purpose of our report was to share the "good and bad" experience of admission HIV screening in a large hospital. The program is well accepted by patients and healthcare workers. Although it is not perfect and a targeted population approach would be much more cost effective, hospitals are practical places for HIV screening, and the benefit is to the patient.

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Clinical Predictors of Infection of Central Venous Catheters Used for Total Parenteral Nutrition

To the Editor:

We were pleased to read the article "Clinical Predictors of Infection of Central Venous Catheters Used for Total Parenteral Nutrition" by Armstrong et al.[1] However, we disagree with the methods used by the authors and, accordingly, with some of the conclusions reached in their study.

The authors support and implement a predictive protocol for catheter sepsis based exclusively on the clinical and microbiological investigation of the skin close to the catheter entry site. This alone could invalidate their study because many of these infections are caused by endoluminal hub contamination.[2] Additionally, there are serious methodological pitfalls, the most important of which are the following. First, no clear criteria for catheter removal are given. Second, the skin is not sterilized after the skin culture has been taken and before the catheter is removed. This may result in spurious extraluminal contamination of the catheter tip. Thiid, because only the semiquantitative extraluminal culture method was used, endoluminal contamination might have been overlooked in some cases.[3]

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Fourth, for the statistical analysis, the authors use all catheters "infections confirmed" and "infections probable" in a single data pool.

These methodological flaws result in unreliable results, particularly those concerning the clinical significance of a positive skin culture and the discordance between clinical findings (temperature) and microbiological results. Thus, we cannot agree with the following conclusion written in the abstract: "Another source of fever is likely if inflammation is absent and there is...colonization by less than 50 colonies of coagulase-negative staphylococci at the insertion site." This approach would dismiss the catheter as cause of fever in all patients with hub-related catheter sepsis.

In our experience (unpublished observations), the sensitivity and specificity of surveillance skin cultures are too low to recommend their routine use. Furthermore, study protocols on catheter sepsis should incorporate means to detect endoluminal catheter contamination in order to properly identify those catheters infected through the hub.[4]

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The authors were asked to respond to this letter

We are pleased to respond to the comments of Drs. Segura and Sitges-Serra. First, we will respond to their methodological concerns, and then we will address the mechanism by which catheter infections develop as it relates to their comments about our conclusions.

The first concern was the criteria we used for catheter removal. These criteria have been published in a related article.[1] The second concern was the method used to remove the catheters to avoid contamination during withdrawal of the catheters. We state in the methods section that "the insertion site was cultured and then cleaned with an alcohol pledget. The catheter was withdrawn at a right angle to the skin to prevent contamination on removal." Alcohol also has been used by other investigators to cleanse the skin prior to catheter removal.[2-5]

Next, we will respond to the fourth comment and then discuss their third comment below when we examine the data on the pathogenesis of catheter infections. Drs. Segura and Sitges-Serra suggest that combining confirmed catheter infections and probable catheter infections could lead to unreliable results. We disagree with this comment. The only difference between confirmed catheter infections and probable catheter infections was that the latter were removed accidently. They were cultured promptly using the same technique. To compensate for possible contamination of these catheters during accidental withdrawal, we set the cutoff for colony counts at more than three times the criterion used for catheters removed under controlled conditions (50 rather than 15).

As stated in the discussion, we showed in a related publication that it was highly likely that these catheters were infected.' First, the lowest colony count on semiquantitative culture of catheters in the group with probable infections was 163. Second, the median colony counts for catheters with confirmed and probable infections were similar (>400 and >310, respectively), and the median colony count for uninfected catheters was zero. Thus, there was a wide margin between the median colony counts for catheters with confirmed and probable infections and catheters that were uninfected. Third, six of the isolates recovered on semiquantitative culture of the catheters with probable infections are common causes of catheter infection, and the catheter infected with *Serratia marcescens* yielded confluent growth on semiquantitative culture. For these reasons, we feel that pooling the catheters with confirmed infection and those with probable infection was entirely appropriate.

The remainder of the comments by Drs. Segura and Sitges-Serra relate to the pathogenesis of intravascular catheter infections. They contend that endoluminal catheter contamination by microorganisms that enter at the catheter hub is an important pathogenetic mechanism for infection of intravascular catheters. They state that failure to take this mechanism into account in our study invalidates our conclusions.

We strongly disagree. The overwhelming bulk of the evidence published in the literature supports migration of microorganisms on the skin surface into the subcutaneous catheter tract with extension to the fibrin sheath on the intravascular portion of the catheter as the primary pathogenetic mechanism for development of intravascular catheter-