Primary bacteremia and short-term catheter-related bacteremia (PB/CRB) are relatively frequent infections in critical patients. In a recent one-day cross-sectional prevalence evaluation of 1265 Intensive Care Units (ICUs) in 75 countries, close to 20% of the 14,414 patients had a diagnosis of bacteremia or vascular catheter-related infection. In Spain, the data collected by the ENVIN-HELICS registry corresponding to the year 2010 indicate that PB/CRB, excluding secondary bacteremia and other foci, account for approximately 17% of all infections controlled by the registry. In the year 2010 an important decrease has been observed from 2.48 episodes per 1000 intravascular (arterial and central venous) catheter days in 2009 to 1.82 episodes in 2010.

The most common etiology of PB/CRB corresponds to coagulase-negative Staphylococcus (CNS), representing close to 40% of all episodes, and 72% of the episodes produced by grampositive strains. Staphylococcus epidermidis causes approximately two-thirds of all PB/CRB due to CNS, this in turn representing 25% of the total. The usual origin is taken to be the surrounding skin and catheter connections.

There is a repeatedly demonstrated significant association between the development of infection during admission to Intensive Care in general and PB/CRB in particular, and morbidity and mortality or severity. For example, mortality odds ratios (ORs) of 1.7 have been seen in patients with PB/CRB. However, there are doubts regarding the repercussions directly attributable to these infectious complications in critical patients, particularly in the case of PB/CRB due to CNS. The described association might not imply causality, but may simply constitute a marker of patient severity and/or prolonged stay subject to risk factors in Intensive Care. For this reason, and because in many cases catheter removal appears to eliminate the origin of the infection, it is common practice to grant less importance to this type of infectious complication than to other infections such as for example ventilator-associated pneumonia (VAP).

The design of a study allowing us to know the morbidity-mortality attributable to PB/CRB due to CNS is full of difficulties. The main problem is to define an adequate control population, i.e., a similar group of patients with the same risk factors, but who do not develop PB/CRB. A partial option in this sense could be the analysis of clinical trials with positive results in which the efficacy of preventive measures against CRB are evaluated. However, one of the main trials of this kind, in which a reduction in CRB was observed, failed to even mention the possible repercussions upon morbidity-mortality of this effect. Another recent and important trial demonstrating the efficacy of chlorhexidine-impregnated dressings in preventing CRB has reported no differences in the duration of stay in the ICU or in mortality between the study groups.

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In the present issue of *Medicina Intensiva*, Olaechea et al. present the results of an original work addressing the repercussions of PB/CRB due to CNS. The study is potentially of great relevance, since it involves an unusually large sample of 85,000 critical patients included in the ENVIN-HELICS registry covering the period from 1997 to 2008. At least in terms of its source data, this study undoubtedly will constitute a reference for future observational studies or trials designed to assess the efficacy of preventive measures. A double analysis is made, comparing the following: 1) PB/CRB due to CNS versus cases caused by other pathogens; and 2) cases versus controls (1:4), respectively defined as the group of patients with an episode of PB/CRB due to CNS as sole infection acquired in the ICU, and the group of patients without infection acquired in the ICU. The findings of the statistical comparison made by Olaechea et al. suggest that the patients who develop an episode of PB/CRB due to CNS do not suffer increased mortality as a result – though their stay in the ICU is effectively longer. These data are important, and confirm previous findings, though in this case the sample size is much larger and the control group has been more carefully selected. However, in attempting to retrospectively “extract” morbidity-mortality attributable to a given infection, it is unfortunately not possible to entirely discard the uneven influence of risk factors for infection and other types of complications in the principal study variables. On the other hand, the comparison of PB/CRB due to CNS with processes of other etiologies may cause the selection of populations with somewhat different characteristics. By definition, the point of entry to the bloodstream in primary bacteremia is not known and is possibly variable, involving the respiratory, digestive or urinary mucosa, and possible false bacteremias secondary to an unidentified focus must also be taken into account. Therefore, such processes may occur in patients with characteristics different from those of subjects in which the point of entry is preferentially a vascular catheter, as in the case of CRB due to CNS. Likewise, the choice of a comparator group “without any nosocomial infection” is an arguable decision, since here again we are unable to discard the introduction of bias on eliminating the risk factors. Perhaps the control group for this second analysis should have been selected from patients with an intravascular catheter who do not develop PB/CRB, i.e., comparing patients with an equal duration of the main risk factor (the presence of a vascular catheter).

In conclusion, the existing data appear to indicate that the development of PB/CRB due to CNS in a critical patient does not worsen survival but significantly extends admission to Intensive Care, and probably also increases the associated costs. Future prospective studies of the repercussions of preventive measures logically preferentially targeted to PB/CRB due to CNS upon morbidity and mortality in the critical patient will allow us to clarify the issue raised by Olaechea et al.

**References**