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surgical indication, timing of operation, and conduct of the operation by an experienced high-volume surgeon and surgical team. Even then, however, the results will never be generalizable to centers that average too few patients with IE to achieve the experience necessary for excellent outcomes.

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<http://dx.doi.org/10.1016/j.jtcvs.2017.09.066>

**KEEP FUMBLING AROUND
IN THE DARK WHEN IT
COMES TO INFECTIVE
ENDOCARDITIS, OR
PRODUCE NEW, RELIABLE
DATA TO REDESIGN THE
GUIDELINES?**

Reply to the Editor:

We thank Dr Bando¹ for his valuable comments and we share his view regarding the construction of an infrastructure for randomized controlled trials and a registry of endocarditis. The scarcity of data emerging from the few available randomized trials significantly restricts our ability to draw conclusions on the most appropriate treatment of complex endocarditis or define a line of demarcation between allogenic tissue and mechanical or xenograft prostheses. The benefit of early surgery over medical treatment remains established,² and a randomized study revealed better long-term survival and lack of thromboembolic complications with the use of allograft over conventional prostheses.³ Additionally, several studies are insisting on the biomechanical and hemodynamic advantage provided by allogeneic and autologous substitutes, which is reflected in the favorable long-term outcomes in terms left ventricular function.⁴

We agree with Bando¹ about the potential unavailability of allogenic substitutes in certain parts of the world. In Africa and Asia, the limitation to their use is exacerbated by other factors, such as lack of specific training and rules imposed by social and religious education. However, when thinking of a revision of the current guidelines on infective endocarditis,⁵ several factors need to be taken into account. First of all, over the past 15 years, infective endocarditis has seen a substantial change in its etiology and pathogenesis. Changes in the characteristics of the patient population—in the geographic distribution of infective endocarditis as well as the selection of antibiotic-resistant germ strains—have significant implications in regard to surgical strategy and approach. An international registry to collect information on the epidemiology, etiology, drug resistance, and geographic distribution is of paramount importance in this context. Also, data on the actual trend of use of synthetic or autologous tissues around the world for the reconstruction of extensive lesions of the annulus and related cardiac structures, along with the data on their long-term outcomes, would be extremely valuable to shed light on a field that is still dominated by expert consensus rather than evidence from adequate trials.

Another important point to consider is the morphology of the vegetation, which represents a discriminatory criterion

Authors have nothing to disclose with regard to commercial support.

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for immediate surgery. Vegetation < 10 mm of 1 aortic leaflet or mitral scallop with transesophageal echocardiographic evidence of low embolic risk may not require immediate surgical treatment. Single or multiple vegetations larger than 10 mm involving more than 1 leaflet or scallop indicates immediate surgery. In these cases, despite negative blood culture, surgery should not be deferred while other microbiologic assessments are performed because of the consistent risk of neurocognitive complications or end-organ damage with clinical deterioration. Urgent surgery should be considered in infectious endocarditis of prostheses.

We agree with Bando's¹ conclusions regarding the need for an international project that effectively addresses all the issues related to endocarditis. The goal should be to obtain reliable data and redefine the guidelines and establish the most appropriate strategy to assist these patients.

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OBESITY AS A CONFOUNDING FACTOR IN A STUDY EVALUATING THE ROLE OF M1 POLARIZATION IN AORTIC VALVE CALCIFICATION

To the Editor:

We recently read with interest the great article by Li and colleagues¹ showing that macrophage infiltration and polarization influence the process of calcification in the human aortic valve. In their study, the infiltration of classically activated (M1) phenotype macrophages was more predominant in the calcified aortic valves than in the noncalcified aortic valves.

It is well known that the M1 macrophage infiltration and M1 polarization are increased in patients with obesity, which could induce chronic inflammation.^{2,3} In the study of Li and colleagues,¹ the body mass index of the group with calcified aortic valves was statistically significantly higher than that of the group with the noncalcified aortic valves. Is there any chance that body mass index is working as a confounding factor in the evaluation of whether M1 macrophage infiltration pattern is different between the groups of patients with calcified aortic valves and with noncalcified aortic valves?

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