follow surgical oncologic principles and should be regarded as a bailout procedure and used judiciously.

Raja M. Flores, MD
Division of Thoracic Surgery
Mount Sinai School of Medicine
New York, NY

Reference

doi:10.1016/j.jtcvs.2012.03.045

WILL THE REAL CULPRIT OF HETEROGRAFT VALVE CALCIFICATION PLEASE STAND UP?

To the Editor:

Sinha and colleagues are not the first to implicate glutaraldehyde as a culprit for tissue valve calcification. Although they are to be commended for their careful methods and the suggestion that glutaraldehyde concentration and exposure times appear to correlate with the degree of calcification, they unfortunately failed to reach a different conclusion; that is, calcific degeneration of heterograft tissues is primarily an immunologic phenomenon.

Alain Carpentier and his wife Sophie, a biochemist, are given credit for the introduction of aldehyde chemistry to the modern manufacture of heterograft tissue valves. At the time, they did not advance an argument that aldehyde preservation rendered heterograft tissues nonimmunogenic. That perception belongs to perspicious marketing departments within the tissue valve industry. The observation that calcific degeneration of heterograft valves is an age-related phenomenon was an early and important clue to the problem of tissue valve durability. These issues notwithstanding, the Carpentiers’ discovery had already spawned the multibillion dollar industry of heterograft tissue heart valves.

More than 30 years ago, Salgaller and Bajpai detected both cellular and humoral immune responses to glutaraldehyde-treated and untreated bovine pericardium. Their data provided the first real proof that glutaraldehyde-preserved heterograft tissues are not biologically inert and remain antigentic. The association between a smoldering immune response and tissue valve durability was never widely recognized, however, and glutaraldehyde continued to be the presumed cause of heterograft calcification, leading researchers and companies to search for new tissue treatments designed to retard calcification.

Love and associates first described the successful use of autologous pericardium briefly treated in 0.6% glutaraldehyde for use as a stent-mounted valve replacement. Since this introduction, multiple investigators have reported wide success with the use of autologous pericardium briefly treated in 0.6% glutaraldehyde for the replacement of semilunar heart valves and the repair of damaged and shortened mitral valve leaflets. All these techniques have proved durable, and in none of the published experiences, including those with pediatric or very young patients, has calcific degeneration been considered a limitation.

The more than 20-year clinical experience with autologous pericardium briefly immersed in glutaraldehyde and used for valve reconstruction should finally dispel any perception that aldehydes are directly responsible for calcific degeneration of tissue valves.

Charles S. Love, BA
Spot On Medical, LLC
Santa Barbara, Calif

References

doi:10.1016/j.jtcvs.2012.01.085

Reply to the Editor:

We thank Mr Love for responding to our article and for emphasizing the importance of immune mechanisms in heterograft calcification. We are in agreement that aldehyde treatment is just one of several factors that can exacerbate calcification of implanted tissues. In the clinical setting of pediatric cardiac surgery, where autologous pericardium is often used and immune mechanisms are presumably not so important, however, it is the factor that can be most readily modified. Unfortunately, our animal model was not suited to autologous pericardium implantation.

We strongly disagree with Mr Love’s assertion that calcific degeneration never occurs in the pediatric setting when glutaraldehyde-treated autologous pericardium is used for