veno-venous support in cases of right ventricular failure. Although veno-venous support is generally not successful in cases of right ventricular failure, the patient in our series was seen with severe pulmonary hypertension and a large atrial septal defect with a right-to-left shunt. Flow from the cannula was therefore preferentially shunted across the atrial septal defect, resulting in functional veno-arterial support.

We also commend their group for the use of the Avalon dual-lumen cannula (Avalon Laboratories, LLC, Rancho Dominguez, Calif) for patients with a body surface area greater than 2.0. In our initial experience, we were concerned that high enough flow rates to fully oxygenate these patients would not be achievable. We have subsequently, however, used the Avalon cannula with surprising success in patients with a body surface area greater than 2.0.

In reply to the comment regarding range of gas exchange rates in this series, we too generally recorded rates between 200 mL/min and 300 mL/min. Only a single time point was found to have a rate greater than 500 mL/min, and this value may simply represent laboratory error.

This remains an exciting time in the evolution of care for patients with severe lung injury, and further study of the use of ambulatory extracorporeal membrane oxygenation is still required.

Jose P. Garcia, MD
Zachary N. Kon, MD
Bartley P. Griffith, MD
Division of Cardiac Surgery
Department of Surgery
University of Maryland School of
Medicine
Baltimore, Md

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CONCERNING EN MASSE LOBECTOMY

To the Editor:

We read with interest the article entitled, "Video-Assisted Thoracoscopic

Surgery (VATS) Lobectomy: Catastrophic Intraoperative Complications," by Flores and colleagues¹ in the December 2011 issue of *The Journal of Thoracic and Cardiovascular Surgery*. We are interested in their experience and would like to make some comments.

For patient 1 among the 12 cases they described, Flores and colleagues¹ said that the posterior ascending branch of the pulmonary artery had calcified lymph nodes that required dissection. Subsequently, the pulmonary artery was damaged irreparably, and intrapericardial proximal arterial control was needed with a thoracotomy. As a result, a planned right upper lobectomy was eventually converted into a pneumonectomy. We do not know the details of the manipulation, because Flores and colleagues¹ did not describe it further.

We would like to recommend in such cases the "en masse lobectomy." Originally, en masse hilar management was the main technique used in lobectomy, because surgeons warned against hilar dissection and individual ligation for fear of spreading infection by opening inflamed tissue planes.² Times have changed, however, and individual hilar management has become the standard procedure in modern lobectomy. Nevertheless, some authors^{3,4} still report that en masse lobectomy is useful under certain conditions.

From an oncologic perspective, care should be taken when handling malignant lung diseases because of the potential for lymph node metastasis. If no lymph node metastasis is detected preoperatively, and there are inflammatory adhesions to the pulmonary artery, we do not think that dissection is required. Even if this is not the case, we think that an octogenarian would welcome an en masse lobectomy as a thorough pneumonectomy. If no calcifications are found, a mechanical stapler could be applied to the divided root of the right upper lobe, as is done with simultaneously stapled lobectomies.⁵

We believe that this technique might help to avoid catastrophic intraoperative complications. Thus, with an understanding of what has taken place in the past, it should be possible to reshape the present and future.

Mitsuhiro Kamiyoshihara, MD, PhD
Hitoshi Igai, MD, PhD
Takashi Ibe, MD, PhD
Department of General Thoracic
Surgery
Maebashi Red Cross Hospital
Maebashi, Japan

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Reply to the Editor:

With regard to our article, "Video-Assisted Thoracoscopic Surgery (VATS) Lobectomy: Catastrophic Intraoperative Complications,"¹ I agree with Kamiyoshihara and associates that en masse lobectomy (tourniquet lobectomy) is a useful tool in the thoracic surgeon's armamentarium. This procedure has the potential, however, to leave N1 nodal disease behind in patients with lung cancer. The key is to identify the potentially dangerous situation before starting the dissection. Heavily calcified lymph nodes and an artery that is inseparable from the bronchus should be clues to avoid further dissection, and a tourniquet lobectomy may prove to be a very useful alternative. Nevertheless, tourniquet lobectomy does not

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

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

To the Editor:

Sinha and colleagues¹ 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 far enough back into the literature to recognize that most of their questions had already been answered by others.² A closer look at the literature might also have led these researchers to arrive at 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.³ At the time, they did not advance an argument that aldehyde preservation rendered heterograft tissues nonimmunogenic. That perception belongs to perspicacious 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.⁴ 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 Bajpai⁵ 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 antigenic. 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 associates⁶ 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 valves⁷ and the repair of damaged and shortened mitral valve leaflets.⁸ 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

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Reply to the Editor:

We thank Mr Love for responding to our article¹ 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