

sizing,⁴ as observed in our in vitro comparison.¹ Moreover, considering the excessively and surprisingly high regurgitant volumes observed in vitro with the Carpentier-Edwards Magna valve, as well as the minimal tolerability of this valve to stent distortion, according to present work and to other experiences in the literature,^{5,6} the upsizing of the Carpentier-Edwards Magna should be, to our mind, carefully considered.

Unfortunately, “in vivo” comparisons between different prostheses are difficult and misleading; several confounding factors, such as blood viscosity (patient hematocrit), heart rate, left ventricle and mitral valve pattern, cardiac output, septum hypertrophy, systemic hypertension, reduced systemic arterial compliance, and effects of angiotensin-converting enzyme inhibitors in hypertensive patients, data that are rarely reported in clinical studies, are frequently present. These factors may confound the data obtained by echocardiographic studies. Other important factors such as aortic root anatomy or variability in surgical skill and implant technique might indeed affect clinical comparisons. Finally, echocardiography parameters (ie, effective orifice area calculated by using the continuity equation) have inherent variability that is mainly related to the techniques used for its measurement, as well as to flow dependency. For these reasons, we maintain that it is hazardous to conclude that a prosthesis model is the gold standard by interpreting only clinical results.

The system that we have used has a virtually rigid arrangement section downstream from the aortic valve, which represents perhaps the single largest distortion from reality. Attachment of a small compliant device to the downstream section could give a significantly different system performance, mimicking an in vivo setting such as an aorta setting. However, if we compare two heart valves in this modified system, we would expect to appreciate the same differences between the two different valves. Therefore, the pulse duplicator device is not really designed to give an accurate representation of the true anatomy; rather, it is a system that provides an extraordinary and unquestionable bench test for comparison of different prostheses.

The most striking finding of our study was the ability to obtain a unique hydrodynamic comparison of different models of supra-annular tissue valves fitting a 21-mm di-

ameter artificial aortic annulus, regardless of the labeled manufacturers’ size. This comparison can be helpful in assisting surgeons’ decisions.

Gino Gerosa, MD^a

Vincenzo Tarzia, MD^a

Giulio Rizzoli, MD^a

Tomaso Bottio, MD, PhD^b

Cardiovascular Institute

University of Padova, Italy^a

Cardiovascular Institute

University of Brescia, Italy^b

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Nitric oxide precursors and congenital cardiac surgery: A randomized controlled trial of oral citrulline. Definition of pulmonary hypertension in Fontan circulation?

To the Editor:

We read with great interest the article from Smith and colleagues¹ describing the effect of citrulline supplementation in reducing postoperative pulmonary hypertension. This is very appealing therapy because it is reported to be effective and without any side effects.

Nevertheless, we have a concern with the definition of pulmonary hypertension. The authors have used the accepted definition of pulmonary hypertension as a mean pulmonary arterial pressure of at least 25 mm Hg² or exceeding 50% of the mean systemic artery pressure. Although acceptable for patients presenting with ventricular or atrioventricular septal defect, as well as transposition of the great arteries, this definition is more questionable for bidirectional Glenn and Fontan procedures.

A prerequisite for a successful bidirectional Glenn or Fontan procedure would be a mean pressure of less than 15 mm Hg. In addition, a pressure of more than 12 mm Hg in a Fontan circulation would be considered a suboptimal result.

Taking 25 mm Hg as a limit to describe high pulmonary pressure in this group induces a significant bias to our point of view. Assuming that we use 15 mm Hg as the limit for the Glenn and Fontan group, all patients (11/11) presented with pressure of more than 15 mm Hg in the placebo group, and 9 of 10 patients presented with this pressure in the citrulline group. Taking 20 mm Hg as a superior limit, 4 of 11 in the placebo group presented with pressure over the limit compared with 4 of 10 in the citrulline group, respectively.

On this basis, we think that groups with shunt lesions and biventricular repair versus single-ventricle physiology should not be evaluated similarly with regard to pulmonary hypertension.

Another concern, and this is applicable to both shunt lesions or single-ventricle circulation, is that nothing is reported with regard to postoperative care, and several confounding factors can increase or decrease pulmonary arterial pressures in this period, such as pH, PO₂, sedation, ventricular function, pulmonary wedge or atrial pressures, and inotropic/vasodilatory support for 48 hours.³

We think these points are of major importance because citrulline levels might not have been the sole responsible variable for reducing pulmonary arterial pressure.

Maurice Beghetti, MD

Yacine Aggoun, MD

Eduardo Da Cruz, BSc, MD

Unité de Cardiologie Pédiatrique

Hôpital des Enfants

Genève, Switzerland

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Carbon dioxide embolism induced by endoscopic saphenous vein harvesting during coronary artery bypass grafting

To the Editor:

We read with great interest the article by Dr Kypson¹ reporting a case of carbon dioxide embolism during saphenous vein harvesting for coronary artery bypass grafting (CABG) in a 43-year-old woman. Saphenous venous conduits were harvested by the endoscopic method with carbon dioxide insufflation. On weaning from cardiopulmonary bypass (CPB), the endoscope was reinserted to control hemostasis and a massive gaseous embolism occurred that did not respond to pharmacologic resuscitation, leading to the need for reinstatement of the CPB. Direct inspection revealed a torn saphenofemoral junction that was resealed. Finally, air locks in the venous circulation and right side of the heart disappeared and the hemodynamic parameters returned to normal.

Our group^{2,3} has previously reported on 2 cases that have occurred in more than 1000 procedures to date. We would like to raise some issues concerning this observation.

First, although carbon dioxide embolism is clearly due to direct entry into the injured vein, the cause of this lesion was not discussed in the article. Reintroduction of the scope and gas reinsufflation in the operating tunnel at the end of the operation to secure hemostasis when liquid and blood accumulation may have occurred over several hours appear, at best, of little use owing to poor visualization, and they may be potentially dangerous as this maneuver was likely responsible for the tear. Hemostasis is best secured at the time of harvesting and

may be revised by direct visualization of each venous end ligation through the skin incisions. We routinely wrap the tunnel site with circular bandaging immediately after vein extraction and for the duration of the cardiac procedure, after which the bandage is removed, hemostasis double checked by evacuation (if necessary) of liquid from the tunnel, and elastic bandaging for 24 hours postoperatively. Need for direct revision of leg hemostasis is rare in our experience of more than 1000 endoscopic vein harvestings.⁴

Second, although quickly suspected, the diagnosis of carbon dioxide embolism was not made before CPB reinstatement, since at the time of the gaseous reinsufflation, the transesophageal echocardiographic (TEE) monitoring was discontinued. Since it has been shown to be the most effective method for detection of gaseous embolisms,⁵ TEE should always be available during insufflation.

Last, the author states that this complication was "unanticipated."¹ Although massive gaseous embolism with significant clinical implications is anecdotal,⁵ it has now been well described by our group² and other authors. Moreover, since the total incidence of introduction of carbon dioxide in the venous circulation reaches a 17% rate,⁵ physicians should certainly be aware of this risk, search and take into account eventual predisposing factors,² provide TEE monitoring during the whole procedure, and avoid repeated gas insufflations. Endoscopic venous harvesting is a safe procedure so long as the necessary precautions are taken.

Arnaud Mommerot, MD

Louis P. Perrault, MD, PhD

Department of Surgery and Research Center
Montreal Heart Institute and
Université de Montréal
Montreal, Quebec, Canada

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Reply to the Editor:

I thank Drs Mommerot and Perrault for their contributions to the literature regarding endoscopic vein harvesting and for their comments regarding my recent case report. I would also like to point out the following issues in response to their comments.

The cause of the lesion was not discussed in the article; however, injury to the saphenofemoral junction consisted of a partial tear occurring at the time of initial vein harvest and not at the conclusion of the operation, when the scope was reintroduced into the leg. Nevertheless, carbon dioxide entry into the vascular space was the result of reinsufflation at the conclusion of the case. Drs Mommerot and Perrault are correct in stating that hemostasis is best secured at the time of harvesting and reintroduction of the scope should be minimized. However, if necessary, we do re-examine the tunnel after protamine administration, albeit carefully.

Intraoperative transesophageal echocardiography (TEE) has become a valuable tool for patients undergoing cardiac surgery. We use this technique on all such procedures and do not discontinue its use until the patient is ready for transport to the intensive care unit. In this case, TEE monitoring had been discontinued because the sternotomy incision had been closed. Drs Mommerot and Perrault are correct that TEE is the most effective method for detection of gaseous embolisms. Had the probe been in place during reintroduction of the scope, it might have sooner revealed the impending problem.

In conclusion, endoscopic vein harvesting is a safe procedure with a very low complication rate. Although the incidence of introduction of carbon dioxide into the venous circulation reaches 17%, the inci-