is beneficial to the lungs of pigs under conditions of CPB. Can that be extrapolated to man?

An important issue to be addressed is whether lung ventilation/perfusion, during conditions of CPB, promotes gas exchange. We³ have performed all valve procedures with the heart beating, perfused simultaneously antegradely and retrogradely with warm blood, and with the lungs ventilated/perfused during the cardiac procedure, in an effort to decrease ischemia-reperfusion injury. End-tidal carbon dioxide (ETCO₂), a measurement of pulmonary blood flow and cardiac output, was continuously monitored. ETCO₂ provides a noninvasive estimate of cardiac output, through the ratio of change in ETCO₂ partial pressure and CO₂ elimination after a brief period of partial rebreathing.⁴ Expired CO₂ is assessed by a noninvasive CO₂ monitor that integrates the differential CO₂ Fick partial rebreathing technique.⁵ Preliminary work shows that ETCO₂ is an excellent indicator of lung perfusion, but its clinical value remains unclear. ETCO₂ continued to be present once CPB was initiated and was unchanged throughout the operations. An example of one valve case is shown in Figure 1. Perfusion/ventilation of the lungs did not interfere with the operation.

Recently, we received institutional review board approval for a prospective randomized clinical trial to evaluate lung perfusion/ventilation during CPB. Results will be forthcoming. On the basis of initial experience by Imura and associates² in pigs and our own experience with humans, in long and complicated procedures, or in patients with pre-existing pulmonary diseases, lung perfusion/ventilation is recommended. Potentially, lung injury can be decreased in these patients.

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doi:10.1016/j.jtcvs.2009.08.062

Reply to the Editor:

We thank Dr Macedo and colleagues for their interest in our study on low-frequency ventilation during cardiopulmonary bypass (CPB) that was recently published in the *Journal*.¹ They share our interest in lung injury during cardiac surgery and how to prevent it. We believe this is relevant to current practice with an ever-increasing population of high-risk patients.

The rationale beyond our interest in lung injury is based on the simple observation that in current clinical practice, once CPB is instituted, the patient's lungs are disconnected from the ventilator and left open to the air, fully collapsed and poorly perfused for the duration of CPB. Our report showed significant ischemic damage and atelectasis in the collapsed lungs during and after CPB, and these were improved by using low-frequency ventilation during CPB.¹ Although

the underlying mechanisms are still unclear, there have been reports suggesting that ventilation during CPB may reduce damage in the lungs.² We have taken this forward and are now about to start a large clinical trial to assess low-frequency ventilation in clinical practice.

The issue of pulmonary perfusion during cardiac surgery is an interesting one. Current practice is based on the assumption that bronchial blood supply is sufficient for lung protection during CPB.³ Yet, during cardiac surgery the bronchial circulation (only 1%–2% of the cardiac output) is jeopardized by CPB and surgery-related factors, including lung collapse, internal thoracic artery harvesting, aortic manipulation, hypothermia, and loss of pulsatile flow. Schellensak and colleagues⁴ showed that there is significant decrease in bronchial arterial flow during the initial phase of total CPB and that flow remains low until the end of CPB, returning to normal by 60 minutes of reperfusion.

This highlights the importance that pulmonary circulation may have for alveolar oxygenation, which is also supported by the observation that during lung transplantation surgery the bronchial arteries rarely are reconnected without obvious lung ischemia.

Pulmonary artery perfusion is an obvious potential intervention to prevent lung ischemia. Along with Dr Macedo and colleagues, we are currently focusing on this in ongoing experimental research.

Others have suggested that ventilation during CPB can reduce lung injury.^{4,5} In our view, the safety, efficacy, and surgical practicality of pulmonary perfusion during CPB remains to be proven beyond any doubt before establishing it in clinical practice. Also, there is still a lot of research to undertake to ascertain the mechanistic insights, the dominance of ventilation versus perfusion or vice versa as a protective intervention, and the best route or mode of delivery of pulmonary perfusion.

With regard to the possible resistance by surgeons to adopt these interventions, we think the availability of compelling evidence in support of these interventions to prevent lung injury will eliminate any practical concerns.

Dr Macedo and colleagues refer to a trial that will assess the effect of lung perfusion/ventilation during CPB. We look forward to their results, which will hopefully add further knowledge to face the continued challenge of lung injury during cardiac surgery.

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doi:10.1016/j.jtcvs.2009.09.002

PULMONARY AUTOGRAFT FOR MITRAL VALVE REPLACEMENT: MAKING A SIMPLE PROCEDURE COMPLEX?

To the Editor:

I read with interest the article by Kumar and colleagues¹ about the use

of pulmonary autografts for mitral valve replacement. I agree with the authors that cardiac surgeons working in Third World countries are faced with a major problem in the management of large numbers of patients with valvular disease. Most of those patients are of low socioeconomic and educational class, living in remote villages and mountains. There is no family practice or general practitioner system to provide early diagnosis and referral, postoperative follow-up, and anticoagulation control. Patients usually present late with advanced disease that makes repair impossible. The magnitude of the problem is escalated in children and women of childbearing age because we have no ideal valve substitute.

The pulmonary autograft concept is attractive but still has its drawbacks and complications. The procedure is complex and technically demanding, requiring at least double the usual crossclamp and bypass times. The procedure is not free of charge because there is a price for the homografts or preparation of the autografts, plus the added cost of complications. The mortality of approximately 15% reported by the authors is still high compared with single mitral valve replacement. Kabbani and colleagues² reported 5% early mortality and 6% late mortality, but as they stated, it is "clearly related to the procedure," which entails higher overall mortality. The issue of converting a single-valve into a double-valve disease is irritating because we have to expect long-term sequelae of pulmonary and mitral valve failures. The main concern with pulmonary autografts or homografts is early calcification. The removal of calcified grafts, as experienced in aortic homografts, is usually disastrous and has a high complication rate, including left ventricular rupture. The same complications are associated with the use of aortic or mitral homografts for mitral valve replacement. All of these autografts and homo-

grafts have to compete with the standard stented bioprostheses, which are improving and technically easier to insert, and require no anticoagulation. At the present time, bioprosthetic valves remain the standard in these patients, and manufacturers have shown us better preservation and longer durability.³

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doi:10.1016/j.jtcvs.2009.08.049

Reply to the Editor:

We are thankful to Dr Khaled E. Al-Ebrahim for his comments and critique. These were the same arguments raised when the Ross procedure for aortic valve replacement was first reported by Donald Ross in 1967.

The patients undergoing operation are young and clearly unsuitable for tissue valves. The surgery is indeed complex but provides superior hemodynamic results with a higher valve area. In addition, these patients only require an echocardiogram at yearly intervals, receive no medication 1 year after surgery, and do not require blood tests. The most important advantage is that it is a living valve, and we now have more than a 15-year follow-up of our own results for the Ross (aortic valve replacement) procedure. There has been no reoperation in the adults with a pulmonary homograft.

We believe these early results will translate into superior outcomes in