Reply to the Editor:

We thank Takagi and colleagues for their interest in our work and their update, which completes and supports our results.¹ Among the RAPCO reports published during the years covered by our research (January 1966–March 2009), we chose to include the study by Buxton and colleagues² published in 2003 and exclude the report by Hayward and collaborators³ published in 2007 because the latter study included patients undergoing serial angiography and, more importantly, because in that analysis saphenous vein was grafted to second-, third- and fourth-order coronary vessels, thus affecting overall patency rate.

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**FONTAN HEMODYNAMICS:
WHAT IS THE PROBLEM?**
To the Editor:

Congratulations to Dasi and coauthors¹ for their valuable study on Fontan hemodynamics. Eleven patients underwent lateral tunnel procedures, and 11 underwent total cavopulmonary connection through an extracardiac Fontan operation. We have previously proposed that contractility and ventricular stress tests should be reevaluated

with different measurement methods,² although our opinions do not coincide with those of some other authors.³ We think that there should be a consensus regarding methods used to measure contractility and ventricular stress with respect to Fontan hemodynamics. Consideration of our opinions on this issue by Dasi and coauthors¹ would be appreciated and would help us in solution of this enigma.

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Reply to the Editor

We thank Kestelli and colleagues for their letter on this important topic. Fontan failure^{1,2}—defined here as poor prognosis after the Fontan operation—may occur at either an early or a late stage. Most late failures are in older patients with the classic atriopulmonary connection, with problems including arrhythmias, thromboembolism, heart failure, and pulmonary venous blockage. Early failures include arteriovenous malformations and protein-losing enteropathy, occurring mostly in patients with a total cavopulmonary connection. Heart failure appears eventually if no other failure mode manifests. These

varying failure modes clearly show that the problem is complex, multifactorial, and still unfolding as more and more patients who underwent total cavopulmonary connection in the mid 1990s are now reaching adulthood. Even though all these patients have a single-ventricle physiology that is inherently preload limited with increased afterload,³ it is not yet clear whether preload limitation is the only cause. There are other independent factors (quality of connection, afterload, underlying congenital defect).

The specific modes of failure for a given patient depend on the severity of the underlying congenital heart defect, efficacy of the palliative approach chosen, and the health and composition of the interacting organ system, including the lungs and abdominal viscera. We have just begun a National Institutes of Health–funded predictive study to help determine the specific criteria for efficient management of these patients. For instance, patients in whom the hepatic flow return is biased toward a single lung may have arteriovenous malformations develop. Those with a highly energy-dissipating total cavopulmonary connection may have decreased preload, poor exercise tolerance, or high central venous pressure, leading to protein-losing enteropathy or heart failure. There is no predictive model currently available that can determine the precise contributors to and mechanisms of failure in a given patient, even when all hemodynamic information is available. We hope that our ongoing research will provide more definite answers to these questions in the coming years.

In the final analysis, for many patients with Fontan circulation, survival into the 4th or 5th decade may require innovative solutions, such as that proposed by Kestelli and associates.⁴ There are many concerns regarding the proposed solution, however, which may be trading one problem for many others. The proposed solution is energy neutral (energy available to drive the circulation is still the same,