To the Editor:

I thank Drs de Almeida, McQuinn, and Sedmera for demonstrating that an increase in ventricular preload is compensated by myocyte proliferation in normal and hypoplastic fetal chick left ventricle. The results of their study clearly provide further impetus to develop fetal cardiac interventions for hypoplastic left heart syndromes with intrinsically normal or only mildly stenotic mitral and aortic valves.

The purpose of my letter is to point out an incorrect concept that the authors put forth in their discussion. Regarding the rationale behind percutaneous ultrasound-guided fetal atrial septoplasty, the authors state from clinical data collected by Marshall et al that “despite technical successful septoplasty the transseptal flow to the left atrium was minimal, pointing out the need of an additional procedure to force the blood into the left heart structures.” The opposite is the case: Atrial septoplasty in fetuses with hypoplastic left heart syndrome with intact or highly restrictive atrial septum is performed on one hand to enable pulmonary venous blood to reach the systemic circulation postnatally and on the other hand to alleviate severe pulmonary congestion by left atrial decompression toward the right side of the heart prenatally. The better outcomes in prenatally treated fetuses with this condition support the rationale that alleviation of pulmonary congestion improves abnormal muscularization of pulmonary veins and, by this mechanism, contributes to a lower risk of intractable pulmonary hypertension in these critically ill infants after birth.

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1. deAlmeida A, McQuinn T, Sedmera D. Increased ventricular preload is compensated by myocyte proliferation in normal and hypoplastic fetal chick left ventricle. Circ Res. 2007;100:1363–1370.

Key Words: fetal atrial septoplasty / pulmonary congestion / pulmonary hypertension
Fetal Atrial Septoplasty Is Performed for Left Atrial Decompression but not for Enhancing Preload to a Hypoplastic Left Heart

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_Circ Res._ 2007;101:e113
doi: 10.1161/CIRCRESAHA.107.164202

_Circulation Research_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/101/10/e113

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