Letters to the Editor

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Determinants of maximal right ventricular function: Role of septal shift

To the Editor:

I read with interest the article by Klima and associates[1] published in the January 2002 issue of the *Journal*. This work represents another valuable contribution by Vlahakes and his coworkers to the understanding of the pathophysiologic mechanisms that underlie right ventricular (RV) failure.

The model used in this study comprised an isovolumetric contracting right ventricle in which the preload was varied by means of incremental increases in volume of an intracavity latex balloon. Systemic arterial pressure and coronary perfusion were maintained by cardiopulmonary bypass support with or without left ventricular (LV) ejection. RV performance was assessed by maximum developed pressure during contraction.

From these experiments the authors suggest that ventricular interaction as opposed to RV free-wall perfusion is the most significant determinant of maximal RV performance. It is difficult to see how this conclusion can be reached from the data they present. In fact, they identified no significant difference in RV performance in terms of maximum developed RV pressure or RV dP/dt when the left ventricle was ejecting or completed offloaded. A myocardial perfusion–limiting phenomenon occurring at maximum RV volume appears a more likely explanation.

The authors found that, at the point of maximum RV preload, the maximum RV developed pressure is correlated with systemic arterial pressure (independent of LV loading) (their Figure 3). It is possible that at this point of RV dilation the increase in RV diastolic pressure is sufficient to critically limit RV myocardial perfusion and subsequent variation in the systemic pressure will produce a corresponding change in contractility. This would be consistent with the findings by Sunagawa and associates,[2] who identified a similar linear relationship between coronary artery pressure and the slope of the end-systolic pressure-volume relationship under conditions of critical perfusion. With increasing RV distention and rise in RV diastolic pressure, increased wall tension will occur with the potential for ischemia of the subendocardium (free wall and septal). In addition, the increased oxygen demand required at the higher developed RV pressure will contribute to myocardial ischemia when perfusion becomes marginal.

In the authors' Figure 4, maximal RV developed pressure increases with increasing RV balloon volume up to 60 mL and then declines thereafter (defined as the point of RV failure). This curve has the appearance of a Frank-Starling relationship whereby the performance of the ventricle is enhanced by the increased ventricular volume. Further increments in RV volume result in RV failure, indicated by a reduction in the maximum RV developed pressure. The authors' discussion does not clarify what the potential and likely mechanisms for RV failure are beyond this ventricular volume. This volume could represent the peak of the Starling curve, that is, the sarcomere length at which the maximum crossbridge/actin interaction occurs. Alternatively, it may result from inadequate myocardial perfusion. It is interesting right coronary artery flow decreased significantly after RV failure.

To gain further insight and in attempt to resolve these issues, the authors could easily construct an RV diastolic pressure-volume curve (this would have to be repeated by distending the balloon alone, so that the contribution of the balloon to the total compliance is determined). An acute increase in pressure-volume gradient with the higher ventricular volumes may indicate a reduction in RV driving pressure, which will reduce coronary artery flow[3] and render the subendocardium vulnerable to ischemia.

Furthermore, the authors identified that maximum RV developed pressure occurs at higher ventricular volumes if the left ventricle is ejecting rather than empty. LV ejection potentially may contribute two

benefits: First, the filling of the LV cavity during diastole will maintain RV geometry. (This was indicted in the article by the displacement of the septum with LV filling, Figure 5). This more physiologic shape in diastole may affect the compliance of the right ventricle with subsequent beneficial effects on wall tension and subendocardial perfusion. Again, the use of the diastolic pressure-volume curve may identify this. Second, LV ejection will add pulsatility to the systemic arterial supply, which may allow greater perfusion at marginal pressure gradients.

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Reply to the Editor:

My colleagues and I appreciate the questions raised by Danton in his letter to the Editor. The relationship between maximum right ventricular (RV) function and systemic pressure was suggested in 1955 by Peter Salisbury.[1] On the basis of this early study, we[2] demonstrated that, at the onset of RV failure, the RV free wall becomes ischemic, and this ischemia and failure can be reversed by raising systemic pressure. Although we believed that the entire matter was simply an issue of RV perfusion, additional studies from our laboratory began to cast doubt on this hypothesis. Page and associates,[3] using a model in which the right coronary circulation perfuses only the RV free wall, demonstrated that the maximal pressure developed by the right ventricle is determined primarily by maximal developed left ventricular (LV) pressure, and not by right coronary perfusion pressure. The issue of RV free wall versus left heart contribution was further explored by Damiano and colleagues[4] in a model in which the RV free wall was electrophysiologically isolated from the rest of the heart. They demonstrated two distinct contributions to global RV function: one derived from RV free wall contraction and one derived from contraction of the left side of the heart. On the basis of these studies, we undertook the study recently published by Klima and coworkers[5] to investigate further the nature of left heart contribution to RV function.

Putting together the findings of our recent study with the prior work has led us to conclude that approximately half of RV function is derived from the contribution of the free wall, which is dependent on perfusion pressure, combined with a significant contribution from the left side of the heart, that is, via the interventricular septum. Furthermore, in our study, even when no LV preload was introduced into the left ventricle, the ventricle still developed pressure because of blood returning to the left side of the heart through the thebesian vessels; in our preparation, peak developed LV pressure closely correlated with aortic pressure, irrespective of left heart output. Although we do not deny the role of RV free wall ischemia in the pathogenesis of RV failure, we do maintain that the process is complex with multiple determinants.

Irrespective of mechanism, our study supports the physiologic principle that developed LV pressure, and hence systemic pressure, must be maintained when treating patients with RV failure.

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Thymolipomas with myasthenia gravis in Japan

To the Editor:

With great interest we read the article, "Thymolipoma in Association With Myasthenia Gravis," by Zambudio and colleagues.[1] They reviewed 18 case reports about thymolipoma with myasthenia gravis, including 3 Japanese cases.

We also reviewed 14 case reports about thymolipoma with myasthenia gravis in 1993.[2] To our knowledge, there are 9 cases in Japan, including our new case (Table 1).[2-4] The patients comprised 2 men and 7 women, with a mean age of 50 ± 14 years (29-76 years). The mean thymolipoma weight was 385 ± 355 g (55-850 g). Thymolipomas with myasthenia gravis appear in older patients and are smaller than thymolipomas without myasthenia gravis (mean age 34 ± 18 years, mean weight 640 ± 650 g) according our previous review.[5]

As Zambudio and colleagues discussed in their comment, we think the best current surgical treatment for thymolipoma with myasthenia gravis is extended thymectomy.[2] We also think that extended thymectomy is the preferable surgical treatment for thymolipoma even without myasthenia gravis, since patients with thymolipoma may have a high titer of serum antiacetylcholine receptor antibodies.[5]

We thank Zambudio and colleagues for calling attention to this subject.

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