Left Ventricular Performance in Ischemic Right Ventricular Dysfunction

To the Editor:

Brookes et al reported decreased left ventricular (LV) systolic performance during right ventricular (RV) ischemia in the pig and concluded that “The hemodynamic compromise seen in association with acute RV dilatation within an intact pericardium is partly attributable to impaired LV systolic performance and cannot be wholly ascribed to changes in either LV preload or compliance.” They also stated that “… no studies to date have examined LV systolic performance in this context.”

However, my colleagues and I reported LV systolic and diastolic performance during ischemic RV dysfunction in the dog. This study was unfortunately ignored by Brookes et al. We demonstrated that ischemic RV dysfunction induced by right coronary artery (RCA) occlusion resulted in a significant leftward shift of the LV end-diastolic pressure-volume relation, which was markedly exaggerated by the presence of an intact pericardium. This finding agrees with that of Brookes et al.

More important, when we compared the indexes of LV systolic performance (ie, cardiac output, LV systolic pressure, peak LV dP/dt, and percent systolic shortening) during RCA occlusion with those during inferior vena caval occlusion at a matched LV end-diastolic volume, they were the same. This indicates that the primary mechanism of decreased cardiac output during RCA occlusion is caused by decreased LV preload (ie, end-diastolic volume) due to decreased LV chamber compliance rather than by decreased LV contractility. Because RCA occlusion in the dog causes isolated RV free wall ischemia, our results represent a pure RV ischemia model. In contrast, because occlusion of a dominant RCA in the pig probably causes ischemia in the inferior interventricular septum, the decreased LV contractility (ie, decreased slopes of both the preload-recruitable stroke work and end-systolic pressure-volume relation) seen in Brookes et al’s study may be explained by concomitant LV myocardial ischemia. Thus, the hemodynamic compromise seen in Brookes et al’s study is attributable to the combined mechanisms of decreased LV preload and acute LV ischemia.

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Response

We thank Dr Goto for his comments. The purpose of our study was to examine the effects of right coronary artery (RCA) ischemia on ventricular interaction in an attempt to understand the significant morbidity and mortality associated with this condition in humans. For this reason, we chose to use the potentially more relevant pig model instead of a dog model. Indeed, we verified that all the animals studied had a dominant right coronary system, as is present in 90% of humans. Thus, as explained in our article, the effects of right ventricular dilatation with an ischemic interventricular septum were an integral part of our findings.

We believe that this methodological difference, which clearly created a greater hemodynamic insult, explains the majority of the discrepancies between the 2 studies. This belief is supported by the fact that the left ventricular (LV) volume changes in response to RCA occlusion reported by Goto et al were very small (<4.1 mL) and that the RCA occlusion caused a paradoxical increase in LV systolic pressure when the pericardium was open, a finding that was not explained in their article. Furthermore, the interpretation of changes in maximum dP/dt and LV systolic pressure as indices of systolic performance has significant limitations given their preload and afterload sensitivity. We chose to examine relatively load-independent indices to separate out these confounding effects. Finally, the data presented by Goto et al do not systematically examine the systolic effects of right coronary occlusion in relation to pericardial integrity.

Goto et al should be commended for their detailed and elegant study on very subtle changes in end-diastolic pressure-volume relations; however, we believe that the issue of changes in systolic performance in a clinically more relevant preparation is more fully addressed in our article.

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