Furthermore, closure of the PDA occurred within 10 days in all of 5 infants demonstrating lower levels of sBNP (<1000 pg/mL), whereas closure occurred in only 1 of 3 infants with higher levels (>2000 pg/mL): One infant who required the largest dose of indomethacin finally required operative ligation for the PDA because it reopened (case number 8 in Table 1).

From these findings, we suggest that the hemodynamic severity of the PDA in addition to the size of the PDA should be taken into consideration to predict the possibility of successful medical treatment and to seek the timing of surgical intervention because no single parameter can be a complete index. The measurement of sBNP in the early postnatal days can be adjunctive and useful for these purposes because it is rather objective than the measurement of PDA diameter by USCG and requires only a small blood sample.

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MECHANISM OF MYOCARDIAL PROTECTION BY PRESSURE-VOLUME UNLOADING INVOLVES STRETCH-INDUCED PATHOPHYSIOLOGY

To the Editor:

I read with interest the recent report by Tamareille and colleagues,[1] who demonstrate in a porcine model of myocardial infarction that left ventricular (LV) unloading with a left ventricular assist device (LVAD) during early reperfusion is associated with both reduced endothelin-1 release by the myocardium and reduced infarct size. They demonstrate a positive correlation between early post-ischemia endothelin release and infarct size but admit that a cause–effect relationship was not established by this study. They found no effect of LV unloading on regional myocardial blood flow. Our laboratory previously demonstrated that LV unloading during early reperfusion ameliorates myocardial stunning in a canine model of regional ischemia-reperfusion.[2] As did Tamareille and colleagues, we found no differences in regional myocardial blood flow between LVAD-treated and untreated animals. By using ultrasonic dimension crystals, however, we observed marked ischemia-induced segmental stretch during both systole (paradoxical bulging) and diastole (creep), phenomena that were completely eliminated by LV pressure-volume unloading. We hypothesized that LVAD-mediated alleviation of abnormal stretch was responsible for improved contractile function. We later tested this hypothesis in an isolated guinea pig papillary muscle model,[3] in which we demonstrated that abnormal stretch caused contractile dysfunction similar to stunning. This dysfunction was abolished by gadolinium, a lanthanide cation that blocks a variety of stretch-induced perturbations in cardiac function through mechanisms that may involve specific stretch-activated ion channels. We then returned to the canine model and demonstrated that gadolinium attenuates regional stunning to the extent of mechanical LV unloading.[4] I would therefore suggest to the authors that the mechanism of infarct reduction with LV unloading observed in their study was not reduced endothelin release, but the elimination of abnormal stretch, which also happened to reduce endothelin release.

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