Figure 1. The opened wall of a large ascending aortic aneurysm. Note that the wall of the aorta has become so thin that the markings on a ruler can be read right through the aortic wall.

strain. In our echocardiographic studies on normal and aneurysmal ascending aortas, we have consistently found that the anterior wall of the aorta (the “greater curve,” so to speak) expands more than the posterior wall. These data were recently presented at the American Society of Anesthesiologists meeting in Las Vegas (October 23-27, 2004). This finding agrees with the mechanical concept that an unsupported tissue layer (the anterior wall) experiences more stress compared with a supported layer (the posterior wall, which is supported by the pulmonary artery and the associated central mediastinal fibrous tissues). These findings validate and support the importance of regional variations in mechanical properties of the aorta that Dr Sundt and colleagues have detected.

We share Dr Sundt’s skepticism regarding ruptured vasa vasorum as the cause of intramural hematoma. In fact, prompted by an insightful observation by Dr Kouchoukos some years ago, we have scrutinized the internal lining of the widely opened aorta during deep hypothermic arrest in cases of intramural hematoma. In most, but not all, cases one can find an intimal tear that was not appreciated even on high-quality preoperative radiographic images. In a small minority of cases, no intimal tear exists. It is specifically to explain such no-tear intramural hematomas that the concept of rupture of the vasa vasorum has been postulated. Also, our

echocardiographic measurements have confirmed Dr Sundt’s hypothesis that stress on the adventitia is greater than stress on the media. We agree with Dr Sundt that uneven distribution of wall stress across the layers of the aortic wall might play a role in the genesis of intramural hematoma. This thesis awaits scientific confirmation.

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Correlation of autologous skeletal myoblast survival with changes in left ventricular remodeling in dilated ischemic heart failure

To the Editor:

McConnell and colleagues¹ present a well-designed study with implantation of skeletal myoblasts into an infarcted area. The infarction was created by interventional closing of the circumflex artery, and this resulted in dilated ischemic heart failure. The results, however, were somewhat disappointing. As assumed in the editorial by

Tang,² the small number of surviving myoblasts could be one reason for this outcome. Of course, there might be several reasons for cell loss of transplanted cells, but one important issue has not been addressed by both authors: ischemia. The cardiomyocytes of the target area were lost because of insufficient blood supply. There is no reason why the transplanted cells should be more tolerant to ischemia than the native myocardial cells! Therefore the restoration of blood supply to an infarcted area is possibly one important measure to improve survival of transplanted cells. Additionally, further cell division and differentiation also depend on sufficient oxygen and nutrition supply.

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Intercostal muscle flap without increase of pain and blood loss after lung surgery

To the Editor:

We read with interest the article by Cerfolio and colleagues.¹ This report demonstrated that harvesting the intercostal muscle flap (ICM) reduces the pain of thoracotomy. We also have some data about the pain in comparison of cases with ICM harvesting and other cases without ICM harvesting (non-ICM), as in the study performed in our institution. ICM is very mobile and vascular rich. Therefore the ICM was used in the reinforcement of perforated esophagus or bronchopleural fistula.² In our series of patients, performing lobectomy with reinforcement of ICM to bronchial stump never resulted in bronchopleural fistula over the decade.

We had 35 patients who had ICM flap with lobectomy (ICM group) and 51 pa-