the borderline range of >13 mm" was in error. Six percent had a wall thickness of >14 mm as stated in the caption of Figure 2. The incidence of intraventricular septal thickness of more than 13 mm (14 to 16 mm) was 11% (17 of 156 subjects) as stated in the Results section. The incidence of either the intraventricular septal wall or left ventricular free wall being more than 13 mm was actually higher (13%; 20 of 156 subjects).

The findings presented in our study demonstrate the chamber size, wall thickness, and ejection fraction of elite professional American football players in the NFL. Because both the size and the types of physical effort of these players are different from those of bodybuilders, rowers, canoists, or cyclists, we believe these observations will be useful in separating physiologic adaptations from pathologic findings when evaluating such players.

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Cellular Myocardial Reconstruction Using Human Myoblasts

The Expedited Review from Pagani et al. (1) and the Research Letter from Hagege et al. in The Lancet (2) are the first reports that provide evidence about the fate of human myoblasts posttransplantation. Before these reports appeared there was no histological evidence on the viability and in vivo behavior of the human myoblasts after engraftment into the myocardium. These are significant findings and may provide clues to the unanswered questions impeding the progress in heart cell therapy using myoblasts. Moreover, these reports will provide impetus to the efforts in defining the basic mechanism underlying cardiac function improvement in postskeletal myoblast transplantation.

Pagani et al. (1) showed that transplanted myoblasts developed into myotubes and expressed human myosin heavy chain (slow isoform) in the myocardium. The myofibers originating from the myoblasts aligned in accordance with the heart muscle architecture. These results may resolve the controversy surrounding the milieu-dependent trans-differentiation of myoblasts into cardiomyocytes. The absence of connexin-43 expression, however, confirms the lack of electrical integration between the skeletal myofibers and the surrounding cardiac tissue. Without such linkage, it will be difficult for the cell graft to synchronize its contractility with the myocardium.

Considering the difficulties of using human subjects as a model, we developed a porcine heart model of myocardial infarction with immune tolerance in order to study the in vivo behavior of the human myoblasts and to define the underlying mechanism of global improvement in the left ventricular pump function. We observed the persistence of lac-z reporter gene carrying human myoblasts for up to seven months in the porcine heart (3). The xenografted myoblasts expressed human skeletal muscle myosin heavy chain (slow isoform) at the site of transplantation. Similar to the results obtained by Pagani et al. (1), we observed that the cells followed the geometric organization of the host tissue. An interesting finding of our study—based on histochemistry and fluorescent in situ hybridization analysis using a cocktail of probes specific for human and porcine chromosomes—is the presence of heterokaryons in the host myocardium resulting from the fusion between the donor myoblasts and the recipient cardiomyocytes. This hypothesis is substantiated by the long-term survival of the xenograft even after the discontinuation of immunosuppression (3). The observation of myofibers expressing both slow and fast isoforms of myosin by Hagege et al. (2) may be due to spontaneous fusion between myoblasts and the cardiomyocytes. We believe that together with other proposed mechanisms, formation of mosaic muscle fibers is the underlying mechanism in the improvement of cardiac function.

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