treatment. Nonetheless, among the subgroup of patients who underwent monitoring during both the initial treatment and study drug discontinuation periods (n = 163), ischemia was less frequently observed during *both* monitoring periods in the enoxaparin group (18.4% vs. 32.2%, p = 0.045 and 25% vs. 46%, p = 0.005, respectively). Further, the time to first ischemic episode was significantly earlier among UFH-treated patients, consistent with a superior *early* antithrombotic effect of enoxaparin and a reduction in the composite clinical end point of death, myocardial infarction (MI), and need for urgent revascularization (3).

Therefore, we believe that enoxaparin has been shown to be superior to UFH based upon the consistent and statistically significant reductions in the composite and double (death/MI) end points in ESSENCE and TIMI 11B (4). In contrast, "extending the duration and slower weaning" of UFH is an unproven method of administration, and, in fact, the cost-savings realized with enoxaparin ultimately make it the *less* expensive option (5).

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Failure of Right Ventricular Recovery of Fallot Patients After Pulmonary Valve Replacement: Delay of Reoperation or Surgical Technique?

We read with great interest and surprise the article published by Therrien et al. in the November issue of the *Journal* (1). These results are extremely disappointing. All the physicians familiar with the treatment of patients with repaired tetralogy of Fallot share the idea that one should not wait too long before implanting a valve in the right ventricular outflow tract of patients presenting with dilated right ventricle. Extremely dilated right ventricles might not benefit from valvulation as much as moderately dilated ones. However, it is extremely surprising that all patients operated on in a major center like Toronto showed neither clinical improvement nor regression of ventricular volumes after reoperation.

Although their patients were operated on quite late, we wonder whether their unexpectedly bad results might not be related to another reason than the delay for reoperation. The Toronto team has the peculiarity of implanting bioprosthetic material in the right ventricular outflow tract of these patients. Almost all patients received a stented bioprosthesis varying in size between 25 and 33 mm in diameter. It is noteworthy that true diameters of the outer rings are even larger than these measurements. The only way to implant these rather bulky bioprostheses at the level of the pulmonary annulus is to cover them with a patch extending from the main pulmonary artery to the infundibulum of the right ventricle. The immediate effect of this patching is to further increase the size of the right ventricular cavity, which may at least partly explain the fact that no decrease was observed in end-systolic and end-diastolic volumes of these patients after reoperation. We have recently shown that pulmonary insufficiency might not be the leading factor causing right ventricular dilation after repair of tetralogy of Fallot, and we suspect that the contractile function of the pulmonary infundibulum may play a role in the preservation of right ventricular function (2). Adding a patch to this already weakened area might further contribute to the deterioration of this function.

Like others, we believe that homografts are the ideal valve substitute for the right ventricular outflow tract because they offer a better effective orifice area, and they do not necessitate a patch enlargement. Although 40% of the patients presented in this series had an aneurysm resection, it is not clear whether the researchers believe that they effectively reduced right ventricular size at the time of the procedure.

In conclusion, we wonder whether the extremely poor results presented by Therrien et al. (1) might not be at least partially explained by their surgical technique rather than by the delay in the reoperation. The insertion of a patch in the already dilated area of the pulmonary infundibulum might further impede right ventricular function and as such increase rather than decrease right ventricular volumes.

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REPLY

We appreciate the opportunity to reply to the letter sent by Dr. d'Udekem and colleagues concerning the article we recently published in *JACC*.

Although we are grateful for their interest in our work, we would like to address the issues they have raised. First, their statement that ". . . all patients operated on . . . showed neither clinical improvement nor regression of ventricular volumes after reoperation . . . " is incorrect. As our article clearly stated, most of our symptomatic patients had a significant clinical improvement postoperatively (24% NYHA class $\geq$ III preoperatively vs. 0% postoperatively, $p < 0.001$) (1). Unfortunately, this could not be confirmed objectively (the same mean duration of exercise and external workload was achieved both pre- and postoperatively). Furthermore, although the mean right ventricular volume and function did not improve postoperatively, about one-third of our patients did show such improvement as depicted in Figures 1, 2 and 3 of our article (1). Unfortunately, the relatively small number of patients made it impossible to determine which of the many factors analyzed favored such a response. Finally, we disagree with the statement that "the . . . effect of this [bulky bioprosthesis] . . . is to further increase the size of the right ventricular cavity" . . . and impede contraction of the pulmonary infundibulum by virtue of its extensive transannular patching. They are correct that we do enlarge the outflow tract to accommodate as large a prosthesis as possible, but the patch to do this is largely from the annulus distally. Proximal to the annulus, the patch extends into the infundibulum a distance of 10 to 15 mm. We do not believe that a 10 to 15 mm incision below the pulmonary annulus would have an important impact on right ventricular volume and function. We do agree, however, that any incision in the right ventricle should be avoided or minimized as much as possible in order to potentiate maximal postoperative functional recovery.

The issue of pulmonary valve replacement in adults late after repair of tetralogy of Fallot remains a controversial one and we again thank Dr. d'Udekem and colleagues for their interest in our work.

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A Real Smoker's Paradox

The article by van Domburg et al. (1) in the September issue of the *Journal* presented detailed but not unexpected findings with regard to the hazards of cigarette smoking. However, I wonder whether the authors are aware that in Figure 2 of their paper, there is a graph which implies that the survival rate of cigarette smokers who quit after their bypass operation is significantly better than those who continue to smoke, but also significantly better than those who have never smoked. One would have to conclude from this data that the best chances of survival are among those who smoke up until the time of their surgery and then quit, rather than never to smoke at all. This would truly be a smoker's paradox if in fact it is correct!

I would appreciate some explanation from the authors.

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REPLY

We appreciate the comments of Dr. Shander regarding our recent article in *JACC* (1). In his comments he concluded that "the best chances of survival are among those who smoke up until the time of their surgery and then quit rather than never to smoke at all." We have proven that patients who quit smoking after bypass surgery are significantly better off than those who continue to smoke. However, in our study we only compared the patients who quit smoking with patients who continued smoking. Because of its irrelevancy, we did not compare the patients who quit smoking with patients who did not smoke. Furthermore, we did not use the term "never smoked" but used the term "nonsmoking." We did not distinguish between patients who have never smoked and ex-smokers (patients who had stopped smoking before the time of surgery), and we combined these two groups into one nonsmokers' group at the time of surgery. The smoking habits at the time of surgery did not influence survival during the follow-up period. This smoker's paradox is partly explained by the difference in baseline characteristics such as an age difference (smokers were four years younger than nonsmokers). Another explanation could be selection bias, as many smokers tend to die of fatal myocardial infarctions before they have the chance to undergo coronary bypass surgery (2).

Finally, the survival rates of the nonsmokers were probably positively influenced by the ex-smokers. In conclusion, the worse condition of the nonsmokers as compared with the smokers at the