cantly, only 2% had mild aortic insufficiency. Comparison of these two series is difficult because of the differences in population and etiology, but in the absence of prospective or randomized studies, we must base our decisions on extrapolation from current data. Thus there is no conclusive evidence that valve-sparing operations for patients with MFS have an advantage relative to Freestyle root replacement. Antimineralization treatment and zero-pressure fixation[8] make this valve more attractive for implantation than a homograft because of the lower rate of calcification with no significant changes in the elastic properties of the elastic wall,[8,9] and reoperation if needed is simpler with the Freestyle bioprosthesis because of lessened inflammatory reaction in the host tissues. A Freestyle root replacement with graft extension could be a reasonable operation for patients with MFS for whom anticoagulation is contraindicated or not acceptable. Further long term data are needed, however, before any of these operations can be recommended with certainty.

Carlos Del Campo, MD
Department of Cardiothoracic Surgery
Western Medical Center Anaheim and
University of California Irvine
Fullerton, CA 92835

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Amiodarone for postoperative atrial fibrillation

To the Editor:

We read with interest the article by Yagdi and colleagues[1] dealing with the use of amiodarone to prevent of postoperative atrial fibrillation (AF). In this prospective, randomized study, 77 patients (amiodarone group) received intravenous amiodarone during the first 48 hours after the operation followed by declining oral dosing over a 30-day period, and 80 patients (control group) received placebo. The authors observed a statistically significant reduction in the incidence of AF (10% in amiodarone group vs 25% in control group) as well as a significant reduction in the mean duration of AF (12.8 ± 4.8 hours in amiodarone group vs 34.7 ± 28.7 hours in control group).

We recently reported similar results in a prospective, randomized study conducted with 200 consecutive patients undergoing CABG.[2] The treatment group received oral amiodarone 4 hours after arrival to the intensive care unit and until hospital discharge. The incidence of AF was reduced from 25% to 12%, and its duration was also reduced.

These two prospective randomized trials constitute additional evidence for the efficacy of amiodarone in the prevention of AF after CABG. Interestingly, although in our series only the oral form of amiodarone was used, the results observed were almost identical to those reported by Yagdi and colleagues,[1] suggesting that the intravenous administration of amiodarone may not offer additional beneficial effects in preventing postoperative AF. If these observations were to be confirmed in future studies, the problem of the cost-effectiveness of the use of amiodarone in this setting, as alluded to in the editorial by Saltman,[3] would be completely resolved.

Alexander Yazigi, MD
Fadia Haddad, MD
Samia Madi-Jebara, MD
Department of Anesthesiology and Critical Care
Ghassan Sleilaty, MD
Victor A. Jebara, MD
Department of Cardiovascular and Thoracic Surgery
Hôtel-Dieu de France
Beirut, Lebanon

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Reply to the Editor:

There is no consensus about the optimal dose and route of amiodarone in prophylaxis against atrial fibrillation after coronary artery bypass grafting. In most of the studies we cited in our article,[1] intravenous amiodarone doses ranged from 10 mg/(kg · d) to 20 mg/(kg · d) through 2 to 8 days. Oral amiodarone doses ranged from 2.8 to 7.0 g through 7 to 20 days. We used relatively short-term, low-dose (10 mg/[kg · d], for 48 hours) intravenous administration, followed by oral tapered doses at a total of 9.0 g through 30 days. We prefer a combination therapy to take the advantage of accelerated loading time with the intravenous amiodarone and to obtain the incremental benefits of the oral amiodarone during the short-term intravenous administration.

We found that the postoperative administration of amiodarone was effective at significantly reducing the incidence of postoperative atrial fibrillation by 14.6%, the duration of atrial fibrillation episodes by 21.9 hours, and the ventricular response rate by 20 beats/min. In addition to the lower incidence of postoperative atrial fibrillation, the amiodarone group had sig-

nificantly fewer and less severe postoperative ventricular arrhythmias than did the control group. In addition, fatal ventricular tachyarrhythmia was not seen in the amiodarone group, whereas 2 deaths in the control group were related to ventricular tachyarrhythmia. The combined intravenous and oral amiodarone regimen used in this study was well tolerated.

We also found that amiodarone prophylaxis is cost-effective even when intravenous administration is used during first 48 hours. Intravenous amiodarone use did not require additional lines or extra intensive care unit stays. Furthermore, the amiodarone group had shorter hospital stays than did the control group (6.8 days vs 7.8 days). In our protocol, the total cost of the amiodarone was only a third the cost of a single day of hospitalization.

Tahir Yagdi, MD
Department of Cardiovascular Surgery
Ege University Medical Faculty
İzmir, Turkey

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Endotoxemia and cardiac function

To the Editor:

We read with interest the article by Aydin and colleagues[1] describing the degree of endotoxemia in patients undergoing on-pump versus off-pump coronary artery bypass grafting. The authors discussed the possible sources of endotoxin (lipopolysaccharide) during surgery, yet the effects of endotoxemia on cardiac function were mentioned only briefly.

Despite the similarity in outcomes between patients who underwent cardiopulmonary bypass and those who did not, we would like to point out that whereas endotoxemia is usually thought to increase cardiac output as a result of decreased systemic vascular resistance, it may actually have a significant negative inotropic effect. In cardiac surgical patients the combination of ischemia-reperfusion injury and myocardial endotoxin-mediated stress probably has a potent effect on myocardial function. The most commonly cited mechanism for this involves vascular as well as myocardial nitric oxide synthesis and takes several hours to reach functional relevance by disturbing intracellular calcium regulation and blunting contractile protein calcium sensitivity.[2]

Recently, we described an alternative mechanism that brings about lipopolysaccharide-induced negative inotropy within minutes. Cardiomyocytes were found to rapidly incorporate endotoxin, which activated the sphingomyelin-sphingosine signaling cascade through the release of tumor necrosis factor α and direct, paracrine membrane receptor activation.[3,4] Sphingosine directly inhibits calcium release from the sarcoplasmic reticulum, thereby reducing the amplitude of the calcium ionic transient and impairing systolic force generation. It is important to note that all the necessary signaling steps for cytokine-mediated negative inotropy in response to endotoxin stress can occur within the myocardium, without involvement of circulating cytokines. Studies that compare the inflammatory response to off-pump and on-pump surgery by measuring cytokine concentrations in blood often provide conflicting results, and the discrepancy between cytokine levels and outcome may be attributable to the lack of direct endotoxin measurements. The article by Aydin and colleagues[1] fills that gap.

Cardiac index, as measured by Aydin and colleagues,[1] is dependent on preload and afterload and therefore does not necessarily reflect the actual myocardial contractility. The increased lactate concentration, in part attributed to inhibition of pyruvate dehydrogenase, may also be interpreted as evidence for a compromised hemodynamic situation resulting from primarily impaired cardiac function. In this context, a more detailed, load-independent analysis of myocardial contractility might help to clarify the functional relevance of endotoxemia.

Christof Stamm, MD
Department of Cardiac Surgery
University of Rostock
Rostock, Germany
Douglas B. Cowan, PhD
Department of Anaesthesia
Children's Hospital Boston
Harvard Medical School
Boston, MA 02115

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Ascending aorta cannulation in type A dissection

To the Editor:

The Hannover group's approach to cannulation of the ascending aorta in the setting of type A dissection,[1] although potentially controversial, has clearly been successful and introduces an unconventional technique in a complex situation. I have used the same technique in 3 cases and also found it safe. I would suggest two additional maneuvers to enhance success. First, epiaortic echography is perhaps more precise in assessing the ideal site for cannulation of the aorta. Second, to be sure I am in the true lumen, I use a Seldinger technique. The USCI cannula (US Catheter and Instrument Co) can be placed over a wire previously passed into the aorta over a large bore needle. Precise positioning of the wire in the distal arch and descending aorta with transesophageal echocardiography ensures its accurate placement. The cannula is subsequently passed over the wire.

Alex Zapolanski, MD
San Francisco Heart Institute
Daly City, CA 94105

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