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.

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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.

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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.

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